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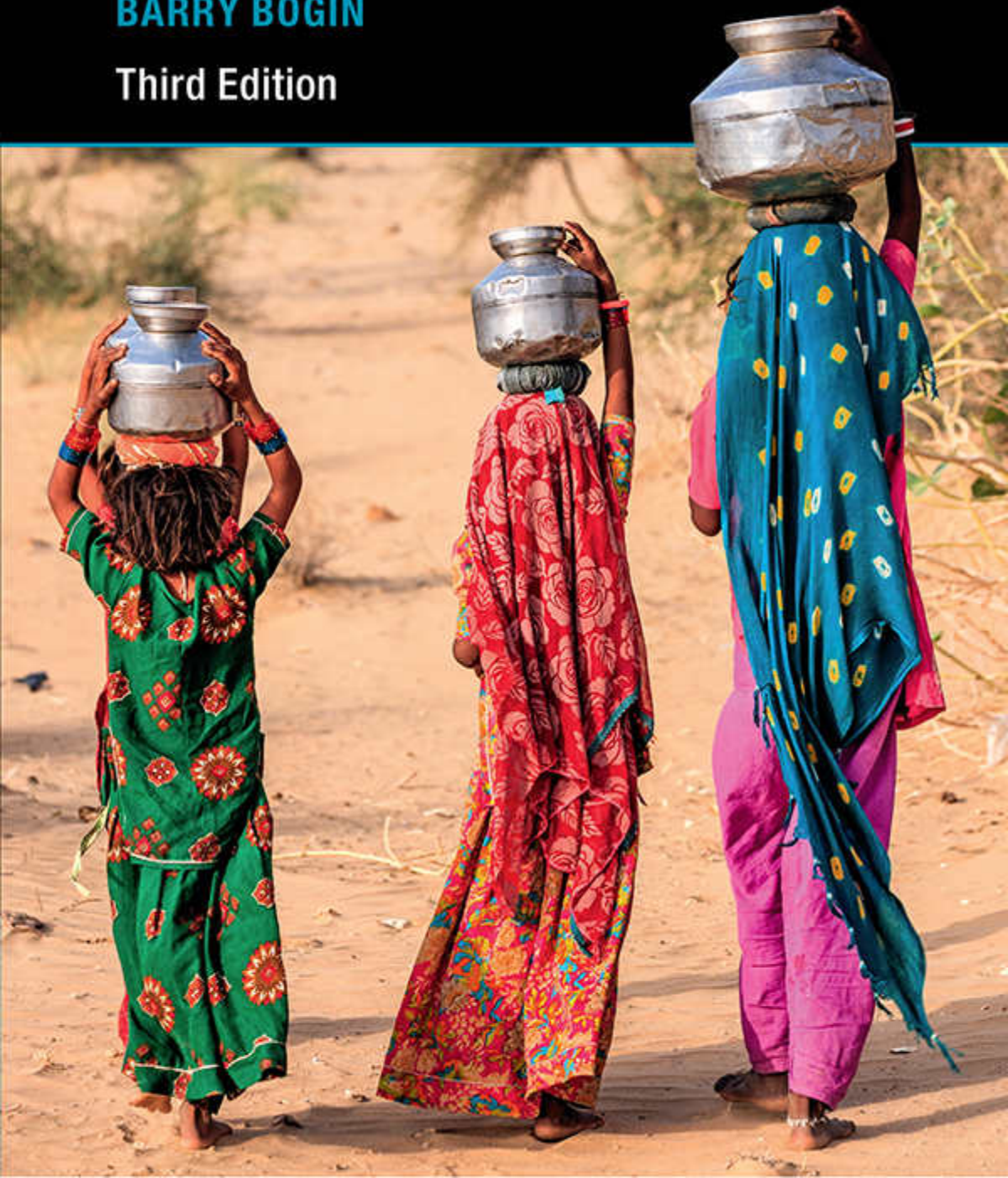
Patterns of Human Growth

BARRY BOGIN

Third Edition



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Patterns of Human Growth

Third Edition

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Dedicated to the memory of my mentor Gabriel Ward Lasker in admiration and eternal friendship.

Dedicated with love to my daughter Rachael and her daughters Anabella and Charlotte, my son Josh, and my daughter Isabel Jian-Ni. Wishing you comfort, learning, productive work, love for each other, and ever-lasting hope.

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Color plates can be found between pages 306 and 307.

Acknowledgments

This third edition of Patterns of Human Growth was a long time in coming. In fact, the 20+ years since the second edition is about equal to the time it takes a fertilized ovum to grow, develop, and mature to adulthood. I have matured a bit over those two decades. I moved from the University of Michigan-Dearborn, an outstanding undergraduate university in the United States, to undergraduate and doctoral training at Loughborough University in the United Kingdom. Over 12 years in the UK I supervised six PhD students who gave back to me much value by expanding my research experiences to new peoples and new places. I also retired from daily university life in 2018, after 42 years of my own post-PhD life. Retirement for me means that a university no longer provides a pay check, but I remain quite active in research, writing, attending, and presenting at conferences, and enjoying the privileges of an academic life.

I am very fortunate to have several colleagues who took time from their own research to read and criticize one or more chapters of this new edition, provide me with results of their own research, and engage with me in biological-anthropological-medical-historical-political-philosophical discussions. For their kindness, for their forthright comments which have improved the presentation of this work, and for their intellectual honesty and stimulation sincere thanks are extended to Drs. Christiane Scheffler, Michael Hermanussen, Will Johnson, and Liina Mansukoski. Liina was my final PhD student and was able to provide critical reviews from both the pre- and post-doctoral perspectives. Liina, Michael, and Christiane read every word of the third edition and contributed some of the final text. My gratitude for their support is beyond words. As must be said, the book you have before you is my responsibility and any errors are mine alone.

Many thanks are due to colleagues who adopted previous editions of this book for their teaching. A few of you, and occasionally some of your students, sent notes of support and helpful comments.

Since the publication of the second edition in 1999 I became a member of the *Center for Academic Research and Training in Anthropogeny* (CARTA). "Anthropogeny" is a word first used in the 1839 edition of Hooper's Medical Dictionary and defined as "the study of the generation of man." Today the word is defined as the investigation of the origin of humans. CARTA is an international and virtual organization, formed at the University of California San Diego and the Salk Institute for Biological Studies, to promote transdisciplinary research into human origins, drawing on methods from many disciplines spanning the social, biomedical, biological, computational and engineering, physical and chemical sciences, and the humanities. Professor Ajit Varki, one of CARTA's founders discovered me by reading *Patterns of Human Growth* and invited me to join. This new edition benefits from the learning provided at many CARTA symposia, both in person and online. I wish to

acknowledge the contributions of CARTA officers Drs. Pascal Gagneux and Margaret Schoeninger, CARTA administrators, especially Ingrid Benirschke Perkins and Linda Nelson, and the late James Handelman for financial and intellectual support.

Final thanks for this third edition are extended to my editors at Cambridge University Press, Dr. Dominic Lewis, Jenny van der Meijden, and Olivia Boult.

I also want to thank, again, those colleagues who helped with the first and second editions of this book, as many of their suggestions for improvement are still found in this third edition. For the first edition critical suggestions were provided by Drs. George Clark, Nancy Howell, Marquisa LaVelle, Michael A. Little, Daniel Moerman, Gerald Moran, Jessica Schwartz, and B. Holly Smith. The professional work of Professor Elizabeth Watts and Professor James Gavin inspired much of my interest in the evolution of human growth. Professor Watts read sections of the first edition and Professor Gavin engaged me in lively conversation about our disparate ideas about human evolution. Sadly, neither of these colleagues lived to see the drafts of the second edition. Nevertheless, their writings and conversations with me leave a mark on this book. Parts of the first edition of this book were written during a research leave from the University of Michigan-Dearborn. The author appreciates the assistance of Drs. Eugene Arden, Victor Wong, and Donald Levin in helping to arrange for the leave. A generous grant from the American Philosophical Society helped to defray research expenses at that time. The second edition was written without the benefits of a leave from teaching, nor special financial assistance. Of course, Cambridge University Press, in the person of my editor Dr. Tracey Sanderson, provided much support, even some money, but especially kind understanding of the inevitable delays in writing. Sandra Bogin contributed several of the line drawings that illustrate the text and, more importantly, provided physical and emotional support and encouragement for the writing. Professor Gabriel W. Lasker read every word of the draft of the text and engaged the author in many discussions about the intellectual and technical content of the book. His involvement in all aspects of the production of the book are appreciated deeply. Gabriel and his wife Professor Bernice (Bunny) Kaplan were my friends and mentors. The many hours I spent with them, in their home and elsewhere, were some of my most rewarding and pleasurable times. Gabriel passed away in 2002 and since then I have lost contact with Bunny. I hope she is well, and I hope she knows how important she was in my professional development.

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Introduction

It is the purpose of this book to describe and interpret some of the evolutionary, physiological, socio-cultural, and mathematical patterns of human **growth**[1]. Throughout, a **biocultural approach** is taken, one that tries to seamlessly meld scientific exploration of the relationships between human biology and culture. Anthropologists have many definitions of human culture. Older proposals viewed culture as the sum of human technology, sociology, and ideology. The observations by Jane Goodall on wild chimpanzees, and by many researchers inspired by Goodall on other primate species, changed that older view. The technology and sociology of many nonhuman animals differs by degree, not kind, from human capacities. Contemporary theorists tend to focus their definitions of culture on ideology, that is, the justification of behavior. To justify behavior, we humans give meaning and purpose to our existence. Human purpose seems unique to our species (Jolly 1999). Ideology and purpose encompass the beliefs, norms, and values of a social group, which are transmitted across generations by means of informal and formal teaching and learning (Boyd & Richerson 1985). Human purpose spurred the technological change from chimpanzee hammer stones to the laptop I am using to write this sentence. Purpose took people to a moon landing, but also to the Nazi extermination of 11 million Jews, Gypsies, homosexuals, Catholic priests, people with mental or physical disabilities, communists, trade unionists, Jehovah's Witnesses, anarchists, Poles, and others. Human purpose underlies the social change from genetically-based chimpanzee hunting parties to socially and ideologically defined human gatherer-hunters and agro-industrialists. Human purpose provides my rationale for revising this book to bring it more "up-to-date" in terms of both scientific fact and my own interpretations of the science and humanities literature about human growth.

Given my purpose for this book, the title requires some explanation. A cell biologist might think of the phrase "patterns of growth" in terms of a series of genetically controlled cell duplication and division events. An embryologist might think of patterns of cell differentiation and integration leading to the **development** of a functionally complete human. The clinician interprets patterns of growth, especially deviations from expected or "normal" growth, as evidence of disease or other pathology in the patient. Each of these concepts of "pattern" may be biologically valid and useful in their own areas of specialization, but this book is about none of

[1] Formal definitions for all words in bold type are found in the Glossary.

them. The goal of this account is to consider the growth of the human body in a unified, holistic, and anthropological manner. The result, it is hoped, will be a synthesis of the forces that shaped the evolution of the human growth pattern, the biocultural factors that direct its expression in populations of living peoples, the intrinsic and extrinsic factors that regulate individual development, and the biomathematical approaches needed to analyze and interpret human growth.

The study of human growth in relation to evolutionary biology, biocultural factors, intrinsic and extrinsic factors, such as genes, **hormones**, the physical and social environment, and mathematics may seem like a strange brew of topics. In fact, it is a common mix for biological anthropologists. The rest of this book is designed to show the reader that the anthropological blend of scholarship and research is, in fact, a practical and rewarding combination.

Introductory students of human growth often assume that the field is primarily a part of pediatric medicine. Indeed, until the publication of the first edition of *Patterns of Human Growth* in 1988, all but one of the leading introductory texts were written by physicians, and were written with the medical student in mind, or as a practical guide for parents. The one exception is the book *Child Growth* (Krogman 1972), written by a biological anthropologist, but focused primarily on pediatric topics. While it may seem logical for human growth to be a subfield of medicine, it is more accurate, however, to view pediatric medicine and "parenting" as subfields of the study of human growth. In turn, human growth is a part of a much broader discipline, namely anthropology. A little bit of history, and an applied example, are provided here to justify this statement. Chapter 1 includes a more detailed history of the study of human growth.

Anthropology and Growth

The study of human growth has been a part of anthropology since the founding of the discipline. European anthropology of the early to mid-nineteenth century was basically anatomy and **anthropometry**, the science of human body measurements (Boyd 1980; Tanner 1981). Early practitioners of American anthropology, especially Franz Boas (1858–1942), are known as much for their studies of human growth as for work in cultural studies, archaeology, or linguistics. Boas was especially interested in the changes in body size and shape following **migration** from Europe to the United States.

At the time of those studies, around 1910, most anthropologists and anatomists believed that stature, and other measurable dimensions of the body such as head shape, could be used as "racial" markers. The word "race" is set in inverted commas here because it refers to the scientifically discredited notion that human beings can be organized into biologically distinct groups based on **phenotypes** (the physical appearance and behavior of a person). According to this fallacious idea, northern European "races" were tall and had relatively long and narrow heads, while southern and eastern European races were shorter and had relatively round skulls. Boas found that, generally, the children of Italian and eastern European Jewish **migrants** to the

United States were significantly taller and heavier than their parents. The children of the migrants even changed the shape of their heads; they grew up to have long narrow heads.

According to Boas, in the new environment of the United States, the children of recent southern European migrants grew up to look more like northern Europeans than their own parents. Boas used the changes in body size and shape to argue that environment and culture are more important than genes in determining the physical appearance of people. Boas used the concept of biological **plasticity**, the responsiveness of the body to environmental change, to account for the changes in size and shape. In Chapter 2 I return to the concept of plasticity in more detail.

In terms of environment, life in the United States afforded better nutrition, both in terms of the quantity and the variety of food. There were also greater opportunities for education and wage-paying labor. These nutritional and socioeconomic gains are now known to correlate with large body size. In terms of culture, especially child-rearing practices, there were other changes. In much of Europe infants were usually wrapped up tightly and placed on their backs to sleep, but the American practice at the turn of the century was to place infants in the prone position. To be "modern" the European immigrant parents often adopted the American practice. One effect on the infant was a change in skull shape, since pressure applied to the back of the infant's skull produces a rounder head, while pressure applied to the side of the skull produces a longer and narrower head. The sleeping position effect on skull shape was demonstrated first in Europe by Walcher (1905).

There has been lively debate about the work of Boas and his colleagues (Gravlee et al. 2003; Sparks & Jantz 2002), but all agree that an interest in human growth is natural for anthropologists. This is because the way in which a human being grows is the product of an interaction between the biology of our species, the physical environment in which we live, and the social-economic-political-emotional (SEPE) environment that every human culture creates. Moreover, the basic pattern of human growth is shared by all living people. That pattern is the outcome of the four-to-seven million-year evolutionary history of the **hominins**, living human beings and our bipedal fossil ancestors. Thus, human growth and development reflect the biocultural nature and evolutionary history of our species.

Maya in Disneyland

The biocultural nature of human growth may be appreciated by examples based on my own research in Guatemala, Mexico, and the United States on the impact of the SEPE environment on the growth and development of **Maya** children. Because this research will be used throughout this book, I provide some background to the Maya people here and then move on to describe the results of the research examples.

The living Maya are the biological and cultural descendants of people inhabiting a geographic region extending from the Yucatan Peninsula of southern Mexico, through Belize, Guatemala, El Salvador, and western Honduras (Figure I.1).

Figure I.1 Map of Maya Culture region.

By about the year AD 250 a Maya cultural identity was well established. Maya society was organized in several state-level groups, each ruled by a priest-king or queen and an elite class of political-religious leaders. Each Maya state group maintained a workforce of peasants that produced food and provided labor for construction, as well as classes of artisans, military specialists, and bureaucrats for government administration. After the year AD 900, many Maya state societies declined, likely due to internecine warfare and overexploitation of natural resources. European contact and conquest in the year AD 1500, found the Maya living in chiefdomship societies, still building monuments, and still fighting each other. Europeans dismantled these societies via a combination of diseases, military action, taxation, and enslavement. By the year AD 1600, Maya population numbers plummeted from about 2 million in 1500 to about 200,000 (Lovell & Lutz 1996). Maya became the underclass of the new Colonial societies of Yucatan and Central America.

The Maya population recovered in size during the following centuries and today there are an estimated 7–8 million Maya living in Mexico and the Central America region (Lovell 2010). This makes the Maya the largest Native American ethnic group. Common features of rural lifestyle, economic activities, kinship and marriage systems, religion, philosophy, and a brutal history of repression since the Conquest of the Americas binds all Maya together into a shared cultural identity. There are, however, 30 or more Maya languages, each associated with a specific Maya group (see Wikipedia, Mayan Languages).

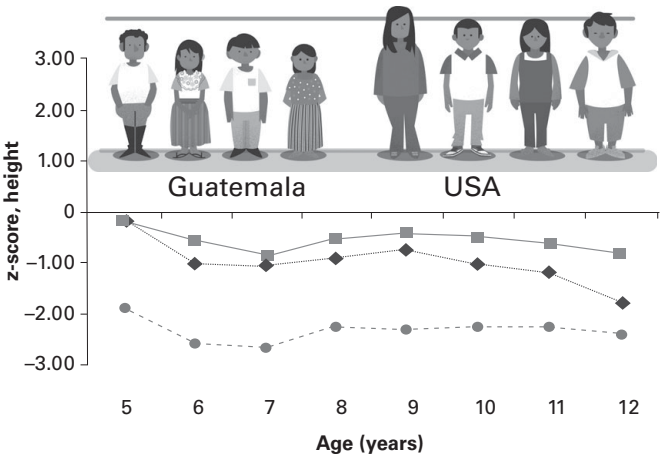


Figure I.2 Mean z-scores for height of Maya children from the GUATE-1998 (circles), USA-1992 (diamonds), and USA-2000 (squares) samples compared with the NHANES III reference means. From (Bogin et al. 2002). The illustrations are of 9-year-old Maya children (illustration copyright with Barry Bogin). The line indicates the average height of 9-year-olds according to the US growth references.

The following describes growth in height of three samples of Maya at the time of our fieldwork in 1992 and 2000 (Bogin & Loucky 1997; Bogin & Rios 2003; Bogin et al. 2002). One is a group living in their homeland of Guatemala, and the other two are migrants living in the United States. All three groups include girls and boys between the ages of 5 and 12 years old. Mean heights by age and sex are shown in Figure I.2. The heights are expressed as z-scores, that is, as a value indicating how many standard deviations a Maya mean height for a given age and sex differ from the mean of a reference for the same age and sex. The reference here is the Third United States National Health and Nutrition Examination Survey, 1988–1994 (NHANES III). This Survey included nationally representative samples of white (European-American), black (African-American), and Hispanic (mostly Mexican-American) people. The version of this reference published by Frisancho (2008) was used to calculate the z-scores.

The sample living in Guatemala are Maya schoolchildren (n = 1,347), measured in 1998 by an anthropometric team from Spain (Dr. Luis Rios of the National Museum of Natural History, Madrid kindly supplied these data). This sample is referred to as GUATE-1998. These children lived in the rural agricultural and fishing communities around Lake Atitlan in the western highlands of Guatemala. These Maya communities are of very low **socioeconomic status** (**SES**, see Box I.1 for definition of SES as used in this book) and many families lived below the poverty level. In these villages, basic human services, such as health care, safe drinking water, and supplementary food programs for women, infants, and children, were either very limited or totally absent. The incidence of infant and childhood morbidity and mortality from infectious disease and undernutrition was relatively high. Other deaths were due to military action and political repression, especially the civil war of 1960–1996. The

Box I.1 **Socioeconomic status**

Throughout this book there is discussion of the association between socioeconomic status (SES) with human growth, development, and **maturation**. In this book, and in the research literature, SES is often treated as a proxy for specific factors known to influence growth, such as nutrition, disease, and workloads. In this text box the concept of SES, as used in this book, is defined.

In its most widespread usage, SES is a concept devised by the social scientists, statisticians, and the governmental tax authorities[2] to measure some aspects of education, occupation, and social prestige of a person or a social group. One early usage was by the psychologist Raymond Cattel who claimed that the essence of socioeconomic status was the "prestige factor" (Cattell 1942). Prestige was derived from a person's occupation and was more important than income, property, or education, but prestige was highly correlated with these. Cattell (p. 300) wrote that, "Social status, in short, is a purely psychological entity. Such a statement must not be taken to mean that it is not real or that it cannot be measured or that it is not a precisely definable concept. It is to be defined and measured in terms of behavior, implying mental states behind behavior. The prestige of an occupation is resident in the minds of all people in the community and is to be measured by assessing their attitudes towards it at a given time." A key concept added by Cattell is that prestige, and its socioeconomic status, is measured by the people of a community – it is a community effect. The influence of community effects on biological growth is a theme of this book and is discussed in detail in Chapter 7.

In practice today, SES is measured in the wealthier industrialized nations by the years of formal education (schooling) and the occupation of an adult. In the United Kingdom the official classification is based on occupation only. In the lower-income nations, SES must often be measured by other criteria because formal education is unavailable to so many people and occupations are traditional sorts of farming or herding. In these traditional communities,

[2] Governments use socioeconomic status to target taxes, e.g., taxes on tobacco products and sugary drinks. In the United States, Mexico, and some other nations, people of lower SES tend to use more of these products. To discourage overuse, governments tax these products. There is another purpose of the taxes on these products – to increase revenue to the government. People of lower SES often earn less money and pay less income tax. Lower SES people purchase fewer expensive items such as houses, boats, and other luxury goods that are taxed at relatively high levels. As lower SES people make up the predominate share of the population (e.g., ~30% of the population in the USA is in poverty or near-poverty and 44% of the population in Mexico live below the poverty line) taxing the products that these low-income people use is one effective means to increase revenue. References: USA, Haymes et al. (2015); Mexico, www.telesurenglish.net/news/In-Mexico-7-out-of-10-Born-in-Poverty-Will-Die-in-Poverty-20180509-0008.html.

the size of land holdings or number of animals owned may be useful indicators of SES. Among the urban poor of the lower-income nations, the quality of the home, as indicated by the number of persons per room, the presence of running water and toilet facilities within the home, the ownership of various electrical appliances, and the type of cooking fuel used, are sometimes used as markers of SES. The SES of infants, children, juveniles, and most adolescents is ascribed to them based on the SES of their parents. Some societies have very rigid boundaries between people of different economic, educational, and occupational statuses. In these societies the boundaries establish well-defined social classes and a person's SES is, in many ways, constrained by that person's social class. Other societies allow varying degrees of mobility between social classes, and often that mobility is linked to the quantity and quality of formal education.

Examples of this relationship between education and social class exist today in the United States, the United Kingdom, Japan, and other WEIRD (Wealthy, Educated, Industrialized, Rich, Democratic) nations (Henrich et al. 2010). Graduates of certain prestigious universities are selectively employed by the largest corporations, by governments, and associate with the wealthiest individuals of their society. Many of the elite university graduates themselves come from well-to-do families, and therefore retain the social class of their parents. This has been well documented in the United Kingdom (Clark & Cummins 2014, discussed further in Chapter 7). Fewer students of elite universities were raised in middle SES or lower SES families, but some of these fortunate few may eventually supersede the social class of their parents. A popular cultural myth of the WEIRD nations is that they are relatively open societies, with equality of opportunity for upward and downward social mobility. But, empirical research shows this is not true and over the past 100 years there has been less social mobility of the lower SES groups toward the wealthiest, higher social status groups. This trend toward greater SES inequality intensified with the 2007–2008 global economic crisis[3] and is reported for most of the nations of the world (Marmot & Bell 2012; Sen 1999; Wilkinson & Pickett 2009b).

In all societies, whether totally rigid or relatively more open to social movement, social class and SES are powerful influences on human physical and psychological growth and development (Batty et al. 2009; Bogin et al. 2017; Mansukoski et al. 2019b; Steckel 2012; Wilkinson & Pickett 2009a). Conversely, stature, **body composition** (fatness and muscularity), and rate of maturation influence the social, emotional, and economic status of children, youth, and adults. An appreciation of these **synergistic** and seamless biosocial interactions between growth and socioeconomic status has been realized only

[3] www.oecd.org/social/inequality.htm

in the past 200 years and are still being researched today. Some of the history of findings, their conventional explanations, and novel mechanisms for SES biosocial interactions are explored in the chapters of this book.

residents of the Lake Atitlan region were caught up in the military hostilities of that time and suffered some of the worst atrocities of the civil war.[4] They also suffered from reduced food availability due to the collapse of the Guatemalan economy during the 1980s and a cholera epidemic of the early 1990s (Bogin & Keep 1999).

The Maya children in the United States are the offspring of Maya adults who emigrated from Guatemala, mostly from the late 1970s to the early 1990s. All the adult Maya refugees were born in Guatemala and prior to migration most lived in rural villages in the Q'anjob'al-speaking language area (northwest Guatemala highlands). My colleagues and I analyzed data for the height of children measured in 1992, called the USA-1992 sample (n = 211). About 50% of the children in the 1992 sample were born in the United States and the remainder were born in Guatemala or Mexico. All had lived for at least two years in the US and there was no significant difference in height between those born in the different countries. We also measured the heights of Maya children in the United States in 1999 and 2000 – the USA-2000 sample consisting of 431 Maya American children, 93% were born in the United States. There were approximately equal numbers of girls and boys in all samples and no statistically significant differences in height, so sexes were combined for analysis.

The Maya families in the United States lived in two communities, one in rural Florida and the other in Los Angeles, California – not so far from the theme parks that name this section "Maya in Disneyland." There were no differences in height between the two communities. In both locales, adult Maya worked as day laborers in low paying jobs. Some Maya worked as teacher aids, nursing aids, or had small businesses such as grocery stores. All the Maya-USA children in the sample qualified for free breakfast and lunch programs at the schools they attended. The Maya-USA samples, although of low SES for the USA, lived under much more favorable conditions for growth and development than did the Maya sample in Guatemala. The Maya-USA children benefited from safe drinking water, medical screening at the schools, medical care in their communities, and supplementary feeding programs. The parents of the Maya-USA children capitalized on the economic prosperity in the United States of the 1990s via relatively steady employment and freedom from state-sponsored violence.

[4] See Cultural Survival Quarterly Magazine, 1991, Massacre In Santiago Atitlan, www.culturalsurvival.org/publications/cultural-survival-quarterly/massacre-santiago-atitlan-turning-point-maya-struggle.

The growth in height (as well as weight, fatness, and muscularity which are not shown here) of Maya children living in Guatemala was significantly retarded compared with the NHANES reference data (Figure I.2).

At all ages the mean z-score for height is near or below −2.0. When average heights of a group of children are less than −2.0 z-scores, that group is considered stunted, that is, within the shortest 2.3% of a growth reference or growth standard.[5] In the 1980s and early 1990s, some researchers argued that the small size of impoverished, and likely malnourished, communities such as the Guatemala Maya is a genetic adaptation to their poor environmental conditions. Being small protected them against the need for even more food to support larger bodies. If this argument were true, then a change in the economic, social, or political environment would not influence growth. The notion that the small size of the Maya is primarily genetic is clearly wrong, for as also shown in Figure I.2, the USA-resident Maya averaged about −1.0 z-scores. While still short in comparison with the US reference, these Maya were significantly taller than Maya children living in Guatemala. We also reported that Maya children in the United States attained virtually the same weight as the US reference due to increased fatness and muscularity.

Our analysis indicated that the USA-1992 sample was, on average, 8.9 cm taller than the GUATE-1998 sample. For the USA-2000 sample, the height difference increased to an average of 11.5 cm. These increases occurred within single generations, that is, as the parents of the children moved from Guatemala to the United States. These are the largest such increases in mean height between migrant children and **sedentes** (those remaining in their homeland) ever reported in the literature. To place this in context, the immigrant children measured by Boas averaged about 2.0 cm taller than their sedente counterparts back in Europe.

Even more impressive is that of the total height increase for the USA-2000 children, 4.7 cm was due to greater sitting height (length of the trunk, neck, and head of the body) and 6.8 cm was due to greater leg length. The growth of the legs of infants and children seem to be more sensitive to the environment than growth of the upper body – a topic that is explored in more detail in Chapter 5. That the Maya children in the United States changed in shape, as well as growing taller, is more evidence of Boasian biological plasticity.

The reasons for the increase in body size of the Maya children are similar to those for the European immigrant children measured by Boas. In the United States there is greater food security, which means not only more food and a greater variety of food than in rural Guatemala, but more importantly the reliable access to a sufficient quantity of affordable food. The public supply of safe drinking water in the United States eliminates the constant exposure to bacteria, parasites, agricultural pesticides, and fertilizers that contaminate drinking water in rural Guatemala. The Maya-USA children benefit from social services that are unavailable in rural Guatemala, including food supplementation programs such as free school breakfast and lunch for low

[5] See www.who.int/childgrowth/en/ for World Health Organization growth standards.

income students and free or subsidized health care. These services improve the biological, social, and emotional environment for human growth. The emotional/psychological environment, discussed in later chapters, is as powerful an influence on physical growth as any nutrient or illness. Part of the emotional/psychological difference in the United States is the absence of overt political repression and the threat of military violence. To be sure, most of the Maya families in the United States were low income and many parents were undocumented and without legal status or citizenship. These impose economic and emotional stress on all members of the family, as parents could be seized and deported (children of immigrants born in the United States are given citizenship). Many families in Los Angeles lived in high-crime neighborhoods. Even so, the relative security and political freedom for Maya in the United States allowed parents to pursue their goals for the healthy growth and development of their children.

Parents around the world share highly similar goals for their offspring. Robert LeVine (1988), an anthropologist of the family and of children, proposed a universal evolutionary hierarchy of human parental goals. The primary goal is to encourage the survival and the health of a child. Secondary goals relate to developing the child into a self-supporting adult and instilling cultural beliefs and behavioral norms. Economic and political conditions in Guatemala make it difficult for parents to achieve these goals for their children. The political economy of the United States offers real possibilities for success, and Maya parents seize upon these, just as other immigrants have done before them.

As Boas argued for nearly 50 years, the study of human growth provides a mirror of the human condition. Reflected in the patterns of growth of human populations are the "material and moral conditions of that society" (Tanner 1987). The forces holding back growth in Guatemala are severe indeed, and the growth differences between Maya of Guatemala and the United States may be used as a measure to assess the magnitude of change in SEPE conditions.

Growth and Evolution

The pattern of human growth serves as another type of mirror; one that reflects the biocultural evolution of our species. **Biological evolution** is the continuous process of genetic adaptation of organisms to their environments. **Natural selection** determines the direction of evolutionary change and operates by **differential mortality** between individual organisms prior to reproductive maturation and by **differential fertility** of mature organisms. Thus, genetic adaptations that enhance the survival of individuals to reproductive age, and that increase the production of similarly successful offspring, will increase in frequency in the population over time.

Human ideology and purpose also evolved and interacted with genetic adaptations. The combined biocultural evolution produced the pattern **of growth and development** that converts a single fertilized cell, with its full complement of **deoxyribonucleic acid (DNA)** and cytoplasm, into a multicellular human organism composed of hundreds of different tissues, organs, behavioral capabilities, and

emotions. That process is no less wondrous when it occurs in an earthworm, whale, or human being. Indeed, many growth processes that occur in people are identical to those in other species and attest to a common evolutionary origin. The discovery of PAX6, a "master-control gene" for of the eye (Halder et al. 1995) common to species as diverse as marine worms, squid, fruit flies, mice, and people, is powerful evidence for the common evolutionary origin of the eye. The PAX6 protein is thought to activate other genes involved in the formation of the eyes, as well as the brain and spinal cord, and the pancreas.[6] Other organs, and the genetic mechanisms that control their growth and development, also are shared among diverse species (Chapters 4 and 6 discuss the evolutionary biology and genetics of growth in greater detail). Nevertheless, some events in the human **life cycle** may be unique, such as distinct childhood and adolescent growth stages (discussed in later chapters), and attest to ongoing evolution of our species.

Dobzhansky (1973) said that "nothing in biology makes sense except in the light of evolution." Human growth, which follows a unique pattern among the mammals and, even, the primates, is no exception to Dobzhansky's admonition. A consideration of the chimpanzee and the human, two closely related (genetically) extant primates, shows the value of taking an evolutionary perspective on growth. Thomas Henry Huxley (1825–1895) carried out careful anatomical studies and demonstrated many anatomical similarities between chimpanzees and humans (Huxley 1863). Mary-Claire King and Allan C. Wilson (1934–1991) proposed in 1975 that such anatomical similarities are likely due to a 99% identity of the alignable structural DNA sequences of the two species. The completion of the human and chimpanzee genome projects shows, however, " . . . that closer to 5% of the total DNA in the chimpanzee **genome** differs from that of humans, if one considers the DNA that is missing in either species but present in the other" (Varki & Gagneux 2017, p. 152). Still, the genomic similarity is striking given the many differences in phenotype.

One interpretation of King and Wilson's findings is that the differences in size and shape between chimpanzee and human are due to the regulation of gene expression, rather than the possession of unique **genotypes**. Of course, humans are not descended from the chimpanzee, but both species did have a common ancestor some five, or more, million years ago. During evolutionary time, mutations and selective forces were at work on the descendants of this ancestor shaping their genomic constitution and its expression in their phenotypes. A systematic and transdisciplinary search for the evolutionary reasons for human–"great ape" (chimpanzee, bonobo, gorilla, and orangutan) similarities and differences was organized several years ago. It is called comparative **anthropogeny** (Varki & Gagneux 2017).[7]

One way that alterations in gene regulation produce anatomical differences between human and chimpanzee is via changes in growth rates. D'Arcy Thompson (Thompson 1917, 1942) showed that the differences in form between the adults of various species may be accounted for by differences in growth rates from an initially

[6] https://ghr.nlm.nih.gov/gene/PAX6 [7] See also https://carta.anthropogeny.org/moca/.

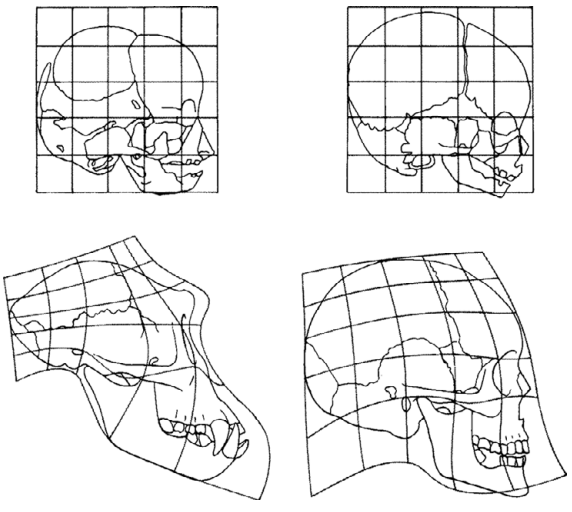


Figure I.3 Transformation grids for the chimpanzee (left) and human (right) skull during growth. Fetal skull proportions are shown above for each species. The relative amount of distortion of the grid lines overlying the adult skull proportions indicate the amount of growth of different parts of the skull. (Inspired by the transformational grid method of D'Arcy Thompson 1917, and redrawn from Lewin 1993)

identical – one might better say "similar" – form. Thompson's transformational grids (Figure I.3) of the growth of the chimpanzee and human skull from birth to maturity show how both may be derived from a common neonatal form. Different patterns of growth of the cranial bones, maxilla, and mandible are all that are required to produce the adult differences in skull shape. Of course, the differences in skull growth are related to size and shape of the brain, and size of the dentition (both species have the same number and types of teeth). In a similar manner, the differences in the post-cranial anatomy between chimpanzee and human being result from unequal rates of growth for common skeletal and muscular elements.

Despite the anatomical and biochemical evidence for the evolutionary origins of the human growth pattern, most works on human growth give little consideration to this topic. A paragraph or two is all that may be found in the current physiological and medical texts devoted to human growth. I am happy that since the publication of earlier editions of this book, many textbooks in biological anthropology and evolutionary medicine do give space and emphasis to the evolution of the human pattern of growth. In this new edition of *Patterns*, Chapters 3 and 4 are devoted to an account of the evolution of mammalian and especially nonhuman primate and human patterns of growth. Moreover, the theme of evolution runs throughout this book because it is the evolutionary perspective which makes sense of the rest.

Growth Theory

This is also a book about a theoretical approach to the study of human growth. The literature in the general area of animal growth is rich in both hypothesis testing and

theory (Bertalanffy 1960; Bonner 1965; Brody 1945; Goss 1964, 1978; Huxley 1972; Snow 1986; Thompson 1917; Weiss & Kavanau 1957). By contrast, until the year 1980 only a few workers had published hypotheses about the course and regulation of human growth (a few examples are Bogin 1980; Bolk 1926; Frisch & Revelle 1970; Gould 1977; Grumbach et al. 1974; Tanner 1963). Examples of some important contributions to growth theory, and some of its impact on later research, are given in Box I.2.

Until about 1970, most research and writing about human growth and development was descriptive and atheoretical. A typical anthropological or public health study would be based on a sample of children, youths, or adults measured for some variable(s) (height, weight, etc.). The data would be presented, described, and compared to similar data for another group of people. Or, in the fields of medicine and psychology, an unusual case study of growth, perhaps resulting from physical or psychosocial pathology, was described. In another realm, growth data derived from statistically representative samples of human populations were used to construct references and standards of height, weight, and other physical dimensions. Growth standards and references have value in public health work to assess the growth, development, and nutritional status of populations that are "at risk" for growth failure or malnutrition, and to monitor the effectiveness of intervention programs designed to improve the health and growth status of such populations.

Historically, most disciplines begin with this sort of descriptive phase. Human growth research was no exception, as documented in two books that detail the historical development of the field (Boyd 1980; Tanner 1981). One sign of a maturing discipline is when it begins to develop hypotheses to examine the nature of the processes that account for the descriptive data. An early example of the use of hypotheses in growth research is the classic series of studies by Boas (Boas 1912, 1922) on the growth of the children of migrants to the United States, as discussed earlier. The scholarly value of the research lies in Boas's use of the scientific method. Boas challenged the dogmatic belief that physical types, or "races," were fixed and biologically distinct. His hypothesis was that a change in environment brought about by migration from impoverished urban and rural areas in Europe to urban centers of the United States would bring about alterations in the amount and rate of growth. The value of his hypothesis-testing method lasted, and is still being used in migration research to test the relationship between migration history and human biology (Bogin & Loucky 1997; Bogin et al. 2014c; Mascie-Taylor & Krzyżanowska 2017; Mascie-Taylor & Lasker 1988).

Mature sciences are noted for their ability to synthesize several hypotheses that have been verified independently into one or more comprehensive theories that can explain the known data and, in turn, indicate the kind of observations that should be made in further research. Despite the early work of Boas, hypothesis testing in human growth research was only sporadic until the late 1960s. Since that time, the relationships between nutrition and growth, physical development and chronic disease, and environmental stress and growth, among other topics, began to be actively pursued.

Box I.2 **Models of the regulation of human growth**

A Conceptual Model of Growth

In 1963, James Tanner proposed a conceptual **model** for the biological regulation of normal human growth. By "normal" it is meant nonpathological growth. Chromosomal pathology, such as Klinefelter syndrome, other genomic mutations, chronic infectious disease, prolonged emotional trauma, and other pathology results in different patterns of growth, development, and maturation. Basic elements of Tanner's model are represented in Figure BI.2.1. A major feature of this model is that growth is target seeking and self-stabilizing. The curve labeled "Time-Tally 1" represents a hypothetical mechanism that provides a "target size" for body growth and keeps track of biological time during infancy and childhood. Biological time is measured in units of maturation, with the clock started at conception and stopped when some functionally mature state is reached.

The curve labeled "Inhibitor" represents the concentration in the body of a hypothetical substance, perhaps a by-product of cell division or protein synthesis, that acts upon the time-tally to regulate growth rate. The amount of

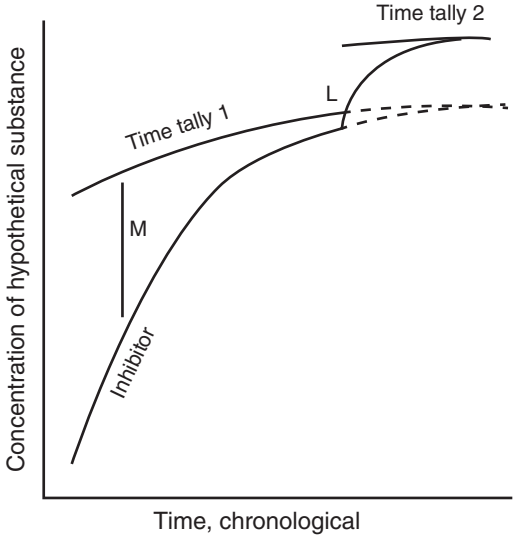


Figure BI.2.1 Tanner's conceptual model for the regulation of human growth. Rate of growth is determined by the amount of mismatch (M) between the concentration of a hypothetical inhibitor substance and a time-tally. Time-Tally 1 controls growth during childhood. Time-Tally 2 controls growth during adolescence. At point L, the switch between time tallies occurs, and the **adolescent growth spurt** is initiated. (Original drawing, based on Tanner 1963)

mismatch, "M," between the two curves determines the rate of growth at each chronological age.

Tanner's model accounts for the deceleration of growth velocity during infancy and childhood and explains the phenomenon of **catch-up growth** (Prader et al. 1963) following serious illness or starvation. During the normal postnatal growth of an infant, there is a large mismatch between the size the infant might attain and its actual size, and growth rate is rapid. As the infant and child grows, the amount of mismatch decreases, and the concentration of inhibitor increases. As a result, the rate of growth slows. Under nonnormal conditions, for instance starvation, the rate of growth slows or stops during the period of insult. The concentration of inhibitor remains constant during this time as well. The time-tally continues to register the mismatch between actual size and target size. When the insult is removed, in this case upon refeeding, there is a rapid increase in growth rate to restore the balance between the time-tally, the expected amount of mismatch, and the concentration of inhibitor. When this balance is restored the rate of growth assumes the normal velocity for that child, as if the insult had not occurred.

To account for the abrupt change in the velocity of growth that occurs at adolescence, Tanner suggested the existence of a second time-tally. "Time-Tally 2" (Figure BI.2.1) operates in the same manner as time-tally 1, but both the mechanism controlling the new tally and its inhibiting substance are assumed to be distinct from the old one. Tanner believed that the switch to the new tally occurs when a minimum velocity of growth, or minimum mismatch, on the old tally is reached. This is labeled point "L" in Figure BI.2.1. After the switch occurs, a new larger mismatch is established and a rapid increase in growth rate results. This is the adolescent growth spurt.

As the mismatch is reduced, and the concentration of inhibitor is increased, the rate of growth slows once more. Variation in the timing of the adolescent spurt between individuals is explained by changing the point at which the switch between tallies takes place.

This model is as conceptually stimulating today as when it was first proposed. Early in my career (Bogin 1980), I published a paper that built on Tanner's model by incorporating the, then popular, mathematical concepts of catastrophe theory. Since 1980, much more has been learned about the biological nature of the growth promotors (Tanner's time-tallies) and the inhibitor substances that regulate growth both before and after **puberty**. This research is reviewed in Chapter 7.

Where Is the Growth Regulator Located?

A complete and exact knowledge of the biological mechanisms involved in the regulation of amount of growth and rate of maturation are unknown. Clinicians treating children with growth disorders often report a strict association between increase in length/size and increase in maturity. Those who do not

increase in height, be it due to illness, starvation, or emotional trauma, also do not increase in bone age. Successful treatment that promotes growth usually also succeeds in increasing the biological rate of maturation, so that in the end there is no or only little effect on final height – these patients just reach final height at an earlier age than untreated patients who, in time, overcome their growth pathology. This tight linkage between size and maturity suggests that the growth and the maturation regulator are either located in the same tissue or organ, or if separate entities they are in intimate communication with each other. Tanner's model proposed that the control of amount of growth is related to the concentration of a hypothetical inhibiting substance that acts on a time-tally located in the brain, perhaps in the **hypothalamus**. Goss (1978) suggested that overall size of the body is regulated by a genetically programmed amount of growth for certain visceral organs. For instance, the heart or the kidneys may grow to a predetermined size and the functional limits of these organs to support the operation of other organs and systems of the body may determine the size to which these tissues and the body, as a whole, may grow. Snow (1986) interpreted the results of experimental studies of growth-controlling mechanisms to support Goss's hypothesis. However, Snow found that growth control in a developing organism is evident before **organogenesis** occurs during embryonic development. This fact, Snow suggested, indicates that control may lie in a tissue that differentiates relatively early in development and is distributed widely throughout the body, rather than in the visceral organs. A tissue composed of cells derived from the neural crest of the **embryo**, such as the central nervous system, was Snow's choice of a likely tissue for this function.

More recent research, some with insects, adds a further complication to the question of how body size is regulated. Nijhout and Emlen (1998) experimented with the growth of body parts in buckeye butterflies and dung beetles. In one experiment they removed embryonic cells that would have developed into the hind wing of butterflies. The adult butterflies' fore wings grew to be significantly larger than expected. Experiments with beetles manipulated the size of their horns by exposing them to hormones that were known to shrink or enlarge horn size. As larger horn size was induced, the size of the eyes decreased, and vice versa. These findings indicated that there is an interaction between body parts in their growth. A greater amount of investment in the size of one part may limit the size of another part. If this is true, then there is no simple genetic determination of the final size and shape of an organism.

Epigenetic (meaning "above" the level of the DNA sequences of the genome) marking of the genome, micro-RNAs, and other elements that were largely unknown to Tanner and Snow may also provide important mechanisms for the regulation of gene-by-environment interaction and phenotypic plasticity in adult height (Simeone & Alberti 2014). **Epigenetics** is the study of **heritable** changes in gene function that do not involve changes in the DNA sequence

(more detail on epigenetics is provided in Chapter 6). Rather, chemicals that interact with the DNA, such as presence of histones or methyl groups, inhibit or enhance the expression of DNA sequences. Still unknown is how the epigenome (the factors that influence gene expression) is marked and regulated to promote a target height for a population or group of people (Simeone & Alberti 2014). What is clear is that there is unlikely to be a single, central time-tally or size-regulating mechanism. Instead, animals may be able to adjust their final size and shape during development in response to many stimuli, both internal and external to the body. One proposal is that competition for social status and other community effects may be a key player in the setting of target height. Evidence for this type of **strategic growth** has accumulated in the past few years and is reviewed in Chapter 7.

Despite the hypothesis-testing purpose of science, a dreadful number of purely descriptive growth studies are still produced today.

One valid reason for the paucity of hypothesis testing and experimentation in human growth research in the past and today is the ethical dilemma. In nonhuman animal research, the genetic and environmental determinants of growth may be studied in the laboratory. The growth of the entire organism, anatomical regions, or even isolated populations of cells may be studied with great precision. Using human beings in experimental research is almost always unacceptable ethically. It is even illegal to use human tissues in research in some countries. Moreover, laboratory-controlled experimentation is conceptually unrealistic for many questions relating to human growth. Normal human growth and development require the complexities of a normal social and cultural milieu.

Despite the needed ethical and legal safeguards, students of human growth and development are not restricted to descriptive studies. Human growth may be studied with the same intellectual excitement, experimental approach, and theoretical research as is done by laboratory biologists. Creative and rigorous field research that makes use of "natural experiments" can be employed. A few examples are the studies of migration of southern Europeans or Maya to the United States mentioned already and research with young people who survived the horrors of World War I and World War II (e.g., Hermanussen et al. 2018a; Ravelli et al. 1976). These natural experiments, in combination with powerful new statistical and computational methods, the study of pathological growth disorders, and controlled experimentation within allowable and ethically justifiable limits, can be used to achieve a sophisticated understanding of human growth.

Advances in technology are also forging a new conceptualization of growth. Scientific advances in fields from tissue engineering to computerized imaging allow for human growth research that can proceed without some of the ethical and moral limitations that restricted experimentation on people. Molecular biology has reached

the stage where the biochemical substances that control growth and development can be identified, their DNA sequences decoded, and synthesized. Using CRISPR-CAS9 technology that was first proposed by Jennifer Doudna and Emmanuelle Charpentier (Jinek et al. 2012), DNA sequences may be edited to add desired DNA or eliminate undesirable DNA. An early breakthrough was the invention of the polymerase chain reaction by Kary Mullis in 1983 (Mullis et al. 1986), which allowed for the nearly unlimited duplication of segments of DNA. This led to the identification of regions of the genome that code for structural proteins and regulatory elements for growth, development, and maturation; so-called master genes for development of body segments and organs, and genome regions that cause specific growth pathologies, all of which were proposed as possible prior to 1983.

Another major advance is the synthesis of data from many branches of biology, including **ecology**, demography, genetics, physiology, **phylogeny** (evolutionary history of a species), and **ontogeny** (growth and development of individuals), into a type of grand unification called **life history theory**. Life history theory is a field of biology concerned with the strategy an organism uses to allocate its energy toward growth, body maintenance, defense against infection, reproduction, raising offspring to independence, and avoiding death (Bogin & Smith 2012; Varea & Bernis 2013). For a mammal, it is the strategy of when to be born, when to be weaned, how many and what type of pre-reproductive stages of development to pass through, when to reproduce, and when to die. The life history approach attempts to unravel the reasons why different species of animals follow different sequences of development and why new life stages evolve, such as the juvenile stage of the social mammals. As will be shown later in this book, some "mysteries" of human growth, such as the nature of the human childhood and adolescence stages and the vigorous post-menopausal stage of women, are best understood in terms of life history strategies for efficient reproduction and offspring survival.

Human Auxology

This book is a synthesis of methods and knowledge about human growth gleaned from evolutionary biology, from reports of the growth of human populations living under various ecological regimes, from statistical and mathematical applications, from medical pathology, and from experimental biology. Theoretical perspectives from anthropology, economic history, political economy, sociology, philosophy, and life history are used to order the data derived from observation and experiment.

Drawing upon these areas of research and theory, this book strives to include human growth within the field of **auxology**. The term "auxology" refers to the study of biological growth. It could be the study of any type of growth, and some botanists and most veterinary and farm animal zoologists use this term to refer to growth research in their fields. Botanists use the term "auxins" to refer to the hormones of plants that promote an increase in size of stems, leaves, and other structures. From about 1970, European human biologists began to use the phrase "human auxology" to refer to human growth research and in 1974 the International Society for the

Study of Human Growth and Clinical Auxology was founded.[8] In the words of James Tanner, one of the founders, the Society is the place where " ... physiology, psychology, and sociology meet" (from the web page). An ideal human auxology will combine the results of descriptive studies, experimental research, and hypothesis testing into a comprehensive theory of the structural and functional elements of growth.

The Organization of This Book

A major purpose of this book is to show that the tempo and mode of human growth are basic to the understanding of our species' place in nature – that patterns of human growth reveal the basis for human life history biology and the basis for much of human behavior. The book approaches its purpose by dividing the understanding of patterns of human growth into five areas. The first area is an overview of the history of the study of growth and the basic biological principles of growth and development during the human life cycle, presented in Chapters 1 and 2. The second area is the evolutionary foundation of the human growth pattern, treated in Chapter 3 and Chapter 4. Chapter 3 focuses on the evolution of the human growth curve. The mammalian foundations of the human growth curve, and the nonhuman primate embellishments upon that foundation, are presented conceptually and mathematically. In Chapter 4 the pattern of human growth is considered from an ecological and evolutionary perspective. The relation of hominin and human growth rates to feeding and reproductive adaptations is examined using data from **paleontology**, **paleoecology**, demography, ethology, and ethnology – in other words from a life history perspective. The unique features of the human pattern of growth, including the evolution of the childhood growth period and the human adolescent growth spurt in height, are detailed. The result is a comprehensive exploration, and, where possible, theoretical explanation for the functional and adaptive significance of human growth patterns.

The third area is variation among human populations in growth patterns. In Chapter 5, several cases of such variation are described and the adaptive value of population differences in growth is discussed from an evolutionary and life history perspective. Chapter 6 has discussion of some aspects of genetic and endocrine regulation of growth, development, and maturation. This chapter continues with the theme of population variation and adds a focus on variation between smaller groups, such as communities, families, and individuals. The genome codes for many structural elements, such as proteins, needed for growth and form. The structural genome also interacts with the **regulatory genome** (noncoding DNA, micro-RNA, etc.), the epigenome, the proteome, and with the environment, including the trillions of organisms that live in and on human bodies (the microbiome). Each of these provide guidelines for genetic expression, but not direct gene-to-phenotype

[8] www.auxology.org/

determination. In Chapter 6 there is a review of this popular but incorrect notion of genetic determination. Much of the current debate centers on the benefits and failings of genome-wide association studies (GWAS). The advent of GWAS technology is part of a 150-year history of the misuse of hereditary and genetic information to promote racism, sexism, social inequality, and other harmful political agendas. Parts of this history relevant to human growth are critiqued. It is shown that genomic factors usually do not directly control growth, development, and maturation. Rather, genomic influences are mediated by the endocrine system. In Chapter 6, the basic workings of the endocrine system are presented, and several examples of hormonal mediation are given, including the effects of nutritional and social-psychological stress on hormones, genomic action, and growth.

The fourth theoretical area is covered in Chapter 7, which asks "What makes people grow?" This chapter discusses impacts on human growth, development, and maturation originating from human biocultural environments. Topics include nutrition, seasonal variation, migration, urbanization and modernization, pollution, sleep, socioeconomic status, **community effects on growth**, and strategic growth. Some aspects of these topics were discussed in previous chapters in the context of SEPE influences on growth but are treated more formally and intensely in Chapter 6. Chapter 7 offers a more formal theoretical model of community effects and strategic growth regulation. These two theoretical areas offer relatively new hypotheses about the complex interactions between material, social, and psychological variables that are known to influence biological growth, development, and maturation.

Interspersed throughout the book is discussion devoted to a fifth theoretical area, mathematical and biological models of the process of human growth and development. A scientific understanding of the pattern of human growth requires detailed information of the biological factors that determine development. However, it also requires the precision and economy of analysis that is provided by mathematics. These are not separate realms of knowledge, for the growth and form of an organism often display a clear relationship between biological structure and mathematical regularity. D'Arcy Thompson (1942) described the biological form of the growth of the *Nautilus* shell, seen in Figure I.4, and several other spiral shapes in nature, with a mathematical function called an equiangular spiral. In a similar fashion, the form of the curve of growth of the human being can also be expressed as one or more mathematical functions.

The mathematical treatment of human growth, or the growth of any other organism, is made possible by the predictability of biological development. Growth must produce a biological form that meets the ecological requirements of life for the species. Thus, in terms of growth and form, including the morphology and physiology of organisms, new individuals resemble other members of the same species more than they resemble members of other species. Due to this predictability, growth and form are amenable to the precision of mathematical description. Where appropriate and when needed there are sections of boxed text that describe some of the classic and recent innovative quantitative and qualitative mathematical approaches to the study of growth and its regulation.

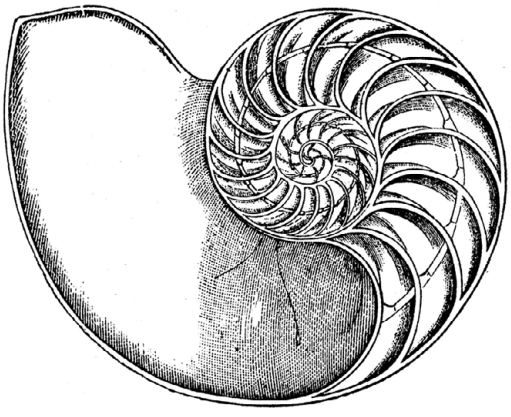


Figure I.4 Shell of *Nautilus pompilius*, sagittal section. Credit ilbusca / DigitalVision Vectors / Getty Images

A recapitulation and synthesis of the major themes of the book are given in Chapter 8. A list of "The most important discoveries about human growth, development, maturation" is offered. As a challenge for the future, Chapter 8 ends with an invitation for readers of this book to consider growth and development research as a professional career. Younger researchers are needed to lead the way toward more accurate, dynamic, and imaginative models and understanding of patterns of human growth, development, and maturation, and their regulation.

1 Background to the Study of Human Growth

People, like most animals, begin life as a single cell, the fertilized ovum. Guided by the interaction of the genomic information provided by each parent, the cytoplasmic environment provided by the mother's ova and her body, and the physical and sociocultural milieu in which people live, this cell divides and grows. The daughter cells differentiate and develop into the embryo, **fetus**, newborn, and with much care from others, and a bit of luck, into the adult who may start the whole process again. Though growth and development may occur simultaneously, they are distinct biological processes. **Growth** may be defined as a quantitative increase in size or mass. Measurements of height in centimeters or weight in kilograms indicate how much growth has taken place since birth. Additionally, the growth of a body organ, such as the liver or the brain, may also be described by measuring the number, weight, or size of cells present. **Development** is defined as a progression of changes, either quantitative or qualitative, that lead from an undifferentiated or immature state to a highly organized, specialized, and mature state. **Maturation**, in this definition, is measured by functional capacity, for example, the development of motor skills of a 6-year-old child that result in a mature state of bipedal walking. Though broad, this definition allows one to consider the development of organs (e.g., the kidney), systems (e.g., the reproductive system), and the person.

Why Grow and Develop?

The human body is composed of about 3.72×10^{13} cells (Bianconi et al. 2013), which result from approximately 2^{38} mitoses (cell divisions) since the moment of fertilization. During **mitosis**, cells differentiate into dozens of types of tissues and organs. From a human perspective, all of this appears to be normal and good. Anthropologists write paeans to the marvels of the specializations of human brain, hand, and bipedal foot. It is not obvious, however, why any living thing should be multicellular. Paleobiologists estimate that enormous numbers of "selfish"[1] single cells, capable of

[1] Use of words such as "selfish" may seem like misguided anthropomorphism. Biologists use words such as "altruistic," "selfish," "cheater" and the like when writing about the evolution of cooperation between cells, organs, and whole organisms. These words are a short-hand way to refer to more complex biochemical mechanisms of the genome. "Selfish" denotes preservation of the genome of a cell or individual. "Altruistic" denotes preservation of the genome of another cell or individual. "Cheaters" are cells or individuals that benefit from the altruism of others without reciprocating. Biologists ascribe no human moral, ethical, or purposeful meaning to these words.

self-replication and sometimes organized into aggregations such as mats, dominated the history of life on Earth between about 4.2–1.5 billion years ago. There are many hypotheses explaining the advantages of multicellularity, all of which revolve around "food and sex." By this is meant the two forces of evolutionary **natural selection** discovered by Charles Darwin (1809–1882) and Alfred Russel Wallace (1823–1913): differential fertility and differential mortality. All living things require nutrients for the building-blocks of their physical structure and, also, require energy to organize, maintain, and power their life. Most living cells are mortal and must replicate to pass on copies of themselves, in whole or part, to the future. On the "food" side of things, multicellularity and specialization of tissues, it seems, were naturally selected to increase the efficiency of food acquisition and the digestion and transport of nutrients between cells. Selection for defense against predators also played a role, for example, being larger than your predators or having surface defenses. On the "sex" side, many types of differentiated cells can still divide by **mitosis** to replace their mortal comrades (think of your skin or bones healing after damage) and specialized gametes produced by meiosis – ova and spermatozoa – allow for repeated reproduction events during the lifetime of the individual.

Multicellularity requires cooperation between cells that were formally autonomous and selfish. Several mechanisms for the evolution of cooperative multicellularity are proposed, the most basic of which is the single-cell bottleneck of sexual reproduction (Levin et al. 2015). The descendant cells are genomic clones of the fertilized ovum. The selfish interest of these clones is the altruistic care of their "sister" cell's genomes, no matter their phenotype as specialized cells, tissues, or organs. The very high genome similarity between the clones promotes inclusive fitness, that is, behavior to increase, " . . . the proportion of replica genes in a relative . . . " (Hamilton 1964, p. 1). Experimental evidence from the laboratory of Joan E. Strassmann and David C. Queller supports both inclusive fitness and another mechanism, called "resistance to cheating," as promoters of multicellularity (Levin et al. 2015).

Multicellularity likely evolved more than once and is present in at least the four Eukaryote Kingdoms of life – Animalia, Plantae, Fungi, and Protista. Even some species in the normally unicellular Kingdom Eubacteria form colonies that seem to divide biological activities and increase metabolic efficiency. The benefits of multicellularity, cell and tissue specialization, and sexual reproduction come with the costs of growth, development, and maturation. The initial contribution to the next generation, a fertilized ovum, does not look or behave like the adult parents. To be like their parents, " . . . the new organisms will have to suffer changes before they become something approaching replicas of the old" (Newth 1970, p.1). Newth, an embryologist, adds that the processes of growth, development, and maturation are arduous, often prolonged, and generally hazardous.

Historical Background for the Study of Human Growth

Like the fertilized ovum, the study of human growth and development has undergone an arduous, prolonged, and hazardous history of intellectual development. Arduous

and prolonged because it has taken more than three thousand years of study to arrive at our present state of knowledge. Some of the highlights of these three millennia are described in the next paragraphs of this section. The history of growth research is hazardous because misinformation and gaps in our knowledge lead to tragic consequences for human beings. The thalidomide drug disaster of the 1960s and the decline in breast-feeding in "modern" societies are just two recent examples of these hazards. Thalidomide is a sedative and hypnotic drug that was used to treat some of the discomforts of pregnancy. It was withdrawn from sale after it was found to cause severe birth defects, especially of the limbs. The thalidomide tragedy promoted tougher standards for testing the safety of new drugs and more research on environmental influences of embryonic development.

A decline in breast-feeding during the twentieth century is a typical occurrence in both developed and developing nations. The development of milk-based formulas to feed infants and their aggressive promotion by the formula makers as "modern," "safer," and nutritionally "superior" to breast milk, led partly to the decline. Anti-nursing support also came from physicians, who were often compensated by the formula makers (Wolf 2003). In the United States, rates of breast-feeding of any duration fell to a low point of $<24\%$ in 1971 (Wolf 2003). During the 1980s and 1990s there was much research showing that there are harmful physical effects for infants who are never breast-fed. These include increased incidence of respiratory, inner ear, and gastrointestinal infections, accumulation of more body fat, later-life risk of heart disease (Cunningham 1995; Scariati et al. 1997) and reduced mental development (Dettwyler 1995). The emotional benefits of breast-feeding, including greater mother-infant bonding and attendant care, likely work to decrease the risk of these physical problems. A systematic review of literature from the twenty-first century finds that over their lifetime, breastfed infants enjoy benefits of lower blood pressure, lower risk for type-2 diabetes, lower risk for obesity, and higher intelligence test scores (an increase of 2.2–3.5 IQ points) (Horta & Victora 2013). This research was incorporated into public health policies and education programs, which raised the incidence of breast-feeding in the United States so that in 2016 81% of infants were ever-nursed and 52% were nursed until 6 months of age (NCCDPHP 2016).

These examples of hazards in the history of the study of human growth and development provide reason for caution today and for the future when prescribing drugs and recommending infant feeding regimes. Over-prescription of antibiotics to growing girls may alter their intestinal biome as women, with consequences for pregnancy and the development of their embryo and fetus. A review by Dunlop and colleagues finds links between maternal microbiome and preterm birth, preeclampsia, gestational diabetes, and excessive gestational weight gain (Dunlop et al. 2015).

Another reason to study the history of any discipline is that one learns what topics have been studied, when such inquiry first occurred, and which problems need further study. Historical study is important from a conceptual perspective as well, as it may help explain why scholars and practitioners have been interested in human development. The history of study of human growth makes clear the relevance of growth research to medicine, **epidemiology**, and public health. More generally, an

understanding of the history of the study of human growth reveals connections between economics, art, law, politics, philosophy, and other fields of knowledge that influence the course of human events.

The following is a review of some of the major historical events in the study of human growth, with special emphasis on those which still have an influence on growth research today. Concise historical reviews were published by Lowery (1986), with a focus on pediatric medicine and by Bogin and Kapell (1997) with an emphasis on topics of anthropological interest, including normal population variation in growth and development and their biocultural influences. Two edited compendia, *The Cambridge Encyclopedia of Human Growth and Development* (Ulijaszek et al. 1998) and the more recent and delightfully illustrated *Auxology: Studying Human Growth and Development* (Hermanussen 2013) provide many entries relating to the history of human growth research from both clinical and anthropological perspectives.

Book-length histories of the study of human growth were published by Boyd (1980) and Tanner (1981). These two books provide comprehensive historical detail and are complementary to each other in the sense that the content and style of each book is distinct. A few comments on these books seem appropriate to place them in historical context. Both are authored by physicians and both specialized in human auxology. Both Boyd and Tanner made contributions to descriptive studies of how people grow, develop, and mature, technology needed to measure the human body, psychological and physiological aspects of growth, statistics needed to analyze growth data, and hypotheses about why people grow and what factors influence growth. Both books have a clinical perspective on growth pathology and the use of growth data to assess public health. Tanner's book is the more conventional, coherent, and formal in style of presentation and is available from Cambridge University Press.

Boyd's book, *Origins of the Study of Human Growth*, was published posthumously through the efforts of her two editors, Bhim Sen Savara and John Frederick Schilke at the School of Dentistry, University of Oregon Health Sciences Center. The book, " . . . entailed fifty years of endeavor by several scholars" (Beall 1984, p. 93). Richard Scammon had started preparation for this book in 1929 and at the time of his retirement in 1947 left, " . . . about one hundred notebooks filled with manuscripts, transcripts, bibliographies, and tables in triplicate, twelve drawers of drawings, and twelve drawers of bibliographic cards pertaining both to human growth and to the history of science . . . [along with] . . . extensive detailed outlines . . . " (Boyd 1980, p. XVII). Upon this foundation Boyd continued to build, starting in 1953 when she was working at the Child Research Council, University of Colorado at Denver and continuing until at least 1966. At the time of her death in 1977 the manuscript remained unpublished and a group of Boyd's former students, colleagues, and friends made the effort to finalize the editing and assure publication. A limited number of copies were published by the Oregon Health Sciences Center Foundation and could be purchased only by special request. Boyd's book is clearly the product of several writers and editors. It presents the history of interest in human growth in a linear

fashion over the past 6,000 years. The book includes many important illustrations, especially of artwork, and takes a global perspective, with chapters on China, Hindu India, Early Jews, Zoroastrians, ancient Greece and Rome, and the Islamic Empire; topics mostly not available in Tanner's book. Boyd's book lacks an index and is not available online, so it is cumbersome to search for specific information in its large format printing with 676 pages of text.

Tanner makes no mention of Edith Boyd's book even though it was published in the previous year. It is possible that Tanner was not aware of Boyd's book due to its unusual mode of publication. More surprising is that there is no index entry for "Edith Boyd" in Tanner's book, but she is described on page 318 as " . . . Scammon's erstwhile assistant . . . " Tanner did cite one of Boyd's articles on measurement error and her " . . . small and valuable monograph on growth of surface area . . . " (Tanner 1981, p. 339). Boyd was more than an "assistant." In addition to publishing the book, *The Growth of the Surface Area of the Human Body* (1935, University of Minnesota Press), authoring many scientific articles in medical and anthropological journals and working at both the University of Minnesota Institute of Child Welfare and the University of Colorado Child Research Council, Boyd served on the Committee on Growth and Development, convened in 1930 by United States President Herbert Hoover. Other notables on the Committee were Franz Boas, Walter Cannon, Charles Davenport, Arnold Gesell, Richard Scammon, Wingate Todd, and the one other woman, B. Holly Broadbent (1894–1977), a pioneer of radiographic studies of growth (see below).

Edith Boyd's many contributions to human growth research and pediatric practice are not as well remembered today as they should be. This is a common fate for many women scientists and academics. To her credit, Boyd's book reviews important research by other women, including another forgotten auxologist Rachel Fleming, whose longitudinal study of child and adolescent growth is described later in this chapter.

Prehistory and Early Historic Period

The hominin fossil record includes immature individuals ascribed to the genus **Australopithecus** and *Homo*, including *H. ergaster*, *H. erectus*, and *H. antecessor*. These fossils date from about 2 MYA (million years ago) to about 800,000 BP (before present). There are fossils of immature *H. neanderthalensis* dating to about 49,000–70,000 BP (Akazawa et al. 1995; Rosas et al. 2017). The earliest evidence for the evolution of our own species, *H. sapiens*, dates to about 300,000 BP (Hublin et al. 2017). Unearthed at this site, in present-day Morocco, were five individuals, including a child and an adolescent. There is little evidence of how these ancient hominin ancestors ("hominin" refers to living humans and extinct species that were capable of bipedal locomotion) may have conceptualized the growth and development of their offspring. The Neanderthal infant from the Dederiyeh site in Syria, perhaps aged 1.5–2.0 years old at death, seems to have been buried purposefully, suggesting ritual behavior that extended to very young individuals. A review of these

fossils and what they may tell us about the evolution of human growth and development is presented in Chapter 4.

It is not until quite recently that human ancestors depicted their own form in artwork. Small stone sculptures, often called "Venus figurines," and cave paintings from Europe depict people and animals that may be pregnant. The earliest of these artistic renderings date from about 30,000 BP. There are also rock paintings from southern Africa, Indonesia, and Australia, older than the European cave art, that depict people's hands of various sizes as well as nonhuman animals of various ages and sexes. Dating from about 20,000 BP there are depictions of what may be women giving birth.

One early modern human skeleton from Italy, dating to the Upper Paleolithic (~18,700 BP) seems to have been from an adolescent with a type of dwarfism caused by acromesomelic dysplasia (Frayer et al. 1987). This type of dwarfism results in severe deformity and physical impairment, but normal intelligence. The affected individual would have been unable to contribute much labor to a hunting and gathering group. Frayer and colleagues believe that this individual's survival to the teenage years indicates both tolerance and care for impaired infants and children. This may be so, but a detailed understanding of how Paleolithic peoples may have interpreted the meaning of pregnancy, normal and pathological growth, and human development in general are matters for speculation.

The earliest written records about human growth date from Sumerian Mesopotamia, about 3,500 BP. Inscribed myths recount the act of fertilization, the nine months of pregnancy, and both full term and premature birth. Concerns about **low birth weight** or **prematurity**, birth defects, and twinning also are recorded. The Sumerians divided postnatal life into several stages that correspond to modern ideas of infancy, childhood, youth, **adulthood**, and old age. There is no direct evidence that Sumerians measured the dimensions of the body. Some of the artworks seem to depict accurate size differences between children and adults. Other works of art depict high status people, such as male elders, as disproportionately taller than lower status people, such as women. Several texts also make mention of a positive relationship between health, social status, and stature. Thus, both in Sumerian art and in life there was a relationship between growth and biosocial conditions. That this relationship appears in the earliest writing on human growth is fascinating, for the study of this association is still a very active area of research today (see Chapters 5 and 6).

The ancient Egyptian, Chinese, Hindu, Greek and Mesoamerican civilizations followed many of these Sumerian traditions. Written records and artwork show that the earliest interest in the biology of children was primarily a concern with the preservation of life. Greek, Roman, and Arab physicians prescribed regimes of physical activity, education, and diet to help assure the health of children. Their advice was more often guided by the needs of their societies for military personnel and by religious dogma about children rather than by empirical observations of the effect of child-rearing practices or child growth, development, and health. Of course, there were marked differences between these societies in specific cultural values, but the universal biological nature of pregnancy, birth, and infancy (this nature is

reviewed later in this chapter) meant that all human societies must converge on some basic strategies for the care and feeding of their young. Some of the early civilizations, such as the Egyptians and the Hindu Indians, showed careful concern for measurement of the body, including children and youths. Egyptians used a grid system to carefully render body proportions correctly. Egyptian relief carvings and statures depict correctly the body shape and limb proportions of people with achondroplasia (normal-sized head and trunk with short arms and legs) and hypochondroplasia, a milder form of achondroplasia (Hermanussen 2013, p. 132).

Other cultures, such as the Chinese and early Jewish tradition, emphasized more spiritual aspects of human development in their concern for the young. One matter of repeated concern for these ancient societies is the number of stages of life. Numbers vary, but by the time of the Romans "seven ages of life" becomes a frequent blueprint for human development. Today, research into biological life history theory, which is concerned in part with the number of stages in the life cycle of organisms, is very popular and productive. Human life history theory is a central concern of Chapters 3 and 4 of this book.

The Latin West and the Renaissance

Egyptian, Greek, and Roman artwork, especially three-dimensional sculptures, depict infants and children quite accurately in terms of body size and proportion (infants have relatively larger heads and shorter arms and legs than children and adults). In some of this art the children are depicted at play. Viewing this art can give a sense that infancy, childhood, youth, and the other "seven stages of life" were each accorded its own special biology and behavior. Scholarly concern with the stages of life continued following the collapse of the Roman Empire, but there may have been a shift in the status accorded to children and youths. Medieval physicians, clerics, and artists began to follow a tradition of treating the child as an "unripe" adult. In this tradition, infants and children had no special biological characteristics. To grow and develop from infant to adult involved only an increase in size and a maturity of behavior and reasoning.

There is considerable debate as to how children of the Medieval Age of Europe were perceived and treated (scholarship focused on the history of childhood in non-European regions is conspicuous by its absence). By the end of the twentieth century, historians seemed to reach a consensus that children lost any special status they may have had in earlier times. Kaplan (Kaplan 1984, p. 46) writes that, "Plague, pestilence, ignorance, extraordinary poverty, drudgery, starvation, perpetual warfare were . . . " some of the reasons why children lost special status. Ariès (1962) reconstructed the social world of that time and concluded that between the thirteenth and eighteenth centuries in Europe a cultural category of childhood was not recognized and that children were regarded as "little adults." After the age of seven years, children were forced to enter the social world of adults. Ariès believed that this accorded children great freedom, but Kaplan viewed this as a kind of abandonment. In either case, this

version of "social reality" holds that young people were expected to become adultlike at a fairly young age.

Anthropologists long rejected the notion of a society without a concept of childhood. Every society studied by anthropologists has definitions of childhood, but these vary considerably, as do cross-cultural child-rearing practices. The variations extend to the terminology of the stages or divisions of pre-adult life, the rituals experienced by young people, and the expectations that older people place on younger people (Lancy 2014; LeVine 1988, 2009). Contemporary historians of childhood acknowledge that this field was invented by Ariès, but do not accept most of Ariès' more dogmatic assertions, for example that children, " . . . heard the same jokes, played the same games, wore the same clothes, and, so far as they were physically able, did the same work as their parents" (Zuckerman 2009, p. 60). Current scholarship in the field of History of Childhood expands interest to new work on such topics as the child's perspective, fatherhood, gender socialization, sibling interaction, and poor families which has greatly enriched the field (Gray & Anderson 2010; Pollock 1983).

Some historians of childhood, including Ariès, were influenced by the artworks of the Medieval times. Quite often the paintings and mosaics of that time depicted young people with the same body proportions as adults. This led the historians to assume that children were thought of as "little" or "unripe" adults in cognition and behavior, as well as in body. A few well-known paintings in this stylistic tradition are the "Rucellai Madonna," attributed to the Italian artist Duccio (1285?), the "Madonna of the Trees" by Bellini (1487), and "Peasant Dance" by Pieter Bruegel the elder (1568?).

A few words of caution need to be interjected at this point. The limited scholarship and current interpretation of the history growth research, and of "children" and "childhood" (in fact human development from fetus to young adult), is distorted by Eurocentric ideals and beliefs, including the perceptions and behavior of people in the past toward children. Sommerville (1982) mentions that the Greeks made note of the absence of infanticide among the Egyptians. Sommerville documents that infanticide was an accepted practice for some Greeks and later for the Romans. After invading the Americas, the Spanish were impressed that all Aztec, Maya, and Inca infants were breast-fed, and that nursing, along with other foods, continued until four years of age. Even women of royal status nursed their own babies (Shein 1992). It seems that in sixteenth-century Spain, and elsewhere in Europe, breast-feeding was viewed as "too natural" and thus not becoming to people. This was especially so for those of high social status who often contracted lower social status women as wet nurses. So, when historians debate the alleged brutalities against children of past ages, readers should be careful not to commit the ethnocentric fallacy of judging other cultures by one's own standards. What late twentieth-century writers note with amusement about the past may be telling us more about our own unconscious assumptions, or wanting behaviors, toward our own children. Anthropologists of childhood and child rearing practices have warned of this Eurocentric ethnocentrism for some time (Lancy 2014; Weisner 1987). More generally, the academic fields of sociology, psychology, philosophy, economics, and political science are too often

based on research with people from the WEIRD societies of the world – Western, Educated, Industrialized, Rich, and Democratic (Henrich et al. 2010). These fields are too often based on experiments and observation of university students, a very WEIRD group, usually with little experience of children and child care. University students are unrepresentative of the majority of the living human population and of past human populations.

It is also important to interpret the art of the past with some caution. A critic of the first edition of this book stated, "Art is a figurative medium and cannot often be used to make objective assumptions about culture" (Kathryn Stark, personal communication). The same critic wonders if it is correct to state that Picasso (1881–1973) paints children "better" than Bruegel (1525?–1569)! Even though it may not be possible to know exactly how infants, children, and youth were treated, and perceived, during the Medieval period, it is clear enough that human growth and development were not studied scientifically. During the Renaissance period there was a revival of the classical Greek concept of the dynamics of growth, which is amusing, as this concept was never accepted popularly by the Greeks or Romans. An example of the Greek revival comes from the scholar Giordano Bruno (ca. 1550–1600) who wrote, " . . . We have not in youth the same flesh as in childhood, nor in old age the same as in youth; for we suffer transmutation, whereby we receive a perpetual flow of fresh atoms and those we have received are ever leaving us" (Boyd 1980, p. 176). This is a remarkably modern statement of the constant turnover of cells and the constituents of cells in the human body (see Chapter 2).

Leonardo da Vinci (1452–1519) proposed that new studies of human growth and development, from conception onwards, needed to be undertaken. Leonardo initiated his own human dissections, including his study of a seventh-month fetus, the **placenta**, and stillborn full-term infants (Figure 1.1). In the year 1502, the physician Gabrielo de Zerbis (ca. 1460–1505) published a description of the anatomical differences between child and adult. Many other medical and scientific publications quickly followed.

Leonardo used his scientific studies of human growth to produce drawings that correctly rendered adult and child body proportions. Building on the work of Vitruvius, a first-century BC Roman architect and writer, Leonardo developed canons, or rules, for drawing human proportions (Tanner 1981). Albrecht Dürer (1471–1528), a German artist, devised a method of geometric transformations that he used to accurately render proportions of the human head and face. With his geometric methods, Dürer could draw not only the canonical types, but any manner of human variation in size or proportion. He applied his method to drawings of men, women, children, and infants (Figure 1.2). Including the depiction of women and children in this type of methodological work was an innovation, as most artists followed the teachings of Cennino Cennini (ca. 400) who wrote that women do " . . . not have any set proportion" (Boyd 1980, p. 202). Children, it seems, were too inconsequential for Cennini to even mention! As a reflection of the art and scholarship of the time, the work of Leonardo and Dürer portended a major change in the concept of children and research into human growth.

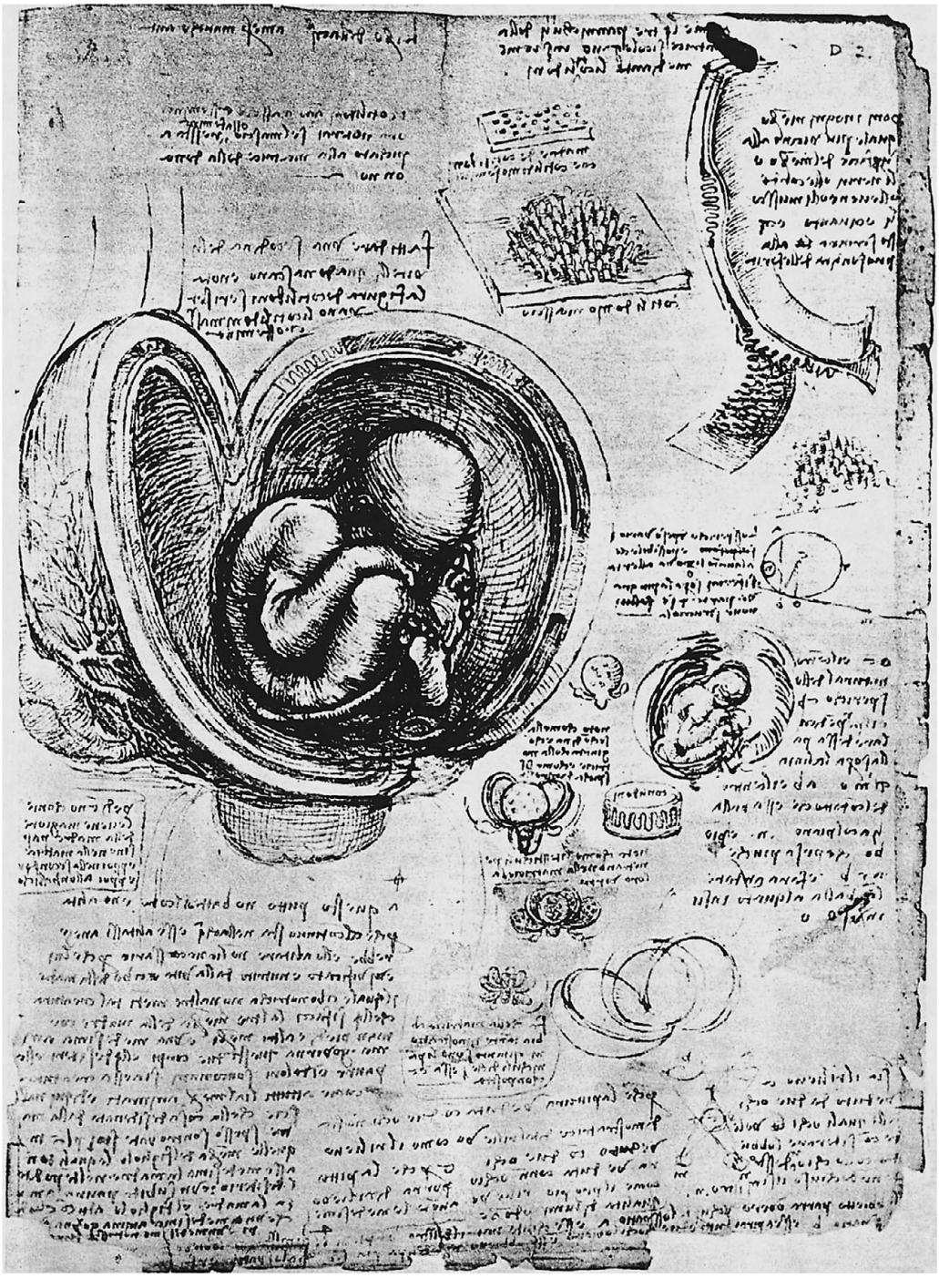


Figure 1.1 Fetal positions and structures of the placenta shown in sketches by Leonardo da Vinci. Windsor collection, folio 8r. From *Quaderni d' anatomia*, volume III

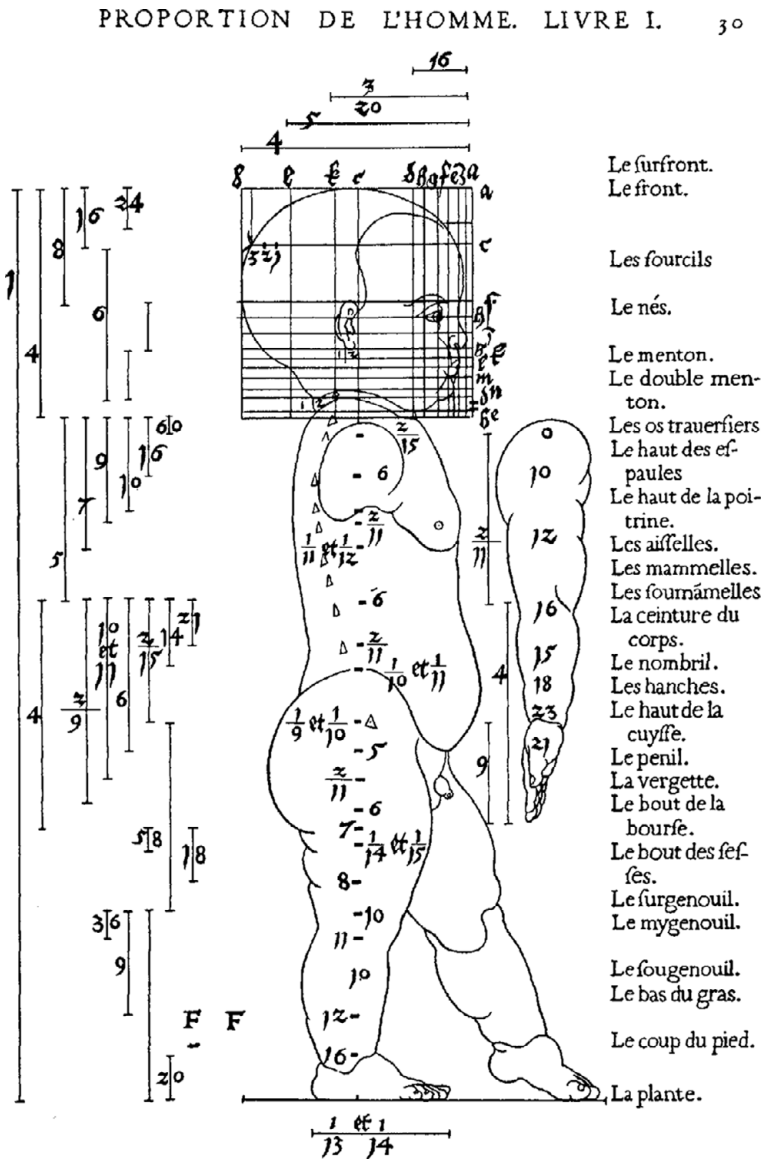


Figure 1.2 Proportions of the infant, diagram by Albrecht Dürer. From the 1557 edition of *Les quarte livres d'Albert Dürer*

After the year 1600, the post-Renaissance painters begin to depict children with normal proportions, as well as with growth pathologies. The Flemish artist Van Dyck depicts three normal children in the painting "The Children of Charles I" (1635). The painting "The Maids of Honor" by Diego Velázquez (1656) centers on a normal child (the 6–7-year-old Spanish princess), includes several "normal" sized and proportioned adolescents and adults, but also a woman with achondroplastic dwarfism and

an adult man with growth-hormone deficiency dwarfism (proportionate reduction in size of all body parts) with his foot on a rather large dog. At the time of these paintings, of course, the biological control of normal and pathological growth was not known. Nor is it clear today how physicians and scientists of that time regarded different types of dwarfism.

Embryonic and Fetal Development

Ancient, Classical, and Medieval scholars had written a great deal on human growth and development prior to birth. Some of this was undoubtedly based on observation of human and nonhuman fetuses, but much was also the product of imagination and myth. The actual process of growth and development from fertilized ovum to the birth of a human child is so counterintuitive to our expectations, based on our experience with child growth after birth, that through much of human history scholars and physicians did not know or believe that it occurred. It was not until the year 1651 that the physician William Harvey helped establish that the embryo is not a preformed adult. Harvey showed that during prenatal development there are a series of embryological stages that are distinct in appearance from the form visible just before and after birth. The Greek physician Galen (ca. AD 130–200) wrote about the appearance of the fetus in the later stages of pregnancy. However, the first accurate drawings of the fetus were made by Leonardo da Vinci, as mentioned above. Other descriptions of fetal anatomy and physiology followed Leonardo's work, notably the studies published by Vesalius in 1555 and Volcher Coiter in 1572. Coiter studied a fetus less than 3 cm in length, indicating that the fetus had developed for about 10 weeks since conception.

During the seventeenth and eighteenth centuries, descriptive anatomical studies continued, with most of the work being done on fetuses in the last trimester of pregnancy (last three months). The fetus of this age is of unmistakable human appearance, so these studies failed to appreciate the physical changes that take place earlier in prenatal life. Some biologists continued to believe in **preformation**, and a few extended that concept beyond pregnancy to the formation of spermatozoa (Figure 1.3). In 1799, S. T. Sommerring published drawings of the human embryo and fetus from the fourth week post-fertilization to the fifth month, clearly showing that the embryo is not a preformed, or miniature, human being.

The scientific study of the cellular mechanisms embryonic development has its roots in the work of Karl Ernst von Baer (1792–1876), published in 1829 (von Baer 1986). He described the "germ layers" of the embryo, properly called the endoderm, the mesoderm, and the ectoderm. In general, the endoderm cells give rise to the inner lining of some internal organs, cells from the mesoderm form the skeleton, muscles, the heart and circulatory system, and the internal sex organs, and ectoderm cells give rise to parts of the skin, the teeth, and the brain and nervous system. The work of von Baer removed the need to invoke mystical "vital forces" to explain embryological transformations, replacing these with more mechanistic processes. However, it was not until the twentieth century that an understanding was achieved of the highly

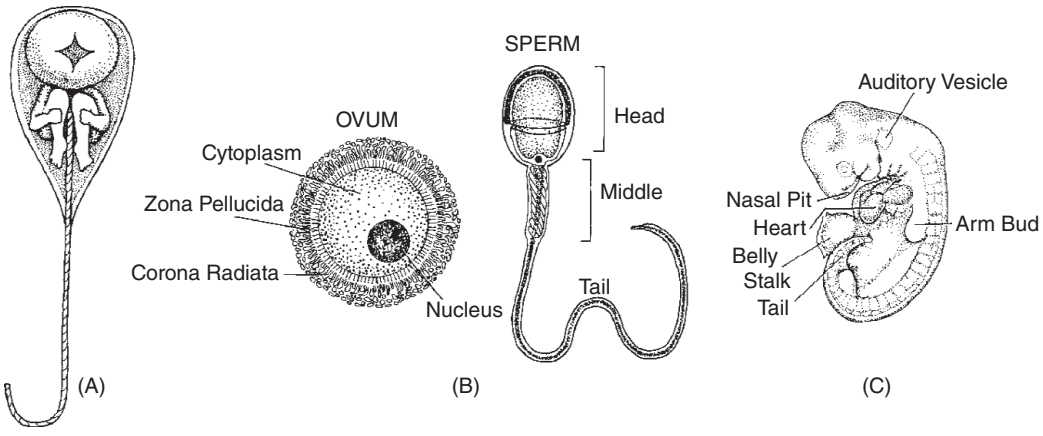


Figure 1.3 (A) Preformationist rendering of a human spermatozoon (Hartsoeker, 1694; from Singer, 1959). (B) Diagram of human ovum and spermatozoon. (C) Diagram of human embryo 32 days after fertilization.

complex nature of the physical, chemical, and biological processes that occur during prenatal growth.

As early as 1651 Harvey declared that "everything comes from the egg." Amazingly, it was not until 1875 that Oscar Hertwig (1849–1922), a German biologist who is mostly unknown today, first proved that fertilization results from a single sperm penetrating an egg cell, fusing with the nucleus, after which the cell begins to divide. He observed this using the sea urchin (Dolnick 2017). In 1885 Hertwig proposed that nuclein (later called nucleic acid) is the substance responsible not only for fertilization but also for the transmission of hereditary characteristics. He was proven correct in 1944 by Oswald Avery, Colin MacLeod, and Maclyn McCarty, who showed that this is indeed the role of the nucleic acid DNA.

Longitudinal Studies of the Eighteenth Century

Another post-Renaissance advance was a growing interest in how early life events could influence later development. For instance, by the 1700s physicians pursued the study of birth weight and its relation to child health. Prenatal and neonatal influences on later development remain topics of research interest today (Gluckman et al. 2007; Hanson et al. 2011; Kuzawa 2005). A new research strategy had to be developed to study the relationship between early influences and later growth outcomes. This is the longitudinal method of research. A longitudinal approach requires that the same individuals be examined on at least two occasions, separated by some amount of time. Prior to the use of longitudinal methods, the predominate strategy was the cross-sectional approach in which each individual is examined only once. The cross-sectional approach has the advantage that many people can be measured in a short period of time. The disadvantage is that the dynamics of growth, such as changes in rate of growth, cannot be properly studied.

The Count Philibert Guéneau du Montbeillard (1720–1785) of France, measured the stature of his son every six months from the boy's birth in 1759 to his eighteenth birthday. George-Louie Leclerc de Buffon (1707–1788) included the measurements, and his commentary on them, in a supplement to his *Histoire Naturelle* in 1777. These data are usually considered to constitute the first longitudinal study of human growth and, due to Buffon's commentary, the most famous study. The growth in height of this boy, both in terms of achieved stature by age and rate of growth at any age, is illustrated in Figure 1.4. The original data were reported in antiquated French units of measurement, but Scammon (1927) converted these to modern metric units. The metric data are drawn here as mathematically smoothed curves (the cubic spline technique was applied to the data given by Scammon by the present author). The smoothing makes the important features of the curve more easily seen. Scammon's original graph of the data, and another graph comparing the aristocratic French boy to infants and school children of lower socioeconomic status (SES), may be seen in Scammon's 1927 article and are reprinted by Miller (2018), who adds commentary on the historical context and legacy of Scammon's research on human growth.

The curve in the figure labeled A is the boy's total height at each measurement. If growth is viewed as a motion through time, then this graph may be called the **distance curve** of growth. The boy's rate of growth between successive measurements is graphed in part B of the figure, commonly called the **velocity curve** of growth. Buffon had earlier written on the adolescent spurt in growth (the rapid acceleration in growth velocity around the time of sexual maturation) and on the general advancement of maturation of girls compared with boys. With the data on Montbeillard's son, Buffon noted the seasonal variation in rate of growth; the boy grew faster in the summer than in the winter. Buffon also wrote of the daily variation in stature; the boy was taller in the morning after lying at rest during the night than he was in the evening after working and playing during the day. Since Buffon's time, it has become necessary to take these variations in seasonal growth and daily stature into account when designing or analyzing longitudinal growth studies.

Except for the adolescent growth spurt, Buffon made little mention of changes in growth velocity that are clearly seen in Figure 1.4. Montbeillard's son shows four distinct phases of growth velocity. The approximate duration of each phase, its name, and its tempo are: (1) from birth to three years of age, the **infant phase**, growth decelerates rapidly from its maximum value of 22 cm/year to 6 or 7 cm/year; (2) from three to about seven years of age, the **childhood phase**, growth rate remains fairly constant; (3) after age 7 years until about 11 years, the **juvenile phase**, growth rate decelerates again, at first rather slowly but then much faster; (4) from about 11 to 18 years of age, the adolescence phase, there is a classic adolescent growth spurt, with an acceleration period from about 11 to 14.5 years followed by a deceleration period that continues until 18 years and beyond. Note that the acceleration period is less steep, that is, the **tempo of growth** changes less rapidly, than the deceleration period. A more detailed discussion of these phases of postnatal growth is presented in the next chapter.

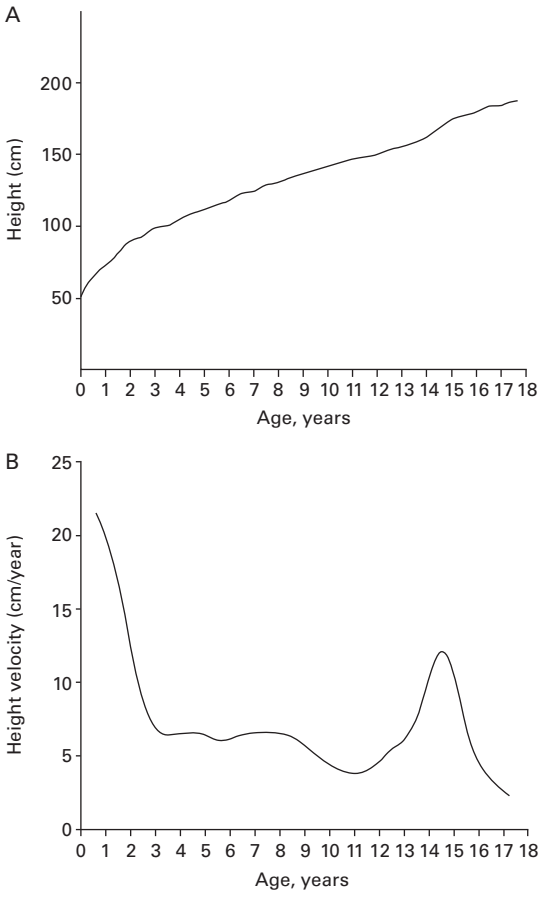


Figure 1.4 Growth in height of the son of the Count Montbeillard during the years 1759–1777. (A) Amount of growth achieved during each six months from birth to age 18 years. (B) Rate of growth in height during each six months from birth to age 18 years.

Tanner (1990, p. 75) explained that the concept of tempo of growth is a metaphor from classical music, " ... some children play out their growth andante, others allegro, a few lentissimo." The work of Buffon, and more recent work by Tanner and others, helped to clarify the important difference between growth distance (size) achieved at a given time and the growth velocity over time.

Another eighteenth-century longitudinal study of growth is that of the students of the Carlschule, conducted between the years 1772 and 1794. The pupils of this high school, founded by the Duke of Württemberg, included sons of the nobility (the high aristocracy) and of the bourgeoisie (the low aristocracy). The growth data were analyzed by Komlos and colleagues (Komlos et al. 1992) showed that the high aristocracy students were, on average, taller than the bourgeoisie during the growing years and remained so at 21 years of age (Figure 1.5). Thus, despite the nearly identical physical, nutritional, sanitary, health, and educational environment at the

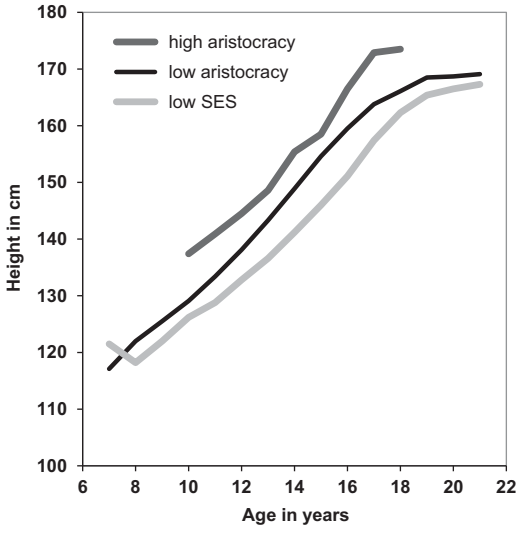


Figure 1.5 Mean heights of Carlschule students measured in the years 1771–1793. Based on data in Komlos et al. (1992)

school, the sons of the nobility were always taller. This is so even today and reasons for differences in height associated with social status are discussed in detail in Chapter 7.

Statistical Approaches of the Nineteenth Century

In 1835 Lambert Adolphe Quetelet (1796–1874) published the first statistically complete study of the growth in height and weight of children. Quetelet was the first researcher to make use of the concept of the "normal curve" (commonly called today the normal distribution, "bell-shaped" curve, or Gaussian distribution) to describe the mean and distribution of his growth measurements, and he also emphasized the importance of measuring samples of children, rather than individuals, to assess normal variation in growth. Quetelet proposed, " . . . that a random sample from a representative diversified group could be used to estimate the total population" (Eknoyan 2007, p. 48). Eknoyan writes that Quetelet, "saw bell-shaped curves everywhere he looked, including in social phenomena and the variables that determine character and aptitudes (Eknoyan 2007, p. 49). Quetelet applied this discovery to the analysis of physical characteristics, including size of the body, to birth and marriage rates, and to population distributions between regions and countries. With these mathematical advances, Quetelet, essentially, invented modern applied statistics.

During Quetelet's academic life, Buffon's observations on puberty and the adolescent growth spurt were, essentially, ignored or forgotten by growth scientists. Quetelet's mathematics worked best on smooth, normal curves of growth data. Growth spurts were to be ignored, he declared, because from infancy onward growth

velocity decreased in a monotonic fashion. His academic and social influence was so powerful that only after Quetelet's death did other growth researchers dare to begin to analyze the "anomalous" adolescent growth spurt (Vandereycken and Deth, 1990). Ludwig W. Kotelmann (1839–1908) was, perhaps, the first to mention again the adolescent growth spurt (Kotelmann 1879). Henry P. Bowditch (1840–1911), a colleague of Boas, published his measurements of Boston school children and reestablished the spurt as a normal human trait (Bowditch 1877). Bowditch reaffirmed Buffon's 1749 comment that girls began their spurt about two years earlier than boys.

Quetelet's statistical approach was followed in Europe by Luigi Pagliani (1847–1932). Pagliani began his studies on the size and fitness of Italian military personnel. He later applied his methods to children, and in 1876 demonstrated that the growth status and vital capacity (the maximum volume of air that can be inspired in one breath) of orphaned and abandoned boys, ages 10 to 19, improved after they were given care at a state-run agricultural colony. Pagliani also noted that children from the higher social classes were taller, heavier, and had larger vital capacities than poverty-stricken children. Finally, Pagliani followed Buffon in taking longitudinal measurements of the same children. From these Pagliani noted that **menarche** (the first menstruation of girls) almost always followed the peak of the rapid increase in growth that takes place during puberty. He concluded that reproduction was delayed in young women until growth in size was nearly finished. This, he considered, was a proper relationship, for the nutritional and physiological demands of growth would interfere with similar demands imposed by pregnancy.

Politics, Heredity, Environment, and Growth

During the nineteenth century, growth research was used for the first time to inform political and legal decisions regarding the treatment of children. The growth of European cities during the eighteenth century led to a flow of rural-to-urban migrants. Urban life dislocated many people from traditional rural family social organization, such as the care of orphaned or abandoned infants and children by family members. Urban poverty and generally high rates of mortality for women associated with pregnancy and birth resulted in a surge of motherless and unwanted infants. If they survived, they often became wards of the parish or were placed in foundling hospitals. The growth and health of these abandoned infants was extremely poor, and many died. Indeed, an estimate of the infant mortality rate (IMR) for London in the year 1740 is 450 per 1,000 live births (Levene 2005). To give this some context, for the year 2017 the highest national IMR was for Afghanistan at 110.6 per 1,000 live births and the lowest was 2.0 for Japan.[2]

The economic, emotional, and moral burden to society of the abandoned and dying babies called for some action. The physician William Cadogan (1711–1797) published *An Essay Upon Nursing and the Management of Children from Their Birth*

[2] www.cia.gov/library/publications/the-world-factbook/rankorder/2091rank.html

to Three Years of Age (1748),[3] that instructed the women working in foundling hospitals and parish orphanages. In a sense, this book was the first practical pediatric guide to baby care. The need for such books has not diminished over time. Indeed, one best-seller, Benjamin Spock's (1903–1998) *The Common Sense Book of Baby and Child Care* (first published in 1946), sold over 30 million copies in its first 30 years in the United States (only the Bible sold more copies). By 2011 the book had sold more than 50 million copies worldwide, in 39 languages.

The publication of Cadogan's Essay reflected a broad concern for infant health in England, where by 1767 laws were passed regulating the operation of foundling homes. On the Continent new concerns for infant care were sparked by the publication of Jean Jacques Rousseau's (1712–1778) book *Emile* in 1762. Rousseau advocated a "return to nature," including the breast-feeding of infants by their own mother. Artificial feeding devices ranging from cow's horns to clay vessels had been used for centuries, but were becoming more common in cities. Such devices were difficult to clean, and the animal milk or other liquids fed to infants was surely not maintained under hygienic conditions. The inevitable result was intestinal infection for the infant. The higher social classes, especially in large cities such as Paris, often "farmed out" their infants to wet-nurses. Since these nurses might have several infants to feed, including her own infant, it is likely that some or all her charges received too little breast milk, or were fed artificially. Rousseau's book was highly critical of these practices and, " . . . profoundly influenced thinking on child care and education throughout Europe" (Boyd 1980, p. 270).

Another socioeconomic and political force working against the welfare of children was the Industrial Revolution. Between the years 1765 to 1782 James Watt (1736–1819) developed a commercially viable steam engine, which forever changed the nature of human labor. The factory system ushered in under steam power reduced the need for human muscle power, allowing children to be employed for many tasks. Children were paid less than adults and were easier to bully and coerce. One survivor of childhood labor described his life to a British Parliamentary investigation in 1832 (quoted from Sommerville 1982):

Have you ever been employed in a factory? — Yes.
At what age did you first go to work? — Eight.
Will you state the hours of labour at the period when you first went to the factory, in ordinary time? — From 6 in the morning to 8 at night.
When trade was brisk what were your hours? — From 5 in the morning to 9 in the evening.
With what intervals at dinner? — An hour [once per day].
During those long hours of labour could you be punctual; how did you awake? — I seldom did awake spontaneously; I was more generally awoke, or lifted from bed, sometimes asleep, by my parents.
Were you always on time? — No.
What was the consequence if you had been too late? — I was most commonly beaten.
Severely? — Very severely, I thought.

[3] https://archive.org/details/anessayuponnurs00cadogoog

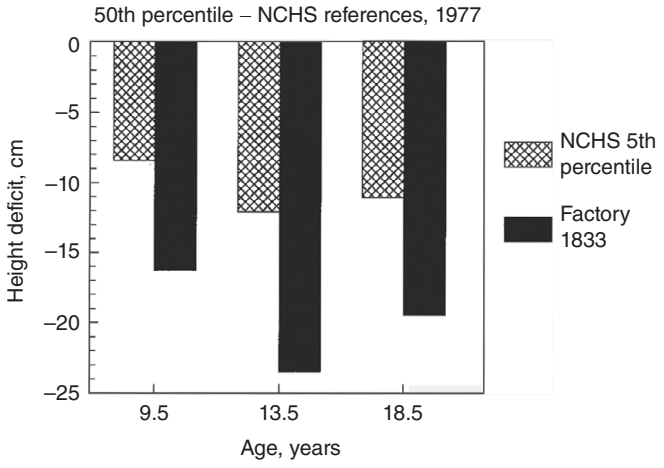


Figure 1.6 Height of English factory children in 1833 compared with the National Center for Health Statistics (NCHS) references. The heights of the factory children are shown as deficits, in cm, to both the 50th percentile and the 5th percentile of the NCHS references.

The testimony continues at some length and describes a life in the factories of fatigue and hunger, punctuated by beatings.

Medical hygienists, or what we call today Social Medicine and Public Health professionals, established the decline of health associated with urbanization and industrialization, by measuring the height and weight of people. In France, Louis-René Villermé (1782–1863) found in 1829 that military conscripts from mill or factory areas were too short and suffered too many disabilities to make them fit for military service. In England, Edwin Chadwick (1800–1890) published data on growth and health of factory children in his Report of the Commissioners on the Employment of Children in Factories (1833). Some of these data are reproduced in Figure 1.6, which compares the average height deficit of the English factory children (as reported by Tanner, 1981) against the international reference data for stature published by the United States National Center for Health Statistics (NCHS) (Hamill et al. 1977). In this figure, the "0" line represents the 50th percentile height (the "average") of the reference population. The factory children are 16.3–23.5 cm shorter than the NCHS 50th percentile, and 7.9–11.4 cm below the NCHS 5th percentile. In a group of children an average height below the 5th percentile is an indication of major growth delay and stunting, meaning very low body length-for-age. This magnitude of stunting is usually seen only in children with serious pathology. The majority of children growing up under conditions of poverty in the least developed nations of the world today have average heights above the 5th percentile of the NCHS references. At 18.5 years of age only the Pygmy populations of central Africa, who have hormonal and nutritional limitations, have smaller average heights – about 150 cm for Pygmies vs. 158 cm for the factory workers.

In response to these findings Friedrich Engels (1820–1895) campaigned extensively against the employment of children in English factories. Engels cited evidence

of stunted growth, spine and bone deformities, and the physical and sexual abuse of child workers. Some physicians, Engels proclaimed, said that the factory districts would produce "a race of pigmies" (Sommerville 1982, p. 146) by a kind of evolutionary decline. Engels may have misunderstood the workings of evolutionary biology, but his social approach to growth stunting was correct. Chadwick's report included Engels' concerns and that report, along with personal testimonies, led to the passing of the Factories Regulation Act (1833) in England. The Act prohibited the employment of children under the age of nine and stipulated that periods for eating and rest must be provided for older children during the work day. Age was determined by the state of dental maturation as assessed by the eruption of permanent teeth.

The relationship between tooth eruption and chronological age was verified in an extensive survey by Sir Edwin Saunders published in 1837. Saunders assessed the state of permanent molar eruption in English school children at ages 9 and 13 years, and showed that dental eruption was a better indicator of chronological age than height. By this work Saunders helped establish that development of the dentition is less influenced by the environment, while height, and therefore skeletal development, is more affected. In later years an appreciation of these differences would lead to the crucial concept of "biological versus chronological age" and research leading to the production of atlases of dental and skeletal development (topics discussed in more detail later in this chapter).

Across Europe much important research in Social Medicine and Public Health was conducted and government policies were influenced. It is not possible to review events in every country, but I do wish to draw attention to some neglected examples, usually because the literature is not in English and not available electronically. Fortunately, some of the forgotten publications have been recovered by medical historians and growth researchers (Hermanussen et al. 2018b; Žalnora & Miežutavičiūtė 2014). Germany and Eastern Europe were beacons for enlightened public health policies.

A Viennese physician, Johan Peter Frank (1745–1821), wrote a nine volume, 6,262-page treatise "*A system of complete medical police*" that investigated many different social and physical factors that impacted on human health. Some of this is translated in English (Lesky 1976). One of the worst factors, according to Frank, was poverty which leads to "ignorance and decay of culture in a society." Frank worried that undemocratic political and social policies, combined with a social Darwinism would lead to discrimination and poor health for the lower social classes (see discussion of eugenics in the next section).

Frank's concerns were amplified by Rudolf Ludwig Carl Virchow (1821–1902), a German physician, anthropologist, biologist, and politician. Virchow incorporated scientific methods into German social medicine, and never forgot the social side of human health with his famous observation that, "politics is nothing else but medicine on a large scale." Virchow was a brilliant pathologist, essentially creating this medical field, but he was an anti-Darwinist, claiming that the first Neanderthal remains discovered were of a modern human suffering from some rachitic disease, and a religious racist, condemning Catholics. Politics and social medicine came

together with the appointment of Tomasz Janiszewski (1867–1939) as Poland's Minister for Public Health in 1919. He believed that the main goal of public health was to prevent physical weakness and promote disease resistance. He focused on reducing the high levels of infant and maternal mortality, alcoholism, venereal diseases, cancer, and mental disability. Janiszewski's approach was to improve hygiene by the development of plumbing and refuse management, and to increase physical activity. This would be accomplished by physical education and hygiene education programs in schools. The later would promote new concepts of restrictive marriage and reproduction following the eugenics movement.

In the city of Vilnius, sometimes part of Poland and now capital of Lithuania, Kazimierz Karaffa-Korbutt (1878–1935), and colleagues at Stephen Bathory University produced much research and publications on the fields of labor hygiene and social hygiene. Labor hygiene included shortening the working day to eight hours, banning child labor, protecting women from dangerous jobs, especially when pregnant, and allowing for education and worker training. Lithuanian leadership in promotion of public health ended after World War II with that nation's incorporation into the Soviet Union.

There was much concern with nutrition among European pediatricians and public health workers. Terrible living conditions of industrializing cities, unsafe food transport and storage, widespread poverty, and nearly constant warfare combined to create a negative synergism between food shortages and infectious disease that diverted available food and nutrients away from healthy growth. Several German and Italian discoveries were made relating to seasonal variation in growth, dissociation of growth in height and weight, the value of mid-upper-arm circumference as a proxy for nutritional status, and the value of the ponderal index ($100 * \sqrt[3]{\text{weight}} / \text{body height}$). The last, proposed by Rudolfo Livi (1856–1920) in 1897 meant to estimate health along a continuum from excessive leanness to excessive fatness, is a precursor of the **body mass index** ($\text{BMI} = 100 * \text{weight/body height}^2$). More about the use and abuse of BMI and other estimates of fatness is discussed in other chapters. Livi applied the ponderal index in his study of the health of 300,000 Italian soldiers, published in several articles and books between 1896–1905. This study of *Military Anthropometry*, as Livi called it, established the methods for future such studies across Europe, North America, and elsewhere.

Extensive surveys of German school children, juveniles, and adolescents during and after World War I focused on starvation, refeeding, and its effects on body growth. Fritz Goldstein (1888–?1939), a German pediatrician, reported in 1922 that the refeeding of starved children resulted in a rapid recovery in height, about 3 cm in only 6 weeks, but no further increase once the deficit was corrected, even when abundant feeding was continued. Another pediatrician confirmed and extended these findings. Eugen Schlesinger (1869–1937, Box 1.1), published *Das Wachstum des Kindes* (The growth of the child) in 1925. He reported that on refeeding there was an immediate and marked increase in height growth, on the order of " . . . 3 or 4, or even 5 cm" and then over the following months there was a slower recovery in weight. The saltation in height velocity is today known as catch-up growth, of which more will be presented later in this book.

Box 1.1 **Persecution of Jewish pediatricians in Nazi Germany**

The work of Goldstein and Schlesinger was representative of other German pediatricians, especially those of Jewish heritage. It is estimated that in 1933, when the National Socialists (Nazis) came to power in Germany, 0.9% of the population was Jewish and that 15% of all medical doctors were Jewish (Saenger 2006). There were 1,253 pediatricians and of that total 611 (47%) were Jewish. Why this is so is not known. Perhaps it was an honorable religious/social tradition to care for the most vulnerable infants and children, reinforced by the rising tide of professional and political interest in social medicine and public health. Or, perhaps it was due to segregation within medical practice that prevented Jewish physicians from more prestigious specialties, such as surgery. Such segregation by gender persisted throughout the twentieth century in the United States, with more men in surgery and internal medicine and a " ... concentration of women in pediatrics, family practice, and obstetrics–gynecology" (Boulis et al. 2001, p. S67).

The Nazis wanted to create a genetically and racially pure Aryan state and a focus on children and pediatrics was vital to this cause. The Nuremburg Laws of 1935 declared that only workers and professionals with "German blood" could serve the state and this excluded Jewish physicians. These laws were the culmination of German anti-Semitism that had been growing since the last decades of the nineteenth century. All Jewish pediatricians were fired from their positions at hospitals and other state medical services. They could practice privately, but not using the title Medical Doctor, rather the title "*Behandler*" – "treater" or "provider." Eugen Schlesinger was one who was so humiliated. He received his medical degree in 1893, served as a pediatrician and school doctor in Straßburg (then part of Germany) until that city was returned to France after World War I. In 1919 he moved to Frankfurt am Main and served as a school doctor and as professor at the pedagogic academy. The Nuremburg Laws resulted in his removal from both positions. He wrote in 1936, "After the local medical society has excluded the non-Aryans from membership, I feel compelled to withdraw from the other medical associations. Moreover, my revenues have declined so much that I can no longer afford to be a member despite my nearly 40-year-old tradition" (Seidler 2007, translated from original German). Schlesinger died in 1937, apparently of natural causes as there is no note of suicide or deportation to a concentration camp in Seidler's book. Many other Jewish pediatricians suffered far worse fates. Readers may consult Seidler's (2007) book, which is a history of persecution against Jewish medical professionals and a compendium of biographies of Jewish pediatricians. Two other sources are Saenger (2006) and Adelsberger (1946), a pediatrician who provides her first-hand account of humiliation, deportation, and experience in concentration camps.

"Race" and Growth

The Nazi social policies and atrocities of the 1930s and 1940s had their origin in other countries, especially Great Britain and the United States. By the second half of the nineteenth century a highly contentious political debate involved the use of growth data. Starting in 1875, Henry Pickering Bowditch gathered measurements of height and weight, taken by school teachers, of 24,500 school children from the Boston, Massachusetts area. In a series of reports published in 1877, 1879, and 1891 (e.g., Bowditch 1877), Bowditch, a physician, followed the social medicine approach of Villermé and Chadwick, and also applied modern statistical methods (following Quetelet) to describe differences in growth associated with sex, nationality, and socioeconomic level between different samples of children. Bowditch was the first person to construct percentile growth charts for children (Bowditch 1891), following the statistical method of Galton (see below). The NCHS growth charts discussed above are the modern descendants of Bowditch's work. Bowditch found that children of the laboring classes were smaller by about 1 inch (2.54 cm) than children from the nonlaboring classes and that across both classes, Anglo-Saxon American children were taller than the children of Italian immigrants. To account for these observations, Bowditch wrote that child height depends " . . . partly on the greater average comfort in which such [non-laboring] children live and grow up and partly upon differences in race or stock" (Bowditch 1877, p. 35).

After further analysis and consideration of English data showing differences of up to 4 inches (10.2 cm) between working-class and nonlaboring-class children, Bowditch revised his "environment x genetics" interpretation to place more weight on the environment, writing that, " . . . comfort and misery affect the growth of children . . . Taking these circumstances into consideration, it will probably be safe to conclude that the importance of mode of life, as a factor in determining the size of growing children in this community, is at least equal to, and possibly even greater than, that of race" (Bowditch 1879, p. 54). The methodological, scientific, and social innovations of Bowditch, as well as his services to medical education, were held in very high regard and after his death a Biographical Memoir was published by the United States National Academy of Sciences, written by the esteemed physiologist Walter B. Cannon.[4]

This conclusion ran counter to that of Francis Galton (1822–1911). In his book *Natural Inheritance* (1889) Galton demonstrated both the use of percentile charts and the **heritability** of adult stature and other physical traits. Galton, a cousin of Charles Darwin, read *Origin of Species* (Darwin 1859) and came to the false conclusion that heredity was the all-powerful determinant of human form and functional capabilities. He misinterpreted the principle of natural selection to mean selective breeding could be used to improve the physical and psychological state of the human species. With this belief, Galton created the eugenics movement, a pseudo-scientific social and political crusade of controlled breeding (Gillham 2001). At its worst, during the 1930s American eugenicists persuaded courts to order the forced sterilization of

[4] www.nasonline.org/publications/biographical-memoirs/memoir-pdfs/bowditch-henry-p.pdf

people with physical and mental handicaps and advised German Nazis, who extended the advice to the extermination of 11 million Jews, Gypsies, political dissenters, and even "Aryan" children with disabilities such as vision and hearing impairments, and other "undesirables" during World War II (Kühl 1994).

Eugenicists held that the laboring classes were genetically inferior to the non-laboring classes. One proof of this inferiority was their short stature. Eugenicists also believed that the "race," or ethnic origin, of American-born children could easily be determined based on physical measurements, as these sizes and shapes of body were, supposedly, fixed by genetics. Very little, of course, was known about genes or their actions in the first half of the twentieth century. Nevertheless, according to the eugenicists, "racial" admixture via intermarriage, especially between tall "Anglo-Saxons" and short people from southern and eastern Europe, would bring about a physical degeneration of Americans.

The words "race," "racial," and similar terms are set in quote marks throughout this book to indicate that for the human species "racial" categories denote socially defined groups and not biologically or genetically justifiable classifications. Several scientific societies, including the American Association of Physical Anthropology, the German Society for Anthropology (Gesellschaft für Anthropologie), and the American Anthropological Association, have published statements on the meaning of the term "race." "Race" may be socially defined, but its influence has measurable effects on human phenotypes in terms of growth, development, maturation, and risk for disease (Gravlee, 2009).

Previously mentioned in the Introduction to this book was the research of Franz Boas, a German-born anthropologist working in the United States. Boas combined the power of anthropometry with the statistics and social medicine of Bowditch to attack and dismantle the prevailing "racial" typologies of the eugenicists (Boas 1892). Boas investigated the anthropometry of immigrants and their descendants in a project supported by the United States Immigration Commission. His report was published in the United States Congressional Record in 1910 and in revised form in the journal *American Anthropologist* with the title "Changes in the Bodily Form of Descendants of Immigrants" (Boas 1912). The title summarizes Boas' findings that so-called racial types were transformed from one generation to the next with a change in environments. These changes occurred in a time frame that was too short for any explanation by natural selection or any other genetic mechanism.

Boas' main findings for changes in height, weight, and head shape were given on the first pages of his 1912 article. The word "type" is a synonym for "race," that is a genetically determined phenotype. His findings, copied verbatim, were:

1. American-born descendants of immigrants differ in type from their foreign-born parents. The changes which occur among various European types are not all in the same direction. They develop in early childhood and persist throughout life,
2. The influence of American environment makes itself felt with increasing intensity, according to the time elapsed between the arrival of the mother and the birth of the child,

3. The observations on intraracial heredity show an increased variability of children of dissimilar parents, which proves a regression of the children to either parental type, not a regression to the mid-parental type,
4. The average stature of children decreases with the size of the family.

Boas wrote that he did not know the causes for the changes in physical growth or the positive association of stature with length of time in the United States and the negative association of stature with greater family size. Quoting Boas again, "As long, then, as we do not know the causes of the observed changes, we must speak of a plasticity (as opposed to permanence) of types . . . " (Boas 1912, p. 557). Boas may have been the first biologist to use the word "plasticity" to describe these changes in bodily form. Boas helped to discover the variability of the **norm of reaction** of human phenotypic development to changing environments. Ever since Boas, studies of human plasticity and reaction norms have been a central focus of biological anthropology and other fields. In many ways, Boas opened the way toward research into an epigenetic basis for human biological variation, which is a topic of Chapter 6.

The **plastic** changes in growth discovered by Boas applied to both the laboring and nonlaboring classes. It is sometimes assumed that Boas ascribed this plasticity to the better health care and nutrition received by the children in the United States. This is not the case: "I do not consider this likely, because the conditions under which the immigrants live are not favorable; but this suggestion is worth following up as one of the possible contributory causes" (Boas 1912, p. 558). Boas did present detailed arguments against genetic, natural selection, and other explanations favored by eugenicists.

Bowditch had earlier found the similar effects of migration on growth but in smaller samples of children, for example, that German-Americans were taller on average than Germans in Germany. What both men were able to show was that the American-born offspring of immigrant parents from different national, ethnic, and religious origins grew up to look more like each other in body size than they looked like their parents. Thus, Bowditch and Boas proved statistically that the eugenicists claim that ethnicity could be determined by physical measurements was not true. Bowditch concluded that his research disputed the " . . . theory of the gradual physical degeneration of the Anglo-Saxon race in America" (Boyd 1980, p. 469). Despite the scientific findings, eugenicists and politicians called for quotas on the immigration of so-called inferior peoples into the United States. The work of Boas may have helped delay the imposition of limitations on immigration. Nevertheless, the American Congress eventually passed the "Immigration Restriction Acts" in 1921 and 1924, which specifically targeted southern and eastern Europeans and Asians for migration quotas (Gould 1981).

Boas and the anti-eugenicists may have lost that political battle, but their work influenced the following generations of anthropologists, public health workers, epidemiologists and others. Their work did establish that undernutrition, poor sanitation, lack of health care, illiteracy, and poverty are correlates of physical and mental growth failure. Documentation of the pernicious effects of these conditions on the growth and development of infants, children, and adolescents continues to be

carried out, and, in the tradition of Villermé, Chadwick, Bowditch, and Boas, recommendations for action to alleviate this suffering are made by researchers who have " . . . a feeling of responsibility for the children's welfare" (Borms 1984).

Twentieth-Century Research

Boas' scientific discoveries also include his research into the methodology of growth studies (Boas 1892, 1930). One of his enduring contributions is the importance of calculating growth velocities from the measurements of individuals rather than from sample means. As shown in Figure 1.7, the former method gives an accurate estimate of average growth rate, while the latter method mixes data from early, average, and late maturing children and results in a mean velocity curve that underestimates the actual velocity of growth of all children during the adolescent growth spurt. Boas also provided a formal appreciation of the concept of the tempo of growth to understand the difference between early and late maturing individuals. The effects of maturational timing are most evident at the time of the adolescent growth spurt, but they are present at all stages of life – even during the prenatal period. Early maturers are always ahead of late maturers in skeletal development and other indicators of biological maturation. With both methodological and conceptual advances such as these, and the descriptive knowledge gained since the time of Buffon, the modern era of growth measurement and analysis began.

In the first half of the twentieth century several large-scale longitudinal studies of growth were started in the United States and Europe. In addition to better quantification of amounts and rates of growth of healthy children, these studies made use of

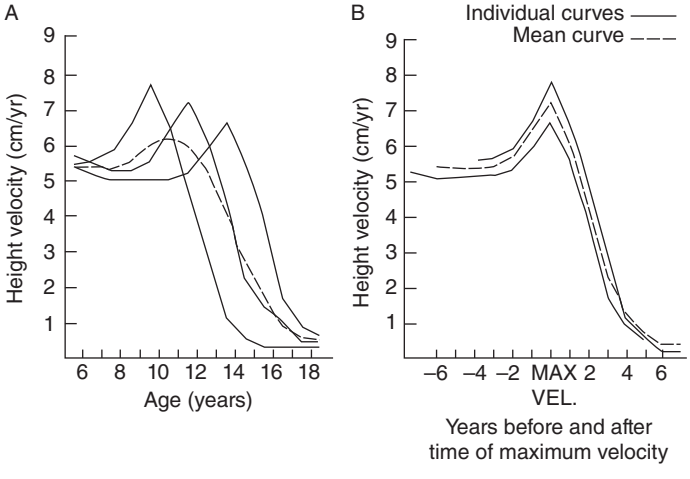


Figure 1.7 (A) Individual velocity curves of growth (solid lines) and the mean velocity curve during the adolescent growth spurt. The mean velocity curve does not represent the true velocity of growth of any individual. (B) The same curves plotted against time before and after peak height velocity (maximum velocity) of each individual. The mean curve accurately represents the average velocity of the group.

new technologies in radiology, physiology, and psychology to characterize the biological maturation of body systems. Equally important, these longitudinal studies represented a new philosophy about human growth and development. Research workers and politicians became interested in the causes of individual differences between people. Perhaps this interest was a consequence of the work of Boas on the plasticity of reaction norms of growth and physical development following a change of environment. It may also have reflected the American cultural ideals of "individualism" and how to ensure continued production of the same. American "individualism" was enshrined in President Herbert Hoover's 1928 speech, "Rugged Individualism" which advocated less government interference in human affairs, including a laissez-faire capitalism. Eugenicists applauded this political philosophy because it promoted a type of Social Darwinism. But even Boasians were attracted to the idea of individualism and its determinants. Lester W. Sontag (1901–1991), who directed the Fels Longitudinal Study in Yellow Springs, Ohio from 1929 to 1970, wrote " ... that modern understanding of the growth, health, behavior, and effectiveness of human beings could only be understood if the nature and significance of individual characteristics of each child's physiological, biochemical, nutritional, educational, and environmental characteristics could be assessed and integrated into a total picture" (Sontag 1971, p. 988). Sontag summarized this approach as the study of "the whole child."

The American Studies

The American longitudinal studies were supported in their early years by private donors. The Rockefeller Foundation and the Laura Spellman Rockefeller Memorial Fund were major sources of financial support. Lawrence K. Frank (1890–1968) was an administrator at both funds and helped start and maintain virtually all the major longitudinal studies. Eventually there was also public support for longitudinal research on human growth. The United States National Research Council created the Committee on Child Development in 1923 leading to several White House Conferences on Child Health. Specialized journals such as *Child Development*, started in 1929, and *Growth*, first published in 1937, also appeared. By this time the study of normal growth was of national importance, both for its scientific and political value.

Several large, expensive long-term studies were initiated. These include the Fels Study mentioned above, the University of Iowa Child Welfare Station Study, the Harvard Growth Study, the University of Colorado Child Research Council Study, the Brush Foundation Study of Western Reserve University (Cleveland, Ohio), and several studies at the University of California, Berkeley. Sontag (1971) stated that in general these research programs shared several features. First, they were interdisciplinary with physicians, psychologists, anthropologists, and others taking a global approach – they studied "the whole child." Second, they were fastidious in terms of the methodology of data collection. Third, they collected data as an end in itself, as they posed no research questions or scientific hypotheses about human growth to be addressed. Fourth, they planned to continue data collection for 15 years or more.

With one exception, all of the American longitudinal studies ended when either funding disappeared, the justification for data collection without purpose could not be sustained, or, most importantly, the philosophy of the "whole child" research approach was abandoned. The only one of these studies still active today is the Fels Institute Longitudinal Study, which began in 1929 with funding by Samuel Fels, president of the eponymous soap manufacturing company. The original sample of the Fels study were healthy, well-nourished boys and girls, living in small urban communities and rural areas of southwestern Ohio. Participants in the study were measured longitudinally, ideally once a year or more often, from birth to maturity for height, weight, and a variety of other physical and psychological characteristics. The study enrolled newborn siblings of the original participants, their children and their grandchildren and great-grandchildren. Measurements were extended to include the adult and old age years. In 1977, the Fels Fund donated the Institute to Wright State University School of Medicine, and the Institute was absorbed into the School of Medicine.[5]

Alex F. Roche (1921–2017) became director of the Fels study after Sontag. Roche wrote a history of the study in which he stated that the Fels study remained viable because members of its staff were willing to use the data to answer important questions about human growth, development, and health (Roche 1992). As an example, both Sontag and Roche cite the work of Stanley M. Garn (1922–2007), a member of the Fels team from 1952 to 1968, on the development of fatness from birth to old age, and the relation of growth in early life to health and disease in adulthood (Garn 1958, 1970). These were new directions in research in the 1950s and 1960s, and have become "normal science" today. As early as 1935, Fels data were used to study the effects of smoking on the fetus – smoking increases fetal heart rate (Sontag & Wallace 1935). New methods of assessing biological age from **skeletal maturation** of the hand and wrist, and from the knee, were developed with Fels radiographs. The United States pediatric growth charts from 1978 to 2000 for children from birth to 3 years of age were produced exclusively from Fels data.

Other notable consequences of the American longitudinal research program include the work of Frank K. Shuttleworth (1899–1958) who used the data of the Harvard Growth Study to design new statistical methods to analyze **longitudinal data**. His first major report on this was published in 1937 (described in the next chapter), and many of his methods are still used standardly today. Howard Meredith (1903–1985), long associated with the University of Iowa Growth study, focused much attention on population, geographic, and sex-related differences in growth. He was instrumental in applying many new mathematical techniques to the description of growth curves and did much to sort out some of the genetic and environmental determinants of human growth.

The Brush Foundation and Spelman Fund supported a longitudinal study at Western Reserve University in Cleveland, Ohio. Katherine Simmons (no dates found)

[5] https://medicine.wright.edu/epidemiology-and-biostatistics/fels-longitudinal-study-collection

and T. Wingate Todd (1885–1938) produced reference standards from ages 3 months to 13 years for height and weight from these data. It is necessary to comment on the lack of life dates for Katherine Simmons and some other women researchers cited here. Women scientists, including Edith Boyd mentioned earlier and Katherine Simmons, were not only under-represented in the research literature, but also are less likely to have biographic information recorded, even in this day of the Internet and Wikipedia. Publications on the history of human growth research have little data on women. I have tried to find and include the contributions of women in this brief historical review.

Simmons and Todd also analyzed the correlation between growth in height, weight, and sexual maturation over time. Simmons and Todd came to the important conclusion that weight was not as reliable an indicator of healthy growth as height. This is because weight is the sum of many body tissues and includes fat and water in the body. Each of these tissues and components of the body can vary with age, sex, nutritional status, state of health, physical activity, etc., which make weight too imprecise a measure of health (Simmons 1944).

Todd also used the hand-wrist radiographs collected during the study, and an extensive series of human skeletons he collected, to publish the first major *Atlas of Skeletal Maturation* (Todd 1937). Todd's method to assess skeletal maturation was based on first selecting a "representative" radiograph for each age and sex. Analysis by sex was required because Todd and others had already found that the rate of skeletal development of girls is advanced over that of boys. For example, from all the radiographs of eight-year-old girls, Todd found the one that had both an equal degree of development of all the bones in the hand and wrist (and there are 28 such bones, or **ossification** centers to consider), and also a degree of development that was average among all the eight-year-old girls (see Figure 2.4 for an illustration of hand-wrist radiographs). This process was repeated separately for eight-year-old boys, and then repeated again at every age. These "average" radiographs then became the standard against which all other radiographs were judged. Todd's *Atlas* was used for many years and was revised in the 1950s into the form still used today (Greulich & Pyle 1959).

In 1929 B. Holly Broadbent, also working with Todd and Simmons, established a longitudinal study of cranio-facial growth and development, including X-rays of the skull. The resulting book (Broadbent et al. 1975) remains one of the fundamental reference works for orthodontists, plastic surgeons, and human biology researchers.

Interest in the study of body composition may have been stimulated by Simmons and Todd's work. Major studies of body weight and its division, "... into 'fat' and 'lean' components ... " (Baumgartner 1997) began in earnest in the 1940s with the work of Albert R. Behnke (1903–1992). In the 1950s Ancel Keys (1904–2004) and Josef Brozek (1913–2004) expanded these studies to separate body composition into components, " ... of water, protein, minerals, carbohydrates, or glycogen ... " (Keys et al. 1950).

In 1928 the first of several longitudinal studies, The Berkeley Growth Study, was started at the University of California, Berkeley. Nancy Bayley (1899–1994) was

director until 1954, and she fully subscribed to the "whole child" philosophy of these studies. Bayley was trained as a psychologist and one of her most notable contributions is the *Bayley Infant Scales of Motor and Mental Development*, which she created in 1940. She also received training in anthropometry from Sarah Idell Pyle (1895–1987) at Iowa. Bayley not only collected physical growth data (22 different measurements) but also analyzed it in novel ways. There were 61 infants recruited for the study, 31 boys and 30 girls, and 47 were followed to maturity. After 3 years of age, and then again after 10 years and at maturity, Bayley calculated correlation coefficients between the measurements. These correlations indicate the amount of relationship between the measurements. Stated another way, the correlations can be used to estimate the degree of predictability of one measurement from another measurement, say the predictability of weight if stature is known.

Bayley was the first to find that correlations are much lower from birth to six months of age than from six months to one year of age. Correlation between measurements continues to rise until the age of two years (Bayley & Davis 1935). Thus, it seemed that an infant's individual pattern of growth is a bit disorganized just after birth, but becomes better organized during the first two years of life. This discovery had important practical implications for the prediction of adult size at early ages. In 1952 Bayley and S. R. Pinneau published the first tables for predicting adult height using height and **skeletal age** measured at earlier ages. These prediction tables are still used by physicians and researchers.

In addition to work on longitudinal patterns of growth in external body size, several studies of the growth of internal organs and chemical composition by Richard E. Scammon (1883–1952) and Edith Boyd (1895–1977) are of importance. Figure 1.8 reproduces one of Scammon's classic illustrations of growth of the internal organs, and the body as a whole, during postnatal life. By necessity, this work was based on cross-sectional samples of cadavers, but Scammon and Boyd took a longitudinal approach when presenting their data. Their studies of the internal milieu of the human body started with the embryo and continued until old age. With this research the scientific and medical world gained its first accurate understanding that in terms of chemical composition, "The organic body is not a closed system. It receives and liberates materials constantly" (from Scammon's "Introduction" to Boyd et al. 1980). These studies of internal organs showed that the parts of the body do not develop or grow according to one grand pattern, rather there are several different curves of growth for different types of tissues.

The European Studies

European longitudinal growth studies were slower to start than in the United States and were probably delayed by the World Wars. Prior to 1949, there were two notable studies with a longitudinal component. The first was a longitudinal study started in 1919 and conducted by Rachel Mary Fleming (1882–1968). For the first eight years of this study Fleming contributed her labor, working in her spare time from her duties as Assistant Secretary of the Geographical Association. In 1927, she received a grant

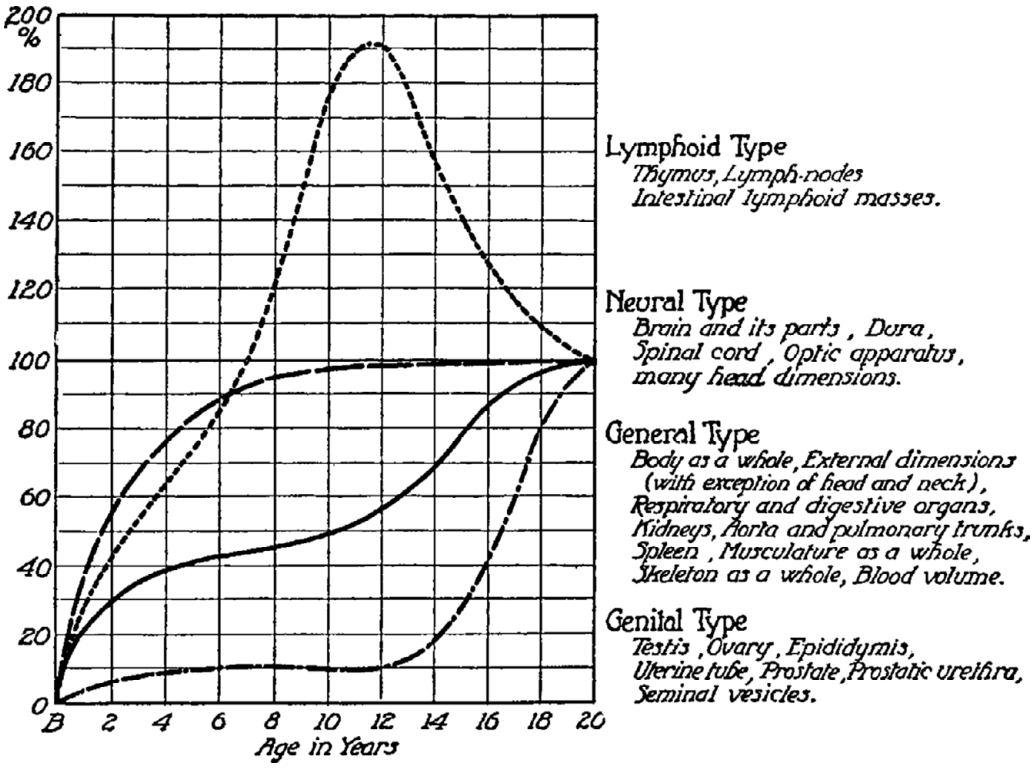


Figure 1.8 Four major types of postnatal growth curves from birth to 20 years, expressed as the percentage of the total increment of growth. (Scammon 1930)

from the Medical Research Council to devote full-time to the data collection, analysis, and publication of a report. The growth study had been proposed by Herbert John Fleure (1877–1969), a zoologist, geographer, Honorary Secretary of the Geographical Association, and editor of *Geography*. Fluere was motivated by the debate between eugenicists and Boasians. Fluere wanted to establish the permanence of "racial types" and considered the idea of plasticity to be extreme and that could be " . . . discarded as serious anthropology" (Fleure & James 1916). Useful historical background to the debate between eugenicists and Boasians and the misuse of anthropometric data by the eugenicists was published by in the book *Mixed Race Britain in the Twentieth Century* (Caballero & Aspinall 2018).

Fluere considered that the Welsh and the English were distinct racial types and that the Welsh were descendants of a crossbreeding in 2,500 BC of Anatolians and Mediterraneans that had created a "race" of restless mariners that Fluere named The Prospectors. This fanciful and totally incorrect idea was published by Sir Arthur Keith (1866–1955), in an essay "How did Britain's racial divisions arise?" (Rich 1990, p. 240). Keith, an advisor to Fleming's longitudinal study, was a prominent anatomist and anthropologist, fellow of the Royal College of Surgeons, a eugenicist, wrote derogatory essays on the nature of Jews and Jewishness, and was the chief supporter

of the fraudulent Piltdown Man fossil as evidence of an English-first evolution of *Homo sapiens*. Keith could serve as the "poster-boy" for a "Do not trust authority" campaign!

The growth study conducted by Fleming recorded the annual measurements of stature and head dimensions on children and youth 3–18 years old in England and Wales. From these data Fluere hoped to substantiate his opinion that, " . . . for in every parish of our country markedly contrasted types live side by side, and yet, in spite of intermarriage as well as substantial similarity of conditions, they remain distinct" (Fluere 1923, pp. 84–85). Apparently, Fluere believed neither in genetics, which even the eugenicists understood would have some effect on "type" via intermarriage, nor in environmental plasticity. Rather, he believed in immutable "racial types."

Fleming published her report of the longitudinal study in 1933 and her analysis included a longitudinal curve of growth in stature, head length, and head breadth for each participant in the study – there were more than 12,616 examinations of 4,293 children, juveniles, and adolescents (Fleming 1933). With statistical advice from the Medical Research Council, especially Mr. W. J. Martin, she produced thousands of graphs, not only for individual participants, but also groupings of participants. These graphs were made with reference to age, sex, stature, cranial measures, hair and eye color, and an attempt at "racial" and psychological classification. Although Fleming and Fluere continued to see "racial types" in the results, they had to divide the participants into 15 such groups, not just Welsh v. English. A modern interpretation of the data, both the central tendencies and variance, comes to a different conclusion, namely that there is abundant evidence of growth plasticity, especially in association with living conditions and food availability. Even Fleming seemed to lose faith in the idea of "racial type permanence," as she wrote, "It is admittedly difficult to classify groups according to racial type (which is, after all, merely a convenient term for summing up predominating physical characters) in the population of these islands where so much intermixture has taken place . . . " (Fleming 1933, p. 54).

With the individual approach Fleming noted that some children grew throughout the teenage years, while others came to an early stop. Some children grew steadily during puberty and others experienced a rapid and distinct burst of growth followed by an end to growth. Some children were tall until puberty but then ceased growing and remained shorter than average as young adults. Today these patterns of growth are well known and reflect normal individual differences in the "mode and tempo" (as Boas, 1930 called them) or "amplitude and tempo" (as Ramsay & Hermanussen 2014 call them) of growth.

Fleming also noted 40 individuals with unusual stature graphs. These children, of different ages, showed a longitudinal pattern of: (1) normal growth; (2) then a period of growth arrest; (3) and finally a resumption of normal growth. None of these children had been identified as growth-delayed during the cross-sectional medical examinations by the school physician. All attended a small village school and lived so far away from the school that they had to bring lunches to school, whereas other children attending the school, but who lived in the village, went home for dinner. The bag lunches were usually bread and jam, while the home dinners, the main meal of

the day, were usually a hot lunch of meat and vegetables. The typical evening meals for all the children were bread, jam, and tea. Thus, only on weekends did the bag lunch children receive a full meal at dinner. The cause of the growth stunting seemed to be a combination of inadequate food intake, plus the energy expended in the commute to school. When the Headmaster of the school initiated a hot dinner program for the "commuting" children the growth differences between the village residents and rural students disappeared.

The growth study found that so-called racial types proved to be a product of nutritional status. This and a century of other research proved Boas was correct about plasticity. Fleming's serendipitous finding of alterations in the pattern of growth associated with a change in school feeding policy was to be repeated by another British researcher, Elsie M. Widdowson (1906–2000), in the aftermath of World War II. Widdowson (1951) found that the psychological well-being of German children, orphaned during World War II, directly affected their growth. Children in two orphanages that Widdowson called "Bienenhaus" and "Vogelnest," took part in a year-long nutrition supplementation experiment. Children of both orphanages were fed their usual ration for six months. In the second half of the study, the children of Vogelnest were given the usual ration plus additional, unlimited servings of bread, jam, and orange juice, but no supplement was given at Bienenhaus. Unexpectedly, during the first six months, when both groups received identical rations, the children of Vogelnest put on more weight, an average of 1.4 kg, than the children of Bienenhaus, an average less than 0.5 kg. Six months later the situation was reversed. Despite receiving extra food, the children of Vogelnest gained less weight (average gain of about 1.2 kg) than the children of Bienenhaus (average gain greater than 2.5 kg). However, eight of the children had gained an average of about 4.2 kg in the supplemented orphanage. Quite coincidentally, they had been transferred from Bienenhaus to Vogelnest just at the time when the supplement was given to Vogelnest. The records also revealed that the headmistress transferred between orphanages at the same time. Further inquiry by Widdowson found that the headmistress, a woman referred to as Fraulein Schwarz, was a stern disciplinarian, who severely punished children for even minor infractions of behavior. She delivered these punishments at mealtime, possibly to provide an example to the assembly of children gathered for the meal. The few children with favorable growth under the care of Fraulein Schwarz, were her "favorites" who received no punishments, and when she transferred between orphanages she took her favorites along. Only these children, provided with positive psychological stimulation, showed the benefits of the extra food.

Widdowson concluded that the emotional environment of the orphanages had a greater influence on physical growth than did the nutritional environment. A serious limitation of the German orphanage study is that carefully controlled observations of behavior, food consumption, and energy expenditure did not take place. Widdowson's conclusion, though important in many ways, cannot be accepted at face value. Since the time of Widdowson's research a great many studies have reported that a negative emotional environment can suppress physical growth, even when an

adequate diet is available and there is no evidence of infection or other illness. Some of these studies are discussed in detail in Chapter 7.

The point to make here based on the work of Fleming and Widdowson is that much scientific discovery is unexpected and found only when the individual researcher is willing to take the risk of exploring new research territory, trying different strategies and methods, and thinking in new ways about how things work. Rachel Fleming is not well known today, but she exemplified these characteristics of noteworthy research. She played a part in changing social policy, especially the introduction of free school lunch programs for undernourished children and the need to identify such children.

Fleming's work also formed a basis for the discovery that children's nutritional requirements are divided between maintenance and repair of the body, work, and growth. When food intake is inadequate to meet all requirements, it is usually growth that suffers first. Later in life, nutritionally growth-stunted children become adults with reduced performance in both physical and mental work capacity. In retrospect this seems obvious, but it took decades of research, hundreds of millions of dollars, and much intellectual debate to reach this conclusion (see Chapter 7). With the advantage of her longitudinal data, Fleming also performed statistical analyses to reveal several features of the human adolescent growth spurt: " . . . The results show that the fast-growing period for girls starts sooner, finishes earlier, and is less intensive than for boys. The sexes are equal up to 11 years of age, between 11–14 girls are taller than boys, but from 14 onwards the boys become steadily taller than the girls" (Boyd 1980, p. 374). These well-known features of human growth at adolescence were new discoveries to late nineteenth and early twentieth century growth researchers. Many were puzzled by the growth spurt, some denied it really existed, and others had their Victorian sensibilities offended by the transitory "ascendancy" in stature (as Tobias 1970 referred to it) of girls over boys during adolescence.

Fleming's study is not mentioned in James M. Tanner's history of human growth research, but is cited in the book by Edith Boyd and colleagues (Boyd 1980). Overshadowed by Fleure and overlooked by Tanner and other twentieth-century male academics, Fleming was a serious and productive scholar in several fields. She and Fleure elevated the field of geography to a central position in British universities. The University of Wales conferred on her the degree of M.Sc., *honoris causa*, in appreciation of the work she had done for geography (in 1917 Fleming attended a summer school in geography at the University College of Wales, Aberystwyth, Wales). In 1922 she published a book of folktales for young readers, *Ancient Tales from Many Lands*. She was a historian of Russia and spoke Russian and other languages. From 1932–1934 Fleming served as Librarian of the Royal Anthropological Institute (RAI) in London. As well as her duties superintending the Library, cataloguing the books, and indexing the periodicals, she was to prepare the RAI journals *Man* (now titled *The Journal of the Royal Anthropological Institute of Great Britain*) and *Occasional Papers* for the press. Perhaps unsurprisingly in view of her heavy workload, by 1934 Fleming was obliged to resign due to ill health. Her friends,

including Fleure, secured for Fleming a Civil List Pension and for the last 20 years of her life she lived in retirement in Ventnor, Isle of Wight, and died there on March 23rd, 1968, aged 86. "Rachel Mary Fleming is one of the category of people who are often invisible even in institutional histories, one of those who do their work with quiet devotion, playing a pivotal structural role in the 'back room' rather than the 'front room' of the discipline" (Maddrell 2009, pp. 132).

The second longitudinal study of the early twentieth century was the Aberdeen, Scotland study conducted by Alexander Low (1868–1950). Low personally measured 21 dimensions of 900 newborn babies in 1923. He then remeasured 65 of the boys and 59 of the girls annually until they were 5 years old. The data were never properly analyzed until Healy and colleagues (Healy et al. 1956) produced a study of correlations between measurements taken at different ages. As part of their analysis, the authors also searched for the original participants of the Aberdeen study and found 42 of the men and 38 of the women. The adults were measured and correlations between the birth to age five-year data and the adult data were calculated. Healy and colleagues generally confirmed similar work by Nancy Bayley (discussed above). However, the Healy group was the first to try to explain the pattern of increasing correlations from birth onwards. They emphasized that late in pregnancy, the fetus may be "deflected very considerably" from its growth trajectory. After birth the infant slowly recovers toward that trajectory, "somewhat after the manner of a growing animal who has passed through a period of not too severe malnutrition" (quoted material from Tanner 1981, p. 347). A discussion of this perinatal "malnutrition" effect is presented in the section on birth, and birth size, in Chapter 2.

During World War II the Oxford Child Health Survey was started by John Ryle (1899–1950). A total of 470 infants and children were recruited and measured between the ages of one month to five years of age. The data include anthropometric measurements, illness histories, social changes in the families of the participants, and radiographs of the hand and wrist, knee, and chest. From an historical perspective, the X-ray data are most important. Roy Acheson (1921–2003) was a member of the team analyzing the data. To make use of the radiographs Acheson developed a new system to rate skeletal maturation (Acheson 1954). He was dissatisfied with the Todd method of assessing a bone age from the entire hand-wrist or a group of bones. Acheson wanted a simpler system and found that individual bones of the hand-wrist could be used to determine maturation. Acheson determined the number of identifiable stages of appearance of each bone as it matured. Each stage was given a number: 1, 2, 3, etc. A maturity score for any hand-wrist radiograph was easily calculated by giving each bone its stage number and adding up all the numbers. A higher numerical total meant a higher skeletal maturity score. The score could be treated just like any other measurement, such as height in centimeters, for statistical analysis. This simplified procedure for skeletal maturity was refined and standardized by Tanner and colleagues into a widely used clinical method (Tanner et al. 2001).

Soon after World War II, longitudinal research in Europe began in earnest. The British Harpenden study started in 1949 and continued until 1971 (Tanner, 1981). Other longitudinal studies were started in Paris, Zurich, Stockholm, London, and

Brussels. These studies were coordinated by the International Children's Center (ICC) in France. In terms of technical excellence and biological value the Zurich study is probably the most important (Prader et al. 1989). The longitudinal growth and development of 137 individuals of each sex were followed from their birth in 1954–1956 until adulthood in 1976. The authors published, "Distance standards of 20 anthropometric measurements such as weight, height and head circumference . . . as mean values and standard deviations or as median values (for weight and skinfold thickness) with smoothed empirical centiles. Velocity standards [were] provided for seven anthropometric parameters" (p. 1). The study also included annual hand-wrist X-rays for the estimation of skeletal age. Many articles were published and continue to be published based on the Zurich study data. There was an ICC sponsored study in the United States as well, the Louisville (Kentucky) Twin Study, which started in 1957 under the direction of Frank Falkner (1918–2003). This was a longitudinal study of twins (results are discussed in Chapter 7) and data collection ended in 2003, but may be renewed (Rhea 2015). The study has a Facebook page.

All the ICC studies followed a standardized procedure for anthropometric measurement and data recording. This was a major advance as earlier studies often used different methods of measurement and recording that made comparisons between data sets difficult. The focus of these ICC studies, and the Harpenden Study as well, was medical, and the research personnel were mostly pediatricians and endocrinologists. James M. Tanner (1920–2010), a member of the ICC board, stated that in contrast to the "whole child" approach of the American studies, the European longitudinal research was concerned more with growth pathologies. Important medical discoveries were indeed made by these studies, and there were also advances in understanding the basic biology of human growth. It is commonplace in biology that we come to understand how a normal system operates by studying what happens when things go wrong, that is, when there is a disease or other medical pathology. Tanner and colleagues advanced the cause of growth research in Europe by promoting many of these studies of growth pathologies and by publishing several major works. His book *Growth at Adolescence* (Tanner 1962), first published in 1955, summarized the state of knowledge of normal and pathological growth derived from all of the research of the first half of the twentieth century. Tanner and colleagues, especially his long-time research partner Reginald Whitehouse (1911–1987), also published some of the most widely used modern references for growth in height and weight, the stages of sexual maturation, and for skeletal maturation (Tanner et al. 2001; Tanner & Whitehouse 1976).

A specific type of longitudinal study is more common in Europe, especially the United Kingdom (UK), than in the United States or elsewhere. This is the birth cohort study. A cohort is a group of people who share a common characteristic or experience within a defined period (e.g., are currently living, are exposed to a drug or vaccine or pollutant, or undergo a certain medical procedure). A birth cohort is a group of people born during a defined period, which may be a week or a year. In the UK there are at least six birth cohort studies still running, the oldest being the Hertfordshire Cohort Study (HCS) that comprises 3,000 men and women born during the period

1931–1939 and still resident in the English county of Hertfordshire. The most recent is the Millennium Cohort Study (MCS), following the lives of around 19,000 girls and boys born in the UK in 2000–2001. The MCS is a multi-disciplinary research project collecting physical growth, family social life, cognitive performance, and other developmental data. Participants and their families were assessed at birth and then at ages 9 months and 3, 5, 7, and 11 years. Key findings from these surveys are that: (1) more than half of the MCS infants and children experienced poverty at some point in their first 11 years; (2) nearly 4 in 10 of the children have lived through at least one change in their parents' relationship status; (3) 1 in 5 children are obese by age 11 years; (4) most 11-year-olds report being very happy with their lives and their families; (5) social class background remains the most powerful predictor of 11-year-olds' cognitive abilities, assessed as their vocabulary, memory, problem solving and decision making. Social class is measured as a combination of parents' highest educational qualification and parental occupation. Not surprisingly, the highest scores on all cognitive measures were for 11-year-olds whose parents have more years of formal education, a higher academic degree, and higher income. A bit more surprising is that across all social classes, children from Indian-origin families scored higher than all other ethnic groups; Pakistani and Bangladeshi-origin children scored consistently worse (Sullivan & Brown 2014). The findings are broadly in line with what is known of the socioeconomic circumstances of the ethnic groups and is a contemporary example of American results reported by Bowditch and Boas for physical growth a century earlier. The UK birth cohort studies and other longitudinal studies are coordinated by the Centre for Longitudinal Studies[6] and CLOSER (Cohort & Longitudinal Studies Enhancement Resources).[7] The major birth cohort studies and other longitudinal studies still active in the UK are illustrated in Figure 1.9.

Longitudinal Studies in the Less Economically Developed Countries

The ICC also sponsored longitudinal studies in Africa: the Dakar and Kampala Studies. By the early 1960s some results of these studies were published, and the findings were puzzling. In some respects, African infants and children were advanced over Europeans, such as in skeletal development. In other measures, such as height, weight, and fatness, Africans were smaller than Europeans. Moreover, the differences between populations increased with age. Some researchers were content to ascribe these growth differences to genetics, but other scholars looked more to the ecology for human growth of Africa and its history. Poverty is the word often chosen to best describe the human ecology of Africa. The history of Colonialism was cited by some workers to explain the origins of much of this poverty. There was little hope for extensive longitudinal research in Africa to settle the question of "genes versus environment." By the 1960s, Europe was losing its colonies in Africa and there was

[6] www.cls.ioe.ac.uk/ [7] www.closer.ac.uk/

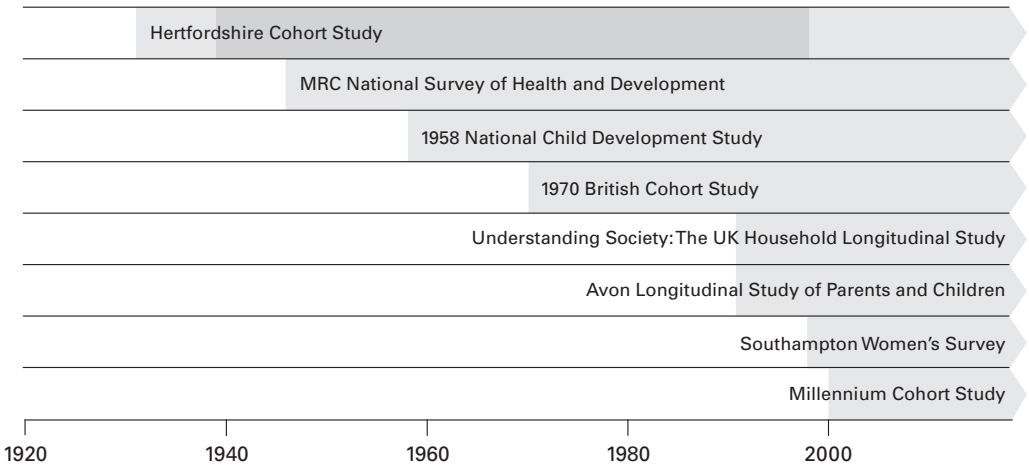


Figure 1.9 The major birth cohort studies and other longitudinal studies still active in the United Kingdom. From CLOSER (Cohort & Longitudinal Studies Enhancement Resources, www.closer.ac.uk/) with permission.

little popular sentiment in Europe, or North America, to invest the resources needed to conduct research.

In Central America research was underway to examine the environmental and genetic determinants of human growth. Robert MacVean (1917–2012), who was born in the United States and settled in Guatemala after World War II, helped to establish the American School of Guatemala as a laboratory school (one that conducts research and training in addition to teaching) in 1948. A longitudinal study of child, juvenile, and adolescent development was chosen as the basic research project of the laboratory school program. Data collection started in 1953 and until 1963 was limited to the students at the American School, an expensive private school. Beginning in 1963 other schools, representing middle and low socioeconomic status (SES) families in Guatemala City, were added to the study. The students attending each school were measured once each year for height, weight, hand grip strength, and number of erupted permanent teeth. A hand-wrist X-ray was taken. Several tests of cognitive development and school performance (e.g., IQ and reading tests) were also administered. As the measurements were taken from the entire school population of each year, this was not a pure longitudinal study. Children might have left or entered the school at any time, although many of the students did continue through the elementary grades and on to complete secondary school. This study, now called "The Longitudinal Study of the Growth and Development of the Guatemalan School Child," became one of the first large-scale mixed-longitudinal investigations of human growth (Bogin & MacVean 1983).

The mixed-longitudinal study is considered a very powerful statistical design for growth research. In this design, subjects of different starting ages are measured for several years. The overlap in age means that data covering much, or all, of the growing years may be collected in just a few years rather than two decades. In

addition, each age group serves as a check against other age groups to ascertain if the data collected are representative, that is, that the different cohorts of individuals of any age group (i.e., different years of birth) are typical in terms of the measurements taken. In a pure longitudinal study, peculiarities of the cohort of children, such as a disease or social stigma, may invalidate application of any findings to the population at large.

The data collection ended in 1999 and masses of data on over 40,000 individuals, some with 14 annual measurements, were collected, but little analysis was conducted until Francis E. Johnston (1931–2020), an American anthropologist, became a consultant to the study in the late 1960s. From 1974 to 1976 the present author (Barry Bogin, 1950), one of Johnston's students, did his doctoral dissertation research in Guatemala and became a consultant to the longitudinal study as well. I suggested adding measures of body composition (triceps and subscapular skinfolds and arm circumference) to better estimate nutritional status, and these have been taken on all participants since 1976. I also suggested adding a school with a Maya population. Maya are the majority ethnic group of Guatemala. Maya ethnicity is characterized by language (there are more than 20 Maya languages in Guatemala), traditional clothing styles, religious practices, and rules for cultural behavior (such as kinship and family organization). In many ways, the present-day Maya of Guatemala are the cultural descendants of the Classic Period Maya who constructed the ceremonial centers of pre-contact Mesoamerica (places such as Tikal and Palenque). Prior to 1976, the schools participating in the longitudinal study were comprised primarily of **ladinos** (attending all the schools) or Europeans/North Americans (attending the American School). Ladinos are the second largest ethnic group in Guatemala. In a sociocultural sense, *ladinos* are descendants of the Spanish conquistadors who ruled Guatemala for the past 400 years. In 1979 a Maya school, comprised mostly of children from very low SES families, was added to the study.

The important results of this study are not discussed here but are incorporated into many discussions of human growth and development throughout this book. From an historical perspective it is important to state now that the American School study, and its enlarged successor, was the first major research program of its type in any developing nation. Analysis of the study data continues today making it one of the longest-lived projects of its type, a resource of unparalleled data which remains to be fully exploited. Until 2016 most of the data existed as paper records and in the case of the hand-wrist X-rays as the original radiograph films. Some of the paper records had been destroyed and the X-ray films were deteriorating. The records and films were salvaged by transferring them to electronic format with funding from the Bill & Melinda Gates Foundation and deposited in the *Healthy Birth, Growth and Development knowledge integration* (HBGDki) project.[8] The goals of this project are to prevent stunting (very low height-for-age), wasting (very low weight-for-age), and enhance neuro-cognitive development (Varela-Silva et al. 2016). Through the

[8] https://kiglobalhealth.org/

HBGDki web portal, researchers around the world may access the Guatemala Longitudinal Study data and many other studies, as well as apply sophisticated searches and modeling techniques.

Guatemala was also the site of another longitudinal study of human development in the developing world. This was the research program of the Institute of Nutrition of Central America and Panama (INCAP), and is sometimes called the "Four Village Study" because participants were recruited from two small (about 500 persons) and two larger villages (about 900 persons) in rural Guatemala (Martorell et al. 1979; Stein et al. 2008). Except for population size, the large and small villages were homogeneous in terms of ethnicity (all people were *ladinos*) and other social and economic factors. This was to be a study of growth and development from birth to age seven years, but there have been follow-ups of the original participants and their offspring (Ramirez-Zea et al. 2010). The original study ran from 1969–1977. Participants were recruited when their mothers were pregnant, at their birth, or soon thereafter. This was also an experimental study. The participants in one large and one small village received a dietary supplement called *atole*, the local term for a gruel usually made with corn meal. The participants in the other villages received a low-calorie *fresco*, the name for a cool refreshing drink.

When the study started in 1969, the *atole* supplement contained protein, carbohydrates, vitamins, and minerals. The *fresco* contained only carbohydrates, water, and artificial flavor. From 1971 to 1977, when the study ended, it was decided to increase the vitamin and mineral content of the *atole* and add equal amounts of these vitamins and minerals to the *fresco*, so that the analysis could be confined to the energy containing nutrients, the protein, and carbohydrates.

The major research question of the INCAP study was to what extent a nutritional supplement, especially of protein, could enhance the physical and mental development of children? The infants and children living in the four villages suffered from both **kwashiorkor**, an acute type of undernutrition that can kill, and chronic mild-to-moderate undernutrition, not severe enough to kill a person directly. In the 1960s it was hypothesized that chronic mild-to-moderate malnutrition could retard growth and development and reduce the body's ability to fight off diseases, which could kill. There were also hypotheses that poor, rural people of less-developed countries like Guatemala had "adapted" in some way to a limited food supply and would not benefit from additional food. However, the INCAP study proved that lack of adequate nutrition is a major factor retarding the physical growth and mental development of Guatemalan children. The infants and children supplemented with *atole* grew significantly taller and heavier than the children receiving the *fresco*. The *atole*-supplemented children also performed better on cognitive tests and were more likely to enter school. Further detail of the findings of the INCAP study, and other intervention studies, will be presented in later chapters. Further analysis of the INCAP results showed that it was the additional energy (kilocalories) in the *atole* supplement, not deficiencies of protein per se (e.g., amino acids) that made the difference. The additional energy from the *atole* came from both the protein and the carbohydrates. The INCAP study showed that one solution to the problem of undernutrition is to give

people more food. This may seem obvious, but as in the 1960s there are people today still clinging to notions of a healthy adaptation to under-feeding. In addition, it is known that poverty is associated with other causes of physical and mental growth retardation, including social inequality, the stress of violence and warfare, and political repression, which are not overcome by more food. This topic is presented in some detail in Chapter 7.

With the INCAP study results, researchers wanted to see if nutrition interventions would help children living in poverty in Africa, and Asia, as well as the developed nations of Europe, North America, Australia, and Japan. To test this, several studies were started, especially in Asian countries, that followed in the steps of the INCAP project. In fact, one birth cohort study began in 1967 is known as the Bacon Chow Study of Maternal Nutritional Supplementation. Bacon Chow (1909–1973) was a professor of biochemistry at the Johns Hopkins School of Hygiene and Public Health. In 1935 Chow helped to isolate for the first time a pure form of an antibody, Type I pneumococcus antibody (Chow & Goebel 1935) and later pure hormones from **pituitary** glands, including growth hormone. In later experiments with rats, Chow showed that a 50% dietary protein restriction during pregnancy and **lactation** resulted in offspring that were 20% to 30% smaller as adults, despite feeding of a nutritionally complete diet to the pups after their birth. These experiments, and his earlier work on protein antibodies and hormones, convinced Chow that more protein in the diet would overcome the effects of malnutrition in people. He undertook a study in Taiwan from 1967 to 1973 to determine the effect of increased maternal protein and calorie intake on the pre- and post-natal growth of offspring (Adair & Pollitt 1985). This was a double-blind controlled intervention, which was quite sophisticated for the time, of two groups of nutritionally at-risk rural village women. One group (n = 114) was given a nutrient-rich dietary supplement and the other a nutrient-free placebo (n = 111). The intervention began after each woman had given birth and continued through the lactation period of the subsequent infant. The variables assessed included infant birth weight and length, postnatal physical growth, motor, mental and dental development, morbidity, and maternal weight and skinfold changes during pregnancy and lactation.

The nutritional supplementation experiment did not provide much support for Chow's main hypotheses as there were no clear differences in anthropometric values between the two groups. Adair and Pollitt evaluated the findings, " . . . the growth of the children and the weight changes in the mother cannot account for all the energy that is made available. Where does this energy go?" (p. 976). They speculate that some of the energy in the food supplement went to more physical activity. Later studies found this to be true and the INCAP follow-up found that supplemented infants and children became more physically and economically productive adults.

An important lesson from Chow's application of the rat findings to humans is that rats do not follow a human pattern of growth. Neither do rats have the anatomy, physiology, hormonal, nor neurological make-up of people. Malnutrition in young rats followed by adequate feeding greatly impairs their growth at the time of starvation and reduces their adult size. In contrast, severe malnutrition in human infants,

for example during war and post-war periods, followed by adequate feeding often has little effect on adult size. A review of the human studies was published by Keys and colleagues in their two-volume work *The Biology of Human Starvation* (Keys et al. 1950). Rats and other nonhuman animals are used in many laboratory experiments to test hypotheses and drugs with possible application to humans and human health. More often than not the results of the "rat" studies have no value for humans or in many cases any positive effect on the nonhuman animals plays out as a harmful effect on humans (Akhtar 2015).

Other birth cohort, longitudinal studies (with the years of recruitment) were undertaken in Pelotas, Brazil (1982), New Delhi, India (1969–1972), Cebu, Philippines (1983–1984), and Soweto-Johannesburg, South Africa (1990). These, along with the INCAP study, are now part of COHORTS, the Consortium Of Health-Orientated Research in Transitioning Societies (Richter et al. 2012).

Other Basic Research Related to Growth

During the twentieth century, scientific research into genetics of growth became possible. The rediscovery of Mendelian principles in the year 1900 and the characterization of the DNA molecule (deoxyribonucleic acid) in 1952, are two of the major historical events influencing growth research. An early emphasis on "racial genetics" and eugenics, à la Galton, gave way to modern population genetics by the early 1950s. Methods to study the influence of genes at the individual and family level were also developed, including studies of monozygotic and **dizygotic twins** and studies of family pedigrees (see Chapter 7 for details). By the 1960s research on the effect of chromosomal variations and abnormalities had appeared. The first evidence that specific genes exist for growth in size, body proportions, body composition, and rate of maturation was derived from studies of sex chromosomes. Garn & Rohmann (1962) proposed that genes on the X chromosome control some aspects of the development of skeletal and dental tissue. J. German and colleagues (German et al. 1973) proposed that genes on the Y chromosome stimulate the growth of skeletal tissue to produce the greater average stature, arm length, and **biacromial breadth** (shoulder width) of men vs. women. The specific sequences of DNA responsible for these effects are being discovered and it is known that the short stature homeobox-containing (SHOX) region of the X chromosome is involved (Crespi 2008). The development of rapid genomic sequencing, a technology propelled by the Human Genome Project (Green et al. 2015; Moraes & Góes 2016), led to a proliferation of genome-wide association studies (GWAS) in the search for "gene-to-phenotype" relationships. Such relationships have proved elusive, for example, there are between more than 700 genomic regions, called short nucleotide polymorphisms (SNPs), associated statistically with human height, but these account for less than 20% of the variability in stature in any human population (Trerotola et al. 2015). Similar findings are reported for human body weight and other anthropometric dimensions. Further discussion of GWAS and human growth is given in Chapter 6.

An important corollary of these genetic studies was the discovery of new ways in which the environment can produce effects on growth that seem to be hereditary, but are, in fact, not due to the direct action of the genome (e.g., DNA, SNPs). In 1964, both in Poland (Malinowski & Wolanski 1985) and the United States (Bloom 1964) researchers found that the heritability of some phenotypic characteristics (such as stature) is higher in groups of parents and children living under low SES conditions, but that the heritability is lower when children live under more favorable SES conditions than did their parents. Benjamin S. Bloom (1913–1999), an educational psychologist, formalized this observation into what he called the "powerful environment hypothesis." By this Bloom meant that when succeeding generations of people grow up under the same, or similar, environment of extreme deprivation, each generation will develop similar and unfavorable physical and cognitive characteristics, even if they could develop otherwise in more fortunate environments. Bloom proposed that powerfully good environments would also constrain phenotypic variation toward a favorable phenotypic similarity between parents and offspring. Older views considered a high correlation between parent and offspring in any measurable trait to be evidence of a genetic effect. In contrast, Bloom's hypothesis predicted that when living under powerful environments the traits of both generations may be altered in similar ways without any genetic contribution.

Polish researchers were able to show that when the negative influence of the environment is ameliorated the correlation between generations declines, sometimes effectively to the point where there is no correlation. This research was reviewed by Napoleon Wolanski (1929–) (Wolanski 1967). Subsequent studies of "powerful environments" and growth variability are, themselves, quite variable in their results, but generally supportive of Wolanski's findings (Nikitovic & Bogin 2013). In many ways the powerful environment hypothesis complemented and extended the work of Boas and other anti-eugenics researchers.

Technological Developments

The technical basis for all of the research on growth lies in the precision of the instruments used to measure lengths, weights, circumferences, and other dimensions and the accuracy and reliability of the methods of measurement. Growth research technology and methods, beginning with the invention of the anthropometer (a device to measure stature) by Johann S. Elsholtz (1623–1688) in 1654, are reviewed by Noel Cameron (1984). Cameron also reviewed the development of skinfold calipers, radiography, **photogammetry**, and data analysis methods.

Anthropometric devices are mentioned here briefly, and then discussed in greater detail where relevant. The anthropometer was developed to measure lengths of the body, especially stature. Eventually, it was modified to measure lengths of body segments (arms, legs, etc.) and then was used to measure body breadths, such as the biacromial ("shoulder") and bicristal ("hip") breadths. Skinfold caliper measurements, first used by Kotelmann (1879) in Germany, have become a widely used method to evaluate **subcutaneous fat**, and its relation to growth, body composition, health, and

behavior. Modern anthropometers and calipers are designed to produce both accurate and reliable measurements, which are needed especially in longitudinal research where the same individual is measured repeatedly.

The discovery of X-rays by Wilhelm Conrad Röntgen (1845–1923) in 1895 was soon followed by applications to document skeletal and dental development. Röntgen made the first public presentation of X-rays, which took place in Würzburg, Germany on January 23, 1896. The X-ray taken that day was of the 78-year old Swiss anatomist Albert von Kölliger, who volunteered for the exposure which may be seen online and in an article by Thodberg and colleagues (2016). A painting of this event was made by Alan Thom and is also available online and in an article by Buzzi (2015).

Several works depicting normal skeletal development appeared starting in 1904 and culminated in Todd's 1937 *Atlas of Skeletal Maturation* (discussed above). Todd's atlas was revised by William Walter Greulich (1901–1987) and Sarah Idel Pyle (1895–1987) and published in 1950 as the *Radiographic Atlas of Skeletal Development of the Hand and Wrist*, which is still widely used for basic and clinical research (Greulich & Pyle 1959). Longitudinally collected radiographs from the Oxford Child Health Survey (see above, Acheson, 1954), the Fels Study, and the Harpenden Study were used to create newer methods for the assessment of skeletal maturation (Roche et al. 1975a; Tanner et al. 2001). These newer methods are more precise in the prediction of adult stature, which is a concern of parents, pediatricians, the military, and the ballet – ballerinas should not be too short or too tall. Automated skeletal age assessment from digitized hand-wrist X-rays is the latest technological advancement and commercial systems are available (Thodberg et al. 2009; Shampinato et al. 2017; Pan et al. 2020).

Photogrammetry is a method of growth evaluation based upon photographs of a person posed in a standardized position for either visual appraisal of size, shape, or maturity or for the measurement of height or body segments. One of the primary areas of use of photogrammetry is **somatotyping**, developed by William H. Sheldon (1898–1977) and colleagues, which relates human morphology to physical and psychological behavior (Sheldon et al. 1940). Progress in imaging, such as PET scans (positron emission tomography) and MRI scanning (magnetic resonance imaging) allowed for the noninvasive examination of internal soft tissue of the living and for the examination of internal structures of fossil human ancestors without damaging the specimen. There are also 3-dimensional scanning devices which automatically derive many standard anthropometric measurements (Lu & Wang 2008). These devices, along with advances in data collection strategies, statistical processing, computerization, and mathematical modeling have, and will continue to, revolutionize growth research.

Endocrines and Growth Control

In the early 1960s, Tanner stated, "There exists at present no entirely convincing and coherent theory of endocrinology of adolescence . . . " (Tanner 1962, p. 176). It may

be added that an understanding of the endocrine regulation of growth at all other stages of life, prenatal and postnatal, was equally poor. In 1974 Melvin Grumbach (1925–2016) and colleagues published *Control of the Onset of Puberty*, which contains several "coherent and convincing" hypotheses of the **endocrinology** of growth (Grumbach et al. 1974) and updated these 18 years later (Grumbach 2002). Today, models and theories of hormonal control exist for all other stages of growth. Box 1.2 describes the meaning and use of models in the study of growth.

The rapid pace of research in this field is due both to technological advances in the assay of hormonal factors and advances in understanding how hormones exert their influences on human growth and development. The history of human growth hormone is a major example. The following discussion is based on medical history articles (Ayyar 2011; Frasier 1997), the Smithsonian Institution blog,[9] and a *Life* magazine photo essay from 1948.[10]

Human growth hormone (hGH) is produced by the pituitary gland and secreted into the general blood circulation. It is a peptide hormone that works with another hormone called insulin-like growth factor-1 (IGF-1) to stimulate growth in cell size, cell reproduction (mitosis), and cell regeneration. hGH and IGF-1 are also stress hormones that raise the concentration of glucose and free fatty acids. The family of peptides that hGH and IGF-1 are parts of is evolutionarily old, with analogues found in living jawless fish (Agnatha), a group that diverged from other vertebrates more than 300 MYA. Mammalian growth hormone was isolated from rats and pigs in the 1940s by Choh Hao Li (1913–1987) and Herbert McLean Evans (1882–1971). These versions of GH were not effective against short stature in humans. Today it is known that GH of each mammalian species is different in terms of its genetic structure and that there are species-specific binding proteins that transport GHs in the circulation as well as transmembrane receptors at the cell level and post-receptor signaling molecules within the cell that participate a cascade of events for action of GH on growth biology (Savage et al. 2011).

hGH first was independently isolated from the human pituitary gland in 1956 in the laboratories of Li and Harold Papkoff. The first human trials to treat extreme short stature took place in 1958. In 1960, The United States National Pituitary Agency (NPA) was established to regulate the collection of hGH and supervise hGH treatment. Canada formed a similar regulatory agency. Between 1963 and 1985 about 7,700 children in the United States and another 27,000 children worldwide were given US or Canadian government hGH, extracted from human pituitary glands, to treat severe growth hormone deficiency. It took about 100 g of human pituitary tissue, extracted from cadavers, to produce 0.3 g of hGH. Due to this rarity, patients were treated for only part of a year and this limited their growth recuperation. Commercially available pituitary hGH, produced by pharmaceutical companies, were approved in 1976. By 1984 there was enough hGH for year-round

[9] http://americanhistory.si.edu/blog/2012/10/human-growth-hormone.html

[10] https://books.google.co.uk/books?id=dEoEAAAAMBAJ&tpg=PA89&source=gbs_toc_r&redir_esc=y#v=onepage&q&f=false

Box 1.2 **Models in biology**

Models are representations that display the pattern, mode of structure, or formation of an object, a process, or an organism. A model may also serve as a standard for comparison, between hypotheses that test human understanding of how some physical or biological phenomenon operates and the actual nature of that phenomenon. For example, models are often employed to study aspects of human growth, such as the adolescent growth spurt, and aspects of maturation, such as the onset of puberty. No model can represent a complete understanding of the regulation of human growth and maturation, for that would require inclusion of all the many genomic, epigenetic, endocrine, and environmental factors – as well as their interactions – that influence the developmental process. However, models are easier to construct, to understand, and to test than is total reality, since the models usually represent only a small portion of total amount of this detail.

Two types of models are used by growth researchers. The first type describes a result. For instance, a series of longitudinal measurements of the height of a child may be compared to a mathematical formula that fits a curve to the growth data. The mathematical parameters of the curve are chosen to model the actual increases in height, and rate or change in growth velocity, of the person over time. The fit of the curve may be quite precise, that is, the model describes growth very well. However, this type of model does not explain why increases in height occur or when changes in the rate of growth in height are likely to take place.

A second type of model attempts to describe a result as well as explain some of the determinants of the observations. An example is D'arcy Thompson's model for the growth of the nautilus, presented in Figure I.4. The equiangular spiral, the mathematical function that describes the growth of the Nautilus shell, implies that the proliferation of shell material occurs at a constant rate; proportional to the amount of tissue already produced. Thus, volumetric growth rate constantly accelerates, producing ever-larger chambers in the shell. Unlike a purely descriptive representation, D'arcy Thompson's model predicts, with great precision, the size and volume of the next chamber to develop.

A predictive model of growth has both mathematical and biological meaning, and is preferred to descriptive models. Applied to human growth, such a model might attempt to describe and predict growth in height from birth to maturity. For instance, if it is assumed that a genetic program aims the growth of a newborn toward an "ideal" adult size, as might be the case with strong genetic selection for size, then the rate of growth should be proportional to the difference between present size and ideal size. This would predict a concave-shape velocity curve, if growth rate slows as the adult target is approached, due perhaps to a decrease in a growth promoting substance. Or the velocity curve

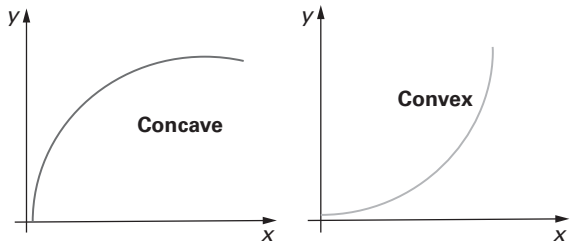


Figure B1.2.1 Concave and convex shaped curves. In the case of growth rate since birth, the x-axis would represent time (age) and the y-axis would represent rate of growth, e.g., mm/month, cm/year, kg/year.

might be convex-shaped if growth rate accelerates as the target is approached, due perhaps to a greater concentration of a growth promoter (Figure B1.2.1). The reality of the human growth curve (Figure 2.5), with a decline in velocity during infancy, the steady rate of growth in childhood, a **mid-growth spurt** at the transition to the juvenile period, and the adolescent growth spurt, may be explained by additional hypotheses about the changing nature of growth regulation. Tanner's conceptual model of growth presented in the previous chapter (Box I.2) proposed a change in the Time Tally with the transition from the juvenile to adolescent periods resulting in the adolescent growth spurt. In a sense, Tanner's model imposed successive concave curves, with the second curve stacked above the end of the previous curve. An alternative would be to insert a convex curve, indicating acceleration toward a new target size, between the two concave curves. Other models are possible, and a few will be presented in Chapter 3. The model building process encourages revision of previous concepts and the incorporation of innovations in biological knowledge, mathematical – statistical methods, technology, such as ways to measure the human body, including its biochemistry and biophysics, and in computation technology (e.g., machine learning and artificial intelligence). Each of these, singly and in combination, leads to the testing of hypotheses against observations of growth and maturation and the eventual formulation of a theory of human development.

treatment, if patients could afford it, and the final heights increased. In the 1984–1985 period hGH was given to 2,450 patients in the United States, and slightly more than 300 patients in Canada. In 1985 several adults who had been treated with hGH as children developed Creutzfeldt-Jakob disease. This is a prion caused illness with symptoms similar to mad cow disease. Death follows within one year of diagnosis. At least 26 people died from contaminated hGH. Treatment with hGH from pituitaries was banned. In retrospect, the conservative clinical use of pituitary hGH fostered by the US and Canadian governments, and its limited supply, was fortunate. As Frasier writes, "If anything can be learned from the use of pituitary GH

in children, it is a healthy respect for the law of unintended consequences" (Frasier 1997, p. S1). Too many other drugs have had, and will have, unfortunate unintended consequences on human growth and development.

Pharmaceutical companies had been developing recombinant hGH (rhGH), derived via the splicing of the human DNA sequence for GH into a bacteria genome. Compared to the pituitary extraction method of the past, vats containing billions of these genetically-engineered bacteria were able to produce large quantities of rhGH that was free of Creutzfeldt-Jakob and other diseases. A new era of GH treatment began, with enormous benefits to patients and enormous profits to the drug companies holding the copyrights to rhGH. The use of rhGH for growth hormone deficiency children was immediate (1985). Treatment for other syndromes of short stature followed: Turner syndrome (1997), Prader–Willi syndrome (2000), Small for gestational age (2001), Idiopathic short stature (short stature without any known cause – 2003), SHOX gene deficiency (2006), and Noonan syndrome (2007). Governmental approval to treat adult growth hormone deficiency came in 1996. Use of rhGH was extended to treat burn victims, to promote cell regeneration and healing. It is also used in treatment of cystic fibrosis (to increase adult height), inflammatory bowel disease, juvenile rheumatoid arthritis, osteoporosis, and in patients receiving chronic glucocorticoid administration. Adult prescriptions for rhGH are given for cosmetic use to reduce skin wrinkles, and some users believe it slows aging. Finally, there is a black market for illegal use of rhGH in professional and amateur sports to increase muscle mass and performance.

There are at least six hypothalamic hormones, eight pituitary hormones, a dozen or more hormones secreted from other endocrine glands (e.g., thyroid, adrenal, ovary, and testis) and a host of **growth factors** produced throughout the body that regulate human growth. Each of these has a history of discovery and understanding similar to hGH (reviewed in Chapter 6). Initially, most of the information on hormone regulation was derived from studies of children with endocrine pathologies. Newer, non-invasive assay methods (e.g., detecting hormones in blood spots and saliva, rather than from intravenous blood draws or from tissue samples) now permit the study of large samples of healthy infants to elderly people. This has led to a more comprehensive understanding of the endocrinology of human growth, development, maturation, reproduction, and aging. These topics are reviewed in greater detail in later chapters.

Growth Theory

The public health work of the nineteenth century and the large-scale longitudinal studies of the twentieth century provided a wealth of growth data. Advances in fields such as molecular biology, **endocrinology**, nutrition, and the social sciences allowed scientists and physicians alike to turn the study of human growth into a research and medical specialty. However, these data and technical advances were primarily descriptive in nature. They told us how people grow and how their growth was affected by heredity and the environment, but they could not tell us why. To

understand the "why" of growth and development a theoretical approach was needed. For example, the cell theory of Matthias Jakob Schleiden (1804–1881) and Theodor Schwann (1810–1882), proposed in 1838, made it possible to understand the earlier work of von Baer, who had described the different germ layers of the developing embryo. These layers were distinct types of cells that gave rise to the different tissues and organs of the body. With the publication of Darwin's *Origin of Species* in 1859 biological research became a modern theoretical science. The scientific method of experimentation and hypothesis testing was increasingly applied to biological questions, including the control of growth and development.

One example is the work of Ernst Haeckel (1834–1919), who proposed the theory of recapitulation during embryological development – that is, the development of the individual organism follows the evolutionary history of life (see online, Haeckel, 1874 *Anthropogenie oder Entwicklungsgeschichte des Menschen*). In contrast, von Baer in his 1827 study *De Ovi Mammalium at Hominis*, emphasized that during development embryos from different classes of animals move away from common forms, thus there is no recapitulation. This dispute stimulated experimentation and refinements to both hypotheses. Eventually, recapitulation was discredited, but Haeckel's work gave rise to interest in growth theory.

Many other scientists contributed to growth theory during the late nineteenth and early twentieth centuries, but the work of one person stands out more than any other. D'Arcy Wentworth Thompson's (1860–1948) book, *On Growth and Form* (Thompson 1917, 1942), is a tour de force combining the classical approaches of natural philosophy and geometry with modern biology and mathematics to understand the growth, form, and evolution of plants and animals. Thompson visualized growth as a movement through time. Scientists from Buffon to Boas had studied the velocity of growth; Thompson made it clear that growth velocities in stature or weight were only special cases of a more general biological process. The development of flower parts in plants or the evolution of antler size in mammals were also examples of growth as a movement through time. Thompson developed the concept and methodology of using transformational grids to quantify the process of growth during the lifetime of an individual or during the evolutionary history of a species (see Figure I.3). Until the advent of high-speed computers, which are needed to carry out the mathematical procedure of the method, the transformational method was difficult and slow to apply and, hence, little used by other biologists (Bookstein 1978; Windhager et al. 2017).

Even so, *On Growth and Form* provided an intellectual validity to growth and development research and stimulated succeeding generations of growth researchers to think about the mathematics and biological control of growth in new ways (Bogin 1980; Huxley 1972; Tanner 1963; Thom 1983). In later chapters there is further discussion of the contributions of D'arcy Thompson to evolutionary biology and human growth.

Conclusion

The foregoing highlights some advances in the study of human growth. Additional historical background is given in later chapters when new topics are introduced. The brief review in this chapter finds that by the 1940s some of the basic principles and patterns of physical growth and development were known. Since then, researchers have been making progress, often slowly, in unraveling the underlying biology of physical growth. One fact is clear – all normal, healthy, and well-nourished children follow the same basic pattern of growth from birth to maturity. Research also shows that a common pattern of human development and growth occurs during the prenatal period as well. Furthermore, important features of the pattern of human growth are unusual, perhaps even unique, to the human species. The next chapter describes these major features of human growth and development from conception to death.

2 Basic Principles of Human Growth

Stages in the Life Cycle

Many of the basic principles of human growth and development are best presented in terms of the events that take place during the life cycle. One of the many possible ordering of events is given in Table 2.1, in which growth periods are divided into developmentally functional stages. For convenience, the life cycle may be said to begin with fertilization and then proceed through prenatal growth and development, birth, postnatal growth and development, maturity, **senescence**, and death. In truth, however, the course of life is cyclical – birth, the onset of sexual maturation in the adolescent boy or girl, and even death is each a fundamental attribute of the cycle of life. From the embryo to the adult, old cells die and degrade so that their molecular constituents may be recycled into new cells formed by mitosis.

At the population level, people grow, mature, age, and die even as new individuals are conceived and born. Declaring one moment, such as fertilization, to be a beginning to life is arbitrary in a continuous cycle that passes through many stages, both in the individual person and in generation after generation.

In the following sections the stages of prenatal and postnatal life are described. The timing of growth events is presented, usually in the form of mean, median, or modal ages. The reader should bear in mind that these ages indicate the central tendency and not the normal range of variation that occurs naturally in the timing of many growth events. Research may find, for example, that the median age at menarche (first menstruation) is 12.6 years in a sample of girls. The actual age at menarche for individual girls in the sample may range from 8.0 to 15.0 years, and both the earliest and latest ages represent perfectly normal individuals. When the range of variation in the timing of growth events is important in terms of the basic principles of human growth, then it will be given in this chapter. Otherwise, individual and population variation in growth are discussed in more detail in Chapter 5.

Prenatal Stages

Human prenatal growth, development, and maturation last, on average, 38 weeks. This is the time in the uterus from fertilization to birth. In clinical practice and popular tradition, the average length of pregnancy is calculated as 40 weeks, because pregnancy is counted from the first day of the woman's last menstrual period, which typically occurs two weeks prior to ovulation and fertilization. Questions of "when

Table 2.1 The stages of human growth, development, and maturation. This is an update from the 1999 2nd edition of this book. This new table incorporates elements of the scheme of Grimm (1966), basic biology and cognitive research since 1999 and the pediatric clinical experience of Michael Hermanussen (Bogin, Scheffler, Hermanussen, 2018a). Ages given in the table are approximate, representing the average or modal ages for the onset of a stage or its range of duration. Names of stages in parentheses are those originally proposed by Hans Grimm. The essential biological, socio-cultural, and cognitive signs of each stage or period are given in the table.

Duration/age	Biological signs	Socio-cultural and cognitive signs
Neonatal stage		
Birth to 28 days	Extra-uterine adaptation of cardio-vascular, pulmonary, digestive, excretory systems from maternal dependence. Motor skills characterized by automatic inborn behaviors (reflexes) and gross motor activity. ***End***: maturity of mother's breast milk at no sooner than 28 days.	Preference for visually following human faces more than other objects. Visual acuity is best at a distance of about 19 cm, about the distance between faces when nursing. All senses operational, preference for sweet taste, able to distinguish the odor of mother's breast milk. Reflexes orient neonate's attention toward sound and light.
Infancy		
Month 2 to end of lactation (usually by 30–36 months in **traditional societies**[**])	Rapid growth velocity with steep deceleration in velocity with time. Many developmental milestones in physiology. Feeding by total or partial lactation in traditional societies, or by human breast milklike formulas in industrial societies, complementary foods added by 6–12 months.	Rapid motor-sensory, behavioral, and cognitive development.
Early infancy		
Month 2–12 month	Eruption of some deciduous teeth. ***End***: bipedal walking typical by the end of the stage (at ~12 months).	Feeding by lactation with addition of complementary foods after 6 months of age. Learning first motor skills, training of sensory systems, social relationship.

Table 2.1 (cont.)

Duration/age	Biological signs	Socio-cultural and cognitive signs
Late infancy		
Month 12 to 30–36 month	***End:*** deciduous tooth eruption is complete (2nd deciduous molar erupts at 20–35 months), **weaning** (termination of breast-feeding) between 30–36 months.	Development of verbal skills associated with more intense social and cognitive development. Use of **shared intentionality** and **theory of mind.**
Childhood		
3 to 6.9 years	Moderate growth rate. Mature level of bipedal walking. Relatively fast rate of brain growth and synaptogenesis, near completion of brain volume growth by end of stage. ***End***: eruption of first permanent molar and incisor complete, mid-growth spurt in many children, **adrenarche**.	Dependency for feeding. Language improvements in phonology, vocabulary, and sentence length. Greater independence in feeding, self-care, and care of others. End of the *kindchenschema* in physical appearance and behaviors.
Juvenile		
Pre-pubertal		
7–9 years in both sexes	Slower growth rate. Adultlike energy efficiency in bipedal walking.	Capable of self-feeding. Cognitive transition leading to learning and practice of economic and social skills (apprenticeships in traditional societies, formal schooling in many societies).
Puberty		
Neuroendocrine change in reproductive system: 9–10 years Somatic signs: girls: 11.0 yrs boys: 11.6 yrs	Neuroendocrine: ***Event*** in the regulation of the hypothalamic-pituitary-gonadal axis from negative feedback to positive feedback of the sex steroid hormones. ***Short*** duration (1–2 months) that reactivates the hypothalamic GnRH pulse generator leading to a massive increase in sex hormone secretion. Somatically: First appearance of secondary sexual characters (darkening	In traditional societies, and many industrial societies, pubertals contribute increasing amounts of time and labor toward food production, food processing, infant and child care, and wage-earning activities; in post-industrial nations most juveniles attend formal school, intensify friendships and social activities and are protected from physical labor.

Table 2.1 (cont.)

Duration/age	Biological signs	Socio-cultural and cognitive signs
	and increased density pubic or axillary hair, development of the breast bud in girls, genital changes in boys). Beginning of the adolescent growth 10–11 girls and 12–14 boys.	Brain growth rate declines in volume, but cognitive organization continues. Additional syntactic advances in language use, an increase in speech-breathing capacity and further increases in speech fluency. Greater socially relevant use of language from gossip to storytelling and greater use of language and cognitive skills in social competition.
Adolescence		
Girls: 11–18 yrs Boys:12–22 yrs	Adolescent growth spurt in height and weight. Further development of **secondary sexual characteristics**.	Intensification of interest and practice in adult social, economic, and sexual activities. Further development and organization of brain associated with changes in language usage, risk-taking behavior, and other cognitive capacities.
Pre-fertile		
Girls: 11–13 yrs Boys:12–13 yrs	Increasing velocity of growth in height and weight until PHV. ***End***: menarche (~12.5–13 yrs) spermarche (~13–13.5 yrs)	Continuation of juvenile behaviors, but with greater skill.
Fertile		
Girls: 13-18 yrs Boys.14-22 yrs	Decreasing velocity in height, weight velocity is variable. Low fecundity in girls due to 1–3 years of irregular ovulations (phase of **adolescent sterility**), sex-specific fat/muscle changes. ***End***: permanent tooth eruption complete (molar 3 eruption at ~18 years, if present). ***End***: epiphyseal fusion of long bones, adult target height achieved.	Improvements in physical and cognitive levels of work capacity. Post-fertile adolescents may be self-sufficient in physical terms but become more socially-emotionally dependent on peers. Linguistic content, including vocabulary, becomes more nuanced, grammatical operations and idiomatic phrases (slang) become commonplace.

Table 2.1 (cont.)

Duration/age	Biological signs	Socio-cultural and cognitive signs
		More refined logical expression of thought as well as joking, deceiving, mollifying, negotiating, persuading, and the use of sarcasm.
Adulthood		
Prime (maximal performance age)		
Women: 18–20 yrs Men: 20–23 yrs to about age 30–35 years in both sexes	Commences with completion of skeletal growth. **Homeostasis** in biology. Optimal reproductive performance and resilience to insults from injury and illness.	Cognitive, physical, social, and economic skills achieve maximum performance. Linguistic abilities in all aspects of spoken language are fully mature, written language (when present) may improve throughout the adult stage. All physical, social, economic, linguistic, and cognitive abilities are applied to success in mating, reproduction, and care of offspring.
Gradual decline		
~35 to ~50 years, **menopause**	First signs of physical degeneration are clinically detectable. Decrease of reproductive performance, fertility cessation. ***End for women***: menopause by age 50. Decline of sperm quality for men.	Both sexes still capable of physical and cognitive work; most women and men can compensate for the degeneration by new biobehavioral strategies.
Transition (degeneration age)		
Age ~50 years to senescence	Decline in the function and repair ability of many body tissues or systems. Decrease of body muscle and bone. Increase of relative or absolute percentage of body fat.	Decline in cognitive functions. Women may adopt a strategy investment in younger generations to enhance reproductive success and human capital, "grandmother effect." Men may also do this or continue with their own reproduction, but risk of unhealthy offspring increases.

Table 2.1 (cont.)

Duration/age	Biological signs	Socio-cultural and cognitive signs
Senescence (old age)		
Variable time of onset and progression, depends on prior level of somatic and cognitive reserves.	Decline in the function and repair ability of many body tissues or systems. Decrease of body muscle and bone. Decrease of relative or absolute percentage of body fat.	More rapid decrease of physical and cognitive working ability and decline in the ability to adopt biobehavioral strategies for compensation.
Death (age dependent physiological death)		
Variable	Reduction of the performance of somatic tissues and organs below that required for life support.	

**Anthropologists define traditional societies as hunter-gatherer (forager), horticultural and pre-industrial agricultural, and pastoral societies.

life begins" are fraught with ambiguities of mathematical, medical, and conventional meanings. The course of pregnancy may be divided into three time-spans, each of almost three-months' duration, called **trimesters**. In fact, the 266 days from fertilization to birth equates to nine lunar months, each of, roughly, 29.5 days. A body of research is devoted to lunar association with pregnancy and birth. The most reliable studies find no such associations (Bueno et al. 2010; Staboulidou et al. 2008). There is good evidence of a daily pattern in the hour of birth, with highest frequency of labor onset at night and births in the very early morning or later morning (Bernis & Varea 2012; Varea & Fernández-Cerezo 2014). This is a primate pattern, with deep evolutionary roots that may relate to selection for lower risk of predation, better conditions for delivery, and better conditions for mother–infant bonding.

During the first trimester, one of the major events is the multiplication of a single cell, the fertilized ovum, into tens of thousands of new cells. At first, cell division may produce exact copies of the original parent cell, but within hours of the first division, distinct groups of cells begin to form. Variations in the rate of cell division may be seen in the separate groups. Eventually these groups of cells form different kinds of tissue (the "germ layers" of endoderm, mesoderm, and ectoderm) that will constitute the growing embryo. Thus growth (an increase in cell number or cell size) and development (cellular differentiation) begin (Goss 1964). After the initial embryonic tissues are formed, the first trimester is taken up with organogenesis, the formation of organs and physiological systems of the body. During the first few weeks after conception the embryo has an external appearance that is "mammalian," that is, many

mammal embryos share these same external features. By the eighth week the embryo has many phenotypic characteristics that may be recognized as human.

Though the human body is composed of dozens of different kinds of tissues and organs, their generation and growth during prenatal life, and postnatal life as well, takes place through a few ubiquitous processes. Goss (1964) described two types of cellular growth, **hyperplasia** and **hypertrophy**. Hyperplasia involves cell division by mitosis. For example, epidermal cells of the skin form by the mitotic division of **germinative cells** (undifferentiated cells) in the deep layers of the skin. Hypertrophic growth involves the enlargement of already existing cells, as in the case of adipose cells growing by incorporating more lipid (fat) within their cell membranes. Giulio Bizzozero (1846–1901), an Italian medical doctor and researcher described three strategies of growth employed by different tissues: renewal, expansion, and stasis (Bizzozero 1894). Goss (1986) updated this classification. **Renewing tissues** include blood cells, gametes (sperm and egg cells), and the epidermis. Mature cells of renewable tissue are incapable of mitosis and have relatively short lives; for example, red blood cells (erythrocytes) survive in circulation for about six months. The supply of red blood cells is constantly renewed by a two-step process: First, the mitotic division of pre-erythrocyte cells of the bone marrow, and second by the differentiation of some of these into mature red blood cells. Goss pointed out that the undifferentiated cells are sequestered in a growth zone that is "spatially distinct from the differentiated compartment" (Goss 1986, p. 5). This two-step process and the growth zone for undifferentiated cells are common physiological features of many types of renewing tissues.

Expanding tissues include the liver, kidney, and the endocrine glands, the cells of which retain their mitotic potential even in the differentiated state. In the liver, for example, there is no special germinative layer or compartment, and most liver cells are capable of hyperplasia to replace other cells lost by damage. Relatively large portions of a diseased liver may be excised surgically, as a proliferation of new cells will eventually occur to meet physiological demands. Perhaps it is no coincidence that in Greek mythology the gods punished Prometheus, who gave the secret of fire to the mortals, by binding him to a rock and sending an eagle to peck out his liver each day for all eternity.

The third category are mitotically **static tissues**, such as nerve cells and heart muscle, that are incapable of growth by hyperplasia once they have differentiated from precursor germinative cells. Central nervous system tissues, such as the brain, the eye, and spinal cord, of most vertebrate species were also considered to be static and nonregenerable. The static nature of these tissues was assumed to be due to either the lack of a reserve of the germ cells or the limited size of the reserve which was usually depleted early in life. Therefore, according to twentieth-century textbooks, but not twenty-first-century research (see below), the body's pool of static tissues cannot be renewed if damaged or destroyed. The good news in this conventional perspective is that unlike renewable tissues, which have short lives, static tissues may survive and function for the lifetime of the individual.

Complex anatomical parts of mammals, such as a limb, were considered static in the sense that these could no regenerate if lost. When Goss was writing in the 1980s it

was well known that the adults of many invertebrate and some vertebrate species, can regenerate body parts. Fish and amphibians can regrow lost tails, fins, and limbs. Some lizard species can regenerate tails, but not limbs. Birds and mammals were thought to be incapable of limb or tail regeneration. Why can some vertebrate species regenerate so-called static tissue, but other species cannot?

Discoveries relating to stem cells, newly discovered growth factors, noncoding RNAs, epigenetic regulation of gene expression, immune system regulation, and more are changing the definition of static tissues. Regeneration of central nervous system tissue and heart muscle is possible. Many pathways are involved, including upregulation of genes for Wnt proteins that regulate cell proliferation, noncoding RNAs (also regulators of cell metabolism), upregulation of hyaluronic acid which may result in immune suppression, and induction of greater tissue hydration. These conditions are similar to the extracellular environment present during embryogenesis and prenatal development (Alibardi 2017). Further review of regeneration is outside the scope of this book, but in the near future regrowing a human finger, hand, and arm may be possible (Nowoshilow et al. 2018); even a brain (English et al. 2013).

Static tissues, regardless of regenerative ability, can grow by hypertrophy, for example, individual nerve cells may grow to relatively great lengths during normal development, and, if not fatally damaged by accidents or surgery, regrow new interconnections. The physique of body-building enthusiasts tends to result from hypertrophy of existing muscle cells and not from the formation of new muscle cells. Even if tissues are static in the sense that they cannot undergo mitosis, they are dynamic due to being reconstituted by a turnover of material at the subcellular and molecular levels. Studies of dietary intake and excretion of nitrogen provide an estimate of protein metabolism in the body. This is because most of the body's store of nitrogen is in protein molecules. As muscle tissue forms the largest mass of protein in the body, measures of nitrogen balance may be used as indicators of muscle turnover and renewal. Data published by Cheek (1968) and Young and colleagues (1975) indicate that in young adult men, about 2–3% of the muscle mass is renewed each day. In infancy, when new muscle tissue is forming by hyperplasia, the rate of protein renewal is about 6–9% per day. The magnitude of this metabolic renewal may be appreciated by the fact that the major contributors to basal metabolic rate of the adult body (which may be measured by the heat that the body produces when at complete rest) brain, liver, and muscle – each responsible for ~20% – and then 11% heart, 9% gastrointestinal tract (Cameron et al. 2016). This leaves only ~20% of metabolic activity (i.e., energy expenditure) from physical activity and immune function. Note that the turnover of cellular material occurs about equally in "static" tissue (brain and muscle) and expanding tissues (liver). The renewing tissues also undergo a turnover of protein molecules during their relatively short lives, but the major metabolic dynamic of these tissues is their mitotic proliferation and eventual death.

Thus, the biological substrate of the individual is not permanent, and from embryonic life through adulthood the human body is in a constant state of decomposition

and reorganization. Tanner (Tanner 1990, p. 25–26) observed that, "This dynamic state enables us to adapt to a continuously changing environment, which presents now an excess of one type of food, now an excess of another; which demands different levels of activity at different times; and which is apt to damage the organism. But we pay in terms of the energy we must take in to keep the turnover running . . . Enough food must be taken in to provide this energy, or the organism begins to break up." To be sure, different tissues turnover at different rates, so that in muscle cells nitrogen is replaced in a few days to a few weeks, while the calcium in bone cells is replaced over a period of months. During the years and decades of life, sufficient turnover and renewal of the molecular constituents of the body's cells must take place to rejuvenate the entire human being.

The metabolic dynamic of the human organism is most active during the first trimester of prenatal life. The multiplication of millions of cells from the fertilized ovum, and the differentiation of these cells into hundreds of different body parts, makes this earliest period of life highly susceptible to growth pathology caused by either the inheritance of harmful genetic mutations or exposure to harmful environmental agents that disrupt the normal course of development (e.g., certain drugs, malnutrition, disease, smoking, alcohol use, etc. that the mother may experience). Because of this, a careful study of the community of Kauai, Hawaii estimated that about 10% of human fertilizations fail to implant in the wall of the uterus, and of those that do so about 50% are spontaneously aborted (Werner et al. 1971). Surveys in 40 developing countries report that spontaneous pregnancy loss in human populations ranges from 25% to 60% (Casterline 1989). Most of these spontaneous abortions occur so early in pregnancy that the mother, father, and others are usually unaware that they have happened.

By the start of the second trimester of pregnancy the differentiation of cells into tissues and organs is complete and the embryo is now a fetus. During the first trimester, the embryo grows slowly in length, often measured as crown–rump length (CRL). At 18 days post-conception the embryo has a CRL of about 1.0–1.5 mm and by 12 weeks post-conception the CRL is about 53 mm (Meire 1986).The rate of growth in length increases during the second trimester. By the fourth month CRL is about 205 mm, by the fifth month 254 mm, and by the sixth month between 356 and 381 mm, which is about 70% of average birth length (Timiras 1972).

Increases in weight during this same period are also rapid. At eight weeks the embryo weighs 2.0 to 2.7 g and at six months the fetus weighs 700 g (O'Rahilliy & Muller 1986). This is about 20% of birth weight (Timiras, 1972), so relative to size at birth the growth in length during the second trimester exceeds the growth in weight. During the third trimester of pregnancy growth in weight takes place at a relatively faster rate. During this last trimester, the development and maturation of several physiological systems, such as the circulatory, respiratory, and digestive systems, also occurs, preparing the fetus for the transition to extra-uterine life following birth.

Measurements of length, weight, and other dimensions of the body are called collectively anthropometry or kinanthropometry, the scientific measurement of the

human body. There are several articles, texts, websites, and other online sources on anthropometry, with definitions of the most common measurements and instructions on how to take these measurements (Cameron 2013; Lohman et al. 1991; Lu & Wang 2008)[1]. Taking body measurements may appear to be simple, but accurate and reliable measurement requires training and practice. Good anthropometry is like good sport or musical performance – it takes knowledge, coaching, time, and a lot of patience. There are many possible sources of error that can make a series of measurements inaccurate and useless. The International Society for the Advancement of Kinanthropometry (ISAK) offers training courses. ISAK has developed international standards for anthropometric assessment and an international anthropometry accreditation scheme. Interested readers may see their website for further information.[2]

Birth

Birth is a critical transition between life in utero and life independent of the support systems provided by the uterine environment. The **neonate** moves from a fluid to a gaseous environment, from a nearly constant external temperature to one with potentially great volatility. The newborn is also removed from the supply of oxygen and nutrients that has been provided by the mother's blood passed through the placenta, and which also handles the elimination of fetal waste products, and must now rely on his or her own systems for digestion, respiration, and elimination. The difficulty of the birth transition may be seen in relation to the percentage of deaths by age during the neonatal period (the first 28 days postpartum), as shown in Figure 2.1. Of course, most of these deaths were not due to the birth process itself; rather the leading factor associated with neonatal death is inadequate growth and development during the prenatal period. Excessive prenatal growth also carries higher risk for neonatal death, but the discussion here is confined to inadequate growth as this is correlated with the leading cause of death in all human populations.

The most common indicator of inadequate prenatal growth is low birth weight (defined as less than 2,500 grams at birth). During the 1980s, about four out of five neonatal deaths in the United States were of low birth weight babies (Emanuel et al. 1989, 1992). An index of relative mortality during the neonatal period by birth weight is given in Figure 2.2. Relative mortality is defined as the percentage of deaths in excess of the number that occur for infants within the normal birth weight range of 3–4.5 kg. These data are for infants at all gestational ages. Prematurity, defined as birth prior to 37 weeks gestation, may cause additional complications that increase the chances of neonatal death. Some infants are small for gestational age (SGA), which is often defined as a birth weight less than the tenth percentile for completed week of gestation. Newborns who are both SGA and premature are usually

[1] wwwn.cdc.gov/nchs/data/nhanes3/manuals/anthro.pdf [2] www.isak.global/

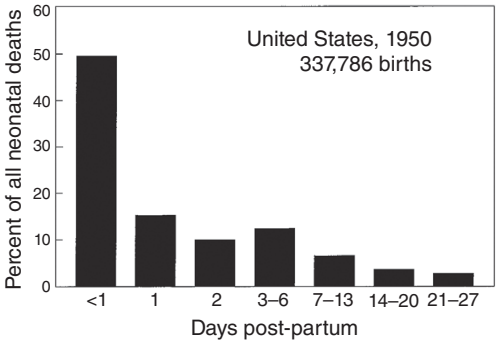


Figure 2.1 Percentage of deaths occurring during the neonatal period (birth to day 28). Data from the United States, all registered births for 1950 (Shapiro & Unger 1965). These data are presented in lieu of more recent data as the technology for extraordinary neonatal medical care in existence today reduces neonatal deaths.

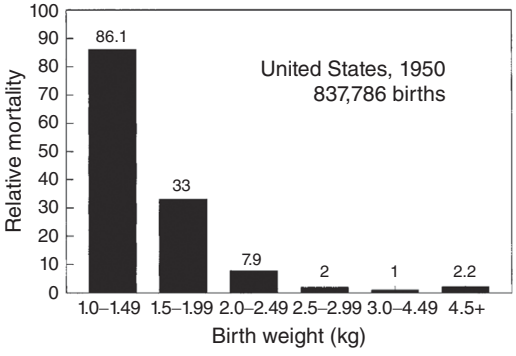


Figure 2.2 Index of relative mortality by birth weight (kg) during the neonatal period. Data from the United States, all registered births for 1950 (Shapiro & Unger 1965). Relative mortality is calculated as the risk of death for an infant born at a given weight. Infants of normal birth weight, 3–4.5 kg, have a relative mortality of 1.0. Infants born at 2.5–3 kg have twice the risk of death as normal birth weight infants, and so on.

at a greater risk of death than a premature child of the expected weight for gestational age (Gould 1986).

The causes of low birth weight, prematurity, and SGA are many and not all are well understood. It is known that one set of causes may be **congenital** (hereditary or inborn) problems with the fetus. Another set of causes may be placental insufficiency, or maternal conditions including undernutrition, disease, smoking, or alcohol consumption. Whatever the cause, low birth weight, with or without prematurity, is associated with socioeconomic status (SES) of the mother. A higher or lower SES does not, by itself, influence birth weight; rather the attitudes and behaviors of people and their opportunities for living conditions, education, occupation, and social status are associated with their SES. It is these attitudes, behaviors, conditions, and

opportunities that influence diet, health, smoking, alcohol consumption, educational attainment, and other determinants of birth weight. Any measure of SES will only partially account for all the social and cultural factors that affect growth. Nevertheless, SES serves as a proxy for these more specific determinants and at a gross level the effect of SES is obvious; for example, in the year 1980, the incidence of low birth weight in the wealthier, higher SES nations averaged 5.9% of all live births; in the poorer, lower SES nations the incidence averaged 23.6% (Villar & Bellizan, 1982). As of 2014, Finland, Iceland, and Republic of Korea reported an incidence of low birth weight of 4% (lowest values), compared with Pakistan (32%), Yemen (32%), and Mauritania (35%) (highest values).[3]

At a finer level, within the developed nations, the socioeconomic relationship with birth weight is still strong. Using nationally representative data from the United States (n = 8,400), the United Kingdom (n = 12,018), Canada (n = 5,350), and Australia (n = 3,452) from the early 2000s, Martinson & Reichman (2016) estimated prevalence rates of low birth weight by income and maternal education. They found SES gradients in low birth weight in all four countries, with the strongest associations in the United States. While all four nations are high income, the authors point to the greater social and economic inequalities of the United States compared with the three other nations. It was suggested that the more generous social safety nets and health care systems in the United Kingdom, Canada, and Australia were able to buffer the harmful effects of lower SES. A more thorough discussion of SES inequalities and human growth is given in Chapter 7.

In Europe and North America, non-European minority groups (nonwhites) show consistently lower average birth weights compared with European-origin people, and part of this difference is accounted for by the lower SES of the nonwhite groups. However, when European-American and African-American women are matched for SES, black women still give birth to a higher percentage of low birth weight infants. To some researchers, the white– black differences suggest that genetic factors are major determinants of birth weight, but others point to nongenetic factors. This debate is reminiscent of the Galtonian vs. Boasian debates on stature and head shape.

To put this debate in perspective it is first necessary to state more clearly what is known about the factors that produce variation in birth weight. Based on a variety of evidence and many research studies, E. Bette Robson (1928–2016, a pioneer of human genetics and gene mapping) estimated the variance in birth weight due to fetal genotype to be 10% and the variance due to parental genotype to be 24%. Most of the remaining 66% of the total variance was due to nongenetic maternal and environmental factors (Robson 1978). Robson's findings have stood the test of time, as the largest Genome-Wide Association Study (GWAS, sampling 153,781 individuals) identified 60 loci where fetal genotype was statistically associated with birth weight and these loci accounted for approximately 15% of the variance in birth weight (Horikoshi et al. 2016).

[3] https://data.unicef.org/topic/nutrition/low-birthweight/

There are many environmental factors that act directly on the developing embryo and fetus. Some obvious factors, briefly mentioned above, are smoking, alcohol consumption, and maternal nutrient deficiencies from poor diet or famine. These reduce birth weight. Maternal hyperglycemia, from unregulated diabetes or other causes, can lead to a higher than normal birth weight (Beaumont et al. 2017). There is also evidence that exposure to urban pollution in the form of noise, lead, and polychlorinated bisphenols can reduce gestation length leading to premature births and cause low birth weight in full-term infants (Schell 1991). Emanuel and colleagues find that these relationships may hold across so-called racial lines, influencing births to both white and black women, but the relative amounts of influence on birth weight are not equal. It is well established that in any given year more black infants are low birth weight than white infants, even when black and white mothers are matched for SES variables such as income, education, housing, and occupation (Gravlee 2009).

Even more curious are the findings for so-called biracial infants. One study examined all "biracial" single births reported in the United States in the year 1983 (Migone et al. 1991). Such births were to either white mothers and black fathers (WMBF), or black mothers and white fathers (BMWF). The percentage of low weight births for these "biracial" couples was compared to samples of single births to same "race" parents. The results, shown in Table 2.2, indicate a statistically significant trend. The "race" of either parent contributes to this trend; however the mother's "race" is a stronger predictor of birth weight than the "race" of the father. The data in Table 2.2 are unadjusted for known confounding variables, but the significant trend remained even when the authors of the study adjusted for mother's age, education, marital status, prenatal care, live birth order, previous fetal deaths, baby's sex, and gestational age. These findings were substantiated in a systematic review and meta-analyses of adverse birth outcomes for "biracial" couples (Srinivasjois et al. 2012). Based on a combined sample of 26,335,596 singleton births the authors calculated the adjusted odds ratio (95% confidence intervals) for low birth weight, preterm birth, and stillbirth. An odds ratio (OR) is a measure of association between an exposure and an outcome. The OR represents the probability that an outcome will occur given an exposure, compared to the probability of the outcome occurring in the absence of that exposure. Odds ratios greater than 1.0 indicate a positive association and ORs less than 1.0 a negative association between exposure and outcome. An adjusted OR accounts for other variables that may influence the outcome. The findings were, that compared with parents identified as white mother–white father (WMWF), the odds for low birth weight (LBW), pre-term birth, and still birth all increased for white mother–black father (WMBF), to black mother–white father (BMWF), to black mother–black father (BMBF) (Table 2.2).

What does the "race" effect seen in these studies mean? The social conditions of life for same "race" or different "race" parents are likely not to be equivalent. Moreover, the differences or similarities in living conditions between the various combinations of parents are likely to be quite complex and not easily summarized by a statistic such as current SES. Nevertheless, Mutambudzi and colleagues (2017)

Table 2.2 Total number of births, number of low weight births (LWB) (<2,500 g), and percentage of low birth weight births for each parental "racial" group reported by Migone et al. (1991) and adjusted odds ratios (Adj. OR) for LBW, pre-term birth, and still birth reported by Srinivasjois et al. (2012).

Parental groups (mother–father)	Total births	Low weight births	%	LBW: Adj. OR (95% confidence interval)	Pre-term birth: Adj. OR (95% confidence interval)	Still birth: Adj. OR (95% confidence interval)
White–white	24,059	1,027	4.3	1.0 (baseline)	1.0 (baseline)	1.0 (baseline)
White–black	18,004	1,108	6.2	1.21 (1.10–1.33)	1.17 (1.05–1.31)	1.43 (0.92–2.21)
Black–white	5,617	459	8.2	1.75 (1.64–1.87)	1.37 (1.18–1.59)	1.51 (1.09–2.08)
Black–black	15,220	1,629	10.7	2.08 (1.81–2.38)	1.78 (1.59–2.00)	1.85 (1.47–2.32)

reviewed the impact of material and psychosocial living conditions associated with "race" and ethnic disparities in birth outcomes in the United States in the years 2000–2014. Neighborhood-level deprivation, measured by quantitative indices derived from the 2000 Census, was the indicator of material conditions and racial discrimination or occupational stressors, measured by interview or questionnaire, as indicators of psychosocial stress. The outcomes of interest were LBW and pre-term delivery. The authors found that material deprivation and perceived racial discrimination in daily life and occupation significantly and negatively affected blacks more than whites, and were associated with increased adverse birth outcomes.

Many studies have assessed the association of current living conditions, including the SES, on birth outcomes of white and black women in the United States, the United Kingdom, and other countries, but even when women have equivalent living conditions, education, and occupation risk of low birth weight and other adverse birth outcomes remains higher for black women. Recognizing the complexities in this research, Irvin Emanuel and colleagues proposed an **intergenerational effect hypothesis** for the persistence of lower birth weights for infants born to black mothers. By this, the researchers mean that the SES matching is valid only for the current generation of adult women. The mothers and grandmothers of these black and white women were less likely to be equally matched for SES. Given the social history of the United States and Britain, previous generations of black women were likely to be of lower SES than their white counterparts. The intergenerational effect hypothesis predicts that the poor growth and development of women from older generations will have a lasting effect on the current generation.

There is considerable support for the intergenerational effect hypothesis. In his earliest study, Emanuel (1986) reported that a woman's health and living conditions during childhood were associated with birth weight of her offspring. In several studies, Emanuel and his colleagues working with both British (Emanuel et al. 1992) and United States data (Sanderson et al. 1995) found that a mother's own birth weight, her health history during infancy and childhood, and her adult stature

(which reflects the total history of her growth and development) are strong predictors of the birth weight of her offspring. These results were confirmed in a similar study of all births in Norway since 1967 (Skjaerven et al. 1997). The authors of the study linked the birth weight records of women with the birth weight of their infants to produce a sample of 101,264 mother–infant birth weight pairs. Mother's birth weight was strongly associated with the weight of infant.

Part of my own research, with a team of colleagues, focuses on the intergenerational effect hypothesis. In one study, we evaluated risk for overweight and stunting in 206 Maya children (4–6 years old) from Mérida, Yucatan, Mexico (Varela-Silva et al. 2009). Stunting is defined as a very short height-for-age, usually less than −2.0 standard deviations of an appropriate growth reference group. We compared the Maya children with growth references for the United States (Frisancho 2008) for height, weight, and body mass index (BMI, defined in Chapter 1 as [weight in kg/height in meters^2, abbreviated as kg/m^2]). Birth weight of the child was available from the national health cards that every Mexican child must have. We also measured the height of mothers of the children. Almost 70% of the mothers were shorter than 150 cm, which is an adult indicator of stunting. We reported that mothers' height and child's birth weight predicted excessive fatness as estimated by the BMI, although now I prefer to state that these predicted variation in body mass rather than fatness. As explained in Box 2.1, the BMI is not a reliable estimator of fatness. Children with a mother shorter than 150 cm were more likely (OR = 0.44) to have a low-to-normal BMI for their age and sex compared to children whose mothers were equal to or taller than 150 cm. Children with birth weights below 3,000 g were less likely to have a high BMI (OR = 0.28) than their peers within the range of normal birth weight (3,000–3,500 g). Children with a mother below 150 cm were 3.6 times more likely to be stunted than children of taller mothers. Stunting was defined as a height-for-age standard deviation score (a z-score) of less than −2.0. Fewer than 3% of children are expected to have a height that short, but we reported that children with birth weights below 3,000 g were over three times more likely to be stunted relative to children with birth weights within the normal range. Mother's age at pregnancy was not a predictor of her child's BMI or stunting. Our findings conform to the intergenerational effect hypothesis: Very short mothers, who were likely stunted as girls, were more likely to have lower birth weight infants who became stunted as children. The low-to-normal BMI of these children may mean they have low lean body mass, that is, less muscle or bone mass, and/or smaller organs, or less fat mass, or any combination of these. The BMI is too crude a measure to differentiate between the components of body composition (see Box 2.1). Later chapters return to these findings, and other similar research, that tries to better understand health risks due to intergenerational effects and birth weight effects.

Other Measures of Growth at Birth

Weight at birth is just one measurement that is commonly taken to indicate the amount of growth that took place during prenatal life. Recumbent length, the

Box 2.1 **The Body Mass Index: The good, the bad, and the horrid**

In 1832 Lambert Adolphe Quetelet, the Belgian mathematician described in Chapter 1, proposed that normal body weight measured in kilograms was proportional to the square of the height measured in meters (Quetelet 1832). This ratio (weight, kg/height, m^2) was given the name Quetelet Index (QI). Quetelet justified the QI based on his empirical analysis of data on weight and height showing that the QI produced the bell-shaped curve (normal distribution) that he sought in all biology and social science. In 1921, a Swiss physician named Fritz Rohrer proposed an improvement on the QI that he called the "Corpulence measure," also known as Rohrer's Index (RI). The improvement was to divide weight by the cube of height. This makes sense because weight is a volumetric measure and height is a linear measure. If we consider the human body to be a cylinder, then $height^3$ best estimates weight. Some debate about using $height^2$ vs. $height^3$, or some value between the square and the cube, ensued and by the mid-twentieth century the QI, the RI, and other related weight-for-height ratios were used by some human biology researchers to assess fatness and by the life insurance industry to apportion risk and insurance premiums (Billewicz et al. 1962; Dublin & Lotha 1937; Khosla & Lowe 1967).

Eknoyan (2008) reviews the use of Quetelet's index and reports that some support for the use of the QI in epidemiological research was based on an analysis of anthropometric data from the fourth examination of the Framingham study published by Charles du V. Florey (Florey 1970). Eknoyan's review does not explain that Florey was not impressed by the QI, the RI, or any index. Florey wrote, "The most likely best ratio in Western male populations is $weight/height^2$ and it seems probable that in Western female populations it is weight/height: the least likely for both sexes is ponderal index. Evidence is also given to show that all three indices are poor measures of adiposity." (p. 93). Florey based this assessment on the relationship of the indices to an independent measure of fatness using skinfolds. Florey noted that the QI, the RI, and the simple weight/height ratio were all dependent on height and that any measure of fatness should be independent of height.

Florey's analysis and conclusion that all the indices "are poor measures of adiposity" should have relegated all weight-for-height ratios to the anthropometric waste bin. Two years later, however, Ancel Keys (1904–2004) and colleagues, using more data from the Framingham Study came to the conclusion that the QI is a marginally better estimate of fatness than RI or WT/HT. The real impact of Keys and colleagues came from their renaming the QI the Body Mass Index (BMI) (Keys et al. 1972). The importance of the Framingham Study must be stressed. It was one of the first well-designed epidemiological investigations of the causes of heart disease, which was then, as now, a major public health concern. Physicians, public health researchers, the insurance

industry, and government policy makers were searching desperately for a cost-effective, that is simple, way to estimate the risk of heart disease and mortality. The prominence of Ancel Keys in nutritional science and public health policy must also be stressed. Together, the fear of heart disease, the economic costs of heart disease to families and the insurance industry, the simplicity of measuring only height and weight, and the reputation of Keys and the Framingham Study elevated the BMI to international prominence. A problem was the mathematics as many people, including medical doctors, have difficulty with the formula for the BMI:

$$\text{BMI} = \frac{(\text{weight in kilograms})}{\text{height in meters}^2}$$

Dear Readers – please stop reading to calculate your own BMI with this formula.

Do you know your weight in kilograms and height in meters? Did you remember to apply the correct order of arithmetic operations in the formula? What does the value of the BMI calculated mean?

To overcome these questions and promote the use of the BMI, the WHO in 1995 published tables of height and weight, in metric units, with the conversion values to the BMI. The BMI conversion tables in the report are an example of a nomogram, defined in the WHO 1995 report as, "A graphical device to allow rapid determination of an index (such as BMI), *avoiding the need for detailed calculations*" (WHO Expert Committee 1995, p 420). I added the italics to emphasize that the nomogram overcomes math phobia. In practice, a nomogram is a table or diagram designed to represent the relations between three or more variable quantities so that the values of the variables are associated via a simple geometrical relationship. BMI nomograms arrange the values of height and weight so that the point of intersection of any two measured values indicates the BMI value that would be obtained using the mathematical formula for BMI. The WHO nomogram was improved upon by making it easier to use and by making color versions – a different color for height, weight, and the BMI. Many such examples are found easily via an internet search.

The same WHO report published recommended BMI cut-off values for "thinness" as $\leq$18.5 kg/m^2 and "overweight" as $\geq$25 kg/m^2. BMIs between the "thin" and "overweight" values are "normal." "Obesity" was given a cut-off of $\geq$30 kg/m^2 but the WHO cautioned that true excessive fatness could be confirmed only if the "obesity" cut-off was combined with triceps and subscapular skinfold measurements that are both $\geq$90th percentile for age (WHO Expert Committee 1995). One may ask why even bother with BMI if skinfold measurements of that magnitude are known? The WHO report recommended the use of BMI as a proxy for nutritional status for adults and adolescents, risk for high blood pressure, women's pre-pregnancy and pregnancy risk for a low

birth weight infant, and other health risks and status. The authors of the report, a committee of prominent, international, public health professionals, were careful to mention some limitations of the BMI, for example, that it is an index of weight-for-height and not a measure of fatness. The committee also wrote that, "The exact significance of BMI is often difficult to determine" (p. 43). But later they seem determined to give BMI significance well beyond its weight-for-height meaning by writing, "As they relate to maternal anthropometry, socioeconomic indicators would include BMI among women (non-pregnant and non-lactating, or standardized for stage of pregnancy and lactation) as an overall indicator of the factors that affect women's energy balance (diet, workload, morbidity, reproductive demands). The importance of nutritional status as a factor in reproductive outcomes as well as maternal mortality makes a strong argument for the validity and usefulness of maternal BMI as an indicator of socioeconomic inequity. The same indicators may be used at the individual level to rank women according to degree of deprivation and to target resources to the most deprived, again using the underlying concept of socioeconomic inequity" (pp. 72–73). Note from this quotation that the BMI is recommended as a social and economic indicator, a nutritional indicator, a measure of workload and illness, a measure of inequality – and all for individuals!

The entire history of BMI is based on the 200-year-old **statistical hack** by Quetelet who came up with his simple index as a quick and easy way to estimate the degree of excessive body weight in the population. He was trying to help the Belgian government allocate health resources. Quetelet was a mathematician, not a biologist or physician, but he fully understood that his index should not be applied to individuals because the components of weight, that is, skeleton, organs, muscle, fat, could vary considerably between people. Keys and colleagues also stressed that the BMI not be used to assess individuals, but the simplicity and ease of measuring only height and weight to conjure an estimate of fatness overrode those warnings. Today the BMI is the most widely used measure of human fatness, even though it does not measure fatness. BMI is so popular because it is an understandable number which by design may only have values between 0 and 100, it is based on a simple mathematical formula, it has sharp cut-off values endorsed by the WHO, and it may be provided to you by your physician. The link to the WHO and the medical profession gives the BMI an air of scientific/medical authority.

The "Good" of BMI

In defense of the use of the BMI it may be stated that, generally, for groups of people who are physically inactive and who eat more food than they need to maintain body function a higher BMI score usually indicates more body fatness. This describes an ever-increasing number of population groups in all

parts of the world. The BMI is well associated – correlation coefficients above 0.80 – with the over-fatness of these groups of people. It is reported that higher BMI values and certain more direct measures of body fatness are about equally well associated with risks for higher systolic and diastolic blood pressure, higher very low-density lipoprotein and low-density lipoprotein cholesterol ("bad cholesterol"), lower high-density lipoprotein cholesterol ("good" cholesterol), higher triglycerides, and higher serum insulin levels (risk for diabetes). In one study, a sample of 2,840 girls and boys were examined at ages 10 and 18 years. Body fatness was assessed by dual-energy X-ray absorptiometry (DEXA), clinical examination, and blood sampling (Bell et al. 2018). The authors of this study reported that girls and boys with more DEXA measured FM index and higher BMI showed higher cardiometabolic risk profiles. The associations were stronger at age 18 years than at 10 years and also stronger for those girls and boys who increased both measured body fatness and BMI. The researchers recommend BMI as a useful tool to detect these types of health risks. These types of applications are the limited "good" of the BMI.

The "Bad" of BMI

A good deal of skepticism in the BMI was generated by the work of Stanley Garn (1922–2007). Garn was my colleague at the University of Michigan and by the late 1970s he was voicing concern about the misuse of the BMI. Garn was fond of saying that taller children and adults usually have more of everything – bone, muscle, and fat. Garn formalized this observation in the article "Three limitations of the body mass index" (Garn et al. 1986). The three limitations of BMI are: (1) BMI is not independent from stature. As a ratio, the calculation of BMI should yield the same result for all combinations of identical weight-for-height. Garn and coauthors showed this is not true as there is a change in the correlation between stature and the BMI from about +0.30 for children to an average of −0.12 for women 20–39 years old; (2) people of the same height have different BMI values according to frame size and relative leg length. People with narrower chests and/or longer legs relative to their total height have lower BMI values; and (3) the BMI cannot distinguish between the amount of lean tissue and fat tissue of a person's body. This is not only a problem for athletes vs. sedentary people of the same height and weight but also is part of the cause for ethnic, age, and sex effects on the BMI.

All three of the limitations highlighted by Garn and colleagues are, in fact, interrelated. Greater stature may be associated with a narrower skeletal frame and relatively longer legs. A narrower frame size may be associated with less total muscle mass. The limitations of BMI described by Garn and colleagues over 30 years ago have been rediscovered by younger researchers (Emerging Risk Factors Collaboration 2012; Wells 2014), but without any citation to Garn. To their credit, these younger researchers have found that the height denominator of the BMI equation is as important, or more important, than

weight nominator. Wells (2014) reviews several studies reporting that the association of juvenile and adolescent BMI with adult blood pressure is primarily driven by height. The Emerging Risk Factors Collaboration reports that the greater adult risk for mortality associated with higher BMI is primarily due to short stature, which increases the BMI of those with "normal" body weight.

As discussed in the main chapter, the effects of relative leg length have been especially well studied. Compared with the effect of fatness on BMI, as measured by skinfolds, the statistical effect size of relative leg length on BMI is small, explaining about 4% of the variation in the NHANES III sample (Bogin & Beydoun 2007). Even so, that effect is both statistically and biologically real and important. In the United States men and women with relatively shorter legs carry more subcutaneous fat, as measured by the sum of four skinfolds, than adults with relatively longer legs. At present it is not understood why this is so.

Other examples of how misleading the BMI ratio may be comes from our Maya research discussed in the Introduction. In addition to the height data depicted in Figure I.2, we also have weight data for the same girls and boys, and we calculated their BMIs. These are shown in Figure B2.1.1. Note that the Maya sample from rural Guatemala has mean BMIs that are greater or equal to the US mean at the ages 6–9 years, then their BMIs are lower than the US mean from 9–12 years but remain within the "normal" range. These rural Maya are, however, not "normal," because, on average, they are very short and very thin. Their BMI ratios mask their suffering in terms of health due to their extreme poverty (Bogin et al. 2002; Smith et al. 2003).

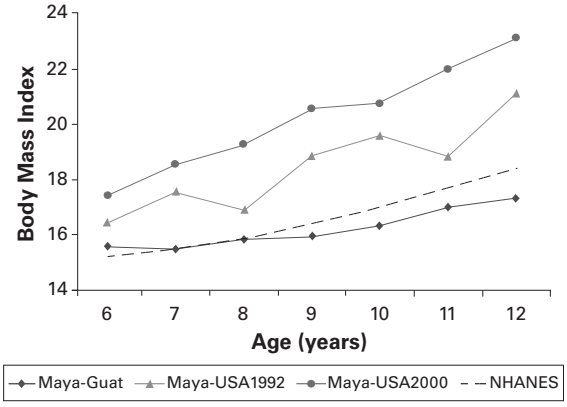


Figure B2.1.1 Mean body mass index (BMI) of Maya girls and boys in rural Guatemala (Maya-Guat), the United States in 1992 (Maya-USA1992), United States in 1999-2000 (Maya-USA2000), and the mean BMI of US reference data for European-Americans and African-Americans from the National Health and Nutrition Examination Survey III (NHANESIII) of 1988–1994. The photograph shows three Maya students in the foreground.

The USA-Maya depicted in Figure B2.1.1 have high BMI ratios at every age, an indication of overweight. Between 1992 and 2000 the BMI scores increased markedly. Higher BMI scores do indicate that an individual has relatively more weight-for-height than a person with a lower score. But a BMI score indicates only this and does not provide any information about body composition, that is, relative amounts of lean tissue (muscle, bone, organs) vs. fat tissue. For the USA-Maya 1992 sample we have the more direct measures of body composition triceps skinfold and mid-upper arm circumference. From these we estimated arm fat area, which is a widely used measure of subcutaneous fat (Frisancho 2008). Median arm fat area of the Maya girls and boys did not differ from US references (Addo et al. 2017) except at ages 7 and 9 years, when the Maya are significantly fatter. Over the entire age range our more direct fatness estimates do not correlate statistically or biologically with the BMI scores. We have shown elsewhere that the high BMI scores of the Maya-American girls and boys are likely due to their body shape, with relatively short legs in comparison to total stature, than due to fatness (Bogin & Beydoun 2007; Bogin & Varela-Silva 2008), a conclusion reached independently for other samples of people with relatively short legs by several researchers (Billewicz et al. 1962; Deurenberg & Deurenberg-Yap 2003; Norgan & Jones 1995).

A second example from our research team in Mexico was published in our article, "How useful is BMI in predicting adiposity indicators in a sample of Maya children and women with high levels of stunting?" (Wilson et al. 2011). The applicability of BMI to populations with high levels of stunting (height-for-age-and-sex below −2.0 standard deviations of an appropriate growth reference) has been questioned. Stunted people can have low levels of body fat, but normal amounts of muscle tissue for their height. Stunted people may also have disproportionately short legs and a relatively larger trunk length for their height. Any of these effects of stunting may increase BMI without an associated increase in body fat. Stunted people may also have low muscle mass and higher than expected body fat, which may also increase BMI. The point is, stunting may be unreliably associated with BMI.

We worked with participants from the Maya ethnic group of the Yucatan Peninsula. Our overall research goal was to understand why the Maya people, both children and adults, show high levels of stunting and at the same time high levels of overweight. This combination of short stature with overweight is known as the nutritional dual-burden. In principle, any group of people that has enough energy intake to grow in body weight should also have enough energy intake to also grow in stature. But, the Maya of Mexico and Central America remain stunted.

One of our projects recruited a sample of 57 urban Maya schoolchildren, aged 7–9 years (31 boys), and 53 of their mothers, mean age 34.44 (SD = 6.3) years. All the children and their mothers underwent anthropometric assessments of height, sitting height, weight, and the adiposity indicators of waist

circumference (WC), sum of the triceps and subscapular skinfolds (SSF), upper arm muscle area (UAMA), upper arm fat area (UAFA), and arm fat index (AFI= UFA / UAMA). We found that 18 (31.6%) of the children were stunted. In all children, BMI significantly predicted WC (e.g., abdominal fatness) and SSF, but not UAFA or AFI (e.g., peripheral adiposity). Stunting status and leg length did not modify the power of BMI to predict the WC or SSF. These findings suggest that BMI is an appropriate tool to estimate central adiposity in this sample of 7–9-year-old children, but that BMI fails to predict their fatness when only arm anthropometry is measured. This is an important finding because in practice the most common anthropometric measures of nutritional status and health are height and weight, and then arm circumference and triceps skinfold. A lack of correspondence between height and weight, used to calculate BMI, and arm anthropometry would lead to incorrect assessments and ineffective interventions to improve health.

In the Maya mothers, BMI significantly predicted abdominal adiposity (WC) but not arm adiposity (AFA, AFI). Stunting did not change the association between BMI and adiposity indicators in any of our statistical analyses. Relative leg length was neither significant nor altered the association between BMI and any adiposity indicator. We concluded again that BMI is appropriate for use in these adult urban Maya women only to predict abdominal adiposity. The Maya women participating in our study had BMIs in the range of 25–29.99, which suggests overweight but not obesity. However, their percentage of body fat was about 42%, which indicates obesity, when we estimated from the skinfolds, circumferences, and other measures. It appears to be inappropriate to use BMI alone to assess fatness in this sample of adult urban Maya women as it grossly underestimates their levels of obesity and may eliminate them from programs to lower body fatness and improve health.

The "Horrid" of BMI

A sizable number of people in all parts of the world are physically active, eat a healthy diet, have more muscle mass than the average and will have a high BMI score due to increased lean tissue. Lean tissue is heavier, per cubic centimeter of tissue, than adipose tissue. Adding a cm^3 of muscle will raise BMI faster than adding a cm^3 of fat. Use of BMI fails to characterize the health status of people with greater than average muscle mass. In addition, there are complex effects of age, sex, and ethnicity in the mathematical determination of the value of the BMI and these all relate to the poor precision of BMI to predict health problems of an individual (Razak et al. 2007).

Government agencies and policy advisory organizations in several countries ignore all these limitations and strongly recommend using the BMI to characterize "healthy" and "unhealthy" people. Public and private organizations reward or penalize people according to their BMI in terms of costs of health insurance and access to medical care. A proposal of this type by the United

States Equal Employment Opportunity Commission prompted Tomiyama and colleagues, " ... to examine cardiometabolic health misclassifications given standard BMI categories. Participants (N = 40,420) were individuals aged 18+ in the nationally representative 2005–2012 National Health and Nutrition Examination Survey. Using the blood pressure, triglyceride, cholesterol, glucose, insulin resistance, and C-reactive protein data, population frequencies/percentages of metabolically healthy vs. unhealthy individuals were stratified by BMI. Nearly half of overweight individuals [BMI between 25 and 29.9]; 29% of obese individuals [BMI $\geq$ 30], and even 16% of obesity type 2/3 individuals [BMI $\geq$ 35 or 40] were metabolically healthy" (Tomiyama et al. 2016, p. 883). The researchers found that 30.8% of individuals with normal BMI were unhealthy. These results were consistent for European-Americans, African-Americans, and Hispanic/Latino-Americans. The authors concluded that, "Using BMI categories as the main indicator of health, an estimated 74 936 678 US adults are misclassified as cardiometabolically unhealthy or cardiometabolically healthy. Policymakers should consider the unintended consequences of relying solely on BMI, and researchers should seek to improve diagnostic tools related to weight and cardiometabolic health" (p. 883). Just to reiterate, that is nearly 75 million people misdiagnosed. That should be considered medical malpractice! The problem is that a high BMI does not mean overfat or obese. The "improved diagnostic tools" to measure fatness recommended by Tomiyama and colleagues already exist and are called the skinfold (a polite way to say "fatfold") and waist circumference. Most medical and public health professionals can be trained to use and interpret these diagnostic tools in a few hours. Or, if that is too burdensome, professional anthropometrists can be employed to do so.

High risk for cardiometabolic disease is associated with "normal weight obesity" (NWO). This is a condition in which a person has an excessive amount of total body fat in combination with decreased lean body mass. The BMI of these people is within the "normal" range of values (Musálek et al. 2018; Oliveros et al. 2014). In adults, NWO increases the risk for cardiovascular morbidity and mortality, metabolic syndrome, a medical status that includes insulin resistance, hypertension, and dyslipidemia (Franco et al. 2016). The NWO phenotype was first described by Ruderman and colleagues as the "metabolically-obese, normal-weight individual" (Ruderman et al. 1981) to emphasize its association with metabolic syndrome. Public health policies based on BMI only to identify people at risk for metabolic syndrome will totally miss identifying people with NWO.

In different studies the prevalence of NWO varies from 0.7% to 36% of people screened. This wide range of prevalence is due to different methods and diagnostic criteria for assessing NWO (Franco et al. 2016). NWO seems to be more common in adult women than men. It is also found in children. One study from Prague, Czech Republic sampled 210 middle-school children (110

girls), 9–12 years old in the year 2015 (Musálek et al. 2018). The researchers divided the sample into three groups using percentile rankings according to age and sex-specific values of international BMI references and Czech national reference for skinfolds: (1) overweight obese (OWOB), n = 72, defined as BMI >85th percentile along with average values from three skinfolds >85th percentile; (2) normal weight obese (NWO), n = 69, defined as BMI of 25–60th percentile, along with average values from three skinfolds >85th percentile; (3) normal weight nonobese (NWNO), n = 69, defined as BMI in the range of the 25–84th percentile, along with average values from three skinfolds within the 25–84th percentile. It is immediately striking that 67% of the sample is obese (groups 1 + 2). The children and the school were described as typical for present-day Prague. The researchers also measured several body dimensions to assess skeletal robustness and muscularity. They found that NWO children had significantly less skeletal robustness on lower extremities and smaller muscle area on the upper arm and calf compared to NWNO children. These effects were more pronounced in boys compared with girls. The researchers suggested further study to determine if the results were due to insufficient physical activity by the NWO children and if NWO children also show a higher prevalence of health problems now and in the future.

Each of these "bad-to-horrid" misuses of BMI pales against the "super-horrid" abuse of the BMI when applied to infants and children, especially the so-called adiposity rebound of children. In groups of healthy, well-fed infants and children the BMI reaches a peak at ~6 months, then decreases over time until ~age 7 years, and then begins to increase again. The change from deceleration to acceleration in BMI has been called the "adiposity rebound" (Rolland-Cachera & Péneau 2013). The decrease in BMI between ages 6 months and 7 years is often interpreted as a diminishing of fat reserves due to increased physical activity with age or due to a trade-off between energy needed for rapid brain growth vs. energy stored as body fat. The rationale for linking BMI to body fat is based, in part, on the similarity in appearance between BMI reference curves and triceps and subscapular skinfold thickness reference curves at the same ages (Tanner & Whitehouse 1975). If the skinfold references are taken to indicate total body fatness, then it is a small step to assume that BMI in infancy and childhood is also representative of total body fatness. The strongest proponents of the BMI adiposity rebound proposal do make this assumption, as shown by statements that during infancy and childhood, "The trajectory of always high BMI could correspond to the so-called 'metabolically healthy obese subjects' while the trajectory of low BMI followed by increasing fatness is associated with insulin resistance and coronary heart diseases" (Rolland-Cachera & Péneau, 2013, p. 127). Note the seamless transitions in language from BMI to fatness, then obesity, and finally cardiometabolic disease in this statement. The language use is "smooth" but there are few data in support of these associations. Despite the lack of direct

causality between BMI, fatness, and illness there are many publications claiming that an adiposity rebound at an early age (<7 years) is associated with later life fatness, insulin resistance, and risk for metabolic disease (Aris et al. 2019 and references therein).

Total body fat content is not measurable directly in a living person. It is measurable from total dissection of the body, but for the living there is no single best method of body composition measurement of humans. Researchers have settled on the four-component method (4C method) as one "gold standard" for body composition estimation of the living. The 4C method divides the composition of the body into fat, water, mineral, and protein. The operation of this method is described by Wells and colleagues (Wells et al. 2012). Several other methods exist, and the present discussion focuses on anthropometry (e.g., skinfolds and circumferences), bio-impedance analysis (BIA), and DEXA. Explanation of the theory and application of BIA and DEXA for the estimation of body fatness are available on the Internet.

An adiposity rebound is not seen when fatness is estimated by the 4C method, BIA, or DEXA. Published reference data on body fatness measured by any method, from age 5 years to 18 years or older, for large samples from India, Europe, and North America show steady increases in total body fatness from the earliest age to adulthood (Khadilkar et al. 2013; McCarthy et al. 2006; Wells et al. 2012). This absence of an adiposity rebound is explained easily because the shape of the BMI and skinfold reference curves for infants and children has nothing to do with body fatness. Rather, that shape, including the rebound at about age 6 years, is associated with allometric changes in body shape and proportions – the type of shape changes analyzed by D'Arcy Thompson as described in the Introduction and the type of **allometry** described in the main text of the present chapter.

Fetal and early infancy are characterized by an "inflation" type of growth: Everything increases in size. It has been well understood since the time of ancient Greek geometry that an increase in the volume of a corpus results in an inverse decrease of the surface area of that same corpus. BMI is simple geometry that relates volume (kg) over surface (m^2). The quotient increases "during the inflation" phase of growth and the BMI curve peaks around age 6–12 months. But all body parts do not increase at the same rate. As was shown in Figures 2.3 and 2.8, the head and the trunk of the body are relatively large at birth and the legs relatively short. By six months of age, legs grow relatively faster than the upper body segments and the simple geometric nature of volume to surface area of early infancy is counteracted by the late infantile and childhood change in proportions that prevails during the following years. In the language of mathematical ratios, the sitting height/total body height ratio decreases from about 0.7 at birth to about 0.55 at age 6 (Bogin & Rios 2003; Scheffler et al. 2017). Stated as the reciprocal, the relative leg length

increases by about 50%, that is, from 30% of total body length at birth to 45% at age 6 years. This 50% increase in the relative leg length directly influences the denominator of the volume/surface quotient. The BMI decreases – not because of fat, but because of the squared leg length proportion. After age 6, sitting height ratios stabilize, and stay close to 0.55–0.53 for the rest of life. The former "inflation" type of growth starts to prevail again, and the BMI rises. All of these changes in BMI are totally unrelated to fatness.

The shape of the triceps and subscapular skinfold reference curves may be explained for a mathematically similar reason, which is that the lean body tissue of the upper arm or the upper trunk is expanding as the infant and child grow. This stretches and thins the subcutaneous fat layer making the skinfold measurement smaller. More subcutaneous fat may be deposited during these phases of growth, but the lean tissue volume of the arm and trunk is inflating faster than the subcutaneous fat surface area. At the end of childhood, the rate of volume growth slows – this is the juvenile deceleration in growth rate. The skinfold measurements "rebound," but not because of a fat spurt, rather because of the steady increase of fat deposition occurring since at least age 5 years that now happens at a relatively faster rate than lean tissue growth.

It is all simple geometry, but the attraction of the BMI is that it obviates the need to remember our school geometry and allows for a simple-minded association of BMI with body fatness, an association that is totally devoid of the well-known biology of body shape changes during infancy and childhood.

BMI is Bad Medicine

The Body Mass Index is, on balance, mathematical, biological, and medical snake oil, that is, a product, policy, or ideology of little real worth or value that is promoted as the solution to a problem. The limitations of ratios such as the BMI in human growth research have been well known since James Tanner's 1949 expose on the topic (Tanner 1949a). Tanner showed that the use of ratios in human biology and medicine is theoretically wrong and misleading as it produced spurious correlations and erroneous diagnosis of ill health (Tanner 1949a, 1949b). Over the years, other researchers have supported Tanner's findings. Despite this, ratios such as the BMI are still widely used because they are simple to calculate and seem easy to understand. But they are based on bad mathematics, fallacious science and result in hardships such as higher insurance costs, job loss, and lack of promotion for those classified as not "normal." In the United States social discrimination against people with a perceived high BMI is as rampant and damaging as racial and gender discrimination (Puhl et al. 2008). As shown in this brief expose, the misuse of BMI by the epidemiological and medical community causes harm by misclassifying health risks. In some cases, this misclassification amounts to professional malpractice.

Table 2.3 Mean size at birth and at age 18 years for children born in the United States. Data from various sources of nationally representative statistics (Bogin 1999b).

	Birth		18 years	
	Boys	Girls	Boys	Girls
Recumbent length/stature (cm)	49.9	49.3	176.6	163.1
Weight (kg)	3.4	3.3	71.4	58.3
Head circumference (cm)	34.8	34.1	55.9	54.9
Triceps skinfold (mm)	3.8	4.1	8.5	17.5
Subscapular skinfold (mm)	3.5	3.8	10.0	12.0
Arm muscle area (mm) at age one year	20.4	19.6	75.0	57.2

circumference of the head, arm, and chest, and skinfolds are others. Recumbent length is similar to stature, however the person measured is lying down and is stretched out fully by having the examiner apply gentle pressure to the abdomen and knees. The maximum distance between the vertex of the head and the soles of the feet constitutes the measurement. This can be measured at a very young age, or under other circumstances when stature (standing height) is impossible to determine. Circumferences measure the contribution made by a variety of tissues to the size of different body parts. For example, head circumference measures the maximum girth of the skull and hence, indirectly, the size of the brain. This is because of the intimate conformity between the brain and the tissues which surround and protect it, including the skull bones. Similarly, arm circumference includes the measurement of bone, muscle, subcutaneous fat, and skin. For infants of the same weight and length, variations in arm circumference are chiefly due to variations in the amounts of muscle and, especially, subcutaneous fat.

Some representative data for several measures of size at birth and at 18 years of age are given in Table 2.3. These average figures show that at birth, boys are a bit longer, heavier, and larger-headed than girls, but the girls have slightly more subcutaneous fat than the boys. There is such a wide range of variation in actual birth dimensions, however, that the small average **sexual dimorphism** in size is biologically insignificant. At 18 years of age, on average, men and women display well-marked sexual dimorphism in all these growth variables, except head circumference. Another difference between the infant and the adult is in body proportions. For children born in the United States, head circumference at birth averages about 70% of length at birth. By age seven years, head circumference averages 42% of length and at maturity the average value falls to about 30%. The reason for this change in percentage over time is that during the fetal, infant, and childhood stages of life the growth of the brain proceeds at a faster rate than the growth of the body (Scammon 1930). For the average healthy child, head circumference reaches 80% of mature size by about 7 years of age, though length of the body is only 68% complete at the same age. There are also proportional changes in the length of the limbs, which become longer relative to total body length during growth. The proportional changes

are illustrated in Figure 2.3. Note how size of the head dominates to body size during prenatal and early postnatal life. This is due to growth of the brain.

The body composition of 77 (41 male) healthy term infants (>37 weeks gestation) born to nonobese mothers in Brisbane, Australia in terms of fat mass (FM) and fat free mass (FFM) has been determined using the PEA POD body composition device (Carberry et al. 2010). The PEA POD is a minimally invasive air displacement plethysmography machine and software that uses whole body densitometry to determine fat and fat-free mass in infants weighing between 1 and 8 kg. Prior to this technology, methods to determine infant body composition required multiple anthropometric measurements or the dissection of infant cadavers. For the Australian sample, the mean newborn total body weight is 10.05% (standard deviation 4.05%) body fat and 84.6% FFM, that is, the weight of the body's water, bone, organs, and muscle content. Newborns are, in fact, mostly water – about 70–75% of total body mass (Fomon et al. 1982). By age 1 year, fatness increases to about 30% of total body mass (Butte et al. 2000a). FFM, especially muscle and bone also increase. The estimates published by Butte and colleagues were derived using another minimally invasive technology, Dual-energy X-ray absorptiometry (DXA).

In the second half of the twentieth century and in the wealthier nations of Europe and North America, adult men who were not overweight or obese (OW/OB) averaged about 15–17% FM and about 40% muscle mass; non-OW/OB women averaged 24–26% FM and 35–37% muscle mass (Holliday 1986). Those numbers come from before the current pandemic of overweight and obesity, which was noted in the wealthier nations by the late 1980s. Today adult men and women in northern Germany classified as non-OW/OB by BMI average about 20% and 35% FM, respectively, and individual variation ranges from 10–45% FM. OW/OB women attain more than 50% FM (Müller et al. 2016). The composition of the newborn, the adolescent, and the adult in terms of other soft tissues, and a variety of chemicals, is also available in the literature cited here and online and shows marked differences between birth and adulthood. The differences are important for many reasons, including energy reserves needed for growth during the pre-adult stages of development vs. energy reserves needed for successful pregnancy and growth of the fetus for adult women. Body composition differences according to age and sex are also important in medicine, for example, drug dosage prescription which needs to be tailored to the metabolic differences associated with muscle mass and fat tissue.

The study of the formation and maturation of hard tissues, such as the skeleton, during prenatal and postnatal life is another means toward describing different stages of development. Most bone forms from cartilage, which becomes calcified (calcium and phosphorous minerals are added) and, then, ossified (hardened) into mature skeletal tissue. Bone formation takes place throughout the growing years. A record of the process can be captured on radiographs, since at certain X-ray exposure levels cartilage is "invisible," but calcified and ossified bone is radiopaque (Figure 2.4).

Radiographs of skeletal development of normally growing children from the United States and England have been compiled into atlases, which may be used to

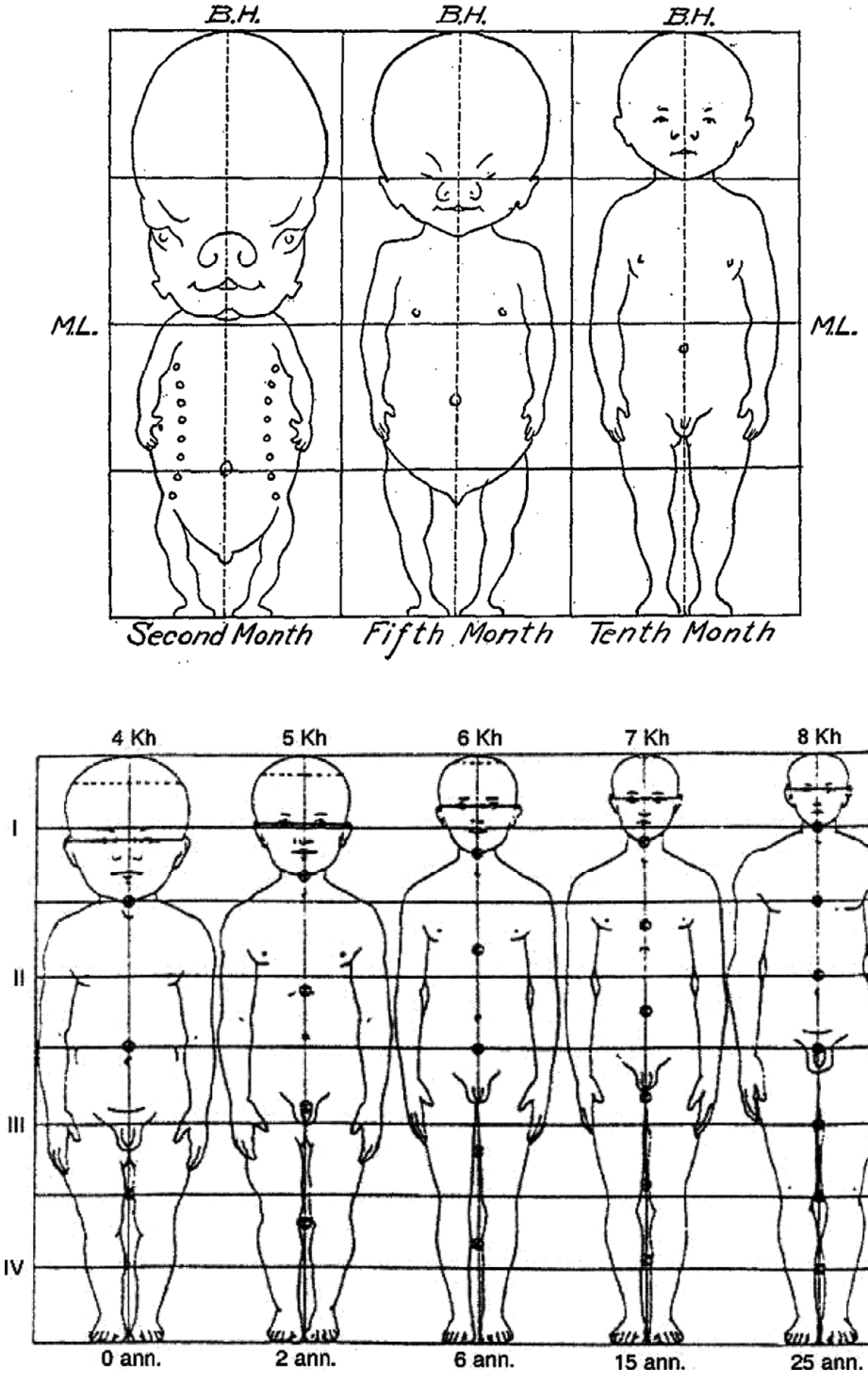


Figure 2.3 Changes in body proportions in fetal life (top illustration). *B.H.* = Body height/length; *M.L.* = Midline. Changes in body proportion during postnatal life (lower illustration). *Kh* = head length as a percentage of total body length, 4Kh = 25%, 5Kh = 20%, etc.; *ann.* = age in years, 0 *ann.* = birth, etc. From Stratz (1909), top illustration modified at www.neonatology.org/classics/hess1922/hess.3.html

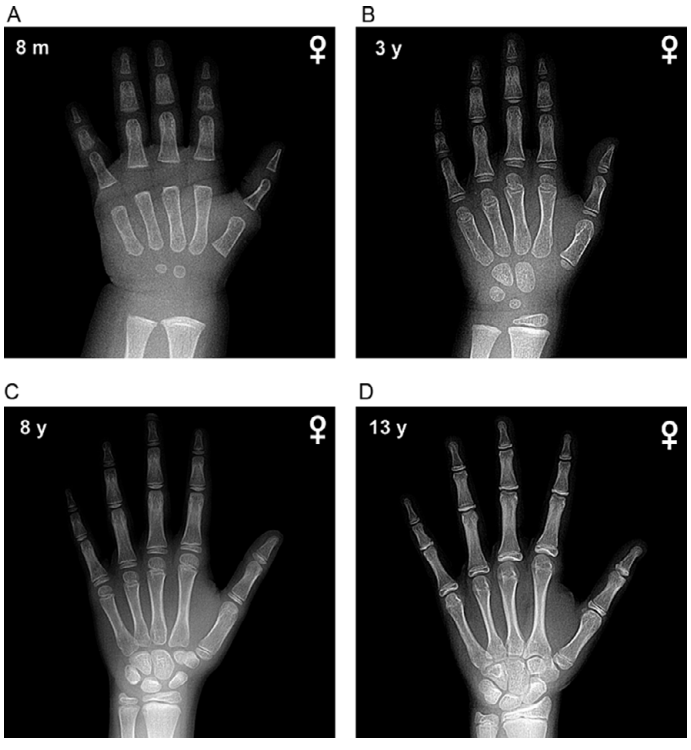


Figure 2.4 Radiographs of the hand and wrist at different skeletal ages, illustrating the sequence of bone maturation events. All radiographs from healthy girls. (A) At 8 months after birth, most wrist bones and the growing ends of the finger bones (called epiphyses) are formed of cartilage. At certain X-ray exposures, this cartilage is "invisible." The image shows few centers of ossification (i.e., places where bone is present) in the wrist and few visible epiphyses. (B) At 3 years old, some wrist ossification centers are present, epiphyses of radius are present, and most epiphyses of the hand are calcified (i.e., forming bone). (C) At 8 years old, all ossification centers are calcified. (D) At 13 years old, all bones have assumed final shape, but growth in size remains to be completed. Source: Original figure created from images published in Gilsanz & Ratib 2005, with permission of the authors

assess the stage of bone maturation of other children (Gilsanz & Ratib 2005; Greulich & Pyle 1959; Hoerr et al. 1962; Roche et al. 1975b; Tanner et al. 2001; Todd 1937). The atlases require a trained technician to manually compare an X-ray of an individual hand-wrist, knee, or foot-ankle to a reference photograph and assess the state of bone maturation and estimate a "bone age." The images in Figure 2.4 show reference photographs for girls from the Glisanz and Ratib atlas. There are also automated systems for estimating bone age, such as the BoneXpert system (Thodberg et al. 2009).[4]

[4] www.bonexpert.com/

The importance of these assessments of body composition and skeletal maturation at different ages is twofold. First, they allow clinicians and researchers to assess a growing person's state of biological maturation for different organs, tissues, or chemicals independent of chronological age. Biological maturation is used to help determine if a young person is developing too slowly or quickly, either of which may indicate the presence of some disorder. Second, the maturational contrasts between early and later life are also conceptually important. They show that the infant may take any one of several paths for growth, maturation, and functional development. Adult human morphology, physiology, and behavior as well, are plastic (Lasker 1969) and in no way rigidly predetermined. Plasticity refers to the ability of an organism to modify its biology or behavior to respond to changes in the environment, particularly when these changes are stressful. People, of course, cannot sprout wings or breath water; but the sizes, shapes, colors, emotions, and intellectual abilities of people can be significantly altered by environmental stress, training, and experience.

When the biology and behavior of people are considered together in biocultural perspective, it seems that human beings are, perhaps, the most plastic of all species. Other species, such as cockroaches, may resist cold, toxic chemicals, radiation, and **hypoxia** (low oxygen) better than humans, but they do so without much change to their basic phenotype. Human beings change during their lifetime. One example is a nearly 23 cm variation in mean height from shortest to tallest populations of people alive today (Bogin et al. 2017), and if pygmy populations of Central Africa were included the range of variation would be greater than 30 cm. In part, the human expression of phenotypic plasticity is due to a long lifespan,relative to most other animals. The adult human phenotype is achieved after many years during which a variety of factors may influence their final outcome. This makes people highly variable and adaptable. A long developmental process also entails many risks, which are discussed in later chapters.

Postnatal Life

In contrast to the widely used trimester system for prenatal growth and development, many ways have been proposed to divide life after birth into distinct periods. An historical discussion of some of these was given in Chapter 1 (such as the Roman "seven stages of life"). A five-stage model of human postnatal growth and development to maturity was proposed in the first edition of this book published in 1988. These five stages are infant, child, juvenile, adolescent, and adult. These stages are included in Table 2.1, which includes additional prenatal and post-adult stages, as well as subdivisions of the infancy, juvenile, and **adolescent stages**. In this book, the word "stage" denotes a biologically and behaviorally definable period of growth, development, and maturation. A chronological age range may be assigned to each stage for convenience of definition. Any stage model of human development is somewhat arbitrary (life is a continuous process), but the use of stages aids description and analysis of the life cycle.

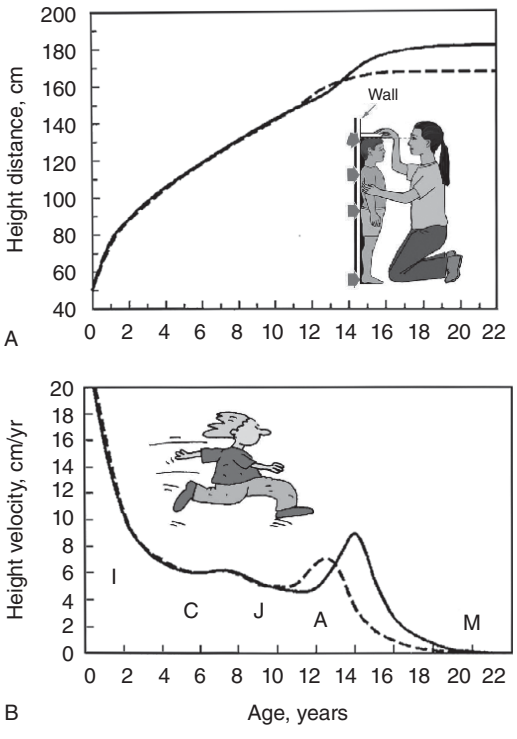


Figure 2.5 Average distance (A) and velocity (B) curves of growth in height for healthy girls (dashed lines) and boys (solid lines). Distance is the amount of height achieved at a given age. In part A, the image shows a child's height being measured. Velocity is the rate of growth at a given time, in this case shown as centimeters per year. In part B the running figure represents "velocity." The velocity curves show the postnatal stages of the pattern of human growth. Note the spurts in growth rate at mid-childhood and adolescence for both girls and boys. The postnatal stages: I, infancy; C, childhood; J, juvenile; A, adolescence; M, mature adult. (Original figure of the author)

The rationale for the stage model presented here begins with an analysis of the amount and rate of growth from birth to adulthood. To visualize the amount and rate of growth that takes place during each of these stages, the growth in height, or length, for healthy boys and girls from North America and Europe are depicted in Figure 2.5 (growth in weight follows very similar curves). In Figure 2.5 the distance curves of growth, that is the amount of growth achieved from year to year, are in panel A. The velocity curves, representing the rate of growth during any one year, are below in panel B. The velocity curve is labeled with symbols indicating the average duration of each stage of development.

From inspection of the velocity curves it is apparent that changes in growth rate and tempo are associated with each stage of development. Such changes in rate and tempo were shown in the previous chapter in relation to the growth of Montbeillard's son (Figure 1.4). Each stage may also be defined by characteristics of the dentition, by

changes related to methods of feeding, by physical and mental competencies, and by maturation of the reproductive system and sexual behavior.

The stages of the human life cycle presented here are an update from previous editions of this book and are based on work with my colleague Christiane Scheffler of Potsdam University. Dr. Scheffler alerted me to the fact that another stage scheme, with some similarity to my work, had been published in 1966 but was buried behind Cold War politics and behind the Berlin Wall in the former German Democratic Republic. The author was Hans Grimm (1910–1995), a German medical scientist and biological anthropologist. Grimm's developmental stage scheme was published in his book *Grundriss der Konstitutionsbiologie und Anthropometrie* (Compendium of Biological Constitution and Anthropometry, Grimm, 1966). Here he gave a classification of biological stages from birth to death based on anatomical, physiological, and behavioral markers. Grimm also included some social aspects of human development. In his book, Grimm referred to a symposium held in Moscow in 1965, where the participants discussed a "scheme of periods of biological stages" and came to a compromise about the biological stage classification. Presumably, the compromise position is the one that Grimm presented in his book. Scheffler and I, with our colleagues Carlos Varea of the Autonomous University of Madrid and Michael Hermanussen a pediatrician and researcher from Altenhof, Germany, republished Grimm's stage scheme in English, along with our update (Bogin et al. 2018c).

Presented here is an update on the stage classification system from the previous edition of this book, incorporating some of Grimm's proposals as well as additional and newer evidence on biology, socio-cultural traits, and cognition from the literature as well as direct research and clinical experience of the Bogin, Scheffler, Varea, Hermanussen team. We hope this new scheme (Table 2.1) better defines the stages of human growth, development, and maturation, as well stages in adulthood. Described here are some of the characteristics of each stage or period of growth and development. The ages of onset and offset for each stage are statistical approximations and averages for healthy individuals. In the real world, healthy individuals may vary considerably in their timing of growth, development, and maturation.

The Neonatal Stage

The **Neonatal phase** is a critical and stressful transition from intra- to extra-uterine environments. We limit discussion to full-term (37–42 weeks gestation) neonates of normal birth weight (2.5–4.3 kg) because preterm, low birth weight, or high birth weight neonates are at elevated risk of mortality. The medical technology needed to sustain such neonates has been available for only 50 years or less. Thus, during almost the entirety of human evolutionary history there was strong selection against neonates born outside the range of full-term and normal birth weight.

There is broad agreement by human biologists and neonatology that the period ends at 28 days after birth. Biologically, the 28-day period makes sense in that both the neonate and the mother make physiological adjustments during this time from pregnancy to extra-uterine life. The neonate must quickly adjust her own metabolism

to the extra-uterine environment, and this involves temperature regulation, breathing, sleeping, eating, digestion, elimination, and other autoregulatory processes (Ward Platt & Deshpande 2005). Even with these adjustments, the human neonate is altricial, that is, born in an undeveloped state and requiring care and feeding by the parents. Trevathan and Rosenberg (2016) characterize human neonate altriciality as due to having a large body relative to other apes, a small brain size relative to the human adult, and a prolonged time period of extreme motor immaturity relative to other ape neonates. These three traits are an unusual combination for a primate newborn. This combination makes human infants costly creatures to carry around, protect, and feed – burdens usually falling on the mother. Trevathan and Rosenberg (2016) emphasize that human altriciality is associated with maternal commitment to the neonate and that this has important behavioral implications for the social group. In Chapter 4 these implications are discussed in relation to the evolution of the human style of **biocultural reproduction**.

The relatively large body of the neonate is in part due to the fact that human infants are born with a greater reserve of fat than any other mammal (Kuzawa 1998). The human fat reserve not only allows for survival during the first few days after birth, but also fuels a rapid brain growth. By day 5 after birth human milk composition begins to mature in terms of energy and other nutrient content and is fully mature by 4–6 weeks postpartum (Ballard & Morrow 2013). The maturity of human milk at no sooner than 28 days after birth is, in our view, the single most compelling reason to define the duration of the neonatal period.

Infancy Stage

The infancy stage spans the next 35 months of human postnatal life, that is, to about age 3 years after birth. Infancy is characterized by the most rapid velocity of growth of any of the postnatal stages. During the first year of postnatal life infants may add 28 cm in length and 7 kg in weight, which represents more than 50% of birth length (about 50 cm) and 200% of birth weight (about 3.4 kg). The rate of decrease in velocity, or deceleration, is also very steep, which makes infancy the life stage of most rapidly changing rate of growth. The infant's curve of growth, rapid velocity and deceleration, is a continuation of the fetal pattern, in which the rate of growth in length reaches its peak in the second trimester and then begins a deceleration that lasts until childhood (Figure 2.6).

As for all mammals, human infancy is the period when the mother provides all or some nourishment to her offspring via lactation, or by some culturally derived imitation of lactation. One reason for this is that infants do not have teeth, and thus cannot eat solid food. During infancy the deciduous dentition (the so-called milk teeth) emerges through the gums.

Infants will erupt five deciduous teeth in each quadrant of their mouth, the central incisor, lateral incisor, canine, first molar, and second molar. Table 2.4 provides the median age of emergence, and range in age, of these teeth for French-Canadian infants. The deciduous teeth of boys emerge about one month earlier than girls. This

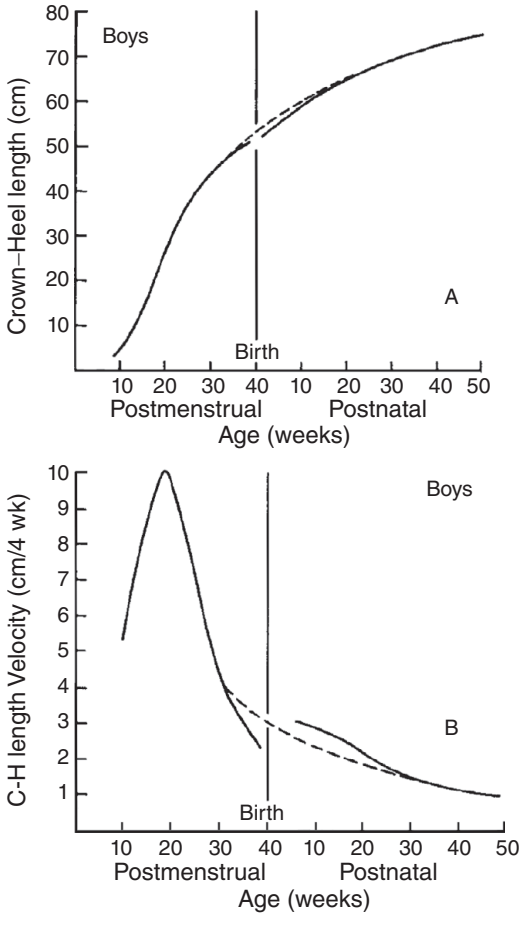


Figure 2.6 Distance (A) and velocity (B) curves for growth in body length during human prenatal and postnatal life. The figure is diagrammatic, as it is based on several sources of data. The interrupted lines depict the predicted curve of growth if no uterine restriction takes place. In fact, such restriction does take place toward the end of pregnancy and this may impede the flow of oxygen or nutrients to the fetus. Consequently, growth rate slows, but rebounds after birth and returns the infant to the size they would be without any restriction. (Tanner 1990)

is noteworthy only because in most other aspects of physical growth and maturation girls are, on average, ahead of boys.

One surprising feature of human growth during infancy is the similarity that most infants show in both amount and rate of growth in length and weight during the first six months of life. One might expect that variation in hereditary and environmental factors between individual infants and populations would lead to marked differences in amounts and rates of growth. However, Habicht et al. (1974) and Van Loon et al. (1986) showed that the growth of infants of normal birth weight, from a wide variety of ethnic and socioeconomic classes, in both the developed and developing nations, is remarkably similar during the first six months of life.

Table 2.4 The median and range in age (in months) of eruption of the deciduous teeth for healthy French-Canadian infants (after Demirjian 1986).

Boys		
	Median age (months)	Range (months)
Maxilla		
I^1	8.49	5.70–11.84
I^2	9.81	5.87–14.74
C	17.56	12.44–23.56
M^1	15.20	11.26–19.74
M^2	27.04	19.95–35.20
Mandible		
I_1	6.86	3.90–10.63
I_2	11.48	6.95–17.13
C	17.77	12.98–23.30
M_1	15.25	11.49–19.54
M_2	26.13	19.19–34.14
Girls		
	Median age (months)	Range (months)
Maxilla		
I^1	9.42	6.19–13.33
I^2	10.53	6.13–16.12
C	18.31	13.28–24.15
M^1	15.06	11.51–19.09
M^2	27.86	20.67–36.11
Mandible		
I_1	7.31	3.96–11.68
I_2	12.54	7.70–18.57
C	18.58	13.14–24.96
M_1	15.32	11.67–19.48
M_2	26.90	20.22–34.54

Abbreviations for the maxillary teeth are: I^1, central incisor; I^2, lateral incisor; C, canine; M^1, first molar; M^2, second molar. The same abbreviations with subscript numbers are for mandibular teeth.

These reports are supported by the World Health Organization (WHO) Child Growth Standards.[5] These growth standards were developed to indicate how healthy infants and children *should* grow, as opposed to growth references which indicate

[5] www.who.int/childgrowth/standards/en/

how those measured for the reference *do* grow, regardless of their health status. The WHO standards are based on the Multicentre Growth Reference Study (MGRS), which was undertaken between 1997 and 2003 to assess the growth and development of infants and young children around the world. The MGRS collected primary growth data at regular age intervals on length, weight, head circumference, arm circumference, subscapular skinfold, triceps skinfold, and motor development milestones from approximately 8,500 infants and children from Brazil, Ghana, India, Norway, Oman, and the USA. These six national samples represent widely different ethnic backgrounds and cultural settings. The requirements for inclusion in the samples were singleton pregnancy (no twins, triplets, etc.), no significant morbidity of the mother, normal gestation length, and a neonate of normal birth weight. The study families had to have socio-economic conditions favorable to growth (i.e., not be living in poverty), be stable in terms of housing with a low probability of mobility to a new home, with mothers willing to uniquely breastfeed for six months, and no maternal smoking. The rationale for these restrictions was that infants and children growing up under these conditions would provide data to establish a new, " . . . single international standard that represents the best description of physiological growth for all children from birth to five years of age and to establish the breastfed infant as the normative model for growth and development" (WHO Multicentre Growth Reference Study Group 2006, p. 7).

The infants from the six countries of the WHO standard grow similarly, not only for the first 6 months, but until age 59 months. Perhaps breast-feeding, which supplies the nutrients, immunity, and psychological needs of the infant, overrides the effect of variations in other aspects of the environment. The studies by Habicht *et al.* and van Loon *et al.* reported that after six months of age, when breast milk alone no longer meets the nutritional demands of the growing infant, and other specially prepared infant foods must be supplemented; infants from the developed nations or higher socioeconomic classes may become significantly larger than their less privileged age-mates from poorer environments. If alleviated by improved nutrition and health status early on, the disadvantaged children may catch up in size (as shown by Pagliani in 1876). Otherwise, the differences in size between the well-off and the deprived become greater and greater, and by childhood the differences may have become irreversible. This seems to be the case for Guatemala Maya children, who are significantly shorter than non-Maya children when all first attend primary school and remain shorter through adolescence (Bogin et al. 1992).

The outline of growth and development of Table 2.1 divides Infancy into two parts: Early and Late Infancy. The distinction is the degree of motor skill acquisition (Figure 2.7) and the use of shared intentionality (defined below) in social behavior after ~1 year of age (Bogin and Smith, 2012; Bogin et al. 2014b).

Motor skills (what a baby can do physically) develop rapidly during infancy. At birth, states of wakefulness and sleep are not sharply differentiated, and motor coordination is variable and transient. By one month the typical infant can lift its chin when prone and by two months lift its chest by doing a "push-up." By 4 months the infant

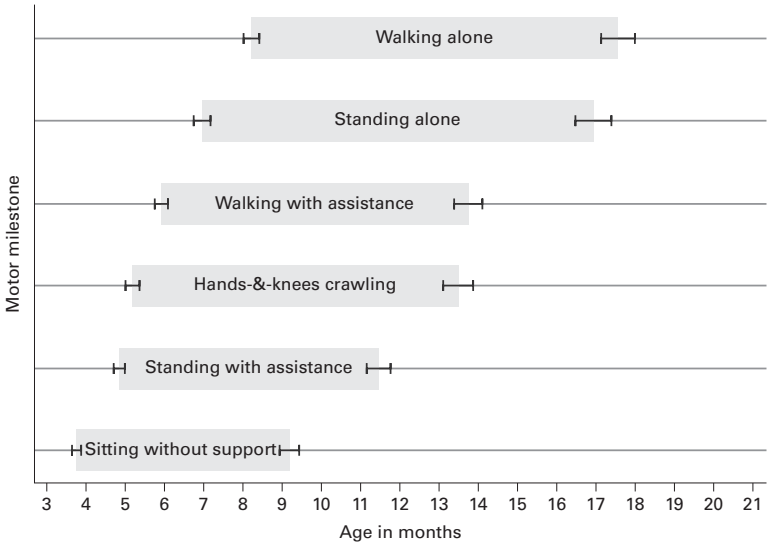


Figure 2.7 Windows of achievement for six gross motor milestones. Source: World Health Organization Multicentre Growth Reference Study Group (WHO Multicentre Growth Reference Study Group 2006) with permission.

can sit with support, by 7 months sit without support, by 8 months crawl, and by 10 months walk with support. Early infancy ends at about 12 months as the first unsupported bipedal steps are taken. With the transition to the Late Infancy period, motor development of walking, running, object manipulation, and other skills continues to take place in a, mostly, gradual manner for many years. By two years of age the infant can walk well and turn the pages of a book, one at a time. By three years of age, the end of the infancy stage, the youngster can run smoothly, pour water from a pitcher, and manipulate small objects, such as blocks, well enough to control them.

There is a similar gradual progression of changes in the problem solving, or general cognitive, abilities of the infant. In contrast, some specific motor-cognitive skills, especially language, develop more rapidly. In Table 2.1 the emphasis is on linguistic development, as it is the outcome of interactions between physical growth, motor-sensory development and control, brain development, and cognitive maturation (Locke & Bogin 2006). Griffiths (1954) observed that an infant is able to recognize her/his own name at an age of 12 months. Within a few months, infants develop a suite of verbal and cognitive traits that improve verbal efficiency and creativity. Locke and Bogin (2006, pp. 261–262) add that by the end of late infancy, at the age of 36 months, " ... the rudiments of a structural linguistic system, and basic components of a functional communicative system, are operative."

The infant's language skills are centered on the ability to infer the intentions of others and the disposition to align these intentions with the infant's own physical and emotional states. These abilities are called "theory of mind" or "shared intentionality" (Povinelli & Preuss 1995; Tomasello et al. 2005). Human infants

develop this skill to a greater degree than any other species and refine it throughout the growing years and into adulthood. Some scholars suggest that the intensity of shared intentionality is the basis of the evolution of the human brain and mind (Hrdy 2009). More about this is discussed in Chapter 4.

The end of the Late Infancy period is marked for most youngsters by completion of the eruption of all deciduous teeth, the transition from breast-feeding to complementary foods (i.e. weaning per Sellen, 2006, 2007), motor abilities such as walking forward and backward easily, and language/cognitive skills such as understanding "same and different," counting, sorting objects by shape and color, speaking more than 250 words, often in sentences of 5–6 words, and telling stories with elements of pretense and fantasy.

The development of the skeleton, musculature, and the nervous system account for these motor and cognitive advancements. The rapid growth of the brain is especially important. The human brain grows more rapidly during infancy than almost any other tissue or organ of the body (Figure 2.8). All parts of the brain seem to take part in this fast pace of growth and maturation, including those structures that control the reproductive system. The hypothalamus, a center of neurological and endocrine control, is one of these brain structures. During fetal life and early infancy, the hypothalamus produces relatively high levels of gonadotropin-releasing hormone (GnRH). This hormone causes the release of luteinizing hormone (LH) and follicle stimulating hormone (FSH) from the pituitary. LH and FSH travel in the bloodstream to the gonads (ovaries or testes) where they stimulate the production and release of estrogen or androgen hormones (see Figure 6.10). These gonadal hormones are, in part, responsible for the rapid rate of growth during early infancy. By late infancy, however, the hypothalamus is inhibited for reasons not completely known. GnRH secretion virtually stops, and the levels of the sex hormones fall, which suspends reproductive maturation (see Figure 2.8 "Reproductive" curve). The hypothalamus is

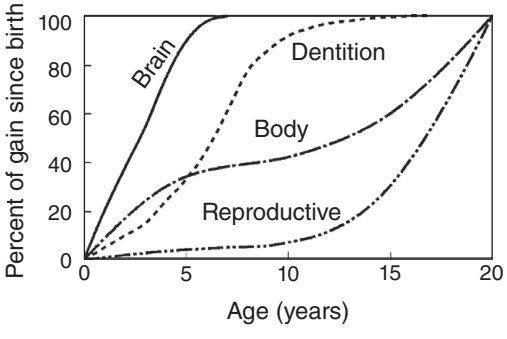


Figure 2.8 Growth curves for different body tissues. This figure was inspired by Richard Scammon's (1930) figure shown in Chapter 1 (Figure 1.8).The "Brain" curve is for total weight of the brain (Cabana et al. 1993). The "Dentition" curve is the median maturity score for girls based on the seven left mandibular teeth (I_1, I_2, C, PM_1, PM_2, M_1, M_2) using the reference data of Demirjian (1986). The "Body" curve represents growth in stature or total body weight and the "Reproductive" curve represents the weight of the gonads and primary reproductive organs.

reactivated just before puberty, the event of development that marks the onset of sexual maturation.

The age of emergence of the last deciduous tooth, usually M^2, is important for this is one of the events that signal the end of infancy. Emergence of all the deciduous teeth allows the infant to switch from dependence on breast-feeding (or formula/infant food feeding) to eating appropriate weaning foods. The strict definition of weaning used in this book is the termination of breast-feeding. In pre-industrialized societies, such as hunters and gatherers, horticulturists, pastoralists, and farmers depending on animal power (also referred to as traditional societies), weaning occurs between 24 and 36 months of age. By this age all the deciduous teeth have erupted, even for very late maturing infants. Thus, by 36 months of age there occur both biological developments (tooth emergence) and behavioral changes in the mother–infant relationship (weaning), as well as the motor, cognitive, and linguistic achievements mentioned above, that signal the end of infancy.

Childhood Stage

The childhood stage follows infancy, encompassing the ages of about 3–6.9 years. Childhood may be defined by its own pattern of growth, feeding behavior, motor development, and cognitive maturation. The rapid deceleration in rate of growth that characterizes infancy ends at the beginning of childhood, and the rate of growth levels off at about 5 cm per year. In terms of feeding, children are weaned from the breast, or bottle, but are still dependent on older people for food and protection. Most mammalian species move rapidly from infancy and its association with dependence on nursing from the mother, to a stage of independent feeding and do not experience a childhood stage or period of growth and development.

Post-weaning dependency is found in several species of social mammals, especially carnivores (such as lions, wild dogs, hyenas) and in some species of primates (Ewer 1973; Goldizen 1988). Lion cubs, for example, are weaned at about 6–8 months old, but remain dependent on their mothers until about 24 months old. During that time the cubs must learn how to hunt for themselves. Many species of primates must also learn to hunt for high-quality foods, such as insects, and learn how to open fruits and seeds with tough skins. This learning also requires a period of post-weaning dependence on the mother and sometimes the father (as for marmosets and tamarins).

Post-weaning dependency is, by itself, not a sufficient criterion to define human childhood. Human children do, of course, learn how to find and prepare food, but there is a suite of features that define the childhood stage. Many of these features are not found for the social carnivores and nonhuman primates. Human children require specially prepared foods due to the immaturity of their dentition, the small size of their stomachs and intestines, and the rapid growth of their brain (Figure 2.8). The metabolic activity of the human brain is especially important. The newborn uses 87% of its resting metabolic rate (RMR) for brain growth and function. By age 5 years the RMR usage is still high, at 44%, while in the adult human the figure is between

20 and 25% of RMR. At the comparable stages of development the RMR values for the chimpanzee are about 45, 20, and 9% respectively (Leonard et al. 2007).

Research finds that energy requirements of the body peak during childhood, measured as RMR per kg body weight or as daily energy requirement expressed in grams of glucose per day per kg body weight (Kuzawa et al. 2014). The brain, which grows rapidly during infancy and childhood, is especially greedy for energy. According to the data presented by Kuzawa et al. (2014), the life history transition from Infancy to Childhood takes place when brain glucose uptake exceeds 100–110 g day^{-1}.

The human constraints of immature dentition and small digestive system necessitate a childhood diet that is easy to chew and swallow and low in total volume. The child's relatively large and active brain, almost twice the size of an adult chimpanzee's brain, requires that the low volume diet be dense in energy, lipids, and proteins. Children do not yet have the motor and cognitive skills to prepare such a diet for themselves. Children are also especially vulnerable to disease and accidents and thus require protection. In past times, and in some areas of the world today, children were also targets for predatory birds and mammals. There is no society in which children survive if deprived of this care provided by older individuals. So-called wolf children (referring to children reared by wolves or children living "wild" on their own) and even "street children," who are sometimes alleged to be living on their own, are either myths or, in fact, not children at all. A search of the literature finds no case of a human child, that is a youngster under the age of six, living alone either in the wild or on urban streets (Bogin 2006).

Some years ago, Johnston (1986) observed that one of the more striking features of human growth during the childhood is its predictability in terms of changes in height, weight, and body composition. The distance and velocity curves for height depicted in Figure 1.4 are examples of the predictability of childhood growth. This French boy, the son of the Count Montbeillard, was raised in the countryside under near-optimal conditions for that time. Though this figure represents but a single child, the pattern of growth of all normal children follows a very similar course. For example, the boy gained 59.9 cm in height between his 2nd and 12th birthdays (Scammon 1927). Children of generally middle socioeconomic class born in the United States during the 1960s and early 1970s, average a 61.6 cm gain in height between their 2nd and 12th birthdays (Hamill et al. 1977). The difference between the gains in height of the French boy and the US sample are within the range of expected biological variation. The similarity in growth between a child and a sample of children, across time periods, and across geographic boundaries, emphasizes the common pattern of growth shared by all normal children and the predictability of this pattern. These features of human growth have important practical implications. For instance, they form the basis of epidemiological and clinical examinations that detect pediatric health disorders by searching for deviations in the expected trajectory of growth.

Though the pattern of childhood growth is predictable, there are several factors which may influence the amount and rate of growth of the individual child or groups of children. These factors include heredity, nutrition, illness, socioeconomic status,

and psychological well-being. Chapters 5 and 6 of this book are devoted to a detailed discussion of the action of these factors, and their combined interactions, on growth and development. Here it may be briefly stated that, all other factors being equal, short or tall parents are likely to have children who achieve similar stature. However, malnutrition, chronic illness, poor living conditions, and chronic psychological stress can retard the growth of a child. An example of all these factors combining at the same time is the case of the "Maya in Disneyland" described in the Introduction. The Maya people of rural Guatemala have the shortest height-for-age, and shortest adult height, of all human populations (Varela-Silva et al. 2016), aside from African and Asian pygmy populations who are short for genetic and neuroendocrine reasons.

Two of the important physical developmental milestones of childhood are: (1) the replacement of the deciduous teeth by the emergence of the first permanent teeth and (2) completion of growth of the brain in weight. Eruption of M1 (eruption is usually defined as first appearance of the tooth through the gingival, or gum, surface) takes place, on average, between the ages of 5.5 and 6.5 years in most human populations. Eruption of the central incisor (I1) quickly follows, or sometimes precedes, the eruption of the first molar. There is some variation between human populations in the age of eruption, but the number of permanent teeth in 7-year-olds varies from about 8 teeth in European "whites" (AlQahtani et al. 2010) to 9 or 10 teeth in African Zulu and Bantu groups (MacKay & Martin, 1952) and urban black Africans in Johannesburg, South Africa and Gaborone, Botswana (Esan & Schepartz 2018). By the end of childhood, usually at age seven years, most children have erupted the four first molars and, in addition, permanent incisors have begun to replace "milk" incisors. Within another year the four lateral incisors will also erupt, replacing the "milk teeth" that had been in that position. Along with growth in size and strength of the jaws and the muscles for chewing, these new teeth provide sufficient capabilities to eat a diet like that of adults. The mean age of eruption of the permanent dentition of boys and girls is given in Table 2.5.

A close association between human dental development and other aspects of growth and maturation was noted in the early twentieth century by anatomists and anthropologists (Krogman 1930; Schultz 1935). More recently, Holly Smith and colleagues (Smith 1991; Smith et al. 1994) analyzed data from humans and 20 other primate species and found that age of eruption of the first molar is highly associated with brain weight and a host of other growth and maturation variables. The correlation coefficient between age of eruption of the first molar and adult brain weight is $r = 0.98$ ($r = 1.00$ is a perfect positive relationship). The big brain of humans predicts a relatively late age of first molar eruption, and humans do have the latest age of all primates. Other research, based on direct measurements of victims of accidents and disease, shows that human brain growth in weight is mostly complete at a mean age of 7 years (Cabana et al. 1993), just when all first molars are erupted and in occlusion. This provides additional support for Smith's statistical analysis. Equally important is that at about age 7 years the child becomes capable dentally of processing an adult-type diet. With a slow rate of brain growth, the child has less risk of starvation because energy and nutrient requirements for brain growth have diminished.

Table 2.5 The mean age and standard deviation (in years) for the eruption of the permanent teeth for North American boys and girls (Smith et al. 1994).

Boys		
	Mean (years)	SD (years)
Maxilla		
I^1	7.34	0.77
I^2	8.39	1.01
C	11.29	1.39
PM^1	10.64	1.41
PM^2	11.21	1.48
M^1	6.40	0.79
M^2	10.52	1.34
M^3	20.50	–
Mandible		
I_1	6.30	0.81
I_2	7.47	0.78
C	10.52	1.14
P_1	10.70	1.37
P_2	11.43	1.61
M_1	6.33	0.79
M_2	12.00	1.38
M_2	19.80	–
Girls		
	Mean (years)	SD (years)
Maxilla		
I^1	6.98	0.75
I^2	7.97	0.91
C	10.62	1.40
PM^1	10.17	1.38
PM^2	10.88	1.56
M^1	6.35	0.74
M^2	11.95	1.22
M^3	20.50	–
Mandible		
I_1	6.18	0.79
I_2	7.13	0.82
C	9.78	1.26
P_1	10.17	1.28
P_2	10.97	1.50

Table 2.5 (cont.)

Mandible		
M_1	6.15	0.76
M_2	11.49	1.23
M_2	20.40	–

Abbreviations for the maxillary teeth are: I^1, central incisor; I^2, lateral incisor; C, canine; P^1, first premolar; P^2, second premolar; M^1, first molar; M^2, second molar. The same abbreviations with subscript numbers are for mandibular teeth.

During late infancy and childhood human locomotive skills develop and mature. Nakano and Kimura (1992) reviewed previous research on human and nonhuman primate locomotive development and presented some of their own new research. At age three years, the beginning of childhood, the human is still a "toddler," that is, able to walk bipedally but without the efficiency and characteristic gait of the adult. Nakano & Kimura find that by age seven years, on average, humans can walk with the adult-type efficiency and gait. A study by Kramer (1998) examined the energy costs of locomotion in children and juveniles. Children use more energy per kilogram of body weight when walking than do adults. Children aged 5–6 years old are about 85% efficient as adults. Juveniles aged 7–8 have more than 90% the efficiency of adults. The onset of adult-style locomotion, along with the eruption of the first permanent teeth, and the end of brain growth, are all indicators that the physically dependent child is moving on to independence.

The end of childhood has also been associated by a small increase in the velocity of height and weight growth, as depicted in Figure 2.5. This change in growth velocity has been called the mid-growth spurt (Tanner 1947). This small increase in growth velocity was first noted by Backman (1934), and later by other researchers. Some studies report the presence of the mid-growth spurt in the average velocity curve of boys, but not girls. Other studies report that about two-thirds of boys and girls had mid-growth spurts. Analysis of data from the Edinburgh Longitudinal Growth Study, 80 boys and 55 girls, found that all the boys and all but one of the girls show a mid-growth spurt at age 7 years for the boys and 6.7 years for the girls (Butler et al. 1990), but also other prepubertal spurts.

Varying methods of statistical analysis may explain the differences in findings between these studies. Especially important are the effects of data "smoothing" techniques, which are often used in longitudinal analysis that would tend to obliterate the mid-growth spurt. Curve fitting is also used commonly in longitudinal analysis and many curve-fitting routines assume that the mid-growth spurt does not exist. Some of the shape-invariant models (SIM) of growth assume that the mid-growth spurt does exist. The concept of SIM is that a population of growing girls and boys has a common characteristic curve of growth, such as that shown in Figure 2.5. This curve can be modeled with a mathematical function, " ... which by shifting and scaling can be

made to have the form of any individual curve" (Beath 2007, p. 2550). SIMs for human growth may assume the existence of the mid-growth spurt and, therefore, force the model to find it, even if it does not exist. Other factors that may influence the ability to detect the mid-growth spurt are the number of serial measurements of height available for analysis (few measurements make detection difficult), and the frequency of measurements (whether annual, semi-annual, or more frequent).

An elegant statistical analysis of prepubertal growth by R. Darrell Bock (1927–) used data from the Fels and Edinburgh longitudinal studies of human growth to search for the mid-growth spurt (Bock 2004). Both studies measured height at six-month intervals. Building on the Edinburgh study analysis, Bock looked for multiple pre-pubertal "spurts," or accelerations and decelerations in the growth of the Fels girls and boys from southwest Ohio, USA. Bock found multiple spurts, varying in number from zero to four in both girls and boys. The later maturing children tended to have more of these mini-spurts. Two sets of triplets, one all boys and the other all girls were analyzed. The monozygotic pairs (identical genome) of the triplets were not more concordant in number of timing of the mini-spurts than they were with their dizygotic sibling. Bock concludes that the finding of multiple mini-spurts occurring at 2–3 year intervals in these two geographically distinct samples, plus the lack of any obvious genetic concordance, indicates that the prepubertal mini-spurts are, " . . . episodes of random change in rate of growth prior to the onset of adolescence . . . " (p. 59). This conclusion supports an earlier study of 73 healthy children, aged 2.9 to 15.9 years old, measured once or twice per week throughout periods of 180 to 306 days. The measurements were of knee height, using the knemometer device invented by the lead author (Hermanussen et al. 1988a). The knemometer provides an accurate lower-leg length measurement (Hermanussen et al. 1988b). Nonlinearity of growth was found for 45 participants, with characteristic mini growth spurts occurring every 30 to 55 days. These mini-spurts seemed to appear spontaneously, " . . . though in some cases we found a marked coincidence between periods of growth arrest and intermittent infectious illness" (p. 103).

It seems that these mini-spurts, including the well-known mid-growth spurt at about age 7 years, are random events. They are likely to be perturbations of growth rate caused by environmental events that either slow or accelerate linear growth, including illness, hormonal events, and emotional factors. These influences on the amount and velocity of growth are discussed in more detail in Chapters 6 and 7. Suffice it to state here that the mid-childhood spurt is one of several possible mini-spurts of the infancy and childhood periods of human growth.

Juvenile Stage

Juvenile mammals are, for the most part, responsible for their own feeding and protection. Juveniles are also not sexually mature and rarely, if at all, practice any mating (Pagel & Harvey 2002; Pereira & Fairbanks 2002). This definition is derived from ethological research with social mammals, especially nonhuman primates. It applies to the human species, except that human juveniles remain part of family and

larger social groups and both receive and contribute food to the group. More about the biocultural nature of human families and societies is presented in Chapter 4. In contrast to infant and human children, juvenile primates can survive the death of their adult caretaker. The human primate is no exception to this, as ethnographic research shows that juvenile humans have the physical and cognitive abilities to provide much of their own food and to protect themselves from accidents and disease (Blurton-Jones 2002; Weisner 1987). Remember those so-called street children mentioned above – they are in fact "street juveniles!"

The Juvenile period is divided into two parts. The first part is called the *Pre-pubertal stage*, from about the ages of 7.0–9.0 years in both girls and boys. The second part of the Juvenile period is called *Puberty*, which is defined as an event of relatively short duration (1–2 months). Some researchers consider Puberty to be a life history stage of several years duration (Ellison et al. 2012), whilst other literature uses the words "puberty" and "adolescence" interchangeably. Here a distinction is made between Puberty as a life history transition event and Adolescence as a life history stage of growth and development.

During the Pre-pubertal period of the Juvenile stage, the rate of growth declines to its slowest rate since birth. This decline follows the mid-growth spurt in those children who experience it. But even in children without a detectable mid-growth spurt the rate of growth declines. This slow rate of growth applies to global measures of size, such as stature and weight, as well as to individual tissues, organs, and body systems. During the Pubertal period of the Juvenile stage growth rates increase.

Juvenile growth, like childhood growth, tends to be predictable, stable, and harmonious. The growth in height of two groups of Guatemalan juveniles between the ages of 7 and 13 years, shown in Figure 2.9, is an example of a predictable and stabilized difference in size. Both groups live in Guatemala City and attend school. The larger juveniles are from high SES families, the smaller juveniles are from low SES families. The high SES group is about the same size as healthy, well-nourished juveniles from the United States. The families of the low SES group are known to

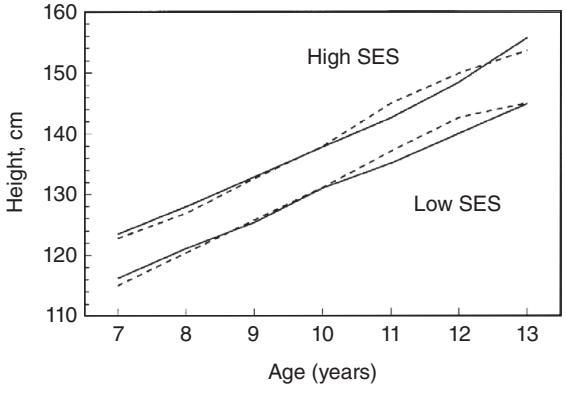


Figure 2.9 Mean heights of Guatemalan boys (solid lines) and girls (dashed lines) of high and low socioeconomic status (SES). (From Bogin & MacVean 1978)

suffer from inadequate nutrition, primarily a shortage of total food intake, poor living conditions in terms of health care, and insecurity in terms of water and sanitation, education, physical safety, and emotional stability (Bogin & MacVean 1978, 1981, 1983). Though unequal in size, these children all display a similar regularity in their growth as may be seen from the mean values plotted in the figures. The differences in size between girls and boys are not of biological importance at these ages. Even so, the pattern of growth by sex is virtually identical in both high and low SES samples, which is further evidence of the predictability of juvenile growth. Generally, changes in size from age to age are similar for both SES groups. Indeed, a formal statistical comparison using the technique of tracking analysis (i.e., does the pattern of growth in one sample parallel, or track, the pattern in another sample?) finds a high degree of tracking between the groups. The parallel tracking means that the differences in height and weight between these two groups were established prior to age 7 years. Most likely this difference is the result of growth retardation in infancy and childhood due to the adverse living conditions of the low SES group.

Growth in weight follows a similar pattern in these Guatemala juveniles and the harmony of the height-weight relationship may be seen in Figure 2.10. Juveniles from both SES groups have the same height for weight proportionality regardless of absolute size. The "regression line" drawn in Figure 2.10 represents the best-fitting straight line (estimated by the statistical method of least squares) drawn through the data points for the high SES juvenile boys and girls. The data points of the low SES juvenile boys and girls show no statistically significant deviations from the regression line. The maintenance of proportionality under the stress of low SES reflects the stability, predictability, and harmony of juvenile growth. Since both height and weight are equally affected in the low SES Guatemalan juveniles, it is likely that

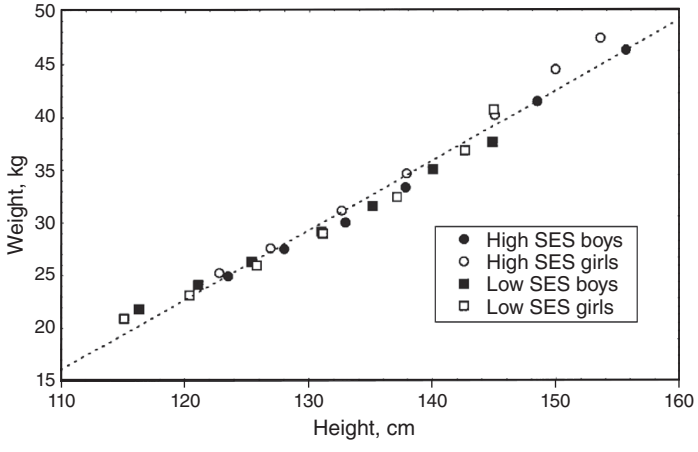


Figure 2.10 Relationship of height to weight for Guatemalan boys and girls, aged 7.00 to 13.99 years old, of high and low socioeconomic status (SES). The broken line is the linear regression, the "best fitting" straight line running through the data points. (From Bogin & MacVean, 1978)

some common mechanism is regulating the growth of several different tissues (e.g., bone, muscle, adipose). The exact nature of this mechanism is not known.

In her article "Harmony of growth," Elsie Widdowson (1906–2000) observed that animal growth is a complex affair, but to some extent a harmony of growth is displayed in the normal development of many body parts not only during the juvenile stage of life, but also during all stages of human growth and development (Widdowson 1970). For example, from childhood to adulthood there is a coordinated growth of the teeth and the craniofacial complex of bones (mandible, maxilla, etc.) that maintains the functional integrity of the masticatory system. Without a harmony of growth of these bones and the teeth the growing individual would not be able to eat. Another example is the phenomenon of catch-up growth (Prader et al. 1963). This is a rapid increase in growth velocity following a short-term period of starvation or illness which slowed or stopped growth. An example is growth failure from Celiac disease, which results from an immune system reaction to gluten, a protein found in wheat, barley, and rye (Figure 2.11). The patient was growing at about the 50th centile of a growth reference chart until age 4 years, but then the rate of growth slows, and the child falls below the 3rd centile by age 6 years. Grow rate remains very slow until the introduction of a gluten-free diet at age 12 years. The rate of growth then rockets upwards and at age 17 years the patient is nearing a return to the 50th centile. Note that the rate of catch-up growth slows each year after the diet change, so that the increase in growth velocity restores a youngster to the size he or

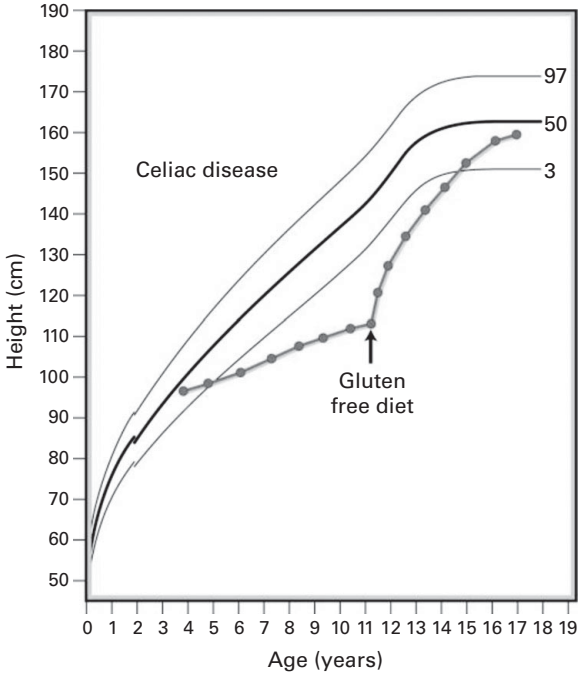


Figure 2.11 Catch-up growth in a patient with Celiac disease (Source: Public Domain)

she would have achieved had there been no growth delay, but no further. This case is a clinical example of Tanner's model of growth control presented in Box I.2.

Many segments of the body, such as the head, neck, truck and extremities, must participate in catch-up growth and each segment tends to maintain its normal pattern of growth, that is, the affected individual does not end up with one leg shorter than the other or with arms stunted but legs of normal length. The nearly global nature of this harmony of growth has stimulated several researchers to propose a theoretical concept of growth as biologically self-regulating (Bogin 1980; Goss 1978; Tanner 1963). This is a topic that is discussed in more detail in Chapter 6 in relation to the physiology of the epiphyseal growth plate. A general discussion of genetic and hormonal regulation of human growth, development, and maturation is also presented in Chapter 6 which may be consulted to better understand the following discussion of puberty and adolescence.

The biology of Puberty, as studied in nonhuman primates and humans, begins in the brain and involves the hypothalamic-pituitary-gonadal axis, its hormones, and its afferent and efferent connections to the entire body (Ellison et al. 2012; Herbison 2016; Plant 2015a, 2015b). Puberty, as defined here, is the reactivation of the hypothalamic GnRH (gonadotropin-releasing hormone) pulse generator leading to a massive increase in sex hormone secretion (Grumbach 2002; Herbison 2016; Plant 2015a). This leads to maturation and the functioning of the gonads – ovaries and testes – which is sometimes called **gonadarche**. The current understanding of the control of gonadarche is that one, or perhaps a few, centers of the brain change their pattern of neurological activity and their influence on the hypothalamus. The hypothalamus, which has been basically inactive in terms of sexual development since about age two years, is again stimulated to produce GnRH. It is not known exactly how this change takes place.

The production of GnRH and its release in a pulsatile fashion is active in the mammalian fetus and neonate. In primates studied, including rhesus monkeys and humans, that activity declines after birth and the system is "turned-off" by about age 2 years. It is reactivated at puberty. The "inhibitor" has not been identified, but is probably located in the brain, and certainly not in the gonads. Human children born without gonads, or rhesus monkeys and other primates whose gonads have been surgically removed, still undergo both the inhibition of the hypothalamus in infancy, and the reactivation of the hypothalamus at puberty (Figure 2.12a). The transition from juvenile to adolescent stages requires not only the renewed production of GnRH but also its secretion from the hypothalamus in pulses (Figure 2.12b). Experimental non-human primate studies and clinical trials with humans show that the frequency of the GnRH pulses must occur each 90 minutes to initiate puberty (Ellison et al. 2012 review the literature). Various "trigger-factors" have been proposed as regulators of the pulse generator inhibition in infancy and its reactivation during the juvenile stage, such as neurokinin-B, melanocortins, kisspeptin, growth hormone, but none has proved to be directly causal (Avendaño et al. 2017; Ellison 2017; Plant 2015b). Signaling by epigenetic marks, microRNA pathways, and metabolic/nutritional factors are newly discovered to be associated with the control of puberty (Avendaño

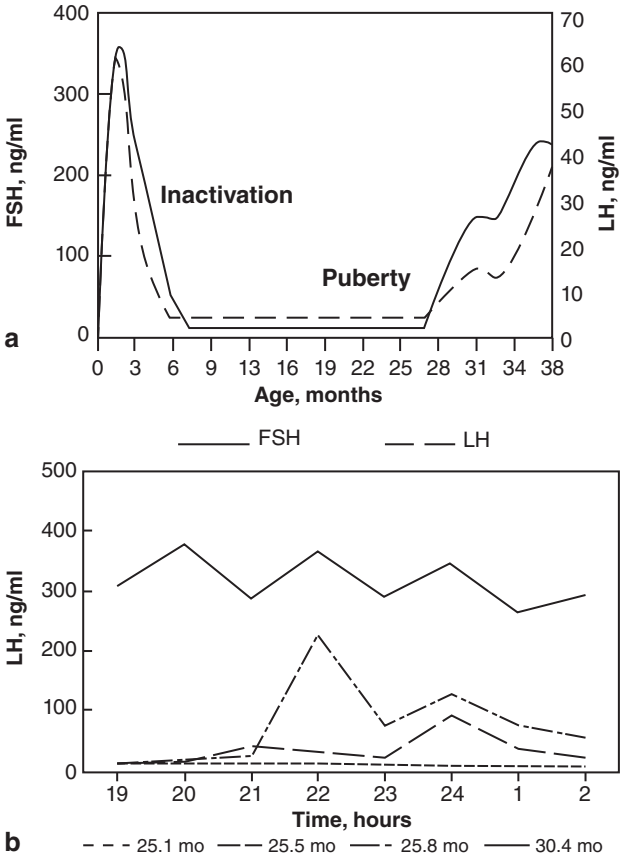


Figure 2.12 (a) Pattern of secretion of follicle stimulating hormone (FSH) and luteinizing hormone (LH) in a male rhesus monkey (genus *Macaca*). The testes of the monkey were removed surgically at birth. The curves for FSH and LH indicate the production and release of gonadotropin-releasing hormone (GnRH) from the hypothalamus. After age 3 months (i.e., during infancy) the hypothalamus is inactivated. Puberty takes place at about 27 months and the hypothalamus is reactivated. (b) Development of hypothalamic release of GnRH during puberty in a male rhesus monkey with testes surgically removed. At 25.1 months (mo) of age the hypothalamus remains inactivated. At 25.5 mo and 25.8 mo there is modest hypothalamic activity, indicating the onset of puberty. By 30.4 mo adult pattern of LH release is nearly achieved. The pattern of LH release shows both an increase in the number of pulses of release and an increase in the amplitude of release. In human beings a very similar pattern of infant inactivation and late juvenile reactivation of the hypothalamus takes place. This figure is redrawn, with some simplification, from (Plant TM 1994)

et al. 2017). According to Avendaño and colleagues, these factors may be arranged into categories of: (1) essential gatekeepers and activators of puberty, such as kisspeptin and many of the microRNAs; (2) permissive and amplifying factors, such as estradiol, leptin, and insulin; (3) inhibitors, such as ghrelin and at least two microRNAs; and (4) redundancies, such as the network of factors throughout the

body that regulate appetite and energy balance. Ellison (2017) emphasized the energy balance aspects of the pubertal transition. Using a life history theory perspective, he presented a mathematical model of puberty onset that is regulated primarily by insulin, growth hormone and their cascading effects on estradiol, prolactin, and testosterone.

Puberty is described by Ellison and colleagues as, " ... a life history transition" (Ellison et al., 2012, p. 352), involving changes in physiology, anatomy, behavior, social interests, emotional attitudes, and cultural values. The transition takes the person from the immaturity of the child and early juvenile to the incipient adultlike phenotype of the young adolescent. Due to the biocultural nature of the puberty transition, there is a long history of research and scholarship by anthropologists, sociologists, psychologists, life history biologists, physicians, epidemiologists, and other human scientists, as well as economists and philosophers. The book *Adolescence: An Anthropological Inquiry* is a primary source (Schlegel & Barry 1991). There are several "Encyclopedias of Adolescence," including two published in 2011, one with a more psychological and sociological perspective (Levesque 2011) and the other with more bioanthropology, including an entry on "Puberty and adolescence" (Bogin 2011).

To conceptually organize all the known regulators of puberty onset, Ellison and colleagues (2012) proposed an "hour-glass" model. The center, or "waist," of the hour-glass is the hypothalamic-pituitary-gonadal (HPG) axis. The various factors of the categories listed above, plus psychosocial stressors and photoperiod information, fill the top of the hour-glass, that is, the brain regions near or in communication with GnRH neurons, with signals and information. These are the upstream factors that flow into the HPG axis and exert their regulatory effects on GnRH neurons. Excitatory and inhibitory neurohormonal signals flow downstream from the HPG axis to synchronize the growth, development, and maturation of the ovaries and testes, as well as secondary sexual characteristics such as adipose and muscle tissue, libido, mating behavior, and the adolescent skeletal growth spurt.

Ellison (2017) refined this model by adding the analogy of "interlocking gears" to conceptualize the way that the different components of the pubertal system drive changes in the entire system. A variant on this concept, by Herbison and colleagues (2016), proposed that the Infancy inhibition and Juvenile reactivation of GnRH production and release is regulated by, " ... a series of interlocking functional modules ... " (Herbison 2016, p. 452), each with a variety on genomic, epigenetic, and environmental regulators.

None of the hormonal changes of puberty and gonadarche can be seen without sophisticated technology, but their effects can be noted easily since visible signs of sexual maturation appear. One such sign is a sudden increase in the density of pubic hair (indeed the term "puberty" is derived from the Latin *pubescere*: to grow hairy). Another sign, for girls, is the development of the breast bud, the first stage of breast development and for boys there are genital changes in coloration and growth of the testes. Skeletal growth rate transitions from deceleration to acceleration and the

point of this change marks the beginning of the adolescent growth spurt. The pubescent boy or girl, his or her parents, and other relatives, friends, and sometimes everyone else in the social group, can observe these signs of transition from the juvenile to adolescent stages of growth, development, and maturation.

In girls, the juvenile period ends, on average, at about the age of 10, two years before it usually ends in boys, the difference reflecting the earlier onset of the Adolescence stage in girls. The juvenile period is often accompanied by a pronounced, but short-lived decrease in rate of growth. The data for Montbeillard's son (Figure 1.4) show such a dip in velocity at about age 8–11 years. Similar dips are known from recent longitudinal studies of growth (Stützle et al. 1980), although not all children display them. The cause of this decrease in growth rate is not known, but may be explained by Tanner's time tally and inhibitor interactions (Box I.2). The nadir of this dip marks the end of the juvenile stage.

Adolescent Stage

For both girls and boys, the reversal in the rate of growth, from deceleration to acceleration at the end of the juvenile stage is one signal of the transition to adolescence (Figure 2.5). There are several other signals, because human adolescence is the stage of life when social and sexual maturation takes place. Human adolescence may be defined by a suite of maturational traits in physiological behavior, emotions, and cognition. Many of these characteristics are sex-specific, or appear in a different sequence for each sex. The production of viable spermatozoa, for example, occurs relatively early in the adolescent development of boys, but the production of mature oocytes is a relatively late event in the adolescence of girls. Every human society has beliefs, values, and practices that interact with the maturational events of adolescence in ways that assure a mutual influence and complex feedback. To understand human adolescence requires a biocultural perspective.

A comment on the current state of adolescence research is needed here. Since publication of the 1999 2nd edition of this book, scholarship and art on the nature of adolescence has flourished. This activity includes research in the biological, social, and medical sciences, and new depictions of adolescence and "the adolescent" in literature, poetry, theater, and film. Anthropology of adolescence, like anthropology of childhood before it, is a thriving field. Much of this "adolescence-ology" is divided into social vs. biological camps, premised on the notion that adolescence is a social construct. Examples of this divide in the bio-science literature may be found in articles such as "Culturing the adolescent brain" (Choudhury 2010) and the literature cited therein. Choudhury is part of a group researching cultural neuroscience, that is, ways in which differences between societies in beliefs, values, and experience influence the adolescent brain, emotions, and behavior. The approach is not biocultural, at least not in the way the word "biocultural" is used here. Other examples are in the February 2018 collection of articles and commentaries coordinated in the *Nature* publishing group (Anonymous 2018a). The most anthropological and evolutionary

Box 2.2 **Human growth and play**[6]

The human postnatal stages of infancy, childhood, and juvenile may be defined by the biological, socio-cultural, and cognitive signs listed in Table 2.1. In addition, styles of play behavior differ between stages. Developmental psychologists and human ethologists describe many types of human play, including pretend play, games of chance and skill, and language play. Each of these play types may be performed in formal or informal settings. But the focus in this box feature is on physical activity play, the type of play shared by all social mammals. Because physical activity play is shared across mammalian species, it is possible to consider such play in a biological and evolutionary context.

Play behavior is correlated with life history. For mammals with only an infant and adult stage of life the rate of play during infancy may be high, but it drops to near zero at the time of weaning. Social mammals – animals with infant, juvenile, and adult stages of life – follow a different trajectory of play behavior. Figure B2.2.1 is a model of the age trend in physical activity play of social mammals. Play is relatively infrequent during infancy; it peaks during the juvenile stage, and becomes infrequent again during adulthood. Well-known examples of this pattern are social carnivores, such as lions and most monkeys and apes. These animals begin to show physical-activity play via interactions with their mother – playing with her tail, and feet, or jumping on her body. By late infancy, the young are playing with each other in games of chasing and dominance. The intensity of this play increases during the juvenile years, but as the young become sexually mature their interest turns from play

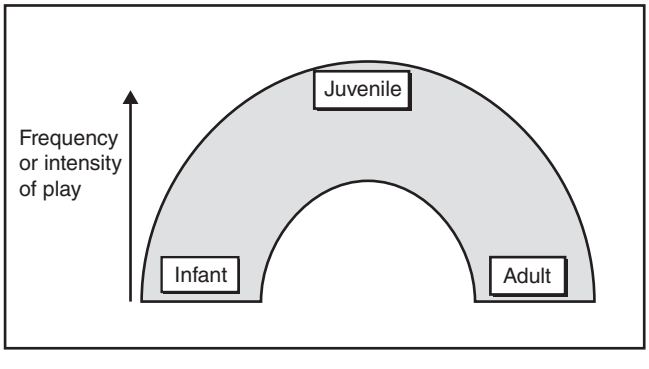


Figure B2.2.1 Age trends in social mammals' play.

[6] This text box is based on the chapter by Bogin (2002) "Childhood, play and growth." Readers may consult this chapter for bibliographic references and additional explanation of the importance of play behaviour for healthy human growth, development, and maturation.

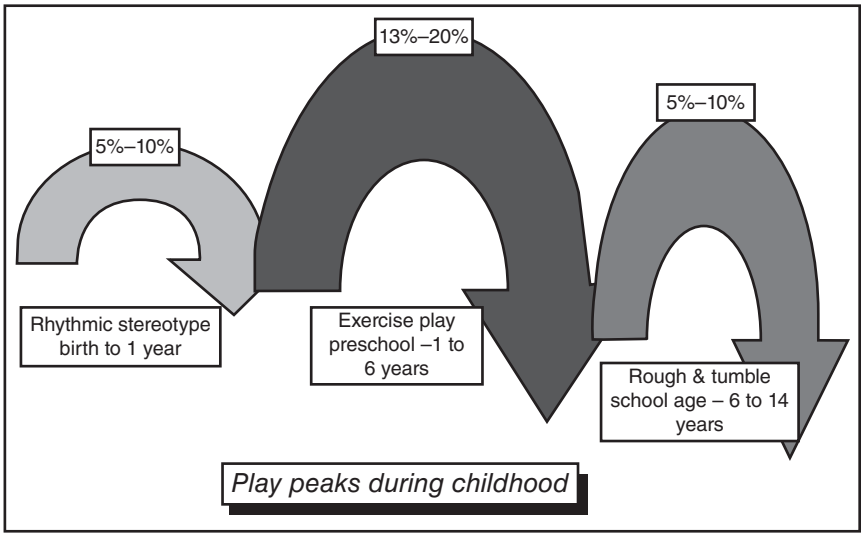


Figure B2.2.2 Age trends in human physical activity play. Data from Pellegrini AD, Smith PK. Physical-activity play: the nature and function of a neglected aspect of play. *Child Development* 1998; 69: 577–598

to adult activities. Nevertheless, many adult mammals continue to show some play behavior, especially with their juvenile young.

Age trends in human physical activity play are shown in Figure B2.2.2. The human age trends are more complex than in other social mammals. Part of the reason for this is the addition of the childhood stage of human development. The human pattern begins with "rhythmic stereotype" play. An example is when an infant, lying in a crib, repeatedly swats at a mobile suspended above its head. Body rocking and foot kicking are other examples of rhythmic stereotypes. The next type of physical activity play is called "exercise play." It begins during late infancy and its frequency increases during the childhood years of life. This type of play involves vigorous movement, such as running and jumping, and the manipulation of moveable objects, such as balls and blocks. The third physical play type, "rough and tumble" play, becomes common during late childhood and the juvenile stage. This type of play involves physical contact with other players, for example wrestling, gymnastic-like movements, and competitive games. From Figure B2.2.2 it is clear that human beings have an unusual pattern of development of physical and social play. Infants spend about 10% of their time in "rhythmic stereotype play." Children spend about 20% of their time in a style called "exercise play." Juveniles make a shift to "rough and tumble play," and reduce total time at physical play to 10%. Human play researchers report that the frequency of all

types of play, but especially physical activity play, peaks between the ages of 4 and 5 years, that is, during the biologically defined childhood stage of life.

The styles of human physical activity play during infancy, childhood, and juvenile stages work synergistically with brain development. The human brain undergoes wondrous transformations during the perinatal period and the first 6 years of postnatal life. This is a period of production of new nerve cells, the formation of neural connections via the synapse (a chemical junction between neurons) and the elimination of neurons by **apoptosis** and neural regression (the death and removal of neurons). The formation of new nerve cells, synapses, and synapse elimination occurs simultaneously and vigorously from the infant to the adolescent stage of life. Both humans and other primates experience a burst of synaptogenesis just before and after birth. The density of synapses in the auditory and visual cortexes in early life exceeds the adult density. The elimination of the "extra" synapses, often called "pruning," is impressive, with one estimate that between the ages of 2 and 5 years, 5,000 synapses are lost every second from the visual cortex.

Why does such a massive overproduction and then elimination of synapses take place? The answer seems to be learning. Synapse connectivity is considered to be the primary means by which knowledge is represented in the brain. It is not only the sheer number of synapses that matters, but also the networks by which the synapses make connections. The sculpting of nerve tissue from birth to adolescence allows for greater leaning because the near constant addition and elimination of synapses leads to greater plasticity in brain development. If synapses formed once, and only once, then learning and behavior could not be modified by new experience. Old knowledge that was no longer useful would have to be suppressed by other means to allow for newer, more useful, knowledge. Plasticity in brain development, allowed for by the overproduction and then pruning of synapses, overcomes these inefficiencies and permits a constant adjustment and improvement of learning and behavior.

The ontogeny of human play interacts with the plasticity of synapse formation and elimination to sculpt a brain that is best suited for the biocultural milieu in which people live. Play helps to make those "calibrations" between body and environment, especially the social-emotional environment. Play is essential for normal human development. Infants, children, and juveniles deprived of play may suffer from poor health, especially cardiovascular fitness, and being overweight. Lack of play also leads to impairments in cognitive function and inappropriate social behavior. It is noteworthy that physical activity play peaks during childhood. Cognitively, the child is in the preoperational stage of development, which accounts, in part, for physical and social dependence of children on a network of older people. Learning via play will be enhanced by the social interactions within this network. The childhood stage of growth and development is unique to our species and the intensity of play during this stage likely underlies many human social and cultural capacities.

Human beings have a longer time period of growth and development than most other mammals. This includes the longest time period for brain maturation. Humans depend on culturally-learned behaviors for survival, including complex technology, social organization, language, and ideology. Due to our biocultural nature, which in large part is due to the evolution and functioning of our brain, play behavior has a greater impact on healthy human growth than in any other species. In the modem world, with physical limitations from intense urbanization and its attendant risks (e.g., crime, motorized traffic, loss of open spaces) there are often fewer opportunities for physical play, and this imposes a risk for the healthy growth and development of young people.

article in that series, by Worthman and Trang (2018), adopts the concept that adolescence is a social construct, in contrast to puberty which is a biological universal of humans and many other mammals and birds. Puberty comprises the physical changes from reproductive immaturity to maturity – adolescence comprises the sociocultural variations on that reproductive transition.

The idea of this biological-social divide originated with American psychologist G. Stanley Hall (1844–1924). In 1904 he published a two-volume treatise, "Adolescence: Psychology and Its Relations to Physiology, Anthropology, Sociology, Sex, Crime, Religion, and Education."[7] Hall described adolescence as a transition stage of the soul (Hall 1904, p. vii). Hall was a firm believer in "races" and eugenics. Choudhury (2010) notes that Hall described people of African, Indian, and Chinese cultures as "adolescent races," whose development stagnated just after puberty. People of western European descent, in contrast, progressed to a fully-adult "race." In addition to his racism, Hall stated that in earlier times of human history there was no adolescence. From an early age, children worked alongside their parents and other adults and transitioned directly to adulthood. Adolescence was invented due to the industrial revolution, which replaced child labor with machines and, concomitantly, with the need for more formal education (schooling) to assure technological and social progress. This technology-based extension of childhood created the time for a social adolescence. As Europeans passed through the industrial age before Hall's other "races," they adapted to this social adolescence and progressed to a fully mature adulthood. Other "races," according to Hall, were still in the process of maturation.

Despite the totally unscientific nature of Hall's "races" and "souls," and his ethically bankrupt denigration of non-European societies, his influence on psychology was so powerful that many current textbooks, including those assigned for courses in anthropology and sociology, include his ideas without comment or

[7] https://archive.org/details/adolescenceitsps002hall

question. Little wonder that academics trained on these textbooks perpetuate the social-biological divide.

In the book before you, as in its previous editions, all "conventional wisdom" based on racism and eugenics is rejected. Here a fully integrated, cross-cultural, and holistic approach is taken to human development. Adolescence is a biocultural stage of human life history and is found universally in all human beings. There is no divide between the biological and sociocultural nature of adolescence. People at all stages of growth and development are biocultural animals. In the following paragraphs some examples of the inextricable biocultural connections and influences of human adolescence are given.

Sexual Development

The adolescent stage also includes development of secondary sexual characteristics, such as the external genitalia, sexual dimorphism in body size and composition (Figure 2.13), deepening of the voice in boys, as well as the onset of greater interest and practice of adult patterns of sociosexual behavior and economic behavior, such as food production in traditional societies. Some of these physical and behavioral changes occur with puberty in many species of social mammals.

What makes human adolescence unique are two important differences. The first is the length of time between puberty and adulthood, that is, full reproductive maturity. Humans take five to eight years for this transition. Monkeys and apes take less than three years. The second human difference is that during this life stage, both boys and girls experience a rapid acceleration in the growth velocity of virtually all skeletal tissue – the adolescent growth spurt (see Figure 2.5). The magnitude of this acceleration in growth was calculated for a sample of healthy Swiss boys and girls (112 boys and 110 girls) measured once a year, near their birthdays, between the ages of 4 and 18 years (Largo et al. 1978). At the peak of their adolescent growth spurt, called peak height velocity– (PHV), the average velocity of growth in height was +9.0 cm/year for boys and +7.1 cm/year for girls. Similar average values are found for adolescents in most human populations. In contrast to humans, other primate species either have no acceleration in skeletal growth or an increase in growth rate that is very small (Chapter 3 includes a detailed analysis of growth spurts in human and nonhuman primates).

Largo and colleagues (1978) analyzed several other features of the adolescent spurt in growth using the Swiss data. First, they ascertained that the age range of the sample, 4 to 18 years, is greater than the variation in the timing of the onset of adolescence. That onset occurred within the range of 6.6–13.5 years in the study sample, and thus the transition from juvenile to adolescent stage could be observed in every individual. This fact and the relatively large number of children measured repeatedly from year to year, gives confidence to the growth statistics derived from the study. Using the data published by Largo and colleagues, the change in acceleration of growth from childhood to adolescence can be calculated. During the juvenile

stage the deceleration in the rate of height growth averages −0.46 cm/year/year for boys and −0.48 cm/year/year for girls. From the point of minimal juvenile velocity to the peak of the adolescent growth spurt the acceleration in height averages +1.66 cm/year/year for boys and +0.88 cm/year/year for girls.

The change in the velocity and acceleration of growth at adolescence affects almost all parts of the body, including the long bones, vertebrae, skull and facial bones, heart, lungs, and other visceral organs. Not all parts of the body experience the adolescent spurt at the same time. Different regions of the skeleton, for example, reach the peak rate of growth during adolescence at different ages. In a nationally representative sample of 18,004 girls from the former East Germany, measured by Holle Greil (Greil 1997), the average age at peak velocity of the spurt occurred at about 9 years for foot length, 10 years for the hand length, 10.5 years for leg length, and 12 years for arm length, standing height, and trunk length. The sample of boys measured by Greil, totaling 18,123 individuals, followed the same general sequence of peak velocities, but the boys reached those peak values 2–3 years later than the girls. A study of the skeletal sequence of adolescent spurts of Japanese boys (n = 15) and girls (n = 20) measured longitudinally from age 9.0–15.0 years was published by Satake et al. (1994). They found that, on average, the spurt in leg length preceded the spurt in trunk length, biacromial diameter (bony shoulder breadth), and biiliac diameter (bony hip width) in both boys and girls. Again, girls reached these spurts at an average earlier age than boys, but the sex difference was only about one year. Satake and colleagues noted that there was considerable variation in this sequence for individual subjects, so much so that order of this sequence could, " . . . provide a unique method of clarifying individual growth variations" (p. 359).

As noted in the previous chapter, Buffon observed that the adolescent spurt began at an earlier age in the higher social classes compared with lower classes. It is unclear if Buffon understood that the spurt is not only earlier in the well-off and better fed, but those so privileged also have a greater amount and intensity of the spurt. Indeed, adolescence is the stage for final adjustments in body size and shape, and often these final adjustments set apart groups of people according to social class (Aßmann & Hermanussen 2013). The timing and **intensity of growth** are separate phenomena and it took some effort during the twentieth century to appreciate the difference. Increasing emphasis has recently been put on separating tempo (the pace, or "time signature," of development and maturation) and amplitude (the maximum rate of growth at a specific state of maturity) (Hermanussen & Bogin 2014; Tanner 1971). Many of the traditional concepts of growth have recently been questioned in view of this dichotomy. For more than half a century, scattered observations exist on both tempo and amplitude in starvation and illness. Starved populations are not necessarily short populations, but they develop at slow pace; well-nourished and economically affluent populations are not necessarily tall. Brundtland and colleagues (Brundtland et al. 1980) published an excellent example that even longstanding starvation does not influence final height. The marked growth impairment in Oslo schoolgirls at the time of the German occupation during World War II was not impairment in amplitude, it was impairment in tempo. The formerly starved cohorts later achieved normal adult height. Similar

observations in war- and post-war school children were published in Germany and elsewhere (Hermanussen & Bogin 2014). Tempo impairment has also been observed in chronic illnesses, for example, cystic fibrosis (CF) and certain endocrine disorders in which patients grow poorly at all ages, but eventually achieve normal final height. More about these topics of growth tempo vs. amplitude and the relation of food shortage or excess and growth is presented in Chapters 5 and 6.

The spurt encompasses more than the skeleton. Muscle mass of boys also undergoes a spurt at adolescence, and it is relatively greater than the spurt for growth in height. After the peak of the skeletal spurt, the rate of bone growth declines more steeply than the rate for muscle growth, meaning that adolescent boys continue to increase their muscle mass at a faster rate than they grow in stature. Expressed another way, the average healthy boy reaches 91% of his adult height by his age at PHV during adolescence, typically about 14 years of age in healthy well-nourished populations. However, the same average boy achieves only 72% of his total muscle mass at the age of PHV and takes about 4 more years to reach 91% of his adult value (Butler 1990).

Some body parts and tissues do not evidence an adolescent growth spurt, for example, adipose tissue, both subcutaneous fat and the deep body fat, decreases in mass during adolescence in British and American boys, and perhaps in many girls as well (Johnston et al. 1974; Tanner 1990). Lymphatic tissues and the thymus show no adolescent increase in growth rates (Scammon, 1930). Another body part unaffected by the adolescent growth spurt is the female pelvis. Marquisa LaVelle (Moerman 1982) measured the growth of the **pelvic inlet**, the bony opening of the birth canal, from X-rays taken on a sample of healthy, well-nourished American girls who achieved menarche between 12 and 13 years. These girls attained adult pelvic inlet size at 17–18 years of age. Quite unexpectedly, the adolescent growth spurt, which occurs before menarche, did not influence the size of the pelvis in the same way as the rest of the skeleton. Rather, the pelvis and pelvic inlet of these girls followed its own slow pattern of growth, which continued for several years even after adult stature is achieved.

One consequence of the pattern of growth of the female pelvis is that adolescent girls under age 17 who become pregnant risk their own health and the health of their fetus. This is due, in part, to the immaturity of the size of their pelvic inlet. Other risks of teenage pregnancies are due to the mother's on-going growth in height and weight (bone, fat, muscle, etc.). Her own growth diverts nutrients away from the fetus. The result may be growth retardation of the fetus leading to a low birth weight infant. These risks, and the biological and social strategies that humans have developed to avoid them, are discussed in more detail in relation to the evolution of the human life cycle in Chapter 4.

Changes in stature, muscle mass, and fatness that typically occur from childhood through adolescence are illustrated in Figure 2.13 for a longitudinally measured sample of French-Canadian children (Baughan et al. 1980). The variable "muscle mass" is an estimate of the amount of muscle at the mid-point of the arm. This estimate is derived from measurements of upper arm circumference and triceps

skinfold. These measurements are usually taken at the mid-point of the upper arm, half way between the olecranon process (the "elbow") of the ulna and the acromion process (the "shoulder") of the scapula (Lohman et al. 1991). The circumference measures the amount of skin, subcutaneous fat, muscle, and bone in a cross-section of the arm. The triceps skinfold estimates the contribution of skin and subcutaneous fat to arm circumference. If it is assumed that the arm is cylindrical in shape, simple geometry may be used to calculate the lean arm circumference, which is the circumference of the muscle and bone at the midpoint of the upper arm. Frisancho (2008) gives the following three formulae to make this calculation (readers with access to a skinfold caliper and tape measure may take the measurements and apply the formulae for themselves):

(1) Arm muscle diameter (mm) = (arm circumference in mm/π) − triceps skinfold in mm.
(2) Arm muscle area (mm^2) = (π/4) x (arm muscle diameter)^2.
(3) Arm fat area (mm^2) = ((arm circumference)^2/4π) − arm muscle area.

If it is also assumed that the circumference of the humerus is equal for all individuals, variation in lean arm circumference represents differences in the amount of muscle at this site. Though the arm is not cylindrical in shape and the circumference of the humerus is not equal in all individuals, the differences between reality and the assumptions of the technique are small enough so that when applied to groups of individuals the estimate of average muscle mass at the mid-arm are reliable and accurate.

In Figure 2.13 fatness is represented by the sum of three subcutaneous skinfolds – triceps, subscapular, and suprailiac. While skinfold measurements do provide a good estimate of the subcutaneous fat, their relationship to the deep body fat is questionable. Some early research found that the amount of fat in the subcutaneous and deep body reserves is positively correlated and that changes in both occur in a similar fashion. However, more recent studies using computed tomography to measure deep fat show a lack of correlation in the amount of fat held in the two reserves (Borkan et al. 1982; Davies et al. 1986). Nevertheless, since during the childhood, juvenile, and adolescent stages most fat is subcutaneous, a measurement of the amount of subcutaneous fat is a fair estimate of total fat (Brozek 1960).

It may be seen in Figure 2.13 that there is little difference in average stature between boys and girls until adolescence, after which boys are typically taller than girls. Girls usually begin their adolescent growth spurt about two years earlier than boys, which means that on average girls are taller than their male age-mates for a couple of years. Boys have greater average muscle mass at all ages, though the differences become absolutely greater, and biologically important, at adolescence. Conversely, girls tend to have more subcutaneous fat at all ages, and again, the difference in fatness increases during adolescence. On average, girls add FM continuously from age 8 to 18, with a slowing or possible loss of fat at the time of the adolescent growth spurt (about age 11–12 years as shown in Figure 2.13). Most boys experience an absolute loss of total FM during adolescence, and may have no more

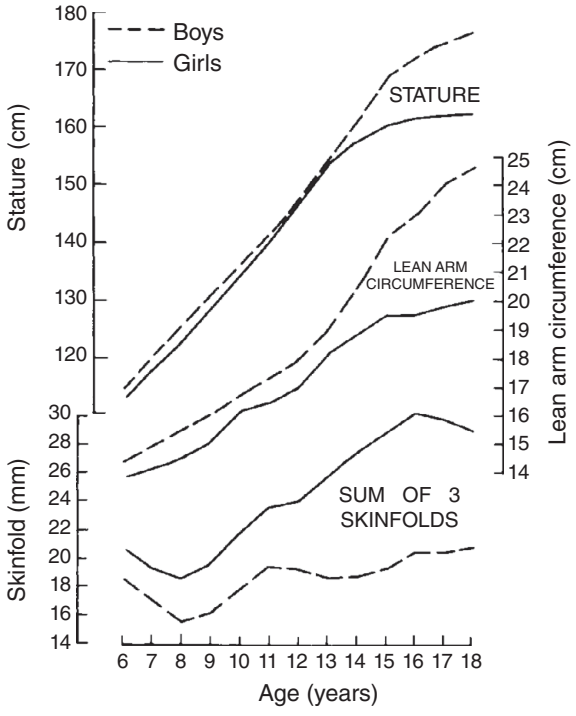


Figure 2.13 Mean stature, mean lean arm circumference, and median of the sum of three skinfolds for Montreal boys and girls (from Baughan et al. 1980). Note that sexual dimorphism increases markedly after puberty, about age 12–13 years.

fat at age 18 than they had at age 6 (Holliday 1986). The adolescent spurt in muscle mass in boys is usually accompanied by an increase in bone density, an increase in cardio-pulmonary function, larger blood volume, and greater density of red blood cells. Increases in each of these also occur in girls, but at levels both relatively and absolutely lower than for boys (Shock 1966).

As indicated in Figures 1.4 and 2.5, the shape of the adolescent growth spurt is not symmetrical. The rise to PHV is relatively slower than the fall after the peak. The size of the spurt is usually greater in boys than in girls, although there is much individual and population variation in this (see Chapter 5 for a discussion of population variation in growth and development). The size of the spurt and the age when peak velocity is reached are not related to final adult height. In fact, some normal, but slow maturing, individuals and people with certain endocrine disorders do not have a growth spurt but may reach normal adult height (Prader 1984). This fact makes the otherwise universal nature of the adolescent growth spurt an even more striking human characteristic. The evolutionary and biocultural significance of the human adolescent growth spurt are discussed in Chapters 3 and 4.

On average, adult men are taller and heavier than adult women. Alexander and colleagues (Alexander et al. 1979) surveyed 93 societies, including Western and

non-Western cultures, and found that the stature of women averages between 88 and 95% the stature of men. In England, women average 93% of the height of men, and this average difference is identical for men and women in the tallest (97th percentile), median (50th percentile), and shortest (3rd percentile) height groupings (Marshall & Tanner 1970). The most recent survey of worldwide adult heights from 169 countries, including national estimates from low income to high income nations, reports a median male height of 170.5 cm and median women's height of 158.8 cm (NCD Risk Factor Collaboration (NCD-RisC) 2016). The difference of 12.05 cm is typical of previous studies and amounts to women having 93% the height of men (Bogin et al. 2017). The biocultural significance of the sexual dimorphism in human adult stature, and in body composition, is discussed in later chapters.

One study in Switzerland (Largo et al. 1978) found that the difference between men and women in adult height is 12.6 cm. Since this study had followed the growth of the subjects longitudinally, from the age of four years, it was possible to calculate how much of the adult difference in height occurred in the various stages of postnatal growth. It was found that 4 factors accounted for the difference: The boys' greater amount of growth prior to adolescence added 1.6 cm, the boys' delay in the onset of adolescence added 6.4 cm, the boys' greater intensity of the spurt added 6.0 cm, and the girls' longer duration of growth following the spurt subtracted 1.4 cm from the final difference.

Due to the interplay of these factors, the regulation of size may be more precisely controlled and the "harmony of growth" evidenced during infancy and childhood is continued during adolescence. For instance, Boas (1930) reported that the age at which adolescent growth begins is inversely correlated with the size of the spurt, meaning that early-maturing children have higher peak height velocities than late-maturing children (Figure 1.7).

This observation has been confirmed for American children (Shuttleworth 1937, 1939), British children (Marshall & Tanner 1969, 1970), Swedish children (Lindgren 1978), and other nationalities. Another compensating mechanism, described for Swiss children by Largo et al. (1978), is that a child with slow growth prior to puberty will tend to have a longer-lasting growth spurt during adolescence than a child who achieves a greater prepubertal percentage of adult height. Where chronic undernutrition, disease, and child labor are endemic, such as in highland Peruvian Indian societies, East African pastoral societies, and Guatemala Maya villages (Bogin et al. 1992), height at every age is reduced compared with less stressed populations. However, the total span of the growth period is prolonged, up to age 25 or 26 years, so that a greater adult height may be achieved than if growth stopped at 18 to 21 years, as it does for many healthy individuals in the United States. Presumably, these growth adjusting mechanisms are present in all children.

In both sexes, the onset of the puberty is followed within a few months by the appearance of the secondary sexual characteristics. In boys these include changes in size of the penis and scrotum, the growth of pubic, axillary, and facial hair, the "breaking of the voice," and seminal emission. In girls the secondary sexual characteristics include the growth of the breasts, appearance of pubic and axillary hair,

menarche (first menstruation), and development of the uterus, vagina, and vulva to their mature size and appearance. One of the common methods used to assess the secondary sexual development of boys and girls is the Tanner Puberty Stage classification system (Tanner 1962). This system divides the development into five stages. For both boys and girls there are five stages of pubic hair development:

PH1 indicates no visible pubic hair, PH2 the first appearance of pubic hair, and PH3 to PH5 the progressive growth of pubic hair to the adult stage. For boys there are five stages for the development of the penis, testes, and scrotum, which rate their changes in size and coloration. This genital rating proceeds from G1, the prepubertal stage, to G5, the adult stage. For girls there is a breast development scale that begins at B1, no breast development, and proceeds to B2, initiation of the breast bud, to B5, the adult form of the breast (including areola and nipple).

Details of the development of the primary and secondary sexual characteristics, interrelationships between these events during adolescence, and sex differences in the timing of these events have been studied since the 1940s and are available for European-Americans, African-Americans, and British (Herman-Giddens et al. 1997, see references cited therein), for Chinese (Chang et al. 1966; Lee et al. 1963), for Swiss, Swedes, and Poles (Gasser et al. 2013 see references cited therein), for Turks (Onat & Ertem 1974; Semiz et al. 2008), and other populations. Differences exist between these populations in the timing of onset of the stages of adolescent maturation. For instance, in the older studies (pre-1980) breast development in European-American and British girls began at an average age of about 11 years, which is about one year later than for Turkish or Chinese girls. Turkish boys began pubic hair development at, on average, 11.8 years, compared with Swedish boys at 12.5 years, and Chinese boys at 13 years. Even so, the results from each of these samples were remarkably similar, despite the variation in ethnicity, geographic areas, and cultural practices of each population, and variation in the methods of measurement and analysis used by the authors. Indeed, the amount of variation in the age at which individual adolescents achieve any maturational stage is greater within the samples studied than between the samples. The average differences between samples were of little biological importance.

More recent studies of US samples indicate changes in the age of first appearance of secondary sexual characteristics that may have biological and social importance. Herman-Giddens and colleagues (Herman-Giddens et al. 1997, 2012) collected data for Tanner stage and testicular volume data on 4,131 boys seen for well-child care at 144 pediatric offices across the United States. Data were analyzed for prevalence and mean ages of onset of sexual maturity markers. They report that, "Mean ages for onset of Tanner 2 genital development for non-Hispanic white, African American, and Hispanic boys were 10.14, 9.14, and 10.04 years and for stage 2 pubic hair, 11.47, 10.25, and 11.43 years respectively. Mean years for achieving testicular volumes of $\geq$3 mL – an indicator that puberty has begun – were 9.95 for white, 9.71 for African American, and 9.63 for Hispanic boys; and for $\geq$4 mL were 11.46, 11.75, and 11.29 respectively. African American boys showed significantly earlier mean ages for stage 2–4 genital development and stage 2–4 pubic hair than white

and Hispanic boys. No statistical differences were observed between white and Hispanic boys." Thus, "US boys appear to be developing secondary sexual characteristics and achieving testicular enlargement 6 months to 2 years earlier than commonly used norms, with African American boys entering Tanner stages 2 to 4 earlier than white or Hispanic boys." (p. e1058).

Findings for girls were based on a sample of 17,077 girls, of whom, " . . . 9.6% were African-American and 90.4% white. At age 3, 3% of African-American girls and 1% of white girls showed breast and/or pubic hair development, with proportions increasing to 27.2% and 6.7%, respectively, at 7 years of age. At age 8, 48.3% of African-American girls and 14.7% of white girls had begun development. At every age for each characteristic, African-American girls were more advanced than white girls. The mean ages of onset of breast development for African-American and white girls were 8.87 years (SD, 1.93) and 9.96 years (SD, 1.82), respectively; and for pubic hair development, 8.78 years (SD, 2.00) and 10.51 years (SD, 1.67), respectively. Menses occurred at 12.16 years (SD, 1.21) in African-American girls and 12.88 years (SD, 1.20) of age in white girls" (p. 505).

What could be the reasons for the decrease in age at onset of secondary sexual characteristics? Is it possible that girls as young as 3 years old, just making the transition from infancy to childhood, are pubertal? The answer may be that the tight association between puberty and secondary sexual development seen in the pre-1980 studies has been disrupted in the more recent studies. Age at menarche, or menses as labeled by Herman-Giddens and colleagues, has declined in virtually all populations since 1980, with a range of median age from about 12.0–14.9 years (Figure 2.14).

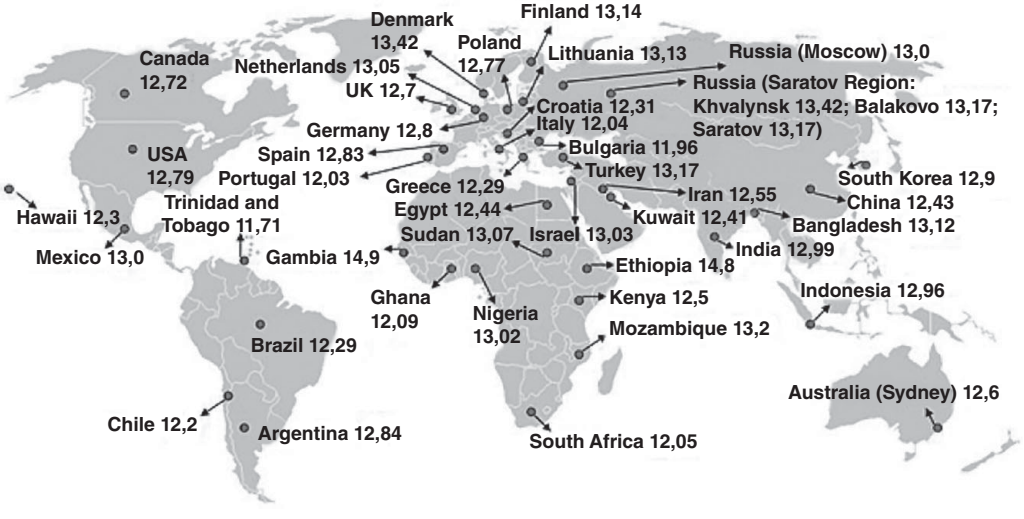


Figure 2.14 Median age at menarche for girls from 41 countries surveyed from 2003 to 2013. The data were compiled by Janina Tutkuviene and Simona Gervickaiteand. The figure is reprinted here with their kind permission. (Originally published in Hermanussen et al. 2014)

Average menarcheal age is a sensitive indicator for various characteristics of population including socioeconomic situation, nutritional status, style of life, and stress level. For a long time, the decline in the age at menarche was explained by improving social and economic conditions, better nutrition, and better health care. However recently, early menarche was linked to overweight and obesity and metabolic changes caused by excessive fatness. Increased social stress, from living in poverty or low-income, insecure environments with associated physical and emotional abuse is another possible cause. Globalization and urbanization are possible influences, as girls raised in urban environments often have earlier menarche than girls from rural environments. Environmental contamination by many industrial pollutants, especially endocrine-disrupting chemicals such as the banned but still ubiquitous polychlorinated biphenyl, as well as Bisphenol A (BPA) and dioxins in plastic bottles or plastic containers, may also lead to earlier age at menarche. To date, the effects of these possible influences and their complex interactions on pubertal timing are poorly understood.

Greater fatness in infancy, childhood, and the juvenile years, for example, is well-related to earlier menarche but not in a simple way. Increasing fatness tends to lower age at menarche, and first signs of puberty in boys, until the fatness is excessive, that is, until clinical obesity levels are reached. Then age at menarche, or boys' puberty, rises with additional body fat (Herman-Giddens 2013; Lee et al. 2016). The possible nutritional, metabolic, endocrine, social, and psychological reasons for this nonlinear relationship are not understood. Even less well understood are the biological and social consequences of changing age at puberty. Are earlier maturing boys and girls healthy? Are they at risk for sexual abuse because of a disconnect between their chronological age and biological maturation? Some research indicates that they are, and also at risk for heart disease, diabetes, some cancers, and other diseases in later life. Research is needed to understand both the causes and consequences of ever-earlier sexual maturation in human populations.

All the studies on pubertal and sexual development reviewed above report findings for, generally, healthy children, juveniles, and adolescents. There are also studies of the sexual development of young people with illnesses and interested readers may find these via search engines. Searching for the publications of Herman-Giddens and colleagues is one good place to start. One study of relevance to the present discussion is of South African adolescents who had been severely malnourished (requiring hospitalization) when they were between 5 months and 4 years 4 months old (Cameron et al. 1988, 1990). These former patients were followed up at 10 years and 15 years after their hospitalization and compared with a control sample of adolescents from similar low socioeconomic and poor nutritional circumstances who had not developed clinical signs of severe malnutrition. Amazingly, both the former patients and the controls show an identical sequence in the order of appearance and timing of the secondary sexual characteristics. It is known that chronic poor nutrition from infancy to juvenility can delay the onset of puberty. From the South African studies, it seems that one can conclude that severe malnutrition at an early age does not seem to disrupt the basic human pattern of secondary sexual development.

In Chapter 5 the population variation in sexual development, and the environmental causes of this variation, are explored in more detail.

The Common Control of Adolescent Development

As the sequence of appearance of secondary sexual characteristics is so highly predictable in people, some researchers have attempted to find a common control mechanism for adolescent development. Two such attempts were by Nicolson & Hanley (1953), who used factor analysis on their American sample, and by Bielicki et al. (1984), who used principal component analysis on their Polish sample. These statistical techniques are similar in that they divide the total variance in a set of data into discrete sources of variation, called "factors" or "components." In both studies the component accounting for the largest % of the total variance in maturation was a general maturity factor. Clustered on this general maturity factor were growth velocities for height and other linear dimensions, stages of sexual maturation (e.g., breast development in girls or pubic hair growth in boys), and skeletal maturation. For the data of Nicolson & Hanley, the general maturity factor accounted for an average of 71% of the variance in maturation in the boys, and an average 73% of the variance in girls. Bielicki *et al.* found that the general maturity factor accounted for 77% of the variance in boys and 68% in girls.

This statistical finding, along with the similarities in adolescent maturation found in different populations, supports the idea that adolescent maturation is controlled by some central organ or system within the body. These data also demonstrate that there is a human pattern of adolescent growth and development which is shared by all people. The search for the central control of maturation is a subject discussed in Chapter 6 (which examines genetic and endocrine regulation of growth). The reasons for the universal pattern of adolescent maturation are the subject of Chapters 3 and 4, which discuss the evolution and ecology of human growth.

Adult Stage

Adulthood is subdivided into the separate stages of *Prime* (also referred to as maximum performance age), *Gradual decline*, *Transition* or degeneration age, and *Senescence* or old age. Description of these stages is given in Table 2.1. Much has been written about these stages, and about aging in general, by physical anthropologists in the past 100 years, including Franz Boas (1940). In recent reviews of research on "Aging," Joy Pearson and Douglas Crews called attention to the work of Raymond D. Pearl (1879–1940), Albert Damon (1918–1973), Stanley Garn (1922–2007), Gary A. Borkan, Cynthia Beall, Charles A. Weitz, and Phyllis B. Eveleth among others (Crews 2003b; Pearson & Crews 1997).

The attainment of adult stature is one of the hallmarks used to mark the transition from adolescence to adulthood. In the United States, young women and men of middle to upper socioeconomic status reach adult height at about 18 years of age and 21 years of age respectively (Roche & Davila 1972). These ages may be close to the

lower limit for onset of adulthood. In other populations growth continues to later ages. Hulanicka & Kotlarz (1983) studied a sample of 221 young men from Wroclaw, Poland, an industrial city of 600, 000 people, and found that only 54% of the subjects reached final adult height by age 19 years. The other 46% added an average of 2.13 cm in height between the ages of 19 and 27 years. It is well known that individuals suffering from undernutrition, chronic diseases, and certain drug therapies may continue to grow in height until reaching about 26 years of age. Though these individuals may grow for a longer period of time, they usually achieve less total growth and end up shorter than their more privileged or healthier age mates.

Height growth stops when the long bones of the skeleton (the femur, tibia, etc.) lose their ability to increase in length. Usually this occurs when the **epiphysis**, the growing end of the bone, fuses with the **diaphysis**, the shaft of the bone. As shown in Figure 2.4, the process of epiphyseal union can be observed from radiographs of the skeleton. In their study of Polish men, Hulanicka & Kotlarz (1983) found that the amount of growth that occurred after age 19 was a function of skeletal maturation, late maturers grew to a later age than average or early maturers. This fact has been known for many years, and an estimate of skeletal maturation, often called skeletal age, is incorporated into equations used to predict the adult height of children (Bayley & Pinneau 1952; Roche et al. 1975a; Tanner et al. 1983).

The fusion of epiphysis and diaphysis is stimulated by the gonadal hormones, the androgens, and estrogens. However, it is not the fusion of epiphysis and diaphysis that stops growth, for children without gonads, or whose gonads are not functional, never have epiphysial fusion, but they do stop growing (Tanner 1990). Rather, it is a change in the sensitivity to growth stimuli of cartilage and bone tissue in the **growth plate region** (Figure 2.15) that causes these cells to lose their hyperplastic growth potential.

Reproductive maturity is another hallmark of adulthood. The production of viable spermatozoa in boys, and viable oocytes in girls, is achieved during adolescence, but these events mark only the early stages, not the completion, of reproductive maturation. For girls, menarche is usually followed by a period of one to three years of adolescent sub-fecundity. That is, there are menstrual cycles, which are often irregular in length, ovulation is also irregular, and there is a low probability of pregnancy. A girl with menarche at age 12 years may not be fertile until 14 or 15 years of age. Fertility, of course, does not indicate reproductive maturation. Becoming pregnant is only a part of reproduction: Maintaining the pregnancy to term and raising offspring to adulthood are equally important aspects of the total reproductive process. Girls in the earlier phases of adolescence who become pregnant have a high percentage of spontaneous abortions and complications of pregnancy and their neonates suffer increased risk of low birth weight and preterm birth. This is true for girls in developed nations such as the United States and other wealthier nations (Black et al. 2012) and lower income nations such as Peru (Frisancho et al. 1985). There are many reasons for the reproductive difficulties faced by teenage girls, ranging from physiological immaturity of the reproductive system to socioeconomic and psychological trauma induced by the pregnancy. Cross-cultural surveys indicate that teenage mothers with

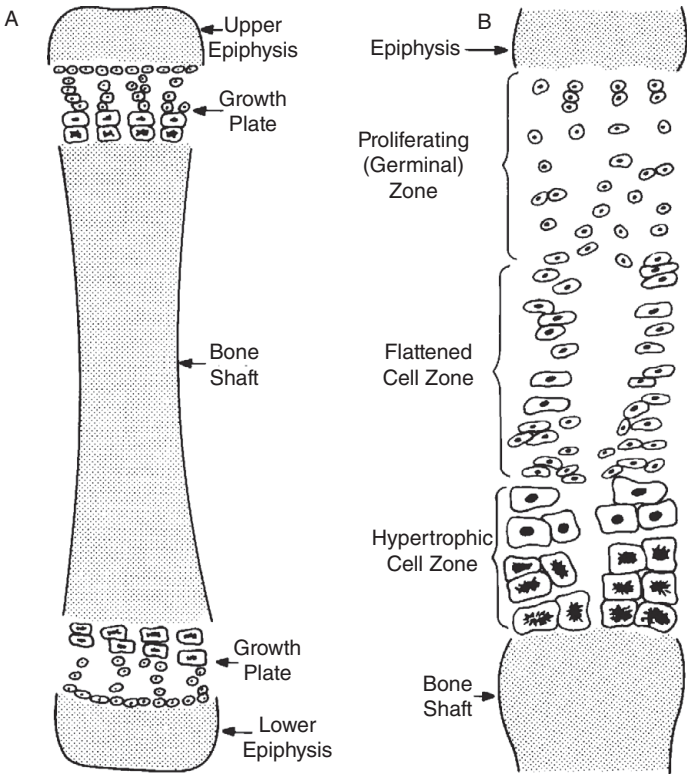


Figure 2.15 (A) Diagram of a limb bone with its upper and lower epiphyses. (B) Diagrammatic enlargement of the growth plate region: New cells are formed in the proliferation zone and pass to the hypertrophic zone to add to the bone cells accumulating on top of the bone shaft. (Redrawn from Tanner 1990)

reliable social support from older women, usually close kin of the mother, may overcome many of these risks (Kramer & Lancaster 2012). Pregnancy is, as for all human experience, a biocultural matter. Even with kinship support, the fact that the mother in the earlier phases of adolescence is still growing means that the nutritional needs and hormonal activity of her body may compete with and interfere with the growth and development of the fetus. This problem was suggested by Pagliani over 100 years ago and confirmed by Frisancho et al. (1985). For all these reasons, most researchers agree that female reproductive maturity is reached at the end of the adolescent stage of life, which occurs, on average, at 18 years. It is no coincidence that the average age at first successful birth averages 19 years in traditional, preindustrial human societies (Bogin 2001).

Analysis of urine samples from boys aged 11–16 years old show that they begin producing sperm at median ages that cluster between 13.4 and 14.5 years (Hirsch et al. 1985; Nielsen et al. 1986; Richardson & Short 1978). This event is called **spermarche** and often occurs before the appearance of any secondary sexual

characteristic. Whether this event marks the onset of fertility is not known. The quality of viable sperm from teenage boys is also unknown, though there are sporadic cases of verified fatherhood for boys younger than 15 years old. Even if fertile, the average 14-year-old boy is less than half-way through his adolescent growth spurt and, therefore, his developmental status is incomplete. In terms of physical appearance, physiological status, and psychosocial development – he is still more of a juvenile than an adult. The cross-cultural evidence shows that few boys successfully father children until they are into their third decade of life and where reliable data exist less than 4% of live-born infants were fathered by men under 20 years of age (Bogin 2001). A notorious exception occurred in 1997 when a 13-year-old schoolboy in Washington State, USA fathered a child with his teacher, a married woman, who had already given birth to four children. The teacher was imprisoned for second-degree rape of a child, the "child" being her student. After her release the pair resumed their relationship, violating a court order to not see each other, produced another pregnancy, the teacher was reimprisoned and gave birth there, she was released in 2004, the young man was then 18 years old and petitioned the court to remove the restraining order, then the pair married in 2005, and in 2017 separated from each other.[8]

The transition to adulthood is marked by dramatic events, such as the cessation of height growth and full reproductive maturity. In contrast the course of growth and development during the prime reproductive years of adulthood are relatively uneventful. Most tissues of the body lose the ability to grow by hyperplasia (cell division), but many may grow by hypertrophy (enlargement of existing cells). Exercise training can increase the size of skeletal muscles and caloric over-sufficiency will certainly increase the size of adipose tissue. However, the most striking feature of the prime adult stage of life is its stability, or homeostasis, and its resistance to pathological influences, such as infectious disease and psychological stress. By about age 35 years, most men and women enter a phase of gradual decline. Both sexes are still capable of important and demanding physical and cognitive performance, but decrements in outputs are measurable. Most women and men can compensate for the degeneration by new biobehavioral strategies, such as doing work in shorter segments and enlisting assistance from others. By about age 50 years the declines in performance are more noticeable.

Transition and Senescence

Following the prime reproductive years of adulthood and the period of gradual decline, the process of aging becomes more noticeable. Aging, or senescence, is the cumulative loss of the ability to adapt to environmental stress. The pattern of decline varies greatly between individuals. Though specific molecular, cellular, and organismic changes can

[8] https://en.wikipedia.org/wiki/Mary_Kay_Letourneau

be measured and described, not all occur in all people and rarely do they follow a well-established sequence. Menopause, the end of ovulations, may be the only event of the later adult years that is experienced universally by women who live past 50 years of age; men have no similar event. The biology, evolution, and possible value of menopause are described in Chapter 4.

There are many hypotheses about the aging process and about why we must age at all. One explanation for why we must age is called the "pleiotropy hypothesis." In now classic works on the biology of senescence Peter Medawar (1915–1987) and George C. Williams (1926–2010) argued (Medawar 1952; Williams 1957) that aging is due to age-specific pleiotropic genes that is, genes which have good effects early in life, but have bad effects later in life. Kirkwood (1977), Charlesworth (1980), and others refined this hypothesis further in terms of a general theory of aging. Some aging theorist proposed that senescence and death are programed in the sense that aging and death are shaped by natural selection. Except in a very few species, such as yeast, there is very little empirical evidence for programed death (Kirkwood & Melov 2011). There is evidence for the limited mitotic (cell duplication) ability of hyperplastic cells. Hayflick reported in 1980 that when raised in tissue cultures, human embryo hyperplastic cell lines double in number by mitotic division only 50 (+/−10) times and then die. Tissue cultures of cells from adult humans have an even more limited mitotic potential, doubling only 14–29 times before dying (Hayflick 2007). This doubling limit is not due to genes, but rather, "... the general loss of molecular fidelity ..." (p. 1) of hyperplastic cells. This loss of copying fidelity provides a theoretical limit to life. In practice, few cells and few people ever reach this limit, because many cells die as part of normal physiology, not senescence (Crews 2003b). These cell deaths appear to be influenced by factors operating at all biological levels, from the DNA sequence, through epigenetic effects, cell metabolism, to behavior and environmental exposures of the whole organism (Crews & Bogin 2010). Another reason for the ubiquity of death in the biological world is likely the inability of one or more cell types, including nerve, muscle, and other cells with limited mitotic ability in adulthood, to use nutrients and repair damage.

Other candidates for "agents of aging" are: (1) free radicals, chemical by-products of metabolic activity, that accumulate with time and can damage DNA, proteins, and cell membrane and (2) the amassed burden of DNA mutations caused by ionizing radiation or chemical pollutants. Undoubtedly, aging is a multi-causal process, but there may not be a biological plan for the aging process. Death is inevitable, but nature may not have had the time or the selection pressures to mold our manner of death into a predictable pattern. Perhaps for humans this is due to the fact that only recently, in the evolutionary history of our species, have an appreciable number of individuals come to live past the prime and gradual decline adult years. Throughout prehistory, death by predation, disease, and trauma caused by violence and accidents was probably more common than death due to old age. Even if this is so, there is evidence that for 99% of our evolutionary history as *Homo sapiens*, our foraging (i.e., gathering and hunting) ancestors were able to live past 60–70 years when conditions permitted (Caspari & Lee 2004).

In contrast to the process of aging and death, growth and development from conception to adulthood follow a predictable pattern. It was during the evolutionary history of our species, and those species ancestral to ours, that selective pressures operated to shape our pattern of growth. To understand why we grow the way we do, we must examine some of the events that occurred during human evolution. Chapter 3 describes the evolution of the human pattern of growth.

3 The Evolution of Human Growth

Biologists and anthropologists have proposed several taxonomic schemes for classifying Homo sapiens. Lovejoy (1981) suggested five characteristics of humans as defining features: bipedality, a large **neocortex**, reduced anterior dentition with molar dominance, material culture, and unusual sexual and reproductive behavior. The development of each of these characteristics can be seen in the ontogenetic unfolding of the human pattern of growth and development. For instance, bipedality is made possible by differential growth of the legs and pelvis, including the bone, muscle, tendons, and ligaments, vs. the arms and shoulder girdle. Our unusual reproductive behavior results, in part, from our species-specific neuroendocrine physiology that adds prolonged childhood and adolescent stages of growth and development. These additional human life course stages delay sexual maturation, and also establish the foundation for the capacity for human culture and **biocultural** reproduction, defined as the set of marriage and kinship based rules for extramaternal cooperation in the production, feeding, and care of offspring (Bogin et al. 2014b, 2018b).

Though these characteristics set us apart from all other species, they have their origins in evolutionary history. In this sense, we share many basic growth patterns with other species, but we differ in other ways due to the contingencies of the evolutionary history of the hominins, the group of primates to which we, our ancestors, and a few other extinct species belong. To better understand both the shared and special features of human growth, this chapter explores the phylogeny of growth of lower vertebrates, mammals in general, and primates – the group of mammals that includes monkeys, apes, and humans.

Vertebrate and Mammalian Foundations for Human Growth

The growth and development of any organism may be viewed as movement through space or time (Thompson 1917, 1942). Growth and development move through time in two ways, first in the conventional sense of the passage of days, months, and years, and secondly in the sense of biological change and maturation from earlier to later stages of life. These temporal movements can be represented in mathematical form, for instance as the distance and velocity curves of growth illustrated in Figures 2.5 and 2.6, or as the curves for maturation of body systems illustrated in Figure 2.8.

Most nonhuman organisms share the same basic curve of growth. It is an S-shaped, or sigmoid, curve (Figure 3.1, A). The growth of chickens, rats, and cattle

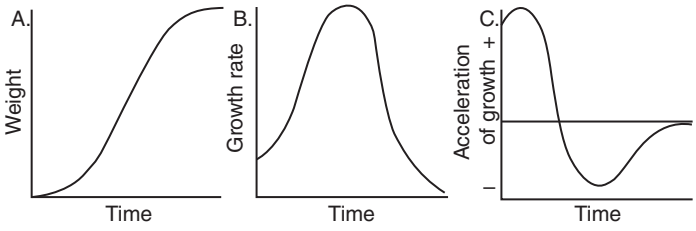


Figure 3.1 General growth curves: (A) weight vs. time, (B) rate of growth (velocity) vs. time, and (C) acceleration of growth rate vs. time. (After Medawar 1945)

follow this curve (Bertalanffy 1960; Brody 1945; Laird 1967). The growth of parts of organisms and colonies of cells, such as yeast, bacteria, and tumors in animals, also conform to this sigmoid curve (Bogin 2001, p. 2, Figure 1.1).

Figure 3.1 B and C illustrate some of the other mathematical features of the general growth curve. In B the velocity, or rate of growth, is given; only a single peak, or maximum rate of growth occurs, meaning that there is an initial acceleration and then a period of deceleration in growth rate. In C the changes in acceleration are more clearly revealed; the point of zero acceleration corresponds to the inflection point in the velocity curve where the rate of growth stops increasing and begins to slow. Growth rate at any subsequent point on the curve is decreasing with time.

In mathematical terms, parts B and C of this figure are related to A as its first and second derivatives, respectively. That the curve of general growth is completely differentiable means, mathematically, that the growth process represented by this curve is smooth and continuous. Continuity means that we can predict changes in amounts and rates of growth during the course of development with precision. Such predictions allow us to make quantitative and qualitative assessments of the growth of any individual organism and to make comparisons between different individuals, groups of individuals, and even different species of animals in terms of the mathematical properties of their growth curves.

An example of this type of smooth and predictable growth pattern is given in Figure 3.2 for the chicken. Only the physical constraints of the egg, around the time of hatching, interfere with a smooth growth trajectory. The rigid shell and the depletion of nutrients from the yolk sac of the egg slow growth before hatching. After hatching the growth rate "rebounds," but only to the point where an averaging of the prenatal and postnatal growth rates would yield a smoothly decelerating curve. A similar pattern of growth occurs for humans just before and after birth (see Figure 2.6). During the last part of the third trimester of human pregnancy the fetus is large enough to press against the inner surface of the uterus and the placenta, which probably constricts blood vessels and inhibits the fetal–maternal exchange of nutrients, gases, and wastes. Fetal growth slows, but rebounds several days following parturition so that the child "catches-up" to the size he or she would have achieved if there had been no prenatal decrease in growth rate.

Brody (1945) and Bertalanffy (1960) showed that the growth of mice and Brahma cattle may be modeled with the same curve used for the chick. Thus, the pattern of

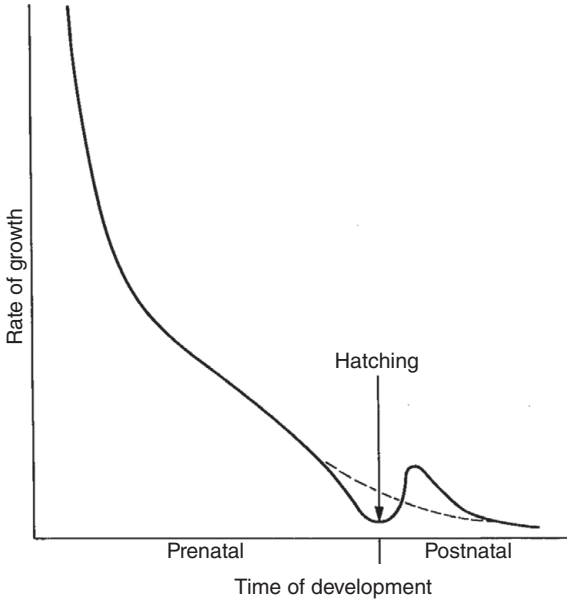


Figure 3.2 Rate of growth of the chick before and after hatching. The interrupted line is the theoretical curve if no growth restriction prior to hatching takes place. (After Timiras 1972)

growth of these animals of very different absolute size, distinct evolutionary history, and ecologically diverse life styles is qualitatively identical. Some important exceptions to the general pattern of animal growth are found when plotting the growth of highly social mammals, including humans. The human pattern was illustrated, and its basic aspects discussed in the Chapter 2 (Figure 2.5). The distance curves in Figure 2.5 are, at first glance, not markedly different from the general sigmoid curve of Figure 3.1 A. However, the human velocity curves illustrated in Figure 2.5 are different from the velocity curves for other animals (Figure 3.1 B). Humans have a series of both rapid and gentle decreases in growth rate after birth punctuated by both small and large increases in growth rate (the mid-growth spurt and adolescent growth spurt). Even a casual inspection of the human and nonhuman curves presented so far finds both qualitative and quantitative differences exist in the pattern of growth.

Unlike the nonhuman curve of growth, the human growth curve cannot be modeled with a single mathematical function: That is, the human curve does not have continuity because it is not completely differentiable over its length. The distance curve requires at least two functions, one for the pre-pubertal segment and one for the post-pubertal, or adolescent, segment (Shohoji & Sasaki 1987) and mathematical models with six or seven functions describe the distance curve even more precisely (Bock et al. 1973; Jolicoeur et al. 1988). The velocity curve requires at least three mathematical functions for adequate description; one for the infancy stage (birth to about three years), a second for the childhood and juvenile stages (from about three years to the beginning of the adolescent spurt), and a third for the

adolescent stage (Bock & Thissen 1980; Bogin 1980; Karlberg 1989; Laird 1967). Further discussion of growth models is given in Chapter 5, especially Box 5.1. Suffice it to state here that the mathematical functions just mentioned do describe the shape of the distance and velocity curves, but the biological meaning of the mathematical expressions, if there is any such meaning, is unclear.

Mammalian Growth

The mathematical shapes of the human distance and velocity growth curves are unusual for mammals, but they have their origins in the patterns of growth followed by other species of social mammals, especially the primates. The growth of mammals differs from that of other vertebrates for two basic reasons. One reason relates to mammalian locomotion and the other relates to mammalian reproduction. Animals, both invertebrates and vertebrates, evolved the capabilities for rapid and flexible movement. This requires muscle tissue and something for it to work against. Most vertebrates (such as fish, amphibians, reptiles, birds, and mammals) utilize bone for this purpose, a tissue that provides support and protection due to its rigidity, but also the developmental flexibility that allows for growth (Goss 1978). Most fish and amphibians make poor use of bone compared to reptiles, mammals, and birds (the bones of birds are specially adapted for flight and are not discussed here in detail). The fish bone grows by **periosteal** deposition, the addition of tissue on all the external surfaces of the bone. As they grow, the bones of fish not only elongate but also widen becoming heavier over time. The buoyancy of water compensates for the additional weight. The skeleton of higher vertebrates does not grow this way. Goss states that "amphibians evolved marrow cavities . . . [and] also acquired cartilaginous epiphyses . . . " (Goss 1978, p. 65). The epiphysis is the growing end of a long bone, and by maintaining the epiphysis in a cartilaginous form growth can continue. If the epiphysis develops into hardened bone tissue, growth ceases. The marrow cavity allows for the transport of nutrients to both ends of a growing bone. With these new structures amphibians were able to grow skeletons that were longer without also growing wider.

The reptiles also took the next step in bone evolution and developed the cartilaginous growth plate, which allows for several improvements in skeletal efficiency. The growth plate of a typical mammalian long bone, including the diaphysis (bone shaft), epiphysis, and growth plate, was shown in Figure 2.15. The growth plate separates the growing part of the bone from the rigid part. A photomicrograph of the growth plate region of a rat is given in Figure 3.3. The figure is labeled to indicate four regions described by Horton and Machado (1992), beginning with the reserve zone (also called the germinal zone or the resting zone), which is farthest from the surface of the growing bone. In this zone are the reserve chondrocytes, which are cells that form the cartilage precursor of mature bone cells. When these reserve cells begin to form bone they first migrate into the proliferative zone where they flatten and undergo mitosis to form clusters of cells. In the hypertrophic zone the cells in these clusters increase in size, especially in vertical diameter. These chondrocyte cells also

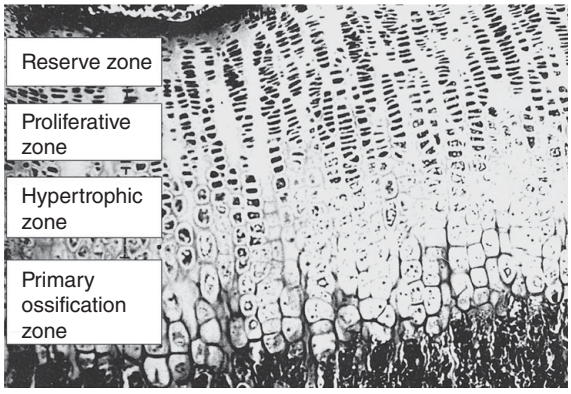


Figure 3.3 Photomicrograph of the growth plate region of the rat. The tissue sample comes from an animal at about 30 days post-conception. The sample was Azan stained and photographed at 6.3 × 20 magnification by Dr. A. A. Missankov, Department of Anatomical Sciences, University of the Witswatersrand, South Africa. Dr. Missankov kindly supplied the photomicrograph. See the text for explanation of the four zones. The reserve zone is also referred to as the germinal zone or the resting zone in the literature.

begin to mineralize, but they do not become true bone cells. That happens when the enlarged clusters finally reach the zone of primary ossification and then die via a process called apoptosis (programed cell death). The dead chondrocytes leave a cartilage core that is invaded by blood vessels and then by bone matrix from osteocytes (bone-making cells). Via this process the original cartilage core is completely replaced by bone matrix (Baron et al. 2015; Mackie et al. 2011).

The growth plate is located behind the bony articular surface, which allows for efficient skeletal operation while growth is taking place. The amphibians must make do with an entirely cartilaginous epiphysis, which is not as hard or durable as bone and limits the range of work and the size of the organism that the skeleton can accommodate. The new structure of reptiles has obvious use in the prodigious size attained by dinosaurs, as well as giant Galapagos tortoises and other large-bodied reptiles alive today.

The evolution of the growth plate overcame many of these problems and allowed reptilian and mammalian bones to perform their functions more efficiently throughout life. The growth plate system also allows for both relatively rapid growth in early life and then the cessation of growth. In fish, amphibians, and some reptiles bone growth never completely stops, although growth continues very slowly after sexual maturity is reached. This pattern of growth is unsuitable for mammals, who must limit growth, for the most part, to the pre-reproductive stages of life. Mammalian reproduction, especially for females, demands so much energy and body resources that it precludes the possibility for significant body growth of the mother and, at the same time, the fetus. One of the dangers of human teenage pregnancy is that the mother's own growth may divert resources from the fetus, resulting in fetal growth retardation (as mentioned before).

Mammals and birds make up for a finite period of growth by being able to grow very rapidly. The maximum rate of bone growth, measured in birds, is 3.0 mm/day per growth plate, or 6 mm/day for bones with growth plates at both ends (Kember 1992). Human infants can achieve growth rates of slightly more than 1.0 mm/day in crown–heel length (Heinrichs et al. 1995). By limiting growth to an early stage of life an organism can develop a strategy of investing first in its own development, and then, after ending growth, become sexually mature and begin to invest in the development of its offspring.

An end to growth is also necessary for terrestrial animals that must support their own body weight without the help of water or any other buoyant medium. The largest terrestrial mammals, the Proboscideans (elephants and the allied extinct mammoths and mastodons), may have reached the limits of size for land mammals. The limbs of these animals are used almost entirely for support of the body and locomotion. The evolution of the nose into a flexible muscular appendage, the trunk, serves the function of a limb for food gathering and environmental manipulation, as well as breathing and vocalization (Shoshani 1998; West 2001). Some smaller mammals, such as rats and other rodents, never fuse their growth plates with the diaphysis and never stop growing. These small, ever-growing rodents increase in size so slowly and die so soon after sexual maturity that they never attain the sizes suggested by certain second-rate horror films, though one rodent, *Hydrochoerus hydrochaeris*, the capybara of South America, may attain 1.3 meters in length and 50 kg in weight. Even so, because of their relatively small size and short lifespan they are capable of flexible and rapid movement throughout their lives.

To maintain efficiency of function, a pattern of limited growth and **remodeling of bone** evolved to meet mammalian needs for movement and feeding. The physiological characteristics of mammals, including **homoiothermy** (self-regulation of a relatively constant body temperature), efficient placentation by which the fetus continuously benefits from maternal blood circulating in the uterus, rapid bodily movement and other features associated with a relatively high metabolic rate, require a diet rich in energy and other nutrients. In the long term, mammalian metabolism requires a constant and high-quality dietary intake rather than episodes of abundant food, like that of a snake gorging itself at a single meal. Mammals must be able to move rapidly and efficiently to find and capture quality foods on a regular basis. This requires an efficient musculo-skeletal system during both the early and the later phases of growth. Bone remodeling helps to maintain such efficiency. As a bone grows in length its surfaces must be remodeled so that its characteristic shape and function can be retained. In a long bone this remodeling is achieved by removing old ossified bone tissue from the periosteal (outer) surface and adding new bone tissue to the **endosteal** (inner) surface (Enlow 1963, 1976). Enlow called this process the "principle of the V" and it is schematically illustrated in Figure 3.4. Via bone remodeling, the evolution of the growth plate, the cessation of growth of the skeleton, and other evolutionary alterations in the function of limb bones (Romer 1966), mammals were able to achieve the efficient, rapid, and flexible mobility that they require.

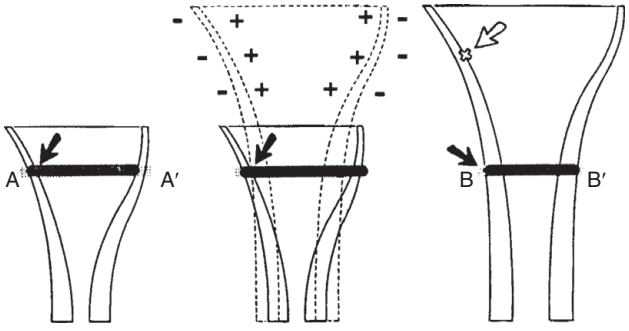


Figure 3.4 Diagrammatic representation of remodeling in a limb bone. As the bone grows in length, the level indicated by the line AA' becomes repositioned into the level indicated by the line BB'. The relative level of AA' in the larger bone is indicated by an X. The structural remodeling of the bone occurs by the process of resorption (− signs) and deposition (+ signs). One result of the subtraction and addition of bone is that the point indicated by the black arrow at level AA' has been relocated from the inner side of the cortex of the bone to the outer side of the cortex in BB'. As the bone continues to grow in length, and new bone tissue replaces the older bone tissue, the point will eventually be lost by continued resorption. Enlow called this process the "principle of the V" and one can see that the growing end of the bone maintains a characteristic "V" shape as it increases in length. (After Enlow 1963)

Mammalian Reproduction

Reproduction is the second aspect of mammalian biology that influences growth. Mammalian reproduction is based upon a high degree of investment in offspring, both before and after they are born. That investment takes the form of energy and time that mammalian parents, especially mothers, provide to their offspring. Mammals were not the first class of vertebrates to evolve parental care and investment in their offspring, indeed such care is found in all classes (fish, amphibians, reptiles, birds), but mammals have carried it to a level of physiology and behavior exceeding that of other vertebrates. The high quality of mammalian **parental investment** may be measured by the efficient internal fertilization and placentation of most living mammals, lactation by the mother during her offspring's infancy, and by the capacity of each individual offspring to help ensure its own survival to reproductive age. Each of these mammalian features has a direct relationship to growth.

Placentas

Mossman (1937, reprinted 1991) described the placenta as " . . . any intimate apposition or fusion of fetal organs to the maternal tissues for physiologic exchange." Though the contact between tissues may be "intimate" there is never any direct connection between mother and fetus and the exchange of substances always occurs through a tissue boundary. The evolution of the placenta removed some of the limitations to prenatal growth, including both growth in size and length of gestation.

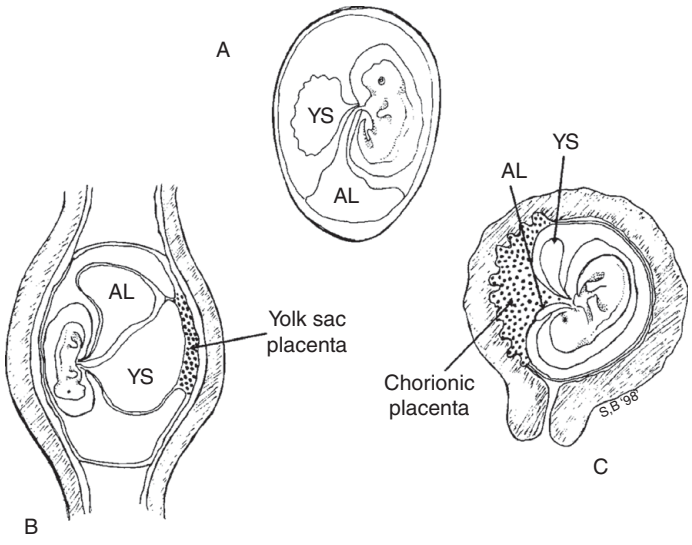


Figure 3.5 Schematic diagrams of the reptilian egg (A), the **yolk sac placenta** of the rabbit (B), and the **chorionic placenta** of haplorhine primates (C). See text for details. (After Hamilton & Mossman 1972; Zihlman 1982)

The prenatal growth and gestation of nonplacental animals such as most reptiles, birds, and monotremes (the platypus and echidna) is limited by the need to "package" fetal nutrients in the **yolk sac** (labeled YS in Figure 3.5) and fetal waste products in a separate compartment called the **allantosis** (AL). In contrast, at least some marsupial mammals (e.g., opossums and kangaroos – see further discussion of marsupial placentas in the next section on Lactation) and in the eutherian mammals (e.g., dogs, horses, primates) the placenta provides for fetal nutrition, respiration, and the removal of metabolic wastes for either the last stage of gestation (marsupials) or continuously throughout gestation (eutherians).

Internal gestation and live birth, or **viviparity**, evolved in all classes of living vertebrates, except the birds. Even placenta-like structures evolved independently in many nonmammalian species, such as snakes. Mammals, however, developed the most efficient types of placentas. Kurt Benirschke (1924–2018) created a useful, well-illustrated website of mammalian placentas (Benirschke 2012). There are several schemes in the literature for the classification of placentas (Bowen 2011). One of these, by Amoroso (1961), seems best related to mammalian phylogeny (evolutionary sequence) and described three basic kinds of placentas for mammals (the first and third are depicted in Figure 3.5). The first is the yolk sac placenta, found in some marsupials and rabbits, in which blood vessels connect the yolk sac with the uterine wall. Nutrients from the mother's circulation may be transferred to the yolk sac to replenish the fetal nutrient supplies. Wastes are still confined to the allantosis and cannot be removed via the maternal circulation. The second type is the **chorionic–allantoic placenta**, characteristic of higher mammals, in which parts of the surface of the allantosis fuse with the chorion (a membrane surrounding the fetus composed of

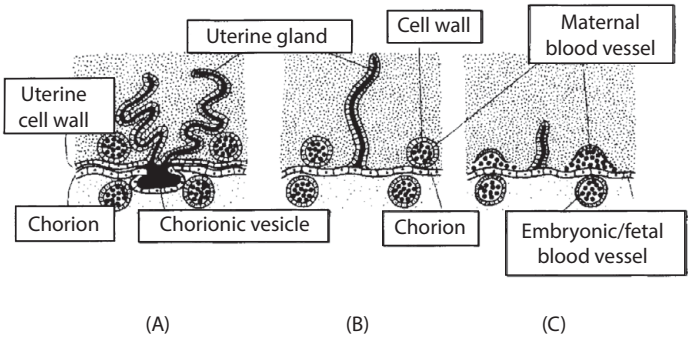


Figure 3.6 Schematic drawings of the three sub-types of chorionic placentae: (A) epitheliochorial, (B) endotheliochorial, and (C) hemochorial. Embryonic tissues are in light stippling and maternal tissues are in heavy stippling. (After Martin 1990)

maternal and embryonic tissues). This apposition allows for a more efficient exchange of substances between mother and fetus than in the yolk sac placenta, because greater surface areas of fetal and maternal tissues are in contact. In the egg-laying birds, reptiles, and monotremes, and in the mammals with yolk sac placentas, the allantosis serves only as a receptacle for embryonic and fetal wastes. The conversion of this waste-sac function to a system for the exchange for nutrients, gases, and wastes in some placental mammals is an example of the conservative nature of evolution. Existing organs are often "retooled" for new functions rather than having organisms develop totally new organs.

The third type Amoroso described is the chorionic placenta, in which there is a more direct connection between the chorion and the fetus via the umbilical cord. There is also, generally, a greater amount of surface area for the exchange of substances between fetus and mother in this type of placenta. The chorionic placenta is the type found in what may be called the "true" placental mammals, including horses, swine, ruminants, monkeys, apes, humans, and rodents. The chorionic placenta presents fewer boundaries between the maternal and fetal blood circulation, but there may still be up to six separate layers of tissue. The chorionic placenta type may be classified into three sub-types based on the number of tissue layers, which are illustrated in Figure 3.6. At the website Placental Structure and Classification,[1] Bowen states that, "Just prior to formation of the chorionic placenta, there are a total of six layers of tissue separating maternal and fetal blood. There are three layers of fetal extraembryonic membranes . . . [and] three layers on the maternal side . . . " The three fetal-side layers are retained in the mature chorionic placenta of all mammals, but the number of layers retained on the maternal side varies greatly among species (Figure 3.7). The number of maternal tissue layers separating maternal blood from fetal tissues describes the **invasiveness** of the placenta – more invasive placentas have fewer layers.

[1] www.vivo.colostate.edu/hbooks/pathphys/reprod/placenta/structure.html

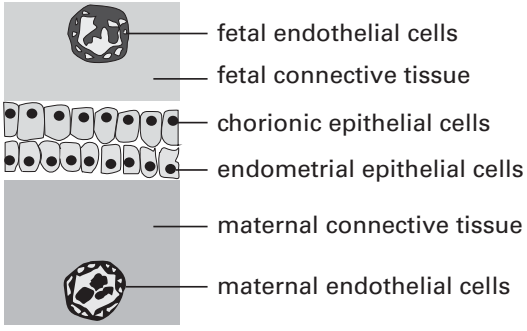


Figure 3.7 Cartoon illustration of the six embryonic tissue layers of the chorionic placenta. (After Bowen 2011)

A brief discussion of these sub-types is important because human beings possess the most invasive and intimate type of placenta of any mammal. The nature of the human placenta helps us to better understand how the mother's environment and her behavior present both benefits and risks to her embryo and fetus. Benefits include the efficient transport of needed nutrients and oxygen to the fetus and the efficient removal of wastes from the fetus. Behavioral risks of the mother include the use of tobacco and alcohol consumption – these behaviors may interfere with the operation of the placenta and may also introduce harmful substances to the embryo and fetus.

The first sub-type, called the **epitheliochorial placenta**, maintains the separation between the chorion and the epithelial lining of the uterus. Maternal blood vessels are separated from fetal blood vessels by four barriers – the two cell walls of the blood vessels themselves and the cell walls of the chorion and uterus. The development of a profuse network of uterine glands and chorionic vesicles increases the area of contact and exchange across epitheliochorial placenta and partially overcomes the barrier to nutrient exchange imposed by the four cell walls. The epitheliochorial sub-type is found in lemurs and lorises (the strepsirrhine primates). The second sub-type is called the **endotheliochorial placenta**, and it eliminates the uterine epithelium barrier. This seems to allow for more direct exchange between the maternal and fetal blood vessels, without the need for the network of chorionic vesicles. The endotheliochorial sub-type is found in rodents. The third sub-type is called the **hemochorial placenta**. This is the most invasive kind of placenta because the cell wall of " . . . the maternal blood vessels has itself broken down so that the chorion is directly bathed by maternal blood . . . " (Martin 1990, p. 446). The hemochorial sub-type is found in tarsiers, New World monkeys, Old World monkeys, apes, and humans (the haplorrhine primates). In human beings, the fetal capillaries fuse with the chorion and protrude through to the maternal side of the placenta where these capillaries sit in pools of maternal blood. The maternal and fetal circulatory systems are never joined directly, but they share a single wall of tissue between them. Such close contact over a relatively large surface area – gross external dimensions of the human placenta average 16–222 cm in diameter and 2–4 cm in thickness with a total surface area of contact of between 10 and 11 square meters (Yetter 1998) – allows for a

greater ease of diffusion and active transport of substances across the placenta of most other mammals.

The critical advance in the biology of placental mammals is that the fetus can develop and grow to an advanced stage protected and well-nourished in the uterus. In general, the length of gestation and the growth in size of the whole fetus (that is, the total body weight) of placental mammals are constrained primarily by the mechanical limitations of the mother's uterus and the size of the birth canal. However, in proportion to the weight of the mother, the primates with hemochorial placentae (monkeys, apes, humans) give birth to heavier newborns than the lorises and lemurs with their epitheliochorial placentae. The "higher" primates also develop offspring with proportionally larger brains than the prosimian primates (Martin 1990). Placental type is not the only reason for these differences in growth of the fetal body and brain, but it is a significant correlate.

The degree of **interdigitation** is another aspect of placental efficiency. Interdigitation refers to the way in which maternal and fetal tissues are spatially arranged with one another. Capellini et al. (2011) described several types of interdigitation. They report that mammals with greater placental invasiveness and interdigitation give birth to neonates with greater body and brain size, but that species with highly interdigitated placentas do so in about half the gestation time of species with less interdigitation. Human placentas are both highly invasive and interdigitated and this helps to explain the relatively high amount of parental investment provided by mothers to their fetus. That investment results in the large body, with a high amount of body fat, and large brain size of human neonates, relative to mother's body size after only 280 days of gestation. Even under fairly severe starvation of the mother the fetus is, generally, secure and its birth weight is unaffected (Keys et al. 1950). More discussion of human parental investment in relation to growth and development is presented in Chapter 4.

Lactation

All mammals, even the egg-laying monotremes, nurse their young. Lactation continues to supply the high-quality nutrients to the newborn that the fetus received via the placenta. Recent research with tammar wallabies (*Macropus eugenii*) suggests that the placenta-lactation divide is less clear than previously believed. The wallabies are marsupials (along with Australasian kangaroos, wombats, koalas, Tasmanian devils, and other species and the American opossums) and were conventionally classified as nonplacental. But some researchers think that this distinction is incorrect, noting that marsupials develop placenta-like structures during the end of pregnancy. These placental structures are simple in form relative to the eutherian mammal placenta. The wallaby placenta is just two cell layers thick, but it provides oxygen, nutrients, and biochemical signals that are needed for proper development of the fetus. The wallaby placenta also protects the fetus from the mother's immune system, which might otherwise attack the fetus as a foreign substance. This function is also served by the eutherian placenta. Tammar wallabies are pregnant for just

26.5 days, about 4–5 days longer than rats. But, joey wallabies (newborns) nurse and seek protection of the pouch for almost one year, during which they grow from about 393 ± 13 mg at birth to about 3 kg – a 10,000-fold increase! Guernsey and colleagues (Guernsey et al. 2017) analyzed tammar wallaby placental tissue in the final days before the fetus was born and found that the tissue expressed the same genomic regions as do the eutherian placentas of mouse and human in the early stages of fetal development. The researchers then analyzed genomic expression in the mammary glands of tammar wallabies that were nursing joeys in the pouch. The researchers found that the mammary tissue expressed the same genes as do eutherian placentas in late fetal development. It appears that the mammary glands and milk of the wallaby are very similar in function to the eutherian mammal placenta. This suggests an evolutionary conservation of the genomics for maternal feeding and investment across the types of living mammals.

Lessons Learned about Lactation in All Its Forms

The high-quality nutrition provided by lactation allows for rapid and sustained growth and development after birth. In addition, lactation provides several more benefits to the newborn. Sellen (2007, p. 125) described four of these basic functions of lactation present in all extant mammal species:

1. "Transfer protective functions of fully developed immune system across generations . . . " The biochemical composition of mother's milk includes a variety of immunoglobins and hundreds of other immune system and bioactive constituents.
2. "Optimize litter size to allow titration of maternal investment across sib sets . . . " Rather than producing many eggs at each reproductive cycle, of which a large percentage may not survive, lactation following efficient placentation allows for an "assembly line" mode of offspring production with waves of pregnancies followed by infant feeding. This helps to increase survival at all stages of growth and development. Women in traditional human societies (e.g., foragers, horticulturalists, pastoralists) will often nurse their current infant until the age of 20–36 months at which time the woman is again pregnant (Bogin 2001).
3. "Facilitate efficient reproduction in unpredictable environments lacking special foods for young . . . " Mothers do no need to forage for foods or prepare "baby foods."
4. "Increase behavioral flexibility and opportunities for learning." Infants are in close contact with their mothers and can learn by observation, imitation, and purposeful teaching.

The evolution of lactation required behavioral changes in both mother and offspring, particularly in mother–infant bonding, which maintains the infant in contact and communication with the mother so that it can be suckled when hungry. The mother–offspring contact ensuing from this feeding method establishes a period of dependency in the young and a reciprocal period of parental investment by the

mother. This time of life for the newborn is called infancy (as defined in the Chapter 2), and it has become a stage in the life cycle and growth curve of all mammals. A similar period of dependency occurs for birds, and in some species includes feeding with crop milk, a nutrient and immune protective secretion of the esophagus, but is of shorter duration and often of less physical intimacy than that for most mammals. Flamingos provide crop milk for up to nine months. A few insect females, including spiders, cockroaches, and beetles, provide a nutrient and immune protective milky food to their offspring while gestating within the mother's body. One insect species, the tsetse fly (*Glossina spp.*), gives birth to a live larva that is full of milk to be used during its 30-day life history transition from newborn larva, to pupa, to adult (Benoit et al. 2015).

Lactation and infancy prolong the period of dependency but allow for rapid and high-quality growth and greater physical and behavioral adaptability (Pond 1977). The nutrient and chemical composition of the milk provided varies by species.[2] Human milk, for example, is 4.5% fat, 1.1% protein, and 6.8% lactose (sugar). This compares with Guernsey cow milk at 5.0% fat, 3.8% protein, and 4.9% lactose. The high protein content of the cow's milk supports the newborn calf's pattern of growth acceleration but would be lethal to the human infant with a deceleration of growth rate. Compared with other animals, infant mammals may be better able to adjust total rates of growth, or the rates for specific body parts, to adapt to environmental stress. For example, in cold environments, growth rates may be adjusted to produce adult mammals with relatively larger bodies (Bergmann's Rule), shorter extremities (Allen's Rule), or both, compared with mammals living in warmer climates (but see Box 5.3 for a critique of these Rules). Large bodies with short extremities conserve heat better than smaller bodies with relatively long extremities; the former body type has relatively less surface area for heat dissipation. The body size and body proportions of polar bears (*Thalarctos maritimus*) and Malayan sun bears (*Helarctos malayanus*) conform to these growth adaptations. Infancy may also increase behavioral adaptability by allowing young mammals the time to practice and improve innate behaviors, such as the stalking of prey in carnivores. The mother–infant bond increases the opportunity for young mammals to acquire learned behaviors by observing and imitating their mothers or other adult animals with whom the mother socially interacts.

Caroline Pond provided an original and highly useful discussion of the evolution of lactation (Pond 1977). She pointed out that viviparity and lactation evolved independently in many species of vertebrates. Only in the marsupial and placental mammals do these both occur regularly as a package. Together, viviparity and lactation protect the mother's investment in ova, embryos, and newborns. Viviparity allows the mother to take the embryo and fetus with her during the whole gestation, rather than leaving an egg unprotected at a nest. Lactation supplies high-quality nutrients to the infant, even if this requires cannibalizing the mother's body reserves

[2] See www.quora.com/What-is-the-difference-between-human-milk-and-cow-milk for a list of mammalian species.

of fat, protein, vitamins, and minerals. Nonlactating species must often confine the birth of their young to places and times of the year when appropriate foods are available, but lactation provides appropriate food at all times and places. The high-quality nutrition provided via lactation allows for rapid postnatal growth, especially of the skull, jaws, and teeth during infancy. By the end of infancy these developments mean that most mammalian species can eat the adult-type diet. Therefore, stated Pond, sexual maturation and reproduction may follow quickly after the end of lactation/infancy, and indeed they do for most species of mammals.

Pond observed that the investments in offspring and patterns of growth and maturation associated with the viviparity–lactation package result in high levels of reproductive success for the mammals.

Brains and Learning

There is more, however, to successful reproduction than what one's mother provides via viviparity and lactation. After weaning (defined here as the cessation of lactation, not the process that leads to cessation) the young mammal must find food and shelter, avoid predators, find a mate, and care for its own offspring until this new generation reaches reproductive age. The way mammals accomplish this is through the growth of relatively large brains and the flexibility in behavior, that is, the capacity for learning, that these large brains allow. The evolutionary record shows that the mammalian brain has undergone repeated selection for increases in size and complexity. Jerison (Jerison 1973, 1991) compared **endocasts** (molds of the interior of the skull which may be used to estimate brain size) of fossil skulls of mammals and found that the brains they contained were, in proportion to body size, smaller in earlier times and have increased in size steadily over the last 60 million years. In contrast, Jerison found that reptiles have not undergone this selection; the brain size to body size ratio of reptiles has not changed appreciably during the last 200 million years.

Mammals have also evolved more complex and functionally diversified brain structures. The mammalian neocortex and its neurologically distinct regions (the motor-sensory region, the auditory and visual regions, etc.) are examples. Jerison (1976) showed that mammalian brains have a system of neurological pathways that bring together, at various locations, information from the visual, auditory, and olfactory senses. The "integrative neocortical system," as Jerison called it, joins neurological regions of the paleocortex (including the olfactory bulb and the limbic system) and the neocortex (including the visual, auditory, and somatic systems) of the brain (Figure 3.8). More recent research shows that there may be several integrative systems in the human brain which allow for more detailed memory and task performance than in other mammals (Müller & Knight 2006). More ancient vertebrates, such as reptiles, rely mostly on the paleocortex for the control of behaviors which Jerison characterized as " . . . fixed-action patterns . . . with few requirements for plasticity or flexibility" (Jerison 1976, p. 101). More recently evolved vertebrates, the birds and mammals, rely on both the paleocortex and the neocortex for the

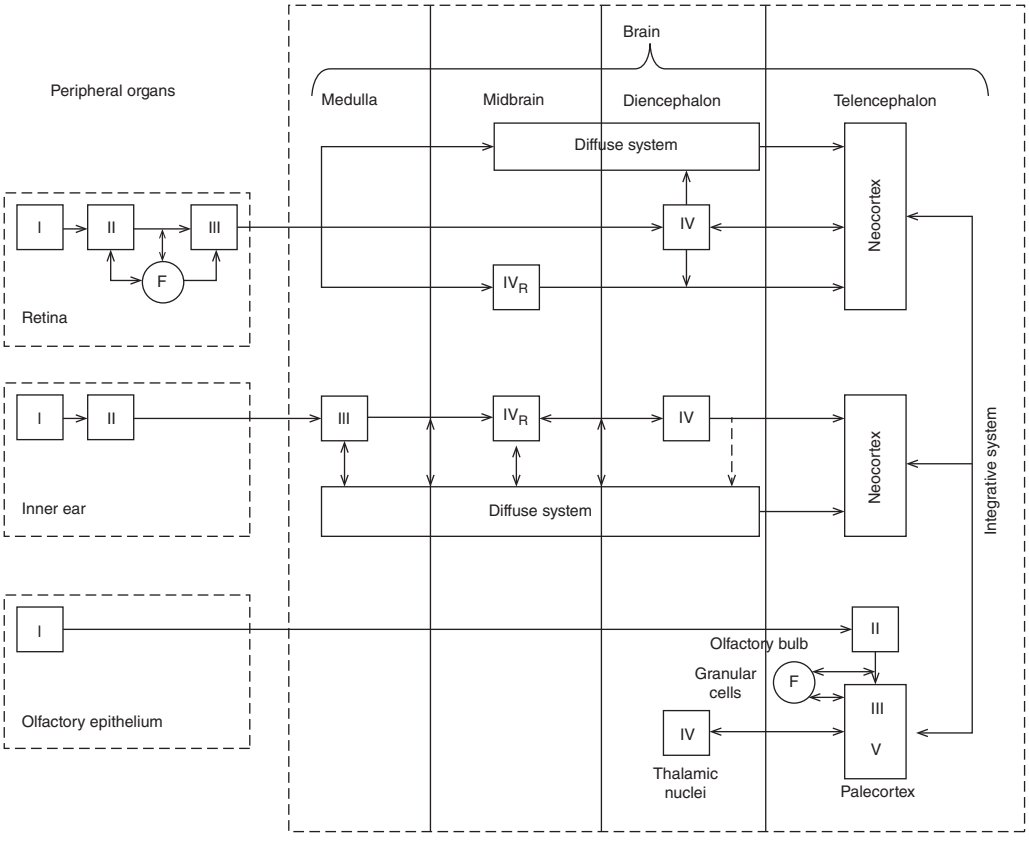


Figure 3.8 Schematic view of the neurological connections between the visual, auditory, and olfactory systems of living mammals. The arrows show the general direction of the flow of information through successive orders of nerve cells (I to V indicates parts of the reflex control systems; F indicates feedback loops). The integrative system (right) is a mammalian characteristic. (From Jerison 1976)

control of behaviors which are plastic and flexible in all species. Birds do not have the integrative neocortical system and, according to Jerison, their behavior displays to perfection the fixed-action pattern of response to stimuli, but he did not address the fact that birds can learn quite complex and lengthy behavior routines.

Mammals have the integrative system, allowing " ... sensory information from various modalities [to be combined] as information about objects in time and space" (Jerison 1976, p. 101). More than other Classes of vertebrates, mammals do not just react to environmental stimuli, they perceive, store, retrieve, and evaluate information and adjust behavior responses according to the present situation and past experience. More neurological tissue is required to accomplish these sensory, brain, and behavioral tasks and mammals do have brains that are larger, in proportion to body size, than the brains of reptiles and most birds. Larger, more complex brains

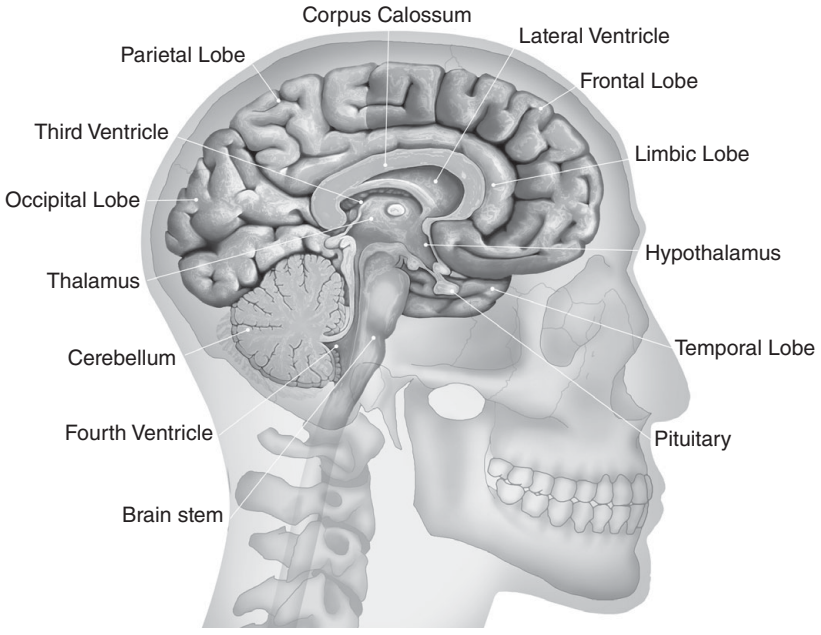


Figure 3.9 Functional areas of the brain. Credit MARK GARLICK / SCIENCE PHOTO LIBRARY / Getty Images. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

allow for a greater capacity for learning and more flexible behavior, because learned behavior may be constantly modified by further learning. In proportion to total body mass, the human brain is one of the largest, with more specialized regions, and is the most complexly integrated of all mammalian brains (Figure 3.9)

The evolution of learning as an adaptive strategy is associated, in a classic feedback manner, with the series of changes in mammalian biology and behavior that have just been described. The tissues of the central nervous system have relatively high metabolic activity, requiring a regular supply of nutrients and oxygen for maintenance and growth. The evolution of the placenta is thus directly related to the evolution of larger, more complex brains and greater learning abilities. The placenta is the organ that constantly supplies oxygen and nutrients to the developing fetal brain and allows that brain to develop to an advanced stage before birth, and the evolution of the placenta and of the brain are clearly correlated in mammals. Yolk sac placentas are found in the lower mammals, chorionic-allantoic placentas are found in higher mammals and chorionic placentas are found in the primates. In turn, each higher mammalian group has a brain that is relatively larger than expected for its body size (Jerison, 1973). It is no coincidence that humans have one of the largest brain to body weight ratios, perhaps the most complex and active brain of any mammal, and the most efficient placental system (the hemochorial variety of chorionic placenta) of any primate.

Other biological changes related to brain evolution occur during postnatal life. One example is a correlation of lactational behavior of the mother in relation to the brain

size and learning capacity of the infant. Robert Martin (1968b, 1968a) described such a case for the tree shrew (*Tupaia belangeri*). Relative to all mammals, tree shrews have a moderate brain to body weight ratio and average learning ability. A female tree shrew may cache her infants (two or three are born per litter) in a nest and leave them for up to 48 hours while she searches for food. The infant tree shrews are virtually silent and unmoving during their mother's absence, which may be a behavioral adaptation to avoid attracting predators. The seclusion and immobility also limit the variety of sensory stimuli that the infants experience. Upon the mother's return the infants are nursed with a milk that is concentrated in calories and other nutrients. This pattern of periodic feeding coupled with sensory deprivation during infancy works well for a species with limited brain growth after birth and a limited learning potential. This feeding style would not work for a species with rapid postnatal brain growth, requiring a constant nutrient supply during infancy, and a greater dependence on learning in later life.

In neurologically more advanced mammals, especially primates, mother and infant remain in virtually constant physical contact for several weeks or months after birth. Most primate females usually give birth to one infant per pregnancy, but South American marmosets and tamarins regularly give birth to twins. Singleton births facilitate intimate physical contact since there is no competition between siblings for the mother. Suckling is done "on demand," 24 hours per day. The concentration of nutrients in the milk of primates is lower than that of the "primitive" mammals, but the efficiency, constancy, and quality of nutrient supply is superior (Widdowson 1976). The newborn primate is highly active compared with the tree shrew infant. The primate infant travels with its mother, clinging to her body, sensing many of the things that the mother experiences and developing motor and sensory skills in the process. This type of early sensory stimulation is known to be conducive toward further learning (Jolly 1985). The infant primate grows more slowly than most other mammalian newborns and is, therefore, dependent for a longer time on this intimate relationship with its mother. Infant dependency extends the period of growth, development, and protection and increases the opportunity for the infant to learn survival skills by observing successful maternal behaviors.

Infancy, dependency, and learning are advantageous to both mother and infant since they lead to a greater probability that the young will survive to reach reproductive age. The drawback of infant dependency is that it is incompatible with some adult behaviors, particularly further reproductive behavior. Competition for nesting space, breeding territories, aggressive encounters with conspecifics for mates, and mating itself are often precluded behaviors for mothers with dependent young, but the limitations to new offspring production by females are partially offset by the higher quality of mammalian reproduction. That is, some nonmammalian species, such as many kinds of insects, fish, and reptiles, rely on prodigious egg production to assure the survival of some of their offspring to reproductive age. In contrast, mammals maximize the probability of survival of each individual offspring to achieve a high degree of reproductive success.

Stages of Mammalian Growth

All placental mammals share the basic skeletal and reproductive adaptations related to rapid and flexible locomotion and to efficient feeding of both the fetus, via some type of placenta, and the infant, via lactation. All placental mammals also share the neurological systems, especially the brain, that allow for intense parental investment and high levels of learning by the offspring. In contrast to these universals of mammalian biology and behavior, different species of mammals can be grouped by patterns of postnatal growth. The velocity growth of the mouse (Figure 3.10) and the Holstein cow (Figure 3.11) show the pattern that is typical for most mammalian species.

The shape of these velocity curves is fundamentally different from the human growth curve (Figures 2.5B, 2.6B) in several ways. First, humans achieve their maximum rate of growth in length and weight during gestation, and after birth growth rates decelerate during infancy. Humans can achieve such rapid rates of growth during gestation by having only one fetus per pregnancy (the most common case) and the highly efficient hemochorial placenta, which provide an optimal environment for rapid growth. In contrast, the mouse and cow reach their maximum rate of growth after birth during the infancy stage. For the mouse this is probably due to the competition between the multiple fetuses for placental resources (litters are the norm) delivered by their less efficient endotheliochorial placenta. For both mouse and cow, the improved nutrition provided by lactation, over that provided via the placenta, may also promote a surge in growth rate, but that surge is short-lived as growth rates begin to decline very soon after weaning.

The second difference is that for the mouse and cow sexual maturation occurs relatively soon after weaning and just after the maximal rate of growth. Within a few days of weaning, female mice have vaginal opening and can become pregnant. Male mice produce sperm about two weeks after weaning. Human growth and maturation

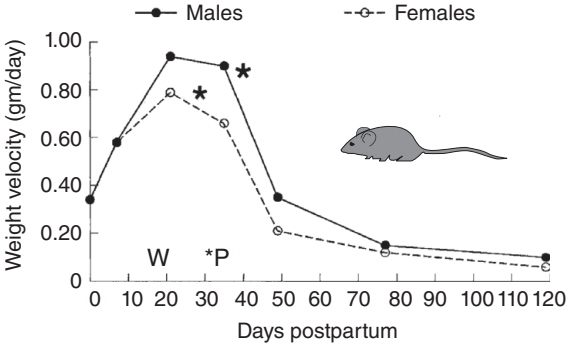


Figure 3.10 Velocity curves for weight growth in the mouse. Weaning (W) takes place between days 15 and 20. In both sexes puberty (P), meaning vaginal opening for females or spermatocytes in testes of males, occurs just after weaning and maximal growth rate. (Redrawn from data reported in Tanner 1962)

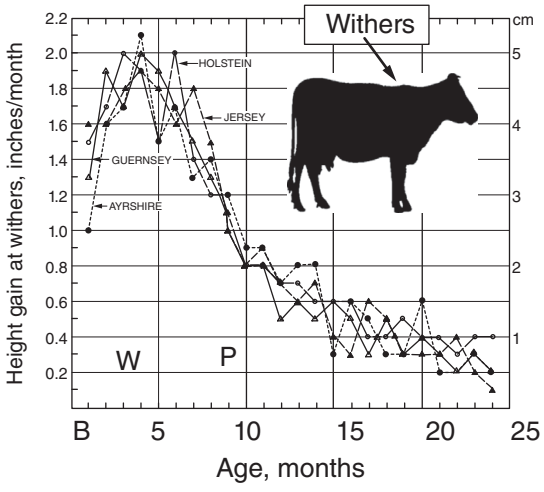


Figure 3.11 Monthly gains in height at the withers for the several varieties of cows. The data are from Brody (1945), who reports monthly values for height as the mean value for many animals (sample size varies from 67 to 239 at each age) measured monthly from birth to 24 months. Note: On commercial dairy farms weaning (W) takes place at age 3–4 months when the calf is separated from its mother so that the dairy farmer can sell the mother's milk. Puberty (P) for female calves takes place at about 9 months. If there were no forced separation, the calf would continue to nurse from its mother until puberty.

stand in sharp contrast to the mouse and cow in that humans delay puberty and sexual maturation for many more years after the end of infancy (weaning). The delay is even longer if measured from the time of maximal rate of growth after conception. The mouse and cow achieve peak velocity after birth, but the human peak velocity is before birth, meaning that humans experience an average delay of more than a decade between peak velocity during gestation and puberty.

A third difference is that for the mouse and cow, puberty occurs while growth rates are in decline but still near the maximal rate. Human puberty occurs when the postnatal rate of growth in height and weight are at their lowest points since birth. The fourth difference is that human puberty, or gonadarche (defined in Chapter 2), initiates a series of changes in endocrine function that lead to the adolescent growth spurt. In contrast, mouse and cow growth rates continue to decline after puberty. The fifth, and final, difference is that soon after puberty, mice are fertile and begin to reproduce, but humans have a delay of years between puberty and the onset of the adult reproductive stage of life.

Many of these differences between human and nonhuman mammalian growth were recognized by other researchers. In a classic analysis of mammalian growth, Samuel Brody (1945) demonstrated that most mammals have a pattern of growth similar to the mouse. Brody's analysis was confirmed by Bertalanffy (1960) and Tanner (1962). Tanner presented many growth curves for mice, rats, rabbits, sheep,

and cattle showing that the size of the mammal makes little difference in the pattern of its growth. The growth curve of Holstein cows, animals with a mean adult weight of about 600 kg and shoulder height of 145 cm (Figure 3.11), is virtually identical to the mouse with a weight of 18–35 grams and body length of 9–10 cm (Figure 3.10). Brody, Bertalanffy, and Tanner all agree that the majority of mammals progress from infancy to adulthood seamlessly, without any intervening stages of development. Human beings, in contrast, follow a very different path from conception to maturity. Brody states this most clearly by writing that his analysis,

demonstrates the close similarity between the age curves of different animal species. The human age curve, however, differs from the others in having a very long ***juvenile*** *period, a long interval between weaning and puberty (approximately 3 to 13 years); this period is almost absent in laboratory and farm animals. In these animals, weaning merges into adolescence without the intervention of the juvenile phase found in man* (Brody p. 495, emphasis added).

Brody's terminology and age for "puberty" are out of date by today's standards, for example, he makes no distinction between puberty and adolescence, and he equates both with sexual maturation. We know today that puberty occurs typically at about age 9 years (not 13 years) and is an event of just days or weeks duration. In humans, puberty occurs at the end of the juvenile period and before the start of the adolescent growth spurt and the adolescent life stage. Nevertheless, Brody correctly identified the fact that of all the animals he studied, only humans and the chimpanzee have a juvenile growth period. Despite this very important discovery, Brody still tried to show that, in essence, all animals, parts of animals, colonies of cells, and even populations of animals followed a common pattern of growth. Brody's own words, again, present this idea most concisely,

The general similarity between the curves of growth of individuals and of populations is not surprising, since ultimately both are collections of individuals. Our bodies are made of cells, and our bodies, in turn, are cells in a social body. Individuals are organisms and also units of a larger organism, an epiorganism (Brody p. 495).

Brody was searching for a type of "grand unification theory" of growth. His specific hypothesis was that all growing organisms and populations or organisms could be modeled with one curve of growth. Brody tried to prove his ideas by aligning the maximum rate of growth after birth for many species, including mouse, cow, and human, and then showing that they all have two phases of growth velocity. The first is a self-accelerating phase, from birth to peak velocity, and the second is a self-inhibiting phase, from peak velocity to adult size. Humans, even with their juvenile growth period and adolescent growth spurt, would have to fit into this unification of growth curves. Accordingly, Brody equated the adolescent growth spurt of humans with the velocity curve of growth for animals such as the mouse, the rat, and many domesticated farm animals (cow, pig, sheep). Brody believed that all mammals followed the same curve of growth, but that some mammals had their growth spurt soon after birth (e.g., the mouse and the Holstein cow) while others delayed the spurt until later in life (e.g., humans).

The Human Difference

In many ways, Brody's methodology and speculative ideas were ingenious and stimulating. His analyses and findings do apply to the laboratory and farm animals in his database, but they do not apply to humans. The main problem with Brody's unification hypothesis is that some animals, especially humans, have more than one phase of self-acceleration and self-inhibition of growth rate. Humans, in fact, have three such phases; the first occurs during gestation, the second is the mid-growth spurt, and the third is the adolescent growth spurt. Brody only knew about the last of these, the adolescent growth spurt, and incorrectly equated it with the solitary growth spurt of his laboratory and farm animals.

Brody's work is mentioned here because it had a strong influence on the study of human growth for many years. Even today, it is common to read about "adolescent growth spurts" or an "adolescent phase" in animals such as rodents or farm animals which have nothing like human adolescence.

While these ideas seem preposterous today, bear in mind that the nature of the human postnatal growth curve, especially the existence of the adolescent growth spurt, was still being debated during the first third of the twentieth century (see Chapter 1), the time when Brody carried out most of his original research. The human postnatal stages of childhood and juvenile growth, as well as the human prenatal curve of growth (Figure 2.6), were completely unknown to most researchers. Moreover, the neuroendocrine control of growth was poorly understood, and the control of puberty was completely unknown.

Juvenile Mammals

Brody's discovery of the human juvenile growth period is one of his lasting contributions to the field. Since Brody's time, juvenile growth stages have been discovered for several other mammalian species. The highly social mammals, such as social carnivores (wolves, lions, hyenas), elephants, many cetaceans (porpoises, whales), and most primates all evolved a new stage of development between infancy and adulthood – the juvenile stage (Bogin 1996). Many species of mammals, and fish, reptiles, and birds, live in groups, but these may be defined as "selfish herds" a term used by William D. Hamilton (1936–2000). Hamilton (1971) proposed that groupings form when individuals of a species attempt to reduce their predation risk by putting other conspecifics between themselves and predators. The individuals of these groupings do not socially interact with each other beyond their competition to move toward the center of the group. In contrast, highly social mammal species interact both competitively and cooperatively, individuals recognize each other and respond to each other based on memories of past interactions. Highly social mammals need each other for their own hormonal and nervous system regulation and will become ill and die if isolated from social contact.

Juveniles were defined in the previous chapter as offspring who are no longer dependent on maternal lactation, but who are still prepubertal. Juveniles are largely

responsible for their own care and feeding. Even though they are members of a highly social group, juveniles must often find their own food, avoid predators on their own, and compete with adults for food and space. Juveniles may even compete with their own mothers, who may be encumbered with another pregnancy or nursing infant. Clearly, the addition of the juvenile stage adds several new risks along the path of growth and development toward reproductive maturity. In fact, the highest rates of post-neonatal mortality (i.e., deaths after the first month following birth) for social mammals occur during the juvenile stage (Pereira & Fairbanks 2002).

Juvenility must have added some benefits to the life of social mammals to have evolved, but there is some debate as to the function of the juvenile growth stage. A stimulating review and analysis of the evolution of the juvenile growth stage was offered by Janson & van Schaik (2002). They propose two benefits. The first is the traditional "learning hypothesis" explanation, that is, the juvenile period allows for the extended period of brain growth and learning necessary for reproductive success in various species of social mammals. Social carnivores, elephants, and primates must all learn how to live within the social hierarchy of the group. They must also learn complex feeding skills such as hunting animal prey, opening fruits or seeds with protective coverings, and where and when to find food. Reproductive skills must also be learned, including competition for mates and care of offspring. In terms of natural selection the benefit of these types of learning is that it permits adaptation to ecological changes that are not predictable. Included in these changes are the common problems of seasonal variability in food availability due to climate, plant growth, and animal migrations.

Human learning is no exception to this and the crisis of the 1943 drought in Central Australia is a prime example. Birdsell (1979) conducted many years of ethnographic research with the native aboriginal peoples of Central Australia. Birdsell was told that during the time of the 1943 drought an old Aborigine man, named Paralji, led a band of people on a 600 km trek in search of water. After passing 25 dry waterholes he led them to a fallback well that the old man had not visited for more than 50 years. That well was also dry, forcing Paralji to trek 350 km on ancient trails, locating water holes by place names learned from initiation rites and ceremonial songs he memorized as a juvenile.

Even the way humans forage for food requires extensive learning during the childhood and juvenile growth periods. Much of the food humans utilize is hidden from view or is encased in protective coatings; tools are usually needed to extract and process these foods. The costs of tool manufacture, the time and energy needed to find and process raw materials, are outweighed by the benefits. Tool-using human gatherers extract twice as many calories from savanna-woodland environments as nontool using primates. Other foods are poisonous before processing by washing, leaching, drying, or cooking. For example, acorns and horse chestnuts, eaten by many North American Indians, and manioc, a staple food of many tropical living cultures, are toxic if eaten raw, and must be leached by boiling in water and dried before consumption. Furthermore, knowledge of the location of these foods and their methods of processing, and the location of raw materials and their manufacture into

tools requires learning. An example is the Arunta, a hunting and gathering people of Central Australia. They are compelled to live in self-sufficient nuclear families by the widely dispersed nature of food resources in their habitat (Service 1978). As soon as they are able, the children follow their mothers and fathers on the daily rounds of food collection and preparation. Boys and girls as young as five years were taken by their fathers on hunting trips and shown how to collect raw materials and prepare them for spears, points, and other tools. Since it takes more than a decade to become proficient in the manufacture and use of these tools, early learning and slow growth and maturation during the juvenile stage are mutually beneficial. Similar ethnographic observations were reported for African and South American foraging societies (Gurven et al. 2006; Schuppli et al. 2012).

Learning seems like a great idea for the evolution of the juvenile stage, but the learning hypothesis does not take into consideration the high juvenile mortality. After all, evolution works by differential mortality and reproductive success, and mortality during the pre-reproductive juvenile stage will not lead to evolutionary success. The second benefit of juvenility, and a complement to the learning hypothesis, is the "ecological risk aversion hypothesis." Janson & van Schaik developed this hypothesis to deal with the risks for juvenile mortality.

The reasoning for this hypothesis is as follows. When the risk of predation is high for the individuals of a species, natural selection often favors the formation of social groups – there is relative safety in numbers. The formation of social groups comes at a cost however, and that cost is an increase in feeding competition within the group – there are more mouths to feed. According to Janson & van Schaik, newly weaned individuals are most likely to be adversely affected by this competition as they have less-developed foraging skills than adults. To reproduce, the young must develop and grow to mature size, and there are two basic ways to get from infancy to adulthood. One is to develop fast and minimize the duration of the nonreproductive period between weaning and sexual maturation – this is the strategy followed by the mouse, the bison, and most mammals.

This strategy places a premium on quantity of reproduction rather than on quality, as there is little time to learn. The other strategy is to develop toward adulthood slowly, increase the time for learning, and produce higher-quality adults. A corollary of each strategy for mammals of medium to large body size is that fast growth and development requires more food in a shorter period of time, while slower growth and development allows for lower intakes of food spread out over a longer time. Janson & van Schaik's feeding-competition hypothesis is that slow juvenile growth reduces the risk of death from starvation. Juveniles must forage for their own food, a skill that must be practiced until mature levels of success are achieved. Furthermore, juveniles are in competition with adults. This competition becomes clear during times of food scarcity, when juvenile primates die in greater numbers than infants or adults. But, not all juveniles die, and there is a relationship between growth and mortality. A small, slow-growing juvenile requires less food input than a larger, fast-growing individual and may survive periods of food scarcity, and there is evidence for this from both primates (Janson & van Schaik, 2002) and elephants (Lee & Moss 1995).

Moreover, slow growth and smaller size allow juveniles to practice feeding skills with less risk of starvation during all seasons of the learning period. In the past, individuals with slow growth and delayed reproductive maturation following infancy may have survived to adulthood more often than individuals with rapid growth and maturation, and thus juvenile stage may have evolved.

An example of the juvenile pattern of growth is given in Figure 3.12 for the elephant. The distance curves are based on cross-sectional means of shoulder height, a common measure of skeletal growth for elephants (see Lee & Moss, 1995 for details of the methodology of measurement). The original means were plotted and fit with a distance-weighted least-squares regression. The curve produced by this statistical technique represents a type of "average" growth and the curve bends to more closely approximate the values of mean shoulder height at each age. The distance curve for both male and female elephants shows the typical juvenile pattern of prolonged and relatively slow growth for many decades. The increments in growth between successive data points were calculated and, as was done for the distance curves, the increments between means were plotted and fit with a distance-weighted least-squares regression. The shape of this fitted increment curve reveals the stages of postnatal growth more clearly than the distance curves. The approximate duration of infancy (I), juvenile (J), and mature adult (M) stages are indicated.

The increment curves must be interpreted with a great deal of caution since they are calculated from means rather than individual data. Increments are presented only to provide a very rough approximation of growth velocity. Overall, the fitted curve shows a fairly rapid decline in rate of growth during the first decade of life and then a slower, prolonged decline to age 25 years for females and age 50 for males. The difference between the sexes in shoulder height seen in the distance curve is explained by the additional 25 years of growth for males. The increment estimates fluctuate widely, and some are even negative. This is due to the cross-sectional nature of the data, that is, the adjacent measurements are from different individual elephants, and the unequal spacing between increments for each individual. The fluctuations also may be due to very different ecological conditions for growth during the life of these individuals. It is possible that negative increments represent the difference in size between individuals who experienced good ecological conditions and those growing up under harsh conditions. Notice that the negative increments, and most of the fluctuations, are confined to the first 20 years of elephant life. That time includes the infancy and juvenile stages – in this sample of elephants, from the Amboseli reserve in Kenya, males do not reproduce until after 25 years of age, females reach puberty at about age 9 years and begin reproduction after 10 years of age. The fluctuations in these increment data, despite their limitations, do add some support to the ecological risk-aversion hypothesis as an explanation for the juvenile stage of growth. In other words, the environment in which these elephants live is unpredictable, and a strategy of slow growth to adulthood may well offer the best chance to avoid starvation in bad years.

Another possible explanation for a juvenile stage for social mammals may be called the "reproductive dominance hypothesis." Research with wild and captive

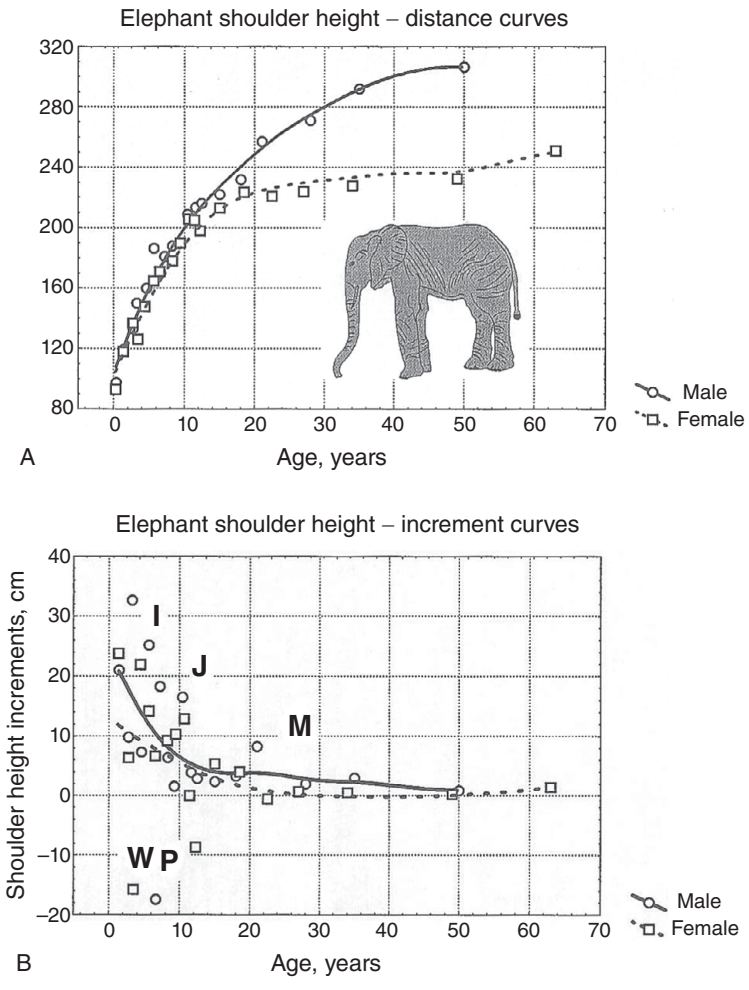


Figure 3.12 Growth curves for elephants illustrating the pattern of growth in social mammals with infant, juvenile, and adult stages of growth. (A) Distance curves, (B) increment curves. Weaning (W) takes place at a mean age of 40 months. Puberty (P), meaning reproductive maturity, occurs at about age nine years in females. Other abbreviations are: I, infancy; J, juvenile; M, mature adult. The mean values of shoulder height were kindly supplied by Dr. P. C. Lee, Department of Psychology, University of Stirling,, UK and published by Lee & Moss (1995)

primates, with elephants, and with social carnivores (wolves, lions, hyenas) shows that high-ranking individuals in the social hierarchy can suppress and inhibit the reproductive maturation of low-ranking individuals (Pereira & Fairbanks, 1993). The inhibition may be due to the stress of social intimidation acting directly on the endocrine system or may be secondary to inadequate nutrition due to feeding competition. Juveniles are almost always low-ranking members of primate social systems. One primate example is that socially dominant male orangutans

Figure 3.13 Social dominance and subordinance in orangutans are associated with male secondary sexual characteristics. A socially dominant male (right) demonstrates secondary sexual characteristics, primarily in the form of enlarged bidiscoid cheek pads. An unflanged, socially subordinate male (center) appears similar in facial morphology to a female orangutan (left). Both male morphs are sexually mature and both can father offspring. Flanged males have more testosterone, cortisol, and growth hormone (Emery Thompson et al. 2012; Maggioncalda et al. 2000). Source: Reprinted by permission from Springer Nature Customer Service Centre GmbH: Banes, G. L., Galdikas, B. M. F. & Vigilant, L. (2015) Male orang-utan bimaturism and reproductive success at Camp Leakey in Tanjung Puting National Park, Indonesia. *Behavioural Ecology and Sociobiology*, 69, 1785–1794.

(*Pongo* spp.) are physically larger than male subordinates. The former also develop conspicuous secondary sexual characteristics such as enlarged fatty cheek pads and throat pouches (Emery Thompson et al. 2012, Figure 3.13). The socially dominant males use their physical characteristics to intimidate other males and attract female mating partners. The subordinate males grow and mature more slowly. Some remain "juvenile" in appearance for many years but are in fact sexually mature and seek mating opportunities without directly confronting the socially dominant males.

The three hypotheses for the evolution of the juvenile stage – learning, ecological risk aversion, and reproductive dominance – are not mutually exclusive. They may all have been at work in the past, perhaps in a sequence of evolutionary processes. One possible ordering of these processes in highly social species of mammals is as follows. The first step was risk-aversion which slowed the rate of growth and prolonged the duration of growth prior to reproductive maturity. A secondary consequence of growth prolongation was time to invest in learning. Thirdly, over evolutionary time there was selective feedback and reinforcement between risk-aversion, growth, and learning. This interactive feedback extended beyond feeding competition to other realms of social life, including reproductive competition and social status. There are implications of these aspects of social life for human physical growth and development, for example, the effects of dominant or subordinate social

status on growth in height of humans (Bogin et al. 2015a; Hermanussen & Scheffler 2016). These implications are discussed in more detail in later chapters.

In summary, whatever the cause of the juvenile growth stage, be it learning, risk aversion, or social inhibition, one scholar points out that in broad perspective, " . . . juvenile life has two main functions: to get to the adult stage without dying and to become the best possible adult" (Alexander 1990). Adding a juvenile stage must have served this purpose well for it to evolve independently in so many species of highly social mammals.

Primate Growth Patterns

Most primates are highly social mammals and, as expected, have a juvenile stage of growth and development. The primates, however, are a diverse group of species including the strepsirrhine (lemurs and lorises) and haplorrhine (tarsiers and anthropoid) clades. The anthropoids include New World monkeys (ranging from marmosets and tamarins to cacajos and howlers), Old World monkeys (such as baboons, rhesus, and colobus), the apes (gibbons and siamangs, orangutans, gorillas, common chimpanzee, and bonobo), and humans. In all there are 12 Families and approximately 240 species of living primates.[3] Primates live in environments that range from semi-arid scrub, to savanna, to woodland, and rain forest, and from sea level to more than 2,000 m. Adult sizes range from the male gorilla, at 160 kg, to the mouse lemur (*Microcebus murinus*) at 0.08 kg. Maximum life spans range from 8.8 years for the dwarf lemur (*Cheirogaleous major*) to about 120 years for humans (Harvey et al. 1986). Given this variation in world distribution, general environments, local ecologies, adult body size, and life span it is reasonable to expect that primates have a variety of patterns of growth and development, and indeed they do.

Three patterns of primate postnatal growth in weight are illustrated in Figure 3.14, based on the work of Steven Leigh (Leigh 1992, 1994). There are differences in growth patterns between the three species (marmoset, baboon, colobus) and between males and females within species. Furthermore, none of these species follows a pattern of weight growth similar to the human species. The marmoset seems to follow the type of growth curve Brody described for most mammals, that is an acceleration phase followed immediately by a deceleration phase. The switch between phases seems to occur just before the time of birth, and the infancy stage merges seamlessly into the reproductively mature stage. Based on the velocity curves of weight growth there is no evidence of a juvenile stage. The lack of a juvenile growth stage may relate to marmoset social behavior. Marmosets, and the closely related tamarins, are **cooperative breeders**, meaning that only one or a few adults of each sex breed and all the nonbreeding adults contribute time and effort toward the feeding and care of the breeders and the offspring (Lukas & Clutton-Brock 2012). Marmosets and tamarins live in a type of "family" group, in which the socially dominant female forms a social bond with one or a few males and with their full- or half-sisters born from the same mother. The dominant female monopolizes all breeding. These "families" with the single

[3] www2.palomar.edu/anthro/primate/table_primates.htm

Figure 3.14 Three primate species showing different patterns of postnatal growth in weight. Abbreviations are: I, infancy; J, juvenile; A, Adult. Marmosets are weaned at 63 days and can breed at one year of age (Harvey et al., 1986). Marmoset growth rate shows no postnatal growth spurt and no change in growth rate, indicating a lack of a juvenile growth stage. Baboons are weaned by 18 months and may breed at about 3.5 years for females and 4.5 years for males (Harvey et al., 1986). Baboons have a juvenile growth stage for both males and females, but only males have clear weight growth spurt. Colobus monkeys are weaned at about 13 months and females have their first birth at about 4.6 years of age. Colobus monkeys show a post-natal spurt for both sexes, but it is not clear if there is a juvenile growth stage. The velocity curves are fit to cross-sectional data collected from captive animals. The curves are fit using statistical estimates based on LOWESS (Locally Weighted Scatterplot Smoothing) regression – see the text for details. (After Leigh, 1996)

polyandrous breeding female establish feeding and breeding territories and exclude marmosets from other "families." The young, usually born in pairs or triplets, are raised cooperatively by the mothers, their male consorts (typically at least two males in a breeding group), and the nonbreeding socially subordinate females. After weaning, which takes place as early as 6 months for some species and by 12 months for other species, the young are protected and provisioned with food by all the adults until reaching sexual maturity at about age 15–24 months (Goldizen 1988).

Leigh (1996) analyzed growth data for five other species of marmosets and tamarins and all of these follow a pattern of growth essentially the same as for the common marmoset. Following Janson & van Schaik's risk aversion hypothesis, a juvenile stage of growth would not be needed in this type of territorial, cooperatively breeding "family" social group. A juvenile stage, at least for the females, might be expected based on the social dominance hypothesis. Garber & Leigh (1997) did not find that risk-aversion from predation and starvation were major factors, rather they offered an alternative interpretation that reproductive costs to parents, especially females, explain the growth and development pattern for marmosets and tamarins.

The baboon velocity curves show evidence for infancy, juvenile, and adult stages of growth. Baboon infants are weaned by about 18 months after birth, but growth

rates remain fairly stable until about age four years – a clear juvenile pattern of growth. Baboons reach a peak in growth rate before birth, just as humans do, but by the time of birth baboons are already growing at the slower, steady rate that characterizes late infancy for humans. Both male and female baboons follow this pattern during infancy, but during the juvenile phase males show a pronounced acceleration in weight growth – a spurt. Baboons live in large social groups composed of both males and females of all ages, all adults attempt to breed, there are hierarchies of dominance and subordinance for both sexes and there is much feeding competition between adults and younger individuals. The baboon growth pattern conforms to the predictions that there will be a juvenile growth stage. Why males, and not females, have a juvenile growth spurt in weight is not known. It is known that the spurt is associated with the eruption of larger canines in males at the time of sexual maturation. The spurt in body weight at this time is due mostly to an increase in muscle mass and may be a consequence of the large canine teeth, the large jaws and skulls needed to contain these teeth, and the upper body musculature required to support the cranial skeleton. Males use their teeth and muscle to compete for scarce resources such as food and mates. An open question is the relationship of dominance status at puberty to the intensity of muscle and dental growth spurts of nonhuman primates.

Colobus monkeys show yet a third pattern of postnatal growth. Both males and females have a clear acceleration in weight growth after birth. What is not clear is whether these accelerations are juvenile growth spurts. It is possible that colobus monkeys grow more like marmosets than like baboons. Like the marmoset, the colobus may have only a single peak rate of growth, but that peak is reached soon after birth. The colobus, then, would be following a growth pattern like that for the mouse and the cow (Figures 3.10 and 3.11). Colobus social behavior and diet do not provide a clear case for the prediction of a juvenile stage. Colobus monkeys live in troops of 3–15 animals; with a typical troop comprising a single adult male, 3–4 adult females, and their offspring. These troops are highly social, there is little evidence for a female dominance hierarchy or female aggression, and infants seem to be groomed and cared for by troop members other than the mother. This mutual infant care, as well as intense allo-grooming between adult females, is believed to maintain a highly cohesive social group (Dunbar 2010; Struhsaker & Leyland 1987). Colobus monkeys eat mostly young leaves from the hackberry tree (*Celtis durandii*) and two other tree species, which together comprise 69% of the total diet. The colobus, and related leaf-eating monkeys of the subfamily Colobinae, digest this large quantity of leaves in a specialized stomach comprising three or four subcompartments. Their digestive system is similar to the ruminant digestive system of the cow. Given the wide distribution and availability of their food, and given their cohesive and noncompetitive social organization, it is possible that young colobus monkeys are exposed to very low risk for mortality. If so, then both the risk-aversion and social dominance hypotheses predict the absence of a juvenile growth stage, or the presence of a very brief stage. Food availability may be the key underlying factor in selection for a juvenile stage. In an analysis of 42 primate species, Leigh (1994)

found a significant correlation between diet and rate of growth. Folivores, such as the colobus monkey, have faster rates of growth than species of nonfolivore monkeys and apes, such as the rhesus monkey and the gibbon. So, perhaps it is not so far-fetched to state that the colobus has both a cowlike stomach and a cowlike pattern of growth.

A more detailed discussion of primate juvenile growth, especially postnatal growth spurts, is presented later in this chapter. Before that review, some of the history and findings of research into primate growth is discussed to better appreciate what primates share with other mammals and what is special about primate growth. Biological and evolutionary comparisons of humans with nonhuman primates are known from the nineteenth century, but the systematic study of primate growth and development in relation to human evolution began with the work of Adolph Schultz (1891–1976). Schultz (1924) published the article "Growth studies in primates bearing upon man's evolution." A year earlier, Schultz published a detailed analysis of human fetal growth (Schultz 1923) and two years later expanded this topic to include nonhuman primates (Schultz 1926). These articles are primarily a descriptive mix of Schultz's quantitative and qualitative assessments of primate ontogeny, based on careful measurement and dissection of cadavers of fetuses, neonates, immatures, and adults. Schultz does not cite Thompson's *On Growth and Form* in these articles, but he summarizes his analysis with the Thompsonian statement that, " ... there will remain the forcible conclusion that the many striking resemblances between man, ape, and, monkey in early development, and their frequently closely corresponding growth changes can only be explained by one common origin, from which they all inherited the tendency for the same ontogenetic processes ... " (Schultz 1924, p. 163).

In later publications Schultz (1935, 1960, 1969) pioneered the analysis of dental maturation and tooth eruption timing as life history markers. Before Schultz, there were publications by Wilton M. Krogman (1903–1987) on the eruption of teeth in Old World monkeys and apes (Krogman 1930). He reported that the first permanent molar (M1) is always the first of the permanent teeth to erupt in all the primates studied. Krogman also discovered that humans take about three times longer than nonhuman primates to progress from M1 to M3 eruption. Anecdotal evidence had led British anthropologists Arthur Keith (1866–1955, chief proponent of the fraudulent Piltdown Man) and Solly Zuckerman (1904–1993) to incorrectly report that apes and humans were nearly identical in the timing of dental eruption. Krogman also seems to be the first anthropologist to report that the correlation between, " ... epiphyseal union with tooth eruption indicates that the growth process in the Anthropoids, while similar in pattern to that of Man, is completed in shorter time" (Krogman 1930, p. 312). Krogman's discovery of: (1) the primacy of M1 eruption in all primates; (2) the significant human delay in molar eruption sequence; and (3) the overall delay in skeletal maturation of humans were major findings that became the basis of all life history research with living and extinct primate species (Smith 1991).

By 1924 Schultz was using the words *embryonic, fetus, newborn, infant, juvenile,* and *adult* as names for distinct stages or phases of primate growth and development.

He used the words "child" and "children" to denote human pre-adults of any age. The word "adolescence" is mentioned one time in 1926 (Schultz 1926). Schultz does not define clearly any of these stages of development; rather the words are used as if the reader understands the meaning. He does state (Schultz 1935) that eruption of the first permanent teeth, most often the M1, indicates the end of the infantile period and, presumably, the start of the juvenile period.

The use of these names for developmental stages became more formally associated with the timing of permanent molar eruption when Schultz published what became one of his most lasting contributions – an illustration of the "Approximate ages of some life periods" of the primates (Figure 3.15, Schultz 1960). In this figure, Schultz defines the Infantile period from birth, " . . . to the first permanent teeth," the Juvenile Period, " . . . to last permanent teeth," and the Adult Period, " . . . to end of mean longevity." More detailed technical definitions of mammalian life course stages were published in the 1960s and these are discussed below. Schultz's original figure included a speculative column on the life periods of "Early Man" and in so doing firmly established the use of molar eruption sequence in the study of the evolution of human life course biology.

From inception, Schultz's studies were aimed at " . . . the relation of the growth of primates to man's evolution . . . " (Schultz 1924, p. 163). Despite the grandeur and vision of Schultz's research on primate growth and primate evolution, very few primate species had been studied in detail by 1960. In fact, until the 1980s details of skeletal, dental, and somatic growth were known from only three species, the rhesus monkey (*Macaca mulatta*), the chimpanzee (*Pan troglodytes*), and humans.

Ana Laird continued Schultz's interest in the "Evolution of the human growth curve" – the title of Laird's (1967) paper. In that paper she reviewed studies of the growth of the three well-studied species; rhesus monkey, chimpanzee, and human. Laird took a mathematical approach to the study of growth. By fitting mathematical functions to the growth data, Laird hoped to reveal more precisely the stages in the evolutionary development of the human growth curve. The curves were fitted to the monthly weight data points by the method of least squares regression. Essentially, this method minimizes the sum of the squared deviations of each data point from the fitted curve, that is, it produces a type of "average" line between the data points.

Laird found that monthly weight increases in the rhesus monkey and the chimpanzee followed two separate growth curves. For the male rhesus, the first curve fit the data from birth to 22 months and the second from 23 months onwards (Figure 3.16). A change in growth rate that occurred between months 22 and 23 necessitated the use of different mathematical curves to model growth in the two periods. Sexual development in the male rhesus takes place during the second growth phase, after month 40. The deviations of weight growth above the fitted curve between months 48 and 54 corresponded to the time of reproductive maturation and the beginning of adult levels of gonadal hormone secretions. The curves of growth for the female rhesus were similar to the male, except that the time of sexual maturation was earlier, occurring at about 42 months. For the chimpanzee, the first curve fit the early phase of growth, from birth to six years. This early phase is

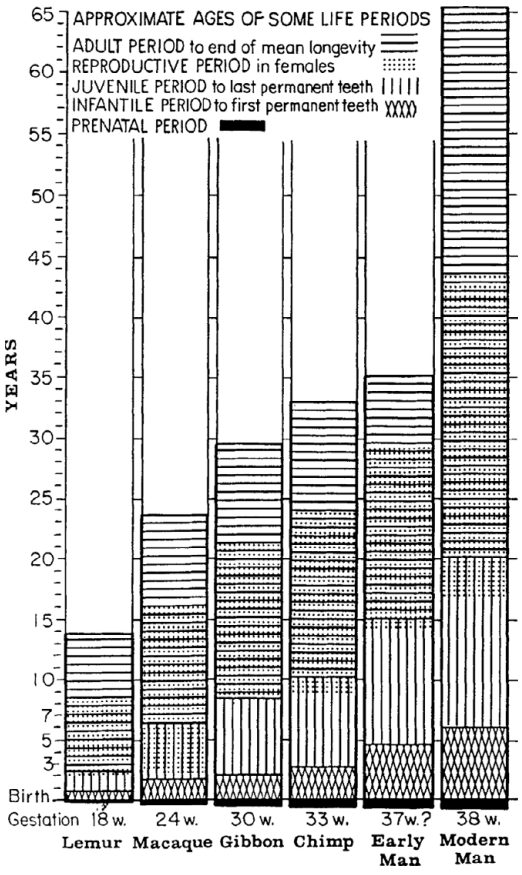


Figure 3.15 Schultz's diagram of the proportional increase in the length of life stages across the *scala naturae* of living primates. Note that Schultz used eruption of the permanent teeth to mark the boundary between life periods. Schultz did not recognize the childhood or adolescent stages for modern humans. Schultz considered that all primate species have the same life stages, which just increase in length from prosimian to human. The estimates for total length of life are based on average expectations rather than theoretical maxima. The column for "Early Man" was entirely speculative as no species is given and very little data were available when Schultz prepared this figure. From Schultz (1960)

essentially the infancy period, which lasts to age five years in chimpanzees (Goodall 1983; Nishida 2011; Teleki et al. 1976). The second curve was fit to what Laird called the "adolescent phase." Male and female chimpanzees followed the same curve of growth from birth to six years. During the "adolescent phase" males grew in weight at a faster rate than females and this required separate mathematical functions for each sex. Due to the different rates of weight growth sexual dimorphism in weight became well marked. The sexual dimorphism reached its greatest level at the age when male chimpanzees begin to sire offspring. Laird's analysis confirmed the work of Tanner (1962), who had shown that the sexual dimorphism in adult chimpanzee weight was largely due to a weight growth spurt for the males (Figure 3.17).

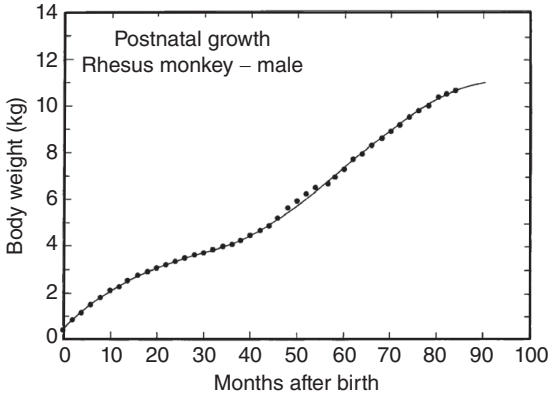


Figure 3.16 Growth in body weight of the rhesus monkey. (From Laird 1967)

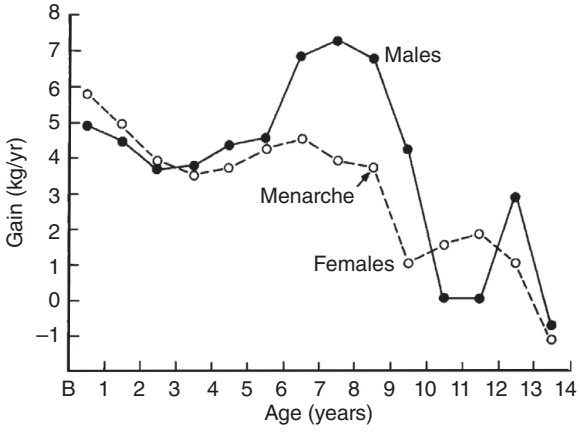


Figure 3.17 Weight velocity curve for the male and female chimpanzee. (From Tanner 1962)

Laird (1967) found that the velocity curve of human growth, as depicted in Figure 2.5B, required three mathematical functions to model its course. This conclusion was confirmed, independently, by others (e.g., Bock & Thissen 1980; Bogin 1980; Karlberg 1989). The need for the third function is one aspect of human growth that makes it different from the growth of the other primates. Laird described the similarities of growth between the rhesus monkey and chimpanzee and the distinct pattern of human growth as follows:

> . . . the curvilinear growth by which the body weight of an organism approaches its mature value and during which sexual maturation characteristically occurs, starts at birth in subprimate mammals and birds, but is deferred in monkeys . . . the preliminary growth occupying about 1/3 and the adolescent growth about 2/3 of the time required to reach fully mature size. In the chimpanzee, adolescent growth is deferred to the last ½ of the total period . . . In the

human a further delay has occurred so that adolescent growth with its concomitant development of sexual maturity occupies only the last 1/3 of a prolonged growth period. The delay in the human can be interpreted as being due to the *insertion*, between birth and adolescence, of two growth phases, rather than the single phase identifiable in the monkey and the chimpanzee . . . (Laird 1967, pp. 351–22, italic emphasis added.)

It seems that Laird considered all three primates to be alike in having a juvenile phase between infancy and what she called "adolescence." Laird did not appreciate the second growth phase that humans add between infancy and puberty, which is childhood. The insertion of childhood into the human life cycle not only requires a new mathematical parameter to fit a growth curve, but, more importantly, childhood changed the biological and social ecology of the human species. It also underlies the human capacity for **culture**. The biological, social, and cultural implications of human childhood are discussed in detail in the Chapter 4.

Of Brains and Bodies

There is another key difference between nonprimate and primate growth, and this involves the relative rates of growth of the body, the brain, and the reproductive system. Most mammals, including the rat (depicted in Figure 3.18) and the social mammals, show an advancement of brain growth relative to body growth. In the rat reproductive maturation occurs before the brain or the body achieve final adult size (Donaldson 1895). Primates delay body growth and reproductive development, but do not delay brain growth. Figure 2.8 illustrated these relationships in humans. The weight of the human brain reaches 80% of adult size by age four and virtually 100% of adult size by age 7. Yet body growth continues to age 18 and beyond. Even more to the point, brain growth is finished before reproductive maturity even reaches 10% of the adult value. This pattern of relative growth of the brain, body, and reproductive system is also found in the rhesus monkey and the chimpanzee (Laird 1967). Other organs of the primate body (e.g., heart, lungs, liver) follow the body growth

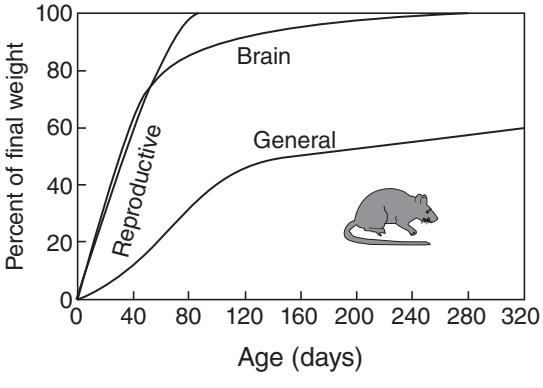


Figure 3.18 Growth of different types of tissue in the rat. (After Timiras 1972)

curve. The primate brain, then, is most unusual in its pattern of accelerated growth in comparison to other organs and the body as a whole.

In one obvious way, this pattern of growth relates to the fact that most anthropoid primates have complex feeding and social environments and need to overcome ecological and social challenges. Large brains and flexibility in behavior and learning are one way to meet these challenges. A clever mathematical analysis of the ecological (metabolic demands for food energy) and social drivers of human brain-size evolution finds that, " . . . a combination of 60% ecological, 30% cooperative and 10% between-group competitive challenges . . . " contribute to human brain size (González-Forero & Gardner 2018). It is not known if these percent contributions apply to other primate species. There are other consequences of the primate pattern of growth that are not as obvious as learning. Some of these are dealt with in the next chapter. At this point it is worth noting that rapid brain growth, deferred body growth, and progressively delayed sexual maturation greatly enhance the quality and quantity of reproductive efficiency in primates. Offspring are endowed with great flexibility of behavior, learning, and survival strategies.

In humans, with our childhood and adolescent periods of growth, reproductive and survival efficiency are further enhanced, and because of this there was strong selection at the genetic level of primate biology to develop and maintain these patterns of growth.

The Human Adolescent Growth Spurt Is Unique

One of the hallmarks of human growth is the adolescent growth spurt, the acceleration in rate of growth in height and weight. Many individual organs and most body dimensions experience the adolescent spurt. Moreover, the adolescent spurt is detectable in both boys and girls, and in virtually every human population so far examined. The adolescent spurt, which is easily visualized from Figure 2.5, is a regular and normal feature of human growth. The size of the adolescent growth spurt strongly depends on mean height of the population. The taller a population, the greater this spurt. In the nineteenth century, Europeans were shorter and had smaller adolescent growth spurts than modern Europeans (Hermanussen 1997). Only chronic and severe illness, certain hormonal disorders, chronic malnutrition, or physiological stress can obliterate the growth spurt. For instance, Frisancho (1977) reported that Quechua boys and girls living at high altitude in the Peruvian Andes have a late and poorly defined adolescent growth spurt. The reason for this is that these children suffer from the combined stresses of hypoxia (insufficient delivery of oxygen to the tissues of the body), energy malnutrition, heavy workload, cold, poverty, and social discrimination. Instead of a clear spurt, the Quechua experience a prolonged adolescent growth period, lasting until age 22 and beyond. Even with their growth prolongation, growth rates are slow and adult Quechua men and women are of short stature.

There was some controversy in the twentieth century as to whether the adolescent growth spurt is a uniquely human feature. On the basis of empirical observations and evolutionary considerations, I proposed that the human adolescent growth spurt in

stature is a species-specific characteristic, that is, a skeletal growth spurt of the human type is not found in any other primate species (Bogin 1994, 1999a; Bogin & Smith 1996b). My proposal stood in contrast to that of previous researchers. As discussed above, Brody (1945) believed that the human adolescent growth spurt, both for height and weight, was homologous with the peak velocity of growth shown just after birth by most laboratory and farm animals. Bertalanffy (1960) and Laird (1967) also believed this to be the case. For these researchers, the special features of primate growth were the prolongation of the juvenile period, found in all haplorrhine primates, which reached its most extreme duration in humans. This progressive prolongation of juvenile growth delayed the postnatal growth spurt common to most primate species until late in the juvenile stage of growth. Newer data and better mathematical analyses showed that this interpretation was incorrect.

Based on the evidence available today, there is no question that some monkeys and apes have pubertal growth spurts in weight, but not in total body length. The work of Leigh (1996) is the most comprehensive study to date that is directed at the question of the primate growth spurts in weight. Some of Leigh's findings were presented above in Figure 3.14. Leigh collected data for chronological age and body weight for 2,395 captive primates, housed at both zoos and primate laboratories. There are 35 nonhuman species in his sample, including representatives of the New World monkeys, Old World monkeys, and apes. He also included a data set for healthy humans (English boys and girls measured in the 1970s and 1980s). Although there are many longitudinal records for individual animals in the data base, the data are analyzed in a cross-sectional fashion. Leigh explains this approach by stating that **cross-sectional analysis** " . . . leads to artificially depressed estimates of growth spurt magnitudes . . . [and] should lead to a conservative diagnosis of the presence of growth spurts" (Leigh 1996 p. 457). Leigh also used the nonparametric mathematical technique called LOWESS regression to fit smoothed curves to the growth data for each primate species. There are many advantages and disadvantages to the use of mathematical curve fitting, and I suggest that interested readers consult books on the theory and application of regression for details. I can state here that Leigh's decision to use the mathematical smoothing technique of LOWESS regression is useful, since it helps to reduce the unimportant variability (often called the "noise") in time-series data sets and helps to reveal the important main trends that occur over time. LOWESS curves were fit to the distance data and then the first derivative of the distance curve was estimated to produce a velocity curve. This follows exactly the procedure used at the beginning of this chapter to construct Figure 3.1.

A summary of Leigh's results for the 35 nonhuman species he analyzed is given in Table 3.1. Only one species of New World monkey showed a spurt, *Cebus apella*, and only for males. All of the Old World monkeys studied showed a spurt in weight for males, and many species also showed a weight spurt for females. Among the ape species, gibbons (*Hylobates spp.*) showed no spurt, orangutans (*Pongo spp.*) showed a possible male spurt, the common chimpanzee (*Pan troglodytes*) showed a male weight spurt but no female spurt, and both the bonobo (*P. paniscus)* and the gorilla (*Gorilla*) had male and female weight spurts. The variation in presence or absence of a

Table 3.1 Summary results of Leigh's (1996) analysis of weight growth in primates. Species evaluated, sample sizes, and presence or absence of a postnatal growth spurt by sex.

Species	Sample size	Growth spurt
	(Male/Female)	(by sex)
Ceboidea (New World monkeys)		
Cebuella pygmaea	36/51	None
Callithrix jacchus	48/71	None
Cailirnico goeldi	42/47	None
Saguinus fuscicollis	18/19	None
Saguinus geoffroyi	9/10	None
Saguinus imperator	11/14	None
Saguinus oedipus	46/18	None
Leontopithecus rosalia	26/31	None
Saimiri sciureus	32/28	None
Cebus apella	26/28	Male
Callicebus moloch	30/23	None
Aotus trivirgatus	25/23	None
Cercopithecoidea (Old World monkeys)		
Cercopithecus aethiops	30/30	Both
Cercopithecus mitis	27/37	Male
Cercopithecus neglectus	29/23	Male
Erythrocebus patas	41/52	Both
Cercocebus atys	38/71	Male
Macaca arctoides	52/58	Male
Macaca fascicularis	13/13	Both
Macaca fuscata	64/71	Both
Macaca mulatta	52/58	Both
Macaca nemestrina	39/64	Both
Macaca silenus	39/41	Male
Papio hamadryas	33/53	Male
Mandrillus sphinx	49/59	Both
Colobus guereza	46/49	Both
Presbytis entellus	29/24	Both
Presbytis obscura	19/17	Male
Hominoidea (apes and humans)		
Hylobates lar	25/25	None
Hylobates syndactylus	19/21	None
Pongo pygmaeus	42/42	Male?
Gorilla gorilla	77/64	Both
Pan paniscus	13/23	Both
Pan troglodytes	22/23	Male
Homo sapiens	Literature data	Both

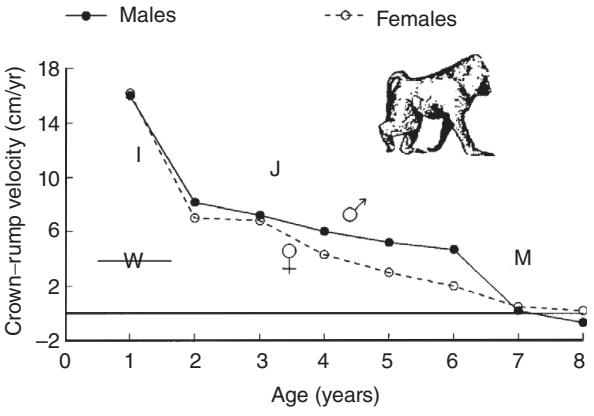


Figure 3.19 Velocity curves for CRL in the baboon. (After Coelho 1985)

postnatal weight spurt among primate species, and between sexes within a species, is in keeping with the diversity of primate biology and ecology. Leigh's data show clearly that a postnatal weight spurt is not a primate characteristic, rather it is a variable trait found in one, or both, sexes of some species.

In contrast to Leigh's comprehensive survey of primate weight growth, there are only a few studies of skeletal growth. Coelho (1985) measured gains in crown–rump length (CRL) and weight in a mixed-longitudinal sample of 250 male and 452 female olive baboons (*Papio cynocephalus anubis*). The animals were part of a laboratory colony living under naturalistic conditions in terms of the physical environment and social group composition. All animals were healthy and well-nourished, and none showed signs of clinical obesity. The date of birth for all individuals was known and the animals were measured once a year. The mixed-longitudinal design of the study provided data on growth between birth and eight years of age, which for this species is the total span of the growing years. Weight velocity is essentially identical to the data for the Red baboon presented in Figure 3.14. Velocity curves for CRL are presented in Figure 3.19 and stand in sharp contrast to the weight velocity curves, for neither male nor female baboons show a spurt in CRL. Baboons do show three phases of declining growth rate in CRL: an infancy phase until two years, a juvenile phase from two to six years, and a post-pubertal phase until eight years. Similar patterns of decreasing velocity in skeletal growth from birth to maturity were found in studies of three other monkey species, *Macaca nemestrina* (Orlosky 1982), *Papio cynocephalus* (Sirianni et al. 1982), and *Macaca sinica* (Cheverud et al. 1992). Based on these studies, there is no evidence for the presence of humanlike adolescent growth spurts in the skeleton of any of these species of Old World monkeys.

Another mixed-longitudinal study of growth with hamadryas baboons offers the suggestion of a pubertal spurt in CRL for males but not for females (Crawford et al. 1997). The male CRL spurt as calculated from pseudo-velocity curves amounts to about a two-centimeter increase in growth rate from pre-pubertal values. Pseudo-velocity curves are estimates based on a varying number of individual baboons, with

as few as two and as many as eight individuals measured on different occasions. It is difficult, if not impossible, to derive an accurate and reliable estimate of growth velocity from data of this type.

In 1990, Tanner and colleagues, analyzing data from rhesus monkeys, reported a pubertal growth spurt in tibia and CRL. The researchers stated, " ... the pubertal growth spurt in female rhesus is very little different from that in man" (Tanner et al. 1990, p. 101).

Chimpanzees Settle the Question

If there were a post-pubertal skeletal growth spurt in any nonhuman primate species, then it should be found for chimpanzees as they are most genomically, anatomically, and behaviorally similar to humans. Based on an analysis of 16 captive chimpanzees Elizabeth S. Watts (1941–1994) and James A. Gavan (1916–1994) suggested an adolescent growth spurt in leg length similar to that of humans (Watts & Gavan 1982). That alleged spurt, however, was on the order of millimeters and not the 2–3 centimeters typical of humans.

Copeland and colleagues (1985) reported on a year-long study of 86 male and female chimpanzees who were measured for body weight and CRL in relation to blood serum concentrations of testosterone (T), estradiol (E2), dehydroepiandrosterone sulfate (DHEA-S), cortisol, and somatomedin-C (now called insulin-like growth factor 1, or IGF-1). In male chimpanzees aged 6 to 8 years old there was a marked increase in serum T that preceded slightly an increase in the rate of body weight gain. There were no detectable increases in serum E2 or the velocity of CRL growth. In female chimpanzees between 6 and 8 years old, serum T increased, and this was concurrent with an increase in the rate of body weight gain, but that gain was much smaller than that in the male. Serum E2 increased only after 10 years of age, and there was no increase in the velocity of CRL growth. Simply put, no evidence of a pubertal skeletal growth spurt.

Steven Leigh (Leigh 1996) reviewed the research on primate growth spurts and reaffirmed that some nonhuman primate species have pubertal spurts in weight growth, but other species do not, and that these spurts may be present in only the males of a species. Moreover, Leigh found that skeletal growth spurts may occur in some regions of the body, such as the face when males erupt large canine teeth, but not in other regions of the body of the same animals. Leigh related these regional spurts to sexual differences in life histories (e.g., rates of maturation, age at first reproduction, age at death).

The question of skeletal growth spurts in chimpanzees was finally settled by Hamada and Udono (2002), who reported that chimpanzees do not have a skeletal pubertal growth spurt (Figure 3.20). Their data were derived from a longitudinal study of captive chimpanzees. The chimpanzees of this study were raised at two research institutes in Japan. None of the chimpanzees was subjected to medical experimentation. This is important, as medical experimentation is known to modify and even stop the growth of chimpanzees. As infants, five were nursed by their

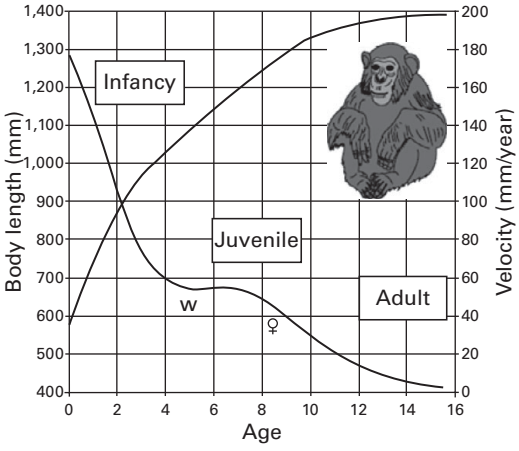


Figure 3.20 Distance and velocity curves for chimpanzee growth in body length (Hamada and Udono 2002). In the wild, weaning (W) usually takes place between 48 and 60 months of age. (Pusey 1983) The ♀ symbol indicates the typical age of puberty, defined as the first sexual swelling, of female chimpanzees in captivity.

mothers and seven were bottle fed by human caregivers. After the nursing or bottle-feeding period, all were transferred to social groups with age-peers, except for one infant chimp who remained with his mother. After weaning, some of the chimpanzees were housed in social groups and some in individual cages. All chimpanzees were measured serially from infancy until adulthood. The measurements, and routine medical examinations, were performed at 3-month or 6-month intervals, with the chimpanzees anesthetized. These chimpanzees were given good care, but their captivity and treatment may have influenced their physical growth.

As indicated in Figure 3.20, postnatal growth of the chimpanzee has only two stages prior to adulthood: infancy and juvenile. Infancy for both human beings and chimpanzees is characterized by the most rapid velocity of body growth of any of the postnatal stages, but also by a steep decline in velocity, a deceleration. Infancy of humans, chimpanzees, and other mammalian species are comparable in many other respects, for example, such as feeding by maternal lactation and appearance of deciduous teeth. In most mammals, including chimpanzees, infancy and lactation end with eruption of the first permanent molars (humans are the notable exception, as is discussed in the next chapter). In chimpanzees, first molar eruption takes place between the age of 48 and 60 months, as indicated by the "W" (weaning, defined as cessation of nursing) in Figure 3.20. Note the change in chimpanzee growth velocity at the time of weaning. At this point the chimpanzee enters the juvenile growth stage. The velocity of growth stabilizes and then decelerates again through puberty and sexual maturation. There was no evidence of growth velocity acceleration, that is a growth spurt, at the time of puberty or post-puberty.

A study of wild mountain gorillas reports a possible pubertal spurt in body length for males only, from about age 7–9 years (Galbany et al. 2017). No spurt was detected for arm length. The body length spurt followed an equally strong

deceleration in growth velocity between the ages about 5.7–7 years, which was not detected in females. It is possible that intimidation and social repression by older males, and females, inhibited the growth of young juvenile males and that the spurt following this inhibition is a type of catch-up growth, a pattern known from orangutans, humans, and other primates (Bogin 1999b; Emery Thompson et al. 2012; Maggioncalda et al. 2002). Catch-up growth results in a temporary acceleration of growth velocity until expected body size is achieved, and then growth velocity declines (Prader et al. 1963). This pattern mimics the normal human adolescent growth spurt. The chimpanzees measured by Watts and Gavan may have experienced catch-up growth following their use in research experiments. It is known that the newborn chimpanzees, "... were separated from their mothers immediately after birth and raised in the [Yerkes] Laboratories nursery where they could be continuously observed and studied" (Gavan 1953, p. 94). The physical and emotional stress of separation followed by experimentation likely stopped or slowed growth.

Some Important Differences between Human and Nonhuman Primate Growth

According to Leigh, the most unusual feature of the human skeletal growth spurt during adolescence is that it occurs at such a late chronological age, even relative to total life span. Other ways in which the human adolescent growth spurt stands in sharp contrast to all other primates are that a well-defined human spurt may be detected in both girls and boys and measured in almost all skeletal elements that comprise body and limb length (Figure 2.5B).

There is no doubt that some nonhuman primates have small increases in the rate of skeletal growth for some body dimensions at the time of puberty. There is considerable evidence that these small increases in skeletal growth represent real and important biological processes, most likely, the onset of adult levels of gonadal hormone secretions. It is well established that testosterone in male primates and low doses of estrogens, such as estradiol, in female primates can increase growth rates. However, more is involved since roughly equivalent amounts of hormone production in both the chimpanzee and humans results in strikingly different rates of growth. In the male chimpanzee, the concentration of testosterone in blood serum prior to puberty (from 1 to 6 years of age) averages 13 nanograms per deciliter (ng/dl). At about 11 years of age, when male chimpanzees are in the full throws of puberty, serum testosterone averages about 400 ng/dl (Martin et al. 1977). For the human male, the pre-pubertal serum testosterone concentration (from ages 1 to 12 years) averages 9 ng/dl (Winter 1978). Peak height velocity in human boys from western Europe occurs at a mean age of about 14 years, when serum testosterone levels average about 340 ng/dl (Winter 1978). Based on these data, serum testosterone concentrations have no association with pubertal or adolescent skeletal growth spurts – humans have a spurt in total body length and chimpanzees do not. Clearly, both chimpanzee males and human boys have large increases in testosterone production after puberty, but the effects on skeletal growth are not similar.

Three major differences between human and nonhuman primate growth may be highlighted at this point. These are: (1) the residual growth potential of the nonhuman vs. the human primate at adolescence; (2) the sensitivity of different body tissues to growth-promoting stimuli; and (3) sex differences in the expression of growth spurts at adolescence. Monkeys, apes, and humans all experience a prolongation in the time for growth and a delay in the age at onset of sexual maturation. In humans, the delay is greater, both relatively and absolutely, than in the monkey or ape and there are rapid increases (growth spurts) in both height and weight. Apes have the weight spurt but not the total body length spurt. These growth differences are likely to be regulated by the sensitivity of neuroendocrine receptors and post-receptors (i.e., biological tissues) to growth stimuli rather than by the rate or amount of production of the stimuli (e.g., hormones) themselves. The lack of linear associations between testosterone concentrations and growth velocities in skeletal and nonskeletal tissue of chimpanzee and human show this. The differences in cellular sensitivity to growth stimuli between nonhuman primates and the human primate are probably regulated, at least in part, at the genomic level. While it is possible that the different patterns of growth seen in the various primate species are the result of the evolution of new **structural genes** (genes that code for specific proteins), it is more likely that the variation in growth control lies in the regulatory genes that initiate and terminate each of the distinct periods of growth and control their duration (Stevens et al. 2013 and Chapter 6).

A Philosophy of Human Growth

Nonhuman primate models are used to study human growth because of similarities in anatomy and physiology between the species. When Schultz started his studies of primate growth he assumed explicitly that there was an evolutionary continuum between the living primate species. His famous illustration of primate life history (Figure 3.15) shows this assumed continuum very nicely. There is an evolutionary connection relating all primate species, but the living monkeys, apes, and humans each have a separate evolutionary history. Cercopithecoids (Old World monkeys) and hominoids (apes) separated some 20 million years ago and the **hominoid** – hominin (ape–human) split occurred about 6–7 million years ago. There is no evolutionary reason to expect that the patterns of growth of these three divergent and ecologically distinct species should be identical or even similar.

The notion of an evolutionary continuum is one legacy of the philosophy of a Great Chain of Being (Lovejoy 1936), a popular cultural construct in western society that has historical roots going back at least to ancient Greek writers. In biology, the Great Chain takes the form of the *scala naturae*, which in its original usage implies (erroneously) that all living creatures, from amoeba to human, form a living evolutionary sequence from the simplest to the most complex creature. We now understand that humans are not the culmination or the goal of evolutionary history. We are just one of the estimated 7.77 million animal species alive today (Mora et al. 2011), each the end-product of its own history and each with its own unique place in

nature. Yet, the *scala naturae* is sometimes misapplied to the connection between human and nonhuman primates.

Some observers tend to see monkeys and apes more as models for human biology and behavior than as creatures in their own right. This point was cogently argued by (Scott 1967, p. 72), "Subhuman primates are not small human beings with fur coats and (sometimes) tails. Rather they are a group which has diversified in many ways, so that they are as different from each other [and humans] as are bears, dogs and raccoons in the order Carnivora." Though Scott referred specifically to psychological attributes of species within the orders Carnivora and Primates, his cautionary remarks apply equally to morphology, physiology and, in the present context, patterns of growth. As mentioned above, within the Carnivora certain social species (dogs, wolves, lions) experience a prolonged period of relatively slow growth between infancy and adulthood, which corresponds to the juvenile growth phase. Nonsocial species of carnivores mature from infancy to adulthood without a juvenile stage of growth. There is no reason to expect, a priori, that the Primates, as an order of mammals, would be any more uniform in growth patterns than the Carnivora.

Laura Newell-Morris and C. F. Fahrenbach reviewed the use of nonhuman primates as models for human development and growth and concluded, " . . . there are problems with the extrapolation from the nonhuman primate model to the human condition because of intergeneric differences in size, growth and development rates, and timing. Although investigators justify direct extrapolation of their findings on the basis of the close genetic relationships of all primates, this assumption in many cases may be little more than absolute faith in the evolutionary argument from which it stems" (Newell-Morris & Fahrenbach 1985, p. 35).

Watts (1990) pointed out that the patterns of growth of the New World monkey *Cebus*, the Old World rhesus monkey (*Macaca*), the chimpanzee (*Pan*), and humans are all derived from some ancestor or ancestors. Each of the living species is likely to be derived from its ancestor in ways that are independent and unequal, due to "different ecological and adaptive circumstances" (Watts 1990, p. 99). Watts argues that the rhesus monkey makes a poor model for human growth due to an advanced state of skeletal development at birth and an early onset of puberty and menarche compared with humans. The rhesus is also a seasonal breeder in nature, with ovulation taking place in the fall or winter months and birth occurring in the spring or summer. When housed under artificial conditions, which was the case for all the laboratory studies discussed above, the sexual maturation of the female rhesus is very much altered as they can ovulate in any season. Several studies show that when experimental animals, including primates, are housed indoors and exposed to 12 hours of light followed by 12 hours of darkness (a standard procedure) many of their normal endocrine system functions are disturbed, or even obliterated (Bogin 1977). As both sexual maturation and growth of the body are influenced by many of the same hormones it seems most unreasonable to expect nonhuman primates reared indoors to serve as models for human growth and development.

My colleague Steve Leigh offered the following comment after reading a draft of this chapter for the 1999 edition of this book: "The presence of a spurt in tibia or in

other anatomical units certainly does not, as your review indicates, imply **homology** with the human statural spurt." In biology, the term "homology" is used to describe anatomical structures, physiological processes, or behaviors that are found in different species due to a common evolutionary origin. The five fingers and toes, and the intimate mother–infant relationship, of almost all primate species are due to homology. Leigh's point is that growth spurts for various parts of the body of different species of primates are not necessarily homologous. Continuing with Leigh's comment, he adds: "For example, my recent studies of baboons demonstrate unambiguous growth spurts in the snout ... The presence of these spurts, however, does not imply homology with human growth spurts in stature. CRL and sitting height would be the closest homologues, but even then, locomotor differences among these primates would greatly complicate assessments of homology. What I derive from the ideas you are presenting here, and in the last section, is that it might be best to think of growth spurts as modular and highly evolvable features of ontogeny. Natural selection (or sexual selection) can effectively 'put' spurts where (anatomically) and when they are needed to increase fitness. I think that the field has labored a little too long under Brody's desire to find a universal pattern. What may be happening here is more akin to a universal process. Perhaps we could propose that the universal process is modularity and evolvability of growth spurts (or decelerations), and that this process need not produce a uniform pattern between any two species or within a particular clade."

Leigh's proposals were reinforced by another of my colleagues, Michael Hermanussen, who commented to me that, "Spurts are usually accelerations in tempo, not in [the final amount of] height/length/weight. They occur whenever a new condition evolves that affects the biological tempo ... " Human growth tempo is slow compared with other primates, even the apes. When in evolutionary history the change in tempo began to occur is not known with certainty, but fossil discoveries since the last edition of this book (1999) allow for more informed estimates. The newer fossils and the interpretation of their patterns of growth are described in the Chapter 4.

An acceptable philosophy of human growth must acknowledge the mammalian and primate foundations for the human pattern of growth. However, that philosophy must also allow for the evolution of variations on common themes, and the evolution of new stages of growth that may be unique to the human species. A robust philosophy of human growth must also account for the ecology to which the human species – indeed any species – is adapted. With this in mind, we may now proceed to a discussion of the evolutionary and ecological pressures that shaped the pattern of human growth.

4 Evolution of the Human Life Cycle

The previous chapter described the evolution of the human pattern of growth in terms of its mammalian and primate foundations. The emphasis was on form and function of anatomical structures such as the placenta, the brain, and the skeletal system and how these structures are developed during the stages of human growth. In this chapter the emphasis is placed on how the human life cycle evolved. The life cycle of any organism includes all the stages of growth, development, and maturation from conception to death. Major events in the evolution of the human life cycle influenced the prenatal stages of development. A brief review of some human prenatal growth and development was provided in previous chapters. For further information of discoveries made in the last century of research relating to primate fetal development readers may consult the review by Richtsmeier (2018). It is worth mentioning here one classic example of human differences in prenatal growth and development published by Schultz (1926). His sketches of the body proportions of hominoid fetuses are reproduced here as Figure 4.1. The human fetus "of the 4th month" has relatively shorter legs than the chimpanzee, orangutan, or gibbon. The accuracy of this difference assumes that Schultz estimated fetal development correctly for the nonhuman apes (see Figure 4.1 legend). Another difference in proportion, not noted by Schultz, is the size of the cranium relative to the face, which is larger in the human fetus than in the chimpanzee, orangutan, or gibbon.

The human differences in the pattern of body proportion development during gestation may be explained, in part, by the evolution of the human large and complex brain. Human newborns are bigger brained than any of the apes (Table 4.1). Growing the human brain requires major inputs of energy, other nutrients, and oxygen – all supplied via the placenta and the fetal circulatory system. That system

Table 4.1 Neonatal and adult brain weight and total body weight for the great apes and human beings. Adult body weight is the average of male and female weight. Data from Harvey et al. (1986).

	Neonatal mass (grams)			Adult mass (grams)		
Species	Brain	Body	Br/Bo ratio	Brain	Body	Br/Bo ratio
Pongo (orangutan)	170.3	1,728.0	0.10	413.3	53,000.0	0.008
Pan (chimpanzee)	128.0	1,756.0	0.07	410.3	36,350.0	0.011
Gorilla	227.0	2,110.0	0.11	505.9	126,500.0	0.004
Homo sapiens	384.0	3,300.0	0.12	1,250.0	44,000.0	0.284

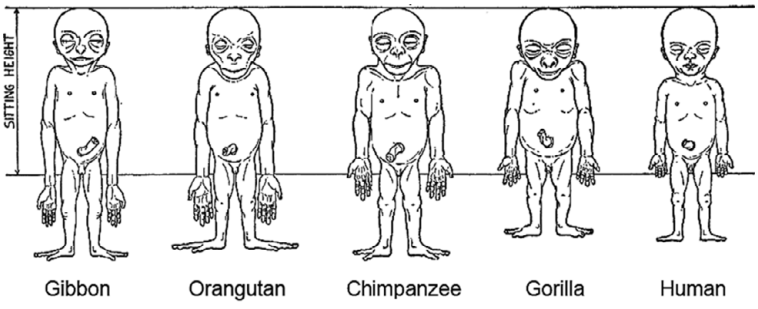


Figure 4.1 Schultz's sketches of the body proportions of hominoid fetuses. The original legend for this figure states, "All the figures have the same sitting height. The human fetus is the 4th month, the gorilla and the gibbon fetus correspond in development to the human fetus, but the chimpanzee and the orang fetus are slightly more advanced in their growth" (Schultz 1926, p. 465–466).

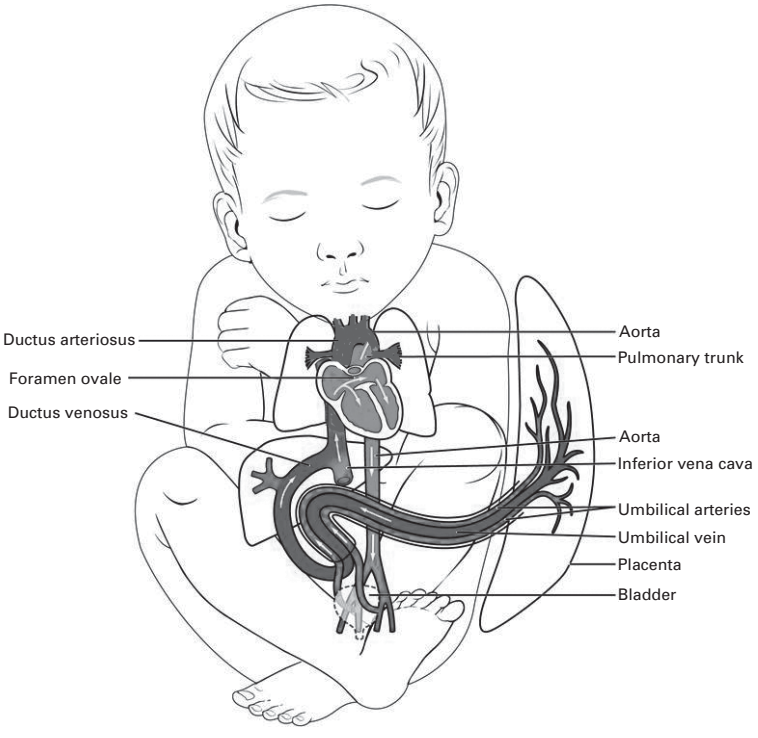


Figure 4.2 Human fetal circulation. The relative amount of oxygen in the fetal blood is greatest in the upper thorax, neck, and head; indicated by the red color of the vessels ascending from the heart. Blood flowing to the abdomen and legs is less well oxygenated; indicated by the violet color of the vessels descending from the heart. Image © Rice University, licensed under a Creative Commons Attribution 4.0 International License. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

is designed to shunt more blood to the trunk and the head than to the legs. In addition, blood in the fetal ascending aorta (toward the brain) has higher oxygen saturation than does the blood in the descending aorta (toward the legs, Figure 4.2). Additionally, the umbilical arteries carry some of the blood descending toward the leg back to the placenta. This pattern of fetal circulation is common to most mammals and is likely to be evolutionarily ancient. Because energy and other growth requirements are limited, there is a trade-off between growth of the upper body and the legs, which results in the mammalian fetal pattern of body proportion of large head relative to short legs. Combined with the more recently evolved metabolic demands of the large human fetal brain, the ancient circulatory pattern may leave the human fetal legs with a more acute reduced supply of oxygen and nutrients than other mammals, further slowing leg growth and development compared with more cephalic regions of the body. Additional details of these ape-human differences in body proportions and brain growth are given later in this chapter.

Human Biocultural Ecology

The pattern of human growth evolved in the context of the biological and social **ecology** of our ancestors. The term "ecology" is used here to refer to the relationship that an individual organism, or group of individuals of a species, has with its physical, biological, and social environment. At the core of any ecological system are two sets of behaviors; the first is directed toward how an organism acquires food and the second is directed toward how the organism reproduces. All organisms are alike in that they share behaviors related to what may be called simply "food and sex." Social mammals, including most primates, satisfy their needs for food and reproduction through a complex ecology of biological and social relationships with their environment. Human beings also share in this biosocial ecology and add to it a significant cultural component. Human beings are **cultural animals**, a point made briefly in the opening paragraph of the Introduction and elaborated upon here. Our species possess all the potentials and limitations of any living creature, and an added cultural dimension. Anthropologists have many definitions of human culture. Mid-twentieth-century definitions of culture included a trilogy of: (1) dependence on technology; (2) codified social institutions, such as kinship and marriage; and (3) ideology (White 1949). As mentioned in the Introduction, elements of the culture trilogy, such as dependence on technology and group-specific social behaviors, may be found in many other species of animals. Only in the human species do all three aspects of the cultural trio become so intensified, elaborated, and universal.

Contemporary theorists of culture focus their definitions on ideology, a point also mentioned in the Introduction. In its anthropological sense, ideology refers to a set of symbolic meanings and representations, which are particular to a society and through which its members view and interpret nature and justify their behavior. The ideology of a social group encompasses their beliefs, norms, and values, which are transmitted across generations by means of informal and formal teaching and learning (Boyd & Richerson 1985). A good deal of attention in the literature treats the

relationship of culture and ideology to human cooperation (Boyd & Richerson 2009; Henrich & Henrich 2007). There is no doubt that human beings can be superb cooperators and can also be highly competitive and combative. To encompass all aspects of human culture, Jonathan Marks shifts the emphasis away from cooperation and toward morality in his humorous and insightful chapter, "Human Evolution as Biocultural Evolution" (Marks 2015). Morality is, for Marks, part of ideology, that is, " ... the knowledge of right from wrong, and the injunction to do what's right" (Marks 2015, p. 129). "Doing what is right," of course, encompasses some of the worst examples of human behavior, as well as some of the most noble cooperation. In Marks' view, human moral ideology serves as the basis for the most uniquely human behaviors, such as symbolic language, religion, marriage and formal kinship systems, with kin terminology and its associated behavioral obligations.

Human moral ideology may be one basis for human culture, but there must also be a biological basis for the capacity for moral ideology. The pattern of human growth, development, and maturation is a key part of that biological basis and may be best understood from **biocultural** and anthropological perspectives.

To understand these perspectives is to understand humanness. Humans are a peculiar species of mammal: bipedal, omnivorous, relatively hairless, massively encephalized, intensely social, and reliant on complex learned behavior for survival. All people are genomically one species with a moderately diverse **genome** (defined below) that varies over clines, that is, geographic gradients in genomic frequencies, rather than well-demarcated boundaries (Livingstone & Dobzhansky 1962). Behaviorally, humans are extremely diverse. Individuals communicate by using thousands of different languages, are organized into societies with widely varying structures, and solve environmental problems with myriad technological solutions.

These human peculiarities have ramifications for how we approach human biology. Any understanding of human biology requires that we attend to the fact that humans are cultural beings. Anthropology is the primary discipline for the study of human culture. Anthropologists and others interested in human evolution, therefore, rely heavily on a biocultural perspective. This approach recognizes that human biology interacts with culture and can only be understood in the light of culture and, reciprocally, culture shapes human biology, human life history, and human growth. Current evidence indicates that human life course biology establishes the foundation for the capacity for human culture.

Biocultural Ecology of the Human Life Cycle

Anthropologists have become increasingly interested in explaining the significance of life cycle characteristics of the human species. This is because the human life cycle stands in sharp contrast to other species of social mammals, even other primates. Several of these contrasts, such as the childhood and adolescence stages of growth and development, were described in previous chapters. Any theory of human growth needs to explain how and why humans successfully combined these novel growth stages, along with a suite of other unusual features such as relatively helpless

newborns, a short duration of breast-feeding coupled with a vastly extended period of offspring dependency, delayed onset of reproduction, and relatively short birth intervals. In addition, humans have unusual secondary sexual characteristics, such as the peculiar distribution of both hair and fat in women and men, and women have a cessation of fertility at menopause followed by one or more decades of vigorous and productive social-economic life. Human men do not experience the reproductive cessation of the menopause but may have fertility decline coupled with vigorous and productive social-economic lives after age 50 years. A central question is, did these characteristics evolve as a package or as a mosaic? The present evidence suggests that the stages and events of the human life cycle evolved as a mosaic and may have taken form over more than a million years.

Life History and Stages of the Life Cycle

Life history theory may be defined as the study of the strategy an organism uses to allocate its energy toward growth, maintenance, reproduction, raising offspring to independence, and avoiding death. For a mammal, it is the strategy of when to be born, when to be weaned, how many and what type of pre-reproductive stages of development to pass through, when to reproduce, and when to die. Living things on earth have greatly different life history strategies and understanding what shapes these histories is one of the most active areas of research in whole-organism biology. Listed in Table 4.2 are several of the more important life history traits and trade-offs of animals, especially mammals.

Human life history, with nearly two decades of infant dependency, extended childhood, juvenile and adolescent stages prior to social and sexual maturation, has long been considered advantageous for our species because it provides:

1. An extended period for brain development.
2. Time for the acquisition of technical skills, e.g., tool making and food processing.
3. Time for socialization, play, and the development of complex social roles and cultural behavior.

These statements are standard "textbook" rationalizations for the value of the pattern of human growth. They emphasize the value of learning, an idea that Herbert Spencer (1820–1903), English philosopher, biologist, anthropologist, sociologist, and political theorist popularized (Spencer 1886), but which goes back to the dawn of written history (Boyd, 1980). Learning as the reason for the evolution of several prolonged life stages prior to maturation was nicely summarized by Dobzhansky: "Although a prolonged period of juvenile helplessness and dependency would, by itself, be disadvantageous to a species because it endangers the young and handicaps their parents, it is a help to man because the slow development provides time for learning and training, which are far more extensive in man than in any other animal" (Dobzhansky, 1962, p. 58). The learning hypothesis for human ontogeny was also invoked by Allison Jolly (1937–2014), author of *The Evolution of Primate Behavior*. She wrote that " . . . human evolution is a paradox. We have become larger, with long

Table 4.2 Life history traits and trade-offs. This is a partial list of the most important traits. The list is based on the discussion in Cole (1954) and Stearns (1992), who provided additional traits.

Traits
1. Size at birth
2. Brain size
3. Growth patterns
• Number of life cycle stages
• Duration of each stage
4. Age at eruption of first permanent molar
5. Rate of maturation
• Age at first reproduction
• Age of last reproduction
6. Size at maturity
7. Number and sex ratio of offspring
8. Reproductive investment in each offspring
9. Length of life
• Rate of aging/senescence
• Age at death
Trade-offs
1. Current reproduction vs. future reproduction
2. Current reproduction vs. survival
3. Number vs. size offspring
4. Parental reproduction vs. growth
5. Brain size vs. body size
6. Parental health vs. offspring growth
7. Parental vs. offspring reproduction

life and immaturity, and few, much loved offspring, and yet we are more, not less adaptable." In an attempt to resolve the paradox of human evolution and our peculiar life history, Jolly concludes in the next sentence that "mental agility buffers environmental change and has replaced reproductive agility" (Jolly 1985, p. 44).

Jolly's reference to reproductive agility meant that we are a reproductively frugal species compared with those that lavish dozens, hundreds, or thousands of offspring on each brood or litter. Later in this chapter it is proposed that the human species is indeed frugal in reproductive effort (the proportion of the total energy budget of an organism that is devoted to reproductive processes), but not because we tend to gestate and give birth to one offspring at a time. Rather, the reproductive effort (RE) of any single women is low because of a complex of biocultural capacities and behaviors that involve contributions of labor and energy from many social group members toward the mother and toward the gestation, birth, growth, and development of each new group member. With this social input humans also have a relatively low wastage of offspring (deaths both before and after birth) compared with many other mammals and this also contributes to human reproductive frugality.

Human reproduction is best referred to as **biocultural** reproduction (defined later in this chapter).

But a paradox remains, for the learning hypothesis does not explain how the pattern of human growth evolved. It does not provide a causal mechanism for the evolution of human growth. Rather, it is a tautological argument for the benefits of the simultaneous possession of brains that are large relative to body size, complex technology, and cultural behavior. Specifically, the learning hypothesis does not answer the following questions: (1) Why not produce more offspring, instead of few mentally agile offspring? (2) Why do our offspring take so long to reach reproductive age? (3) Why is our path of growth and development from birth to maturity so sinuous, meandering through alternating periods of rapid and relatively slow growth and development? (4) Why do human women, and men, often live for many years past the age of reproduction? A theory of human growth must answer these and other questions about human life history traits and events, and their timing during the life cycle.

The Evolution of Ontogeny

Ontogeny refers to the process of growth, development, and maturation of the individual organism from conception to death. It is virtually axiomatic that every species has its own unique pattern of ontogeny (Bonner 1965; Gould 1977). Behind this truism lies what may be the "secret" to life, hidden in the process that converts the fertilized ovum, with its full complement of deoxyribonucleic acid (DNA), into a multicellular organism composed of hundreds of different tissues, organs, behavioral capabilities, and emotions. During human evolution, the form and function of our ancestors' structural and regulatory DNA was reworked to produce the genomic basis for the ontogeny of the human species. The literature is replete with proposals for how the reworking occurred. One tradition in the study of human evolution looks for a single major cause or process. It has been argued that humans evolved when we became big-brained apes, terrestrial apes, killer apes, hunting apes, aquatic apes, tool-making apes, symbolic apes, monogamous apes, food-sharing apes, and, even, apes with ventral–ventral copulatory behavior. None of these, or any other single factor hypothesis, proves to be helpful to understand human evolution, for a nonhuman primate exception can always be found. Another tradition looks instead at the pattern of ontogeny. John T. Bonner (1920–2019) in his book *Size and Cycle* (1965) developed the idea that the stages of the life cycle of an individual organism, a colony, or a society are " . . . the basic unit of natural selection." Bonner's focus on life cycle stages followed from the research of several nineteenth- and twentieth-century embryologists who proposed that speciation is often achieved by altering rates of growth of existing life stages and by adding or deleting stages. Bonner stated that we should not think in terms of organisms with a life cycle, but rather think of organisms as life cycles. "The great lesson that comes from thinking of organisms as life cycles is that it is the life cycle, not just the adult, that evolves. In particular, it is the building period of the life cycle – the period of development – that is altered over

time by natural selection. It is obvious that the only way to change the characters of an adult is to change its development" (Bonner 1993, p. 93).

A history of research on life cycle evolution was published by Stephan J. Gould (1941–2002) in the book *Ontogeny and Phylogeny* (1977). Gould handily summarizes the mechanisms for biological change over time by stating, "Evolution occurs when ontogeny is altered in one of two ways: when new characters are introduced at any stage of development with varying effects upon subsequent stages, or when characters already present undergo changes in developmental timing. Together, these two processes exhaust the formal content of phyletic change . . . " (p. 4). Gould contends that it is the second process that accounts for human evolution. This process is called **heterochrony**. Quoting Gould again, " . . . this book is primarily a long argument for the evolutionary importance of *heterochrony* – changes in the relative time of appearance and rate of development for characters already present in ancestors" (p. 2, author's italics). In the discussion that follows, the focus will be on whether the human life cycle evolved by altering "characters already present" in our ancestors or whether it evolved by introducing new characters. The conclusion is that both were involved but that the evidence argues strongly in favor of childhood and adolescence as new characters in the human life cycle.

Gould explains that there are several types of heterochronic processes, but only one accounts for human evolution. This is **neoteny**, defined in the glossary of Gould's book as "Paedomorphosis (retention of formally juvenile characters by adult descendants) produced by retardation of somatic development." In a subsequent publication, Gould provides a somewhat more readable definition: "In neoteny rates of development slow down and juvenile stages of ancestors become adult features of descendants. Many central features of our anatomy link us with the fetal and juvenile stages of [nonhuman] primates . . . " (Gould 1981, p. 333). Following Gould, we must add neoteny to the list of single-cause hypotheses as the reason, or at least the mechanism, for human evolution.

Another heterochronic process, called **hypermorphosis**, was favored by other researchers as the mechanism for human evolution. Hypermorphosis may be defined as an extension of the growth and development period of the descendant beyond that of the ancestor. The differences between neoteny and hypermorphosis may be summarized as follows. Neoteny is a slowing down of the rate of development. Neoteny produces an adult descendant that retains the immature body shape, and even the immature behavioral characteristics, of its ancestor. Hypermorphosis is a prolongation of the time for development, and this extra time for ontogeny produces a descendant with features that are hypermature compared with the ancestor.

The most clear, logical, and convincing case against any single heterochronic process as the cause of human evolution was published by Shea (1989). Shea rejects both neoteny and hypermorphosis as a "grand unification theory" for all human growth and evolution. Shea was not anti-neoteny or anti-hypermorphosis per se, rather he argued that a variety of heterochronic and allometric processes are responsible for human evolution. Allometry is the study of the relationship of body size to shape. It has applications to anatomy (illustrated in Figures I.3 and I.4), physiology (e.g., body size

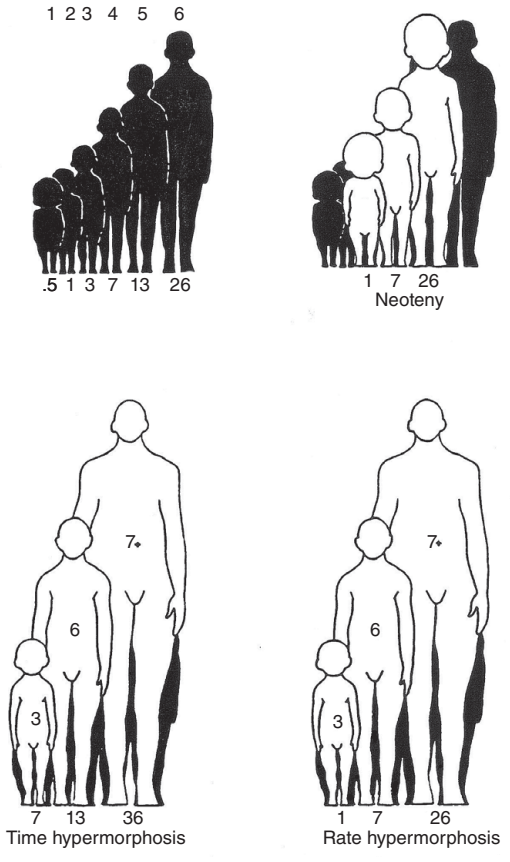


Figure 4.3 Silhouettes of size and shape change during human growth. Numbers under silhouettes indicate age in years. Numbers above or on silhouettes indicate relative shape. Top left: actual size and shape change during normal human development. Top right: neoteny, note that at adult size shape 3 is still maintained. Bottom left: time hypermorphosis, the growth period is extended to 36 years yielding a peramorphic giant (size and shape of the descendant beyond that of the ancestor). Bottom right: rate hypermorphosis, growth ends at age 26 but proceeds at a faster rate producing another peramorphic giant. Note that in both cases the adult shape at 7 is outside the range of normal development. From Shea (1989) with permission

and metabolic rate), and behavior (e.g., leg length and running speed). Much of the work of D'Arcy Thompson discussed in the Introduction is based on allometry.

In Figure 4.3 are illustrated Shea's estimates for body size and shape due to neoteny and two types of hypermorphosis. Shea's estimates begin with a real size and shape morphology for a human infant or child. Neither neoteny nor hypermorphosis acting as a single process can produce a biologically reasonable human allometry of adult size and shape from the initial morphology. Shea found that other heterochronic processes, including acceleration and hypomorphosis, also fail. According to Shea, there are at least two reasons why no single type of heterochronic process can account for human evolution. The first reason is that, in

agreement with Schultz, Shea points out that " . . . we [humans] have extended all of our life history periods, not merely the embryonic or juvenile ones . . . " (1989, pp. 84–85). We are not "permanent children" or juvenilized apes, and while we take longer to reach sexual maturity than any primate we do not become sexually mature while in a juvenile stage. Rather, we humans have our own adolescent and adult stages of life. Furthermore, the adult stage has been prolonged beyond that found in any other primate species. The consequences of that adult stage prolongation, such as menopause in women, are not discussed by Shea, but are discussed later in this chapter.

From Heterochrony to Evo-Devo

The heterochrony literature is enormous, and the terminology is bewildering, with article titles such as "Paradox of peramorphic paedomorphosis" (Godfrey & Sutherland 1996) and "Heterochrony repolarized" (Bonett et al. 2014). Despite their titles both are important contributions to the field. The latter article is focused on salamanders, the favorite group of vertebrate organisms for heterochrony research. This is because salamander life cycles are undisputed examples of evolution via changes in developmental timing. The most celebrated species is the axolotl (*Ambystoma mexicanum)*, which typically becomes sexual mature while still retaining the external gills typical of larval amphibians. The axolotl is a good example of neotony in ontogeny; humans are not a good, or even appropriate, example (Figure 4.4).

More than 20 years ago, I reviewed the pros and cons of heterochrony as an explanation for human evolution (Bogin 1997). The 2nd edition of this book, published in 1999, included an updated discussion, mostly focused on the limited support for a role of neoteny in the evolution of human anatomy, physiology, or life history. Today, there is still interest in heterochrony as a process involved in human evolution (Somel et al. 2014) and some scholars and journalists cling to neoteny as the mechanism for human evolution (Tuomisto et al. 2018). One review of the place of heterochrony in evolutionary biology concluded that, " . . . heterochrony is an effective descriptor of many patterns of morphological variation among related taxa [but] . . . an exclusive focus on heterochrony is unwarranted except in isolated cases . . . " (Hanken 2015, p. 108). Hanken's review emphasized that newer discoveries of molecular-genetic mechanisms of development, which are distinct from heterochrony, provide a more direct and complete understanding of the evolution of morphological diversity and life history variation. The new discoveries helped to create the field of Evo-Devo, or evolutionary developmental biology.

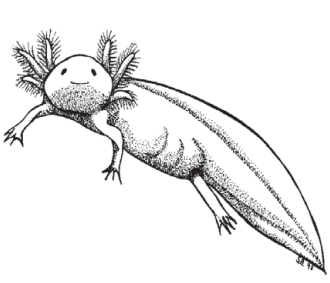
Evo-Devo is not a new idea; nineteenth-century biologists were looking to embryos and the way they developed to understand evolution. It was Haeckel (see Chapter 1) who, in fact, coined the term "heterochrony." Evo-Devo is more than heterochrony. Sean Carroll is one of the leaders of Evo-Devo and his essay *The Origins of Form* (2017)[1] provides a sensible overview of the field. Carroll explains

[1] www.naturalhistorymag.com/features/061488/the-origins-of-form

Neoteny

ne·ot·e·ny – noun

1. Retention of juvenile characteristics in the adults of a species, as among certain amphibians.



2. The attainment of sexual maturity by an organism still in its larval stage.

— ne´o·ten´ic or ne·ot´e·nous adjective

[New Latin neotenia : NEO- + Greek teinein, ten-, to extend. See tenesmus.]

te·nes·mus – noun

A painfully urgent but ineffectual attempt to urinate or defecate.

[Medieval Latin tenesmus, variant of Latin tenesmos, from Greek teinesmos, from teinein, to strain, stretch.]

Figure 4.4 Definitions and etymology of the term "neoteny." The sketch of the amphibian represents the axolotl, which often becomes sexually mature while still retaining the external gills typical of larval amphibians. The cartoon of Betty Boop illustrates some human features which are sometimes labeled as neotenous, such as a large head, short arms and legs relative to total height, and clumsy, childlike movements. Definitions from The American Heritage Dictionary, 3rd edition. Betty Boop © King Features Syndicate.

that until the late twentieth century biologists thought that development was hopelessly complex. Each species of animal, it seemed, required its own explanation for development. Some shared genetic basis might have a role for closely related species, but the differences in form of flies, flamingos, and foxes must arise from distinct genomes, or so it was believed. Moreover, rules of development would need to differ between such different forms. It was proposed, for example, that the eyes of different species of invertebrates and vertebrates had evolved independently, reinvented as it were between 40–65 times, for each species.

One discovery that changed this thinking was the PAX6 gene mentioned in the Introduction. Walter Gehring (1939–2014) and colleagues (Gehring & Ikeo 1999; Halder et al. 1995) discovered that despite the differences in structure and optical properties of the compound eyes of flies, crabs, and other arthropods vs. the camera-type eyes of vertebrates, all animals with eyes have the PAX6 gene. PAX6 belongs to

a family of genes that play a critical role in the formation of tissues and organs during embryonic development. The members of the PAX gene family are also important for maintaining the normal function of certain cells after birth. To carry out these roles, the PAX genes provide instructions for making proteins, one of which is called the Paired box protein, that attach to specific areas of DNA and help control the activity (expression) of other genomic regions. PAX proteins are called **transcription factors** and fulfill one of the roles of the regulatory genes proposed by King and Wilson (1975), see Introduction.

During embryonic development, the PAX6 protein activates genes involved in the formation of the central nervous system, including eyes, the brain and spinal cord, and the pancreas (Shaham et al. 2012). Within the brain, the PAX6 protein is involved in the development of a specialized group of brain cells that process smell (the olfactory bulb). Mutations in the PAX6 gene and the protein it encodes produce a variety of eye pathologies.[2]

Basic body building genes such as PAX6 have been in animal genomes for more than 500 million years and there has not been an evolution of new genes. So, how do a few basic genes result in so much diversity in eye forms and even greater diversity of body forms? The answer is that genes do not directly cause growth and development. Rather, the expression of a genetically inherited pattern of growth is regulated by many proteins that genes produce, and the entire process is mediated by several biological systems, especially the endocrine and neurological systems. This regulation effects the number of body segments, such as paired legs in insects, repeats of color patterns, such as the zebra's stripes, and the size and shape of body parts by altering the amount of growth as well as its tempo (the timing of growth and development events) and intensity (the amplitude and duration of a growth or development events).

A marvelous example of the interaction of genes, proteins, and the endocrine system may be seen in the action of **homeodomain genes** (formally called **homeobox genes**) and **Hox genes**. The description and elucidation of homeodomain and *Hox* genes is one of the most important advances of molecular biology of the past four decades. Gehring and colleagues (McGinnis et al. 1984) first reported the structure of homeodomain genes and demonstrated their role in the development of fruit flies (*Drosophila melanogaster*). The homeodomain is a sequence of about 180 DNA base pairs that codes for a 60 amino acid segment of a protein. Homeodomain sequences are found in all eukaryote organisms so far examined. These highly conservative DNA sequences – the same homeodomain sequences are found in organisms as diverse as hydra, nematodes, all arthropods (the group that includes insects), and all chordates (the group that includes human beings) – produce proteins that regulate the expression of other genes " ... and control various aspects of morphogenesis and cell differentiation" (Mark et al. 1997, p. 421). *Hox* genes are a category of homeodomain genes (e.g., PAX6) that encode transcription factors (Holland &t

[2] https://ghr.nlm.nih.gov/gene/PAX6#

Garcia-Fernàndez 1996), which are proteins that initiate and regulate the conversion of the DNA code to the RNA sequence that is used to make amino acid polypeptide chains.

Homeodomain genes seem to have their greatest impact during the earliest stages of development. The proteins that homeodomain genes produce are needed to regulate the expression of other DNA to " . . . sculpt the morphology of animal body plans and body parts" (Carroll 1995, p. 479). The DNA affected by homeodomain proteins will, in turn, produce other proteins that mediate cellular differentiation, growth, and development. These "down-stream" proteins do not act alone. Some of them must combine via a process called molecular zipping before they have any effect on a given segment of DNA (McKnight, 1991). These and other proteins need an appropriate environment to have any effect. The biochemical environment of the egg cell, and a bit later in time the wombs of seahorses, reptiles, and mammals, provide a host of factors needed for growth, including nutrients and hormones.

In multicellular animals, homeodomain and Hox genes act to delimit the relative positions of body regions, for example the head, thorax, and abdomen of insects, or the general body plan and limb morphology of vertebrates. The twentieth-century British theoretical biologist Joseph Henry Woodger (1894–1981) coined the term *bauplan*, which in German means floor plan or blueprint. In biology it means the basic anatomical architecture of related organisms, that is, the, " . . . suite of characters shared by a group of phylogenetically related animals at some point during their development" (Willmore 2012, p. 219). The idea of a *bauplan* may be traced back to Aristotle, but the nineteenth-century German embryologists von Baer and Haeckel made the evo-devo connections. Perhaps this German history is why Woodger chose a German word to encapsulate his idea. The nineteenth- and early twentieth-century research led to quantitative and qualitative descriptions of what the biologists believed must be true about the underlying regulation of development. The discovery of *Hox* genes of the fruit fly *Drosophila* (Figure 4.5) and of the human species (Figure 4.6) are the empirical support of the *bauplan* of animal life.

Hox genes need to be carefully regulated. A small perturbation in regulation can result in extra wings or legs instead of antennae, which might be lethal to fruit flies in the wild. To assure correct development of the phenotype the groundwork for the fly's body plan is established in the fertilized egg by organizing cells into a head end vs. a tail end. The structures of the head, thorax, and abdomen are gradually refined into broad sections, then sub-sections, and finally into functional body segments. This process involves different classes of genes with increasingly narrow and specific patterns of expression. Broadly speaking, earlier-acting groups regulate later-acting groups in a sort of molecular domino effect. *Hox* genes are turned on in specific places through the activity of genes in this cascade.

The diversity of the form, function, and number of appendages of arthropods (lobster claws vs. antennae, spider legs, millipede "feet," etc.) is derived from repetition of activation of their segments. This same regulation of repetition may be seen in species of vertebrates, which differ fundamentally in the number and kind of vertebrae (cervical, thoracic, lumbar, and sacral). Humans have 12 paired ribs, each pair

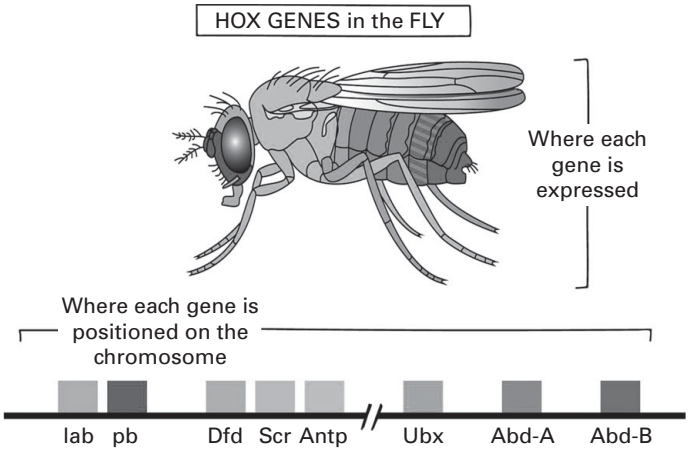


Figure 4.5 Genomic organization and colinear expression patterns of *Drosophila* HOX genes. The figure indicates eight major homeotic genes in flies. The upper part of the diagram shows where each gene is most strongly expressed in the mature fly, while the lower part of the diagram shows where the genes are located on the chromosome. The colors indicate correspondence between HOX region and body region. The order of HOX genes on the chromosome mirrors, approximately, their order of expression along the head-tail axis of the fly. The break mark (//) in the chromosome indicates that these two clusters of genes are separated by a long intervening region that's not shown. Image credit: from https://commons.wikimedia.org/wiki/File:Hoxgenesoffruitfly.svg modified by www.khanacademy.org/science/biology/developmental-biology/signaling-and-transcription-factors-in-development/a/homeotic-genes, public domain. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

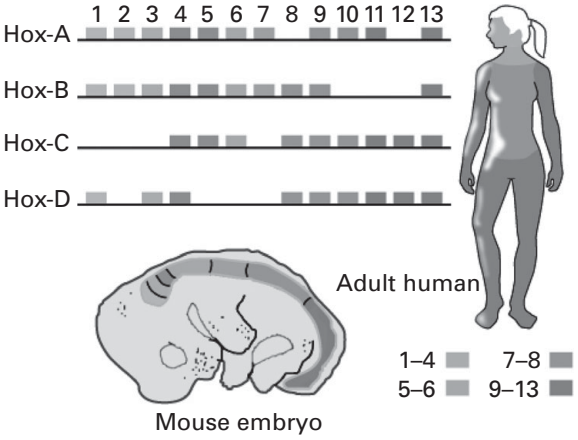


Figure 4.6 Genomic organization and colinear expression patterns of mammalian Hox genes. A schematic representation of the four mouse Hox complexes is shown, which are inferred to be nearly identical for the human species. As in flies, this order roughly maps to the parts of the body whose development is controlled by each gene. From: www.khanacademy.org/science/biology/developmental-biology/signaling-and-transcription-factors-in-development/a/homeotic-genes, public domain. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

articulated with its thoracic vertebra. Snakes have hundreds of rib-bearing vertebrae and essentially no neck. Humans typically have a vertebral formula of 7 cervical, 12 thoracic, 5 lumbar, and 5 sacral. About 10% of people have six sacral vertebrae and this often causes pain and pathology as it interferes with human bipedal locomotion. The other African apes (chimpanzees, bonobos, and gorillas) have a vertebral formula 7-13-4-6 as the norm, and occasionally one more or less vertebra in the thoracic, lumbar, or sacral region (Williams et al. 2016). The human-African ape differences attest to an evolutionary divergence in *Hox* regulation associated with the shift from quadrupedal locomotion and brachiation of the apes to human bipedalism.

A difference between invertebrates and vertebrates, such as mice and humans, is that *Hox* genes have been duplicated over evolutionary history and now exist as four similar gene clusters, labeled A through D in Figure 4.5. The duplicated *Hox* gene sequences evolved novel functions, for example in tetrapods (e.g., frogs, lizards, bats, and people), *HoxD* genes regulate the formation the limb and the number of digits of the forefoot (the hand of primates) and the rearfoot (Montavon & Soshnikova 2014).

Evo-Devo Is Not Enough to Explain Human Growth and Development

It may be fair to state that heterochrony is a part of the larger set of regulatory processes encompassed by evolutionary development biology. Homeodomain and *Hox* gene regulation explain a large percentage of phenotypic variation between and within major groups of animal life, but not all variation. Rarely discussed by researchers of heterochrony or Evo-Devo is that there is another process by which evolution works. To requote Gould, "Evolution occurs when ontogeny is altered in one of two ways": the first is " . . . when new characters are introduced at any stage of development with varying effects upon subsequent stages . . . " Heterochrony and Evo-Devo can do a lot to rearrange existing body parts and behaviors, but they do not create truly new characters. Much evidence supports the hypothesis that the evolution of human brains, language, ideology, and culture are the result of the introduction of new life stages into the general pattern of primate growth and development. Between birth and adulthood, the new life stages are childhood and adolescence.

As discussed in previous chapters, human growth and development from birth to reproductive maturity may be characterized by five stages: (1) infancy, (2) childhood, (3) juvenile, (4) adolescence, and (5) adulthood. It was explained in Chapter 2 how each stage may be defined by rate of growth, by characteristics of the dentition, by changes related to methods of feeding, by physical and mental competencies, and by maturation of the reproductive system and sexual behavior. It is possible that *Hox* and homeodomain gene regulation are part of the process to make new life stages. There seems to be little information on this possibility, but one study found that homeodomain proteins serve as transcription factors to regulate the metamorphosis from larva to pupa in the silkworm (*Bombyx mori*, Deng et al. 2012).

Humans do not undergo the type of metamorphosis of the insects, but the transitions from one human life stage to the next are no less remarkable in terms of

biological, psychological, and behavioral changes. The following discussion focuses on the stages of human childhood and adolescence and why and when these were added to the human pattern of growth.

Human Childhood

Human beings are not the "permanent children" of a neotenized ape, but we do pass through a childhood stage of growth and development between the ages of about 36 to 83 months after birth. The onset of the childhood stage is marked by a change in growth rate, from the rapid decline during infancy to a period of more steady growth rate, the eruption of all the deciduous teeth, weaning (the end of breast-feeding), and maturation of both new motor and cognitive skills (Figure 2.5, Table 2.1). Though weaned, children are still dependent on older individuals for feeding and protection. The end of childhood is proclaimed by several biological events; eruption of the first permanent molars and incisors, the end to brain growth in weight, attainment of adult style locomotion, and adrenarche (adrenal gland maturation, described in later in this chapter). Childhood's end is also associated with new levels of cognitive maturity, the so-called "5-to-7-year-old shift" and for some children the mid-growth-spurt. Some of the milestones that mark the beginning and end of childhood were discussed in Chapter 2, but in the present chapter these, and other events, are considered in terms of the evolution of human life history.

Weaning

Childhood begins after the infant is weaned. Weaning is defined here as the termination of lactation by the mother (other researchers may define weaning as the process of shifting from lactation to eating solid foods). In human societies the age at weaning varies greatly. Industrialized societies provide a poor indication of weaning age because bottle feeding and the manufacture of "baby foods" allow either early termination of breast-feeding or no breast-feeding at all. Pre-industrialized human societies provide a better indication of the age at weaning, and hence the transition from infancy to childhood. One survey of such a societies found that the termination of breast-feeding occurs at a median age of 36 months (Dettwyler 1995). Another review of the age at human weaning (Lee et al. 1991) found that in so-called "food enhanced" societies, those where nutritional intake is good, weaning took place as early as nine months of age. In "food limited" societies, where chronic undernutrition occurs, weaning took place at a median age of 36 months.

There are two fascinating corollaries of this comparison. The first is that in both the "food enhanced" and the "food limited" societies the mean weight of weaned infants was about the same, 9.0 kg and 9.2 kg respectively, or about 2.7 times birth weight (Lee et al. assume a mean birth weight of 3,400 grams for full-term human beings). The second is that some solid foods are introduced into the diet when the infant achieves about 2.1 times birth weight. Lee and colleagues (Lee et al. 1991;

Bowman & Lee 1995) compared the human data with data from 88 species of large-bodied mammals (32 nonhuman primates, 29 ungulates, 27 pinnipeds). They found that for all these species solid food is introduced, again, at about 2.1 times birth weight, but weaning takes place when the infant achieves between 3.2 to 4.9 times birth weight. For all primates the mean value is 4.6 times birth weight, with a range from 2.3 for the talapoin monkey (*Microcebus talapoin*) to 9.4 for the gorilla (*Gorilla gorilla*). The other great apes average at the following multiples of birth weight: *Pan troglodytes* (chimpanzees), 4.9; *P. paniscus* (bonobos), 6.1; *Pongo pygmaeus* (orangutans), 6.4.

Humans are like other mammals in that we introduce solid foods at about 2.1 times birth weight. However, humans are unlike other mammals, even other species of primates, in that pre-industrial and traditional societies, including "food limited" groups such as African hunter-gatherers, wean at a relatively early stage of growth and development – before reaching 3.0 times birth weight. Even more unexpected is that human infants are weaned years before the first permanent tooth erupts. For most other primates, and virtually all other mammals, weaning is broadly coincident with first molar eruption (Smith et al. 1994). Both weaning and molar emergence may be considered as processes (Smith et al. 2013). It is important to understand when event vs. process is being used by researchers. In this book both weaning and molar eruption are treated as events with weaning being the termination of feeding by lactation and molar emergence being the appearance of the tooth crown through the gum.

The apes both conform to the rule of weaning at first molar (M1) eruption in the case of gibbons (*Hylobates*), siamangs (*Symphalangus*), and gorillas, and are important exceptions to the rule in the case of chimpanzees, orangutans (*Pongo*), and humans (Table 4.3).

Chimpanzees and orangutans wean up to three years after M1 eruption and humans wean about three years before M1 eruption. A likely reason for "late" weaning is that young chimpanzees and orangutans with M1 are not able to acquire and process enough food by their own foraging to meet their nutritional needs (Smith et al. 2013; Van Noordwijk et al. 2013). Van Noordwijk and colleagues emphasize the immaturity of locomotor skills as a primary reason for the feeding dependence of the

Table 4.3 Weaning and molar emergence time in hominoids. All ages are in years. From Humphrey (2010).

Ape species	Age at weaning	Age of first molar emergence
Hylobates lar	1.8	1.8
Symphalangus syndactylus	2.2	2.3
Pan troglodytes	5.0	3.1–4.1
Pongo pygmaeus abelii	7.0	3.5–4.6
Homo sapiens	2.4	4.7–7.1
Gorilla gorilla beringei	3.2b	3.2

young orangutan or chimpanzee. Human young at five to six years of age will have M1 eruption but cannot acquire and process enough food to survive for the same reason of motor immaturity as well as dental immaturity, and a three-year-old is even more immature. How then is human weaning by age 36 months possible?

Human mothers can wean their infants at this early stage of growth because the mother, or other people, will provide her child with specially prepared post-weaning foods. These are called complementary foods and are sufficiently processed to make them soft enough so that human children can masticate them (Sellen 2006, 2007). Children require specially prepared foods due to the immaturity of their deciduous dentition, often called "milk teeth." These teeth have thin enamel and shallow roots compared with the permanent dentition. Smith and colleagues (Smith 1991; Smith et al. 1994) report that given this dental morphology, young mammals with only the deciduous dentition cannot process the adult-type diet. With eruption and occlusion of the first permanent teeth the infant mammal can process the adult diet and can be, at least dentally, independent in terms of feeding. The young mammal moves either to adulthood (most mammals have only two postnatal growth stages) or to the juvenile stage (social mammals).

When human infancy ends, children are not only dentally immature, but also the small size of their digestive tracts relative to that of the adult necessitates a special diet. This diet must be dense in **essential nutrients** (those not manufactured in the human body, e.g., vitamin A, iron, certain lipids, and amino acids) and dense in energy (kilocalories from carbohydrates, lipids, and proteins). This need can be appreciated most acutely when it is not met. Béhar (1977) studied the causes of growth retardation and undernutrition of children living in rural villages in Guatemala. He found that the children had access to food, but this was mostly the traditional adult diet of corn and beans. During my fieldwork experience in urban and rural Guatemala between 1978 and 1998, I witnessed 3-4 years old children receiving these foods, and they were specially prepared by being cut into small pieces, mashed to make softer, and with added liquid, usually water or coffee. Children of that age could not eat enough of these foods to receive all the needed essential nutrients and energy. Nutrition surveys carried out in Mexico and Guatemala between the 1960s and 1990s found that low income rural people consumed only 75–80% of the total food energy needed for adequate physical growth (Bogin 2012). The outcomes measured by both Béhar and by me were growth failure and increased susceptibility to infectious disease for most of the children due to chronic undernutrition (Figure I.2, the growth curves for rural Guatemala children).

Bailey and colleagues (Bailey et al. 1984) studied the growth of more than 1,000 infants, children, and juveniles living in 29 villages in northern Thailand. The participants in the study lived in rural agricultural villages, with rice as the basic subsistence crop. The participants were between the ages of six months and five years old at the start of the study, and they were measured for height, weight, and several skinfolds about every six months for five years. It was found that, compared with local or international reference standards for growth, the rural Thai children and juveniles were delayed in growth for all the dimensions studied. Careful

consideration of several factors that may influence growth, including disease or the lack of specific nutrients, such as protein, vitamin A, or iron, showed that the delays in growth were not due to any one of these; rather the delays were due to a deficiency in the total intake of calories. The most dramatic fall-off in growth occurred at 18 months of age, which corresponds with the average age at weaning in these villages. Weaning foods were usually watered-down versions of adult foods. Although there were no food shortages in the villages, Bailey and colleagues reasoned that the small gastrointestinal tracts of the weaned infants and young children may not have been capable of digesting enough food to meet their caloric demands for maintenance and growth of the body. The work of Béhar in Guatemala, Bailey et al. in Thailand, and many other similar studies show that without the use of appropriate weaning foods children will suffer calorie insufficiency leading to undernutrition, developmental delays, and growth retardation.

Complementary feeding is a human strategy to meet the energy and nutrient needs of vulnerable infants and children. Complementary feeding may begin between the ages of 6 and 9 months, depending on infant growth rates and energy needs that are not met by breast-feeding, as well as cultural practices relating to infant care. Complementary foods encompass a wide range of liquids and solids, based on fruits, vegetables, carbohydrates, and animals. Sellen (2001) surveyed ethnographic and demographic reports published between 1873 and 1998 and found that the modal age of introduction of both liquids and solids was 6 months, but varied from birth to 12 months in particular societies. In some societies, insects are an especially important source of complementary food, as reported by (Schiefenhövel & Blum 2007). Per unit weight, insects provide protein and fat comparable to vertebrates (Dufour 1987).[3]

Feeding the Greedy Brain

Another reason that children need a special high-energy diet is due to the rapid growth of their brain relative to the body (Figure 2.8). Indeed, body growth is constrained to a steady 5–6 cm per year from about age 3.0 to 7.0 years as an energy trade-off to channel more available resources to the brain. In this regard, the research of William Leonard and colleagues (Leonard et al. 2007; Leonard & Robertson 1992), discussed briefly in Chapter 2, is most relevant. Leonard and colleagues estimated that due to rapid brain growth; "A human child under the age of 5 years uses 40–85 percent of resting metabolism to maintain his/her brain [adults use 16–25%]. Therefore, the consequences of even a small caloric debt in a child are enormous given the ratio of energy distribution between brain and body" (Leonard & Robertson 1992, p. 191). In a related study, Leonard and Robertson (1994) also showed that the size of the human brain relative to total body size necessitates an energy dense diet. At all stages of life after birth, human beings have brains that are significantly larger than

[3] www.the-scientist.com/?articles.view/articleno/34172/title/why-insects-should-be-in-your-diet/

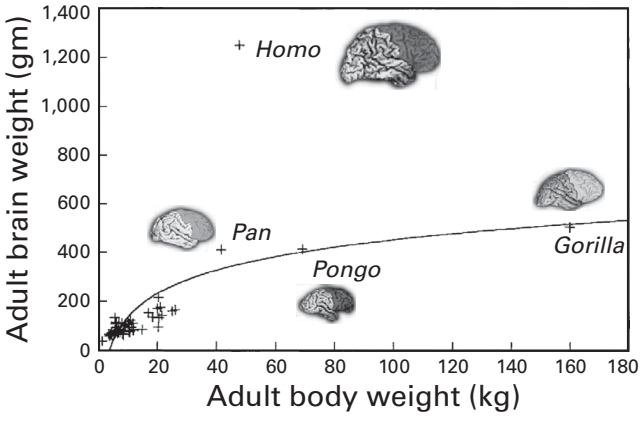


Figure 4.7 Adult body weight and brain weight plotted for 61 species of Cercopithcidae (Old World monkeys, apes, and people). The curve is logarithmic regression fit to the data for all species. The drawing of brains shows the relative size by species. Each part of the human brain enlarged during evolution, especially the size of cerebral cortex. Data from Harvey et al. (1986), brain images reprinted by permission from Springer Nature: Passingham, R. The frontal cortex: does size matter?. *Nat Neurosci* 5, 190–192 (2002). https://doi.org/10.1038/nn0302-190.

expected given the human body size (Figure 4.7). These large, metabolically active brains demand a larger percentage of energy than any other primate.

Big, fast growing, energy greedy brains are the primary reason for feeding dependency during childhood. In fact, recent studies show that total energy requirements peak during childhood, measured as resting metabolic rate per kg body weight, or as daily energy requirement expressed in grams of glucose per day per kg body weight (Kuzawa et al. 2014). The brain, which grows rapidly during infancy and childhood, is especially greedy for energy. As the infant's brain grows its energy demands eventually exceed the ability of mother's milk with additional complementary foods to meet that demand. At that point, infant feeding practices must shift to a new strategy of child feeding practices. According to the data presented by Kuzawa et al. (2014), the life history transition from infancy to childhood takes place when brain glucose uptake exceeds 100–110 g day^{-1} which occurs at an average age of about 3.0 years after birth – just the age at which a change in growth velocity takes place (Figure 2.8).

Aiello and Wheeler (1995) refined the relationship between human brain size, body size, and metabolic costs by noting that relative to total body size, adult human beings have an unexpectedly small gastrointestinal tract (gut) as well as an unexpectedly large brain (Figure 4.8). For children the relative disparity between brain and gut size is even greater. In contrast, the size of other organs, such as the liver, kidney, and heart are about as big as expected. Aiello and Wheeler showed that both brain tissue and gut tissue are "expensive," meaning that both types of tissue have relatively high metabolic rates. Aiello and Wheeler presented estimates of the percentage of total body basal metabolic rate for several tissues utilized by the typical 65 kg adult human male. For the brain the value is 16.1% and for the gut the value is

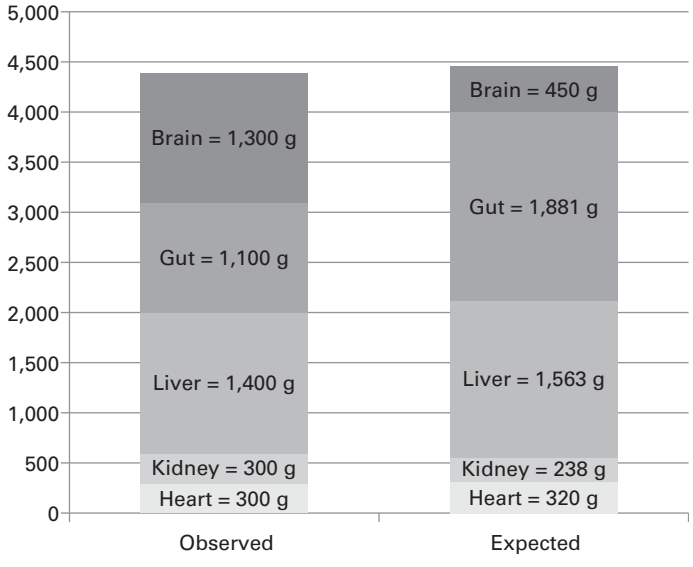


Figure 4.8 Expected and, observed weight of several hu,man organs for a 65 kg adult human male. The histograms provide a comparison of the weight of the heart, kidney, liver, digestive system (gut), and brain. The observed size of the human heart, kidney, and liver are about equal to the expected size. The human gut is much smaller than expected, while the human brain is much larger than expected. The expected sizes of the organs are based on the actual sizes of these organs in more than 20 species of monkeys and apes. The data are from Aiello and Wheeler (1995), where the details of data selection, sample sizes, and methods of analysis are described.

14.8%. Given these values, Aiello and Wheeler proposed that during human evolution the gut decreased in size as a trade-off allowing, in part, for expansion of brain size. Had the gut remained as large as expected for a primate of human size the metabolic costs would have been too great to also support a large brain. The trade-off in size between brain and gut means that humans have a total metabolic rate that is about average for a placental mammal of our size.

Even with this trade-off the large and metabolically expensive human brain still requires a constant supply of energy. A smaller than expected gut size for humans means that less total food can be processed in a given amount of time. This presents humans with the problem of eating just the right kind of food to meet the nutritional demands of brain and the rest of the body. Both Leonard and Robertson (1994) and Aiello and Wheeler concluded that the way human beings satisfy nutritional demands is to consume a diet that is nutrient dense, especially in energy, and easy to digest. Foods for human children need to be especially nutrient dense and digestible. Human beings meet these dietary requirements in two ways. The first is by selection of appropriate foods. Leonard and Robertson compared the diet of five human foraging societies – !Kung, Ache, Hiwi, Inuit, and Pygmies – to 72 nonhuman primate species and found that diet quality, in part measured by energy density, of the human groups was almost twice that of other primates of the same body size.

The human ability to include foods such as seeds, roots, and meat in the diet increases quality, as these are nutrient dense foods. Most other primate species rarely or never eat seeds or roots.

Some nonhuman primates do eat meat, but meat comprises less than 10% of their diet. Human foragers studied in the twentieth century were found to obtain at least 30% of their dietary energy from animal foods. This dietary pattern is ancient in that it is estimated that Paleolithic foragers living in western Europe between 15,000 and 20,000 years ago consumed about 30% of total energy from animal protein (Bogin 1998a, 2001). More ancient species of hominids seem to have eaten less animal protein, but the archaeological evidence indicates that they expended significant effort to acquire as much animal food as possible (Bogin 1998a, 2001).

The second way humans meet nutritional requirements is by processing food to extract, concentrate, and enhance its nutritional content. Humans, and human ancestors, expend great effort in food preparation, using a variety of culturally learned methods (i.e., recipes) to cook, combine, flavor, and detoxify natural ingredients (Pelto & Pelto 1983). Since at least the time of *Homo habilis* hominids have depended on technology, especially stone tools, to do this preparation. *H. erectus* added fire to its repertoire of technology. Fire, which may have been used as early as 1.4 million years ago (MYA), and was certainly controlled by 750,000 years ago, provided warmth, light, protection, and a new way to process foods. Where and how cooking was invented is a matter for speculation. Cooking, by roasting or boiling, increases the nutritional benefit of many vegetable foods by helping to break down the cellulose, which is indigestible by people. Fire may also have been used to open large seeds that resist even stone tools. Cooking, especially drying or smoking, helps to preserve foods for storage. Fire may also be used to obtain foods, for instance by driving game toward a convenient killing site. All of these uses of fire did not appear simultaneously, and many seem to be the invention of *H. sapiens* rather than *H. erectus*. What is certain is that the controlled use of fire was a significant addition to hominid technology, with profound consequences for nutritional status (Wrangham & Carmody 2010).

A third nutritional characteristic of human evolution was the development of cuisine, defined by George Armelagos (1936–2014) as, " ... a momentous event in history ... a biocultural construct, defines which items found in nature are edible, [and] how these products are transformed into food ... " (Armelagos 2014, p. 1330). Armelagos also discussed the importance of etiquette, the ideological rules of how, when, and with whom we eat.

Both careful food selection, intensive food preparation, and etiquette are important for the survival of people of all ages, but they are particularly crucial for the survival of children. Children, of course, have a relatively larger disproportion between brain size and body/gut size than do adults (Figure 2.8) and an immature dentition, which make children dependent on older individuals for care and feeding. Added together, each of the constraints of childhood – an immature dentition, a small digestive system, a calorie-demanding brain that is both relatively large and growing rapidly, and feeding dependency – necessitate a special diet. It is a diet that must be procured, prepared, and

provided by older members of the social group and fed to infants and children under appropriate rules of etiquette; such as feeding on demand for infants, and feeding children within nonthreatening, emotionally secure physical and social circumstances.

Living humans do this via a behavioral strategy called biocultural reproduction (Bogin et al. 2014b). As practiced by human societies, biocultural reproduction may be defined as the set of marriage and kinship-based rules for extra-maternal cooperation in the production, feeding, and care of offspring. With the assistance of other group members, human women not only have more births than other apes, but also keep alive more of their offspring until they reach adulthood. Further discussion of the evolution of childhood and the central role of biocultural reproduction in human behavior is given later in this chapter.

The Passage from Childhood

Important developments that allow children to progress to the juvenile stage of growth and development are the eruption of the first permanent molars and completion of growth of the brain (in weight). First molar eruption takes place, on average, between the ages of 5.5 and 6.5 years in most human populations (AlQahtani et al. 2010; Esan & Schepartz 2018; Smith 1992). Functional occlusion occurs some weeks to months thereafter. Morphological and mathematical investigation shows that brain growth in weight is complete at a mean age of seven years (Cabana et al. 1993; Caviness et al. 1996). Thus, significant milestones of dental and brain maturation take place at about seven years of age. At this stage of development, the child becomes much more capable of processing dentally an adult type diet. Furthermore, nutrient requirements for the maintenance and the growth of both brain and body diminish to less than 50% of total energy needs.

A possible neuroendocrine event of the transition to the juvenile stage is called adrenarche. This is the postnatal onset of secretion of the androgen hormones dehydroepiandrosterone (DHEA) and DHEA-sulfate (DHEA-S) from the adrenal gland. The mechanism controlling adrenarche is not understood because no known hormone or genomic transcriptional process appears to cause it (Nakamura et al. 2009). In humans, chimpanzees, bonobos, and gorillas adrenarche occurs between the ages of 5–10 years, with a median age of 7 years (Behringer et al. 2012; Bernstein et al. 2012; Campbell 2011; Edes 2017). In some other primates, such as the rhesus monkey, the upregulation of DHEA and DHEA-S begins just before or after birth. DHEA acts as an anti-glucocorticoid with a wide variety of effects, including promoting immune function, altering glucose metabolism, and being neuroprotective, all suggesting a selective benefit, but the evolutionary origins of adrenarche are not known. It is suggested that adrenarche and DHEA-S may play a role in ape and human evolution in terms of extended brain development, brain plasticity, cognitive attention, working memory, and prolonged life span compared with other primates (Campbell 2011; Nguyen et al. 2017).

Adrenal androgens do seem to produce the appearance of wisps of axillary and pubic hair, and changes in body odor (Campbell 2011; Kaplowitz et al. 1986; Leung &

Robson 2008). Earlier research suggested that adrenarche regulates the development of body fatness and fat distribution (Katz et al. 1985), but the body fatness association is not supported by newer research (Mouritsen et al. 2013). Current evidence indicates that there is no connection between the occurrence or timing of adrenarche and the mid-growth spurt (mentioned in Chapter 2) or the onset of puberty (Remer & Manz 2001). Adrenarche does not have any causal relationship with the so-called adiposity rebound, which was described in Box 2.1.

The association of adrenarche with some physical changes in hair growth and body fat distribution seem to mark the progression of the child to the juvenile stage of development. These physical changes may be noticed by the child, his/her parents and other kin, and by close friends and recognized as markers of developmental maturation.

Research from the laboratory of Gillian Bentley at Durham University has explored some of the environmental associations with age at adrenarche. Their work focuses on Bangladeshi girls and women living in the region of Sylhet, Bangladesh and Sylheti migrants and their children in London. In one study it was found that the median age at adrenarche of first-generation migrant girls was 5.3 years. These girls had come to the UK between the ages of <1 to 15 years (median age was 4.0 years). Their age at adrenarche was significantly earlier than nonmigrants in Sylhet (7.2 years), second-generation Bangladeshi girls in London (7.4 years), or European girls in London (7.1 years). Adjusting the analyses for differences in height and weight attenuated the results, but the differences were still significant and biologically meaningful (Houghton et al. 2014). Many infants and young children who migrate from southern and southeastern Asia or Latin America to Western countries of Europe, Canada, Australia, and the United States experience a rapid rate of growth, which may be a catch-up from growth delay in the old country or a stimulation to the tempo of growth from the new country (Bogin et al. 2018a). The authors of the Bangladeshi study suggest that rapid catch-up growth experienced by first-generation girls during early childhood may explain their advanced adrenarche. The environmental conditions leading to an earlier adrenarche, as well as the health implications of this early transition, are not well understood and merit further exploration.

There is a story that links the mid-growth spurt with neoteny. Louis Bolk (1926) originated the hypothesis for human evolution via neoteny. Bolk speculated that for our early human ancestors, sexual maturation took place at about six to eight years of age. The mid-growth spurt was first reported by Backman (1934) and following its discovery, several of Bolk's followers, without any additional supporting evidence, opined that the mid-growth spurt and adrenarche are vestiges of sexual maturation from our evolutionary past. Much research, from clinical medicine to anthropological fieldwork, shows that there is virtually no connection between adrenarche and gonadarche (sexual maturation), as each are independently controlled events (Ibáñez et al. 2000).

Bolk's idea about sexual maturation in the past may be wrong, but a connection between adrenarche and the evolution of the human pattern of growth is still a

possibility. Perhaps the evolution of adrenarche may be explained as a mechanism for mental maturation. The physical changes induced by adrenarche are accompanied by a change in cognitive function, called the "5-to7-year-old shift" by some psychologists, or the shift from the preoperational to concrete operational stage, using the terminology of Piaget.[4] This shift leads to measurable changes in brain architecture and cognition (Boersma et al. 2013; Ellis et al. 2014) and new learning and work capabilities in the juvenile (Rogoff et al. 1975; Sameroff & Haith 1996). In this sense adrenarche may function to mark the transition from the childhood to the juvenile growth stage. Weisner (1996, p. 295) wrote that, "The 5-7 shift involves changes in internal states and competencies of the maturing child – shifts in cognitive capacities, self-concept, visual/perceptual abilities, or social abilities. The transition marks the emergence of increasing capabilities for strategic and controlled self-regulation, skills at inhibition, the ability to maintain attention and to focus on a complex problem, and planfullness and reflection."

Using the terminology of Piaget, the 5-7 shift moves the child from the preoperational to concrete operational stage of cognition. This 5-7-year-old shift is found in all human societies and cultures so far investigated but has never been reported in any other primate species. The 5-7 shift seems to be a human species phenomenon and may be associated strongly with another human phenomenon – symbolic spoken language. A detailed review of language and life history, including perspectives on the ontogenetic development of human language from birth to adulthood and human language evolution was published by Locke and Bogin (2006). Essentials of that review as they relate to childhood are given here (and below for adolescence). Some of the communicative skills arising in childhood do so in tandem with certain cognitive advances. One such development is the "theory of other minds," which typically emerges between ages two and four years (discussed in Chapter 2), enabling children to take the perspective of others. Another is an improvement in autobiographical memory, which usually occurs between ages three and eight years, allowing children to describe sequential events and to share memories of their own experience. Other skills that improve during childhood include discourse and narration, meaning the telling of stories and the expression of perception and feeling about the story and the actors in the story.

Additional developments that occur in childhood relate to verbal competition and performance. These include joking and the use of preassembled verbal routines, such as verbal games played with peers using material learned from family members earlier in infancy, or from unrelated children and juveniles. These games mark the beginning of various sorts of verbal competition and creativity that have been proposed to have played key roles in the evolution of human language.

Since it begins with weaning, childhood would also have liberated the young from continuous maternal restraint. The freedom of irresponsible progeny to range unsupervised over greater distances elevated the need for parents to warn and

[4] https://psychology.iresearchnet.com/?s=five+to+seven+year+old+shift

instruct, giving them and other kin selfish reasons to send honest signals. But childhood also put the young in a position, for the first time, to know about, and thus to convey information about, events occurring in the absence of others. It is possible, therefore, that childhood handed children and their extended families a key ingredient – displacement – in the form of new opportunities, and needs, to talk about things not immediately present. Displacement is one of Charles Hockett's (1916–2000) four design features of language that are uniquely human, the other three being grammatical productivity, duality of patterning, and **cultural transmission** (Hockett 1977).

Juveniles Feed Themselves and Become "Helpers at the Nest"

Ethnographic and psychological research show that juvenile humans have the physical and cognitive abilities to provide some, but not all, of their own food and to protect themselves from environmental hazards such as predation and disease (Blurton-Jones 2002; Kramer 2002; Weisner 1987). In addition to self-care and feeding, human juveniles become increasingly involved with domestic work and "caretaking interactions with other children" (Weisner, 1996, p. 296). Juveniles contribute to the welfare of their social groups and often become "baby sitters" especially for children (Kramer 2002). This baby sitting is part of the story of . . .

How and When Did Human Childhood Evolve?

The stages of the life cycle may be studied directly only for living species. However, there are lines of evidence that may be used to reconstruct the life cycle of extinct species. The fossil evidence of skeletons and teeth provide direct and tangible clues as to the life cycle of extinct species. Indirect evidence comes from the fields of comparative anatomy, comparative physiology, comparative ethology, and archaeology. We know from fossil evidence that one human characteristic, bipedalism, appears relatively early in hominid evolution – about four MYA. There are many hypotheses for the evolution of hominid bipedalism, but in the context of this discussion one of these is most important. Bipedalism "allows individuals to walk long distances and carry objects" (Zihlman 1997, p. 185). Zihlman explains that "objects" include infants. There are significant connections between human infancy and bipedalism. During infancy, when the child is dependent on the mother for care and feeding, the rate of leg growth and the maturation of corresponding nerve cells in the motor-sensory cortex of the brain are slow relative to the growth of the head, the arms, and the nerve cells that control movement in the upper half of the body. By about two years of age, there is an acceleration in both the rate of leg growth and the maturation of the motor-sensory cortex region devoted to the legs. At the time the child is weaned (about three years of age in many pre-industrial societies), independent locomotion takes on greater importance for the child and the rate of leg growth becomes faster than the rate of arm growth (Bogin & Varela-Silva 2010).

Another method used to reconstruct life histories for extinct hominids is by analyzing teeth. Teeth are covered by enamel, the hardest substance in a mammal's body, which protects teeth from destruction. The jaw (mandible and maxilla) that support the teeth are composed of dense and durable bone, and therefore both teeth and jaws (either in whole or part) are more likely to be preserved in the fossil record than any other body parts. This is fortuitous for the study of life history because the morphology and development of the dentition is highly conservative in evolution and the pattern of tooth development reveals a great deal about life history. The use of teeth to reconstruct the evolution of hominid life history is a very active area of research with a burgeoning literature that cannot be adequately reviewed in this book. Interested readers should consult several articles (Dean et al. 2001; Dean & Liversidge 2015; Smith 1992; Smith et al. 2015) for details of methodology and alternative interpretations of the evidence.

One example of this research is the work of B. Holly Smith. As discussed in the previous chapter, Smith (1991) found a significant correlation between age of eruption of the first molar (M1) and adult brain size across a large number of mammalian species. She also found high correlations between age at M1 eruption and age at weaning ($r = 0.93$) and age at sexual maturity for both males ($r = 0.93$) and females ($r = 0.93$). Such high correlations ($r = 1.0$ is a perfect positive relationship) mean that these life history events are linked to some more fundamental developmental processes, and that a change in the timing of one of these events will probably result in a change in the timing of them all. We may conclude, then, that even though age at weaning and sexual maturity cannot be seen directly in the fossil record, they can be estimated based on the dentition.

Another example of the methods used to reconstruct the evolution of life history may be found in the work of Martin (1983, 1990) and Harvey and colleagues (1986) on patterns of brain and body growth in apes, humans, and their ancestors. Martin showed that Old World monkeys and apes have a pattern of brain growth that is rapid before birth and relatively slower after birth. In contrast, humans have rapid brain growth both before and after birth (Figure 4.9). This difference may be appreciated by comparing ratios of brain weight divided by total body weight (in grams). At birth this ratio averages 0.09 for the great apes and 0.12 for human neonates. At adulthood the ratio averages 0.008 for the great apes and 0.028 for people. In other words, relative to body size human neonatal brain size is 1.33 times larger than the great apes, but by adulthood the difference is 3.5 times. The human–ape difference is not due to any single heterochronic process, that is, not the result of delay, prolongation, or acceleration of a basic apelike pattern of growth. Rather it is due to new patterns of growth for the human species. The rate of human brain growth exceeds that of most other tissues of the body during the first few years after birth (Figure 2.8). Martin and Harvey et al. also show that human neonates have remarkably large brains (corrected for body size) compared with other primate species. Together, relatively large neonatal brain size and the high postnatal growth rate give adult humans the largest encephalization quotient (an allometric scaling of brain to body size) of all higher primates (Figure 4.7).

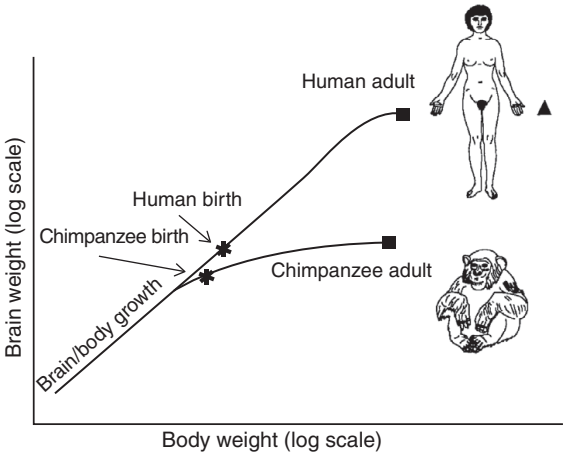


Figure 4.9 Growth curve for human brain and body compared with the chimpanzee. The length of the human fetal phase, in which brain and body grow at the same rate for both species, is extended for humans. Chimpanzee brain growth slows after birth, but humans maintain the high rate of brain growth during the postnatal phase. In contrast, the rate of human body growth slows after birth. If human brain/body growth rate were equal to the chimpanzee rate, then adult humans would weigh 454 kg and stand nearly 3.1 m tall (indicated by the ▲ symbol). After Martin (1983).

Martin (1983) hypothesized that a "humanlike" pattern of brain and body growth becomes necessary once adult hominid brain size reaches about 850 cm^3. This biological marker is based on an analysis of head size of fetuses and birth canal dimensions of their mothers across a wide range of social mammals, including cetaceans, extant primates, and fossil hominids (Martin 1983, pp. 40–41). Given the mean rate of postnatal brain growth for living apes, an 850 cm^3 adult brain size may be achieved by all hominoids (living and extinct apes and humans) by lengthening the fetal stage of growth. At brain sizes above 850 cm^3 the size of the pelvic inlet of the fossil hominids, and living people, does not allow for sufficient fetal growth. Thus, a period of rapid postnatal brain growth and slow body growth – the human pattern – is needed to reach adult brain size. Following this line of reasoning, any fossil human, or any of our fossil hominid ancestors, with an adult brain size above Martin's "cerebral Rubicon" of 850 cm^3 may have included a childhood stage of growth as part of its life history.

Martin's analysis is elegant and tenable. Nevertheless, the difference between ape and human brain growth is not only a matter of velocity; it is also a matter of life history stages. Brain growth for both apes and human beings is mostly complete at about age 7 years, but human brains are both bigger at birth, grow at a much faster rate, and become about 3.8 times larger by age 7 years (Figure 4.10). Steven Leigh explained the different brain growth trajectories of chimpanzees and humans in terms of maternal metabolic adaptations (Leigh 2004). Leigh reviewed data for

several primate species and reported that primates have two major metabolic adaptations. "In the first, brain growth occurs mainly during the prenatal period, reflecting heavy maternal investment. In the second, brain growth occupies large portions of the postnatal period. These differing patterns have important implications for maturation age . . . " (Leigh 2004, p. 139). Prenatal investment requires late maternal maturation so that the mother has accumulated enough physical, social, and cognitive capacity to feed and protect herself as well as her fetus with its high energy demands that can only be met by the mother. Post-natal investments allow for early maternal maturation as the mother need only worry about lactation, which can be terminated at a relatively early age of the infant. Leigh's analysis found that these two different metabolic adaptations are associated with maternal life history more than with the pre- or post-natal life history of her fetus-infant. In a sense, the fetus-infant just goes along the metabolic ride of its mother. This is an important finding and helps support the interpretation that human childhood evolved, primarily, as an adaptation for mothers and not for the child. This is discussed in more detail below.

Chimpanzee mothers invest heavily in pre-natal brain growth and have relatively late maturation – first birth takes place at about age 13 years in the wild. Human mothers invest even more heavily than chimpanzees, giving birth to infants with more than double the brain size of chimpanzee newborns (means neonate brain size: human = 341 cm^3; chimpanzee = 155 cm^3, Holland et al. 2014). Human mothers, and others, then provide enough energy, other nutrients, and the needed social-emotional care to maintain the pattern of fetal brain growth through infancy and childhood. As defined above, and discussed below, humans do this via the biobehavioral adaptation of biocultural reproduction.

Given this background, Figure 4.11 is my own Schultz-inspired summary of the evolution of the human pattern of growth and development from birth to age 20 years (the evolution of adolescence is discussed later in this chapter). Figure 4.11 must be considered as "a work in progress" for two reasons. The first is that only the data for the first and last species (*Pan* and *Homo sapiens*) are known with some certainty. The second is that this version of the figure supersedes earlier versions published that were prepared without the advantage of more recent information about patterns of growth for fossil hominids. Even with the latest information available, the patterns of growth of the fossil hominid species are tentative reconstructions, based on published analyses of skeletal and dental development of fossil specimens that died before reaching adulthood. Known ages for eruption of the M1 are given for *Pan* and *H. sapiens*. Estimated ages for M1 eruption in other species were calculated by (Smith & Tompkins 1995). Age of eruption of M1 is an important life history event that correlates very highly with other life history events. Known or estimated adult brain sizes are given at the top of each bar; the estimates are averages based on reports in several textbooks of human evolution. Following Martin's analysis (1983, 1990), brain size is another crucial influence on life history evolution. One major message to take from the figure is that the prolongation of the total time for growth that plays such a prominent role in the

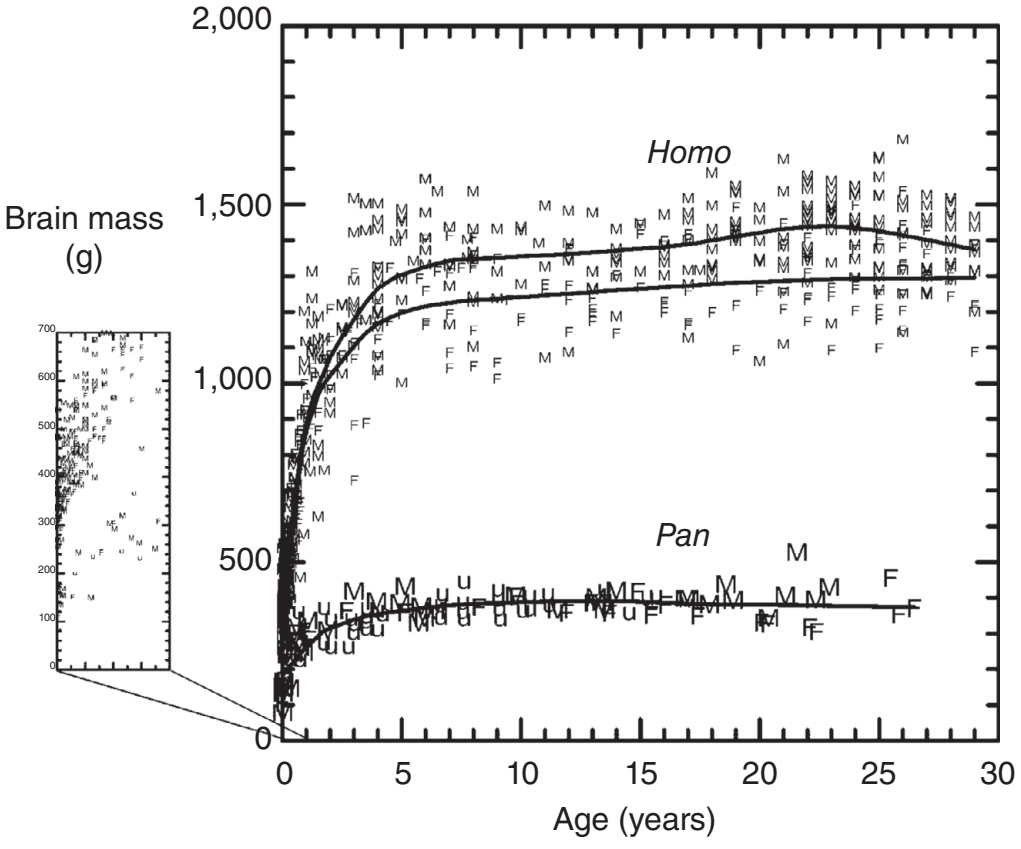


Figure 4.10 Brain-mass growth data for humans (*Homo sapiens*) and chimpanzees (*Pan troglodytes*). Brain mass increases during the postnatal period in both species. Lines represent best-fit LOWESS regressions through the data points. "M", males; "F", females; "U", sex unidentified (Vrba 1998). The human regressions separate into male (upper) and female (lower) curves. The inset shows brain-mass growth for each species during the first postnatal year. Vrba proposed that the human difference in brain mass growth was due to hypermorphosis, that is, the heterochronic process of prolongation of the rapid fetal growth seen in the chimpanzee. Vrba described the chimpanzee pattern as ancestral to the human pattern. Leigh (2004) reanalyzed the data with more appropriate mathematical and statistical models and found no evidence for prolongation or heterochrony. Rather, humans evolved new patterns of rapid growth before and after birth. See the text for more details. Reproduced from Leigh (2004) with permission

hypotheses of neoteny and hypermorphosis is a part of human evolution. However, I find that time prolongation is not sufficient as an explanation or mechanism to account for the insertion of the new stages of childhood and adolescence that are part of human growth.

The hominin fossil record now appears to stretch back more than 6 million years with the appearance of *Sahelanthropus, Orrorin,* and *Ardipithecus* in Africa (Wood &

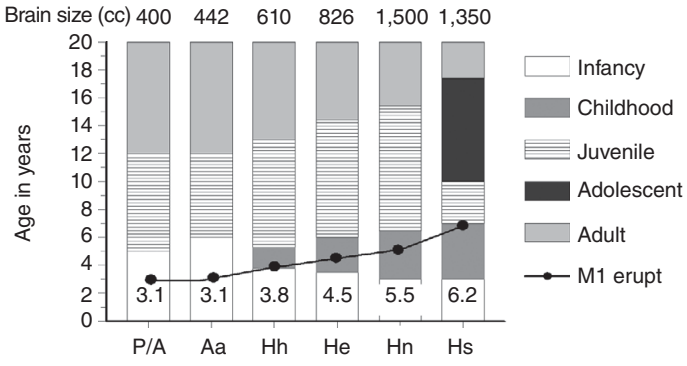


Figure 4.11 The evolution of hominid life history during the first 20 years of life. Abbreviated nomenclature as follows: P/A, *Pan* and *Australopithecus afarensis*; Aa, *Australopithecus africanus*; Hh, *Homo habilis*; He, *Homo erectus*; Hn, *Homo neanderthalensis*; Hs, *Homo sapiens*. Mean adult brain sizes are given at the top of each histogram. Mean age at eruption of the first permanent molar (M1) is graphed across the histograms and given below the graph.

Lonergan 2008). These earliest hominins are known from adult skeletal remains that appear to have been capable of some bipedal locomotion and had brains no larger than the living bonobo and chimpanzee, a mere 300–350 cm^3 in *Ardipithecus ramidus* (ca. 4.4 MYA) and less than 400 in *Sahelanthropus*. We begin to know something more directly of infant and juvenile life by the appearance of *Australopithecus afarensis* about 3.3 MYA. Here the Dikika infant, about 3 years of age at death by its teeth, already has a brain size of 330 cm^3 (Alemseged et al. 2006); adult *A. afarensis* reached an adult brain size of about 400 cm^3 with a pattern of dental development little different from extant apes. Therefore, the chimpanzee and *A. afarensis* are depicted in Figure 4.11 as sharing the typical tripartite stages of postnatal growth of social mammals – infant, juvenile, and adult. The duration of each stage and the age at which each stage ends are based on empirical data for the chimpanzee.

The first permanent molar (M1) of the chimpanzee erupts at about 3.0–3.5 years, but chimpanzees remain in infancy until about age 5 years. Until that age the young chimpanzee is dependent on its mother and is highly unlikely to survive if the mother dies or is otherwise not able to provide care and feeding. After erupting M1 the young chimpanzee may be able to eat adult-type foods (Smith et al. 2013), but is still learning how to find and process foods. Learning to successfully open fruits that are protected by hard shells and to extract insects from nests (such as ants and termites) requires more than one year of observation and imitation by the infant of the mother. For these reasons, chimpanzees extend infancy for more than one year past the eruption of M1.

With a body and brain size near that of the chimpanzee, it is likely that australopithecine hominins followed a pattern of growth and development very similar to

chimpanzees. This is the consensus of existing evidence for the formation in size and shape of tooth crowns and roots suggests that *Au. afarensis* and *Au. africanus* had a pattern of maturation more akin to chimpanzees than to modern humans (Kuykendall 2003; Schwartz 2012). This interpretation is supported by detailed study of microscopic anatomy of incremental enamel formation in tooth crowns which confirms that australopiths, like chimpanzees, erupted M1 at a mean age of 3.1 years (Dean et al. 2001; Dean & Liversidge 2015).

Australopithecine postcranial growth and development is known from few specimens. Christine Berge (2002) employed a heterochrony approach for comparative analysis of the pelves of juvenile and adult African apes (N=150), modern humans (N=60), and two adult pelves, fossil catalogue numbers Sts 14 – *Au. africanus* 2.5 MYA, AL 288-1 - *Au. afarensis* "Lucy" 3.2 MYA, and a single juvenile hip of MLD 7 - *Au. africanus* 2.6–2.7 MYA. Berge noted that MLD 7, as a juvenile, is similar in pelvis shape to juvenile humans, whereas adult Australopithecus represented by Sts 14 and AL 288-1 are much flatter and wider side to side. Berge took this pattern of ontogenetic change with maturation to match an apelike pattern of ilium shape growth. Christine Tardieu (1998) proposed from analysis of the femur of modern humans and australopiths that extended time for growth and lengthening of the lower limb was an essentially *Homo*like trait and that the juvenilized features of the femur of adult *Au. afarensis* is evidence of its apelike pattern of growth. Analysis of postcranial evidence from DIK-1-1, the infant *Au. afarensis* from Dikika, supports this apelike pattern (Green & Alemseged 2012). Based on development and locomotor loading of the scapula, especially the morphology and orientation of the scapular spine of this infant as compared with adults of *Au. afarensis*, the growth is more apelike than humanlike. The immature hominin of the new species *Australopithecus sediba* was discovered at the 1.9 MYA Malapa site in South Africa. The specimen, catalogued as MH1, includes substantial post-cranial skeletal material, and provides a unique opportunity to assess its skeletal maturation (Figure 4.12). I was part of the analysis team (Cameron et al. 2017) that examined eight centers of ossification in the limbs, pelvis, hand, and foot. We reported that in comparison to humans the skeletal maturational ages for *Au. sediba* at the time of death correspond to between 12.0 years and 15.0 years with a mean (SD) age of 13.1 (1.1) years. In comparison to the maturational pattern of chimpanzees the *Au. sediba* indicators suggested a skeletal maturational age of 9–11 years. Based on either of these skeletal maturity estimates and the body length at death of MH1, an adult height of 150–156 cm was predicted, which is more chimpanzee-like than humanlike. We concluded that the skeletal remains of MH1 were consistent with an apelike pattern of maturity, which is consistent with dental age estimates.

Summarizing all of these findings suggests that australopiths did not grow, develop, and mature in a manner consistent with the distinctive and delayed pattern of dental and post-cranial pattern of the genus *Homo*, especially living humans. Most likely, the australopiths, like the living chimpanzee, may have extended infancy for at least one year beyond the age of M1 eruption and then moved on to the juvenile stage.

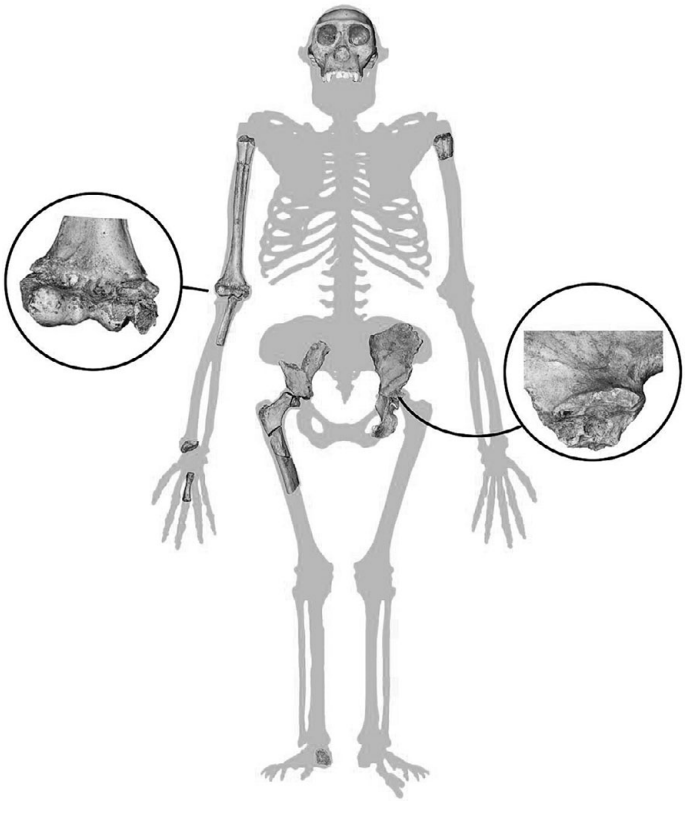


Figure 4.12 *Australopithecus sediba* MH1 skeletal elements used in assessment of maturity. Left circle: Pull-out of right distal humerus showing fused three elements of composite epiphyses, and fusion to the diaphysis. Right circle: Pull-out of left partial ilium, unfused acetabulum. From: Cameron et al. 2017

Homo "Rocks"

More intensive use of technology, especially stone tools, may have been an important next step in the evolution of human life history. The intentional breaking of rocks into flakes with sharp edges, which are superficially like stone tools, can be traced approximately 3.3 MYA, although which species made these and their intended use is not known (Harmand et al. 2015). Living capuchin monkeys smash rocks and produce what looks like stone tool flakes with sharp edges, but the monkeys do not use these as tools. Rather they lick rock dust from the broken surfaces in what may be a way to ingest needed minerals (Proffitt et al. 2016). More clearly recognizable stone tool use dates to about 2.2 MYA. Fossil hominins of the genus *Homo* also date to this time. These earliest *Homo* species possessed several humanlike traits, such as larger cranial capacities and greater manual dexterity. The stone tools associated with these fossils are of the Oldowan tradition and show greater consistency in manufacture and clear use in food processing – they were used to make cut marks on bone (Kivell 2015).

Researchers split early *Homo* into at least two species, *H. habilis* and a larger *H. rudolfensis*. The rapid expansion of adult brain size during the time of *H. habilis* (650 to 800 cm^3) might have been achieved with further expansion of both the fetal and infancy periods, as Martin's "cerebral Rubicon" was not surpassed. However, the insertion of a brief childhood stage into hominin life history may have occurred. Tardieu (1998) suggested that *H. habilis* had a pattern of growth of the femur that was distinct from that of the australopiths, but consistent with that of later hominins. This distinctive femur shape could be due to the addition of a new pattern of growth, as this is what is seen for living children. *H. habilis* may have had a short childhood stage of growth.

For these reasons, a brief childhood stage for *H. habilis* is indicated in Figure 4.11. This stage begins after the eruption of M1 and lasts for about one year. That year of childhood may have provided the time needed to learn about finding and processing adult-type foods. But, more crucially, the shift from infancy to childhood would have freed the mother from lactation and allowed her to become pregnant again. This reproductive advantage to the mother is described in more detail below. *H. habilis* children would have needed to be supplied with special weaning-foods by other members of the social group. There is archaeological evidence for just such a scenario. *Homo habilis* seemed to have intensified its dependence on stone tools. There are both more stone tools, more carefully manufactured tools, and a greater diversity of stone tool types associated with *H. habilis* than with the australopiths. There is considerable evidence that some of these tools were used to scavenge animal carcasses, especially to break open long bones and extract bone marrow (Pante et al. 2018). This behavior may be interpreted as a strategy to feed children. Although there is no direct evidence, such scavenging may have been needed to provide the essential amino acids, some of the minerals, and, especially the fat (dense source of energy) that children require for growth of the brain and body.

A childhood stage of growth for the earliest members of the genus *Homo* is also supported by a comparison of human and ape reproductive strategies. There are limits to the amount of delay possible between birth and sexual maturity, and between successful births, that any species can tolerate. The great apes are examples of this limit. Chimpanzee females in the wild reach menarche, first estrus with sexual swelling, at 10 to 12 years of age and have their first births at an average age of 14 years – later than any other mammal on earth, excepting elephants and living humans. The average period between successful births in the wild is 5.6 years, as infant chimpanzees are dependent on their mothers for about five years. Actuarial data collected on wild-living animals indicate that between 35% and 38% of all live-born chimpanzees survive to their mid-twenties. Although this is a significantly greater percentage of survival than for most other species of animals, the chimpanzee is at a reproductive threshold. Goodall (1983) reported that for the period 1965 to 1980 there were 51 births and 49 deaths in one community of wild chimpanzees at the Gombe Stream National Park, Tanzania. During a 10 year period at the Mahale Mountains National Park, Tanzania, (Nishida 2011) observed, 74 births, 74 deaths, 14 immigrations and 13 emigrations in one community. Chimpanzee population size

in these two communities is, by these data, effectively in equilibrium. Any additional delay in age of females at first birth or the time between successful births would likely result in a decline in population size.

The great apes and extinct hominins such as *Australopithecus*, may have reached a demographic limit by extending the length of the infancy stage and requiring enormous direct maternal investment in each offspring and long interbirth intervals. Somewhere in our history, however, hominins began to reverse the trend, producing offspring in more rapid succession. An often cited example, the !Kung, are a traditional hunting and gathering society of southern Africa. A !Kung woman's age at her first birth averages 19 years and subsequent births follow about every 3.6 years, resulting in an average fertility rate of 4.7 children per woman (Howell 1979). Women in another hunter-gather society, the Hadza, have even shorter intervals between successful births, stop nursing about one year earlier, and average 6.15 births per woman (Blurton Jones et al. 1992). The key seems to be that humans wean infants before they can feed themselves, freeing mothers from the demands of nursing and the physiological brake that frequent nursing places on ovulation, allowing mothers to reproduce again much sooner. These early-weaned infants are by definition "children," still dependent on others for feeding, but no longer supplemented by mother's milk.

Further brain size increase occurred during *H. erectus* times, which began about 1.8 MYA. The earliest adult specimens have mean brain sizes of 826 cm^3, but many individual adults had brain sizes between 850 to 900 cm^3. As shown by an adult female *H. erectus* pelvis 1.8 MYA from Gona Ethiopia, pelves were more obstetrically capacious for giving birth to a larger-brained infant, as large as 315 cm^3 (Simpson et al. 2008). Judging from Gona, *H. erectus* may have given birth to offspring with 35% of adult brain size, intermediate between chimpanzees (40%) and humans (28%) (Simpson et al. 2008). With adult *H. erectus* at or above Martin's "cerebral Rubicon" and the new and direct evidence for an intermediate-brain-sized, more helpless neonate, it may be expected that *H. erectus* required some degree of a postnatal rapid catch-up in brain growth. Although a more helpless infant would have required even more intense care and an even longer infancy, at some point, hominins evolved a growth life history pattern that shrank infancy, substituting and expanding childhood rather than ever-increasing the infant stage, eventually dropping the transition from infancy down to before the eruption of M1 and the other permanent teeth.

From other fossils of *H. erectus*, we even know that the timing of M1 eruption has evolved, changing from the ca. 3.0 to 3.5 years of earliest hominins to about 4.5 years (Dean & Smith 2009). In all, the fossil record indicates that *Homo erectus* evolved a slowed pattern of general maturation, more helpless infants, larger adult brains, and increasing sophisticated tools – all of which adds to an adaptive network of higher quality offspring, long and intense learning, and reliance on complex behavior. Taken together with evidence of larger body size in females, Aiello and Key (2002) pointed out the enormous energy demands for reproduction in a *Homo erectus* female, demands that necessitated change in energetic strategies. Whenever it did evolve, a childhood period would at first diminish the reproductive cost of this "high

quality strategy," by spreading the burden of feeding children to other social group members, and eventually allow the strategy to become even more extreme. At some point, perhaps even with *H. erectus*, hominins shrank the infancy period to below that of chimpanzees, which would have given them a greater reproductive advantage than any previous hominin. The fact that *H. erectus* populations certainly did increase in size and began to spread throughout Africa, and into other regions of the world, suggests that fundamental changes in life history had already begun.

Later *H. erectus*, with average adult brain sizes of 983 cm^3, are depicted in Figure 4.11 with further expansion of the childhood stage. In addition to bigger brains (some individuals had brains as large as 1,100 cm^3), the archaeological record for later *H. erectus* shows increased complexity of technology (tools, fire, and shelter) and social organization. These techno-social advances, and the increased reliance on learning that occur with these advances, may well be correlates of changes in biology and behavior associated with further development of the childhood stage of life – an interpretation I advocated many years ago (Bogin & Smith 1996a). The evolutionary transition to archaic, and finally, modern *H. sapiens* expands the childhood stage to its current dimension. Note that M1 eruption becomes one of the events that coincide with the end of childhood (Figure 4.10). This is roughly the point that many mammals become independent juveniles, and as discussed earlier, in humans is the period that introduces significant biological, cognitive, behavioral, and social changes.

With the appearance of *H. sapiens* comes evidence for the full gamut of human cultural capacities and behaviors. The technological, social, and ideological requisites of culture necessitate a more intensive investment in learning than at any other grade of hominin evolution. The learning hypothesis for childhood, while not sufficient to account for its origins, certainly plays a significant role in the later stages of its evolution.

The Evolution of Adolescence

"Adolescence is a new birth, for the higher and more completely human traits are now born" G. Stanley Hall (1844–1924). Hall was a racist, eugenicist, psychologist, and author of the two-volume work *Adolescence: Its Psychology and Its Relations to Physiology, Anthropology, Sociology, Sex, Crime and Religion* (1904), from which the quote is taken. Hall believed that human growth and development recapitulated human evolution. Childhood represented the time when human ancestors were selfish savages. Adolescence was a stage of evolution when human ancestors had more altruistic tendencies and that living adolescents needed strict indoctrination to channel those tendencies toward the good of society. Hall popularized the phrase "storm and stress" to characterize the emotional state of the adolescent. A charitable evaluation of Hall's work is that it represented the sociology of adolescence, economics, and politics of his own nineteenth-century United States.

Hall was wrong about recapitulation, but he was correct that the *H. sapiens* grade of evolution added the adolescent stage to post-natal development. The single most important feature defining human adolescence is the skeletal growth spurt that is

experienced by virtually all living boys and girls. There is no evidence for a human-like adolescent growth spurt in any living ape. There is no evidence for adolescence for any species of *Australopithecus*. There is some tentative evidence that early *Homo*, dating from 1.8 MYA, may have a derived pattern of growth that is leading toward the addition of an adolescent stage of development. As mentioned above, this evidence is based on an analysis of shape change during growth of the femur (Tardieu, 1998). Modern humans have highly diagnostic shape to the femur, a shape that is absent in fossils ascribed to *Australopithecus*, but present in fossils ascribed to *Homo habilis, H. rudolfensis, H. ergaster*, or early African *Homo erectus*. The human shape is produced by growth changes during both the prolonged childhood stage and the adolescent stage, but whether the two are inextricably linked remains conjectural.

A remarkable fossil of early *Homo erectus* is both of the right age at death and complete enough to allow for an analysis of possible adolescent growth. The fossil specimen is catalogued formally by the name KMN-WT 15000 but is called informally the "Turkana boy" as it was discovered along the western shores of Lake Turkana in 1984. This fossil is 1.6 MYA, and clearly an early variety of *Homo erectus*. The skeletal remains are almost complete, missing the hands and feet and a few other minor bones. B. Holly Smith (1993) and more recently Dean and Smith (2009) analyzed the skeleton and dentition of the Turkana fossil and ascertained that, indeed, it is immature and most likely a male. The youth's deciduous upper canines were still in place at the time of death, and he died not long after erupting second permanent molars, an event that occurs about the time of puberty in male higher primates (Smith 1993: Fig 9.2). These dental features place him firmly in the juvenile stage by comparison with any primate. Even though the Turkana boy skeleton is one of the most complete, it is not possible to determine an exact stature at his time of death: Earlier estimates of 160 cm have been lowered to 154 cm (Graves et al. 2010). The revision lowered estimates of adult stature to 163 cm (∼ 5'4″), not 185 cm (∼ 6'1″) as previously reported. The shorter male stature may have implications for energy requirements, sexual dimorphism, and mating behavior of *H. erectus*. Even with the revised stature estimate, the Turkana boy is one of the tallest fossil youths or adults ever found.

The Turkana youth is sufficiently complete to study his pattern of growth and development, and ask "Did early *H. erectus* have an adolescent growth spurt?" At present, the best answer seems to be "no." Judged according to modern human standards, the Turkana boy's dental age of 10.5–11.0 years is in some conflict with his bone age (skeletal maturation) of 13.0 years and his stature age of 15.0 years. If the Turkana boy grew along a modern human trajectory, then dental, skeletal, and stature ages should be about equivalent. Skeletal and dental ages this discrepant are known in less than 1.5% of normal living boys aged 6–15, and it is particularly rare to see the skeleton advanced over the dentition (Dean & Smith 2009). By chimpanzee growth standards, however, the boy's dental and bone ages are in perfect agreement, both suggesting 7 years of age. So was the Turkana boy 13 or 7? Recently, Dean and Smith (2009) answered this question by counting evidence of time passing in the

growth increments preserved in the enamel of the teeth of the Turkana boy (analogous to counts of growth rings in trees[5]). They found that dental microanatomy could account for about 8.5 years of life for the youth. If correct, then death at age 8.5 means the Turkana boy followed a timing of growth that is neither that of a modern human nor that of a chimpanzee. His relatively large stature-for-age becomes understandable if we suppose that the distinct human pattern of moderate to slow growth prior to puberty followed by an adolescent growth spurt had not yet evolved in early *Homo erectus*. Rather, the Turkana boy followed a more apelike pattern of growth in stature, completing nearly 95% of his estimated adult height by the onset of puberty and emergence of second molars (see also Graves et al. 2010). This is even greater than chimpanzees, who at the onset of puberty have usually achieved 88% of body length. Living humans achieve only 81% at the same stage of maturation. "Because of this, any early *H. erectus* youth would seem to us to be too large" (Smith & Tompkins 1995, p. 273).

Unfortunately, there are no appropriate fossil materials of later *H. erectus* available to analyze for an adolescent growth spurt. There are several fossils of a species called *Homo antecessor*, found in Spain and dated to about 800,000 BP (years before present) (Bermúdez de Castro et al. 1999). *Homo antecessor* was once proposed as a possible last common ancestor between modern *Homo sapiens* and Neandertals and to have an adolescent growth spurt. The recovery of new material, better dating, and biochemical/ancient DNA analysis have rejected both ideas. At present there is uncertainty as to the contribution of *H. antecessor* to later European populations and reconstruction of the formation of teeth and growth of the jaws suggests that *H. antecessor* did not have an adolescent growth spurt or other features of human adolescence (Bermúdez de Castro et al. 2017).

Did Neandertals Have Adolescence?

According to Chris Stringer, "We know more facts about Neanderthals[6] than any other extinct humans. Many thousands of their artefacts and fossils have been found, including several nearly complete skeletons."[7] Stringer adds that extraction of ancient DNA obtained from their fossils allowed for the reconstruction of several Neandertal genomes. Even so, questions about Neandertal behavior and biology, including life history stages of growth and development, remain to be answered.

[5] This method is based on detailed analysis of dental microanatomy, including the lines and prisms in tooth enamel that are visible to microscopic study. Details of the method are given in the articles cited in the text.

[6] The two accepted spellings are "Neanderthal" and "Neandertal." The "-thal" spelling persists most strongly in England, but this book uses U S English orthography. Both spellings can be pronounced with either a "t" or a "th" sound – both are acceptable and widely used in English. The German spelling was "-thal" but the pronunciation has always been "t" (German has no "th" sound) www.talkorigins.org/faqs/homs/spelling.html. None of this affects the taxonomic name of the Neandertals as *Homo neanderthalensis*, as this is the genus and species name given to the type of specimen fossils in 1864 by William King.

[7] www.nhm.ac.uk/discover/who-were-the-neanderthals.html

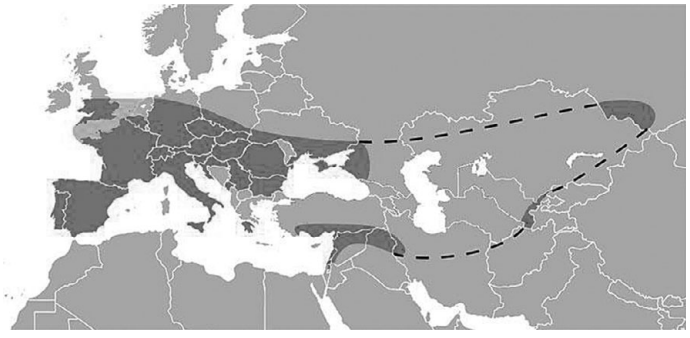


Figure 4.13 Map showing the known range of Neanderthals © I Ryulong licensed under CC BY-SA-3.0, via Wikimedia Commons (https://commons.wikimedia.org/wiki/File:Range_of_Homo_neanderthalensis.png).

The most fundamental question is, "Were the Neandertals human?" The answer is, "almost, but not quite!"

What is generally accepted is that the species *Homo neanderthalensis* lived from about 400,000 to 40,000 BP across Europe and southwest and central Asia (Figure 4.13). Neandertal remains are found in places that were in cold steppe environments at about 60,000 BP, such as in what is today England and Siberia, and in warm temperate woodlands from about 120,000 BP that are today part of Spain and Italy. This broad eco-geographic range suggests that Neandertals had a flexible repertoire of technological and social adaptations. The archaeological record bears this out as there is abundant evidence for complex tools and tool kits, often called the Mousterian tool complex which included Levallois stone point-making technology. Neandertals had controlled use of fire by 200,000 BP and likely much earlier and practiced social care of injured, ill and disabled individuals. Did Neandertals have a humanlike ideology? This is not knowable with certainty but there is evidence for burial of the dead with possible grave goods, and art in the form of red and black paintings on the walls of rock shelters and caves. The paintings include images of, " . . . animals, linear signs, geometric shapes, hand stencils, and handprints," suggesting that, " . . . Neandertals possessed a much richer symbolic behavior than previously assumed" (Hoffmann et al. 2018).

Neandertals and their Asian contemporaries the Denisovans seem to be our genetically and behaviorally closest ancient hominin relatives. Fossil and DNA evidence are interpreted to indicate that the ancestors of both Neandertals, Denisovans, and modern humans separated at between 500,000–650,000 years ago, but their ancestor species (single or plural) is unknown. There are so few fossils of Denisovans that they are not discussed further here in the context of the evolution of the pattern of human growth. Despite many similarities, Neandertals differed from ancient and living *H. sapiens* in several important ways. Neandertals had shorter and stocky bodies with a male height ranging between 150–175 cm and weight from about 64 to 82 kg. Their brain size was larger at about 1,200–1,750 cm^3 (modern humans are typically between 1,200–1,450 cm^3) and perhaps differently shaped.

These differences in body and brain size and shape, along with genetic differences, support the classification of *H. neanderthalensis* as a species distinct from *H. sapiens*.

There are a few immature fossils of Neandertals that can help us understand their growth and development. Most are known from teeth and skulls and analyses of these indicate that, on average, Neandertals had significantly faster dental maturation than modern humans (Smith et al. 2010). Perhaps this also indicates that Neandertals completed their growth faster and lacked the adolescence stage characteristic of living humans. Other analyses of immature Neandertals leave open the possibility of a more humanlike life history of growth. The Dederiyeh 1 infant is a nearly complete skeleton of an individual who died at about 2.0 years of age discovered in 1993. The Dederiyeh 2 infant, also died at about age 2.0 years, was discovered in 1997. The remains are from Syria, and dated to 70,000–50,000 BP. The fossils are ascribed to *H. neanderthalensis* based on skeletal morphology, including bone cortical thickness greater than expected for *H. sapiens* (Sawada et al. 2004). These fossil infants were otherwise growing in size and developing their teeth at about the same rate as modern humans. Due to their young age at death it is not possible to know what their growth at later ages might have been.

The Scladina cave (Sclayn, Belgium) juvenile Neandertal was discovered in 1971 and additional excavations produced nearly complete maxillary and mandibular dentition. The cave site is dated to between 80,000–127,000 BP. Tanya Smith and colleagues ascribed an age at death to this juvenile of 8.05 years based on dental enamel microanatomy. An exact age for first molar eruption is not possible to determine in the Scladina juvenile, but it likely occurred before 6.0 years of age based on the amount of wear on this tooth, the estimated age at death, and other juvenile Neandertal remains (Smith et al. 2007b). If this is the case, then Neandertals had M1 eruption that was at least intermediate in age between *H. erectus* and *H. sapiens*. This is indicated in Figure 4.11 in the "Neandertal" column as M1 eruption at age 5.5 years.

Compared with the average tooth eruption ages of living human juveniles, the Scladina Neandertal's age at death is 2–3 years younger than expected for its overall dental maturation. The disconnect between age at death estimates and dental maturation is found in other samples of immature Neandertals. This indicates that Neandertals grew and developed at a faster rate than living *H. sapiens*. If this were true, then Neandertals would have experienced an accelerated life history, with a shorter childhood, relative to *H. sapiens*. Another life history difference between living people and the Scladina juvenile relates to its feeding during infancy. Biochemical and microanatomical analysis of primate teeth can provide an indication of diet and age at weaning. The Scladina juvenile was exclusively breastfed from birth to age 7 months and then fed a mixture of breast milk and other foods for another 7 months (Austin et al. 2013). This pattern of infant feeding is shared with living humans, but at 1.2 years of age the Neandertal infant was weaned by an abrupt cessation of nursing. Living human infants may also abruptly cease nursing at this age, however, analysis of historic samples of modern human teeth (from times before infant formula) typically show weaning at ages between 2.0–3.0 years. The early weaning

of Scladina is, perhaps, another indication of relatively rapid Neandertal life history. Two molar teeth from the site of Payre in southeastern France are ascribed to Neandertals dated to about 250,000 BP. Biochemical analysis indicates that these two individuals were weaned at about age 2.5 years, more in keeping with living humans (Smith et al. 2018). Some of the same researchers were involved in both the Scladina and Payre tooth studies and they conclude that more research is needed to establish reliable and typical ages at weaning for Neandertals.

Excavations in the 49,000 BP El Sidrón cave system in Asturias, Spain resulted in the discovery of more than 2,500 Neandertal fossil remains. These remains represent seven adults and six immature individuals. Analysis of DNA from the bones indicates that they had close genetic relationships and likely belonged to a single social group. One of the immatures is El Sidrón J1, most likely a boy, is known from a partial immature skeleton about 36% of its left side is complete (Rosas et al. 2017). Based on dental microanatomy his age at death is estimated at 7.69 years (range: 7.61–7.78 years). His skeletal maturity and skeletal age were assessed against modern human children and juveniles from six ossification centers from the elbow, hand, wrist, and knee. This provided an estimated skeletal age of 7.62 ± 2 years, with a possible range from 6 to 10 years. In terms of both dental and skeletal development El Sidrón J1 fell well within the modern human range of variation. This juvenile boy differed from living 7- to 8-year-old juveniles in brain growth. At his death there was still active brain size growth as evidenced by visible bone resorption on the inner surface of the skull bones which is a sign of brain growth in living children. He had an estimated endocranial volume of 1,330 cm^3 which is about 87.5% of average adult Neandertal brain size of 1,520 cm^3. Modern human juveniles complete 95% of brain size growth by 7 years of age. As there is a trade-off between growth of the body and the high energy demanding brain this may mean that the El Sidrón J1 juvenile Neandertal continued to grow and develop at a slow pace for several more years longer than modern humans. If Neandertal brain size growth was completed at puberty, the post-pubertal juveniles may have needed a growth spurt to rapidly achieve adult body height, or they may have continued a steady growth trajectory to final height. The second possibility is more likely because Neandertal adults are shorter, on average, than most living humans. In addition, the analyses by Tanya Smith and colleagues (Smith et al. 2007b, 2010) found dental evidence for more rapid growth and development in Neandertals than expected for modern humans.

There is one fossil of a Neandertal in which the associated dental and skeletal remains needed to assess adolescent growth are preserved. Le Moustier 1, found in 1908 in western France, was most likely a male and still growing at the time of his death. The specimen is dated at between 42,000–37,000 BP. Thompson and Nelson (2011) used information on crown and root formation of the molar teeth to estimate a dental age of 15.5 ± 1.25 years. His dental development – beyond M2 emergence and with third molars well developed – clearly indicates a late adolescent. Compared with modern human standards for length of the long bones of the skeleton, Thompson and Nelson estimate that Le Moustier 1 has a stature age of about 11 years and had achieved about 87% of adult femur length. The dental age of 15.5 years and the

stature age of 11 years are in very poor agreement. Thompson and Nelson could not find any modern human cases that matched the Le Moustier pattern of development. All this evidence indicates that like the Turkana boy, Le Moustier 1 did not follow a human pattern of adolescent growth.

While it is possible that the Turkana boy, Scladina, El Sidrón J1, and Le Moustier 1 are "atypical" individuals, perhaps having suffered from disease or malnutrition that may have deranged the synchrony between their dental and skeletal growth, the most parsimonious conclusion that one may draw from these findings is that all the features of the modern human life cycle, including the adolescent stage and adolescent growth spurt, evolved only with the appearance of *Homo sapiens*. Quite likely this would be no earlier than the appearance of archaic *H. sapiens* in Africa. In fact, the earliest hard evidence of more modern growth and development comes from archaic *Homo sapiens* fossils from Jebel Irhoud, Morocco. These fossils are dated at least to 160,000 BP and possibly as old as 315,000 BP. The remains include partial skulls, jaws, teeth, leg, and arm bones from at least five individuals, including a juvenile and a possible adolescent (Hublin et al. 2017). The Irhoud 3 juvenile consists of a nearly complete mandible. The age at death, estimated from enamel microstructure was 7.78 years. Analysis of tooth formation and eruption sequence showed a prolonged modern humanlike period of dental development, with an estimated age at death of 7.2–7.3 years for a female or 7.3–7.6 years for a male juvenile (Smith et al. 2007a). The Irhoud 3 juvenile age of death based on enamel microanatomy and age at death from dental development and eruption match closely with each other and with modern human dental ages.

An archaic *Homo* juvenile with an estimated age-at-death of 6.5 years was discovered in northern China at the Xujiayao site. The remains are imprecisely dated to the rather large range of 224,000–104,000 BP (Xing et al. 2019). The specimen has an archaic skeletal morphology, but most aspects of dental development fall within the modern human range, including prolonged crown formation time and delayed first molar eruption. The authors stated that, "These findings suggest that several facets of modern human dental growth and development evolved in East Asia before the appearance of fully modern human morphology" (Xing et al. p. 1).

The genus *Homo* is proving to be much more diverse and complex in biology than ever expected. Several new species with small body and brain size are proposed, especially *H. naledi*. This species is known from more than 1,500 skeletal and dental fragments discovered in the Rising Star cave system of South Africa (Berger et al. 2015). The age of the fossils is estimated at 236,000–335,000 BP (Dirks et al. 2017). A team of paleontologists and anthropologists, including your humble author, analyzed the first batch of tooth and jaw remains collected from the floor of one of the cave chambers (Bolter et al. 2018). Our analysis was based on a total of 190 teeth or partial teeth recovered during excavations in 2013 and 2014, which were used in the present study. Of these teeth, 60 are in situ within 7 partial mandibles and 1 partial maxilla. From these we estimated that there were at least 15 individuals. Based on tooth formation of deciduous and permanent teeth, eruption, and wear we assigned 13 of these individuals to age classes. There were 3 infants (no permanent

molars erupted), 3 early juveniles (M1 erupted), 1 late juvenile (M2 erupted), 1 subadult (M3 erupted, unworn), 4 young adults (all molars erupted, moderate wear), and 1 old adult (all fully erupted, wear well into dentin on M2 or sufficient to obliterate most occlusal detail on M3). The other 2 individuals lacked molar teeth making it impossible to estimate age at death, but one was adult and the other immature. It is likely that *H. naledi* had an *Australopithecus*-like life history due to *H. naledi* having a brain size within the range of australopiths and an estimated stature within the range of both small-bodied humans and the largest known australopiths. Two *H. naledi* individuals with teeth in the jaw show a mixture of human and chimpanzee-like stages of permanent tooth eruption (Cofran & Walker 2017).

One way of summarizing the differences in life history between *Homo sapiens* and all other hominins is depicted in Figure 4.14. The dental estimate or known age at death of all fossil and recent *Homo sapiens* generally matches a predicted age at death from modern human tooth formation and eruption references. Other hominin

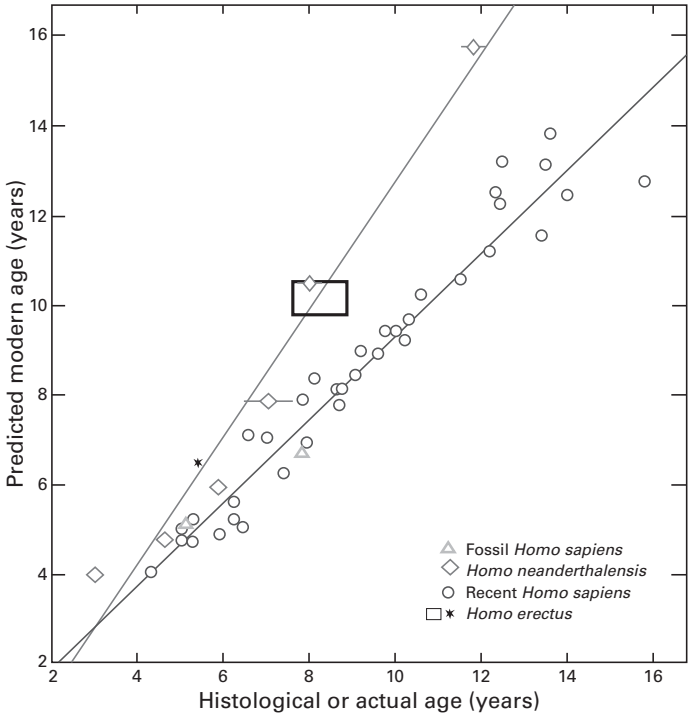


Figure 4.14 Comparison of fossil hominin ages at death determined from tooth microanatomy vs. modern human tooth formation and eruption references. Fossil *H. sapiens* are represented by the juvenile fossils Qafzeh 10 and Irhoud 3 (triangles); Neandertals (diamonds) are represented by Engis 2, Gibraltar 2, Krapina Maxilla B, Obi-Rakhmat 1, Scladina, and Le Moustier 1 (from left to right). Estimates from two early *Homo* fossils of juveniles are shown: asterisk: KNMER 820; box: KNMWT 15000, Turkana boy, both ~1.5–1.6 MYA). Comparisons of known and predicted ages are given for 36 living humans for comparison (circles). The lines through the data points are fit by linear regression. Modified from Smith (2013)

species, however, show more rapid development as indicated by the steeper slope of their regression line. It seems that modern human life history evolved only with archaic *Homo sapiens* and not with any older or even contemporaneous species, such as *H. antecessor, H. naledi*, Neandertals, or Denisovans.

Who Benefits from Childhood?

Brain sizes, the shape of long bones, dental microstructure, and tooth eruption patterns provide some idea of when human life stages may have evolved, but do not explain why they evolved. Bonner (1965) showed that the presence of a stage, and its duration, in the life cycle relates to such basic adaptations as locomotion, reproductive rates, and food acquisition. To make sense out of the pattern of human growth one must look for the "basic adaptations" that Bonner describes.

The most basic of these adaptations are those that relate to evolutionary success, traditionally measured in terms of the number of offspring surviving to reproductive age. Biological and behavioral traits do not evolve unless they confer upon their owners some degree of reproductive advantage, in terms of survivors a generation or more later. Three "textbook" reasons for the evolution of human childhood were listed at the start of this chapter. These reasons emphasized the role of learning in human adaptation, and they are valid reasons since learning does confer an adaptive advantage to pre-adult individuals. However, the "textbook" explanations cannot account for the initial impetus for the insertion of childhood into human life history. A childhood stage of development is not necessary for the type of learning listed here. The prolonged infancy and juvenile period of the social carnivores and apes can serve that function (Clutton-Brock 2016). Rather childhood may be better viewed as a feeding and reproductive adaptation for the mother. Since human mothers live within larger social groups of many mothers, the evolution of childhood is a reproductive adaptation for the human species. The following discussion connects the evolution of childhood as a basis for the human style of making babies within a complex cultural ecology of families and kinship networks that was defined above as biocultural reproduction.

Cooperative Breeding vs. Human Cooperation in Reproduction

Compared with most species of primates, especially the apes, humans have an unusual style of producing and raising offspring that is often described as cooperative breeding (Burkart et al. 2014; Hrdy 1999, 2009). To be ''cooperative'' in breeding is often defined to mean that individuals of a species live in groups and that members of the group help to feed, care for, or protect offspring that they did not bear (Clutton-Brock 2016). Individuals providing these services are called alloparents. Another commonly cited criterion is that the provisioning, care, and protection that alloparents provide must come at some cost to the alloparents. That cost may be measured in assisting others to gain access to food or in terms of reducing the

alloparents' opportunities to reproduce (Lukas & Clutton-Brock 2012; Solomon & French 1997).

Cooperative breeding species are not common in the Order Primates and, indeed, marmosets and tamarins of South America are the only widely-recognized species of cooperative breeding nonhuman primates (Díaz-Muñoz 2016; Fite et al. 2005; Tirado Herrera et al. 2000). Some of the costs incurred by members of these New World monkey species are that only a few females in a social group reproduce, two or more males mate with the same female, which reduces male reproductive success and mothers without sufficient allomaternal support abandon or kill their dependent young.

Ethnographic work among contemporary human foragers provides detailed examples of the extent of cooperative allocare and its role in human societies and shows that this care comes at a cost to the care provider. As one instance, Marlowe (2010) reported that during the first year that Hadza women are breastfeeding an infant the mothers' ability to produce food is reduced to an average of 1,713 kcals/day. This compared to an average food production of 3,016 kcals/day for women without offspring less than 8 years old. Food contributions of married men increased from an average of 2,990 kcals/day to 3,851 kcals per/day during the first year of breastfeeding by their wives. The women's decrease in food productivity averaged 1,307 kcals/day and the increase of food provisioning by husbands averaged only 861 kcals/day. The shortfall of 446 kcals/day is made up by increased provisioning by other members of the camp, especially maternal grandmothers of the nursing infant. Courtney Meehan lived with the Aka, tropical forest foragers of the Central African Republic, and observed mothers with infants $<$35 months old. She recorded behavior and calculated quantitative measures of the mothers' energy expenditure. Assistance to mothers by alloparents significantly reduced mothers' working energy expenditure by up to 216 kcal during the 9-hour observation period. Assistance from grandmothers provided a one-to-one exchange of maternal direct care, while direct infant care from fathers decreased maternal care by almost 4 to 1 (Meehan et al. 2013). These are important intergenerational resource transfers between alloparents to mother and child which carry nontrivial and measurable costs for the alloparents.

In a review of 45 studies of mostly natural fertility societies, (Sear & Mace 2008) found universal evidence of assistance in rearing offspring, leading them to suggest that alloparenting is a human universal. They also found that the benefits of alloparenting varied greatly and often depended on the kinship relation. Assistance from maternal grandmothers and siblings living with the mother tended to improve offspring survival rates. Assistance from paternal grandmothers was associated with both greater offspring survival and mortality, depending on the population, whereas help from fathers showed benefits in only about one-third of the studies. Sear and Mace mention the likely importance of matrilocal vs. patrilocal residence as a determinant of these costs and benefits, but do not provide a formal analysis.

These examples illustrate that, while allocare is ubiquitous in human societies, the benefits vary widely across societies. In some instances, what may seem to be alloparental care may, in fact, involve costs to the recipient that could detract from

reproductive success. Hrdy (1999, 2009) described the loss of tens of thousands of infants who died due to the practice of using wet nurses in seventeenth- and eighteenth-century Europe. Wet-nurses provided a type of allomaternal care by breast-feeding the infants of other women. Mothers living in more urban areas would often send their infants to a rural wet nurse for up to three years due to the need to work – there was no nursing support for working mothers – and because of an ideological aversion to the "bestial" nature of nursing by wealthier women. These same women also believed that nursing would ruin their figures and not allow them to wear fashionable clothing. Unless employed or enslaved by wealthy families, the rural wet nurses often provided too little milk to too many infants, under neglectful conditions, resulting in high infant mortality.

Surveys of the literature on breeding systems reported that between 5–10% of mammalian species are cooperative in breeding (Hrdy 2009). Building on decades of research, Hrdy (2009), Kramer (2007), Meehan et al. (2013) and others make the case that humans are the only species of cooperatively breeding ape. A systematic review by Lukas & Clutton-Brock (2012) provided a framework for characterizing breeding systems that recognizes only 1.8% of mammal species as cooperative breeders and, in contrast to the others just cited, they do not include humans among these species. They make a distinction between cooperative breeders, communal breeders, and social breeders, with cooperatively breeding species being those in which most of the females do not breed regularly and instead provide alloparental care to the offspring of a breeding female (typically only one) to whom they are genetically related, often as siblings or half-siblings. Humans clearly do not meet this definition as virtually all women in traditional forager, horticultural, and pastoral societies reproduce regularly if fecund (Bogin 2001).

Lukas and Clutton-Brock (2012) define other characteristics of cooperative breeding species, such as near-exclusive monogamous breeding and birthing of litters of altricial young. Interested readers may consult their article for further details. Only nine species of nonhuman primates,[8] all members of the New World Callitrichidae (marmosets and tamarins), meet all of their criteria for cooperative breeding.

Lukas and Clutton-Brock (2012: 2151) define communal breeders as those species in which, " . . . most adult females breed regularly and share care such as allonursing or feeding offspring . . . " They identified four species of primates as communal breeders (two New World monkeys and two lemurs). They do not include *Homo sapiens* in their list of communal breeders because in communal breeding species the females are close genetic relatives, usually sisters or half-sisters, as the females remain in their natal social group. Human alloparents may be close genetic relatives, but often are not due to the variety of post-marital residence patterns. Surveys of traditional foragers and nonforagers find that human societies most often practice patrilocal post-marital residence which requires women to live with their husband's

[8] 1.5 percent of the 612 total primate species listed at www.alltheworldsprimates.org/Home.aspx

family. This separates sisters from each other. Matrilocal residence, which would keep sisters together, is the least common type of human residence (Marlowe, 2010).

Lukas and Clutton-Brock (2012) define social breeders as species in which the females live in groups and virtually all breed, but rarely, if ever provide allomaternal care to others. The majority of the nonhuman primate species are social breeders. It is important to emphasize that the ranks of the social breeding species include all of the African apes, which are closest phylogenetically to humans. Humans, in contrast, are not social breeders, as alloparental care is the rule in human societies.

Human Biocultural Reproduction vs. Cooperative and Communal Breeding

Humans certainly show cooperative and communal assistance, support, and mutual aid in relation to reproduction, care of pregnant women, and the rearing of offspring. Indeed, humans have been called ''super cooperators'' in these regards (Nowak & Highfield 2011). Lukas and Clutton-Brock (2012) observed that, as none of the African apes practice either cooperative or communal breeding, it seems probable that these cooperative tendencies in raising offspring evolved in hominins after they split from the last common ancestor with living apes. They are not original in this proposal, as it was also offered by Hrdy (1999, 2009) and Burkart et al. (2009). I agree that the ''super cooperation'' practiced by humans is an evolutionarily derived trait. I differ, however, from Hrdy and Burkart et al. in that they equate the human style of reproduction with cooperative breeding in nonhuman mammals, whereas I find fundamental differences that set human reproduction apart from any other type of cooperative/communal breeding.

The most important difference between the human system and that of nonhuman cooperative breeders is that the provisioning of human allocare and related resource transfers is often uncoupled from genetic relatedness. This is rare in other species. The reason for this rarity is the restriction of breeding to usually one female and one, or only a few, males amongst cooperative breeders (Lukas & Clutton-Brock, 2012). This restriction leaves the siblings and half-siblings of the breeders with little choice for direct fitness enhancement. William Hamilton (1936–2000) formally identified the next best strategy for the nonbreeders, which he called inclusive fitness (Hamilton 1964) and is also called kin selection. By assisting their close genetic relatives, the nonbreeding alloparents help to ensure that copies of parts of their own genome survive in their close genetic kin.

Hamilton expressed inclusive fitness as an equation, now known as Hamilton's Rule. The formula is $C < rB$; where C is the cost in fitness to the actor, r is the genetic relatedness between the actor and the recipient, and B is the fitness benefit to the recipient. If the reproductive cost to the actor is less than the product of genetic relatedness multiplied by the reproductive benefit to the recipient, then a ''helpful'' behavior toward the recipient's reproductive success is expected to be favored by natural selection. If reproductive costs are greater than the product of rB, then apparently altruistic behaviors such as alloparenting will never evolve. The review

of evidence presented here shows that human biocultural reproduction is a system of alloparenting which violates Hamilton's Rule in that alloparents often incur costs greater than the product of *rB* because, unlike in other cooperating species, the genetic relatedness between the human actor and recipient is often small or, essentially, zero. As the value of *r* approaches zero the costs of alloparenting far exceed the benefits.

People are able to "violate" the rule because human biocultural reproduction is based upon social relationships that are qualitatively distinct, in their definition, extent, and complexity, from those of cooperative or communal breeding systems. Due to human nongenetically based behavior the cost in fitness to the alloparent becomes quite difficult to measure, as the actor is part of elaborate kinship networks and families created by marriage, which structure the flow of energy and other resources in ways that do not always map onto genetic relatedness. Equally important, the obligations and prohibitions entailed by kinship categories result in emotional costs and benefits which may offset any material or biological costs. The fitness benefit *B* to the recipient also becomes more complicated to measure for the same reasons. In addition to the metrics used to understand evolutionary selection pressures in nonhuman animals, such as survival, growth, and future reproduction, human reproduction also involves nonbiological contributions to the members of the social group, such as emotional affection, the teaching of culturally acquired skills, and transfers of material goods and other wealth (Gurven et al. 2012). In these ways, reproduction differs from cooperative and communal breeding because it not only contributes to the inclusive fitness of parents and genetically-related helpers, but it also serves to continually reproduce and recreate the social, economic, political, and moral cohesion of social group members.

Humans, of course, rely upon genetic kin to support and help raise dependent offspring, but human alloparenting is unusual, perhaps even unique, among mammals in relying on many nonkin for care and provisioning. Human care for dependent infants and children is structured by sets of local, culturally-defined rules and the phrase *biocultural reproduction* seems a better description of human behavior. It seems likely that some form of genetically-driven cooperative/communal breeding strategy in the earliest species of *Homo* would have been necessary to promote and sustain the evolution of childhood, with its increased requirements for prolonged and more intensive care. At present, this is speculation as the needed evidence may not be reconstructed based upon current fossil evidence. What does seem clear is that, over evolutionary time, the bonds of genetic relatedness which initially fostered the ancestral hominin strategy of raising offspring were replaced with the current flexible, culturally-based systems of kinship and marriage that prescribe that a range of individuals provide care.

Childhood and Biocultural Reproduction

Humans are highly unusual among mammals in weaning offspring before they are nutritionally independent. Extensive provisioning of dependents by genetic and

social (nongenetic) kin allows human mothers to ''stack'' offspring, that is to give birth at relatively short intervals, and spread the burden of provisioning across alloparents, thus facilitating both relatively high fertility, despite the intensity of investment, and high survival of each offspring (Bogin 2006; Gurven & Walker 2006).

Human reproductive behavior increases genetic fitness of parents over that of any other ape by enabling women to give birth to new offspring while allowing existing dependent offspring to receive care and feeding from close kin and other members of the social group (Bogin, 2001, 2006). The evolution of human childhood may be viewed as critical to this human reproductive strategy. The biological constraints of childhood – including an immature dentition, small digestive system, and a calorie-demanding brain that is both relatively large and growing rapidly – necessitate care and feeding from older individuals, which greatly expands the opportunities for allocare within human societies. Although women tend to provide most of the care to infants for at least 3 years in most traditional societies (Marlowe, 2010; Sear & Mace, 2008), many other individuals are also involved in the care, provisioning, and social lives of infants and young children. Indeed, by the stage and age at which human mothers tend to wean their infants – between 30 to 36 months in forager and other subsistence societies – a greater percentage of the care of offspring tends to be provided by other family members including fathers, older siblings, aunts, and grandmothers (Gettler et al. 2012; Hawkes et al. 1997; Hrdy 1999; Kramer 2002; Meehan et al. 2014; Valeggia & Ellison 2004).

The Allometry of the Growth of the Human Child Releases Nurturing and Care-Giving Behaviors

The pattern of growth of infants and children may be a bio-psychological stimulus to release parental and alloparental behaviors from older members of the social group. The central nervous system, in particular the brain, follows a growth curve that is advanced over the curve for the body as a whole (Figures 2.3 and 2.8). The brain achieves adult size when body growth is only 40% complete, dental maturation is only 58% complete, and reproductive maturation is only 10% complete. The allometry of the growth of the human child maintains an infantile appearance (large cranium, small face and body, little sexual development). A series of ethological observations (Lorenz, 1971) and psychological experiments (Alley, 1983; Todd et al. 1980) demonstrate that these growth patterns of body, face, and brain allow the human child to maintain a superficially infantile appearance longer than any other mammalian species (Box 4.1). The infantile appearance of children is perceived as both "cuteness" and "helplessness," which stimulates and facilitates parental investment. In biology, acts of parental investment are measured by the allocation of resources, such as time or energy, to offspring that occurs at some cost to the parents. These acts relate to life history stages of growth, development, and maturation and are centered on decisions regarding when to begin reproducing, how many

Box 4.1 **The evolutionary psychology of childhood**

Reproductive success is the major force behind the evolution of all species. Part of the reproductive success of the human species is due to the intense investment and care that parents, and other individuals, lavish on infants and children. In the course of human evolution, at least since the appearance of the genus *Homo* in the last two million years, patterns of growth were shaped by natural selection to promote and enhance parental investment. One way this was accomplished was by stimulating what may be called the "psychology of parenting."

Konrad Lorenz (1903–1989) stated that the physical characteristics of mammalian infants, including small body size, a relatively large head with little mandibular or nasal prognathism, relatively large round eyes in proportion to skull size, short thick extremities and clumsy movements, inhibit aggressive behavior by adults and encourage their caretaking and nurturing behaviors (Lorenz 1971). Lorenz believed that these infantile features, which he called the *kindchenschema* (baby schema), trigger "innate releasing mechanisms" in adult mammals, including humans, for the protection and care of dependent young. Gould (1979) questioned the innateness of the human response to infantile features. Such behavior may be, " . . . learned from our immediate experience with babies and grafted upon an evolutionary predisposition for attaching ties of affection to certain learned signals" (p. 34). The important point is that whether innate or learned the resultant behavior is the same.

There seems to be a pan-human ability to perceive the stages of human postnatal development, from infant to post-reproductive adult, and respond appropriately to each. An elegant series of experiments performed by Todd and colleagues (Todd et al. 1980), show that human perceptions of body shape and growth status are consistent between individuals. The participants in the experiments were 40 or so North American university undergraduates, all childless and with little experience in childcare. The findings of psychology experiments in North America and much of Europe are usually based on university undergraduates, who are likely not representative of the larger population and certainly not at all like most people in the world today and throughout human history. The acronym WEIRD – Western, Educated, Industrialized, Rich, Democratic – was coined to characterize the uncharacteristic nature of North American/European undergraduates and the bulk of the psychological and sociological research in which they are engaged (Henrich et al. 2010).

The WEIRD undergraduates of Todd and colleagues' experiment were shown a series of profiles of human skull proportions and they could easily arrange them correctly into a hierarchy spanning infancy to adulthood. The students could also ascribe maturity ratings to skull profiles that were geometrically transformed to imitate the actual changes that occur during growth

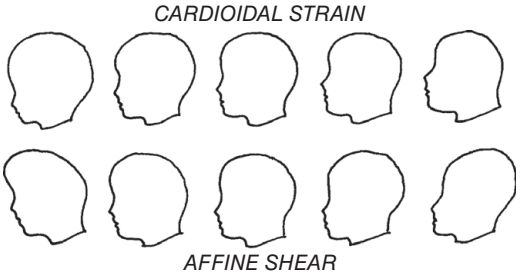


Figure B4.1.1 Two of the mathematical transformations of human head shape used in the experiments of Todd et al. (1980). The middle profile in each row was drawn from the photograph of a ten-year-old boy. The transformations were applied to this profile of a real child. The cardioidal strain transformation is perceived by most adults as growth. The affine shear transformation is not perceived as growth.

(Figure B4.1.1). This perception was selective because a variety of other types of geometrical transformations elicited no reports of growth or maturation. When the growthlike mathematical transformations were applied to profile drawings of the heads of birds and dogs, human subjects reported identical perceptions of growth and maturation, even though in reality the development of these animals does not follow the human pattern of skull shape change. Even more surprising is that subjects reported the perception of growth when the growthlike mathematical transformations were applied to front and side-view profiles of Volkswagen "beetles," objects which do not grow.

In another series of experiments with 120 US undergraduates, Alley (1983) studied the association between human body shape and size, and the tendency by adults to protect and "cuddle" other individuals. In the first experiment, participants were shown two sets of drawings. One set was based upon two-dimensional diagrams depicting changes in human body proportion during growth. Alley's versions of these diagrams were called "shape-variant" drawings (Figure B4.1.2a). Alley's second set of figures were called "size variant" drawings (Figure B4.1.2b). He used the middle-most, "six-year-old" profile in the shape-variant series, to construct sets of figures that varied in height and width, but not in shape. Note that these figures have no facial features, or genitals. Perceptual differences between figures are due to body shape or size alone.

In the first experiment, the participants were shown pairs of the shape-variant drawings (i.e., profiles of a newborn and a 6-year-old, a 2-year-old and a 12-year-old) or pairs of the size-variant drawings and asked to state which one of the pair they, " . . . would feel most compelled to defend should you see them being beaten." In another experiment they were asked about their feelings to "hug or cuddle" the person depicted. The results of both experiments, summarized in Table B4.1.1, found a fairly strong reported willingness

Table B4.1.1 Mean reported willingness to defend or cuddle persons of different body proportions (Alley, 1983).

Age portrayed (years)	Defend	Cuddle
Newborn	7.7 (1.7)	3.4 (2.3)
2	7.1 (1.7)	3.4 (2.0)
6	5.8 (1.8)	3.2 (2.2)
12	5.0 (1.9)	3.0 (1.9)
25	4.3 (1.9)	2.7 (2.2)

Note: Standard deviations are given in parentheses.

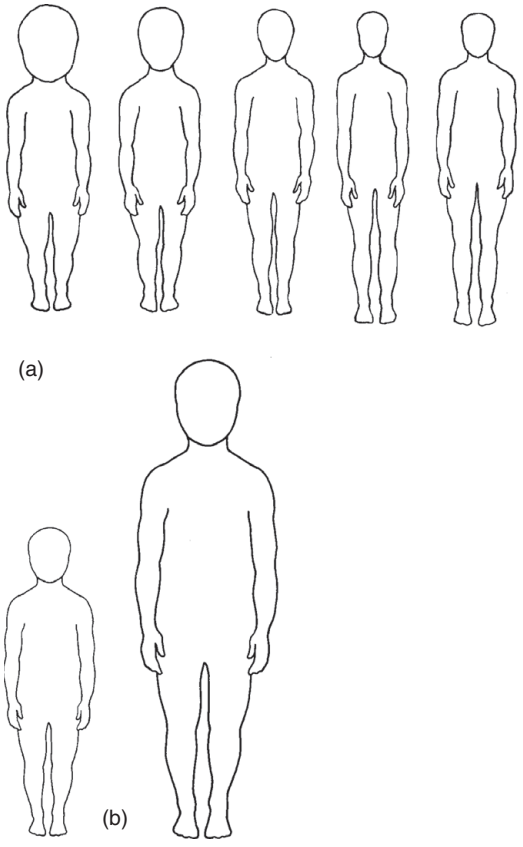


Figure B4.1.2 (a) The series of five shape-variant drawings used in the experiments of Alley (1983). These drawings show the typical body proportions of a male at (from left) birth, 2, 6, 12, and 25 years of age. (b) An example of the size-variant pairs of drawings used in the experiments of Alley (1983).

to defend "newborns" and "two-year-olds," and a moderate willingness to defend "older" persons. The reported willingness to cuddle decreased with the "age" of the drawings. Placed in the context of the ethological study of parental caregiving in mammals and birds, Alley believed that his results demonstrated a general tendency, to protect or cuddle others based on the perception of maturational status.

McCabe (1988) reviewed the work of Alley and other similar studies and concluded that this research indicates that adults are more likely to protect or nurture individuals with "neotenous" facial features. McCabe defined such features as having a relatively large ratio of cranium size to lower face size. He also noted that even if adults are not aware of these facial features they stimulate caretaking feelings and behaviors. McCabe's review included studies of the facial features of nursery school-aged children under court protection for abuse compared with nonabused age-matched controls. The abused children had smaller ratios of the cranium/lower face – that is, they were less "neotenous" or "cute" – than the nonabused controls.

More recent research has begun to elucidate the neurological and hormonal basis of the *kindchenschema's* effect on potential infant and child caretakers. In one series of experiments, Glocker and colleagues (2009) used controlled manipulation of the baby schema in images of infant faces. The researchers showed participants (16 nulliparous women aged 20–28 years old) 3 images of the same baby. One image was the original frontal face photograph, a second image was enhanced to a "high baby schema," with rounder face, higher forehead, bigger eyes, smaller nose and mouth, and the third image was manipulated to a low baby schema, with narrower face, lower forehead, smaller eyes, bigger nose and mouth. The participants observed the images and rated them for "cuteness" while being neuroimaged with functional magnetic resonance imaging. The researchers reported that with baby schema enhancement there was greater activation of the nucleus accumbens, " . . . a key structure of the mesocorticolimbic system mediating reward processing and appetitive motivation, in nulliparous women. Our findings suggest that engagement of the mesocorticolimbic system is the neurophysiologic mechanism by which baby schema promotes human caregiving, regardless of kinship" (p. 9115). In other words, people with little experience with infant and child caregiving have "built-in" brain mechanisms to promote alloparenting. Other research has extended these findings to men with little experience with infants and children and also shown that the hormone oxytocin is positively associated with motivation to protect and care for youngsters (Luo et al. 2015). These mechanisms are adversely influenced by opioid drug use (Wang et al. 2018), which is an increasingly pernicious problem in many parts of the world and results in neglect and abuse of infants and children under the care of the drug abusers.

These psychological experiments and case control studies provide support for the arguments developed in this chapter for the evolution of human

childhood. Despite the "WEIRDNESS" of the participants in most of these studies, it is reasonable to conclude that the *kindchenschema* of human infants and children promote appropriate parental behavior by older individuals, regardless of their experience or genetic relationship.

offspring to have, how often to reproduce (i.e., birth spacing), and how much time and energy to give to each offspring (Lancaster & Lancaster 1983; Stearns 1992).

The allometry of human growth promotes parental investment by maintaining the potential for nurturing behavior of older individuals toward both infants and dependent children. The desire to feed and protect children with infantile features is common to many human societies, but not all. As described in Chapter 1, children have been exploited, abused, and enslaved throughout history (see also Lancy 2014; LeVine 2009; Sommerville 1982).

The relatively slow rate of body growth and small body size of children reduces competition with adults for food resources, because slow-growing, small children require less food than bigger individuals. A 5-year-old child of average size (the 50th centile of the NCHS reference curves for growth) and activity, for example, requires 22.7% less dietary energy per day for maintenance and growth than a 10-year-old juvenile on the 50th growth centile (Guthrie & Picciano, 1995; Ulijaszek & Strickland, 1993). Thus, provisioning children, though time consuming, is not as onerous a task of investment as it would be, for instance, if both brain and body growth were both progressing at the same rapid rate.

Nonfamily members of the social group also provide care because they see the need to do so. Recent work among Hadza foragers found that physical proximity between unrelated individuals was as likely to result in cooperation, including childcare, as was genetic relatedness (Apicella et al. 2012). Apicella and colleagues also reported that Hadza camps are comprised of people with distant genetic relationships: First order genetic relatives comprised less than 10% of residential camps. Hill and colleagues (2011) explained the reason for the low percentage of first-order genetic relations. The researchers surveyed 32 present-day foraging societies, including the !Kung (Ju/'hoansi), the Ache of South America, and the Hadza and reported that human hunter-gatherer societies have a social structure that is unique among all primates. Hill et al. found that, on average, 75% of individuals in residential groups were genetically unrelated or at least not genetically related by descent from common parents or grandparents. This is due to the practice by both men and women of dispersing or remaining in their natal group. Migrations to new groups dilute genetic relationships and require social kinship designations to help structure new relationships.

Another way that humans are distinct from other primates is that people will marry and reproduce according to the rules of kinship, often with disregard to their degree of genetic relationship. Nonhuman primates tend to avoid consanguineous

matings by having either males or females disperse from the natal groups after puberty. Cross-culturally, people may prefer marriage partners of almost any degree of genetic relatedness and use the flexibility of social kinship naming systems to allocate potential marriage partners to acceptable social categories (see Bogin et al. 2014b for examples). The ways in which human social groups form necessitates flexible forms of childcare. Indeed, all human social groups include a variety of childcare and feeding arrangements which make use of both biological kin and socially-defined relationships such as in-laws by marriage, **fictive kin**, servants, friends, or employees paid in cash or kind (Ellen 2018; Lancy 2014). These socially-based childcare arrangements, which operate in addition to genetic kinship, are the essence of what sets human biocultural reproduction apart from the forms of cooperation found in other species.

The Nature of Human Biocultural Reproduction

As practiced by human societies, biocultural reproduction may be defined as the set of marriage and kinship-based rules for extra-maternal cooperation in the production, feeding, and care of offspring. It is vitally important to state here that I am not discussing human mating, which takes place within and outside of stable pair bonds and marriage. Instead, I focus on the cultural rules for the care and provisioning of offspring, regardless of the genetic relationship to the caregivers. Marriage and kinship-based rules include delegating responsibilities for feeding, clothing, housing, and educating the infants, children, and youth of the society, and often similar rules for the care of women of reproductive age.

Reiches et al. (2009) conceptualized the human style of care and feeding for offspring and their mothers as the "pooled energy budget" hypothesis. They defined the pooled energy budget as the combined energetic allocations of all members of a reproductive community that might result in direct or indirect RE (reproductive effort). "These transactions can take many forms at one time and vary across the life course . . . Individuals draw on the pooled energy budget by consuming calories and by diverting time and energy [to] reproduction. They contribute by diminishing their own energetic costs and by contributing to the energy budgets of others. The output of the pooled energy budget is the production of new individuals" (p. 424). According to Reiches and colleagues, the pooled energy budget allows human women to sustain a higher fertility and greater survival of their offspring than would be possible for energetically isolated individuals (Figure 4.15).

It seems reasonable that human childhood and biocultural reproduction coevolved as derived characteristics of our species. In addition, human biocultural reproduction likely also required the evolution of other human-specific characteristics, such as the cognitive capacities that allow for what is often described as ''theory of mind'' (Povinelli & Preuss 1995; Povinelli & Vonk 2003; Premack & Woodruff 1978). This phrase is often taken to mean the ability of one individual to impute or attribute mental states to self and to others. To have ''theory of mind'' requires an appreciation that others may have desires, intentions, or beliefs that may be different from one's

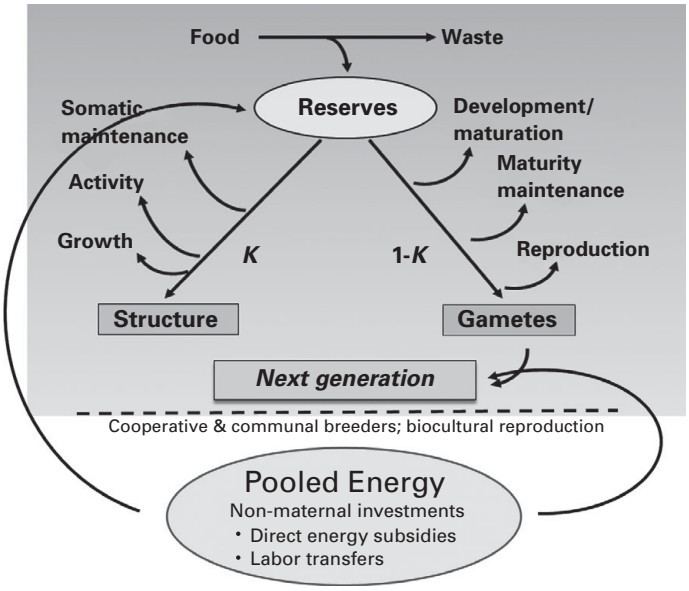


Figure 4.15 Energy trade-offs in life history. For all animals, energy is extracted from food consumed, digested, absorbed, and metabolized. Otherwise, the energy is lost in waste (feces, metabolic heat loss, etc.). Available energy may be used to build biological reserves, which may be either used by the individual's body for maintenance (including basal metabolic activity and immune activity), physical activity, or growth. These energy uses are labeled "K." The remaining energy, labeled "1-K" (with 1 = 100%), may be used for developing and maintaining the reproductive system and for all aspects of reproduction (gamete production, mate acquisition, gestation, parental investment, etc.). For animal species practicing cooperative and communal breeding, and for human biocultural reproduction (below the broken line) there is an additional input of energy from social group members who directly contribute food or provide labor to breeding females and to their offspring that reduces the energy expenditure of the breeding female (reduces her "K"). The helpers contribute to a "pool" of energy that is shared to enhance reproductive success. Nonhuman animals share the pooled energy with close genetic relatives. Humans share the pooled energy with both genetic kin and socially defined kin. Original figure.

own and, based upon this appreciation, the ability to predict behavior on the basis of such states. There are debates as to which animal species have ''theory of mind.'' Rather than enter into these debates here let us accept that living humans have a ''theory of mind'' and related behaviors which surpass those of other primates. Let us also assume that these human cognitive and behavioral capacities evolved, or were at a minimum augmented substantially, since the hominin split from the last common ancestor with chimpanzees.

Burkart et al. (2009) proposed that cooperative breeding by human ancestors facilitated the evolution of larger brains and cognitive development. Lukas and Clutton-Brock (2012) seem to reject this proposal as they find no evidence of an association between cooperative breeding and brain size in mammals, especially

primates or birds. Alternative proposals are that other selection pressures, perhaps related to greater reliance on stone tools, the use of fire, and other extractive technologies (Wells & Stock 2007), the development of language skills (Locke & Bogin 2006) or the increasing size and complexity of social groups (Dunbar 2009) initiated brain/cognitive expansion. All these proposals are speculative, but all assume an evolutionary trend within the hominin lineage toward greater cooperation in tool production, foraging, and social relations which may have fostered alloparenting and social cooperation in reproduction. The transition to biocultural reproduction may have coevolved with the biological, behavioral, and cognitive characteristics which coalesced with the appearance of *Homo sapiens*.

Human ''theory of mind'' emerges during development in a sequence from infant to child, juvenile, adolescent and adult. Two of the crucial changes in cognition which underlie theory of mind emerge at the transition from human infancy to childhood, at ~3–4 years of age. These are the capacities for pretense and the attribution of false belief in other people. Bananas provide an example of pretense. Infants accept only the reality of the banana as a fruit that may be eaten. Children are capable of the pretense of a banana as a telephone or some other imagined object. A common test for false belief is to allow a child and another person to see a banana hidden in a basket. The other person then leaves the room; the banana is rehidden in a box and then the other person returns to the room. Infants and younger children will expect that the other person knows that the banana was rehidden. Children with theory of mind will make the attribution of false belief and say that the other person thinks that the banana remains in the basket. Videos of theory of mind experiments are available online. With these new abilities, the child may use pretense and attribution of false belief in many contexts. A kindly neighbor may be called an ''aunt,'' while two unrelated but socially-close girls may call each other ''sisters.'' Older individuals also make use of pretense toward the child, as illustrated by the kindly neighbor accepting the kinship name of ''aunt'' as well as the associated responsibilities for allocare, even though she is genetically unrelated to the child. Many of the human social rules for alloparental care of offspring are a ''pretense'' of the genetic relatedness which drives cooperative breeding in other species. In the larger social arena of human behavior, these types of pretense and false beliefs are essential supports for the rules for kinship and marriage. The essential point is that, in the human species, relationships defined by marriage and kinship rules often take the place of genetic closeness.

Humans, of course, build on the foundations of primate biology and psychology that foster intimacy between mother and offspring. These foundations are necessary for human biocultural reproduction, but the degree of mother–offspring intimacy in nonhuman primates would not likely be sufficient to sustain the more complex and expansive human networks of affection and alloparental care that extend to many kin and nonkin. A deeper ability to impute or attribute mental states to self and to others is required. Tomasello and colleagues (Tomasello et al. 2005) proposed that this new, deeper ''theory of mind'' is found only in the human species and they label it as ''shared intentionality.'' To evolve from communal or cooperative breeding to

the human practice of biocultural reproduction would have required the new emotional capacities of shared intentionality, and these may have required the stages of childhood and adolescence for development.

One universally human way to share intentionality and make sure that others understand one's own mental state is to tell stories. The word "story" is meant to describe a narrative that may relate information, emotional states, moral guidance, or a combination of these. Storytelling is the central way in which people create and maintain pretense in kinship and all social relationships. Research into storytelling has a long and rich history in the humanities, philosophy, and sociology, but only recently have evolutionary biologists and bioanthropologists turned their attention to this subject. In one analysis, Daniel Smith and colleagues explored the association between storytelling and hunter-gatherer cooperative behavior. They report from field studies of the Agta, a Filipino forager population, that good storytellers are socially desirable social partners and have greater reproductive success than others (Smith et al. 2017). Good storytelling helps to organize cooperation, maintain social and sexual equality, and reduce conflict – all are needed traits for survival in foraging societies. Skilled storytellers promote their own reproductive fitness and because their narratives often convey messages about the moral value of cooperation, they all promote benefits for group selection.

Human beings are compelled to tell stories and storytelling is a human universal. Many of the findings of Smith and colleagues are applicable to all types of human societies. Anyone with experience with infants, children, juveniles, and adolescents knows that these young people love to listen to stories and produce their own narratives. Could it be that the human capacity for storytelling coevolved with the life history stages of childhood and adolescence? Young people become more skilled at narration in terms of content, fluidity of speech, manipulation of syntax and grammar, and the rapidity of narrative production as they mature. These observations and others led Locke and Bogin (2006) to propose that the whole of human life history from birth to adulthood is required for the development of language in every individual person and the evolution of symbolic language in our species.

Walter Goldschmidt (1913–2010) emphasized the importance of another human emotion called **affect hunger**, which he defined as " . . . the urge to get expressions of affection from others" (Goldschmidt 2006, p. 47). For humans this often means affection from many others. Konner (2010) discussed examples of the importance of affection with many people, in addition to the mother, for the healthy development of human children in forager societies. Especially noteworthy is the affection between children with members of the social group who serve as alloparents and also as teachers of essential survival skills. These people may be quite distantly related to the child in any genetic sense. Hrdy (2009, 2016) focuses on the ways in which intense alloparenting by human ancestors "changed our minds," reflecting the fact that infant attachment was spread across many alloparents rather than just the mother. She provides evidence that having a broader range of alloparents and attachments changes mental phenotypes and promotes the ability and desire to adopt the mental perspective of others (i.e., shared intentionality, pretense, social kinship,

and storytelling). Hrdy also proposes that the new emotional capacities of hominin ancestors promoted those human cognitive accomplishments which distinguish our species from other apes.

This point was elaborated by Silk and House (2016, p. 7) in their cultural group selection hypothesis which,

> . . . helps to explain how human societies are able to combine high levels of altruism with low levels of relatedness, a combination that is not observed in other mammalian species. Cooperative breeding in human groups may be part of a broader system of group level cooperation in which mothers receive help feeding and caring for their offspring; the families of unsuccessful hunters are fed; aid is given to the sick and the aged; and collective action problems are resolved.
>
> If the constellation of selective pressures that have shaped prosocial behaviour in humans and cooperatively breeding mammals differ, then it is likely that human prosocial behaviour may also be motivated by a distinctive set of social preferences and psychological adaptations. As predicted by cultural evolution and interdependence models, prosociality in our species is highly influenced by social norms. Cultural variation in cooperation emerges in middle childhood, a time when children seem to become sensitive to social norms within their communities.

Based upon these analyses, one may conclude that human emotion and cognition are among the features that set our species apart from other mammals, even the apes, and undergird our capacity for emotional attachments to a range of individuals other than direct genetic kin. Our ability to form strong social bonds, not only with mothers but also with many other people through social institutions such as marriage and kinship, are critical to understanding how biocultural reproduction operates.

Why Do Humans Rely upon such Diversity in Kinship and Allocare Strategies?

One hallmark of the human system of biocultural reproduction is extreme flexibility in behavior and thinking. Local ecological conditions vary across the range of environments that traditional subsistence-level human societies inhabit today and have inhabited for millennia. It is likely that the effective patterns of allocare, including kinship and marriage-defined rules for provisioning, holding, protecting, and educating the young varied remarkably over time and place. One well researched ethnographic example is the Hadza, who rely on tubers as a key food resource. Tubers are thickened underground parts of a plant stem or rhizome. The potato is an example of a tuber. Tubers are difficult to find and difficult to dig and collect. Hadza grandmothers have the knowledge to find tubers and the physical strength to dig them, allowing them to play a central role as allocaregivers (Hawkes et al. 1997).

Another common pattern of childcare in many traditional cultures is to have juveniles assume caretaking responsibilities for younger children. This occurs among two well-studied African hunting and gathering societies, the !Kung (Konner, 2010) and the Mbuti (Turnbull 1983). In these societies, mothers carry their infant and still

nursing child (nursing a child to age 4 years is common in these cultures) with them while foraging. Weaned children must stay "home," that is at the base camp, as pre-adolescent children have neither the strength nor stamina to follow their parents while gathering or hunting. At !Kung camps children of various ages play together within the camp boundaries while juveniles discharge many caretaking functions for younger children. The children seem to transfer their attachment from parents and other adults to the juveniles, behaving toward them with appropriate deference and obedience. The **age-graded play group** functions to transmit cultural behavior from older to younger generations and to facilitate the learning of adult parental behavior (Konner, 2010). Of course, the children and juveniles are never quite left on their own, as there is always one adult or more in camp at any time, but this person is not directly involved in childcare. Rather, he or she is preparing food, tools, or otherwise primarily engaged in adult activities.

The Mbuti, who are nomadic hunters and gatherers of central African rain forests, have a similar childcare arrangement. After weaning, toddlers enter the world of the *bopi*, the Mbuti term for the children's playground, but also a place of age-graded childcare and cultural transmission. Between the ages of 2–3 years to 8–9 years of age, children and juveniles spend almost all of their day in the *bopi*. There they learn physical skills, cultural values and, even, sexual behavior. Turnbull (1983, pp. 43–44) notes, "Little that children do in the *bopi* is not of full value in later adult life."

The age-graded play group provides for both the caretaking and enculturation of the young, freeing the adults from these tasks so that they may provide food, shelter, and other necessities for the young who may be at various stages of development. A formal mathematical treatment of the reproductive benefits of an age-graded division of labor for alloparenting was provided by Kramer (2014) and showed that a woman may be pregnant, have a child weaned within the past year and have one or more older offspring simultaneously. Kramer carried out ethnographic fieldwork with Maya horticulturalists of Mexico and found that the average Maya woman will be able to give birth and rear 7 offspring during a 20-year reproductive career. As children, juveniles, and adolescents, those offspring contribute time and labor toward the alloparenting of their younger siblings, cousins, and others. It is not until the offspring are ~20 years old that they achieve independence in terms of the ability to leave their natal social group to begin their own families.

The selective advantage of a greater number of surviving offspring afforded by age-graded caretaking may, in part, account for the evolution of childhood in the hominin lineage. In addition, the play group, in the protective environment of the home base or camp, provides children and juveniles with the freedom to explore and experiment, which tends to encourage learning, tool using, socialization, physical development, and emotional well-being (Bogin 1999a, 2002). These examples illustrate how humans, unlike other species, have adopted a system of biocultural reproduction and offspring care that is flexible and responsive to local opportunities and constraints. Having our kinship and marriage systems, including fictive kinship and many styles of marriage practices, emerge locally, allows greater opportunities for plasticity in behavioral accommodation of local ecological realities. In contrast,

other species that practice cooperative or communal breeding have relatively narrowly constrained systems of collective care based on close genetic relationships.

There is, of course, no simple correlation between types of marriage and kinship systems and local ecologies. Styles of human alloparental care likely developed due to many historical factors that are often challenging to reconstruct from the fossil record. I only mean to emphasize that human alloparental care based on biocultural rules for kinship and marriage responsibilities provide human populations with enormous flexibility to adapt effectively to the unusually wide range of ecologies that our species has colonized.

Biocultural Reproduction and Lifetime Reproductive Effort

The biocultural nature of human reproduction and the childhood stage of life history have consequences for **lifetime reproductive effort** (LRE). LRE may be defined as the metabolic energy devoted to reproduction, relative to maintenance costs, over the average adult life span. To explain this definition of LRE it is best to take a step back and discuss RE. Within the life history theory literature, RE is often defined in terms of expenditures of energy and time in: (1) mating, that is, searching for, finding/attracting and keeping a mate(s); (2) offspring production, including gametogenesis, siring, and gestating offspring along with (in mammals) milk transfers during infancy; and (3) parental investment, which includes all expenditures of the parents' time and energy on any one offspring. All these expenditures are viewed as coming at a cost. That is a trade-off, to parental abilities to invest in their own somas and in other current or future offspring.

Human RE is highest between the ages of 18–40 years in most societies. This is the stage of maximum performance as defined in Chapter 2. In traditional societies, especially foragers, reproductive careers are typically initiated after 17 years of age, with 19 years being the modal age at first birth for women (Bogin, 2001; Marlowe, 2010). After about 35 years of age, women's biological capacity to conceive (fecundity) begins to decline slowly until menopause, which can be defined as the cessation of monthly menstrual cycling and the absence of ovulation in adult women. Menopause is usually reached by ~50 years of age, although there is evidence that it may occur earlier when women have been subjected to poor-quality environments in terms of nutrition, infection, or heavy workloads (Murphy et al. 2013; menopause is discussed in greater detail later in this chapter). Menopause is part of the stage of transition or degeneration (see Chapter 2). In societies in which there is no parity specific limitation on the number of children and hormonal contraception is not used, completed fertility rates may exceed 15 per women (Bogin, 2001). Relatively high fertility likely characterized much of human history, as opposed to modern high-income settings in which birth control is common and fertility typically falls below two per woman. Men's reproductive success follows closely that of women in the same population, although in some traditional settings a larger variance in men's total fitness is observed due to inter-male competition for access to mates via

aggression and/or cultural rules which channel women to older, wealthier, or more socially dominant men (Crews 2007; Marlowe 2010).

Extensive, high quality parental investment is a hallmark of human RE and it sets humans apart from all other mammals and other large-bodied apes (Lancaster & Lancaster 1983).[9] The intensive parental investment that characterizes human bio-cultural reproduction is made possible by kinship and marriage rules, noted above, which organize patterns of energy and labor transfers by alloparents to mothers and their offspring. This style of alloparental care allows human societies to maintain extremely high investment in each offspring, while also allowing human women to achieve the highest fertility and shortest inter-birth intervals of all the apes (Bogin 2001; Bogin & Smith 2012; Reiches et al. 2009). An example of parental investment based on my fieldwork with Maya families is given in Box 4.2.

Box 4.2 **Parental investment and growth**

In previous chapters my research with Maya people from Guatemala has been presented. In the Introduction the focus was on the Maya families that migrated from Guatemala to the United States. Those families fled Guatemala due to a war of genocide during the 1970s and 1980s by the Guatemalan military government that destroyed Maya villages and murdered at least 250,000 people (Lovell 2010). The Guatemala Human Rights Commission estimated that 70% of the Maya villages in the Ixil region were destroyed between 1981 and 1983 and 5.5% of the Ixil population was killed.[10] Thousands of Maya families fled to refugee camps in southern Mexico, but these camps were so squalid and unsuitable for living that the United Nations closed them. The Maya refugees could either return to the violence and poverty of Guatemala or move northward into Mexico and toward the United States. Migration to the United States was physically, economically, socially, and emotionally difficult. Many of the Maya refugees walked the 3,000 km from southern Mexico to the southern border of the United States (Burns 1993). Some died along the way.

The act of migration to the United States is based, in part, upon parental investment decisions. Interviews with Maya parents in both Guatemala and the United States reported that children raised in the United States were more likely to survive, grow better, and be healthier than children raised in Guatemala. Maya women stated that their infants and children are nearly twice the size as they would be if raised in Guatemala. The mothers of Indiantown,

[9] This chapter by Jane Lancaster influenced my thinking about the evolution of human life history more than any other book, article, or chapter. The content of the Lancasters' chapter is still up-to-date and very much worth reading.

[10] www.ghrc-usa.org/our-work/important-cases/genocide-cases/genocide-in-the-ixil-triangle/

Florida often ascribed the infant size difference to the infant formulas they use (Stebor, 1992). One mother explained,

> My daughter, Rosita, is four years old and is very small, I think she will be small all her life because she was so sick in Guatemala when she was a baby. She still doesn't eat well. Now look at my son who is almost a year old [born in the U.S.]. Already he is walking, which means his legs are very strong. He is twice the size of Rosita when she was a baby. I tell you the difference is milk [formula] (Stebor 1992, p. 106).

Maya women, both those pregnant and those with infants, received free or low-cost health care and nutritional supplements from the WIC (Women, Infants and Children) Program. Stebor reported that Maya women acknowledged the value of the WIC program, and justified the investment of time and money (lost wages) required to enroll in the program by pointing to their bigger, healthier babies and children. Maya infants in Florida, and probably Los Angeles, were fed more total food, including some breast-feeding along with formula feeding. The WIC program educates Maya mothers to follow hygienic practices when preparing and storing formula, and to use safe drinking water to mix the formula. In rural Guatemala, prenatal and postnatal infant medical care, childcare education, and safe drinking water were usually not available to the Maya families. Due to chronic poverty, infectious disease, and undernutrition for the rural poor in Guatemala, many women may not have been able to produce a sufficient quantity of breast milk. Infant formulas or cow's milk were too expensive for most Maya to purchase. Poor growth and development for Maya infants and children was the outcome. Given the unsafe and damaging environment of rural Guatemala it is easy to understand the parental investment decision to migrate to the United States, despite all the costs.

The final words on this topic come from a Maya father who moved his family to Los Angeles, California. The father, and his wife, had only primary school educations in Guatemala and worked as semi-skilled laborers in the garment industry of Los Angeles. All Maya adults in Los Angeles were short compared to European-Americans, but this man and his wife were shorter in height than most Maya parents in the same community. Yet, they had an 11-year-old daughter who was one of the tallest in the sample of Maya children and juveniles that I measured in 1992 (Bogin & Loucky 1997). The parents invested much of their resources and love into their children, who were expected to finish high school and go on to post-secondary education. The father explained that he hopes the investments he makes in his children will pay off for him when he is old and needs his children's support:

> What is in the future depends on what my children do. Here the children will stay, eat well, live better than in Guatemala. But many adults are returning to Guatemala, because children here abandon their parents. So, it depends on your children here. If you have good work, and children study well, and you earn well, it is possible to rent or even buy a good house, and not have to think about returning to Guatemala.

Lifetime Reproductive Effort

Early historical ''seeds'' of the modern notion of RE were planted by Ronald Fisher (1890–1962), who formulated the concept of ''reproductive value,'' which he described as the direct reproductive contribution that an individual of a given age, on average, will make to future generations. Fisher wrote that this is '' ... of some interest, since the direct action of Natural Selection must be proportional to this contribution'' (Fisher 1930, p. 27). David Lack (1910–1973) extended this concept when he analyzed egg clutch size in birds and reported a trade-off between the number vs. size of offspring, concluding that, ''The parental feeding rate tends to increase if the brood is larger, but not proportionately, so that each nestling gets a smaller share of the food in a large than a small brood'' (Lack 1947, p. 331). Lack's observations have been cited by subsequent theorists and are often considered the basis of the development of life history theory. Building on Fisher, Lack, and others, George Williams (1926–2010) proposed an elegant way to express the propagative part of these basic trade-offs. He called this ''Reproductive Effort'' (RE) and defined it as that portion of adult body mass devoted to reproduction per unit time (Williams 1966). There has been much discussion of how best to measure RE (reviewed in Bogin et al. 2014b), but William's definition remains the most commonly used method.

Eric Charnov and colleagues (Charnov et al. 2007) further extended the RE concept by devising a method for estimating a species' average LRE by taking the RE of an average reproductive bout multiplied by the average number of bouts across the reproductive lifespan of a female of that species. It is important to distinguish LRE from a related measure called lifetime reproductive success (LRS). The LRS of a female equals the total number of surviving offspring that she produces in her lifetime. It is well known that humans have the highest RS of all primates and a RS higher than many mammals (Bogin 2001; Walker et al. 2008). In contrast, LRE encapsulates the direct metabolic burden of reproduction over a female animal's life course and, thus, is a way of expressing the total amount of energy a typical female member of a species allocates to reproduction, on average, over her life course.

Specifically, Charnov et al. (2007) express LRE as:

(litters per year) × *(litter size)* × *(average reproductive lifespan)*
× *(offspring mass at independence / adult mass at first reproduction)*

Their use of the word ''litter'' also includes other terms to denote birthing events, such as clutches and broods. Calculating LRE for 54 lizard species, they find a mean LRE of 1.43 (1.3–1.5, 95% confidence interval) and, for 40 mammal species, a mean LRE of 1.41 (1.2–1.6, 95% CI). No primates were included in the analysis. These results indicate that, across this range of animal species, with varying growth and production rates, the average female in a typical mammal or lizard will generate a mass of offspring ~1.4-times her own body weight. Charnov et al. (2007: E135) explain that LRE is of theoretical importance because it '' ... is a key component of fitness ... and it encompasses the central core of 40 years of life history thought – reproductive allocation, size at maturity, and adult life span. This places it central to the study of life histories.''

Following the approach developed by Charnov and colleagues, Burger et al. (2010) calculated the average LRE of human women using data from 17 small-scale, traditional societies. By multiplying the average fertility rate, offspring size relative to maternal body mass, and reproductive lifespan of women within these populations, they calculate an average LRE of 1.45 (±0.12, 95% CI). This value is statistically indistinguishable from the predicted average LRE value calculated by Charnov and colleagues for other mammals, suggesting that humans may be typical mammals in this regard.

While an important initial step, the assumptions employed by Burger et al. do not fully account for the energetic and reproductive benefits accrued to human mothers through the processes of biocultural reproduction discussed above. Burger et al.'s analysis relies on the assumption that maternal per offspring RE is reflected in the size of an offspring at nutritional independence from maternal metabolism. Nutritional independence from the mother is relatively simple to estimate for a reptile – when the egg is laid or the viviparous offspring are birthed (Somma 2003 describes some rare exceptions). For most mammals, nutritional independence from the mother is achieved at weaning (end of lactation). Cooperative breeding mammals are exceptions, as some food provisioning may be common to weaned young.

Charnov et al.'s (2007) definition of nutritional independence is central to their estimation of LRE in nonhuman species. That definition of nutritional independence clearly does not apply to species with extensive nutritional transfers from alloparents. The human species is most notable in this regard. For humans, weaning is not equivalent with nutritional independence from the mother as she is likely to continue to supply food for many years. A further complication for humans is that both prior to weaning and for many years postweaning other people will supply food to infants, children, juveniles, adolescents, and even adults (Kramer & Ellison 2010; Reiches et al. 2009). Accordingly, the definition of LRE likely needs modification when applied to the bioculturally reproducing human species.

In their estimation of human LRE, Burger et al. treat humans as if there were no pre-weaning alloparental transfers. Burger and colleagues also assume that the age of independence in humans occurs at the age of complete weaning, which they define as 3 years after birth. They then use values for average offspring weight (in grams) at that age for the variable "litter size" in their empirical estimation of LRE. These assumptions are not warranted for humans (Bragg et al. 2012) and other strategies for calculating human LRE that account for more of the features of our unique strategy of biocultural reproduction are needed.

Here is a summary of what seems to be required. Across all placental mammals, three distinct phases of maternal investment in reproduction can be identified: (1) direct metabolic transfers during gestation, (2) a period of exclusive breast-feeding when all of the infant's nutritional needs are met via breast milk; and (3) in many mammals, a period of "mixed" feeding during which infant nutritional requirements are met by a blend of infant self-provisioning and breast milk. The nature of this third, transitional feeding phase is greatly modified in humans compared to all other great apes and noncooperatively breeding primates (Humphrey 2010). Humans,

rather than shifting from milk to a combination of milk and self-provisioning, shift from milk to "complementary feeding" wherein still-dependent and nursing offspring are fed nutritionally-rich, specially prepared foods (Bogin1999a, b; Lancaster & Lancaster 1983; Sellen 2006). Complementary feeding of human infants generally begins ~6 months of age and often earlier, which means that, for the majority of the period of lactation, infants receive both their mother's milk and foods supplied from outside the mother's body. These complementary foods *must not* be included in the calculation of LRE which, according to Charnov et al. (2007), is reserved for the energy that mothers provide as direct metabolic transfers to offspring.

The highest energetic output that an average human woman achieves during lactation is ~2.7 MJ/day (Prentice et al. 1996; Sellen 2007), which is already outstripped by infant energy requirements at 6 months of age (Butte et al. 2000b).

Because energy needs continue to increase with age, the proportion of infant needs met by breast-feeding will tend to progressively decline following peak lactation (Lee et al. 1991; Sellen 2007). All of this theoretical discussion is consistent with empirical observations, from ethnographic research of human societies, of the role of both mothers and alloparents in providing a variety of foods to still-breastfeeding infants, which can even include breast milk from wet nurses (e.g. Hrdy 1999 for historical sources and Tronick et al. 1989 contemporary forager society examples). Taking such factors into consideration, Sellen (2006, 2007) has estimated that supplementary feeding could account for 1.8 MJ/day of infant needs during the first year, which is the equivalent of about two-thirds (67%) of the energetic cost of peak lactation.

Using the same dataset as Burger et al. (2010), Jared Bragg, Chris Kuzawa, and I reestimated human LRE by modifying the equation for LRE to reflect these unique, species-defining characteristics of the human life history (Bragg et al. 2012). We recalculated LRE by "adjusting" offspring size at weaning to reflect the proportion of offspring weight gained from conception through size at weaning "paid for" by maternal metabolism, as well as the portion paid for by complementary feeding (Figure 4.16). We followed Burger et al. (2010) and assumed age 36 months to be the age at weaning. We used the same data set of 15 small-scale forager and traditional subsistence-based societies. We recalculated human LRE based on both "high" and "low" estimates of the energy savings afforded by complementary feeding, which were derived from the empirical data for infant energetic requirements and maternal breast milk production. With these modifications, we estimate that the LRE of human women is on the order of 1.02–1.23, assuming high (67%) to low (33%) proportions of the energetic needs of weanlings older than 6 months being met by complementary foods in lieu of breast milk.

Our estimates of human LRE are 14–29% lower than that calculated by Burger and colleagues. This difference represents a significant saving of energy expended on reproduction in both statistical terms and also in biological function. Statistically, the difference between Burger et al.'s mean LRE of 1.44 (SD = 0.22) and our conservative mean estimated LRE of 1.23 (SD = 0.20) results in a $t = 2.65$ with $p < 0.01$. Biologically, our new human LRE values, ranging from 1.02 to 1.23, result in

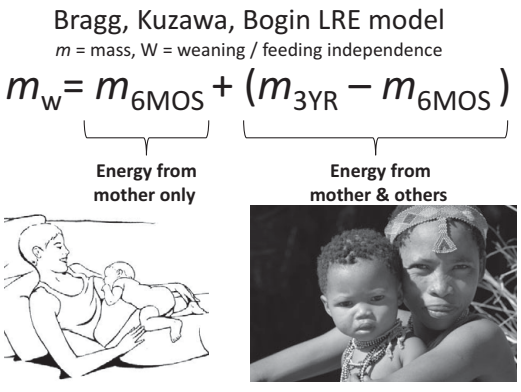


Figure 4.16 Human Lifetime Reproductive Effort model. Most infant weight gain from conception up until 6 months after birth is due to direct energy transfer from maternal metabolism. This full amount (m_{6MOS}) can be included LRE. However, the weight gained from 6 months, when complementary foods are introduced, to 3 years (m_{3YR} - m_{6MOS}) is the product of both maternal and nonmaternal energetics, and as such needs to be adjusted to essentially "factor out" the nonmaternal contribution for infant body mass at weaning (m_w). Image credits: mother nursing from www.llli.org/faq/positioning.html, mother and child Kerstin Geier / Getty Images.

substantial energy savings that may be invested in other functions or needs. These include defense against infection, maintenance of the mother's body, food production, and social interactions.

The evolution of the childhood life history stage in the human lineage made possible new opportunities for reproductive success by allowing infants to be weaned earlier and shortening inter-birth intervals. This strategy would only be favored by natural selection if the increased rate of offspring production was also followed by the survival of the offspring to adulthood. Perhaps in service of this need, the evolution of human childhood was accompanied by a shift from the typical mammalian pattern of a unique attachment dependence between the infant and the biological mother to the human pattern, in which attachment is expanded outward to other members of the social group in a locally- and culturally-defined fashion via rules of kinship and marriage. It is this decoupling of allocare from genetic relatedness, more than anything, which sets humans apart from cooperative breeding species.

In summary, biocultural reproduction describes a suite of biological and sociocultural adaptations, including: (1) cognitive capacities for nongenetically-based marriage and kinship behavior that provide demographically and ecologically flexible, but culturally universal, alloparental care for offspring; (2) early weaning, leading to an increased rate of reproduction; (3) a life history phase of childhood, characterized by the absence of nursing but considerable ongoing nutritional dependence, creating extended opportunities and needs for the provision of care by individuals other than the parents; and (4) decreased LRE, which likely contributed to decreased mortality for mothers and their infant/child.

The excess maternal energy freed up through this strategy may have played an important role in the demographic success of humans over that of other ape species. Lower human LRE may have contributed the metabolic "fuel" necessary to slow the pace of ageing and extend lifespan, thereby expanding the pool of late life allocaregivers and possibilities for inter-generational transfers, which are critical to human biocultural reproduction. This possibility is explored in more detail later in this chapter.

Why Adolescence?

Q: What happens at adolescence to a boy?
A: He says goodbye to his childhood and enters adultery.

(From an undergraduate exam prepared by Bogin and incorrectly answered, mostly, by a student.)

Human adolescence is the stage of life that begins with the relative immaturity of the juvenile and ends with the fundamentals of social, economic, and sexual maturation. All three are needed for successful mating and reproduction. The case was presented in the previous chapter and earlier in the present chapter for the special growth and development characteristics of human adolescence and when human adolescence evolved. Discussion here focuses on why adolescence evolved.

The single most important feature defining human adolescence is the skeletal growth spurt that is experienced by virtually all boys and most girls – up to 10% of healthy, well-fed girls do not have a marked adolescent spurt in height.

One often cited reason for the adolescent growth spurt is the prolonged time required to learn technology, social organization, language, and other aspects of culture during the infant, child, and juvenile stages of growth. At the end of this period, so the argument goes, our ancestors were left with proportionately less time for procreation than most mammals, and therefore needed to attain adult size and sexual maturity quickly. But surely this cannot be the whole story. Consider first that there is no need to experience an adolescent growth spurt to reach adult height, achieve fertility, and reproduce successfully. There are healthy, normal individuals, for the most part very late maturing boys and girls, who have virtually no growth spurt. Nevertheless, these late maturing individuals do grow to be normal-sized adults, and they become fertile by their early twenties – not significantly later than individuals with a spurt. Other boys and girls who are born without gonads or have them removed surgically prior to puberty (due to diseases such as cancer) do not experience an adolescent growth spurt, but do reach their normal expected adult height, but not fertility (Prader 1984). Historical sources describe the *castrati*, male opera singers of the seventeenth and eighteenth centuries who were castrated as boys to preserve their soprano voices. They lacked a growth spurt but were noted as being unusually tall for men (Peschel & Peschel 1987). The same hormones that cause the growth spurt also end it, so the *castrati*

without these hormones grew more. Of course, castrati, whether or not opera singers, do not become reproductively successful.

Another problem with the "lost time" argument for the adolescent growth spurt is that it does not explain the timing of the spurt. Girls experience the growth spurt before becoming fertile, but for boys the reverse is true. Why the difference? The reason relates to reproductive success. Human childhood evolved as a benefit for the mother and not the child, that is, so that the mother could resume reproduction more quickly by weaning early. Similarly, adolescence is likely to have evolved as a reproductive adaptation for adults, and not directly for the adolescent. The reason for this is that natural selection works on differential fertility and differential mortality between individuals. An additional 5 –10 years of infertility, or reduced fertility, associated with adolescence could not evolve for all humans, since those individuals who "cheated" by terminating growth at an earlier age would begin reproducing sooner and would be at a reproductive advantage. All other primates do, in fact, begin reproducing at earlier ages than humans, and none of the nonhuman primates has a humanlike adolescent growth spurt, nor many of the other biological and behavioral features of human adolescence. Clearly, a juvenile primate does not need to pass through a lengthy period of adolescence, with apprenticeship-type learning, just to be reproductively successful. What factors, then, could give rise to adolescence and further delays in reproduction?

The answer may lie in a type of multilevel selection model of mating and parenting. Multilevel models in evolutionary biology include selection at the level of the individual and at the level of the social group (see Bogin 2009 for more background on multilevel models). Such models allow for time lags between the stage of life when selection takes place and the accrual of reproductive benefits later in life. The complex pattern of human individual growth and development, combined with equally complex human social and cultural behavior, seems to be better explained by multilevel evolutionary models rather than simpler models, for example, those focusing only on fertility or mortality of the adolescent.

The multilevel model presented here builds on the nature of biocultural reproduction and human mating behavior. Reproduction and mating are of course related, but they are not identical, as not all mating opportunities result in fertilization and offspring. But, successful mating and reproduction depend on the two types of biological selection identified by Charles Darwin: natural selection, and **sexual selection**. Both are likely to be involved in the evolution of human adolescence. Sexual selection is all about opportunities for mating, while natural selection is, in part, about making and parenting offspring. Darwin (1871, p. 256) defined sexual selection as " . . . the advantage which certain individuals have over other individuals of the same sex and species, in exclusive relation to reproduction." Today we would replace the word reproduction with mating. Darwin also wrote of the many structures and instincts developed through sexual selection, including, " . . . weapons of offence and the means of defense possessed by the males for fighting with and driving away their rivals – their courage and pugnacity – their ornaments of many kinds – their organs for producing vocal or instrumental music – and their glands for emitting

odors; most of these latter structures serving only to allure or excite the female." It is known today that sexual selection also works for females, meaning that female-specific physical and behavioral traits may evolve via competition between the females for mating opportunities with males. Some human examples are the waist-to-hip ratio and childlike voice pitch of women that may be alluring to men.

Adolescent Contributions to the Reproductive Success of Adults

One level of the model for adolescence relates to the work that teenagers do for their social group. Human children and juveniles may hunt, gather, or produce some of their own food intake, but overall, they require provisioning to achieve energy balance. In contrast, human adolescents can produce sufficient quantities of food to exceed their own energy requirements (Bogin 2001; Kaplan et al. 2000; Kramer 2002, 2014). Some of the food that adolescents produce may be used to fuel their own growth and development, creating larger, stronger, and healthier bodies. The surplus production is shared with other members of the social group, including younger siblings, parents, and other immediate family members (defining families in the broad anthropological sense). Adolescent contributions enhance the fertility of adults and the survival of infants, children, and juveniles. The biological trade-off is the delay of years between puberty and first birth for the adolescents. For their valuable services in food production, the adolescents receive care and protection to safeguard their health and survival. This is important because while adolescents may be adult-like in some aspects of physique they are immature in terms of sociocultural knowledge and experience, placing them at risk for damage and death.

Girls and Boys: Separate Paths through Adolescence

Another level of the model for the evolution of human adolescence may be seen by considering the trade-offs related to the different sequence of biological and behavioral events experienced by adolescent girls and boys. The differences allow each sex to improve opportunities for mating and parenting. Mating will eventually lead to the birth of offspring, but producing offspring is only a small part of reproductive fitness. Rearing the young to their own reproductive maturity is a surer indicator of success. The developmental paths of girls and boys during adolescence may be key in helping each sex to both produce and rear its own young successfully.

The order in which several pubertal and adolescent events occur in girls and boys is illustrated in Figure 4.17 in terms of time before and after peak height velocity (PHV) of the adolescent growth spurt. The Tanner Maturation Staging System for the development of secondary sexual characteristics is used in this figure. This system is based on 5 stages, with stage 1 being pre-pubertal. In both girls and boys, puberty begins with changes in the activity of the hypothalamus and other parts of the central nervous system. These changes are labeled as "CNS puberty" in the figure. Note that the CNS events begin at the same relative age in both girls and boys, that is, three years before PHV. This is also the time when growth rates change from

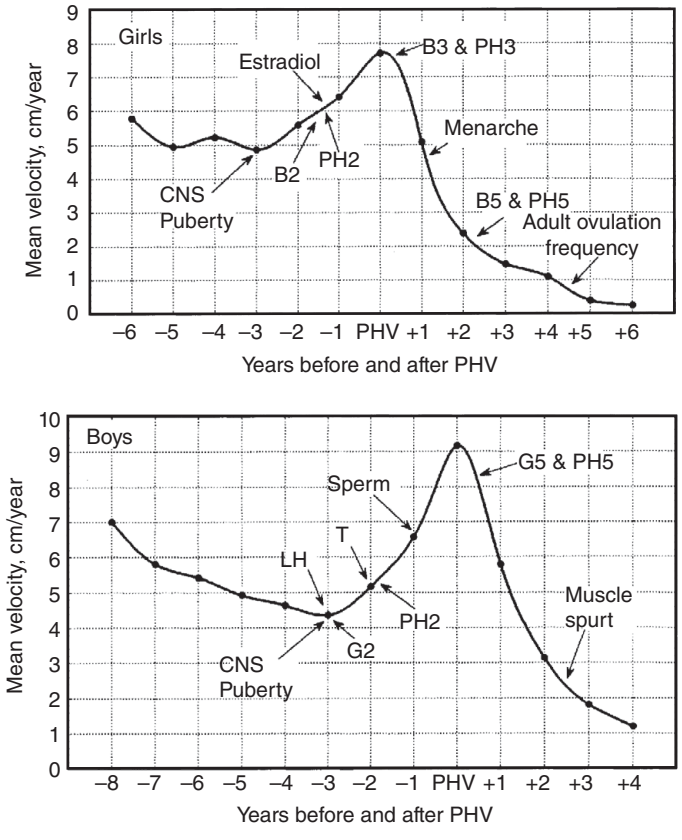


Figure 4.17 The ordering of several sexual maturation events for girls (top panel) and boys (bottom panel) during the adolescent growth spurt. The velocity curves are calculated using data derived from a sample of healthy, well-nourished girls and boys living in Guatemala. See text for an explanation of each labeled event.

decelerating to accelerating. In girls, the first outward sign of puberty is the development of the breast bud (B2) and wisps of pubic hair (PH2) (see Chapter 2 for an explanation of the system of staging the breast and pubic hair development of girls, and genital and pubic hair development in boys). This is followed, in order, by: (1) a rise in serum levels of estradiol which leads to the laying down of fat on the hips, buttocks, and thighs; (2) the adolescent growth spurt; (3) further growth of the breast and body hair (B3 & PH3); (4) menarche; (5) completion of breast and body hair development (B5 & PH5); and (6) attainment of adult levels of ovulation frequency.

The path of pubertal development in boys starts with a rise in serum levels of luteinizing hormone (LH) and the enlargement of the testes and then penis (G2). This genital maturation begins, on average, only a few months after that of girls. However, the timing and order of other secondary sexual characteristics is unlike that of girls. About a year after CNS puberty, there is: (1) a rise in serum testosterone levels (T) which is followed by the appearance of pubic hair (PH2); (2) about a year later

motile spermatozoa may be detected in urine; (3) PHV follows after about another year, along with deepening of the voice, and continued growth of facial and body hair; (4) the adult stages of genital and pubic hair development follow the growth spurt (G5 & PH5); and (6) near the end of adolescence boys undergo a spurt in muscular development.

The sex-specific order of pubertal events tends not to vary between early and late maturers, between well-nourished girls and boys and those who suffered from severe malnutrition in early life, between rural and urban dwellers, or between European and African ethnic groups (Bogin et al. 1992; Cameron et al. 1988, 1990). In addition to these biological events there are behavioral and social events that also follow a predictable course during adolescence. Indeed, the biological and cultural events are usually tightly correlated. A comparison of the biocultural timing of adolescent events in two societies is given in Figure 4.18. Girls from a London, England sample and a Kikuyu (African) sample are compared in the upper panel and boys from the same two samples are compared in the lower panel (the data are reported by Worthman 1993).

The Kikuyu are a Bantu-speaking, agricultural society of the central highlands of Kenya. The London sample represents adolescents who are relatively well nourished and healthy. The Kikuyu sample represents adolescents who suffer from periodic food shortages and, likely, a higher incidence of infectious and parasitic diseases than adolescents in London. The adolescent events for each sex are placed in chronological order and the bars indicate the median age at which each event occurs. These comparisons show two things. The first is how differences in health and nutrition between human societies may influence the timing, but not the order, of adolescent events. The second is how societies as diverse as urban Londoners and rural Kikuyu adjust the timing of some social events to the timing of the biological events of human adolescence. The links between the biological development and maturation events of adolescence and the sociocultural events are often so strong that it seems best to consider both from an integrated biocultural perspective.

For both London and Kikuyu girls, the first biocultural event is breast development (bars 1-B), and the second event is pubic hair development (bars 2-PH). The numbers on these bars indicate median age of entry to each stage of development (B2, B3, B5, etc.). The third event is a rise in serum estradiol concentration (bars 3-E). This hormonal event, and similar hormonal changes in boys, can only be detected by special tests, not by the adolescents or their parents. However, the rise of estradiol leads to biological and behavioral changes that are easily detectable in the form of fat deposits on hips, thighs, and buttocks and new levels of cognition (Piaget's formal operations stage). The fourth event is peak height velocity (bars 4-PHV). Note that for the Kikuyu, PHV occurs about two years later than for the London girls. For many Kikuyu girls the fifth biocultural event is clitoridectomy; at the time of Worthman's research in 1979 and 1980 about 40% of girls underwent this operation, which removes the tip of the clitoris. Clitoridectomy takes place just after PHV, at about breast stage 3, and just before menarche. Worthman reported that the operation was timed so that it precedes the onset of sexual activity and marriage which follow

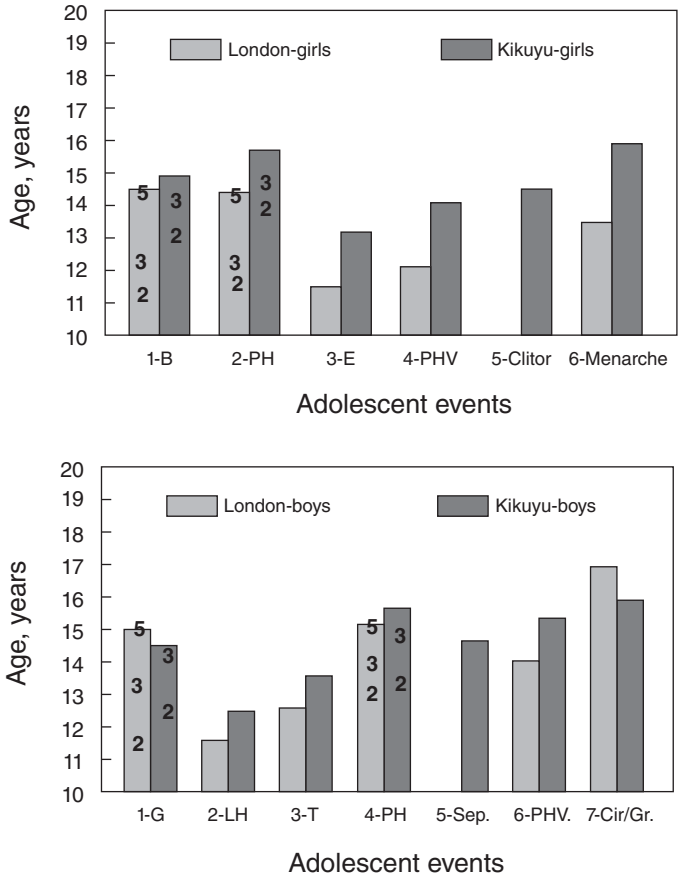


Figure 4.18 Comparison of biocultural events during adolescence in a sample of London and Kikuyu girls and boys. The data are abstracted from Worthman (1993), who provides references to the original studies. The events are presented in order of occurrence. The bars indicate the median age at onset of each event. Explanation of each event is given in the text. The abbreviations for the girls' events are: 1-B, breast stages; 2-PH, pubic hair stages; 3-E, first notable rise in serum estradiol concentration; 4-PHV, peak height velocity; 5-Clitor., clitoridectomy; 6-Menarche, age at menarche. Abbreviations for the boys are: 1-G, genital stages; 2-LH, first notable rise in serum luteinizing hormone concentration; 3-T, first notable rise in serum testosterone concentration; 4-PH, pubic hair stages; 5-Sep., separation to "boys' house" for Kikuyu; 6-PHV, peak height velocity; 7-Cir/Gr., circumcision for Kikuyu, or graduation from secondary school for London boys.

menarche. London girls may experience some adolescent rites of passage after PHV, such as "coming-out" parties or "sweet sixteen" events, but these are usually less well-defined and less traumatic than clitoridectomy. The sixth event is menarche, which is taken as a sign of impending sexual maturation in all cultures. In many cultures, menarche often precipitates intensified instruction about sexual behaviors and the practice of these behaviors (Schlegel & Barry 1991).

For both London and Kikuyu boys, the first two biocultural events are enlargement of the testes, or genital stage 2 (bars 1-G), and a rise in serum concentration of luteinizing hormone (bars 2-LH). In fact the order of the two events could be switched as it is the rise of LH that leads to testes enlargement. Here these two events are considered to be coterminous. The third event is a rise in the serum concentration of testosterone (bars 3-T), which precipitates a cascade of physical and behavioral changes. The fourth event is public hair development (bars 4-PH). For the Kikuyu, the fifth biocultural event is separation (bar 5-Sep.), which means that the adolescent boys leave their nuclear household and begin living in an age-graded adolescent male household. Worthman reported that separation to the "boys' house" is closely correlated with age at first emission, and that separation takes place at about the same age that girls undergo clitoridectomy. These events show that Kikuyu parents are able to recognize and respond to the sexual maturation of their adolescents. The sixth event is peak height velocity (bars 6-PHV). The seventh biocultural event for Kikuyu adolescents is circumcision (7-Cir), which is done to all young men and marks their entry into training for adulthood. Circumcision is timed to occur along with the spurt in muscle mass, which allows boys to perform physical labor at adult levels. London boys do not undergo a circumcision rite of passage, but within that same year they usually graduate from secondary school (7-Gr.). Graduation, which London girls also experience, is a rite of passage in most industrialized societies and often marks entry into the social world of adults.

More will be said about the biocultural significance of these adolescent events in the next few pages. At this point it is important to focus on two general issues. The first is that the adolescent growth spurt is a biologically and socially significant event for both sexes. The second is that the order of adolescent events is different for each sex, for example the growth spurt occurs earlier in the sequence, as well as at an earlier age, in girls than in boys. Given this, the sexual dimorphism expressed in the sequence and timing of these events may be considered human species-specific characteristics. Evolutionary biologists usually find that species-specific traits evolve to enhance survival and reproductive success. Thus, the human adolescent growth spurt must have its own intrinsic evolutionary value and is not just a by-product of slow prepubertal development.

Why Do Girls Have Adolescence, or Why Wait So Long to Have a Baby?

In human societies, adolescent girls gain knowledge of sexuality and reproduction because they look mature sexually, and are treated as such, several years before they actually become fertile. The adolescent growth spurt serves as a signal of maturation. Early in the spurt girls develop pubic hair and fat deposits on breasts, buttocks, and thighs. They appear to be maturing sexually. About a year after peak height velocity, girls experience menarche, an unambiguous external signal of internal reproductive system development. However, most girls experience 1–3 years of anovulatory menstrual cycles after menarche. Two studies of girls and young women living in Switzerland and Finland examined the frequency of ovulation for 4.5 years following

menarche (reviewed in Worthman, 1993). Ovulation frequency varied from 0 to 10% of menstrual cycles at 6 months post-menarche. The frequency increased to about 30% after 1.5 years, varied between 40 and 55% after 2.5 years, and leveled off at 60 to 65% after 4.5 years. Since the mature level of ovulatory frequency is about 65% of menstrual cycles, it appears that it takes about 5 years for healthy, well-nourished girls to achieve adult maturity for fertility.

Adolescent girls, and the adults around them, may or may not be aware of this period of subfecundity. Everyone in the social group is aware of the dramatic changes taking place in the adolescent girl, and these changes certainly stimulate both the girls, and adults around them, to participate in adult social, sexual, and economic behavior. For the post-menarche adolescent girl this participation provides the learning and experience she will need to be a successful woman and mother, and it is "low risk" in terms of pregnancy for several years.

It is noteworthy that female chimpanzees and bonobos, like human girls, also experience up to three years of post-menarche infertility, so this time of life may be a shared hominoid trait. As with human adolescents, the post-menarchial but infertile chimpanzees and bonobos participate in a great deal of adult social and sexual behavior. Primate researchers observing these apes point out that this participation, without pregnancy, allows for practicing many key behaviors that are needed to rear an infant successfully (Goodall 1983; Nishida 2011). Although ape and human females may share a year or more of post-pubertal subfecundity, apes reach sexually mature adulthood at about 12 years of age, much sooner than humans. This limits the learning and practice period for the apes.

Some girls, of course, may become pregnant and there are other social and psychological risks of adolescent sexual behavior. Teenage mothers and their infants are at risk because of the reproductive and emotional immaturity of the mother. This often leads to a low-birth-weight infant, premature birth, and high blood pressure in the mother. The likelihood of these risks declines, and the chance of successful pregnancy and birth increases markedly after age 15 years, and reaches its nadir after age 18 years. Due to these biological and social risks, most human societies carefully regulate, according to age and sex, the onset and type of sexual behavior that is permitted by adolescents.

Another evolutionary reason for the delay between menarche and adulthood in girls is that human female fertility tracks the growth of the pelvis. Marquisa LaVelle Moerman reported in 1982 that the crucial variable for successful first birth is size of the pelvic inlet, the bony opening of the birth canal. Moerman (1982) measured pelvic X-rays from a sample of healthy, well-nourished American girls who achieved menarche between 12 and 13 years. These girls did not attain adult pelvic inlet size until 17–18 years of age. Quite unexpectedly, the adolescent growth spurt, which occurs before menarche, does not influence the size of the pelvis in the same way as the rest of the skeleton. Rather, the female pelvis has its own slow pattern of growth, which continues for several years after adult stature is achieved.

Why the pelvis follows this unusual pattern of growth is not clearly understood. Perhaps another human attribute, bipedal walking, is a factor. The evolution of

bipedalism is known to have changed the shape of the human pelvis from the basic apelike shape. Apes have a cylindrical-shaped pelvis, but humans have a bowl-shaped pelvis. The human shape is more efficient for bipedal locomotion but less efficient for reproduction because it restricts the size of the birth canal. It may take human women longer than an ape to grow a large enough pelvis to achieve full reproductive maturity. Cross-cultural studies of reproductive behavior show that human societies acknowledge (consciously or not) this special pattern of pelvic growth. The age at marriage, and first childbirth, clusters around 19 years for women from such diverse cultures as the Kikuyu of Kenya, Mayans of Guatemala, Copper Eskimos of Canada, and the United States from the colonial period to the 1950s. That time of waiting from menarche to motherhood provides adolescent girls with many opportunities to practice and learn important adult behaviors that lead to increased reproductive fitness in later life. All the while these girls are contributing food, labor, and alloparental care to infants, children, pregnant women, and elderly/disabled group members.

Why Do Boys Have Adolescence?

The adolescent development of boys is quite different from that of girls. Boys become fertile well before they assume the size and the physical characteristics of men. Analysis of urine samples from boys 11–16 years old show that they begin producing sperm at a median age of 13.4 years. Yet cross-cultural evidence indicates that few boys successfully father children until they are into their third decade of life. In the United States, for example, only 3.09% of live-born infants in 1990 were fathered by men under 20 years of age. In Portugal, for years 1990, 1994, and 1999, the percentage of fathers under 20 years of age was always below 3%. In 2001, Portugal stopped presenting results concerning the percentage of fathers below 20 because there were too few of them. Among the traditional Kikuyu of East Africa, men do not marry and become fathers until about age 25 years, although they become sexually active after their circumcision rite at around age 18.

The explanation for the lag between sperm production and fatherhood is not likely to be a simple one of sperm performance, such as not having the endurance to swim to an egg cell in the woman's fallopian tubes. More likely is the fact that the average boy of 13.4 years is only beginning his adolescent growth spurt. Growth researchers have documented that in terms of physical appearance, physiological status, psychosocial development, and economic productivity, the 13-year-old boy is still more a juvenile than an adult. Anthropologists working in many diverse cultural settings report that few women (and more important from a cross-cultural perspective, few prospective in-laws) view the teenage boy as a biologically, economically, and socially viable husband and father.

The delay between sperm production and reproductive maturity is not wasted time in either a biological or social sense. The obvious and the subtle psychophysiological effects of testosterone and other androgen hormones that are released after gonadal maturation may "prime" boys to be receptive to their future roles as men.

Alternatively, it is possible that physical changes provoked by the endocrines provide a social stimulus toward adult behaviors. Whatever the case, early in adolescence, sociosexual feelings including guilt, anxiety, pleasure, and pride intensify. At the same time, adolescent boys become more interested in adult activities, adjust their attitude to parental figures, and think and act more independently. In short, they begin to behave like men. However – and this is where the survival advantage may lie – they still look like boys. One might say that a healthy, well-nourished 13.5-year-old human male, at a median height of 160 cm (62 in.) "pretends" to be more childlike than he really is. Because their adolescent growth spurt occurs late in sexual development, young males can practice behaving like adults before they are actually the size of an adult and perceived as mature by other adults.

Even more to the point is that the spurt in muscle mass of adolescent males does not occur until an average age of 17 years (Bielicki et al. 1984). At peak height velocity the typical boy has achieved 91% of his adult height, but only 72% of his adult lean body mass. Since most of the lean body mass is voluntary muscle tissue, adolescent boys cannot do the work of men. This is one important reason why the Kikuyu, the Inuit, and many other cultures do not even think of younger adolescents as manlike. Schlegel and Barry (1991) found in their cross-cultural survey that young adolescent boys are usually encouraged to associate and "play" with their age mates rather than associate with adult men. During these episodes of "play" these juvenile-looking adolescent males can practice behaving like adult men before they are perceived as adults. The activities that take place in these adolescent male peer groups include the type of productive, economic, aggressive/militaristic, and sexual behaviors that older men perform. However, the sociosexual antics of adolescent boys are often considered to be more humorous than serious. Yet, they provide the experience to fine tune their sexual and social roles before either their lives, or those of their offspring, depend on them. For example, competition between men for women favors the older, more experienced man. As such competition may be fatal, the juvenile-like appearance of the immature, but hormonally primed, adolescent male may be life-saving, as well as educational.

Summary of Adolescence

Adolescence became part of human life history because it conferred significant reproductive advantages to our species, in part, by allowing the adolescent to learn and practice adult economic, social, and sexual behaviors before reproducing. In equal measure, adolescents contribute to the pooled energy of their social group and enhance the survival of individuals younger and older than themselves. The basic argument for the evolution and value of human adolescence is this: Girls best learn their adult social roles while they are infertile but perceived by adults as mature; whereas, boys best learn their adult social roles while they are sexually mature but not yet perceived as such by adults. Without the adolescent growth spurt, and the sex-specific timing of maturation events around the spurt, this unique style of social and cultural learning could not occur. Over the course of time and space, the styles of

learning these behaviors have come to vary considerably cross-culturally. The evolution of human adolescence, therefore, must be modeled in terms of both its biological and cultural ramifications.

Viewing human adolescence in this multilevel, life history, and biocultural perspective has significant implications. Some of these may pertain to medical treatment of growth disorders, for example, the effects of early or delayed puberty on the physical and psychological well-being of the adolescent. Other implications relate more to economic, social, and legal policies that impact the lives of adolescents. These issues are investigated in more detail in Chapter 6, with reference to environmental influences on human growth and development.

The "Valuable Grandmother," or Could Menopause Evolve?

In addition to childhood and adolescence there is another unusual aspect of human life history: menopause. One generally accepted definition of menopause is, " . . . the sudden or gradual cessation of the menstrual cycle subsequent to the loss of ovarian function . . . " (Timiras 1972, p. 531). The process of menopause is closely associated with the post-reproductive stage of life of adult women, which is also unusual because unlike most mammals that are basically decrepit at the end of their reproductive life, human women are likely to be otherwise healthy, vigorous, and productive socially and economically for many more years.

Menopause is distinct from the loss of fecundity as reproduction usually ends some years before menopause. In traditional societies, such as the !Kung (Howell 1979), the Dogon of Mali (Strassmann & Warner 1998), rural Bangladeshis (Towner et al. 2016), and the rural-living Maya of Guatemala (INCAP 1989), women rarely give birth after age 40 years and almost never give birth after age 44 (Figure 4.19). Infectious disease, parasites, food insecurity, and heavy physical labor may take their toll in these societies and shorten the years of fertility. Menopause, however, occurs after age 45 in these three societies. In the industrialized nations, with better health for girls and women, the median age at menopause is 49–52 years (Murphy et al. 2013).

In the United States, between the years 1960 and 1990, data for all births show that women 45 to 49 years gave birth to less than one out of every 1,000 live born infants. In contrast, there were 16.1/1,000 live births to women aged 40–44 years. Similar statistics exist for Canada and the United Kingdom for the same time period (Bogin 2001). The Old Order Amish are a high fertility, noncontracepting population residing primarily in the states of Pennsylvania, Ohio, and Indiana. Amish women aged 45–49 years, born before 1918, gave birth to an average of 13 infants per 1,000 married women, while those women between 40 and 44 years of age gave birth to an average of 118 infants per 1,000 married women (Ericksen et al. 1979). Thus, even in the United States of 1960–1990, with modern health care, good nutrition, and low levels of hard physical labor, and even among social groups attempting to maximize lifetime fertility, women rarely give birth after age 45 years. As for the !Kung, Dogon, Bangladeshis, and Maya, menopause for Amish women occurs one or more years after this fertility decline. After age 50, births are so rare that they are not reported in

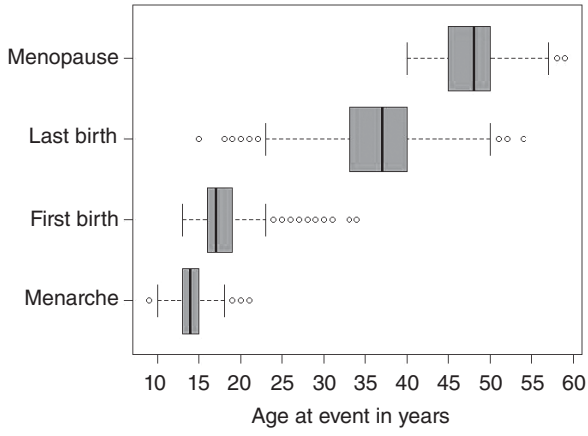


Figure 4.19 Critical life-history events for Bangladeshi women (n = 1037 aged 50–69 years old from rural agricultural households in Matlab, Bangladesh). Boxplots give the median event age (line within rectangle), first and third quartile age values rectangle boundaries), and minimum and maximum age values (brackets), excluding outliers. Summary statistics: age at menarche (mean 14.0, median 14, range 9–21, SD 1.4), first birth (mean 17.9, median 17, range 13–34, SD 3.0), last birth (mean 36.2, median 37, range 15–54, SD 5.5) and menopause (mean 47.4, median 48, range 40–59, SD 3.5). From Towner et al. (2016) with permission

the data of the United States National Center for Health Statistics or for the Amish (but are sensationalized in the tabloids sold at supermarket checkouts).

These ages for the onset of human female post-reproductive life vs. the ages for menopause are reported here for two reasons. The first is that some scholars incorrectly equate menopause with the beginning of the post-reproductive stage. Furthermore, some scholars define menopause as the process of decline in fecundity while other scholars define it as the moment when fecundity ends. So, one must read the literature carefully to interpret in what sense the term "menopause" is used. The second reason is that menopause, and a significant period of life after menopause, are claimed by some scholars to be uniquely human characteristics. Other scholars assert that menopause is a shared trait with other mammals.

The consensus of evidence reports that menopause may be the only event of the later adult years that is experienced universally by human women who live past 50 years of age; men have no similar event. The only nonhuman species with verified menopause in the wild are four species of toothed whales – beluga (*Delphinapterus leucas*), narwhal (*Monodon monoceros*), killer (*Orcinus orca*), and short-finned pilot whales (*Globicephala macrorhynchus*). A possible menopause may exist for the false killer whale (*Pseudorca crassidens*). A common life history trait of all these whale species is that females usually live past 50 years of age (Brent et al. 2015; Croft et al. 2017; Ellis et al. 2018). The length of the post-reproductive lifespan for these female whales averages about 20 years, with a range from about 8 years in false killer whales to 40 years in belugas. African and Asian elephants, the largest land-living mammal, do not have menopause and have median life expectancies of 36 years and 43 years,

respectively (Clubb et al. 2009). No ape species has menopause nor a median life expectancy over 50 years (see further discussion below).

The extraordinary duration of the whale and human female post-reproductive life stage correlates with ethological and cross-cultural ethnographic research showing the crucial importance of the post-reproductive females as repositories of ecological and/or cultural information that leads to inclusive fitness and kin selection. For the whale species, the kin selection seems to have a direct genetic basis. In these species both males and females tend to remain in the natal group. Adult males mate outside their group, but they feed and compete for social dominance within the group, which leads to mating success. Post-menopausal mothers help their sons by leading them to feeding grounds and providing social support for their dominance. Through their sons, therefore, the infertile older mothers promote their own genetic contributions to following generations.

In addition to the few species of whales and humans, with true menopause, there are some exceptional species with grandmother caretaking, for example, hyenas (*Crocuta crocuta*, *Hyaena brunnea*, *Hyaena hyaena*). Grandmother hyenas are observed to nurse the cubs of their daughters. Indeed, when both are still fertile, mother and daughter hyenas take turns nursing each other's young (Mills 1990). Allomaternal nursing is known from at least one other social carnivore species, lions (*Panthera leo*), bottlenose dolphins (*Tursiops truncates*), several primate species, also dwarf mongooses (*Helogale parvula*), gray mouse lemurs (*Microcebus murinus*), galagos (*Galago senegalensis braccatus*), and humans (Hewlett & Winn 2014). The golden snub-nosed monkey (*Rhinopithecus roxellana*), a species of the African leaf-eating colobine monkeys, and the South American white-faced capuchin (*Cebus capucinus*) are also reported to practice allomaternal nursing (Sargeant et al. 2015; Xiang et al. 2019). Some of the nonhuman primate species practice allomaternal nursing by biological sisters and, less frequently, by lesser related females, as well as by grandmothers. Most of the human cases of allomaternal nursing are by maternal grandmothers. Barry Hewlett and Steve Winn (2014) reviewed the literature on allomaternal nursing and found 13 human forager societies with ethnographic data. In six of these societies, from Africa, Philippines, and Andaman Islands (Aka, Efé, Bofi, Chabu, Agta, Ongée), allomaternal nursing was normative; in two societies from South America (Aché, Pumé) it was reported to occur; and in five societies from Australia, Africa, and India (Martu, Nyaka, !Kung, Hadza, Paliyan) it was absent. Where it is normative, biological kin, especially grandmothers, most frequently provide nursing. Hewlett and Winn reported that several factors influence the nature and frequency of allomaternal nursing: infant age, mother's condition, and the society's ideological models about colostrum taboos, about the meaning of women other than the mother nursing an infant, and about when allomothers may nurse. But, allomaternal nursing is not a common human behavior. Other types of care by post-menopausal women to their children, to daughters- and sons-in-laws, and their grandchildren are much more common.

The biology and possible value of menopause are topics of much interest, empirical research, and speculation. The most parsimonious explanation for menopause is that by 50 years of age, women, and the four species of whales, have out-lived their

supply of primary oocytes, which was established during their pre-natal development. This "oocyte depletion" hypothesis is described in more detail in Box 4.3. If this is the cause, then there is no need for an evolutionary explanation for menopause. Rather, the need is for an evolutionary explanation for the human and whale capacity to live healthy, vigorous lives to age 50 years and, in the human case, for decades past menopause. Various ideas to explain latter life vigor have been proposed and a few are described below.

Box 4.3 **Oocytes, pleiotropy, and menopause**

Another model for menopause may be termed the "pleiotropy hypothesis." In now classic works on the biology of senescence Peter Medawar (1952) and George Williams (1957) proposed that aging is " . . . due to an accumulation of harmful age-specific genes . . . [or] . . . pleiotropic genes which have good effects early in life, but have bad effects later . . . " (Kirkwood & Holliday 1986, p. 371). Kirkwood (1977), Charlesworth (1980) and others refined this hypothesis further in terms of a general theory of aging. Mary Pavelka and Linda Fedigan (Pavelka & Fedigan 1991) applied this line of reasoning to menopause. According to their version of the "pleiotropy hypothesis," menopause is a secondary consequence of the female mammalian reproduction system. This system has a physiological "life" of about 50 years because of limitations on egg supply or on the maintenance of healthy eggs. Female mammals produce their egg supply during prenatal development but suspend the meiotic division of these oogonia, also called primary oocytes, in anaphase. Up to seven million oogonia are formed by the fifth month of fetal development (Sievert 2006). Various causes of cell death reduce this number to about 2 million at the time of birth and to about 400,000 at the start of puberty (~9 years of age). During human adolescence and adulthood, the remaining primary oocytes continue to die, but some complete their maturation and are released in series during menstrual cycles during the ovulatory phase. By about age 50 years all primary and mature oocytes are depleted. If the woman lives beyond the age of depletion, then she will certainly experience menopause. But, many women around the world experience the gradual or sudden cessation of menstrual cycles years before age 50. As oocyte numbers decline there is a dysregulation of the production of estrogen and progesterone, especially the surges of these hormones that are required to maintain the menstrual cycle and ovulation (Finch 2014; Kirchengast & Rühli 2013; Sievert 2006, 2014). Even before their cessation, cycles become longer. There is also an increased risk of oocyte abnormalities leading to either infertile oocytes or defects of development for the fetus and the newborn causing spontaneous abortion or neonatal and infant deaths. Men do not have menopause, but after age 45 years

or so they do experience a decline in the number and quality of spermatozoa production, leading to a decline in fertility.

Menopause, then, seems to be an inevitable consequence of the age-limited reproductive capacities of all female mammals. The menopausal transition from the prime fertility of adulthood to infertility is not the desired time to become pregnant. The pleiotropy hypothesis, while explaining the reason for menopause, does not account for the fact that human women, and men, may live another 25–50 years and for much of that time have a healthy, vigorous, and productive post-reproductive life stage. For most other mammals aging, senescence, and death happen before the end of the oocyte supply. Any nonhuman primates that survive to age 50 years are decrepit, barely able to take care of themselves. For the four species of whales with menopause and for humans the order of aging events is changed. At some time in their evolutionary history aging and senescence became decoupled from the termination of reproduction.

A hypothesis to account for this decoupling is related to the discussion in the main text on Biocultural Reproduction. The few species able to outlive their supply of oocytes are large-bodied, large-brained, and acquire a good deal of socially-based learned behavior during their lives. In these species (humans and whales), post-menopausal females shift their reproductive investment to assisting their adult offspring to reproduce. As explained in the main text, this type of allocare may be explained by a combination of kin selection (Darwin 1859; Hamilton 1964) and the value of post-reproductive females, and older males, as repositories of ecological knowledge and contributors of food and other material and social resources that buffer kin against environmental hardships (Brent et al. 2015; Hawkes & Coxworth 2013; Zihlman 2012).

The primate context of human women's later-life biology is illustrated in Figure 4.20. Shown are the timing of the onset of the adult female reproductive stage for the chimpanzee and three hominins, and the post-reproductive stage for *Homo sapiens*. It is unlikely that any hominin prior to *H. erectus* lived to age 50 years. From their analyses, Hawkes and colleagues (Hawkes & Coxworth 2013; O'connell et al. 1999) propose that *Homo erectus* was the first hominin species with grandmaternal allocare. It is possible that a small percentage of *H. erectus* and *H. neanderthalensis* (not pictured) women lived past age 50 years, but the evidence suggests that a post-50-year life span became a regular life history trait of only *H. sapiens* (Caspari & Lee 2004). As for Figure 4.11, the data for fossil hominins are speculative, extrapolated, in part, from evidence provided by extant chimpanzees and human beings. Chimpanzees begin their reproductive career at an earlier age than humans and the chimpanzees continue to reproduce until their death, with only the very oldest chimpanzees, over age 50 years, losing fecundity (Emery Thompson et al. 2007; Herndon et al. 2012). Few wild-living female chimpanzees live to reproduce past age

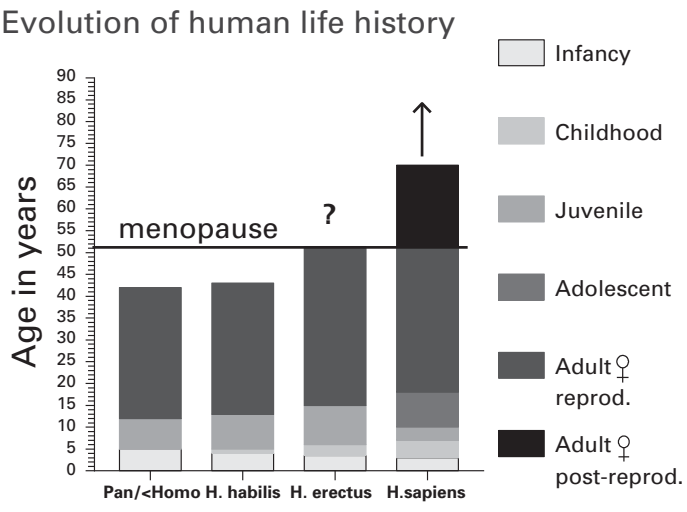


Figure 4.20 The evolution of human female life history emphasizing the post-reproductive stage. Life expectancy estimated by the formula of (Smith, 1991). The arrow above the *H. sapiens* column represents Sacher's (1975) estimate of maximum longevity to 89 years. Increased human longevity extends the post-reproductive stage, not earlier stages of the life cycle. Abbreviations as in Figure 4.11.

30 years. A **meta-analysis** of existing data published by Emery Thompson and colleagues reported that by age 30 years only about 30% of live-born females were alive. By age 40 years less than 20% survived and by age 50 years virtually none survived. Female chimpanzees over 40 years old show signs of rapid senescence and loss of physical function. In contrast, at age 50, about 55% of !Kung women were alive, declining to 45% at age 60 and 18% at age 70 years. For Ache foragers of South America the numbers were about 40% survival to age 50, 25% to age 60, and virtually no surviving women at age 70 years (Emery Thompson et al. 2007). Equally important is that human women over age 50 tend to be physically and mentally vigorous, producing much edible food and providing much needed alloparental care of youngsters. These older women also know a lot of important things, such as where to find food and water during times of scarcity, are excellent story-tellers, and help mediate social disputes.

Much has been written about why human women have considerable longevity beyond menopause. Several versions of "mother," "grandmother," and "grandfather" hypotheses have been proposed (Caro et al. 1995; Hawkes & Coxworth 2013; Voland et al. 2005). One model posits that a post-reproductive life stage could evolve if there are major risks to reproduction for an older female and if the old female can benefit her younger kin. Hamilton (1966) formulated the mathematics of this "grandmother hypothesis" based on his previous work on kin selection theory, but it took 25 years to test this hypothesis with human data. Hill and Hurtado (1991) reported that it would never be advantageous from a purely genetical perspective to stop reproducing altogether. The authors used several hypothetical models that covered the range

of reasonable estimates of maternal cost vs. grandmother benefits. They also tested their predictions against objective ethnographic data derived from their work with the Ache, hunter-gatherers of South America. The Ache data show that offspring with grandmothers survive at somewhat higher rates than those without grandmothers, but the effect is not nearly enough to account for menopause. In a review of the Ache data and other cases derived from hunting–gathering and agricultural societies Austad (1994, p. 255) found no evidence " . . . that humans can assist their descendants sufficiently to offset the evolutionary cost of ceasing reproduction." In contrast Kristen Hawkes and colleagues published several analyses that found substantial benefits from older women trading-off their own reproduction to assist the reproduction their biological daughters (Hawkes 2003; Hawkes et al. 1997, 1998; Hawkes & Coxworth 2013).

One question not addressed by these analyses relates to older women helping nonbiological "daughters," that is, younger women with the kinship name of "daughter," or any other social kinship name, but of no close genetic relationship. Another issue not addressed by the proposals focused on selective advantages to older women, and men, is the role of early life growth and development. The various hypotheses for an evolutionary explanation for menopause and post-reproductive longevity are silent on the role of human childhood and adolescence in human biology, learning, cultural behavior, and ideology.

A simple genetic kin selection mechanism alone cannot account for the evolution of the long, productive, but post-reproductive life stage of women. A consideration of the nature of human biocultural reproduction offers a better perspective. Without question, there is great value from post-menopausal women, and older men, for food production, childcare, and social support. "Grandparenthood" may be defined as an important biological and sociocultural stage in the human life cycle. For women, the universality of menopause makes it possible to develop biocultural models to support a combination of the "oocyte depletion" and "grandmother" hypotheses. Basically, if a 50-year age barrier exists to human female fertility, then the only reproductive strategy open to women living past that age is to provide increasing amounts of aid to their children and their grandchildren. This strategy is compatible with Hamilton's kin selection hypothesis and with the biocultural reproduction model discussed earlier in this chapter.

Holly Smith and I (Bogin & Smith 1996b) were among the first to propose a biocultural model for the evolution of the post-reproductive stage of life history for human women. In our view the focus on genetic selection for menopause of other hypotheses is misplaced. To be sure, there is evidence for "longevity genes" which may account for up to 25% of variation in human lifespan between individuals (Anonymous 2018b; Sebastiani et al. 2017). The remaining 75% of variation, however, is associated with early life health, diet and nutrition, behaviors such as smoking, alcohol consumption, exercise, and reactivity to stressful life events. People with healthier lifestyles from an early age tend to live longer. In another article I proposed a greater emphasis of the lifestyle determinates of longevity via the **reserve capacity hypothesis** (Bogin 2009). The definition of reserve capacity (RC)

as it relates to life history theory was given by Crews (2003) and Larke and Crews (2006) as those somatic, cognitive, and emotional resources that exceed the minimum required for sustaining life and allowing reproduction. Individuals with greater RC have a redundancy and higher quality of cells, tissues, and organ system function and are better able to avoid predation, mount immune defense, recover from trauma, and withstand periods of hunger. These individuals will, on average, have greater reproductive success (RS) and in classic Darwinian selection the '' ... demand for increased RC to achieve greater RS drives the system'' (Larke and Crews 2006, p. 122).

People with greater RC have acquired during their years of growth and development increased biocultural resilience to physical, biological, and emotional stressors. That resilience promotes greater survival to adulthood, adult survival, longevity, and RS. The evolution of childhood and adolescence added five or more years of additional time to human development prior to the onset of reproduction. When environments for growth and health are favorable, this extra time allows humans to develop substantially more RC than any other primate species.

I reanalyzed the "grandmother hypothesis" (GH) of Hawkes and colleagues (1998) in light of my discoveries about the evolution of human childhood and adolescence and the RC hypothesis of Crews and colleagues (Bogin, 2009). I incorporated the human life history stages and the RC hypothesis into a **multilevel selection model** to provide a more complex, and more comprehensive, perspective on some aspects of the evolution of human life history. Some researchers have criticized multilevel selection models as ''circular reasoning,'' but this is not correct. Multilevel selection is part of a maturing view of biological evolution in terms of a nested set of selective processes (Wilson & Wilson 2007). The model I developed begins with a fundamental Darwinian-type selection for decreased birth intervals via the evolution of early weaning (wean early compared with apes) and childhood. This selection operated to first shorten the infancy stage, then prolong the growth period, delay the onset of reproduction, lower women's LRE, increase reproductive success, and, finally, result in greater longevity.

The building of RC during human childhood and adolescence, combined with biocultural reproduction, explain in large part why a greater percentage of human young survive to adulthood than the young of any other primate species. The new life history stages also help to account for the greater RS and greater longevity of human adults over other primates. The "grandmother hypothesis" of Hawkes and colleagues proposes that lower adult mortality had to occur first in order to prolong the period of human growth and development. My multilevel selection model reverses the causality – it is the building of a better, healthier body and the development of greater biological, behavioral, and cultural resilience prior to sexual maturity that leads to greater adult health, fitness, and longevity. Another critical component of this multilevel selection model is human biocultural reproduction, which is derived from associations between the evolution of childhood and the human ability to disconnect alloparental caregiving from direct genetic relatedness between caregiver and care recipient. As detailed earlier in this chapter, biocultural reproduction significantly reduces the LRE of most women. The resources saved by this may be channeled into

greater RC. In turn, the energy, somatic, and psychological savings could be invested into body and cognitive maintenance which may have slowed senescence and prolonged human lifespan by several decades more than our closest great ape kin.

In sum, "valuable grandmothers'' are a consequence of the limitations of mammalian biology in terms of the senescent decline of the reproductive system after age 40 years combined with human biocultural strategies to take greatest advantage of this situation. Viewed in this context, human grandmotherhood may be added to human childhood and adolescence as distinctive stages of the human life cycle.

Conclusion

Perhaps the best summary of the importance of taking a life history perspective of human evolution was stated by Bonner (1993, p. 93): "The great lesson that comes from thinking of organisms as life cycles is that it is the life cycle, not just the adult, that evolves. In particular, it is the building period of the life cycle – the period of development – that is altered over time by natural selection. It is obvious that the only way to change the characters of an adult is to change its development." The stages of human postnatal life from birth to maturity – infancy, childhood, juvenile, and adolescent – shape the biology and behavior of adults and confer upon them greater reproductive success than any other mammalian species.

Human reproductive success is due to the biocultural adaptations of our species. These adaptations may have arisen as both a consequence of, and a response to, the evolution of the human life cycle. The stages of the life cycle and the growth patterns of the human body, the face, and the brain facilitate parental investment in offspring by releasing the potential for nurturing behavior of adults toward infants and older, but still physically dependent, children and socially dependent juveniles. Human culture, in large part, is a response to the need to nurture, protect, and teach these young people. The physical features of childhood and juvenile stages are lost during the time of the adolescent growth spurt. At the end of adolescence, boys and girls enter the social world of men and women. In physical features, interests, and behaviors these young adults are more similar to their parents than to their pre-adolescent selves of just a few years ago. Human adults have two or more decades of prime reproductive life and this is followed by a decade or more of productive post-fertile life. Each new generation follows the cycle of reproduction, growth, maturation, and senescence that was phylogenetically set in place tens of thousands to millions of years ago and continues to be expressed in the ontogenetic development of every human being born today.

5 Growth Variation in Living Human Populations

Population differences in stature, body weight, and other physical dimensions have been documented throughout recorded history. Ancient Egyptian sources mention groups of very short stature people living near the headwaters of the Nile River, possibly ancestors of central African "pygmy" populations alive today (Hiernaux 1974). Museum displays of medieval armor and fashion often provoke visitors to comment on how much bigger European people are today than in the past. Human biologists have recorded the variation in size that exists between living populations and found that it is relatively easy to describe the differences in size, but much more difficult to explain why this variation exits. The causes of population differences in body size, including variation in amounts and rates of growth, are associated with a wide range of hereditary, physical environment (such as temperature and altitude), nutritional, social, economic, emotional, and political factors. Examples of some of these factors were given in previous chapters. The present chapter reviews population variation in growth and development and ends with a consideration of the evolutionary value of population variation in body size.

Population Differences in Body Size

People from different countries grow to different heights. This may be partly due to genetics, but most differences in height between countries have other causes. For example, children and adolescents who are malnourished, or who suffer from serious diseases, will generally be shorter as adults. This is important because taller people generally live longer, are less likely to suffer from heart disease and stroke, and taller women and their children are less likely to have complications during and after birth. Taller people may also earn more and be more successful at school. However, they are also more likely to develop some cancers. (NCD Risk Factor Collaboration (NCD-RisC) 2016, p. 2)

NCD-RisC is a network of health scientists around the world that provides rigorous and timely data on risk factors for noncommunicable diseases (NCDs) for 200 countries and territories.[1] To better understand the worldwide associations between adult height, risks for poor health, and opportunities for higher education and earnings, the NCD-RisC set out to find out how tall people are, on average, in

[1] http://ncdrisc.org/

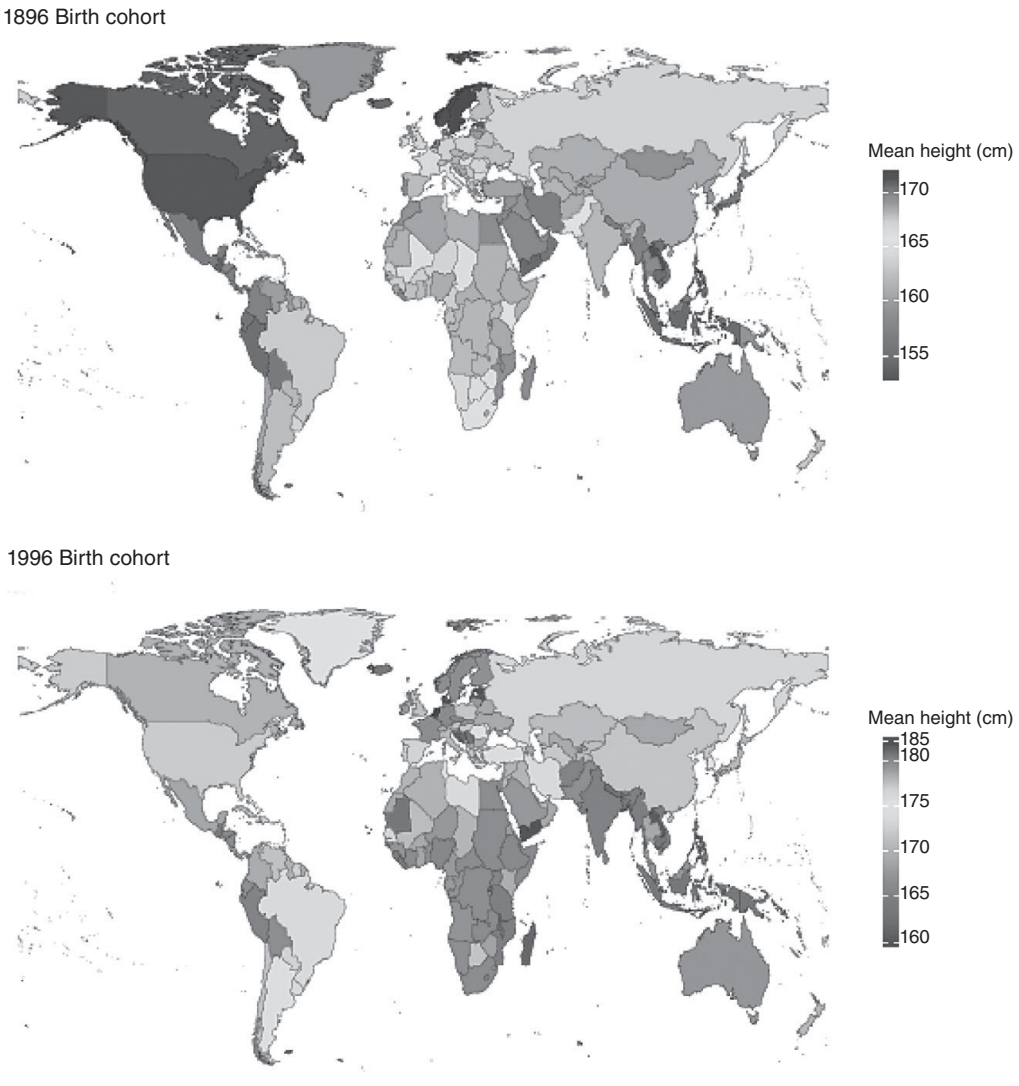


Figure 5.1 Adult height for the 1896 and 1996 birth cohorts for men. See www.ncdrisc.org/height-map-mean.html for interactive version. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

every country in the world in the year 2014, and how average height changed over the past 100 years. The analysis revealed large differences in height between countries (Figures 5.1 and 5.2). The tallest men were born in 1996 (the last birth cohort included in the analysis) in the Netherlands and were nearly 183 cm tall on average. The shortest women were born in 1896 in Guatemala and were on average 140 cm tall. The difference between the shortest and tallest people in all countries is about 20 cm, both for men and for women. This means there are

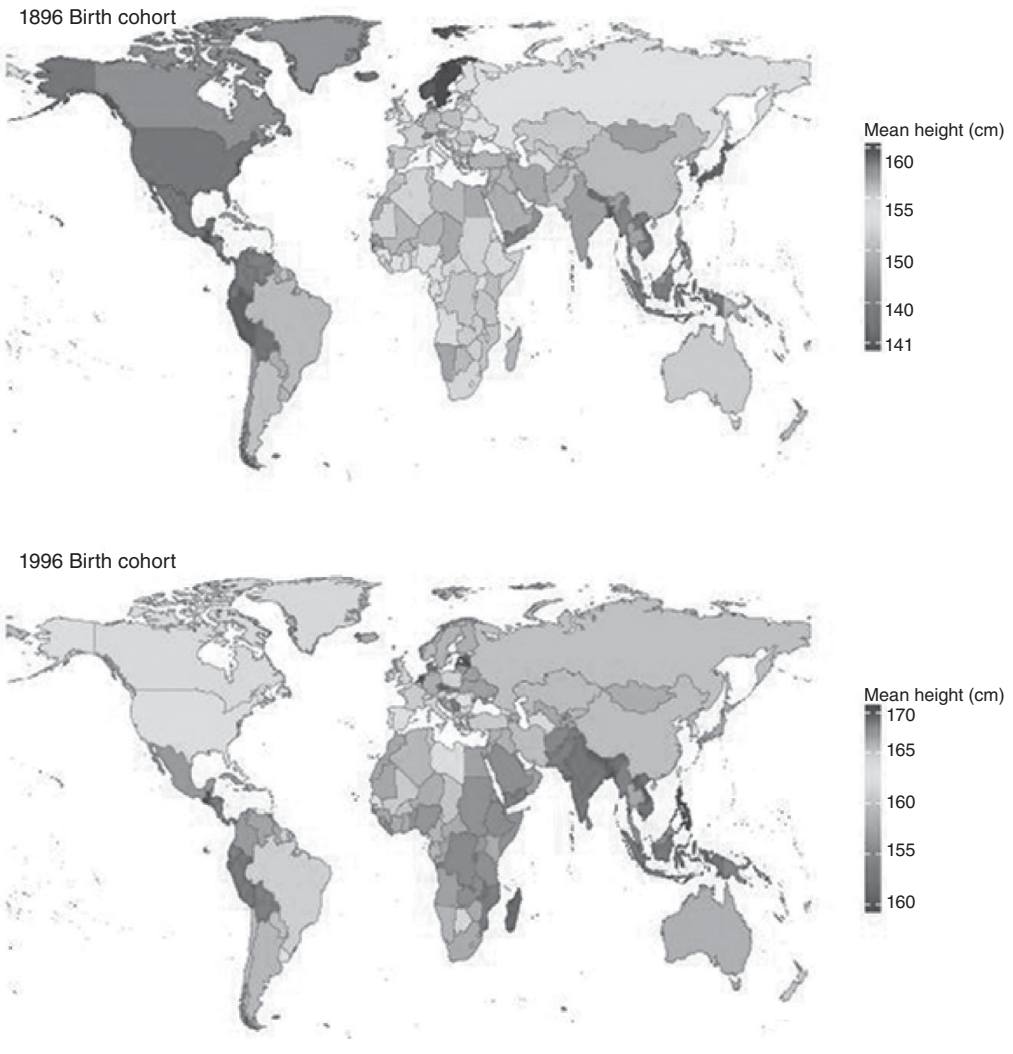


Figure 5.2 Adult height for the 1896 and 1996 birth cohorts for women. See www.ncdrisc.org/height-map-mean.html for interactive version. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

large differences between countries in terms of the risk of developing some diseases.

The way in which height has changed over the past 100 years also varies from country to country. Iranian men born in 1996 were around 17 cm taller than those born in 1896, and South Korean women were 20 cm taller. In other parts of the world, particularly in South Asia and parts of Africa, people are only slightly taller than 100 years ago, and in some countries people are shorter than they were 50 years ago.

A century ago the tallest men and women were living in the United States, Canada, Norway, and Sweden. Today, the tallest people reside in northern, central, and eastern Europe. Canadians are as tall as some of those Europeans, but men and women in the United States are relatively shorter and about the same height as the Spanish.

The NCD-RisC analysis was based on 1,472 population-based studies, with measurement of height on more than 18.6 million participants. There were differences in the selection of participants in the different countries. In the United States all residents, of all ethnic origins, were measured, including Mexican-Americans who tend to be shorter than European-Americans and African-Americans. In the Netherlands ethnic Dutch were measured, but not immigrants from Turkey or Indonesia who are known to be of relatively short stature. These sampling differences introduce some bias into the mean adult height estimates. The data were used to estimate mean adult height for people born between 1896 and 1996 in 200 countries. Adult height was defined as height at age 18 years. For many women this is likely to be the adult height, but not for all women and not for most men. The estimates do not take into account differences in rates of maturation and growth tempo. In some populations men and women may have a slow growth tempo and not reach final height until their early-to-late 20s. Another issue is that there were no measurements of any population prior to 1926 and the adult height for people born before 1926 was estimated by a statistical model. Despite these limitations, the NCD-RisC analysis of trends in height is the most comprehensive and reliable investigation available.

The NCD-RisC publication provides much more information – too much to be adequately presented in this book. A smaller, more manageable list of average height, weight, and body mass index (BMI) for several human populations is given in Table 5.1. As discussed in Box 2.1, BMI was developed as a measure of weight-for-height for populations and is presented here in that context only, and not as a proxy for fatness. This table is a very simple list and includes only average values (means or medians) and not measures of variation about the average, such as variances, standard deviations, or ranges. The simplicity of the table is designed as a teaching aid to explore some concepts of population variation in body size and weight-for-height.

The data are from the years 1970 to 1995, representing the late twentieth century. The samples are listed in descending order according to the average height, an order followed by both the men and the women. Young adults in The Netherlands were then, and are still today, on average, the tallest people in the world. Some of the shortest young Dutch men, those at the third percentile, have a height of 169.3 cm and, thus, are taller than the mean stature of the Aymara men in Bolivia and Maya men in Guatemala listed in the table. Young men in the United States are, on average, shorter than the Dutch and today are shorter than 36 other nations. US women are shorter than young women in 41 other nations. Data for average heights and BMIs for 200 nations, spanning nearly 100 years, are available from the NCD Risk Factor Collaboration (NCD-RisC 2016, 2017). Relative to all 200 nations the United States is

Table 5.1 Average height (cm), weight (kg), and body mass index (BMI), of young adult men and women in several populations measured in the late twentieth century.

		Height		Weight		BMI	
Population, years of measurement	Age	Men	Women	Men	Women	Men	Women
Netherlands, national sample, 1980 (medians)	20	182	168.3	70.8	58.6	21.4	22
United States, national sample, 1977 (medians)	20–21	177.4	163.2	71.9	57.2	22.8	21.5
Africa, Turkana Pastoralists, 1970s (means)	20	174.3	161.6	49.8	47.4	16.4	18.2
Japan, Univ of Tokyo students, 1995 (means)	c.20	171.6	159.1	63.3	50.7	21.5	20
Bolivia, Aymara subsistence farmers, 1970s (means)	20–29	162	149	58.1	52.4	22.1	23.6
Guatemala, Maya subsistence farmers, 1980s (means)	17–18	158.7	146.9	52.2	49.3	20.7	22.8
Africa, Efe Pygmy, 1980s (means)	19–29	144.9	136.1	43.3	40.6	20.6	21.9

Maximum difference in height, Netherlands vs. Efe, Men = 37.1 cm; Women = 32.2 cm.
The data were compiled from published and unpublished sources. Complete bibliographic citations were given in Bogin (1999b).

a "tall" group of people. The Turkana, who are nomadic, animal herding pastoralists living in rural Kenya, are one of the tallest populations of Africa. The Tutsi of Rwanda are about 2 cm taller, on average, than the Turkana. It is a myth that Tutsi (sometimes called the Watutsi) are the tallest people in the world, averaging more than 213 cm (7 feet) tall. In fact, the tallest people of African origin are African-Americans (Cameron 1991). The greater average height of African-Americans compared with Africans in Africa is likely due to the better standard of living in the United States. Even so, African-Americans are, on average, shorter by about 1 cm than European-Americans (Komlos 2010). The reason for the mean difference in height between whites and blacks in the United States is likely due to historical and current-day racism, discrimination, and economic inequality against blacks. More detail on the effects of racism and prejudice on growth is presented in Chapter 7.

The sample of Japanese represents reasonably affluent university students. They are the tallest and heaviest, on average, of any group of young Japanese adults measured in the twentieth century, but they are considerably shorter and lighter, on average, than the Dutch or the Americans. The Aymara of Bolivia and the Maya of Guatemala are native American peoples. Both groups are of very low socioeconomic status (SES). Both live in rural areas and many individuals suffer from mild-to-moderate malnutrition, along with repeated bouts of infections of the gastrointestinal and respiratory systems. Undernutrition and infectious disease are associated with growth retardation, and these are likely to be factors that account for the relative short stature of the Maya and Aymara. The African Efe pygmies may be, on average, the shortest people in the world, and their short stature appears to have a strong genetic component (Hattori et al. 1996; Merimee et al.1987). However, there is a wide range of variation in the stature of individual pygmies. Barnicot (1977)

compared the distribution of male stature of the pygmies with the Tutsi, who have a mean male stature of 176.5 cm, and found that the tallest pygmy men were larger than the shortest Tutsi men. This analysis shows that average figures may be quite misleading for individuals within a population. Even so, the statistics for average size given in Table 5.1 indicate patterns of growth that are useful for descriptive purposes and provide a starting point for the analysis of the causes of such variation.

The rank order of mean weights in Table 5.1 does not follow the same order as stature. On average, US men are the heaviest and Efe women are the lightest of all the populations listed. The relatively tall Turkana men have a lower average weight than that of any other samples, save the Efe. Turkana women have a lower average weight than all samples, except the Efe and the Maya women. The BMIs show that the Turkana and the Efe have the lowest mean values, reflecting their linear body build. The Turkana, Efe, and many other sub-Saharan African peoples have arms and legs that are relatively long in proportion to their total stature (Eveleth & Tanner 1976, 1990). The Turkana and the Efe are also absolutely lean, meaning that their bodies have less fat tissue than other human populations. Together, body proportions and body composition give these two groups a linear body build. The similarity in the proportion of height to weight between these African samples is striking, since the Turkana are, on average, 25.5cm taller than the Efe pygmy sample.

Dutch and American men 20–29 years old have the highest average BMIs, meaning that there is, on average, relatively greater weight for height in these two populations than in the other samples of young men. Little et al. (1983) compared the Turkana with a US reference population and found the greater weight for height of the Americans was due to both more fat and more lean tissue (e.g., muscle), but especially more fat. Since the year 1977, when the US data were published, young adult Americans, 20–29 years old, have increased in mean stature by a few millimeters (Komlos 2010) but men increased in mean weight from 76.3 kg to 83.4 kg and women increased from 61.7 kg to 71.1 kg (Ogden et al. 2004). By the year 2002 the mean BMI of young adult American men and women rose to 26.6 and 26.8, respectively. Virtually all the added weight and increase in BMI is due to fat and today Americans are the fattest population of all the high and middle-income industrialized countries (OECD 2017). Japanese university students, both men and, especially, women have lower average weight-for-height ratios than similarly aged Dutch or Americans. In fact, the Japanese population has the lowest BMI values and the lowest percentage of people classified as overweight or obese of all high-income nations (OECD 2017). Why this is so is not known exactly. The Japanese sample represents a highly educated group, while the Dutch and American data are based on national samples. More highly educated people tend to be less fat than the population at large, at least in many industrialized nations. Aymara and Maya men had BMIs that were lower than for Japanese men, but the women have ratios that are higher than for Japanese women and are even greater than the BMI for US women. One factor influencing the BMI is that relative to the Dutch, Americans, and Japanese, the Aymara and Maya have short arms and legs in proportion to total stature (Bogin et al. 2002; Eveleth & Tanner 1976; Gurri and Dickinson 1990). This means that the

head and trunk of the body contribute disproportionately more to total weight (Bogin & Beydoun 2007). The Maya, and probably the Aymara as well, have less total body fat, on average, than Europeans or Americans, but they have more of it concentrated on their trunks (Bogin and MacVean 1981; Johnston et al. 1984). This results in what is sometimes called a "short and plump" physique, which elevates the weight-for-height ratio. In reality, the adult Maya and Aymara measured in the 1970s and 1980s were not "plump," rather they developed the physique of a short, stocky body as a result of malnutrition and growth retardation in early life, and as adults they were absolutely shorter and had less body fat than populations of the industrialized nations.

Population Differences in Rate of Growth

Rates of growth, or growth tempo, also vary considerably between human populations. Growth tempo is distinct from the amount of growth. Some children are tall for age because they grow at a faster rate than others who follow a slower tempo. Depending on the total amount of time for growth, fast growers may be taller than slow growers as adults, or both fast and slow growers may end up with the same adult height, or those with slow tempo may end up as taller adults. All combinations of amount and rate of growth are possible and seen in groups of healthy, well-nourished infants, children, juveniles, and adolescents.

Velocity of growth in height is presented in Figure 5.3 for four groups of boys: two samples from Guatemala, one of high SES Ladino boys and one of low SES Maya boys (Bogin et al. 1992), a sample of low SES boys from The Gambia, West Africa (Billewicz & McGregor 1982), and a sample of low SES boys from rural India (Satyanarayana et al. 1989). The velocity curves were calculated from longitudinal measurements of height by fitting the measurements to the Preece–Baines model 1 function using algorithms developed by Brown (1983). Today, the Preece–Baines

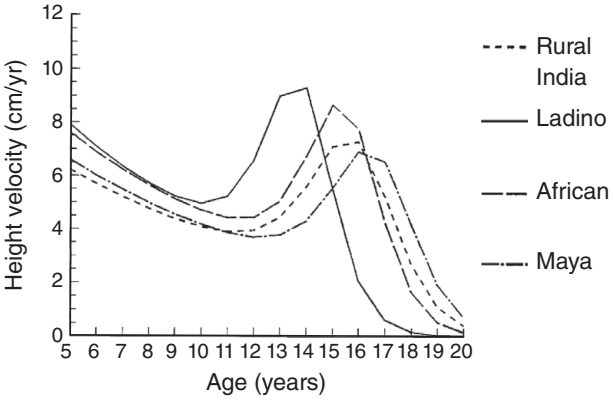


Figure 5.3 Mean-constant curves, estimated by the Preece–Baines model 1 function, for the velocity of growth of Ladino and Maya boys from Guatemala, rural Indian boys, and rural Gambian boys (author's original figure).

function is best implemented via the R Project for Statistical Computing or other commercial statistical packages such as STATA. This mathematical function estimates the mean-constant velocity (that is, the true average curve for longitudinal data) of growth from childhood to the attainment of adult height. Further detail about the Preece–Baines function is given in Box 5.1.

Box 5.1 **Mathematical models of human growth**

Mathematical models of human growth are useful both to describe the shape of growth distance and velocity and to test hypotheses about the influence of early growth events on health later in the life course. One example is the association between rapid weight gain in infancy and risk for overweight and metabolic disease in adulthood. Another example is the effect of exposure to toxins or emotional stress in childhood and its impact on the acceleration or delay of puberty and the timing and size of peak height velocity (PHV) in adolescence.

Michael A. Preece and Mike Baines (Preece & Baines 1978) derived a series of mathematical functions that describe the distance and velocity curves of growth in height from the ages of two years to maturity. The authors described their method of derivation as " . . . purely empirical and has made no pretense to true biological meaning" (p. 17). Marubini and Milani (1986) described the Preece–Baines curves as being based on the assumption that the rate of growth is proportional to the difference between height at any age prior to maturity and height at maturity. The rate of growth, however, is not a simple constant of proportionality; rather it is a function of age. This means, that at different ages the rate of growth may be relatively slow, or relatively fast, compared with other ages, which is the manner in which children actually grow. A set of differential equations was used to calculate the age function for the rate of growth, and the solution yields three functions, of which the following is preferred by Preece and Baines for application to growth data:

$$h = h_1 - 2(h_1 - h_c)/\{exp\,[S_0(t - c)] + exp\,[s_1(t - c)]\}$$

There are five parameters to be estimated in this model: (1) h is height at time t; (2) h_1 is final (adult) height; (3) s_0 and s_1 are rate constants; (4) c is a time constant; and (5) h_c is height at $t = c$. Although the model was derived empirically, Preece and Baines were able to correlate each of the five parameters of the model with "biological" events that occur during growth. The rate constants s_0 and s_1 are highly correlated with the minimal prepubertal velocity of growth and the **peak growth velocity** during adolescence, respectively. Time c has a very high correlation to the age at PHV during the adolescent growth spurt and h_c has a similarly strong association to height at PHV. The relationship of these parameters to the velocity curve of growth is illustrated in Figure B5.1.1. By computation, other growth events may be estimated, such as

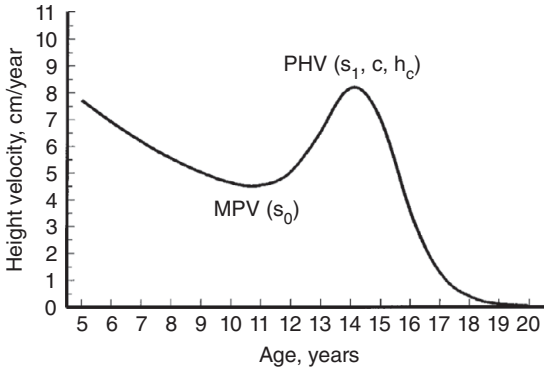


Figure B5.1.1 Height velocity curve of a boy with parameters of the Preece–Baines model indicated. MPV, minimal prepubertal growth velocity; PHV, peak height velocity during the adolescent spurt. Other terms as defined in text (author's original figure).

age at the minimal prepubertal velocity (MPV, also called age at take-off of the adolescent growth spurt), height at MPV, and amounts of growth between MPV, PHV, and final height. The values of these estimates are useful when comparing the pattern of growth of one individual to another, or one population to another.

There are several other mathematical functions that attempt to describe growth. One is called the triple-logistic function (Bock & Thissen 1980). The authors built on ideas proposed by T. Brailsford Robertson in 1908 and Cyril Burt in 1937 that human growth could be described with the use of two or three logistic curves. Since these curves have the same functional form they may be added together to produce a smooth model of human growth. Bock and colleagues developed a triple-logistic function to describe growth in length. This model is the summation of three logistic curves: The first describes growth in infancy, the second term describes growth during the childhood and juvenile stages, and the third adds the contribution of the adolescent spurt. This model fits the distance and velocity curves of growth with reasonable precision. A disadvantage of the triple-logistic model is that it has 10 parameters to be estimated, and adult height must be known. In many cases, the analysis of longitudinal growth data for children does not include a measurement of final adult height and, often, there are 10 or fewer measurements of growth. Thus, more mathematical parameters must be estimated than there are empirical data points, which is statistically undesirable from both a practical and theoretical standpoint.

When appropriate data are available, the triple-logistic model proves to be useful in describing distance and velocity curves of growth. The model is also of value since different growth periods, such as infancy, childhood–juvenile, and adolescence, may be described with precision. Velocities of growth during each period, the age of maximum velocity, and the contribution of each period

of growth to final adult height may be estimated. The transition from one growth period to the next is smooth, and the contributions of each period may overlap, that is, the contribution of prepubertal growth is still active during the early phase of the adolescent growth period. Bock and Thissen (1980) speculated that the genetic and endocrine determinants of development that characterize each period of growth operate in a similar fashion; making a smooth transition and overlapping from one period to the next.

Another group of mathematical models worth mentioning were developed by Jolicoeur and colleagues (Cabana et al. 1993; Jolicoeur et al. 1988). Some of their models estimate growth in length from fertilization to adult height. These models are useful when prenatal growth in length needs to be studied. Other versions of their models estimate growth in height from birth to adulthood. The latter model, called the JPA-2 function, has only eight parameters. Jolicoeur and colleagues find that the JPA-2 model achieves a more accurate goodness-of-fit to real growth data than the 10-parameter triple-logistic model. As is the case for the Preece–Baines function, the JPA-2 model is purely mathematical, with no pretext that its parameters have any biological meaning.

A newer, and possibly more powerful, approach to growth modeling was developed by Tim Cole and colleagues and is called the SuperImposition by Translation And Rotation (SITAR) model (Cole, Donaldson & Ben-shlomo 2010). The SITAR model utilizes a shape invariant approach to characterize differences in growth pattern between individuals. A shape invariant model assumes that growth curves have a common shape, which is the case for human distance and velocity growth. The Preece–Baines model also is based on the human shape invariant curve of growth. One advantage of the SITAR model is that it takes the longitudinal growth data for individuals and matches them to three mean curves for the sample: (1) a *Size* curve (amount of growth) calculated by how much the mean curve is shifted up or down to best match the individual's curve. An example of output from the SITAR model is given in Figure B5.1.2. Larger size (taller, heavier, etc.) is an upward shift and smaller size a downward shift, (2) a *Timing* curve (tempo of growth) which indicates the age at peak growth velocity (APV) and is calculated by shifting the mean curve left or right to best match the APV on the individual's curve, where a rightward shift indicates later maturation and leftward shift earlier maturation, measured in years, and (3) an *Intensity* curve (growth velocity) which indicates the peak growth velocity (PV), and is calculated by how much the mean curve is "squeezed" together or "stretched" apart to best match the PV on the individual's curve, where "squeezing" is for greater intensity and "stretching" for lesser intensity. These translations and rotations of the data result in parameters for the three mean curves for the entire sample, these are called "fixed effects," and parameters for the three curves per individual – *size*, *timing*, and *intensity*, called random effects - that summarize the individual

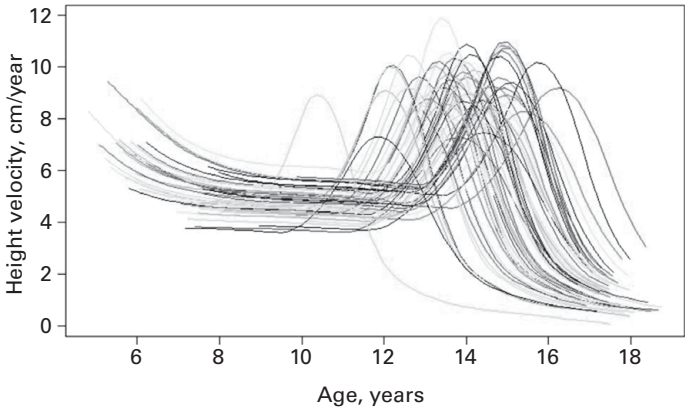


Figure B5.1.2 SITAR model output for longitudinal measurements of height from the Universidad del Valle de Guatemala Study of Child and Adolescent Development. Each curve is a SITAR estimate of growth velocity for one participant in the study. Variation in growth (a) size (height), (b) timing (age at peak velocity), and (c) intensity (amount of peak velocity) is captured by the model. A mean curve for all the study participants may also be estimated and that curve would resemble Figure B5.1.1. Original figure by Liina Mansukoski, with permission. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

growth patterns. A SITAR statistical package in the R computing environment is available.[2]

A list of ages at peak height velocity (APHV) during adolescence in several populations estimated with the SITAR model is presented in Table B5.1.1. The comparison helps to contextualize differences in adolescent growth between girls and boys, as well as between earlier and later maturing populations. Sex differences are smallest for African-Americans. Guatemalan boys and girls of, on average, middle SES have mean APHV similar to that of twenty-first-century Tsimane forager-horticulturalists from Bolivia and urban non-African-American US adolescents. Girls and boys in the rural Gambian population studied intensively by the UK Medical Research Council (MRC) since 1947 have the latest mean APHV. Why these similarities and differences by sex and population exist are not well understood. Some possibilities are discussed in Chapter 7. The Gambian estimates are especially puzzling. The APHV for girls and boys are significantly later than the other populations listed, even the other African-origin groups. The late APHV of the Gambians suggests poor health, poor nutrition, and high emotional stress as likely causes. The MRC Gambian Research Unit website states, "The MRC Unit The Gambia ... is the ... single largest investment in medical research in a low and middle income country ... [and] represents a unique concentration of scientific expertise and high quality research platforms in the West African region."[3]

[2] https://cran.r-project.org/web/packages/sitar/vignettes/Fitting_models_with_SITAR.html
[3] www.mrc.gm/about-us/

Table B5.1.1 List of some studies using the SITAR growth model to estimate age at peak height velocity (APHV). From Mansukoski (2019), where the full citation for each Reference may be found.

Studies reporting a SITAR age at peak height velocity (APHV)				
Sample	Data collected	Reference	Female APHV	Male APHV
Qom, Argentina	2011–2015	(Martin & Valeggia 2018)	10.8	–
South Korea[a]	2005	(Cole & Mori 2018)	~10.9	~12.5
African-American, USA[b]	2002–2010	(McCormack et al. 2017)	11.0	11.6
Colorado, USA	1992–2002	(Hockett et al. 2019)	11.1	12.9
Tsimane, Bolivia	2002–2015	(Blackwell et al. 2017)	11.3	13.2
Guatemala City	1953–1999	(Mansukoski, 2019)	11.4	13.3
Non-African American, USA[b]	2002–2010	(McCormack et al. 2017)	11.6	13.4
ALSPAC, UK	1991–2011	(Frysz et al. 2018)	11.7	13.6
Timor-Leste	2009–2016	(Spencer et al. 2018)	11.7	14.2
Somerset, UK	1981	(Cole et al. 2015)	12.5	13.9
South Korea[a]	1965	(Cole & Mori 2018)	~12.5	~14.8
Gambia	1995–1996	(Prentice et al. 2012)	13.1	16.1

[a] APHV estimated from SITAR velocity curves, no exact value reported by authors.
[b] The samples live in five major US cities: Philadelphia, Cincinnati, Los Angeles, New York City, and Omaha.

Despite the investment, expertise and more than 70 years of activity by the MRC, the Gambian people subjected to the research do not appear to have reaped much benefit in terms of their physical growth and maturation. Further discussion about the failure of nutrition and health interventions to improve growth and development of groups such as these rural Gambians is found in Chapter 7.

There are several analyses of the pros and cons of each of the growth models described here. One recent article reports that both the Preece–Baines and the SITAR models provide unbiased estimates of age at PHV (Simpkin et al. 2017). SITAR estimates are less reliable when measurement error in the raw data is large, which is a common issue in hospital-based measurements of body length/height, weight, and other body dimensions. The Preece–Baines model is computationally inefficient, taking up to 24 hours to fit data sets with hundreds of individuals. SITAR can also take hours to run. Both models assume that an adolescent growth spurt is present, but some perfectly healthy individuals, especially girls, and some human groups suffering from moderate-to-severe nutritional stress lack an adolescent growth spurt. For these individuals and groups the Preece–Baines and SITAR models are inappropriate.

The mathematical parameters of some of the mathematical functions described here have association with the biology of human growth, but all the models are devoid of fundamental biological meaning. These models describe the shape of growth curves and the timing of some growth and maturation events, but not why or how growth takes place. A discussion of mathematical models of growth with biological meaning is presented in Chapters 6 and 7.

The Maya, the rural Indians (from the Hyderabad region of India), and the Gambians were described as suffering from poor living conditions, including high rates of disease and chronic undernutrition. In contrast, the high SES Ladino boys are generally healthy and well nourished. The Maya, rural Indian, and rural Gambian boys, shown in Figure 5.3, have slower velocities of growth during all stages of development. These three slower growing groups also have a longer period of childhood, juvenile, and adolescent growth; note that the Preece–Baines function estimates that growth continues beyond age 20 years. Despite this the Maya, Indians, and Gambians end up significantly shorter than the high SES Ladinos (estimated mean adult heights are: high SES Ladino, 176.9 cm; Maya, 169.15 cm; rural Indians, 158.2 cm; Gambians, 170.8 cm).

Mean-constant velocity curves for high SES Ladina girls, Maya girls, and rural Gambian girls are shown in Figure 5.4. Compared with Ladina girls, the Maya and Gambian girls show a general pattern of slower growth and delayed maturation, although this is more pronounced for the Gambians. An exception to this general pattern is that Maya girls grow more rapidly than the Ladina girls at age eight. Despite that anomaly, Maya and Gambian girls are shorter at all ages, including adulthood, than Ladina girls (the estimated mean adult heights are: high SES Ladina, 162.95 cm; Maya, 151.8 cm; Gambian, 158.8 cm).

The difference in height between Maya and Ladinos may be due, in part, to genetic determinants of amount of growth. However, it is not possible to assess that determination in these samples. Moreover, an explanation that relies heavily on the genetic limitation of Maya growth is not particularly useful, as shown by the enormous plasticity in growth of Maya refugees in the United States (described in the Introduction and reevaluated later in this chapter). The low SES of the Maya of Guatemala correlates with their chronic mild-to-moderate undernutrition as measured by body composition, higher rates of disease, and generally unfavorable environment for growth. In this regard the Maya pattern of growth in height is similar to that of other disadvantaged populations, such as the rural Indians and Gambians depicted in Figures 5.3 and 5.4. The result is shorter stature at all stages of growth for the Maya, rural Indians, and Gambians compared with the high SES Ladinos. Because of the wide geographic, ethnic, and sociocultural differences between these groups, and despite possible genetic differences, it seems that it is

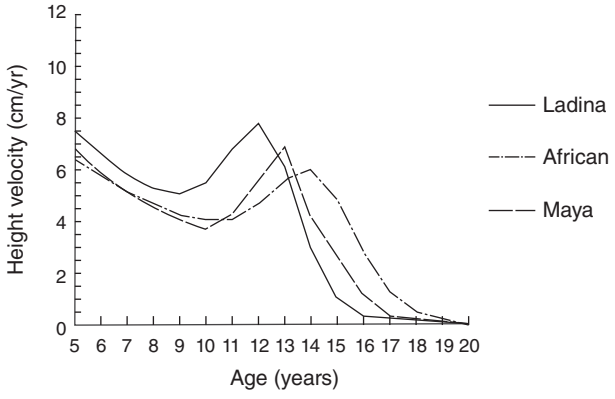


Figure 5.4 Mean-constant curves, estimated by the Preece–Baines model 1 function, for the velocity of growth of Ladina and Maya girls from Guatemala and rural Gambian girls (author's original figure).

the shared negative environment for growth that produces the similar pattern in amount and rate of growth in height in the Maya, Indian, and Gambian samples.

Why Are Pygmies Short?

As described earlier in this chapter, African Pygmies have the shortest average stature of all human groups. It is likely that there are important genomic, epigenetic, and endocrinological reasons for pygmy short stature, as the few studies of Pygmy physiology report low levels of growth hormone (GH) and/or insulin-like growth hormone-1 (IGF-1) and impairment of the IGF-1 binding protein-3 (IGF1BP3, Merimee et al. 1987). Growth in height is strongly associated with GH and IGF-1 levels as well as healthy functioning of IGF1BP (reviewed in Chapter 6). It is well established that adult Pygmies are short-statured, but the pattern of Pygmy growth from birth to adulthood was, until 2015, largely unknown. There was only one mixed-longitudinal study of Pygmy infants and children of known age. In that study, Bailey (1991) found that Efe Pygmies were smaller at birth than their non-Pygmy Lese neighbors and the size difference increased up to the age of five years. Warfare in the region prevented continuation of the study. From this one study it seems that Pygmy rate of growth is slow prenatally and continues to be slow through childhood.

There were also speculations that the short stature of Pygmy adults is due to the absence of an adolescent growth spurt (Merimee et al. 1987). If this were correct, then Pygmies would be the only human population without an adolescent growth spurt which, as explained earlier in this book, is a defining characteristic of the human species. Other speculations were proposed that Pygmies mature sexually much earlier than other human populations and the trade-off between early reproduction and body growth causes Pygmy short stature. A report published in 2015 by Ramirez Rozzi and colleagues demolished both of these fanciful speculations. The researchers

discovered medical records at a Catholic Mission that served the Baka Pygmies of Cameron, who are part of the West Africa cluster of Pygmy groups (located in Cameroon, Central Africa Republic, Congo, Gabon, and Western Democratic Republic of Congo [DRC]). The Efe Pygmies studies by Bailey and colleagues are part of the East African cluster (Rwanda, Uganda, and Eastern DRC).

The Mission records included birth weights since the year 1988. Starting in 2007 and ending in 2014 the researchers measured height and weight of 275 boys and 274 girls of known age living in the locality. About 50% of the participants were measured on two or more occasions, allowing for the calculation of growth velocities. The researchers reported that, "Notably, growth in the Baka is characterized by the presence of growth spurts during adolescence, and they attain maturity at a similar age than other populations: the Baka therefore have the same human life cycle as all other human populations" (p. 2). Also reported was a difference between Baka and Efe growth rates in the first two years after birth, "We find that the short adult stature in the Baka results from a low rate of growth during the first 2 years of life, which produces a lasting delay in growth compared with European reference figures. Baka children are no different in size compared to other non-Pygmy African populations during the first two years of life, but they become smaller from the age of 3 years" (p. 2). The Efe, in contrast, were already small at birth and grew more slowly than non-Pygmies thereafter.

The two clusters of African Pygmies – Efe in the East and Baka in the West – are estimated to have been reproductively separated for about 20,000 years. Ramirez Rozzi and colleagues note that small stature human populations exist in many parts of the world and their common phenotype is likely due to convergent evolution. This implies that different patterns of growth could produce the same short stature. The Efe study shows a growth pattern of slow growth rate and small size pre-natally and post-natally, while the Baka study shows a pattern of slow growth only after age two years post-natally. There are known growth regulating mechanisms, such as the action of the hormone IGF-2 which is most active in the pre-natal and immediate post-natal periods, that may explain the Efe vs. Baka difference.

Differences in Growth between Boys and Girls

Population differences in growth may also be considered in relation to sex, in that we may treat boys and girls as belonging to separate biological and social populations. In general, boys and girls differ in growth tempo by approximately 15%, with girls, on average, advanced over boys in skeletal and sexual maturation. Of course, some boys are in advance of some girls in the same population, but the differential pace by sex is quite constant, on average, throughout childhood and adolescence in all human populations. In the analysis of Guatemalan boys and girls just described, my colleagues and I found that the effects of the environment on growth interact with sex. The difference in height between Maya and Ladino boys is established during childhood and remains fairly constant during juvenile, adolescent, and adult stages. In contrast, the difference in height between the Maya and Ladina girls

increases from childhood to adolescence and reaches its maximum at adulthood. The cause of this interaction is not known. However, the joint effect of two hypotheses about human growth may be considered to propose a reason for the interaction.

One hypothesis is that growth during the childhood and juvenile stages is more sensitive to environmental factors and growth during adolescence is determined more by genetic factors. There are several empirical studies which tend to support the hypothesis (Frisancho et al. 1980; Johnston et al. 1976; Martorell et al. 1977). More recent investigation finds that growth during adolescence may also be highly sensitive to the social environment (Aßmann & Hermanussen 2013; Hermanussen et al. 2014b). The discussion of the evolution of childhood and adolescence plasticity of growth in Chapter 4 lends theoretical support to the hypothesis that both of the uniquely human stages of development, childhood and adolescence, are highly sensitive to environmental influences. Both stages encompass many years of growth and both stages take place in the intensely social context of human biocultural reproduction. There will, of course, be some genetic influence as growth at all stages of development is regulated by the extensive interactions between genomic, epigenetic, nutritional, social, emotional, and other environmental factors.

The other hypothesis is that girls are better "buffered" against environmental factors that are harmful to growth, especially negative influences such as undernutrition and disease, than boys. In the present context, "buffering" means that the growth of girls is less likely to be impaired, reduced, or stunted by harmful environments. There is theoretical and empirical evidence in favor of this hypothesis based on physical growth, morbidity, and mortality (Elsmén, et al. 2004; Nikitovic & Bogin, 2013; Stini 1985; Stinson 1985; Zhao et al. 2017). Suffice it to state here that the growth of boys, from prenatal life to adulthood, appears to be more "sensitive" to environmental factors than the growth of girls. Why this is so is not known with certainty. One line of thought is that from conception onwards the growth and development of girls is closer to their adult state, because girls mature, on average, at a faster tempo than do boys. This means that girls have less developmental time to respond to environmental pressures. Other possibilities exist in terms of hormonal-stress interactions and other influences. Sex-differences in "buffering" will continue to be actively researched.

The multiple and complex ways in which the growth of real people takes place, as opposed to theoretical or statistical models, may be observed for the Guatemala Maya and Ladinos. Both Maya boys and girls are shorter than Ladino children during childhood. The environment for growth of the low SES Maya may be so powerfully negative that despite the girls' "buffering" (hypothesis 2) their growth deficit is about the same as that for the Maya boys. Even so, some evidence of that buffering does exist, in that the Maya and Ladina girls do not differ in growth velocity at the start of the adolescent spurt, the so-called age at "take-off" (defined in Box 5.1 as that point in time at the end of the juvenile stage when growth velocity reaches a minimum and then changes from decelerating to accelerating). In contrast to the girls, Maya and Ladino boys do differ in growth velocity at "take-off." However, neither Maya boys nor girls differ from Ladino boys or girls in the velocity of growth at PHV. Compared

with Ladino boys, the Maya boys continue to show developmental delays as adolescence proceeds, for instance Maya boys have a later age at PHV. The Maya boys' delayed maturation is probably due to the continuation of the negative environmental milieu in which they live. The developmental delay is not prolonged enough to compensate for the reduced rate of growth during childhood. Consequently, the height difference between Maya and Ladino boys is maintained until adulthood.

In contrast to the Maya boys, Maya girls pass through adolescence at about the same rate as Ladina girls. The age at "take-off" and PHV for the Maya girls is a bit later than for the Ladinas, but the difference is not significant statistically. Perhaps this is due to the girls' "buffering" against the environment. Thus, Maya girls proceed through adolescence more rapidly than the Maya boys, which is in accordance with the typical sex differences in rate of maturation. The Maya girls' amount of growth is less than that of the Ladina girls during adolescence because the Maya have a shorter time interval between "take-off" and PHV. As a result, the difference in height between Maya and Ladina girls increases to a greater extent than the difference in height between Maya and Ladino boys. These patterns of Maya and Ladino growth provide no clear evidence in favor of any single hypothesis relating to child vs. adolescent or boy vs. girl growth and development.

Population Variation in Skeletal, Dental, and Sexual Maturation

Other aspects of biological maturation may differ from one population to another. There are many studies on age at menarche (first menstruation), which is a single event of reproductive system maturation, but fewer longitudinal studies of skeletal, dental, and sexual maturation of Europeans, including North Americans and other European-origin populations (described in Chapter 1). Longitudinal studies of non-European samples are even less common. Two African studies from the 1950s and 1960s were conducted in Dakar, Senegal and Kampala, Uganda. Analysis of the Dakar data by Massé and Hunt (1963) found that the dental and skeletal development, as measured by radiographs of tooth formation and of the appearance of ossification centers in the hand and wrist, of African blacks were, on average, more advanced at birth than European whites living in Dakar. However, by two to three years of age the Africans fell behind Europeans in these developmental measures. An important early study of the effects of kwashiorkor on African infants 6 to 30 months of age from the Kampala region was published by Peter R. M. Jones and Reginald F. A. Dean (Jones and Dean 1956). Kwashiorkor is a form of severe malnutrition requiring medical intervention to save the sufferer. It is caused by a lack of protein and other essential nutrients in the diet and is most commonly seen in infants and young children during times of famine. The main sign of kwashiorkor is too much fluid in the body's tissues. In their article, Jones and Dean review the available studies of skeletal maturation of Africans and report that in comparison with European-origin references two studies found the Africans matured more slowly and one study found that girls but not boys were "considerably in advance" of the references. In their own study Jones and Dean compared the severely malnourished

infants and age-matched controls of nutritionally healthy infants to the US skeletal maturation references published by Greulich and Pyle (1959) for European whites. Jones and Dean used a version of the references published in 1950. They assesed the state of maturation of each bone in the hand and wrist and compiled a list of infants showing retardation, that is, nonappearance of the bone or ossification center at the age it is present in the reference. The severely malnourished cases were, for their chronological age, all much delayed in the appearance of hand-wrist ossification centers. About two-thirds of severely malnourished boys and girls lacked the appearance of ossification centers expected for their age. In contrast, the skeletal maturation of boys in the healthy sample was, essentially, identical to the European references and the healthy girls were slightly advanced in skeletal maturation.

Despite the heterogeneity of findings from these mid-twentieth-century reports, the more recent literature presents a strong case for African precocity in skeletal maturation. In reviews of the topic, Tanner (1962, 1981), Eveleth and Tanner (1976, 1990) and Garn and Bailey (1978) state unequivocally that Africans in Africa and African-Americans are consistently advanced in the tempo of skeletal maturation compared with European-origin people. A careful inspection of these reviews suggests that the authors selectively reported only those studies finding that blacks were advanced over whites in the first two–three years of life. Garn and Bailey (1978) suggested that African blacks are "genetically programmed" to develop more rapidly than European whites, but adverse nutritional, disease, and socioeconomic environments for growth of African children results in delays in development after age three years compared with the Europeans. The neat partitioning between genetic and environmental determination was, perhaps, an overly simple assessment of the data. As discussed in the Introduction of this book there is no simple genetic vs. environmental determination of any aspect of growth, development, or maturation. Rather, there are many complex interactions that occur at many levels from cells to society.

Nevertheless, twenty-first-century studies continue to search for support for the early-life skeletal advancement of African-origin people over European-origin people. One study assessed skeletal ages of healthy Americans aged 0–19 years old of European (n=260) and African (n=274) descent born after the year 1980. There were nearly equal numbers of boys and girls of both ethnicities (Mora et al. 2001). The participants were recruited from schools in Los Angeles county, California and were assed to be healthy via a clinical examination by a pediatric endocrinologist. Individuals outside the 5th and 95th percentiles for height- and weight-for-age were excluded to control for effects of under- and over-nutrition on skeletal age. There was no control for SES. The authors used the Greulich and Pyle references and calculated the difference between skeletal and chronological ages as their measure of advancement or delay in skeletal maturation. They reported that, " . . . prepubertal American children of European descent have significantly delayed skeletal maturation [by about 3 months] when compared with those of African descent [who are advanced of the reference by one-tenth of a year]; and, postpubertal [European-American] males have significantly advanced skeletal maturation when compared with postpubertal [African-American] males" (Mora et al. 2001, p. 624). What do

these findings mean? The answer seems to be "not much." Both groups end up as skeletally mature adults. The three-month average advancement of the pre-pubertal African-Americans may be significant in a statistical sense, but is unlikely to have much practical biological or health importance. Since the African-Americans are a bit advanced early in life they have to be delayed later in life because the skeletal maturation references are based on a type of 0–100% scale.

A study from South Africa reported that black Africans are delayed in skeletal maturation compared with white Africans. The data come from the Birth to Twenty cohort of births during a one-week period in Johannesburg and Soweto, South Africa. Bone maturity was assessed between the ages of 9 to 20 years, using the Tanner-Whitehouse III method (Cole et al. 2015). It was reported that black African boys were delayed by seven months compared with boys of white ethnicity. Black and white girls showed no differences in the pattern or timing of skeletal maturity. The seven-month delay is of both statistical and practical importance. The authors suggest that reasons for the delay may include the much lower social and economic status and poorer living conditions of black South Africans compared with whites in combination with a greater sensitivity of boys than girls to harsh living conditions.

Teeth

When infants and children of European ancestry (whites) and predominantly African ancestry (blacks) living in the United States are matched for SES variables, such as mother's occupation and education, it is found that the black infants and children are, on average, consistently advanced over white infants and children in the formation and emergence of the permanent teeth (Garn & Bailey 1978). A tendency for precocity in dental development of blacks over whites has also been observed during the prenatal period. These findings were used to further support the authors' hypothesis of a difference in the "genetic programming" for rate of maturation existing between people of African and European descent.

A review by Gillett (1998) of dental development, including her own study of healthy Zambian urban schoolchildren (n=543), found that the average age at tooth emergence of populations in Africa is consistently advanced over that of populations in North America and Asia. The African populations live under generally less favorable environmental conditions than the other groups, and to the extent that poor living conditions tend to delay both growth and development the findings are somewhat surprising. Especially puzzling is the fact that African-Americans are delayed relative to Zambians and Ugandans in the emergence of several teeth.

There are some important factors that confound understanding of the possible role of genes vs. environment in the age at tooth emergence. One is that tooth emergence is a brief and fleeting event. The criteria used to assess when, and by how much, a tooth has emerged through the gum can, and does, vary greatly from one study to the next. Data reviewed by Gillett come from eight different studies, spanning the years 1919 to 1995. Methodological variations between studies can significantly alter the reported mean ages of emergence. Dental maturation researchers such as Arto

Demirjian (1986) reject tooth emergence and insist on analysis of tooth crown and root formation as the only valid method for dental maturation. A second problem is that the loss of a deciduous tooth can accelerate the emergence of the underlying permanent tooth. If children from poorer African families lose more deciduous teeth, then they would also have earlier permanent tooth eruption compared with African-Americans, Europeans, and Asians. Accordingly, it is difficult to make a clear case for the relative importance of genetic and environmental influences on dental development in these geographically diverse groups of people.

Another issue is statistical. All of the studies reviewed so far used mean or median age to estimate the age of tooth eruption. Forensic anthropologists who specialize in chronological age estimation of young people based on their skeletal and dental maturation currently recommend other statistical methods, such as probit regression analysis, to more precisely estimate the age of transition into maturity stages. Also recommended is the use of a range of chronological age, rather than a single average age, to compare groups (Elamin et al. 2017). Elamin and colleagues calculated mean ages of entry into tooth formation stages using probit regression in two samples of healthy people from Khartoum, Sudan between the ages of 2 to 23.99 years old. One sample was of Arab ethnic origin (848 boys, 802 girls) and the other sample was of non-Arab African ethnic origin (846 boys, 402 girls). The researchers reported no statistically significant difference in the mean age at entry into each stage. Moreover, even where there was an age difference, there was no consistent or clear pattern in the tooth stage transition. The researchers reviewed other literature and reported that their findings are in agreement with two other studies: (1) comparison of mean age at stage-entering of children from Australia, Belgium, Canada, England, Finland, France, South Korea and Sweden; and (2) comparison between whites and Bangladeshi groups in London. Compared with these two previous studies, the Sudanese groups entered some stages earlier and other stages later. There was no consistent evidence for the dental maturation precocity of African or African-origin groups.

A systematic review of the topic by Almonaitiene, Balciuniene and Tutkuviene (2010) concluded that, "Permanent teeth eruption is a complex process that can be influenced by a number of general factors: genetics, nutrition, preterm birth, socio-economic factors, body height and weight, craniofacial morphology, hormonal factors and various systemic diseases" (p. 71). The most important of these factors were genetic/hormonal disorders and systematic diseases, such as vitamin D deficiency, cerebral palsy, HIV infection, Celiac disease, and heavy metal intoxication. Skeletal maturation is also influenced by many of these same factors. Future research will need to include a full consideration of these factors as well as appropriate statistical analysis and its interpretation to assess population differences in both skeletal and dental maturation.

Secondary Sexual Characteristics

Rates of maturation of different populations may also be compared by examining the stages of development of the breast, the genitals, and pubic hair. Assessing the rate of

maturation by using these secondary sexual development events serves as a complement to skeletal and dental maturation and may be used as an alternative test of the "genetic programming" hypothesis. The surveys of secondary sexual maturation stages in US girls and boys conducted by Marcia E. Herman-Giddens and colleagues were discussed in Chapter 2. Those surveys found that African-American girls and boys showed statistically significant earlier mean ages for entry into Tanner Stages of genital and pubic hair than white and Hispanic children. African-American girls showed the first signs of breast development and achieved menarche at younger average ages that whites or Hispanics. These US-based studies might be interpreted to support the "genetic programming" hypothesis, at least for the onset of sexual development.

The timing of appearance of Tanner Secondary Sexual Maturation Stages has been carefully studied for South African boys and girls by Noel Cameron and colleagues. In Chapter 2 their findings were discussed in relation to the sexual maturation of severely undernourished South African children. In a related study, Cameron et al. (1993) assessed the development of secondary sexual characteristics in 300 urban and 352 rural South African girls and boys. The sample ranged in age from 6 to 19 years and the ethnicity of both groups was described as "black African." The rural children were of low SES, but of apparent good health as no child showed "any overt sign of illness" (p. 585). The urban sample all attended private, fee-paying schools in Soweto and Johannesburg, indicating they come from middle- to upper-middle class families. The South Africans were compared with Swiss and British children, who represent healthy, well-nourished populations. The results are presented in Figure 5.5 for the girls and Figure 5.6 for the boys.

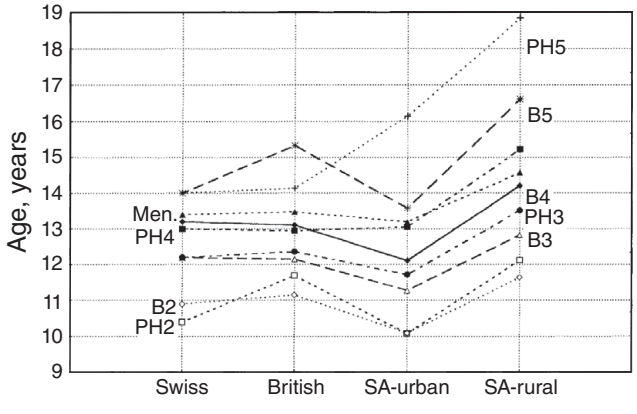


Figure 5.5 Pubertal stages for girls. The Swiss and British samples represent healthy, well-nourished European populations. The SA-Urban sample represents middle to high SES South African blacks living in the Johannesburg area. The SA-Rural sample represents low SES South African blacks living in an impoverished rural area. The median age of attainment of the stages of breast development are indicated by B2 to B5. Median ages for the stages of pubic hair development are indicated by PH2 through PH5. The median age at menarche is indicated by "Men." Based on data published by Cameron et al. (1993)

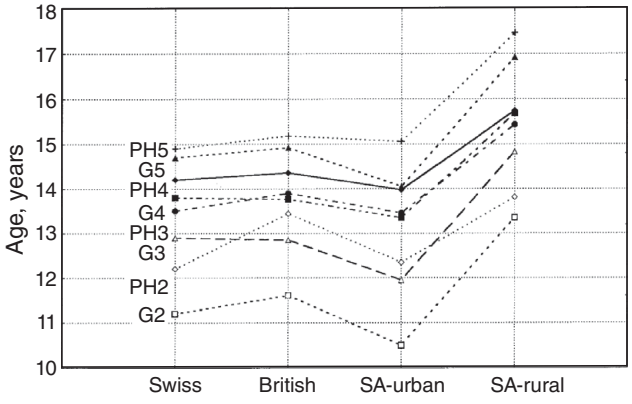


Figure 5.6 Pubertal stages for boys. The Swiss and British samples represent healthy, well-nourished European populations. The SA-Urban sample represents middle to high SES South African blacks living in the Johannesburg area. The SA-Rural sample represents low SES South African blacks living in an impoverished rural area. The median age of attainment of the stages of genital development are indicated by G2 to G5. Median ages for the stages of pubic hair development are indicated by PH2 through PH5. Based on data published by Cameron et al. (1993)

The South African urban girls showed signs of sexual development, both PH2 and B2, at the youngest ages. The PH2 stage occurred 0.32 years earlier than for the Swiss girls. The first sign of sexual development for the British girls was the B2 stage, but even this occurred 1.06 years later than the B2 stage of the South African urban girls. Of these three well-fed and healthy groups, the South African urban girls completed their sexual development at the latest age, reaching the PH5 stage 2.14 years after the Swiss girls. In fact, it took 6.1 years for the South African urban girls to proceed from PH2 to PH5, but it took the Swiss and British girls only 3.6 years to complete all stages of sexual development.

The data for boys showed the same basic pattern as for the girls. South African urban boys had the earliest onset of sexual maturation, with G2 occurring 0.7 years earlier than for the Swiss boys and 1.11 years earlier than for the British boys. All three groups reached the PH5 stage within 0.28 years of each other. The South African urban boys took 4.56 years to complete their sexual development, which was longer than the Swiss boys by 0.85 years, and longer than the British boys by 0.99 years. The rural South African boys began puberty at the latest age and reached each stage of development at the latest age. Even so, these rural boys completed all the stages in 4.1 years, meaning that once started, their rate of sexual maturation was more rapid than the urban South African boys.

What do all these comparisons between urban South Africans, Swiss, and British mean? The answer is, probably not much. The average differences may be influenced by a complex matrix of interacting factors, from genomes to society, but the variation between the different and far-flung geographic groups is within the normal range of variation found within any one of the regions.

Some healthy adolescents mature faster and some slower, but all eventually become sexually mature adults.

Turning to the data for the rural South Africans we see differences of biocultural importance. Every stage of sexual maturation for both the girls and the boys is delayed compared with the other three samples. The poorer living conditions of this rural sample seems to override any of the other possible factors that influence the tempo of sexual maturation. So, what is one to make of the "genetic programming" hypothesis for earlier maturation in Africans, or African-Americans? The answer is, possibly of importance in the clinical assessment of normal vs. pathologically early or delayed sexual development. It may be inappropriate to assess the maturation of African-origin patients using references developed for Northern Europeans.

If we broaden our coverage to include samples of healthy boys and girls from other geographic regions, then the African vs. European comparisons become less interesting. Data compiled by Eveleth and Tanner (1990) indicate that Turkish girls begin sexual maturation, that is achieve the B2 stage, at a median age of 10 years, which is 0.09 years earlier than for urban South Africans. Girls in São Paulo, Brazil have an even earlier mean age for the onset of puberty, 9.7 years for the B2 stage. São Paulo boys have the earliest mean age for puberty onset of any population, with a G2 stage at 9.1 years. Brazilians have a diverse heritage, including African, European, Native American, and Asian genetic and cultural admixture. Viewed in this more global context, the African vs. European differences in rates of maturation are just a small part of the larger variability and plasticity in development that is found across many human populations. The same is true for the ethnic variation in skeletal and dental development described above. Genetics accounts for only a small portion of any of this population variation and is of little biocultural interest unless the genomic influence is medically important.

The Extensive Interacting Matrix of Variables Associated with Population Variation in Growth, Development, and Maturation

The research literature and textbooks about human growth are fond of stating that human size is the outcome of gene-by-environment interactions. This nineteenth-century orthodoxy was part of the previous editions of this book and the section you are now reading was titled "Hereditary and environmental interactions as the cause of population variation." I used the word "hereditary" in place of "gene" (or "genetic") because heredity may include contributions from beyond the genome, such as from family behaviors related to child rearing, exercise, food attitudes, and more that are passed from generation to generation. I now feel that my use of "hereditary" was unclear and that many readers assumed I meant "genetic." To be clear, this section has a new title modified from an article by Herman-Giddens (2013, p. 1126) about the factors associated with sexual development. She wrote, "Extensive interacting variables are known to be associated with earlier development in addition to weight and genetics: certain intrauterine conditions and exposures, preschool high-meat diets, dairy products, low fiber intake, isoflavones, high-stress families, absent fathers,

certain endocrine disruptors, the microbiome as it influences weight, epigenetics, light exposure, hormone-laced hair products, insulin resistance, activity level, geographical location, and others."

Human growth, development, and maturation may be influenced by any, and all, physical, social, economic, emotional, and political factors and forces that people experience. The timing of menarche is, perhaps, the best-studied adolescent event known to be affected by this wide range of factors (no similarly well marked and dramatic event occurs for boys). From a study of monozygotic (MZ) and dizygotic (DZ) twin girls, Tisserand-Perier (1953) and Fischbein (1977) showed that the difference in age at menarche was 2.2 months for MZ twins and 8.2 months for DZ twins. The premise of these comparisons is that the MZ twins are genetically identical while DZ twins share, on average, only half of their genes. It is worth mentioning that there are rare cases of MZ twins not genetically identical due to chromosomal abnormalities (Schmidt et al. 1976) and less rare cases of discordant gene expression due to epigenetic marking of the DNA sequence. Nevertheless, accepting the premise, it is most parsimonious to conclude that the genetic identity of the MZ twins is responsible for their much greater concordance in menarcheal age.

Other studies show that the age of menarche is also highly sensitive to nongenetic influences. Nutrition, illness, and SES are often linked together in human populations. For instance, people living in poverty in the lower-, middle-, and high-income nations almost always have higher rates of poor nutritional status, including undernutrition and/or overnutrition, higher rates of infectious diseases, and a later age at menarche compared with wealthier people. At a national level, greater urbanization, greater gross domestic product (per capita), and more diversity in food consumption indicators are related to earlier menarcheal age, but these interactions are poorly understood. In the twenty-first century the median age at menarche varies from about 11.7 to 15 years of age in the 41 nations surveyed by Janina Tutkuviene and shown in Figure 2.14. Closer analysis finds that menarche is achieved at about 12 years of age in girls of the middle-class from many nations, and at 14–15 years of age or later in girls of the lower socioeconomic classes (Eveleth & Tanner 1976; Krzyżanowska et al. 2016). The latest median age of menarche on record is 18 years of age for girls from the Bundi tribe of highland New Guinea (Malcolm 1970). Malnutrition, heavy labor, infectious disease, and living at high altitude are some of the reasons for delayed maturation of this group. Since the 1970s, the living conditions of the Bundi have improved, especially in relation to nutrition and medical care, and the median age of menarche declined by almost one year by 1990 (Worthman 1993). That is a ten-times faster rate of decline per decade than experienced by European groups since the nineteenth century.

Finally, it has long been known that physical stress and psychological factors influence growth and development, including menarche and menstruation (Choi & Yoo 2013). Highly competitive female track athletes and highly trained and competitive ballerinas, who enter training before puberty, reach menarche later than girls in the general population. One explanation for the delay in menarche of these groups of girls is the stress of exercise. Several hormones, including progesterone, prolactin,

and testosterone, are elevated by strenuous physical activity. These same hormones are known to delay the onset of menstrual cycles. Psychological stress may also be a cause of delayed menarche. Athletes and prima ballerinas are extremely sensitive about their weight and body image, and compulsive behavior in relation to these is often reported (Arcelus, Witcomb & Mitchell 2014; Turton, Goodwin & Meyer 2017). Post-menarchial ballerinas may stop menstruating before a performance, even before they begin intense training or dieting. Synchrony and suppression of menstruation occurs among women living together in a university dormitory as does menstrual migraine in women sharing a residence (Ferreira et al. 2017; McClintock 1998) suggesting that social interaction can have a strong effect on menstrual biology, though this phenomenon is not ubiquitously found.

A "collective social amenorrhea" was proposed for the extremely late age at menarche for mid-nineteenth-century Europeans. In a review of 30 German studies from 1848 to 1924, including more than 200 adolescent girls, Hermanussen and colleagues found that mean menarcheal age decreased from 18 to 12–13 years (Hermanussen et al. 2012). The data were from private medical practices whose patients were upper-middle and upper-class families. Nutritional, health, and economic factors could be excluded as reasons for the late age at menarche. Especially important is that even during World War II, adolescent girls known to be the stressed and malnourished menstruated earlier than the healthy women of the mid-nineteenth century. The authors proposed that social and psychological constraints inhibited sexual development until late adolescence when the girls were of marriageable age. Hermanussen and colleagues cite the " ... the 19th century bourgeois ... restrictive codes of sexual behavior" (p. 240). Historical studies note that these upper-class girls were often confined to home, prevented from attending schools and interacting socially with boys and, often, even other girls.

It is possible that a complex interaction between nutrition and psychology was at work. Many late nineteenth- and early twentieth-century teenage girls suffered from an illness called chlorosis, which was due, in part, to iron deficiency, but was also of psychosomatic origin (Brumberg 1982; Guggenheim 1995). Chlorosis was part of the medical literature and defined as a distinct disease between the years 1870 and 1920. An historical review of chlorosis by Joan Jacobs Brumberg (1982, p. 1468) emphasizes the biocultural nature of chlorosis by noting that during these years the disease:

> ... was widely reported in female adolescents in the United States. Diagnosis occurred on both the clinical and popular levels, yet neither the etiology nor the symptoms were precisely clear. Treatment generally included rest and large doses of iron salts. In large part, chlorosis was a cultural construction embedded in the context of Victorian medicine and family life. Physicians expected to see chlorosis in adolescent girls in the process of sexual maturation; girls learned to have the disease from family, friends, the popular press, and their doctors. Changes in diet and nutrition after 1900, coupled with increased understanding of ovarian function and iron deficiency anemia, provide only a partial explanation of the disease's eventual decline. By 1920, a changed social environment made chlorosis a social liability for girls and their mothers.

Martha McClintock, who first reported menstrual synchrony in 1971, wrote that, " ... social regulation of ovulation throughout the lifespan [is] a creature made up not only of menstrual synchrony, but various forms of the timing of spontaneous ovulatory cycles in adults. It also includes social regulation of ovulation at other points during the reproductive lifespan: puberty, inter-birth intervals and reproductive senescence" (McClintock 1998, p. 77). To date, the effects of socio-endocrinology on pubertal timing and the other reproductive phenomena listed by McClintock remain incompletely understood. Adequate studies to determine the psychological component of menarche and menstrual regularity remain to be carried out. A social-emotional perspective on the timing of menarche and other aspects of human growth, development, and maturation is taken in Chapter 7. This perspective emphasizes the community effects on growth – a new area of research.

Ego Crescere, Ergo Sum Phaenotypo

The nineteenth-century debate between the relative importance of heredity ("genes") vs. the environment in human development is largely ignored by most researchers today but is a recurring topic of debate in the popular and pseudo-scientific media. In Chapter 6 the topic of "genetics and growth" is discussed in more detail and some of the more egregious examples of genetic pseudo-science are exposed. Today, serious scientists accept that the reality of biocultural development of the human being is always due to the interaction of "extensive interacting variables." It is erroneous to consider whether one or a few variables is the most important. As James Tanner wrote, genes are inherited and, " ... everything else is developed" (Tanner 1978, p. 117). The sub-heading for this section plays on Descartes' well-known philosophical proposition about thinking and being (*Cogito, ergo sum*) to growing and becoming – "I grow, therefore I am a phenotype." The human phenotype is the outcome of a multitude of extensive interacting variables. All the measurable characteristics of the human body, of human behavior, and of the human mind are phenotypic traits. Although it is now possible to sequence and describe human genomes with precision, such knowledge by itself provides very little information about how a human being will develop without also knowing about the environment with equal precision. The discussion returns to this point again and again in the remainder of this book.

Body Proportions

More than height or fatness, the relative length and shape of body segments, such as the head, chest, and abdomen (also called the body trunk), arms, and legs have been the focus of extensive study to better understand interactions between genomes and environments. Much of the historical discourse focused on concepts of "race," a topic discussed in Chapter 1. Not mentioned in that discussion was the concern of racist biologists and anthropologists as to the degree of "humanness" of various living groups of people (Gould 1981; Marks 2015). Racist proposals about a hierarchy of from-lesser-to-greater humanness appeared as recently as 1962 with the publication

of *The Origin of Races* by Carleton Coon (Coon 1962), Professor of Anthropology at the University of Pennsylvania. Coon divided living peoples of the world into five "races" based, in part, on body size and proportions. The Australian Aborigines (designated "Australoids" by Coon), had exceptionally long legs in proportion to stature, and African pygmies ("Congoids" in Coon's taxonomy), had exceptionally short stature, long arms relative to leg length, and especially short lower legs. In Coon's words, "Their manner of dwarfing verges in the achondroplastic . . . " (p. 653). Moreover, Coon asserted that both "races" crossed the species threshold between *Homo erectus* and *H. sapiens* only in the last 10,000–50,000 years. In contrast, Coon proposed that ancient Europeans (called "Caucasoids" by Coon), had crossed the *H. sapiens* threshold about 200,000 years ago. According to Coon, the ancient Europeans were "normal" in size and shape, able to " . . . sit in any western European restaurant without arousing particular comment except for their table manners" (1962, p. 582).

These claims of race-based human taxonomy, including Coon's time thresholds for "homo-sapienation," have been discredited by paleontological and genomic research. All living humans share the same antiquity and origins within Africa, as was discussed in Chapter 4. Coon's claim that African pygmies have "achondroplastic proportions" was also wrong. Shea and Bailey (1996) showed that African pygmies are reduced in overall size, but have a body shape that is proportional to the size reduction.

Discarding the racist history of the study of human morphology allowed research to focus on more meaningful biological, medical, social, and aesthetic implications of human body size and shape. In this section evidence is presented that human body shape, especially the length of the legs relative to total stature, is an important indicator for epidemiology and environmental public health. A discussion of how to measure leg length and how to define relative leg length is given in Box 5.2. Across the human species, as well as within geographic, social, and ethnic groups of people, relative leg length reflects nutritional status and health during the years of physical growth, and also has biologically and statistically significant associations with risks for morbidity and mortality in adulthood.

Box 5.2 **Measuring leg length and defining relative leg length**

A strict anatomical definition of leg length (LL) is the length of the femur + tibia. Due to the bipedal nature of the human species, "leg length" often is measured as: (femur + tibia + the height of the foot, from the tibia-talus articulation to the ground). Alternatively, the phrase "lower limb length" may be used to denote this linear dimension. In this book "leg length" may denote several possible measurements such as iliac height (IH) and subischial leg length (SLL, Figure B5.2.1). This is because in a living human being it is difficult to measure anatomical LL. The maximum length of the femur is measured from its head, at the proximal end, to its medial condyle, at the

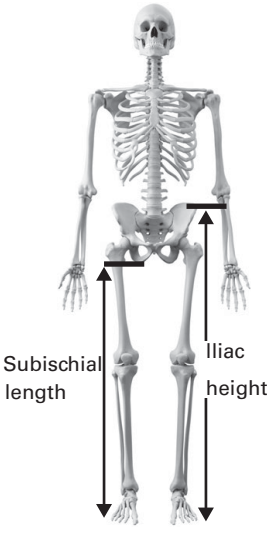


Figure B5.2.1 Iliac height and subischial length. Credit SCIEPRO / Science Photo Library / Getty Images.

distal end. In life, the femur and pelvic bones overlap, and the head of the femur is difficult to assess due to its articulation within the acetabulum. A high degree of body fatness may make these bony landmarks difficult, or, impossible, to access. Consequently, LL is often defined by the easier to measure IH or SLL. It is also possible to measure an estimate LL via the combination of thigh length (TL) and knee height (KH). Some studies employ only one of these measures as the indicator of LL.

Each of these measurements can be transformed in ratios, generally in relation to total stature and sitting height (SH) to give indications of body proportions. Perhaps the most common is the sitting height ratio (SHR= [sitting height / height] $\times$ 100) and this is used here. The SHR is preferred to stature minus sitting height because it standardizes for total height making it possible to compare individuals with different statures. This provides a relative leg length. Practical methods and techniques required to obtain various measures of leg length may be found in the literature (Bogin & Varela-Silva 2010; Lohman et al. 1991; NHANES Anthropometric Manual[4]).

Size and Shape in Living Humans

As discussed in Chapter 4, the human species is distinguished from the nonhuman primates by proportions of the arms and legs relative to total body length. The human difference was illustrated in Figure 4.1. Human body proportions develop from fetal

[4] www.cdc.gov/nchs/data/nhanes/nhanes3/cdrom/NCHS/MANUALS/ANTHRO.PDF

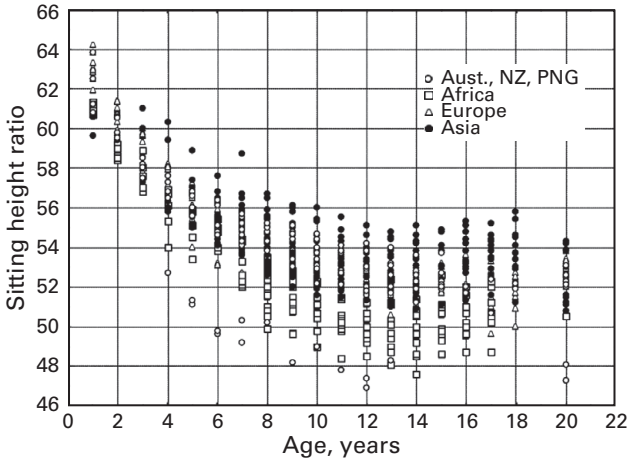


Figure 5.7 Sitting height ratio by age for the four geographic groups defined by Eveleth and Tanner (1976, 1990). Age 20 includes data for adults over the age of 18 years. A larger SHR indicates relatively shorter legs for total stature (author's original figure).

to adult stages of growth, as was illustrated in Figure 2.3. The general pattern of human body shape development is a species-specific characteristic. Historical artwork, sculpture, and anatomical drawings from Renaissance Europe (Boyd 1980; Tanner 1981) and pre-Columbian Mexico (Tate & Bendersky 1999) show fundamental commonalities in the depiction of body shape of late term fetuses, newborns, and infants. Discrete populations of living humans, however, present a diversity of body sizes and shapes. Mean stature for populations of adults varies from minimum values for the Efe Pygmies of Africa at 144.9 cm for men and 136.1 cm for women to the maximum values for the Dutch of Europe at 184 cm for men and 170.6 cm for women. There are also biologically and statistically significant variations between human populations in body shape. Eveleth and Tanner (1976, 1990) published data for height, body proportions, and leg length, estimated via the SHR, from dozens of human populations, distributed across most geographic regions of the world (Figure 5.7). Mean SHR for populations of adults varies from minimum values, i.e., relatively longest legs, for Australian Aborigines (SHR = 47.3 for men and 48.1 for women) to the maximum SHR values, i.e., relatively shortest legs, for Guatemala Maya men and Peruvian women (SHR = 54.6 and 55.8).

Making sense of these worldwide comparisons is difficult because of the differences in lifestyle, environment, and genomics. Two well-known ecogeographic principles, Bergmann's and Allen's Rules, are often cited as primary causes for the global patterns of human body shape variation. Bergmann, in 1847, observed that closely related mammalian species, such as bears, have greater body mass in colder climates. Allen added in 1877 that the limbs and tails of such species tend to be shorter in cold climates and longer in warmer environments. The "rules" of Bergmann and Allen are examples of thermoregulation in relation to body shape. Large body mass and relatively short extremities increase the ratio of volume-to-surface

area and provide for a body shape that maximizes metabolic heat retention in a mammal. Conversely, in warmer temperatures, relative long extremities increase surface areas relative to volume and allows for greater heat loss. It has been shown experimentally that mice and other nonhuman mammals raised in warmer temperature experience greater bone tissue growth and longer limb bones (Serrat et al. 2008). The usual explanation for this is greater vascularization, allowing for greater oxygen and nutrient perfusion. Serrat and colleagues' experimental research showed, however, that even in the absence of vasculature, in vitro culture of chondrocytes from mouse metatarsal bone show a positive correlation between environmental temperature with, " ... greater proliferation and extracellular matrix volume ... " (2008, p. 19348).

Bergmann's and Allen's rules have been applied to the human species. In 1953, Derek Roberts (1925–2016) published an analysis showing a significant relationship between body mass and latitude for human beings, with groups of people living at higher latitudes having greater body mass than those living closer to the equator (Roberts 1953). Twenty-five years later, Roberts et al. (1978) updated and reaffirmed these findings. Other research shows that people living in colder regions also tend to have shorter limbs relative to total stature, compared with groups of people living in warmer regions (Ruff 2002).

These climate relationships, however, are only correlations, with values in the range of r = -0.5 to -0.6. Correlations cannot prove causality or, even, the direction of association. In statistics the square of the correlation value is often taken as an indication of the percentage of explained variance. Doing so means that climate "explains," at maximum, about 36% of variation in human body mass. Other factors explain the other 64% of the variance.

A reanalysis of the Roberts data by Katzmarzyk and Leonard (1998) modified the importance of climate as the primary molder of human body shape. Katzmarzyk and Leonard analyzed the sitting height ratio of 165 human groups studied between 1960 and 1996. The human data analyzed by Roberts were collected prior to 1953. Katzmarzyk and Leonard showed that the more recently studied groups still follow the ecological principles of body shape, but that the association with climate has been attenuated since Roberts's study. The slopes of the best fitting linear regression lines for the relation of mean annual temperature to sitting height ratio are half those reported by Roberts, that is the correlation between body and mean annual temperature declined to r = -0.3 or less in both sexes. Statistically, the variance explained has dropped to only 9%, which is not a biologically meaningful percentage. Katzmarzyk and Leonard pointed out that changes in nutrition and health are the likely reasons for the reduced correlation between body shape and temperature. The authors defined the nutritional changes as modifications in diet and lifestyle, especially the introduction of western foods and reduced physical activity. They also pointed out that " ... climate may shape morphology through its influence on food availability and nutrition [meaning that] linear builds of tropical populations are the consequence of nutritional [factors] rather than thermal stress ... " (pp. 491–492). In this case, during the years of growth and development the intake of more or less total food, more or

less of any essential nutrient, more or less physical activity (and the type of activity) could influence body shape.

The influence of changes in the social-economic-political-emotional (SEPE) environment on the correlations of body size and shape with climate were the subject of two presentations, one by Michael Hermanussen and another by Christiane Scheffler, at the 2017 Society for the Study of Human Biology Symposium. These researchers showed that body size, shape, and composition are modified relatively quickly in response to change in the SEPE climate. Moreover, these modifications are more due to the SEPE climate than they are responses to the temperature climate. A synopsis of the presentation by Hermanussen and Scheffler is given in Box 5.3.

Box 5.3 **No evidence of Bergmann's rule in human body size: a reappraisal of pre-Darwinian biology between creation and evolution.**

By Professor Dr Michael Hermanussen, Aschauhof 3, 24340 Aschauhof, Altenhof, Germany and Privatdozent Dr Christiane Scheffler, Institute of Biochemistry and Biology, Human Biology, University of Potsdam, Potsdam, Germany.

Carl Georg Lucas Christian Bergmann was a German anatomist and physiologist born May 18th 1814, in Göttingen. He died April 30th 1865, in Geneva. He was the son of the lawyer and professor Friedrich Christian Bergmann (1785–1845). After graduation in 1832 in Holzminden he studied medicine and natural sciences at the universities of Göttingen and Würzburg. He worked as Privatdozent of medicine. In 1843 he was appointed associate professor in Göttingen. In 1846 he accompanied Sartorius von Waltershausen and Robert Bunsen on a research trip to Iceland. In 1847 he published his most famous work *On the Proportions of Heat Economy of Animals to their Size* where he discussed the relationship between heat balance and body size that has later been named Bergmann's rule. Bergmann's book appeared 12 years before Darwin's *On the Origin of Species* went on sale in November 1859. Bergmann's considerations are pre-evolutionary, and partially include creationistic ideas.

Bergmann knew about the work of Justus von Liebig, who systematically investigated the chemical foundation of the life processes, classified the constituents of foodstuffs and tried to explain nutrition, metabolism, heat generation, and respiratory gas exchanges in chemical terms. Bergmann was aware that the heat production of warm-blooded animals (*calor animalis*) is limited by their volume, and that all losses of heat depend on characteristics of the animal's surfaces, temperature difference between skin and air, and heat conduction. He concluded that the texture of the surface, and the proportion of surface and volume of an animal are subject to the laws of physics. He ranked animal surfaces according to heat conductivity, with skin-to-water being most thermoconductive to skin-to-air and fur/plumage-to-air as least thermoconductive. He then ranked the animals known to him, from marine

mammals and water-loving pachyderm, other terrestrial animals, and finally birds to determine the largest and the smallest possible sizes for species within God's creation. Based on his considerations Bergmann concluded that " ... there must be a not crossable limit of smallness for endotherm (warm-blooded) animals caused by the proportion that the (heat producing) volume when decreasing in size, will decrease to a greater extent than its (heat loosing) surface." Bergmann believed that hummingbirds are close to this lowest possible limit for size in endotherms. He then asked " ... whether in all places extremes in size have been reached or not ... " and looked for the "largest and smallest homeotherm creatures." He realized " ... that in the temperate and cold regions, extreme sizes even though possible according to this law, have not been reached ... " and he concluded that " ... nature did not fully complete its limits which would have been offered according to this law ... The largest animals of the cooler zones have for whatever reasons not been created." Almost modern in his thinking, he further stated that it " ... is not understandable, and rather looks like random particularly when remembering the large distribution of elephant-like and other very large animals *of earlier creations*" [emphasis added to note the pre-evolutionary thinking]. He formulated his hypothesis that " ... if we could find two animal species which only differed in size ... the geographic distribution of these two species should be relatively determined by their size; which absolutely taken would be their homeland, the smaller one should ask for a warmer, the larger one for a colder climate." Bergmann found support for his hypothesis by comparing body size and wingspan of various birds known to him, of different species but the same family, and showed that in many species, the ratio volume-to-surface is associated with their geographic distribution. Using an approach that may be considered statistical, he found that a majority of species are subject to what is known today as Bergmann's Rule.

Bergmann did not discuss human size. This was done some 100 years later by Roberts (1953) who was interested in the heat production of people according to the climate of their habitats. In his famous compilation of studies, he synthesized data from different ethnic groups living at mean annual temperatures between 10° and 80° Fahrenheit and with adult men weighing between 40 and 77 kg. He found a negative relationship between body weight and mean environmental temperature with $r = -0.59$.

During the years between Bergmann's work and Roberts's compilation, average body height of populations of many nations, and particularly of the industrialized countries, significantly increased. In a meticulous collection of global height changes between the mid-nineteenth and the mid-twentieth century, Kenntner (1963) showed that the European populations had on average increased in height by 8–10 cm, and The Netherlands by 15 cm, since 1850. Similar increases in adult height had not been observed in the tropical European colonies. A recent analysis on a century of trends in adult human height

confirmed that little gain in average height in the countries of sub-Saharan Africa and South Asia at the time of Roberts's compilation (NCD Risk Factor Collaboration (NCD-RisC), 2016). The body height gap between countries of the northern hemisphere and their tropical colonies was considerable. This changed in recent decades as adult body height significantly increased since the independence of many former European colonies.

In 2010, Leonard and Katzmarzyk published more recent data on the relationship between body weight and mean annual temperature of their habitats. In a further update, Leonard summarized that " ... the correlation between mass and temperature was much lower in later samples, and the slope of the regression was significantly shallower than that reported by Roberts" (Leonard 2018, p. 816). He concluded that, " ... these differences partly reflect secular changes in growth and body size, and the development of improved technology that moderates extreme temperature exposure ... [and that] ... these findings underscore the importance of both nutritional and temperature stresses in shaping human variation in body size and shape" (p. 816). These statements are certainly intuitive, but are symptomatic of human thinking regarding traditional knowledge, of which Bergmann's rule is a conspicuous example. It is not Bergmann's Rule itself that is questioned, rather the weakening of the association between size and temperature is ascribed to changes in those factors which, according to the Rule, are believed to influence body height.

Bergmann's Rule has been popular for more than 170 years and belongs to the list of what German-speaking people call "Lieschen Müller[5] knowledge." This type of knowledge refers to common concepts of understanding the world that are transmitted early in life, often already at or even before school age. These concepts are beyond doubt and largely independent of later academic achievements. They are based on prevalent cultural perception. Bergmann's Rule is in this category of knowledge because it is included in almost every introductory textbook and university course related to animal physiology and ecology. Bergmann's Rule, and its corollary Allen's Rule relating to limb proportions, are fundamental concepts taught in undergraduate and post-graduate anthropology and human biology. The belief in the fundamental truth that Bergmann's Rule applies to humans is based on the accidental association of height, weight, and average temperature variation by latitude that existed at the time of Roberts's analysis. Before and after that time, that is before 1953, the situation was different, and Bergmann's Rule did not apply.

Roberts was, in a sense, lucky when he did his 1953 analysis because he was really describing the effect of **secular trends** for increased height in the rich northern nations vs. the stagnation or decline in height of people in the

[5] *Lieschen Müller is a placeholder for the average person in the German-speaking world. The average is often also referred to as "Lieschen Müller." German parents tell their children Lieschen Müller stories to explain how the world works.*

colonial southern nations. In 1953 this difference in average adult stature was at its maximum. The height difference had everything to do with differences in the standard of living and the quality of the SEPE environment. The differences had nothing to do with temperature or climate.

Neither can short stature be considered a synonym of inadequate nutrition; stunting is not a synonym of malnutrition (Scheffler et al. 2019). Nor is the increase in height of recent Europeans explicable by indoor domestic temperature. Quite in contrast, indoor temperature has significantly increased in the last decades, and thus, according to Bergmann's Rule, should have rather given rise to a decline instead of a further elevation of body size in the northern Europeans (Mavrogianni et al. 2013).

Bergmann assessed God's creation in a time before Darwinian evolutionary biology. Bergmann considered species according to heat production and loss of temperature due to size and surface thermoconductivity. He particularly considered the extremes as he believed they represented the upper and lower possible limits for size in endotherms. Roberts and Bainbridge (1963) reported average height of 182.6 cm for men in a Southern Sudanese sample of 52 Dinka-Agaar and 181.3 cm in 227 Dinka-Ruweng measured in 1953–1954. In view of these measures which may be considered close to the upper limit for height for equatorial populations, Bergmann would have postulated numerous taller groups living in the circumpolar regions. Yet, these groups never existed. Quite in contrast, Andersen and coworkers (2004) published median height of 164 cm for 20–29-year-old Inuit men, and of 153 cm for 20–29-year-old Inuit women. Similar heights were reported from Alaskan Eskimos, with mean height of women and men, respectively, 153.4 and 165.2 cm (Risica et al. 2000).

Living under extreme geographic circumstances, such as cold, heat, or altitude, exerts evolutionary pressure that those who live in other regions do not experience. In his 2018 review article, Leonard summarized several mutations being associated with enhanced fatty acid oxidation that are prevalent in northern populations. Yet, size is a complex trait. Bergmann himself realized that by far not all northern species are larger than their southern counterparts.

In social mammals, the regulation of growth does not follow some simple genetic program, but strongly includes social aspects (Clutton-Brock 2016). In humans, growth and final height appear to have affiliations to peer groups, social networks, and dominance within the group (Huchard et al. 2016; Hermanussen & Scheffler 2016). These and other contributing factors have given rise to the exuberant increase in height within a few generations in the contemporary industrial world. The shortness in height currently observed in the low- and middle-income countries may indicate their delay in modernization when compared to the modern Western world, but it must not be mistaken as an example of Bergmann's Rule. There is no evidence that Bergmann's rule applies for present-day humans.

Scheffler and colleagues' presentation was titled "Change of political climate and the plasticity of the human body." They used the phrase "political climate" as an analogy to the meteorological climate and described the political weather of the last 30 years in Germany. This was a period of notable modification of anthropometric parameters during the break-up of the former Soviet Union and the transition of the political, socioeconomic, and environmental circumstances. The authors placed particular emphasis on the economic and political situation in the former German Democratic Republic (GDR, i.e., East Germany) during the early 1990s. At the time of reunification in 1989 the body height of military conscripts of East Germany was significantly shorter than that of conscripts in West Germany, but the difference nearly disappeared within four-five years. Pelvic and elbow breadth of all conscripts decreased at the beginning of 2000, and the fat distribution pattern of conscripted men became more "feminine" after 1997, that is relatively more fat on hips and lower extremities (Scheffler & Dammhahn 2017). Scheffler and colleagues explained the increase of height as due, in part, to the community effect following the political reorganization of the former East Germany. The feminization of fat distribution pattern occurred in parallel with an increase of endocrine disrupting chemicals that were introduced along with other Western consumer products after the collapse of GDR industry. The decrease of skeleton breadth measurements was associated with a decrease in daily physical activity.

Two additional examples add support to the interpretation that population variation in body shape is primarily related to nutritional and SEPE influences. The first is based on studies conducted since the 1960s with Guatemala Maya people. It was reported that the rural Maya consumed only approximately 80% of the total energy needed for healthy growth, and 20.4% were also iodine deficient (Bogin & Rios 2003). Iodine deficiency during infancy and childhood results in reduced leg length growth, especially at the epiphyses of the distal femur, the tibia, and the foot. Maya children and adult participants in those studies spent considerable time and energy at heavy labor, which diverted available energy in the diet away from growth. This nutrition and lifestyle combination is known to reduce total stature and leg length. The second example comes from similar findings reported for native Peruvian highland children of the Andes Mountains by Emma Pomeroy and colleagues (2012). Pomeroy's research group reported trade-offs in relative limb length. Those children exposed to greater nutritional deficiencies and work had significantly shorter limbs, hands, and feet compared with less stressed lowland children. The more stressed participants also lived at high altitude and suffered more cold stress. The cold likely diverted further energy away from growth and toward basic body maintenance. Differences between the groups in head–trunk length were smaller. The ulna and tibia bones were the most sensitive to stressful environmental conditions. All this points to the body size and shape of the Maya and native Peruvians being an unhealthy response to stress and not an adaptation to climate.

The body shape of some groups of people may have a genetic basis, especially for human groups who resided in the past in relative social isolation in far-flung geographic regions for many generations. A comparison of stature and body

proportion between blacks (African-Americans) and whites (European-Americans) in the United States provides an example of genome-environment interactions and their effect on growth. Open access data are available from the First National Health and Nutrition Examination Survey (NHANES I) of the United States, which gathered anthropometric data on a nationally representative sample of blacks and whites aged 18–74 years for the years 1971–1974. When the data are adjusted for differences between the two ethnic groups in income, education, urban or rural residence, and age, there is no significant difference in average height between black and white men. Nor is there a significant difference in average height between black and white women. These statistical adjustments are necessary because of the history of racism in the United States which relegates blacks, on average, to lower SES than whites. Although white and black adults in the United States have the same average stature, when education, income and other variables are controlled, the body proportions of the two groups are different. Blacks, on average, have shorter trunks and longer extremities than whites, especially the lower leg and forearm. This is found for black and white youths 12–17 years old, and for adults 20—49 years old. The same body proportion differences were found for the NHANES III survey of 1988–1994 (Bogin & Varela-Silva 2008).

A genomic contribution to the body proportion differences between blacks and whites may seem likely, as the blacks tend to have more sub-Sahara African genomic origins than the whites. In a statistical pedigree analysis of two human samples, Livshits and colleagues (2002) estimated that between 40% and 75% of inter-individual variation in the body proportions they studied (adjusted for age and sex) are attributable to "genetic effects." But ascribing these effects to genes is highly questionable. These are better described as familial effects because the authors analyzed families and because they found significant common environmental effects for siblings. The researchers also reported significant sex-by-age interactions. This range of sources of variation in the analysis makes it difficult to compute simple genetic variance.

Even if specific genotypes are discovered, their direct contribution to normal geographic origin (so-called "racial") variation in human body shape may be relatively small. At 40 weeks gestation, fetuses identified as African-Americans have, on average, relatively longer legs than fetuses identified as European-Americans (Schultz 1926). But the difference, as measured by (total length/crown–rump length) is less than 1%. In an analysis of the data shown in Figure 5.7, Bogin and colleagues (2001) estimated the contribution of geographic origin to the variance in the sitting height ratio to be 0.04, which accords well with genomic estimates for variation in total stature of 0.04–0.06. Forensic anthropologists and physicians in the United States have often used "race-specific" body proportions, to ascribe an African-American or European/Asian-American ethnicity to a skeleton. Feldesman and Fountain (1996) tested the utility of the femur length/stature ratio to correctly identify 798 femur/stature pairs of skeletons of known ethnicity. Using the statistical methods of discriminant function and cluster analysis they found that coherence to groups defined by geographic origin, meaning blacks or whites, was poor, with

results barely better than chance. Using "race-specific" body proportions to identify unknown skeletons would result in a high number of incorrect attributions of ethnicity.

A more promising approach to understanding the control of human body proportions comes from genomic research on *Hox* genes and homeobox sequences described in previous chapters. It is well known that these genomic elements and their related cell signaling factors, which regulate the growth of body segments, are shared across many taxa of organisms (Mark et al. 1997). There is observational and experimental evidence that *Hoxd* expression is linked with forearm, hand, and digit length differences in the apes (Reno et al. 2008). The short stature homeobox-containing gene (SHOX), mentioned in Chapter 1, is another genomic region that may be relevant to human body proportions. Werner Blum and colleagues reported that "SHOX, located on the distal ends of the X and Y chromosomes, encodes a homeodomain transcription factor responsible for a significant proportion of long-bone growth" (Blum et al. 2007, p. 219). In addition to other growth pathologies listed in Chapter 1 associated with SHOX, Turner syndrome (45, XO **karyotype**) results in approximately 20 cm deficit in stature. Some studies find that legs are disproportionately affected, but other studies find no disproportion (Ogata et al. 2002). Other candidate genomic regions for body shape are known from some nonhuman mammals and in insects (Bogin & Varela-Silva 2010), but these are not connected clearly with human growth.

Another very active area of research is epigenetic regulation of body growth. Epigenome effects may act through several genomic (e.g., DNA methylation and histone modification), proteome (e.g., micro-RNA regulation of gene expression), and environment (e.g., climate, diet, and physical activity) interactions and may well play the major role in determination of human size and shape. More details about the epigenetics of growth are given in Chapter 6.

Secular Trends

. . . "secular trend." This rather curious phrase denotes both the tendency to get larger and the tendency to become more early-maturing, tendencies which are usually, though not invariably, linked. (Tanner 1981, p. 116).

In *A History of the Study of Human Growth* Tanner devotes only 4 of 402 pages to the topic of secular trends. This is as curious as the phrase itself, considering that the description and analysis of secular growth is one of the most frequent topics of human growth publication. Few of those publications explain the origin of this "curious phrase" as applied in human biology. So, a few words on etymology and usage are in order. One dictionary definition of "secular" is just once in an age, indicating a relatively long span of time. A search of PubMed found such a usage of "secular" in a physical scientific context in 1880, in an anonymous article in *Science* about geology. A few years later astronomers use the term. Geology and astronomy work on cosmic time scales. The other dictionary meaning of secular is "worldly,"

pertaining to the material, nonspiritual world. As used today in human biology, these two definitions are apt because the factors influencing the secular changes in growth are related to the material conditions of life, and these conditions do act on human growth over long spans of time.

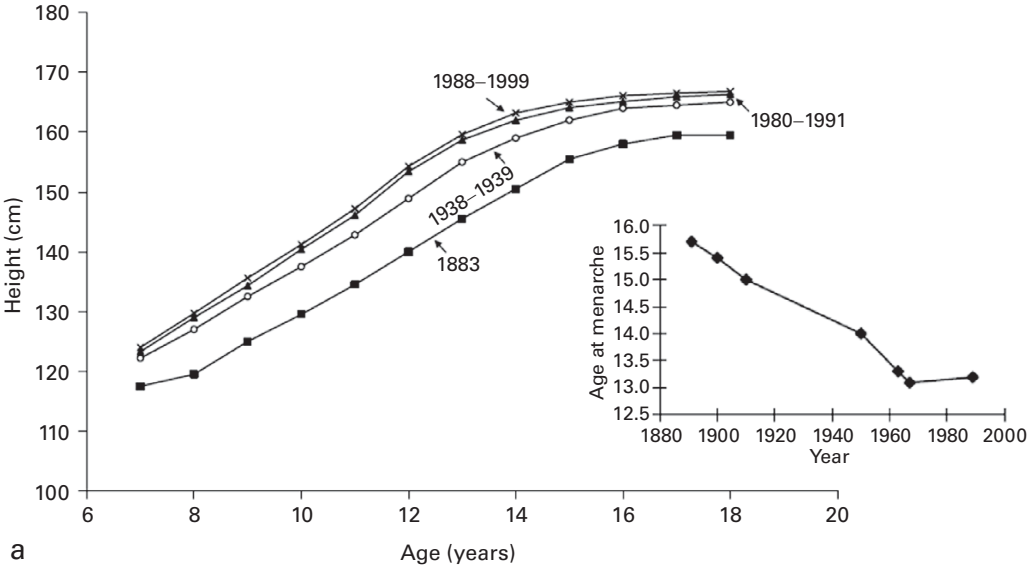
The first paper that clearly defines how to use the term "secular" seems to be an economics report by Burns, in 1934, who wrote "The secular trend of an industry's production may be considered as the persistent, underlying movement of its output over a period which is 'long' in relation to the changes associated with the 'business cycle'. So viewed, the secular trend is irreversible within the periods of a business cycle, though it may be reversible within longer periods. The secular trend can be represented graphically by a curve and may be given algebraic expression by a mathematical equation ... Secular trends of production express the growth (including decadence under this term) of industries" (Burns 1934, p. 31–34).

As may be seen from Figure 5.8, with few changes this quote applies to the study of secular changes in human growth. Indeed, economic growth and human growth are correlated, and both economists and human biologists study these interrelationships. Changes in growth over decades and centuries usually lag behind improvements or declines in economic productivity (Floud et al. 1990; Komlos, 2009; Steckel 2012).

The earliest publication on secular changes in human growth seems to be by Quetelet (1832). He explained urban/rural and European national differences in average height as "secular," meaning the differences were determined strictly in terms of environmental and economic factors. Other nineteenth- and early twentieth-century publications on human size and shape seemed not to use the word "secular." This is despite the fact that by the 1930s human biologists had published many articles on the topic of changes in growth between generations and between immigrant parents in the United States and their offspring, but there is no use of the word "secular."

The next use of the term in relation to human growth seems to be by Lawrence Frank (1935), where the phrase "secular trend" referred to changes in size and biology during the growth of the individual rather than its current usage as a change in growth of groups of people over time or generations. In the same year, a German medical doctor, Walter Koch, referred to trends in the "Akzeleration" of growth, meaning that children, juveniles, and adolescents were growing up faster (Koch, 1935). A few years later it was realized that not only the tempo of growth was accelerated but also final adult height was greater than in previous generations. Howard Meredith (1941) used the term "secular changes" and **secular trends** in a study of growth of two groups of people measured a decade apart. Meredith provided a good review of the topic and concluded by writing that " ... no one really knows the cause [of secular changes] – it is a research frontier" (p. 37). Today, the causes are better understood and are discussed following an overview of the changes themselves.

In the remainder of this discussion the phrase "secular change" refers only to increases or decreases in the mean anthropometric values of human populations



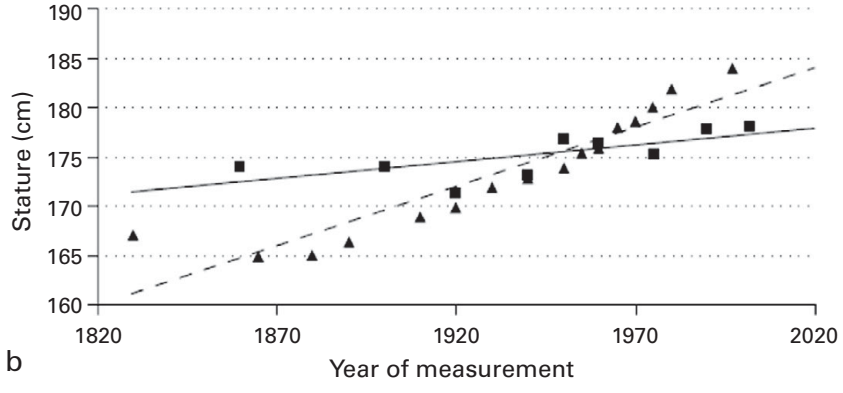


Figure 5.8 (a) Secular changes in the height of four cohorts of Swedish females between 1883 and 1999. During this period, average height at age 14 increased by almost 13 cm (5 inches), and final height at age 19 increased by about 8 cm (3.2 inches). Increases in the height of Swedish males during the same time were even greater, almost 20 cm (7.8 inches) at age 14 and 16 cm (6.3 inches) at adulthood. The magnitude of the positive secular trend in Sweden is similar to that in other industrialized countries (e.g., in part (b)). The inset shows that as height has increased, the age at first menstruation has decreased until about 1970 and then has stabilized.

(b) Secular trend in average stature (mean or median) of men 20–39 years of age in the Netherlands (▲) and the United States (■) between 1830 and 2002. The data points are fitted with linear regression lines (author's original figure).

between generations. The focus is on skeletal dimensions, especially stature, because body mass (fatness, muscularity) is labile to short-term (i.e., within generation) influences. Roland Hauspie (1948–2017) and colleagues (Hauspie et al. 1997) reviewed the evidence for these secular changes over the past century in 17 nations, including many European countries as well as Japan, Cuba, Brazil, North America, and Taiwan. They found that on average the per-decade increase in adult height was ~1 cm. The greatest per decade increase occurred in Japan, ~4 cm from 1950 to 1960, and the smallest increase occurred in Sweden and Norway at ~0.3 cm/decade between 1952 and 1985. In these diverse populations, it seems that most of the final adult increase in stature was achieved during childhood. The average adult increase was ~1.9 cm, with ~1.3 cm achieved by the end of childhood and only ~0.6 cm more achieved during adolescence. As indicated in Figure 5.8 part a, 8-year-old girls in 1999 averaged nearly 130 cm, a stature achieved only at 11 years of age in the nineteenth century. Over the same century the age at menarche declined by nearly three years.

Not all secular change is positive. In fact, an especially useful approach to understand the reasons for secular trends was derived from the discovery and analysis of **negative secular trends**, that is, examples of decreases in body size or delays in the rate of skeletal and sexual maturation from generation to generation. The landmark publication in this area was by Phillip Tobias (1985) in which he presented a worldwide review of growth data for stature from the twentieth century for succeeding generations of people living under deteriorating conditions of low SES and/or under political repression. For each of the cases Tobias examined he found either no secular change in stature or negative secular change. His own data from South Africa demonstrated a clear decline in mean stature for blacks from the late nineteenth century to the present day. The stature declines were linked to the deterioration of the social, economic, and political environment for blacks both prior to and during the apartheid era in South Africa.

The purpose of the apartheid policies was to guarantee economic, social, and political domination of the country by the white minority. Apartheid laws existed in South Africa from 1948 until the early 1990s, but discriminatory practices and policies were common since the nineteenth century. Apartheid institutionalized and legalized racial segregation, economic exploitation, and exclusion from medical, educational, and other essential social services of African blacks and other people of color. The dream of apartheid for whites was a paradise of boundless prosperity, health, and happiness. South African white children would live and grow up under socioeconomic conditions equal to, or superior to, those of the industrialized nations of Europe and North America.

Apartheid brought only misery. People of all social groups, including whites, suffered. Between the years 1880 and 1970, South African white men and women averaged an increase in mean height of 4.5 mm/decade and South African black men had a mean increase of 2.4 mm/decade (Henneberg & van den Berg 1990). White South Africans are predominantly of Dutch ancestry and quite unexpectedly the increase in stature of the South African whites was significantly less than that for the

Dutch living in the Netherlands – 15 mm/decade – and measured in the same years. The three-fold greater secular increase in stature of the Dutch in the Netherlands shows that the apartheid policies were more nightmare than dream. The most likely explanation for the superior secular increase in stature of the Dutch in the Netherlands as opposed to the Dutch in South Africa is that the deterioration of living conditions for the nonwhite population in South Africa caused by the apartheid policies could not be confined to those ethnic groups. Of the almost 39 million citizens of South Africa in 1991, 75.2% of the population were classified as Black Africans, 8.6% were known as Coloureds, 2.6% were Asians, and 13.6% were Whites. When more than 85% of a population lives under repression and poverty, the economic and social development of the entire country is likely to be arrested, and even the privileged social classes will be affected.

The most meaningful statistic in this regard is the infant mortality rate (IMR), which is, perhaps, the best indicator of the overall health environment for any population. In 1980, the estimated IMR was 12/1,000 for whites and 120/1,000 for blacks (Chimere-dan 1992). By comparison, the IMR in 1987 for the Netherlands, for all ethnic groups, was only 7/1,000, a statistically and biologically significant difference from white South Africans. Life for the black majority was horrendous and for South African Dutch it was more precarious than in their homeland.

Another South Africa study compared a secular trend in skeletal maturity of blacks and whites from the 1962 Pretoria National Nutrition Survey with cross-sectional data from 2001 derived from the Johannesburg-Soweto based Birth-to-Twenty birth cohort study. Girls and boys aged 9–11 years from each survey were included in the analysis (Hawley et al. 2009). The authors reported that skeletal maturity of white girls and boys in 2001 was slightly and not significantly advanced over the 1962 cohort by an average of 2 months and 3.4 months respectively. Black girls and boys in 2001 showed significant advancement over the 1962 cohort; on average 15.8 months and 9.7 months and 15.8 months, respectively. The authors wrote that "The increase in skeletal maturity may reflect the removal of growth constraint, particularly in black children" (p. 584). The obvious growth constraint was apartheid.

An even clearer case of the negative trends in height comes from Guatemala during the period from 1974 to 1983, a time of intense civil war and political repression (Bogin & Keep 1999). Working with Ryan Keep, who was then an undergraduate student of economics, we found economic decline and political unrest due to the war was associated with a significant decline in the mean stature of cross-sectional samples of 10- and 11-year-old boys and girls from families of very high, moderate, and very low SES. In order to establish a more direct link between the trends in growth and socioeconomic indicators, we looked more closely at the pattern of change in per capita gross national product (GNP, the value of all finished goods and services produced in a country in one year by its nationals) and stature (Figure 5.9). For this analysis we calculated a moving average of per capita GNP. The moving average smooths the year-to-year fluctuations in the data and was calculated for the percent change in the per capita GNP of Guatemala from year-to-year expressed as the average of the previous 10 years. For example, the per capita

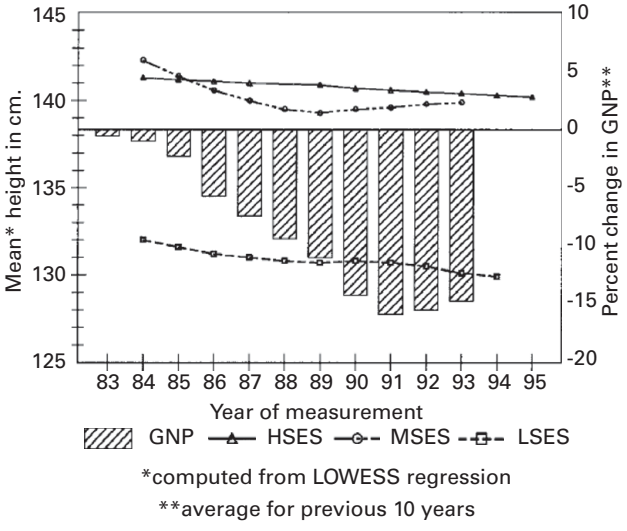


Figure 5.9 Mean height of Guatemalan 10- and 11-year children of high, middle, and low SES in relation to the 10 year moving average for percent change in GNP per capita in Guatemala (author's original figure).

GNP value for 1983 is the mean of the annual percent change for the years 1974 to 1983. Also charted are the mean heights of the children of the high, middle, and low SES groups, as estimated by LOWESS regression, another statistical smoothing method, in the year they were 10 or 11 years old.

Negative trends in stature and in percent change of per capita GNP are evident in Figure 5.9. The downward trends are statistically and biologically significant for all three SES groups. The decline in stature was likely due to biological and psychological factors, especially declines in the quality of nutrition and health and increases in social and economic insecurity of the entire Guatemalan population. The Pan-American Health Organization (PAHO) country profile for Guatemala for the period 1988–1992 reported the following information:

> the two leading causes of death were respiratory infections and intestinal infections . . . The death rate from measles rose during the years 1989–1990, when a major epidemic occurred . . . The number of malaria cases totaled 41,711 in 1990, 57,829 in 1991, and 57,560 in 1992 . . . Nutritional assessments of children under 5 reveal a deterioration in their nutritional status, with an alarming rise in acute malnutrition (weight-for-height). In 1979 the prevalence of endemic goiter was 8%. By 1989 it had increased to 20.4% as a consequence of notable deterioration in the salt iodization program.[6]

By 1990 Guatemala was economically bankrupt and the country was literally falling apart.

[6] www.paho.org

As was the case of South Africa, even the very wealthy in Guatemala were not spared from the decline in public health which led to neglect of municipal water supply systems and an outbreak of cholera and other epidemic diseases. The rich in all nations depend on the services provided by the poor, including work as housekeepers, cooks, and gardeners in their homes and caretakers of their children. In Guatemala, infectious disease of these low-paid workers spread to the families of their wealthy employers. The rich were also plagued by a sharp rise in kidnappings of children and adults for ransom by criminal gangs. These physical and emotional threats are known to increase the production of biological stress hormones, such as cortisol, which are antagonists to growth promoting hormones (Blum et al. 2011; Bogin et al. 2015). Together, increases in nutritional deficiencies, illness, and emotional stress resulted in negative trends in height growth.

Wealthy nations are no exception to negative secular trends. John Komlos (2010) analyzed height trends in the United States since World War II for adults aged 20 years or older. The data came from nationally representative surveys from the years 1976–1980, 1988–1994, and 1999–2006. All the participants were US-born individuals. The data were analyzed separately by sex (women and men) and ethnicity (black and white) and adjusted for family income. Adult white men were at all times taller, on average, than black men and that difference increased from 0.4 cm for men born 1940–1949 (mean heights, white = 177.1, black = 176.7) to 1.1 cm for men born 1975–1986 (mean height, white men average 179.1 cm, black men = 178 cm). The mean height of white women reached a peak of 164.8 cm for the birth cohort 1955–1959 and remained more or less the same to the birth cohort 1980–1986. In contrast, during the same period the mean height of black women declined by 1.3 cm from its peak of 163.8 cm for the earlier birth cohort to 162.5 for the 1980–1986 birth cohort. Black women increased faster in weight than did white women over the same period, so that the difference for women in 1986 aged 20–39 years old was 9.5 kg. Komlos suggested that "Two hypotheses are worth considering, namely, (a) that the decline in their height is related to the obesity epidemic and to inadequate dietary balance, and (b) that their future health will be subject to a double jeopardy in the sense that both their increasing weight and decreasing physical stature are likely associated with negative health consequences." A third hypothesis is that despite the civil rights movement of the twentieth century, the long-standing racism within the United States continues and the social, economic, political, and emotional impact of this racism further divided the well-being of blacks and whites as evidenced by trends for increased division in mean adult height. The harmful pressure on black women was so great as to produce a negative secular trend in adult height.

These examples from South Africa, Guatemala, the United States, and many, many other countries that could be cited, provide further evidence that the growth of human populations is a sensitive indicator of the quality of the social, economic, political, and emotional environment.

Eight Thousand Years of Secular Trend

The studies of recent changes in size and maturation of living populations reviewed here, the case of Maya-American refugee children described in the Introduction, and many more such studies in the literature show that human growth and development are dynamic phenotypic traits and responsive to relatively short-term changes in the quality of the environment for growth. To better understand the relationship between the stature of a population and its environment for growth, it is useful to examine long-term secular trends. Ryan Keep and I (Bogin & Keep 1999) did this for the adult stature of Native Latin Americans. Our use of Native Latin Americans has two connotations. First, it means those people identified as the descendants of pre-Colombian forager, tribal, chiefdom, and state societies – such people are also referred to in the literature as American Indians or Amerindians. Secondly, Native Latin Americans may also be people of social groups that formed after European contact, but came to identify themselves culturally (by language, dress style, kinship organization, etc.) as Latin American. For example, these groups include both Amerindian societies that formed post-contact, as well as groups of rural Mestizos, Ladinos, and others, who are people of mixed Spanish and Native American heritage. We excluded from our analysis people of primarily European, Asian, and African descent living in the Americas.

In total, 322 samples of adult height for men, representing 20,808 individual measurements, and 219 samples of adult height for women, representing 9,651 individual measurements, examined between the years 1873 and 1989 were found in the literature. In all samples, adult height refers to the stature of people who are reported to be 18 years old or older at the time of measurement. In addition to these data for people measured in life, we also assembled estimates of stature for archaeological samples of pre-Conquest and early post-Conquest populations from the present-day Latin American region. These estimates were based on the measurement of skeletal remains of individuals of higher social status (burials from tombs) and lower social status (nontomb burials). There were 29 samples for men and 27 samples for women, representing 1,305 and 1,158 individual measurements respectively. The pre-Conquest data are used to provide a deeper historical perspective on the dynamics of stature variation for Native Latin Americans.

The adult height data were analyzed by plotting the mean height data for each sample and then fitting a distance weighted least square regression to the data – this procedure fits a smooth curve that, basically, passes through or near the mean height for each year with data. Separate regression equations were fitted to the data for men and for women. The data for entire series of adult statures, and the regressions estimated by distance weighted least squares, are presented in Figure 5.10. For the archaeological samples, that is, the data prior to 1873, these regressions were calculated to present an idea of trends in "average" stature over time. Because intervals between archaeological data points are not equidistant, and since sample sizes are often small, and certainly not representative of all people alive at those times, it is not possible to perform formal statistical analyses. Nevertheless, a

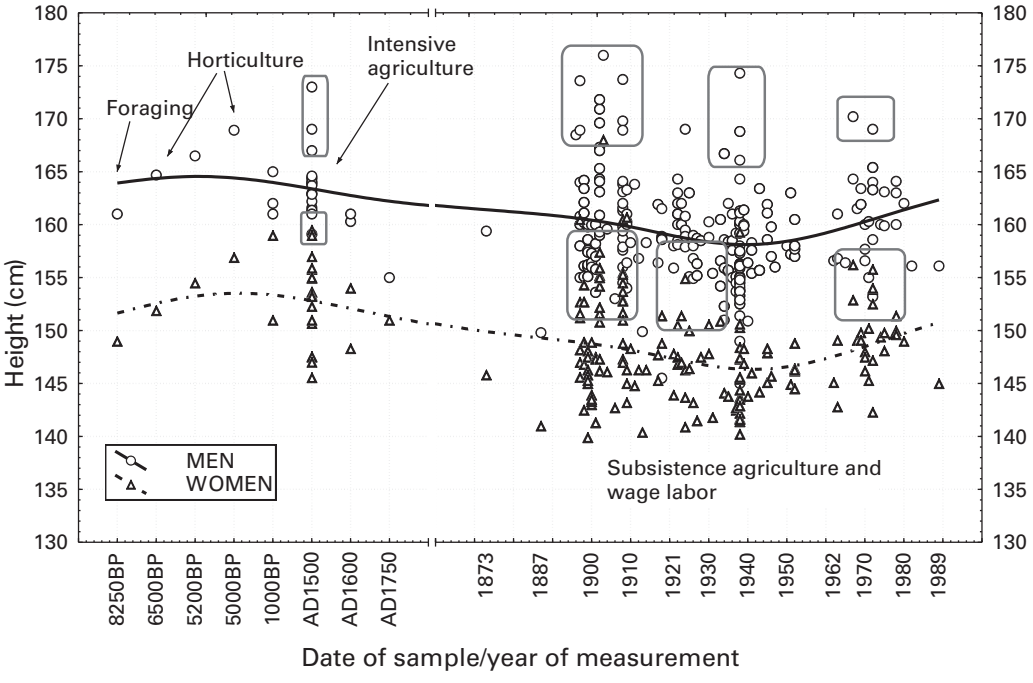


Figure 5.10 Mean statures of Latin American men and women during the past 8,250 years. Prior to 1873 the data are estimates of adult stature based on small samples of skeletal remains excavated from burials. From 1873 onward the mean statures are based on measurements of samples of the living. The fitted curves are trends in mean stature estimated by distance-weighted least-squares regression (see text for details). High SES individuals are in the boxes, men are above and women are below. From Bogin and Keep (1999)

narrative analysis revealed several important associations between estimated stature and the biological and sociocultural conditions for life.

The oldest data are for skeletal remains from the Vegas culture, a foraging people living along the southwest coast of Ecuador from 8,250 to 6,600 BP (the dates given in Figure 5.10 for the skeletal samples are always for the earlier point of a time range). These ancient foragers seem to have been taller than average, as the mean stature for the entire sample presented in Figure 5.10 is 159.2 cm for men and 147.6 cm for women. Archeological remains and chemical analysis of skeletons by Douglas Ubelaker and colleagues (1994) indicate that these foragers ate a wide variety of foods, including abundant fish and shellfish.

The next three archaeological samples are from the Paloma site of coastal Peru studied by Robert Benfer (1990). This is a pre-ceramic period site with many indications that it was permanently settled from 6,500 BP to 4,500 BP. The inhabitants of Paloma were horticulturists, producing a wide variety of garden foods, and also hunted and gathered wild animal and plant foods. The density of the population at Paloma was low to moderate relative to archaeological sites from later time periods. Socially, the Paloma people seem to have been organized into tribal-type political

groups, with minimal social stratification. They were economically and politically autonomous from any other social groups in the region. Mean estimated stature increases from the earliest period (6,500–5,300 BP), to the middle period (5,200–5,000 BP), and finally to the latest period (5,000–450 BP). Benfer pointed out that the increases in stature occurred along with declines in skeletal and dental indicators of stress, such as bone loss or enamel hypoplasias (defects of the teeth in which the enamel is deficient in amount, caused by health stress). Benfer interpreted these biological changes as evidence of increasing adaptation to sedentary life and improvement of the nutrition and health of the Paloma people. Indeed, by the latest period, the mean stature of Paloma men and women would be considered as tall, even by modern Latin American reference values.

The samples dated 1,000 BP are from several coastal and montane sites in Ecuador, spanning the time from 1,000 BP to AD 190. Archaeological remains indicate that the people at these sites subsisted mainly from intensive agriculture. Ubelaker stated "By this time, agriculture was well established in both the highlands and the coast, a shift toward increased sedentism had occurred, and population densities were higher" (1994, p. 148). Several lines of evidence indicate that the reduction in stature of these samples, compared with the earlier pre-ceramic period Paloma samples, was the result of economic, social, and political changes associated with intensive agriculture. Agriculture may have produced a decrease in diet quality for most people. An agricultural diet is usually restricted to a few intensively cultivated crops. Essential nutrient deficiencies are a common result of the restricted diet (Cohen & Armelagos 1984). In addition to dietary restriction, the social and political control that was necessary to efficiently organize agricultural labor almost invariably led to an increase in social stratification (i.e., workers vs. ruling elites), and then to economic and political inequality. Further exacerbating the plight of the lower social classes are the effects of warfare and military conquest, which were common in the Andean area at that time. Together these nutritional and social factors may have brought about a decline in health for the lower social classes, a decline preserved in skeletons of shorter stature.

The next group of data (marked as <AD 1,500) are for several pre-Conquest sites in Mexico, Guatemala, and Ecuador spanning the time from about 200 BC to the time of the European Conquest at about AD 1,500. All were complex, state-level societies (e.g., Toltec, Aztec, and Maya) with dense populations. It is noteworthy that by this late pre-Conquest period the variability in mean stature is almost as great as for the twentieth century. This variability was due to at least two factors; (1) the social status of the individuals within any sample; and (2) social, economic, and political changes over time between samples. Because these skeletons were recovered from state-level societies, there are marked social status differences between individuals within samples. High social status, the data points in shaded circles, is indicated when individuals were buried in tombs made from stone or other durable material. These burials were also likely to have grave goods, such as possessions of the deceased or items especially made to be buried with the deceased. Lower social status is indicated when individuals were recovered from nontomb graves, such as pits under a house or

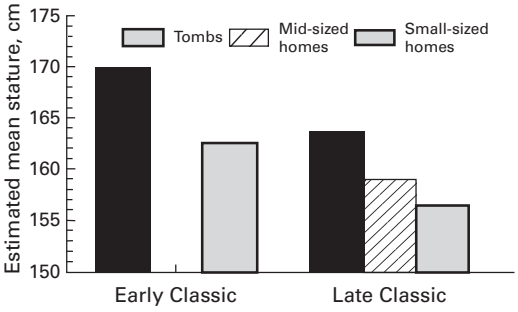


Figure 5.11 Mean stature of skeletons recovered from tombs, mid-sized houses, or small-sized houses at Tikal during the Early Classic or Late Classic Periods. Redrawn from Haviland and Moholy-Nagy (1992)

from rubbish middens. For all the archaeological samples, the tomb burials were taller, on average, than the nontomb burials.

The second source of variation in stature was due to changes in the conditions for growth over time. This effect is illustrated in Figure 5.11 for skeletons recovered from Tikal, a major Maya city-state and center of cultural life (agriculture, trade, religion, etc.) during the Classic Period (AD 250–900). During both the Early and Late Classic period, skeletons from tomb burials average greater stature than burials from small-sized homes or mid-sized homes (Late Classic period only). The size of homes is an indication of the wealth and social status of both the occupants and the burials (occupants of the home were usually interred under the floor of the house). Over time, the mean stature of both tomb and nontomb burials declines by about 5 cm. The decline in stature occurs during a time of increasing population growth, increasing warfare between Maya city states, increasing investment in militarization (larger armies, weapons production, construction of fortifications, etc.), and declines in food production and public building (Bogin et al. 2014a). The material and moral condition of that society were directed away from the environmental factors that would promote growth and toward those factors that would inhibit growth (Bogin & Varela-Silva 2015).

Returning to the data shown in Figure 5.10, the samples dated at AD 1600 are from the Tipu site in Belize and from post-Conquest cemeteries in Ecuador. Tipu was a mission site, periodically visited by a Spanish priest, but otherwise entirely inhabited by Maya. The cemetery at Tipu was in use from 1567 to 1638. The Ecuadorian cemetery samples come from two historic churches in Quito. The remains date from 1500 to 1725 and include Indian, Mestizo, European, and some Africa slave remains. Tipu males were slightly taller than the Ecuadorian males, but Ecuadorian females were noticeably taller than Tipu females. Given the mixture of ethnic and social groups represented by these samples, it may be best to note only that, on average, all these skeletal samples are shorter than the majority of the pre-Conquest samples.

The trend for a decline in average stature continued with the samples dated at AD 1750, which are burials dating from AD 1750 to 1940 from the same two Ecuadorian cemeteries just discussed. Indeed, estimates of the statures of pre-Conquest Latin

Americans (prior to 1500) are significantly greater than stature any time after the Conquest (pre-Conquest mean = 163.4 cm, SD 3.4 cm for men and 152.9 cm, SD 3.8 cm for women; AD 1600–1989 mean = 159.5 cm, SD 4.7 cm for men and 148.6 cm, SD 4.8 cm for women).

Stature of the Living

The samples sizes for people measured in life (after 1873) were means of 68 (SD, 89) individuals for men and 45 (SD, 76) for women. The large standard deviations about the mean samples size is due to a few samples having less than 10 individuals while others had more than 680 individuals. Overall, the sample sizes were large enough to be representative of the larger native Latin American population. Moreover, the data could be organized by equidistant time intervals. These characteristics of the data allowed for more formal statistical analysis of upward and downward trends in the regression curves for mean height. If only the endpoints are considered, the change in mean height from 1873 to 1989 was negligible and the linear regression coefficient for this entire time period was not significantly different from zero. However, mean statures decreased between the years 1898 to 1939 and then increased from 1940 to 1989 (the data from 1873 to 1897 were excluded as there were only four samples). For men and women, the decline from 1898 to 1939 amounted to about 4.5 cm and 3 cm, respectively. From 1940 to 1980 the increase is about 5 cm for men and 4 cm for women. These positive and negative secular trends were biologically significant. Separate linear regression coefficients for these two time periods also show that these trends were also statistically significant. The pattern of average change in stature was virtually identical for men and for women, and the difference in height between the sexes were almost constant. At the year 1900 the difference was 12 cm, at 1939 the difference was 11.5 cm, and at 1980 the sex difference was 12.5 cm.

What Do Secular Trends Mean?

Many plausible and fanciful proposals exist to explain secular changes. A few of these are transportation technology such as bicycles and railroads leading to genetic hybrid vigor as people from formally isolated villages met and married, changing climate and seasonal effects, the availability and price of sugar or other commodities as a cheap form of food energy, environmental toxicants and endocrine disruptors, such as PCBs, which may accelerate puberty, and the development of public utilities to provide heating – allows greater energy investment in growth vs. keeping your body warm – and artificial lighting – may stimulate growth, somehow. It is also proposed that psychosocial changes in the family, in schools, via media, etc. expose ever-younger children to sexual stimulants that accelerate growth and maturation. Each of these changes may play some small role, but it is now well accepted that modifications of the social-economic-political-emotional (SEPE) environment leading to transformations in the quality of life are the principle causes of secular changes of growth status. The quality of life may be measured by SEPE variables such

as education and literacy levels, food availability/market prices, cost of living, real wages, gross domestic product (GDP), social class and gender stratification/discrimination, rules for voting participation leading to democratization of society, and public expenditures on health. No matter which measures are used, the feelings of safety, security, and a hopefulness for the future are always greater in those populations that have more, and a more equal distribution, of these factors. Human height almost always follows the upward trend of physical well-being, emotional security, and hopeful expectation of a better life. These complex interacting variables are discussed, with supporting literature, in Chapter 7. The causal relationship between better SEPE environments and greater mean stature is so strong that mean stature itself is used to characterize the SEPE environments of historic and prehistoric populations before the invention of statistics such as IMRs, GDP, literacy rates, or cost of living indices. Some human biologists and economists call the relationship between the S and E (social and economic) components of SEPE environment with height the **biological standard of living** (Baten & Blum 2012; Komlos, 2009; Steckel 2009). This is a valid perspective, but far too narrow to appreciate the important impact of the political and emotional components that complete the SEPE model.

Pioneering researchers such as Franz Boas and others from the nineteenth and twentieth centuries (see Chapter 1) emphasized the importance of the SEPE environment as the cause of secular changes but could at best only correlate relationships. The first paper to test the SEPE hypothesis was by Acheson and Fowler (1964). They studied lower- and moderate-income parents and their children, ages 2–14 years old from South Wales and upper income London families, all sampled in 1960–1961. The Welsh families formed two groups: low-income coal miners and moderate-income shop owners and professionals. Current skeletal age was assessed for each child and an adult height predicted. The authors wrote, "The Welsh parents, for some or all of their childhood, were exposed to the privations of the economic depression of 1930s, but their children were born in more prosperous times since 1945. In contrast, the parents in the London sample did not suffer unduly in the late 1920s and 1930s and their children too enjoyed relative prosperity. Our working hypothesis was, then, that the between-generation difference in stature in the Welsh group should be greater than that in the Londoners" (p. 25).

This paper is quite "modern" in terms of statistical analysis and interpretation of findings. The authors found that within the parental or child generations and for both sexes, mean heights declined in a linear and statistically significant manner from upper income Londoners, to moderate- and then low-income Welsh. London boys grew more, faster, and showed more rapid skeletal maturation than the Welsh boys (no such effects for the girls). Between the generations, the children of Welsh miners grew more (about 1 cm) than the other two groups, which supports the authors' hypothesis. The authors considered and rejected alternative hypotheses that the mean height of each group reflects genetic or "racial" influences (recall the study by Rachel Fleming in Chapter 1). They conclude that findings reflect the SEPE environments of the two generations.

Acheson and Fowler's conclusions are amply supported by work from around the world. The Swedish data shown in Figure 5.8 may be explained almost entirely via improvements in public health (drinking water, sanitation), food policies, social welfare policies, democratization, reduction in social inequalities, and improved feelings of security. On a global basis, Hauspie and colleagues (1997, p. 20) emphasize the importance of SEPE factors by writing that, "The secular trend in attained height and in the tempo of growth is usually more pronounced in children from low socioeconomic backgrounds, in those with poorly educated parents or in those from rural areas. More marked secular changes appear to occur in the lower height centiles . . . " These are the groups who are most vulnerable to SEPE effects as they are at the margins of society.

Our own recent analysis of "Global effects of income and income inequality on adult height and sexual dimorphism in height" (Bogin, Scheffler & Hermanussen 2017) examined the relationship between height and income inequality. We analyzed data from 169 countries for national average heights of men and women born in 1996 published by NCD-RisC (2016) and national-level economic factors published by the World Bank to test two hypotheses: (1) income inequality has a greater association with average adult height than does absolute income; and (2) income inequality decreases sexual dimorphism in height. Hypothesis 1 is derived from the SEPE analyses finding that despite greater average wealth, income inequality can skew most of that wealth to the upper few percent of people. The Credit Suisse Bank Global Wealth Report for 2018[7] states that, in terms of inequality between poorest and richest people globally, " . . . the bottom half of adults collectively owns less than 1% of total wealth, the richest decile (top 10% of adults) owns 85% of global wealth, and the top percentile alone accounts for almost half of all household wealth (47%)." If you are not in that richest 1% then you also confront SEPE insecurities, to a greater or lesser extent, and if you are in the bottom 50% then you suffer every day. The Credit Suisse report also notes that "The ranking by median wealth per adult favors countries with lower levels of wealth inequality." This means that distributing wealth more equitably in a nation improves the average economic well-being of everyone. Greater income and wealth inequality make everyone poorer, except the top 1%, and overwhelmingly impacts the lower SES groups. As these groups make up the majority of the population in most nations, their shorter stature will bring down the average height of the nation as a whole. Hypothesis 2 is derived from analyses reporting diminished differences in adult height between men and women due to relatively greater male sensitivity to adverse environments (Stini, 1985; Stinson, 1985).

Income was assessed as GDP and gross national income in per-person purchasing power (GNI_PPP). Income equality was assessed by the Gini coefficient calculated by the Wagstaff method. Definitions of these economic indicators are given in the Box 5.4. The findings are graphically presented in Figures 5.12a for women and 5.12b for men. Hypothesis 1 was supported. Greater income equality was most

[7] https://www.credit-suisse.com/media/assets/corporate/docs/publications/research-institute/global-wealth-report-2018-en.pdf

Box 5.4 **Definitions of economic indicators. All definitions are based on The World Bank usage (Index Mundi data portal www.indexmundi.com/facts/indicators/). Values for these indicators are most commonly calculated in current US dollars**

GDP (Gross Domestic Product) is the monetary value of all the finished goods and services produced within a country's borders in a specific time period. GDP is the sum of gross value added by all resident producers in the economy plus any product taxes and minus any subsidies not included in the value of the products. It is calculated without making deductions for depreciation of fabricated assets or for depletion and degradation of natural resources.

GNI (Gross National Income) is the sum of value added by all resident producers plus any product taxes and minus subsidies not included in the valuation of output plus net receipts of primary income (compensation of employees and property income) from abroad. The GNI has replaced the GDP for many economic analyses.

GNI per capita PPP (GNI_PPP) is the GNI adjusted for population size and purchasing power parity (PPP). GNI_PPP represents gross national income converted to international US dollars using purchasing power parity rates. An international dollar has the same purchasing power over GNI as a US dollar has in the United States.

It important to express GNI per capita in purchasing power parity (PPP) international dollars when comparing the more than 200 countries and territories with different currencies and with very different price levels. To compare economic statistics across countries, the data must first be converted into a common currency. Unlike market exchange rates, PPP rates of exchange allow this conversion to take account of price differences between countries. In this way, GNI per capita (PPP $) better reflects people's living standards uniformly. In theory, 1 PPP dollar (or international dollar) has the same purchasing power in the domestic economy of a country as US$1 has in the US economy.

Gini coefficient measures the extent to which the distribution of income (or, in some cases, consumption expenditure) among individuals or households within an economy deviates from a perfectly equal distribution. A Lorenz curve plots the cumulative percentages of total income received against the cumulative number of recipients, starting with the poorest individual or household. The Gini coefficient measures the area between the Lorenz curve and a hypothetical line of absolute equality, expressed as a percentage of the maximum area under the line. Thus a Gini coefficient of 0 represents perfect equality, while a coefficient of 100 implies perfect inequality. The Gini coefficient is applicable only where and when data for monetary income are available – generally since the twentieth century.

Giniw also called the Wagstaff index or Concentration index. It is a standardized Gini coefficient calculated by dividing the World Bank Gini value by the maximal attainable Gini coefficient. The latter is computed based on the maximal level of a health attribute an individual could achieve. In this book, maximal lifespan is the health attribute as estimated by Petrie and colleagues (Kjellsson et al. 2015; Petrie & Tang 2008; Petrie et al. 2015).

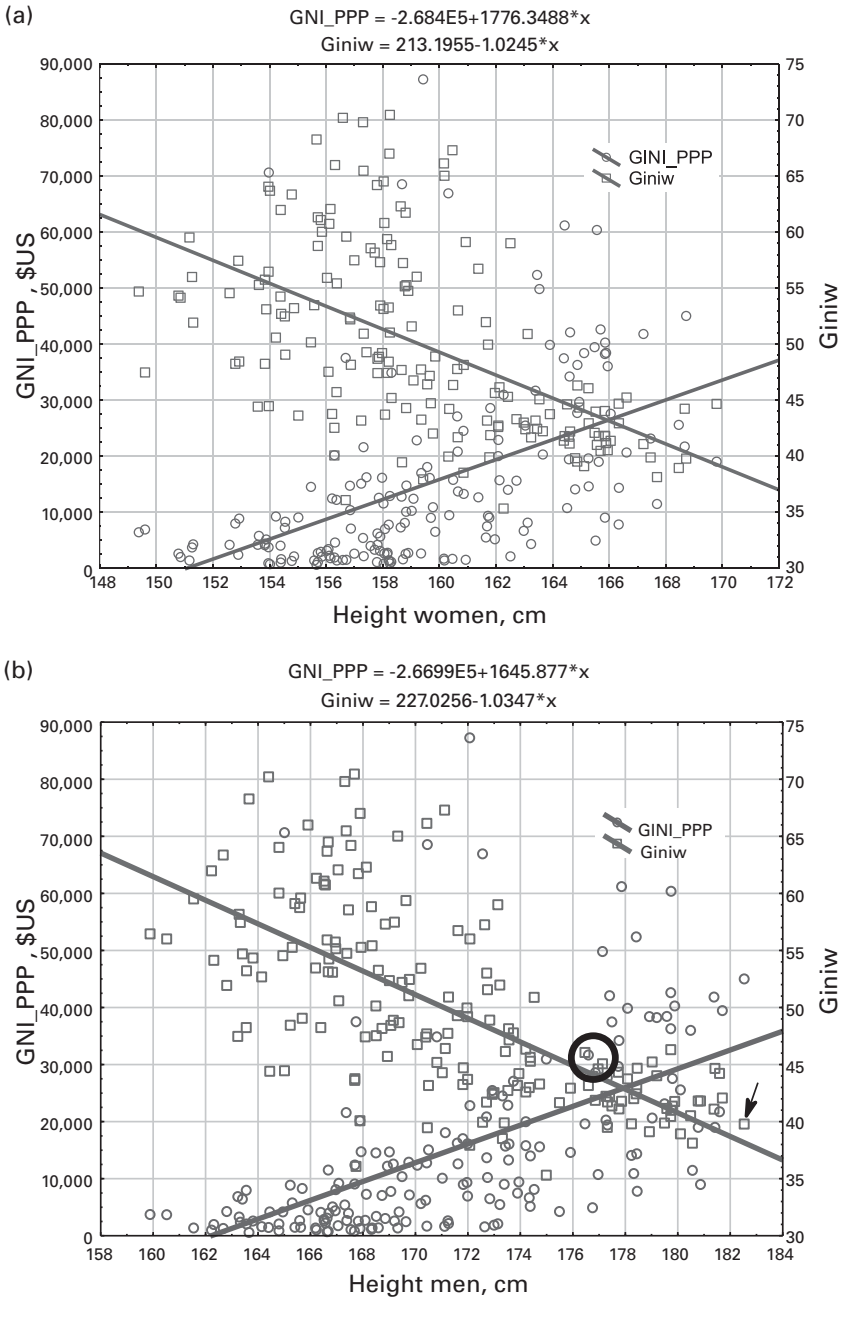


Figure 5.12 Scatter plots of mean national height for women (a) and for men (b) by Gross National Income per capita adjusted for Personal Purchasing Power (GNI_PPP in $US) and Gini standardized by the Wagstaff method (Giniw). The data are fit by linear regression; regression equations shown above the graph. In graph (b) the black arrow points to the height-Giniw data point for the Netherlands; with the tallest men, 182.54 cm, and a relatively low Giniw of 39.77. The black circle encloses the United States (177.1 cm, Giniw = 45.05), Grenada (176.97, Giniw = 44.61), and the Russian Federation (176.46 cm, Giniw 46.05). Author's original figure. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

predictive of average height for both sexes. GNI_PPP explained a significant, but smaller, amount of the variation. National GDP had no association with height. Hypothesis 2 was rejected. With greater average adult height there was greater sexual dimorphism. It is not known why this should be so but may support a modification of the "sex sensitivity to the environment" hypothesis. Boys and men may be more sensitive than girls and women to both adverse and advantageous environments. Our findings support a growing literature on the pernicious effects of inequality on growth in height and, by extension, on health. Gradients in height reflect gradients in social disadvantage. Following the work by Amartya Sen (Sen 2002), Richard Wilkinson and Kate E. Pickett (Wilkinson & Pickett 2009), and Michael Marmot (Marmot, 2015); economic, social, and political inequalities should be considered a "pollutant" which causes emotional stress and disempowers people from the resources needed for their own healthy growth and development and for the health and good growth of their children. Further discussion of the impact of the SEPE environment on growth is presented in Chapter 7 in the context of the theory of community effects and strategic growth.

Population Differences in Body Composition

Population variation in body composition is fascinating, sometimes contentious, but always an important focus of research. One aspect of its importance is due to the association between body composition and disease. Of special significance is the relationship of both the amount of fat on the body, and the placement of this fat at specific sites, to risks for cardiovascular disease, diabetes, and some cancers. For more than a century these diseases have been major causes of death in the wealthier nations. During the last 30 years or so, the spread of the overweight and obesity (OW/OB) epidemics to the middle- and low-income nations means that these diseases are major killers globally (NCD-RisC, 2017). Research, publications, new biomedical journals, popular media coverage, and SEPE activity relating to OW/OB grows at an exponential rate and it is not feasible to attempt to review that activity in this book. Instead, the discussion here focuses on the late twentieth-century studies that examined population variation in body composition. These studies were from a time before OW/OB became ubiquitous and fatness per se became the dominant biological feature of so many populations.

In twentieth-century United States, African-American children and youths had, on average, less total subcutaneous fat (the fat layer just under the skin) than European-American children and youths and were especially less fat on their extremities (Harsha et al. 1980). Robson et al. (1971) measured the triceps and subscapular skinfolds of children of African descent, 1 month to 11 years old, living on the Caribbean Island of Dominica. Only healthy, well-nourished children were included. The data were compared with similar measurements taken from a sample of English children of the same ages. The Dominica children were, on average, leaner than the English children, and this difference was entirely due to the Dominicas having significantly smaller triceps skinfolds. There were no significant differences between

the populations in the mean subscapular skinfold thickness. The results of these studies suggest that during childhood and adolescence, African-origin people have less total body fat, and a different anatomical distribution of subcutaneous fat, than Europeans.

Similar average differences in fatness and fat distribution have been found when other samples of African black children and American or European white children were compared (Eveleth & Tanner 1976, 1990). Based on these studies, some researchers conclude that a genetic difference in mean fatness and typical fat distribution may exist between children of African (black) and European (white) origin. Of course, many individual children do not follow the mean tendency for body composition of their natal population. Mueller et al. (1982) showed that genetics, or so-called "racial" differences, explain relatively little of the mean differences in body composition between black, white, and Mexican-American secondary school female athletes living in the United States. In this study, Mueller used multivariate statistical analysis to mathematically identify factors that influence fatness. A factor Mueller and colleagues called "ethnicity" (i.e., black, white, or Hispanic) was found to be unrelated to amount of fatness. In contrast, age, type of sport, and unspecified factors, of both genetic and environmental origin, accounted for most of the variance in fatness.

Since this study, there have been several discoveries of fatness-associated genes, such as the FTO genes. These genes are found in all human populations and do not cause fatness or fat patterning. Rather they are part of the multitude of extensive interacting interactions with food intake, physical activity, and other SEPE variables that influence human phenotypes. Further discussion on genomic influences on growth, development, and maturation is in Chapter 6.

Two studies conducted in Guatemala lend support to an environmental determination of body composition. In the first study, Johnston et al. (1975) examined samples of children of European ancestry and Guatemalan Ladino ancestry living in Guatemala. Both groups of children were of high socioeconomic class, attending the same private school, so in several ways they were exposed to a common environment. As measured by skinfolds, there were no significant differences in fatness between the ethnic groups at either the triceps or subscapular skinfold sites. Since the triceps site correlates highly with other measures of extremity fatness and the subscapular site correlates highly with other measures of trunk fatness, it may be inferred that there is little ethnic difference in fat distribution in this case. Johnston et al. suggested that the common pattern of fatness and fat distribution of the Europeans and Guatemalan Ladinos might be due to their living under similar environmental conditions.

In the second study, Tim Sullivan and I (Bogin & Sullivan 1986) found that the environment is the major determinant of the average amount of fatness and the fat distribution of children. We compared the fatness and fat distribution of four groups of children, age 7–13 years, living in Guatemala. The groups (and sample sizes) were: Guatemalan Ladinos of high SES (320 children), Europeans of high SES (164 children), Guatemalan Ladinos of low SES (340 children), and Guatemalan Maya of very

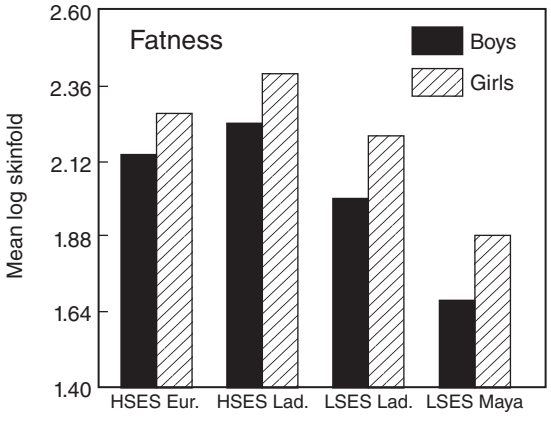


Figure 5.13 Mean values for the sum of the triceps and subscapular skinfolds of children living in Guatemala. The raw data were log transformed to normalize the distribution of the values. Larger log values indicate greater fatness. Sample abbreviations are: HSES Eur. – high SES Europeans; HSES Lad. – high SES Ladinos; LSES Lad. – low SES Ladinos, LSES Maya – very low SES Maya. From Bogin and Sullivan (1986)

low SES (669 children). Triceps and subscapular skinfolds were measured for each child. Previous research with these same groups had shown that SES was significantly associated with nutritional status, as reflected by skinfolds and other measures of body composition (Bogin & MacVean, 1981, 1984). So, as expected, and as shown in Figure 5.13, the high SES Ladinos and Europeans had, as a group, larger skinfolds than the low SES Ladinos or very low SES Maya.

It was hypothesized that SES would also be associated with fat distribution. Trunk fat maybe more physiologically important than extremity fat; for example, trunk fat may serve to protect the internal organs, and sufficient trunk fat is associated with successful reproductive development in women (Bogin et al. 2012; Pond 1998). Excessive trunk fat, especially abdominal fat, is more closely associated with risk for metabolic disorders, such as diabetes, heart and vascular disease, high blood pressure, higher triglycerides, and higher low density lipoproteins (Bell et al. 2018). Accordingly, if total body fatness is reduced in children from lower SES populations, there may be an evolutionary strategy for a relatively greater reduction of extremity fat and a relatively greater retention of trunk fat. As shown in Figure 5.14, Sullivan and I found that high SES Ladinos and high SES Europeans in Guatemala had a similar distribution of fat between the triceps and subscapular skinfold sites. The low SES Ladinos had significantly less fat at the triceps site than the two high SES groups, and the very low SES Maya had significantly less fat at the triceps site than any of the three other groups. Thus, as SES decreased fat distribution became more centripetal, that is, relative amounts of arm fat decreased and relative amounts of trunk fat increased. In accordance with the evolutionary hypothesis, the relative fat distribution changed even as the absolute amount of subcutaneous fat decreased at both arm and trunk sites from high, to low, to very low SES. It seems that fat distribution in these samples of children was determined, at least partly, by SES, which is a proxy

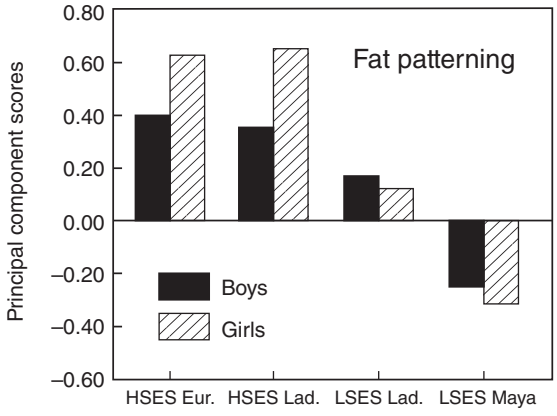


Figure 5.14 Relative distribution of subcutaneous fat, the fat patterning, at the triceps and subscapular skinfold sites for children living in Guatemala. Fat distribution is expressed as principal component scores; larger scores indicate relatively greater triceps fatness, smaller scores indicate relatively greater subscapular fatness. Sample abbreviations are: HSES Eur. – high SES Europeans; HSES Lad. – high SES Ladinos; LSES Lad. – low SES Ladinos; LSES Maya – very low SES Maya. From Bogin and Sullivan (1986)

measure for nutritional adequacy and many other SEPE environmental variables, interacting with evolutionary strategies to maintain health and reproduction. No evidence for an ethnic effect could be demonstrated for these samples of children.

Adult differences in body composition have been studied in several populations. Johnson and colleagues (1981), using data from a national sample of the United States, found that the average pre-adult black–white differences in fatness and fat distribution persisted into adulthood for males, from 1 to 74 years of age. That is, at all ages and at all levels of fatness, black males were leaner, and especially so on the extremities, than white males. Black females were found to be leaner, on average, than white females from the ages of 1 to 24 years, but from age 25 to 74 years, black females had larger mean values for the triceps and subscapular skinfolds than white females. The greater fatness of African-American women was mentioned earlier in the context of their negative secular trend in height.

A study by Robert Malina and colleagues (1982), compared athletes participating in the Montreal Olympic games of 1976 for skinfold measures of subcutaneous fatness and fat distribution. The sample included 264 white male and 133 white female athletes, 38 black male and 10 black female athletes, and 7 Asian male and 4 Asian female athletes, representing 46 countries, 20 major sports, and 68 different Olympic events. The median age of the male athletes was 21 years and of the female athletes was 23 years. It was found that the black athletes were significantly less fat than the white or Asian athletes. The total variance in fatness was statistically partitioned into the following factors (and percentage of variance): sex (31%), sport (19%), ethnicity (3%), and age (3%). The other 44% of the variance in fatness is not explained by these factors and must be due to other (uninvestigated) causes. In terms of fat distribution, white athletes had significantly more fat located on their

extremities than Asian athletes, who had more fat located on the trunk of their bodies. The fat distribution of the black athletes was intermediate between that of the white and Asian athletes. The factors, and percent of the variance, associated with fat distribution (as contrasted with the amount of fat) were: sex (35%), age (7%), ethnicity (2%), sport (2%), and residual (unspecified) factors are associated with the remaining 54% of the variance.

Malina and colleagues pointed out that the patterns of fatness and fat distribution of these highly selected and trained Olympic athletes follow the same pattern as that of the general population. With sex accounting for more than 30% of the variance in fatness and fat distribution, there seemed to be strong genetic and hormonal regulation. A 2017 article by Karen Reue provides experimental evidence for this. The article title "Sex differences in obesity: X chromosome dosage as a risk factor for increased food intake, adiposity and co-morbidities" summarizes the findings (Reue 2017). Typically, women have two X-chromosomes and men an X and Y chromosome. Some men have a double X and a Y chromosome. Laboratory mice also have these sex chromosome arrangements. In the mice, the "double X dosage" of females and some males is associated with greater food intake and greater fatness. The mouse findings parallel human observations of X chrmomosome dosage, eating behavior, and body fatness. In the Olympic athletes, the between population genetic determinants of body composition appear to be relatively weak, with the percentage of variance due to ethnicity being only 2–3%. Perhaps uninvestigated factors related to the many environmental conditions for growth and development, and their extensive interactions with the genome, which differed between the athletes, accounted for the unexplained variance in fatness and fat distribution.

The Significance of Population Variation

From this brief review of population variation in growth, it is possible to conclude that the differences between human groups in the average values for size, body proportions, and body composition are due to an interplay of that multitude of extensive interacting interactions discussed repeatedly in this chapter. Variations in the morphology of the human species have their own intrinsic fascination and scholarly appeal for study, but there are also important practical reasons for the analysis of these variations in growth. Cities and nations are becoming increasingly composed of people from many different geographic and ethnic origins. To monitor the health and welfare of the children of these diverse groups of people, clinicians and public health workers need to know about the normal range of amounts and rates of growth of children from different physical and cultural environments.

Furthermore, reference data for height, weight, body composition, and skeletal and dental development are used to assess health status, nutritional status, obesity, progress during treatment for disease, and relative risks for acquiring several acute and chronic diseases. For accuracy and reliability in their work, health care professionals may need population-specific reference data that reflect

both the hereditary and environmental determinants of growth and development for the people they serve.

Adaptive Value of Body Size in Human Populations

Implicit in the foregoing discussion of factors that influence population variation in growth is the fact that such variation occurs within the limits of biologically possible human phenotypes. Some of the evolutionary and ecological influences on the pattern of growth and development of the human phenotype were described in Chapters 3 and 4. In addition to research on the evolution of the pattern of human growth, there has been considerable interest in the evolution of human body size. One popular, but incorrect, notion is that the average height and skeletal mass of modern humans is greater than that of any of our ancestors. Based on several studies of more than 200 individual skeletons of early to late Pleistocene age (1.8 MYA to 10,000 BP), it seems that our ancestors, from *Homo erectus* to modern *H. sapiens*, were on average about 10% taller and 30% heavier than living humans (Mathers & Henneberg 1996; Ruff 2002). Other research, including the analysis of 8,000 years of secular trend in Latin America presented in this chapter shows that human beings today have about the same stature as our ancestors of the last 10–30 thousand years ago. It seems that the range of normal body size found in contemporary populations reflects the human condition of past populations of modern humans (*Homo sapiens sapiens*). Some researchers proposed genetic explanations for the decline in body size since the time of early *H. sapiens* (>10,000 BP), but Ruff (2002) explained that the difference was not due to any genetic change, rather it is due to the way of life of ancient and modern humans. Our ancestors were required to do more heavy labor, associated with a hunting and gathering way of life, and this imposed more mechanical loading (i.e., physical stress) on the skeleton. The increased mechanical stress seems to have occurred from an early age, and the skeleton responded by growing larger and more massive during the years of development. The human body seems to respond quickly, between succeeding generations, to changes of mechanical stress and other external exposures. An increase in mean height and a decrease in skeletal robusticity, measured as shoulder breadth, pelvic breadth and, especially, relative elbow breadth (elbow breath divided by height) was noted for children and youth from the former German Democratic Republic (East Germany) in the generation after the 1989 reunification with West Germany (Scheffler & Hermanussen 2014). A "feminization" of fat distribution pattern, meaning relatively more waist, hip, and leg fatness for boys and girls, was also noted between generations (Scheffler & Dammhahn 2017). The researchers suggested these changes were due to decreased physical labor (less mechanical stress) narrowing pelvic breadth and elbow breadth relative to height, along with better living conditions and, possibly, endocrine-disrupting chemicals in the general environment influencing stature and fat patterns.

As human beings alive today, we retain the sizes and shapes that were best suited for the way of life of our ancestors who lived as foragers (hunter-gathers) for 99% of

human evolutionary history. Today, fewer than 1% of people live by foraging. The advent of horticulture, pastoralism, agriculture, and permanent human settlements during the past 10,000–15,000 years significantly changed ways of life. Much archaeological and recent research shows that agriculture led to a decline in nutritional status and an increase in nutrient deficiency diseases and infectious diseases, especially for the lower social status groups within any society. There was also an exponential increase in local and worldwide population size leading to competition for land and other resources (reviewed by Bogin, 1998a). Each of these changes contributed to shifts in the quality of life, which was often reflected in reduced growth in height for groups with lower social status and, often, increases in height for those of the upper, dominant social classes (Figure 5.10; Hermanussen & Scheffler 2016). However, the question remains as to whether to some degree the reductions or increases in body size, shape, and mass represent a genetic adaptation. There has been much debate, for example, if the small size and altered shape (such as disproportionately reduced leg length) of people living under adversity is evidence of adaptation, or if these changes are evidence of suffering and failures of biological competence?

The meaning of adaptation is contentious within biology and anthropology. The perspective taken in this book is that for mammals as a group, the biological adaptation of the individual has three components. These are: (1) *survival*, meaning development of the individual to adulthood (reproductive maturity); (2) *productivity*, meaning adequate somatic growth, motor development, and brain/cognitive development for necessary physical activity and social behavior; and (3) *reproduction*, meaning both the quantity of offspring and their quality in the sense of continuing survival, productivity, and reproduction toward future generations. These are all part and parcel part of adaptation because death at any point prior to reproduction negates survival. Insufficient productivity at any stage of life may lead to low, or no, reproduction and death. Nonreproduction by an individual mammal or its offspring is, in a genetic sense, equivalent to death of that individual.

Because of the biocultural nature of human beings, we must add an additional cultural domain to human adaptation. As discussed in the Introduction, the members of all human societies require ideological systems for survival, productivity, and reproduction. This brings us back to the question of if the small size and altered shape of people living under adversity is evidence of adaptation or suffering. Everything about being human is biocultural, even human suffering. Human adversity often is the result of ideological systems, such as justifications for ethnic/racial discrimination, warfare, colonialism, and slavery. These ideological systems often cause disruptions to local systems of food production, the distribution of other critical resources, disorder to family and community, and the demeaning of the local ideology of the subordinated. Human adversity caused by war, racism, and religious/ethnic oppression may lead to malnutrition, exposure to infection, economic oppression/poverty, heavy workloads, forced migration to marginal environments (e.g., high altitude, deserts, refugee camps, urban slums). As may be seen from this list, the biological, economic, sociocultural, emotional, and political aspects of

hardship are concomitant. Accordingly, it is best to consider human growth under adversity and under privilege from a biocultural perspective.

Human Growth under Adversity

Human beings growing up in adverse environments tend to have reduced survival, productivity, and reproduction – the three aspects of the definition of adaptation given already. In terms of survival, there is a greater risk for fetal wastage as well as infant and child mortality. In terms of productivity, those infants who do survive may begin life with low birth weight or grow less in height and muscularity, have asymmetric body proportions (e.g., relatively short legs and arms for total stature), be wasted (low body weight) during the growth years, be overweight as adults, and be at greater risk for both infectious and metabolic diseases at all ages (Frisancho 2003; Varela-Silva et al. 2007). Additionally, during the years of growth and as adults, the survivors of adversity are at elevated risk for physical and cognitive impairments to work capacity (Hoddinott et al. 2013; Victora et al. 2008). Finally, reproduction may be reduced as women with short stature, high energy expenditure, low energy intake, and infectious disease burden often have lowered fecundity and fertility (Ellison 2017).

The dominant biomedical and clinical paradigm of the past 50 years or more views these outcomes as evidence of pathology and not as adaptation. There have been attempts, however, to conceptualize the presence of any fertility, combined with the small body size of women and men, living in these adverse environments as an adaptation. A controversial example was the ''small-but-healthy'' hypothesis (Seckler 1982). That hypothesis was dismissed on biological, economic, and humanitarian grounds (Pelto & Pelto 1989; Sen 1999). The critics of the ''small-but-healthy'' hypothesis cited the evidence just discussed that people growing up under adverse environments have reduced survival, productivity, and reproduction. Sen's critique added perspectives from political-economy and the psychology of human emotional affect showing that people living under adversity are not able to realize their material and emotional desires for survival, productivity, and reproduction for themselves and for their children.

With the demise of the ''small-but-healthy'' perspective, the dominant biomedical and clinical paradigm of ''small-and-unhealthy'' received new invigoration via a field of inquiry called ''developmental origins of adult disease.'' This field originated in the 1920s–1930s (Kuzawa 2005) but became more noteworthy when Ravelli, Stein and Susser (1976) found evidence of elevated risk for adult obesity among those exposed to the Dutch Famine of 1944–1945 during their first trimester of gestation. Building on this key study, many other related findings, and original research, David Barker (1938–2013) and colleagues (1989) extended the list of risks of poor fetal and early post-natal development to heart disease in adulthood and other adult metabolic disorders.

These relationships have been organized into the ''developmental programming hypothesis'' (DPH). In essence, the DPH predicts that exposure to adverse

environments during gestation results in a body that is smaller at birth and will be unhealthy in adulthood. The smallness is a marker of physiological disruption of the prenatal development of one or more physiological systems, including the cardiocirculatory, neuroendocrine, and renal systems. Smaller body size may be in total birth weight, head circumference, body length, organ size, or some combination of these. Barker's group made the important observation that the affected neonates may be within the clinically accepted range for "normal" birth weight or other dimensions, but they are at the lower end of that range. Affected neonates might also be disproportionate in size, for example, at the lower end of desirable birth weight but the upper end of birth length. Any of these, in Barker's view, indicate that prenatal development had been compromised. The DPH predicts that these alterations in growth have permanent maladaptive consequences that place people at risk for disease later in life.

Soon after the DPH was proposed, some researchers found that smaller size at birth plus a greater than expected amount of growth after birth worked synergistically to place people at elevated risk for adult heart disease, glucose intolerance, and other metabolic diseases. Peter Gluckman, Mark Hanson, and colleagues (Gluckman & Hanson 2004; Gluckman et al. 2005, 2007) conceptualized the mismatch between reduced fetal growth and accelerated postnatal growth into the hypothesis of "predictive adaptive response" (PAR). The PAR hypothesis posits that the small size at birth is the result of fetal development under some type of adversity.

Not properly discussed by the proponents of the PAR hypothesis is the population variation in the normal range of birth weight. The birth weights of neonates of South Asian origin, including India, Pakistan, and Sri Lanka, are generally lower than European neonates regardless of social strata. These lower South Asian birth weights are not always due to adversity and are typical of South Asian origin neonates born to well-off families in the United Kingdom, Canada, and the United States. In the United Kingdom, the birth weights of South Asian boys and girls are 9–15% lighter than white British newborns and require separate centile reference charts to correctly identify low birth weight for each ethnic group (Seaton et al. 2011). The clinical standard of a universal range of desirable birth weight does not reflect the biological reality of population variation. The PAR hypothesis does not consider this variation.

The PAR hypothesis defines two levels of adaptation: (1) "short-term adaptive responses for immediate survival"; and (2) "predictive responses required to ensure postnatal survival to reproductive age" (Gluckman & Hanson 2004, p. 68). Fetal growth faltering under conditions of nutritional constraint, such as placental insufficiency or maternal, is an example of the first level. Gluckman and Hanson (2004) stated that such short-term responses may be reversible. The second level PAR is of longer duration and less likely to be reversible. As an example, Gluckman and Hanson cited the work of Chisholm (1999) who reported that human girls, born to women living under adversity, experience relatively rapid growth after birth, early sexual maturation, and teenage pregnancies. The researchers considered this to be the result of two life history trade-offs: (1) between investment in one's own growth

and health vs. Reproduction; and (2) current reproduction vs. survival. As explained by Coall and Chisholm (2003), " ... trade-off underlies the prediction that under conditions of environmental risk and uncertainty (experienced subjectively as psychosocial stress) it can be evolutionarily adaptive to reproduce at a young age" (p. 1771). Coall and Chisholm (2003) pointed out that earlier maturation and reproduction in these girls, especially when less than 16 years old, came at the expense of their own health quality and that of their offspring (e.g., greater risk for low birth weight). But, despite the risks these relatively young mothers fulfill, at least at a minimal level of health, the survival, productive, and reproductive components of human adaptation.

Coall and Chisholm's (2003) work, and Gluckman and Hanson's (2004) use of it to defend their PAR hypothesis are controversial. The predominant view in human biology is that early life adversity may be more likely to delay growth and sexual maturation, as was discussed in the section on secular trends. Indeed, delayed sexual maturation may be viewed as a preferred trade-off to early maturation, as slower growth and a later age at first reproduction could serve to improve the growth of mother and her fetus/infant. As discussed in Chapter 4, the evolution of prolonged human maturation results in a young adult mother, vs. a teenage mother, who is likely to be better developed both physically and emotionally, if the needed material and social resources during growth are available. Part of the PAR controversy may be due to the distinction between material adversity (e.g., undernutrition and disease), which is assumed under the biomedical and secular trend perspectives, and psychosocial adversity (e.g., unstable family life, physical and/or emotional abuse), which is assumed under the Coall and Chisholm (2003) hypothesis. Because of the biocultural nature of human beings, it seems incumbent to consider both types of adversity, but each may have independent effects on trade-offs of growth and development as well as on adaptive responses.

Classic life history theory is derived from nonhuman species, especially insects, fish, amphibians, reptiles, and birds. For these nonideological species (some bird species might possess a type of ideology) it is necessary to consider only the physical adversity position. A human life history theory requires the combination of physical and psychosocial adversity caused by cultural behaviors and beliefs such as racism, nationalism, capitalism, and communism. Biocultural interactions between the four components of human adaptation – survival, production, reproduction, ideology – are likely to create diverse and unexpected outcomes in human growth and development. This leads to major questions about the PAR hypothesis. Can human embryos, fetuses, and infants make predictive adaptive responses in the context of the hugely complicated matrix of factors that comprise biocultural environments? Indeed, are the terms "predictive" and "adaptive" the correct terms to use? Biological responses will occur when the fetus or infant is exposed to adversity, but are these responses in any way foretelling of the most appropriate path for future development? Are these responses in any way beneficial, or are the responses evidence of deranged developmental physiology and risks for pathology? My answer to the last question is that most responses are pathological and not beneficial. The next section explains my answer.

Trade-Offs in Human Growth and Development

Predictive adaptive responses (PARs) require trade-offs (TOs), such as reduced fetal growth in favor of survival, or rapid maturation and early reproduction but with low quality offspring. These TOs correspond to Gluckman and Hanson's first and second levels of adaptation. Gluckman and colleagues see TOs as beneficial if they occur early in development to bring greater success later in life, such as during the reproductive period. Conversely, they see TOs as harmful when there is a significant mismatch between the prenatal/neonatal environment and the postnatal environment. In this case, the fetus/neonate makes incorrect adaptive responses that lead to disease later in life. One example is the greater glucose intolerance, heart disease, and obesity in adults who were exposed to the Dutch Famine as embryos/fetuses. None of these adaptive responses is a conscious decision, rather they are possible physiological and metabolic pathways to follow during development.

This discussion of predictive adaptation and TOs in human health must be evaluated against the available data for biological outcomes for growth and development. Gluckman and Hanson (2004) provide many human examples of such biological outcomes and some of these are organized in Table 5.2, following the life history model of human growth stages proposed in Chapter 2 of the present book. None of the outcomes seems ''good,'' in fact all are indicators of pathology. The mortality rate

Table 5.2 Human life history stages and associated growth and development outcomes under adverse conditions. The ordering of "Life History Stage" in the left column follows Table 2.1 in this book. The "Growth/Development Outcomes" in the right column are based on the examples given in Gluckman and Hanson (2004).

Life History Stage – adverse conditions	Growth/development outcomes
Prenatal – maternal nutritional deprivation; stress of hypothalamic-pituitary-adrenocortical (HPA) and/or HP-Thyroid axes.	Insulin resistance, more omental fat, reduced skeletal muscle mass, reduced bone mineralization, reduced capillary density in many tissues, impaired endothelial cells in heart and vasculature, reduced nephron number, reduced negative feedback of HPA axis (greater stress response), elevated adrenocortical and thyroid hormones.
Birth/neonate (to 28 days postpartum) – outcomes related to adverse prenatal conditions above.	Possible combination of low birth weight, prematurity, reduced brain growth/head circumference, reduced arm and leg length, impaired immune function.
Infant: 28 days to 2.9 years, and Child: 3–6.9 years, undernutrition, lack of play/stimulation, infection, neglect/abuse; overfeeding and/or low physical activity.	Growth faltering, short extremities especially the legs, infection-malnutrition synergism, motor and cognitive delays, HPA precocious sexual development, infant-child mortality; high BMI with excess fat, incipient diabetes and cardiovascular disease, reduced bone and muscle mass.
Juvenile: 7–10 years (pubarche), as above plus excessive physical labor.	Continuation of above responses with possible exacerbation of responses due to additional physical labor and greater exposure to pathogens due to increased independence.

for under 5-year-olds (listed as ''infant-child mortality'' in Table 5.2) is one of the most sensitive and widely used epidemiological indicators of how ''bad'' these outcomes are. A specific example is the nation of Guatemala, which in the year 2017 had the highest under 5-year-old mortality rate of all Central American nations at 27 deaths per 1,000 live born. That rate has declined year-on-year since the late twentieth century, but Guatemala has had the highest rate throughout the period.

In Guatemala, the majority of the population live under the conditions of material, political, social, emotional, and ideological adversity, as described previously and summarized in Table 5.2. Poverty is the main correlate of this adversity, with 60% of the rural population living below the poverty line.[8] The Maya ethnic group suffers more, with 79% living in poverty. The under 5-year-old mortality in the year 2006 for the total Maya population was estimated at 46/1,000, but in isolated rural areas the number doubled.[9] In a narrow sense, the women producing these infants and children are ''adapted'' because these women live long enough to reproduce. But, far too many of their offspring die and those who live are impaired with poor physical growth, reduced cognitive development, and diminished socioeconomic productivity. Given these outcomes, the trade-off between reproduction vs. reduced rates of survival and productivity (i.e., growth and work capacity) indicates, at best, a low level of adaptation for the Maya in Guatemala. More realistically, it indicates high levels of pathology in physical growth, economic productivity, educational attainment, and emotional well-being.

These measures of pathology for the Maya are, unfortunately, similar to other impoverished and lower-income groups around the world. Poverty cuts through all differences in political and religious ideology. Poverty, as measured by family income in monetary currency, is the most direct cause of human adversity, reduced adaptation in terms of survival, productivity, and reproduction. In the perspective of the poverty, the empirical data and the predictions of life history theory may be used to assess the relative merits of the developmental programming (DP) hypothesis vs. the PAR hypothesis for human development. At present, it seems that the focus on pathology and disease in adult life of the DP hypothesis has greater merit than the adaptationist view of the PAR hypothesis. In accord with predictions of life history theory, poor quality of life caused by poverty necessitates TOs in human growth and development, productivity, reproduction, and ideological health. In the case of ideological health, poverty and its associated social and economic inequalities promote environments of criminal behavior, violence, and political extremism. Rates of inequality, poverty, and crime in Guatemala are very high. An average of 101 murders per week were reported in 2018, making the country's violent crime rate one of the highest in Latin America. Also common are kidnappings, sexual violence against children and women, human trafficking, illegal drug business, and military oppression.[10] In this context, it is inappropriate to consider the biocultural

[8] https://borgenproject.org/exploring-poverty-rate-in-guatemala/
[9] UNICEF, https://data.unicef.org/country/gtm/
[10] https://en.wikipedia.org/wiki/Crime_in_Guatemala

TOs of the PAR hypothesis as adaptation in any sense of that word. The growth and development consequences of living in poverty at the margins of the wealthy world is simply pathology.

Hope for the Future

Readers of this book are urged to undertake further development of a biocultural human life history theory. The goal is to discover and promote policies and practices that will break the recycling of poverty and other inequalities across generations. We already know that even small reductions in poverty improve human development. An important example is the beneficial impact of a cash-for-work program on food consumption and nutrition among women and children facing food insecurity in rural Bangladesh (Mascie-Taylor et al. 2010). The research reported findings from a 10-week study that compared an intervention group of 895 households receiving cash-for-work with a control group of 921 households that did not receive any cash payments. At week 1, there were no significant differences between the groups in height, weight, or body composition for women and their under 5-year-old children. By the study end at week 10:

> . . . the difference in mean mid-upper arm circumference between women in the intervention and control groups had widened by 2.29 mm and the difference in mean weight, by 0.88 kg. Among children, the difference in means between the two groups had also widened in favor of the intervention group for: height (0.08 cm; $P < 0.05$), weight (0.22 kg; $P < 0.001$), mid-upper arm circumference (1.41 mm; $P < 0.001$) and z-scores for height-for-age (0.02; $P < 0.001$), weight-for-age (0.17; $P < 0.001$), weight-for-height (0.23; $P < 0.001$) and mid-upper arm circumference (0.12; $P < 0.001$). Intervention households spent more on food and consumed more protein-rich food at the end of the study (Mascie-Taylor et al. 2010, p. 2010).

Other types of interventions, such as giving people specific nutrients, or cooking classes, or a toilet, do not have this immediate and beneficial impact (Goudet et al. 2019; Scheffler et al. 2019; discussed further in Chapter 7). The last thing that rich people give to the poor is money and the services that correlate with money such as high quality education, health care, and housing, but as the Bangladesh study shows giving a bit of money for work immediately benefits the most vulnerable people – mothers and their infants and children. It was well known to early twentieth-century pediatricians and health workers that providing the things that money can buy, especially food and secure housing, allows for catch-up growth following starvation (Hermanussen et al. 2018b). This knowledge was forgotten for several reasons, not least of which was because the publications were mostly in German, a language that fell out of favor in science and medicine. The articles can be found on library shelves but are not translated to English and are not available on the Internet. Some of these old German-language reports are reviewed in Chapter 7 in the context of nutrition and growth.

Even with a bit of money, the poor Bangladeshi families will continue to face undernutrition and high disease loads but will be a bit better able to survive and

strive to make improvements for future generations. An important component of survival is due to the biological plasticity of the human species (Boas 1930; Lasker 1969). As explained in previous chapters, human plasticity allows the individual to adjust to a very wide range of adverse and stressful environmental conditions. Biocultural plasticity is a more powerful and beneficial phenomenon than are predictive adaptive responses. Biocultural plasticity gives the human species an adaptive advantage not found in those species obligated to develop according to a rigid, and predetermined, genetic plan. Plasticity also means that when environmental conditions improve, individuals can recover quickly and return to a more optimal size and shape and a more optimal social, economic, political, and emotional ecology. These permit people to realize their desires and hopes for themselves and their children.

6 Genetic and Neuroendocrine Regulation of Human Growth

Children tend to resemble their parents in stature, body proportions, body composition, and rate of development. It is often assumed that, barring the action of obvious environmental influences of growth, these resemblances reflect the influence of genes that parents contribute to their biological offspring. Genes do not directly cause growth and development. Rather, the expression of the inherited genome into a person's pattern of growth is regulated and mediated by several biological systems, especially the neurological and endocrine (neuroendocrine) systems.

Several examples of these interactions between genes, proteins, and the endocrine system were discussed in previous chapters. In the Introduction the derivation of word *auxology* and its relation to growth hormones was discussed. The PAX6 gene was also described. Recall that PAX6 codes for a protein that is thought to activate other DNA sequences involved in the formation of the eyes, the brain and spinal cord, and the pancreas. PAX6 protein is also involved in the development of the brain's olfactory bulb, a specialized group of brain cells that process smell. In Chapter 1 some of the historical notions of "genes vs. environment" and a brief history of discovery of growth hormone were presented. Chapter 2 reviewed the genetic control of limb regeneration, the genetics of birth weight, and the hormonal control of puberty. In Chapter 3 some differences between primate species in genomic sensitivity to hormones and the impact of those differences on species-specific curves of growth were reviewed. Chapter 4 discussed Evo-Devo, homeodomain genes, and more on the endocrinology of puberty and adolescent growth. Discussion in Chapter 5 focused on hormonal-stress influences, regulation of growth by hormones and signaling factors, and the many *extensive interacting genetic-hormonal-environmental* variables.

A summary of the previous chapters is given in Figure 6.1, titled "Biocultural domains of influence on human life history." The domains are in the two boxes to the left side of the figure. The relative size of each domain box indicates its proportionate influence. The "Phylogeny" domain, referring to human evolutionary history, has the most direct impact on genome, on the limits of the functional proteome, and on some aspects of human morphology and ethology (i.e., behaviors), such as the capacity for bipedalism and tool manufacture. The influence of "Phylogeny" interacts with and is mediated by the "Social-Economic-Political-Emotional" (SEPE) domain. Some of the SEPE variables discussed in Chapter 5 are listed in Figure 6.1. All human beings share a highly similar "Phylogeny" domain but live their lives under a diversity of SEPE conditions. Both domains influence the production and expression of the various

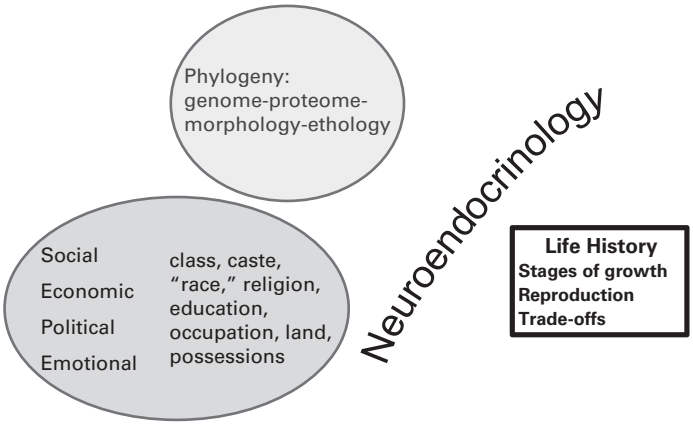


Figure 6.1 Biocultural domains of influence on human life history. See text for explanation (original figure).

neurological and endocrine products of the human body. The unevenness of the SEPE domain – some people have plenty and others poverty – interacts with the phylogenetic domain to produce a range of variation in norms of reaction of neuroendocrine activity. Neuroendocrine products have the most direct influence on the regulation of human life history, directing development through the stages of growth, adjusting the timing and frequency of sexual maturation and of reproduction, and modulating trade-offs in biology and behavior.

This chapter describes some aspects of the nature of the genetics of growth, the endocrine system, and the interaction of genes, the hormones, and the environment on human development. This is a very active area of research with a very large literature. Moreover, knowledge in this area of research is changing rapidly. Only a few aspects of the research being conducted in genomics and endocrinology can be covered in this book. Accordingly, the emphasis will be on research relating to topics discussed in previous chapters, especially those that relate to the biocultural nature of human development.

Genetics of Human Development

First some definitions are in order. The **genome** is the genetic material of an organism. In humans it consists of DNA, both the structural DNA, that is, the amino acid coding regions, and the noncoding regions which may be regulatory DNA or nonfunctional, so-called "junk" DNA. The genome also includes the genetic material of the mitochondria within most cells and epigenetic factors such as microRNA, DNA tagged with methyl, acetyl, ubiquitin, phosphate, poly(ADP)ribose, and other biochemical groups.

There is no universal definition of the "gene." One definition is "... a specific sequence of nucleotides in DNA or RNA that is located usually on a chromosome and that is the functional unit of inheritance controlling the transmission and expression of one or more traits by specifying the structure of a particular polypeptide and

especially a protein or controlling the function of other genetic material."[1] The concept and definition of the "gene" continues to be refined as new phenomena are discovered. For example, regulatory regions of a gene can be far removed from its coding regions, and coding regions can be split into several exons, that is, code regions along a strand of DNA that are separated by noncoding regions called introns. More complexity is added by the fact that under some conditions of gene expression the exons and introns exchange roles meaning that the same sequence of DNA chemical bases may be used to code many, many different gene products (colorful illustrations of the foregoing are found on the Internet). If we look to a wider array of living things, then we find that some viruses store their genome in RNA instead of DNA and some gene products are functional noncoding RNAs. Perhaps a better way to define a "gene" is "... any discrete locus of heritable, genomic sequence which affect an organism's traits by being expressed as a functional product or by regulation of gene expression" (Pearson 2006).

In 2003 it was announced that the human genome had been sequenced with an accuracy of 99.99 percent. Prior to the completion of the human genome project it was often suggested that more than 100,000 "genes" were needed to make a person. The current estimate is 19,000–20,000 structural genes which is less than 1% of all human DNA. The function of the other 99% is still being elucidated; it is known that some is regulatory DNA. The human genome is relatively "small" – about the size of the dandelion genome – and many insects, fish, amphibians, and flowering plants have larger genomes (Figure 6.2).

A General Concept of Heredity and Growth

Before further discussion of recent developments in the genomics of human growth, an overview of evidence for genetic influences on growth is in order. The general concept of heredity and growth was a theme treated by R. Darrell Bock (b. 1927). He analyzed longitudinal data from boys and girls, raised under favorable environmental conditions, participating in the Fels Research Institute Study, which was described in Chapter 1 (Bock 1986). As of 1983, 214 boys and 234 girls had been measured to maturity. The families of the Fels sample lived in the state of Ohio, United States and were largely of middle socioeconomic class and of European cultural background. Thus, the sample was not representative of all children of the state of Ohio or of the United States; however the Fels study provided a wealth of data about the growth and development of normal children.

Bock analyzed the data for individuals representing the extreme variants of the normal range of growth and development. His purpose was to describe variation in the inheritance of patterns of growth. In Figure 6.3, the height and velocity of growth in height of the tallest girl and the earliest maturing girl (defined by the rate of skeletal maturation) are compared. Both girls were tall for age throughout infancy

[1] www.merriam-webster.com/dictionary/gene

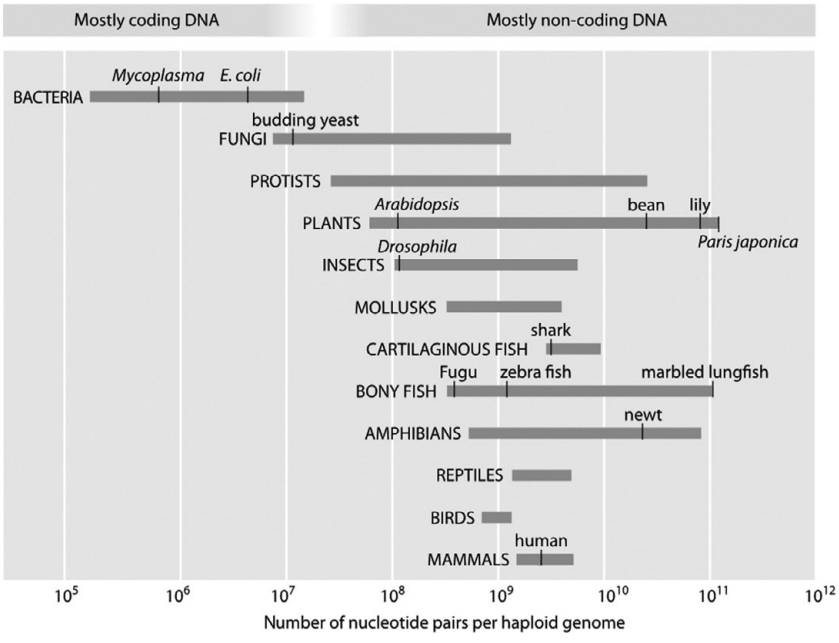


Figure 6.2 Graph of variation in estimated genome sizes in base pairs (bp). Human genome size is at the lower end of the range for mammals. Copyright © 2015 From Cell Biology by the Numbers by Ron Milo and Rob Phillips. Reproduced by permission of Taylor and Francis Group, LLC, a division of Informa plc.

and childhood, being above the 95th percentile for height for all girls in the Fels sample. The early maturing girl entered the adolescent growth period in her 7th year, reached peak height velocity at 9.4 years, and stopped growing by age 13 years, when she reached the 75th percentile of height for girls living in the United States. The skeletal maturation of this girl, as estimated from hand-wrist radiographs, was advanced over her chronological age by about three years. Thus, her tallness during childhood was due, in large part, to her advanced maturation.

The tall girl entered adolescence at about 10 years, reached peak height velocity at 12.2 years, and ceased growing at about 18 years at a height of 185.1 cm, which is at the 99th percentile. The parents of this girl were also tall, compared with other adults of like sex and age, which lead Bock to state that the growth of this girl is a " . . . typical example of intrinsic tall stature of familial origin." By intrinsic Bock seems to mean that the girl's tallness is a genetically determined characteristic, inherited from her parents.

Presented in Figure 6.4 are growth curves for the shortest girl and the latest maturing girl (again, defined by rate of skeletal maturation) that Bock analyzed from the Fels study. During childhood both girls were of similar stature, being at about the fifth percentile for the Fels sample. The short girl entered adolescence just a little later than the earliest maturing girl (shown in Figure 6.3), stopped growing in her fourteenth year, and reached 149 cm, which is below the fifth percentile. This girl

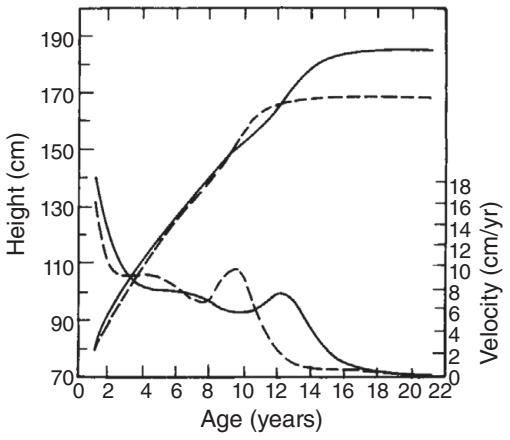


Figure 6.3 Distance and velocity curves for the growth in height of the tallest girl (solid lines) and earliest maturing girl (dashed lines) participating in the Fels Research Institute Study of growth. (Redrawn from Bock, 1986)

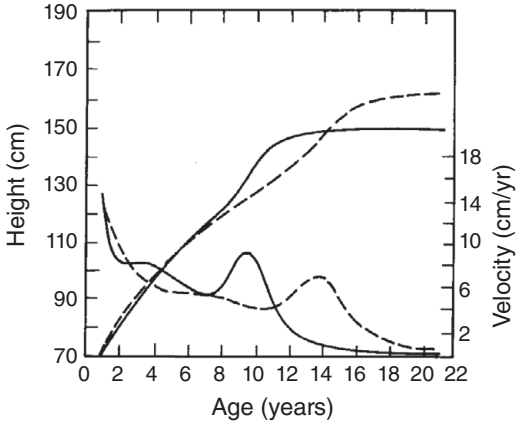


Figure 6.4 Distance and velocity curves for the growth in height of the shortest girl (solid lines) and the latest maturing girl (dashed lines) participating in the Fels Research Institute study of growth. (Redrawn from Bock, 1986)

was advanced in skeletal maturation from age 8 to age 16 years. Bock explained that her short stature is a family characteristic, inherited from her parents. Her stature was influenced by both a reduced amount of growth in size, combined with a developmental pattern for early maturation timing (a fast tempo).

The late maturing girl entered adolescence about two years after the short girl and, with this extra time for prepubertal growth, reached 160.1 cm, which was just under the 50th percentile. Her skeletal maturation was delayed in comparison with her chronological age. The delay fluctuated between one-half year and two years throughout her childhood and adolescence. This slow tempo of development allowed for a prolonged growth period and attainment of average stature.

Bock described these four cases of growth as "unusual," meaning that they represent the limits of the range of normal variation in amounts and rates of growth for the sample of the Fels Research Institute. The four girls were raised under favorable environmental conditions and showed no evidence for acute or chronic diseases that influence growth and development. Given this, and measurements of height of the parents of each girl, Bock's analysis indicated a major heritable component in the determination of size and rate of growth in these girls but could not determine the relative contribution of genomes or environments.

A similar study was published by Prokopec and Lhotská (1989) based on a sample of 81 boys and 78 girls. The subjects, all from Prague, were measured annually from birth to age 20 years. The Preece–Baines growth curve was fit to the longitudinal data of each subject. From these fitted curves for all the boys and girls, the three tallest, the three shortest, the three slowest maturing, and the three fastest maturing of each sex were selected. None of these extreme cases was known to have any major chronic or acute diseases. Neither the subject's history of common childhood diseases, nor the occupation of the fathers influenced patterns of growth and development. In contrast, the **mid-parent height** did predict the stature of offspring. Mid-parent height is the average of the stature of the mother and the father. It has been used for more than a century to predict offspring final height. Francis Galton (1886) noted in his famous article, "Regression towards Mediocrity in Hereditary Stature" that the parental average is not quite correct because it ignores that "mediocrity," which is now called regression toward the mean. An analysis by Hermanussen and Cole (2003) recommended that the adult target height of young people (tHT) be estimated by the formula: tHT = mean parental hSDS * 0.72, where "parental hSDS" is the standard deviation score for height of the parents. A standard deviation score is equivalent to a z-score. The positive impact of mid-parent stature on offspring growth attests to the role of hereditability, which likely has genomic and environmental contributions as parents and children were living in the same households and shared very similar environments. Inspection of the fitted growth curves showed that tall or short stature at age 20 could be predicted from stature at age 4 years. The predictability of adult height from stature at age four attests to the early establishment of individual patterns of growth and their stability over time.

Studies such as these have been interpreted to support the concept of a **genetic potential** for body size. Some researchers extend this concept to genetic potentials for body composition and body proportions. There is a narrow evolutionary definition of genetic potential as " . . . a heightened sensitivity to the effects of mutation that facilitates rapid evolution to novel states" (Meyers et al. 2005, p. e32). In this narrow sense, genetic potential allows populations or organisms to evolve their phenotypic mutation rate without changing their genetic mutation rate. Few growth researchers use the phrase "genetic potential" in this narrow sense. Rather, in the scientific and medical literature and in the popular media the phrase "genetic potential" usually means that every human being has a genetically determined upper limit to adult stature, the ratio of leg length-to-sitting height, muscularity, and other anthropometric dimensions. This concept is so well entrenched in the field of human

growth research that it is almost always used without definition or justification from research, that is, without reference to any data or appropriate data analysis. Interested readers may peruse recent issues of journals dealing with human biology, physical anthropology, pediatric medicine, and related fields, where they will find the phrase "genetic potential" used in this way (for example, Hanson & Gluckman 2014 discussing fetal growth). The problem with this casual usage is that the assumption on which it is based is not true. In Chapters 1 and 5 I reviewed research showing that adult stature, body proportions, and body composition are highly plastic. The short stature and the relatively short legs of past generations of the Japanese or the Maya, compared with Europeans, were once assumed to be genetically determined traits, but now we know that they are indicators of the quality of the SEPE environment. An upper limit for human growth in height has never been determined, not even for the tallest populations in the world today as "Nordic populations are still getting taller" in the twenty-first century (Holmgren et al. 2019).

In a more general sense, the whole concept of "genetic determination" is seriously flawed. That notion implies that the flow of information about how any human trait is developed, be it height, body fatness, personality, or intelligence, originates in the DNA and then unfolds into the phenotype. Within this scenario, one may allow for a greater or lesser amount of environmental influence on the phenotype, but the flow of information basically begins with the DNA and moves one way. In the following sections of this chapter I will show that the role of DNA in human development is much more complex, and often much less direct, than this. I am not going to enter a debate at the low level of "genes versus environment." Rather, my primary goal is to explain that the interactions between genes, hormones, and the environment may flow in all directions. A secondary goal is to indicate where we lack knowledge concerning the factors that produce the wide range of human phenotypes. Taken together, these goals should lead to the rejection of the simplistic and incorrect concept "genetic potential" and wider acceptance of the many *extensive interacting genetic-hormonal-environmental* variables that regulate human growth.

Back to the Homeodomain: Genes, Evolution, and Growth

Is there a "major genetic component" for growth? If it exists, then where might it be located? If found, then would this help to understand how it guides growth and development to produce a human being, rather than a chimpanzee, or a whale, or an oak tree? In the broader perspective of evolutionary time, what sort of genetic changes are required to produce new patterns of growth? Specifically, were new genes needed to evolve the new human life cycle stages of childhood and adolescence? In Chapter 4 I indicated that few, if any, new genes were needed, rather changes in the regulation of the expression of existing genes within the primate genome were all that was needed. However, new genes also evolve. Humans and the living great apes have similar genomes with 95% identity as described in the Introduction, but the ape genome is arranged on 48 chromosomes while humans have only 46 chromosomes. A fusion of two ancestral ape chromosomes created

human chromosome 2. It is possible that these were chromosomes 12 and 13 in the ancestral ape but this is not a certainty. It is estimated that the fusion took place ~3.5 MYA, with a range of ~2.5–4.5 MYA (Miga 2017). There are other major chromosomal differences between chimpanzees and humans, including inversions of DNA base sequences on nine human chromosomes and differences in transcription factors, such as the *forkhead-box P2 transcription factor* (FOXP2) which is involved in speech development. Other DNA sequences involved in hearing are also known to have changed during human evolution. These DNA changes at loci associated with speech and hearing suggest natural selection involving human language-related behavior (Varki & Altheide 2005). Another noteworthy human difference relates to the biology of sialic acids which are glycans, a molecule made from the combination of polysaccharide and protein (so-called "sugar-proteins") found in great abundance on most cell surfaces. There are more than 10 unique genetic changes in human sialic acids compared with great apes (Varki 2010). One association with these changes is the human propensity for meat eating. A high meat diet will cause illness and death in the great apes, but some human groups, such as traditional Eskimo and Inuit, can consume a diet that is 95% derived from animal sources (Leonard 2002). This animal-based diet includes high amounts of protein, fats, and the stomach contents of hunted herbivores. Human sialic acids may be one example of meat adaptive genes (Finch & Stanford 2004).

In all, the genetic differences at the level of chromosomes between humans and common chimpanzees are estimated to be about 10 times the typical difference between pairs of humans. Even this order of magnitude difference cannot account for all the changes in pattern of growth, morphology, and behavior between chimpanzees and people. Some additional discussion of homeodomain genes may provide a more satisfactory theoretical understanding of how the evolution of gene regulation led to the human pattern of growth.

Feet to Hands and Hands to Feet

The great apes and human beings have fore- and hindlimbs that are distinct in shape and function. Apes have relatively long forelimbs and are brachiators, while humans have relatively long hindlimbs and are bipedal. There is clinical and physiological evidence that homeodomain gene regulation plays an important role in the evolution of these differences (Goodman 2002; Muragaki et al. 1996). A mutation in the amino-terminal end of the HOXD13 gene sequence causes the condition called synpolydactyly. People with this condition have the carpal bones of the hands and the tarsal bones of the foot transformed into short carpal-like and tarsal-like bones. Some people also have deformed or duplicated digits (several X-ray photographs are given in Muragaki et al. 1996). The mutation that causes these phenotypes is an expansion of a polyalanine region of the HOXD13 gene.

This mutation, and the malformed hands and feet it produces, do not explain the evolution of human bipedalism from ape brachiation. But it is through the study of mutations that we often come to understand how genetic systems work normally. It

is quite possible that other regions of HOXD13 regulate the growth of ape vs. human limbs. It is known that HOXD13 and HOXD11 regulate the rate of cell division in the proliferative zone of growing cartilage of the chick tibia and fibula (Goff & Tabin 1997). Goff and Tabin proposed that all HOX genes are growth promoters, but that some are more effective than others. Since several HOX genes may compete for the target site of their regulatory action, " . . . the overall rate of growth in a given region is the result of the combined action of all of the Hox genes expressed in that region competing for the same target . . . " (Goff & Tabin 1997, p. 627). Goff and Tabin's model may account for the growth differences in size, timing, and intensity of limbs and other body segments between closely related species, such as chimpanzees and human beings. It is also possible that this model might explain how the timing, duration, and number of life cycle stages are regulated. I could not find more recent evidence evaluating the Goff and Tabin growth model, but there is a lot more literature on growth regulation by HOX genes – more than is possible to review in this book.

Genome-Wide Association Studies

In the year 2007 a technological advance allowed for the rapid and relatively inexpensive survey of whole genomes to search for genetic material associated with variation of phenotypes. These genome-wide association studies (GWAS) scan the genome for small variations, called single nucleotide polymorphisms or SNPs (pronounced "snips"), that occur more frequently in people with a particular phenotype. GWAS has been applied with some success to identify genomic variants associated with several human diseases, such as diabetes, heart abnormalities, Parkinson disease, and Crohn disease (Visscher et al. 2017). GWAS has also been used to search for genetic association with variation in human height, fatness, and BMI. The outcome of a 2015 GWAS of height and BMI that grabbed media attention with some outlandish associations between genetics and income is described in Box 6.1. The most recent GWAS at the time of writing this book, a survey of about 700,000 individuals of white, European ancestry, identified 3,290 SNPs associated with height variation and 716 SNPs associated with variation in BMI (Yengo et al. 2018). The authors describe the SNPs as "near-independent." Nonindependence between SNPs reduces the association between a given SNP and a phenotype. Just how "near-independent" the SNPs for height and BMI were in the analysis was not described, but the use of several new mathematical techniques especially created for GWAS analysis was required to estimate the associations. Using these newly created statistical and bioinformatic methods, the researchers reported that the genome-wide significant SNPs explained 24.6% of the variance of height and 5% of the variance of BMI.

Is there a genetic basis to differences between people in height – yes! The most recent GWAS finds that this basis accounts for less than one-quarter of all variation and is made up of several thousand SNPs, each contributing a very small amount to the overall modest variation (24.6% variance / 3,290 SNPs = 0.008% per SNP; this is

Box 6.1 **Genetic determinism is still alive at the BMJ**

The commentary that follows was cowritten with Michael Hermanussen, a clinical pediatrician, endocrinologist, and auxologist working from Altenhof, Germany, and Christian Aßmann, a biostatistician and econometrician at Otto-Friedrich-Universität, Bamberg Germany. It was originally prepared as an invited commentary for the journal *Annals of Human Biology* (*AHB*). We submitted the manuscript but more than a month later the Editor of the *AHB* claimed to have "lost" it and then declined to publish it because too much time had passed. The same Editor of the *AHB* also rejected a paper we submitted despite having been peer-reviewed by two colleagues of the Editor's choosing who recommended publication. That paper was peer-reviewed and published in another journal (Bogin et al. 2018a). As described in the main text of this chapter, science is a human endeavor which makes it subject to the personalities, emotions, biases, and limitations of the scientists – including journal editors. We publish our commentary here because it is relevant to both past and current GWAS research and the misuse of this research to promote incorrect notions of genetic determinism.

On March 8, 2016 the BMJ (*British Medical Journal*) published an article (Tyrrell et al. 2016[2]) that grabbed the attention of the media, with one newspaper headline reading, "For every 2.5 inches in height resulting from a man's genetics, his annual income increases by nearly £1600, while heavier women lose out on £3000 a year" (Davis 2016). This is not newspaper hyperbole as the abstract and text of the article includes almost identical sentences such as "'genetically determined' greater fatness causing a £2940 . . . lower annual household income . . . "

The news media are suckers for word combinations such as "genetically determined" and "caused by," but scientists should know better – especially biomedical researchers such as Tyrrell and colleagues. Decades of human genetic research, culminating in the Human Genome Project, failed to discover any simple, unidirectional pathway from genes to body height or weight. Instead, researchers consistently find complex, multilevel pathways that depend on how genes are regulated by nutrients, infection, environment temperature, sunlight, social and emotional interactions, and more factors.

The Tyrrell et al. article comes from a team lead by Professor Timothy M. Frayling of the Genetics of Complex Traits laboratory at the University of Exeter Medical School. Human height and body mass are certainly complex traits and certainly have some genetic basis, but can these biomedical

[2] This article preceded the GWAS analysis by Yengo et al. (2018) cited in the main text of this chapter. Yengo and colleagues use the same data analyzed by Tyrell et al., along with newer data, for their revised GWAS estimates of variance explained in human height and BMI.

scientists really derive such precise "inch by earnings" links between genetics, body size, and income?

The authors do not limit their predictive powers to only income, extending their analysis to education, job class, and deprivation. Variation in each of these SES proxies is claimed to be causally dependent on a suite of single bits of the genetic code, called SNPs, associated with human height and body mass index (BMI). Tyrrell and colleagues use the words "causal," "causally," or "causality" 50 times in their article. This is far too many times for there to be any misinterpretation of their intended meaning, which is that the SNPs *cause* people to be taller or shorter, or have greater or lesser BMI, and that these differences in height and BMI *cause* people to have higher or lower SES.

The authors use complex mathematical and statistical methods of bioinformatics that were specifically invented to analyze massive sets of SNP data to extract an estimate of genetic determination. Some of the bioinformatic techniques utilized are called Mendelian randomization, instrumental variable analysis, and genetic imputation. The last is a statistical method to infer the existence and function of genes (SNPs) that were not measured. GWAS researchers call these unobserved SNPs the "hidden genetic variance." The GWASers assume that those SNPs must be somewhere in the genome because it has been known since the time of Galton that 80–90% of the variance of height (or IQ, or gardening ability, or any trait of interest) is due to "heredity." Most readers will not be specialists in any of the analytical methods and may not even understand the assumptions and limitations of the methods. Readers will have to accept the authors' results as likely correct because to do so is easier than questioning. The mathematical manipulations of the GWASers, especially the imputation of "hidden data," provide an aura of sophistication and pseudo-precision that misdirects the attention of readers away from the main message of simplistic genetic determinism.

We question. How many times and how many ways did the authors process their data until they produced their meager results of 12.3% of the variance in adult height and 1.5% of the variance in adult BMI explained by the measured and imputed genetic variants? These results mean that 87.3% of height and a whopping 98.5% of BMI are not explained by genetics.

A devastating limitation of the Tyrrell et al. article is that only current adult height and weight are known. These are the heights and weights of the participants measured when they were, on average, 57 years old. They ranged in age from 37 to 73 years when the measurements took place, from 2006 to 2010. We know little to nothing of the height and, especially weight, of the participants when they were secondary school children and adolescents. That was the time when they were being influenced by personal and the societal biases concerning their height and weight. Decisions that the participants, and their families, made in relation to education and job training are completely unknown. Some of the boys and girls with a normal-slow tempo of growth

may have added inches of height after their education and job path decisions were made. Weight is quite likely to have changed a great deal between the teenage years and age 57 years.

The article by Tyrrell and colleagues is an adventure to read, but not all adventures end happily. This article passed not only the review process but was strongly endorsed by the *BMJ* editors who gave it Open Access and an engaging video abstract. Despite all this fanfare, the message we derive from the article is that the interactions between height, BMI, and social status are far from explainable by genetics, not even when using specially created statistical and mathematical methods of bioinformatics. It was well known prior to the publication of this article that height and BMI are influenced by a hugely complicated matrix of factors. Some of these factors are associated with the genome, but in view of the complexity of probable nutritional, health, social, economic, political, and emotional interactions with the genome, the genetic determination of body height, BMI, and household earnings is far from convincing.

my simple-minded estimate, as an unknown number of the SNPs will have interactive effects). The findings for BMI, only 5% of explained variance, add more evidence to the criticisms given in Box 2.1 about "bad BMI." A person's BMI tells us little about their genomic makeup and if BMI is misused to estimate fatness, then there is little genomic basis for human variation in fatness. Even when GWAS is focused on fatness the surveys for associated SNPs, especially "obesity genes," reveal little if any understanding. An international team of physicians, psychologists, statisticians/bioinformaticians, physiologists, and human biologists reviewed the findings from GWAS on BMI as an indicator of body fatness and wrote, " ... we argue in favor of a broad discussion between scientists from the areas of integrative physiology and of genomics. This discussion should aim at better conceived studies employing biologically more meaningful phenotypes based on in depth body composition analysis" (Müller et al. 2018). It is refreshing to read the words "body composition analysis," as this implicitly acknowledges the futility of further BMI analysis.

GWAS Estimates Variance, but Not Size

There seems to be a misconception about the meaning of GWAS results. It must be emphasized that GWAS is calculating the variance in height, BMI, or other traits associated with SNPs within a sample of people, but not the measured height or BMI of any one person. Knowing the details of every SNP associated with human stature cannot possibly indicate the adult height of any individual. The height of an individual woman or man is a phenotype and will be influenced by the physical environment and the SEPE variables reviewed in Chapter 5. An example of the difference between individual height and the variance of height for a sample is given

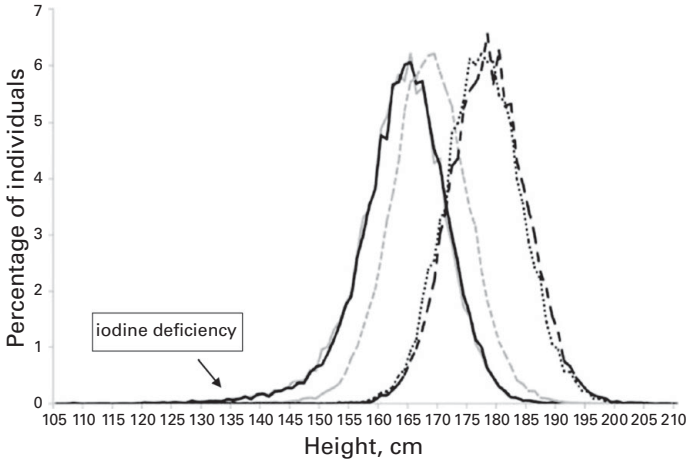


Figure 6.5 Height distribution of 5 cohorts of 19-year-old Swiss conscripts from 1878/1879 to 2008/2009 (representing ~90% of the total census population). Solid grey line: conscripts born in 1859–1860 and conscripted in 1878–1879, with a mean height of 163.3 cm. Solid black line: conscripts born in 1872 and conscripted in 1891, with a mean height of 163.7 cm. Broken grey line: conscripts born in 1908–1913 and conscripted in 1927–1932, with a mean height of 168.6 cm. Dashed black line: conscripts born in 1974–1975 and conscripted in 1993–1994, with a mean height of 177.4 cm. Broken black line: conscripts born in 1989–1990 and conscripted in 2008–2009, with a mean height of 178.2 cm. This figure is reprinted with modification from Staub K. et al. The average height of 18- and 19-year-old conscripts (N=458,322) in Switzerland from 1992 to 2009, and the secular height trend since 1878. *Swiss Med Wkly*. 2011;141:w13238. doi: 10.4414/smw.2011.13238, with permission from EMH Swiss Medical Publishers Ltd.

in Figure 6.5 for 5 samples of Swiss army conscripts over a 130-year period (Staub et al. 2011, 2015). The variance of the height distributions of the conscripts (the width of the curves) tended to remain constant but the height of the individual conscripts increased with time. In the late nineteenth century, height clustered around 163.3 cm; modern height clusters around 178.2 cm. Iodine deficiency in some regions of Switzerland was a major public health problem for the two earliest cohorts and the long "tail" of short stature is due to this nutrient deficiency prior to the distribution of iodized salt beginning in 1922. Only 8 individuals of the 89,800 men (0.009%) conscripted prior to the year 1900 were $>$190 cm tall, but 3,387 were $<$150 cm tall (3.8%). This compares with 1,367 out of 51,831 men (2.6%) taller than 190 cm, but 5 men (0.01%) $<$150 cm for the 2007/2008 cohort of conscripts. Because of Switzerland's policy of universal army conscription these cohorts represent more than 90% of the total population of Swiss 18–19-year-old men alive in each time period. There are ethnic and language differences between Swiss geographic regions, but it may be argued that within each region the cohorts shared the same common genomic SNPs. At any rate, 130 years is too short a time difference for major genetic selection of height to have occurred. The increase of height with time for all conscripts is due to nongenetic causes, but the near constant variance in the height distribution may be due to the SNPs shared across the cohorts.

Many questions remain about the accuracy and reliability of GWAS. Some questions stem from the interpretation of GWAS results using the newly created and complex statistical and bioinformatic techniques. These have assumptions which need to be examined and tested across many human populations. But, the GWAS search for the genetics of human height and BMI has been focused on Europeans. The SNP associations for variance of height or BMI of Africans, Asians, and others are unknown. Results derived from a GWAS in one population (e.g., Europeans) may have limited portability to other populations (Martin et al. 2017).

Even if there is population-specific validity to GWAS, the SEPE status of the European participants is not discussed in the GWAS literature. Environmental contributions to phenotypes are not taken very seriously or not considered at all. The participants are not considered to be people, rather they are genomes. The largest number of participants studied so far for height and BMI, more than 450,000, come from the UK Biobank Study of individuals aged 40–69 years. Only 5.5% of the 9.2 million people invited to participate in Biobank accepted. Analysis of the sociodemographic and health data of that 5.5% found that, "UK Biobank participants were more likely to be older, to be female, and to live in less socioeconomically deprived areas [meaning they are wealthier] than nonparticipants. Compared with the general population, participants were less likely to be obese, to smoke, or to drink alcohol on a daily basis and had fewer self-reported health conditions. At age 70–74 years, rates of all-cause mortality and total cancer incidence were 46.2% and 11.8% lower, respectively, in men and 55.5% and 18.1% lower, respectively, in women than in the general population of the same age. UK Biobank is not representative of the sampling population; there is evidence of a 'healthy volunteer' selection bias" (Fry et al. 2017).

As a case example, I am a participant in the UK Biobank Study and I am not representative in height (186 cm at my maximum), BMI (about 21), or health (generally much better) for my birth cohort (1950). The age of the Biobank participants is problematic because of "shrinkage." The measured heights of people aged 40–69 is not their achieved height at adulthood because height decreases by about 1 cm per decade after age 30 years due to deterioration of vertebral discs and ligaments throughout the body. But the amount of decline varies between individuals. The SNP associations with height cannot be accurate given that both height at young adulthood and the variable shrinkage are not known.

In summary, GWAS may serve to estimate the relative genetic association with variation of a phenotype, in this case relatively more variance explained for height and less for BMI, but the seeming precision of the variance estimates cannot be accepted at face value. GWAS cannot tell us much, or anything, about how short or tall an individual person will be. The best that GWAS can tell us is that the variance in height of groups of people is a complex phenotypic trait which is influenced by many, many hereditary factors, and some of these are genetic in nature with very small individual effects. All this was known in the late nineteenth century. It had been described by Francis Galton using, in part, the findings of his studies of twins.

Twin Studies As an Approach to the Genetics of Growth

Twin studies offer a traditional methodology, used long before homeodomains, HOX genes, or GWAS were discovered, for trying to tease apart the influences of biology and environment on human development. In the mid-to-late 1800s, Galton collected reports, case studies, and anecdotal information on 55 twins, 35 twin pairs of "close similarity" and 20 pairs of "great dissimilarity" (Rende et al. 1990). Today we understand that twins of "great similarity" are likely to be **monozygotic twins**, the product of a single fertilization with highly similar genetic makeup, but not genomic identity as will be explained. Less "similar" twins of the same sex are likely to be dizygotic twins, resulting from separate fertilizations and being about as similar genetically as any two full siblings. Galton could not know the zygosity of these twins, including which of the pairs were monozygotic twins ("identical twins") or dizygotic twins (so-called fraternal twins). Gregor Mendel's (1822–1884) work on genetics was carried out between 1856 and 1863, but the findings were largely ignored because most biologists believed in blending inheritance, in which the traits from each parent were somehow averaged. Even Darwin accepted a version of blending. The importance of Mendel's work was "rediscovered" in 1900. The nature of DNA was not elucidated until 1953 (Watson & Crick 1953) and confirmation that codons (groups of three DNA bases) determined amino acid sequences of polypeptide chains that composed protein molecules was published in the mid-1960s (Nirenberg et al. 1966; Söll et al. 1965). So, when Galton wrote about twins of "close similarity" and "great dissimilarity" he was referring to phenotypic appearance and not genetics. When Galton and others in the early twentieth century wrote about "heritability" they were not imagining genetics, rather they were guided by the observation that "children resemble their parents," for reasons that were as much due to "social breeding" as due to biology. Galton's interest in twins stemmed from his concern with the dichotomy of "nature" vs. "nurture." He thought that twins could separate the unique hereditary influences on human biology and behavior from the environmental influences. Galton published his observations on his sample of twins in an 1875 article, "The History of Twins, as a Criterion of the Relative Powers of Nature and Nurture."[3]

Galton felt that the histories of twins that he reviewed supported strongly the biological determination of most physical, behavioral, and mental traits. He concluded the article by writing, "We have only to take reasonable care in selecting our statistics, and then we may safely ignore the many small differences in nurture which are sure to have characterized each individual case." Indeed, with selection of the statistical methods of analysis it is quite easy to discard, even ignore, the impact of the environment, but this statistical denial is not good science and is not even close to the biological truth. In Box 6.2 I review some of the history of heritability statistics and their misapplication the human genetics.

[3] http://galton.org/bib/JournalItem.aspx_action=view_id=68

Box 6.2 **Some history of statistical heredity and genetics**

The hereditary effect in growth was first measured by the product-moment correlation, also called the Pearson correlation. Karl Pearson (1857–1936) was an English mathematician and eugenicist. He was a protégé of Francis Galton. Inspired by Galton's claims that heredity was all powerful over the environment and by social Darwinism – the eugenic notion that superior "races" of people would naturally dominate and exterminate inferior "races" – Pearson created the statistical models needed to measure inheritance, variation, natural selection, and sexual selection. His mathematical work and many of his ideas about physical science were highly original – Albert Einstein's theories of special and general relativity were influenced by Pearson – and his derivations of the chi-squared test, standard deviation, correlation and regression coefficients, principle component analysis, the *p*-value, and statistical hypothesis testing are fundamental to all statistical analysis. Like Galton, whose racist and eugenic politics were described in Chapter 1, Pearson was also a racist who wrote, "History shows me one way, and one way only, in which a high state of civilization has been produced, namely, the struggle of race with race, and the survival of the physically and mentally fitter race" (Pearson 1901, p. 19). Pearson presaged the writings of Adolf Hitler by adding, "If you want to know whether the lower races of man can evolve a higher type, I fear the only course is to leave them to fight it out among themselves, and even then the struggle for existence between individual and individual, between tribe and tribe, may not be supported by that physical selection due to a particular climate on which probably so much of the Aryan's success depended" (pp. 19–20). Pearson used the word "Aryan" a lot in his writings and juxtaposed the Aryan "race" to people of the Jewish religion who Pearson claimed were part of a " . . . parasitic race . . . inferior physically and mentally to the native population" (Pearson & Moul 1925). Pearson was not just an anti-Semite; he was against immigration into the British Isles by all "inferior races" from the European mainland.

I contrast Pearson's mathematical and physical science achievements with his political racism to give historical context to the use of statistics in the study of human heredity and genetics – just as I did for the anthropometric studies of Galton, Bowditch, Boas, and Fleming in Chapter 1. Doing a correlation analysis does not make one a racist, but the statistical techniques created by Pearson are mathematical tools designed to support the beliefs and social policies of racists and eugenicists. The motivation for Pearson's mathematical creations was the " . . . reasonable care in selecting our statistics . . . " advocated by Galton. Had Boas or another anti-eugenicist created the statistical tools to understand human heredity they may have been different. Researchers today apply different statistical methods and come to different conclusions about the same sets of data. The most relevant example to the present discussion of

human growth is the reanalysis of Boas's data on cranial shape of immigrant children to the United States mentioned in the Introduction. In one reanalysis Corey Sparks and Richard Jantz argued that Boas was incorrect regarding the plasticity of cranial form of the immigrant children (Sparks & Jantz 2002). In a rejoinder, Clarence C. Gravlee, H. Russell Bernard and William R. Leonard confirm Boas's conclusions (Gravlee et al. 2003) and claim that Sparks and Jantz posed a different set of questions and used different, and incorrect, statistical methodology. This type of disagreement is common in the literature and is often related to the choice of statistical analysis and the interpretation of the statistical results.

In the context of Galton's admonition, " ... to take reasonable care in selecting our statistics," it is important to know that the practitioners of GWAS are creating new bioinformatic and statistical tools to find the "missing heritability" in their studies. The phrase "missing heritability" is the one that the GWAS researchers invented in their publications to describe the difference between the percent of variance explained in a trait, such as height or BMI, and the percent of variance explained that they expected to find. The expectation is based on the historical analyses by Pearson and his followers of simple correlations between different classes of relatives, such as twins, siblings, cousins, etc. The expectation is also based on past pronouncements by "experts" that, for example, "90% of height variation is due to genes," even when there was no empirical support for these pronouncements (Feldman & Ramachandran 2018). Prior to 2018, GWAS research reported variance explained of less than 15% for height, but the 2018 article described earlier (Yengo et al. 2018) was able to extract an explained variance of 24.6% by applying a battery of newly created statistical tools.

This is not science; rather this is the denial of science. Creating new mathematical methods to find "missing variance" is a denial of the empirical findings of the SNP screening technology of GWAS (Feldman & Ramachandran 2018). The search for "missing variance" is driven by Galton's and Pearson's pre-genetic notions about "heritability" and is steeped in racism and eugenic politics. The work of Mendel was not known when Galton wrote about twins and heritability of stature and neither Galton nor Pearson used the term "genetics" – they used "heritability" and "inheritance." Today we casually interchange the terms "genetics" and "heritability," but Galton's heritability may best be understood as "phenotype similarity" and has nothing in common with what we associate with genes and heredity. Thus, when modern geneticists refer to the mathematics of Galton and Pearson, they simply ignore that these pre-Mendelian researchers were limited to "phenotype similarity," which is the result of a hugely complicated matrix of biocultural factors, including social, economic, and emotional components within the same family.

With the elucidation of DNA in the 1950s and 1960s the definition of "heritability" moved from phenotypes to that proportion of variation in a trait

that might be *caused* by DNA and then to variation that might be *attributed* to DNA (or RNA). In its current form "heritability" is a hypothesis about possible genetic influences in opposition to nongenetic influences. But in any form the "heritability" hypothesis is flawed because of the false opposition between genetics and nongenetics. The heritability hypothesis has not kept pace with biology and the discoveries of the simultaneous activity of extensive interacting variables happening within people, via social connections within bioculturally reproducing families, throughout communities based on kinship, marriage, friendship, economic interests, and ideology, via intergenerational effects, and with very deep phylogenetic and evolutionary roots.

The GWAS method is based on the heritability hypothesis and on technologies created to identify regions of the genome that are associated with the phenotypic expression of a trait. Taken at face value, GWAS provides a sensitive test of the "heritability" hypothesis and the results show that the pre-genetic era heritability estimates for height and other physical and behavioral traits were incorrect. But some of the practitioners of GWAS deny their own findings – they seem to be "married" to the heritability estimates of Galton and Pearson, so they follow Galton's advice and take much care in selecting and inventing new bioinformatic techniques to inflate the low genetic associations from GWAS into numbers that more closely approach the old estimates of "heritability."

The Racist History of IQ Heritability

I must give credit to Stephen Jay Gould for the basis of my critiques of the history of "heritability" from Galton's eugenics to the twenty-first-century search for the "missing variability." Gould's 1981 book, *The Mismeasure of Man*, critically documented the history of the racist pseudo-science of "intelligence." Gould showed how Galton and his disciples, including Pearson and Charles Spearman (1863–1945), took the many traits, abilities, behaviors, and aptitudes that comprise human cognitive capacities and reduced these into one reified concept – "intelligence." Once created as a thing, "intelligence" could be tested, people could be given an intelligence quotient score (IQ) and then sorted onto a linear scale of IQ points from Imbecile (20–49), Moron (50–69), Deficient (70–79), Dull (80–89), and Average (91–110). If people scored above 111 points they were assigned to categories of Bright, Superior, and Very Superior. The ranking of people was made possible by Spearman's invention of factor analysis. Gould described in elegant and comprehensive detail the historical background, the statistical methodology, and the valid biological and social science applications of factor analysis. Gould also details the errors and misapplications of factor analysis to estimates of "heredity."

As Gould noted, science is a human endeavor which makes it biocultural and dependent on technological-social-ideological interactions. The eugenicists created the statistical technology they needed, created learned societies to

give credence to their beliefs, and created academic and popular journals (e.g., *Annals of Eugenics*, founded in 1925 by Pearson) to propagate the ideology that heredity was all powerful in determining human phenotypes. One such learned society was the Eugenics Society, founded in 1907 and still exists as the Galton Institute in London. The roots of modern statistics are traced to proponents of scientific racism. The social interpretations of their methods and findings were guided by a pernicious ideology that infected popular thought, social policy, and the education of subsequent generations of researchers. Major scientific frauds were conducted in the study of the inheritance of IQ (mental ability), with results published in prestigious journals, and applied to political policy. These crimes including the fraudulent statistical analysis published repeatedly by Cyril Burt (1883–1971) were based on nonexistent twin data collected by nonexistent assistants. Burt's estimate that the "heritability" of IQ was 80–90% genetic was "common knowledge" and taught to generations of university students without question until Leon Kamin (1927–2017) showed that Burt's statistics were mathematically impossible (Kamin 1974). Even with this revelation, disciples of Burt, such as Hans Eysenck (1916–1997), continued to claim that Burt was correct about the genetic determination of "race" differences in IQ even if Burt's research was a fraud. Eysenck supported political campaigns against "racial" integration of schools in the United States based on the alleged intellectual inferiority of African-Americans. During the 1960s to 1980s these campaigns were waged using Pearson's statistics and Burt's discredited "heritability estimates" by William Shockley (a Nobel awarded engineer), Arthur Jensen (an educational psychologist), and Richard J. Herrnstein and Charles Murray (psychologist and political scientist, respectively).

Most recently, Robert Plomin and colleagues claimed that academic achievement of students in the UK is 75% due to genetic determinism of "intelligence" and other cognitive and personality traits and that height is up to 83% genetically determined. Plomin is a life-long proponent of racist and eugenic ideology and has published in both the scientific literature and in popular books (Jelenkovic et al. 2016; Krapohl et al. 2014; Plomin 2018). Plomin and colleagues base their work on twin studies. Plomin ignores the findings of GWAS that no more than 25% of height variation and no more than 4.8% of "intelligence" variation may be found in the "genes" (the SNPs). Plomin even ignores the latest meta-analyses twin studies showing the "heritability" of the variance in several cognitive domains averages no more than 50% (Feldman & Ramachandran 2018). Meta-analysis of published literature is a recently created statistical tool derived from the correlation and factor analysis methods of Pearson and Spearman. One would think that Plomin would gravitate towards the results of meta-analysis, but it seems that GWAS and meta-analysis are just inconvenient for the economic success of Plomin's

book sales and his political purposes. Other failings of Plomin's racist approach are detailed in a review of his book by Nathaniel Comfort.[4]

A further serious problem with heritability estimates of IQ, height, or any other phenotypic trait is that most often only a single measurement is available or is used for analysis. In the case of childhood IQ and height this is done in the belief that these traits are stable across the life course – if a person has a high IQ or is tall at age 12 years, then they are likely to remain so later in life. We know that this is absurd in the case of height because people grow at different tempos and stop growing at different ages. We also know that height shrinkage occurs after age 30 years. The same may well apply to IQ and the few studies with repeated IQ tests at different ages find considerable variation between tests. Our research group examined the longitudinal stability of individuals' IQ test scores derived from school-age tests and later-life intelligence scores (Mansukoski et al. 2019a). The longitudinal pre-adult IQ scores of 42 high socioeconomic status Guatemalans born 1941–1953 were analyzed and showed low stability. Year-to-year fluctuations of individual IQ scores of >1SD were found for 59.5% of the sample. The same participants were reassessed at ages 64–76 years and average pre-adult IQ explained only 12% of variance in the older age intelligence score. The reasons behind the longitudinal instability in test scores reported in this study were not known, but the results suggest IQ is not a stable trait and that single point measurements of intelligence must be treated with caution.

Mathematics May Be Beautiful, but Beauty Does Not Equal Truth

The mathematical tools developed by Galton, Pearson, and other racist-eugenicists are still used by Plomin and other researchers to "prove" their hypotheses about the genetic basis of human heredity. Deep and divisive differences in the interpretation of statistical findings are common and due, perhaps, to a persistence of eugenic expectations by some researchers vs. Boasian predictions of plasticity by others. At face value, Pearson correlations and Spearman factor analysis indicate a mathematical type of association between variables that is independent of social or political meaning. But, statisticians do not agree on even the mathematical interpretation of their statistical tests. Currently, there is a heated debate about the validity and usefulness of Pearson's creation of the *p*-value as an indication of statistical, biological, or social significance (Amrhein et al. 2019; Yaddanapudi 2016).

The most recent criticisms of GWAS and heredity come from genetics researchers themselves and concern the biology, not the statistics (Boyle et al. 2017). The researchers reanalyzed data from GWAS of height, schizophrenia, rheumatoid arthritis, and Crohn's disease. They found that GWAS was identifying DNA regions that are expressed in the cells relevant to height and

[4] www.nature.com/articles/d41586-018-06784-5

the diseases, but in addition, the GWAS analyses were identifying as many or more regions of DNA active in many other types of body tissues, especially DNA variants associated with gene regulation. These regions have broad biological functions and are potentially active in most biological activity in the human body. In response to the Boyle et al. findings one human geneticist quipped, "We might not actually be learning anything hugely interesting until we understand how these networks are connected" (Callaway 2017). The "networks" are the extensive interacting variables, from biology to behavior, nutrition, social practices, emotions, and ideology that simultaneously influence every human being during growth and development. The impact of these biocultural networks is discussed in some detail in Chapter 7.

As described in Box 6.2, disciples of Galton further developed the use of twins into the typical strategy to compare the growth of monozygotic (MZ) and dizygotic (DZ) twin pairs. Both MZ and DZ twins share similar uterine environments, and if raised in the same household share similar postnatal environments, so the differences between them in growth and development during fetal and early post-natal life will have a genetic component. Many studies of MZ and DZ twin pairs indicate that there is, indeed, a statistically strong genetic component in human growth. Based on a sample of 45 MZ and 67 like-sex DZ twin pairs measured for skeletal craniofacial dimensions, body girths (e.g., arm or thigh circumference), and skinfolds, Sharma and Sharma (1984) used the statistical technique of principle component analysis to estimate the hierarchy of genetic associations with phenotype. They reported that the genetic associations were greatest for skeletal dimensions, more moderate for girths, and weakest for skinfolds. The researchers noted that girths and skinfolds are more environmentally labile. How to properly assess gene-environment interactions has been a concern of twin studies since their inception. To make sense of the complex interactions requires careful and comprehensive consideration of how we conceive our research studies, identify participants, collect our data, and place analytical findings in their biocultural context.

An example comes from the Louisville, Kentucky Twin Study, which began in 1962 and by 1979 had recruited and studied more than 900 twins (Wilson 1979). Correlations in height and weight between MZ and DZ twin pairs at ages from birth to eight years are given in Tables 6.1 and 6.2. If amounts and rates of growth are totally controlled by the genotype, then correlation coefficients for MZ twins, who are assumed to be genetically identical, should be close to or equal to 1, a perfect positive correlation, at all ages. The correlation coefficient for DZ twins, who it is assumed share an average of 50% of those genes that are free to vary, should be equal or close to 0.50 at all ages. The numbers in Tables 6.1 and 6.2 show that by one year of age, and thereafter, the correlation coefficients predicted from the genetic model are very nearly found for this relatively large sample of twins. Ascribing these

Table 6.1 Correlation coefficients for height between monozygotic (MZ) and dizygotic (DZ) twin pairs from birth to age eight years.

Age	Total N	MZ	DZ same sex	DZ different sex
Birth	629	0.62	0.79	0.67
3 months	764	0.78	0.72	0.65
6 months	819	0.80	0.67	0.62
12 months	827	0.86	0.66	0.58
24 months	687	0.89	0.54	0.61
3 years	699	0.93	0.56	0.60
5 years	606	0.94	0.51	0.68
8 years	444	0.94	0.49	0.65

From Wilson (1979).

Table 6.2 Correlation coefficients for weight between monozygotic (MZ) and dizygotic (DZ) twin pairs between birth and eight years.

Age	Total N	MZ	DZ same sex	DZ different sex
Birth	992	0.63	0.68	0.64
3 months	766	0.74	0.66	0.40
6 months	819	0.81	0.63	0.39
12 months	828	0.88	0.55	0.37
24 months	779	0.88	0.53	0.50
3 years	713	0.88	0.52	0.54
5 years	606	0.85	0.48	0.62
8 years	444	0.88	0.49	0.46

From Wilson (1979).

correlations to genetics assumes that parents are randomly selected from the population of potential mates. This is usually not the case as positive assortative mating for height, for IQ, for social and economic status, and for many other biocultural characteristics occurs in all human populations. Assortative mating can have major impact on estimates of heritability, and more will be said about this.

The coefficients from birth to six months of age, however, do not correspond with genetic expectations. At birth MZ twins are less concordant in size compared with DZ twins. One reason for this may be that MZ twins often share a monochorionic placenta during the prenatal period. Frank Falkner (1918–2003) reviewed data from 7 studies of twin placentation and found that about 70% of MZ twins have monochorionic placentae (Falkner 1978). Vascular anastomoses (arterial and venous connections) between the parts of the placenta supplying blood to each twin occur in monochorionic placentae. This results in a transfusion of blood, and the oxygen and nutrients carried by the blood, between the twins. The transfusion of blood is usually not equal, which means that the twins do not receive an equal maternal blood supply,

possibly resulting in undernutrition and hypoxia (low oxygen availability) for the disadvantaged twin. In the condition of dichorionic placentation, each twin receives a separate supply of maternal blood and nutrients.

Falkner (1966) found that in a sample of 92 MZ twins, the within-pair difference in birth weight averaged 326 grams in monochorionic twins and 227.8 grams in dichorionic twins. Many subsequent studies confirm that monochorionic twins are more discordant in birth weight than dichorionic twins. These studies also show that monochorionic twins are generally of lower birth weight than dichorionic twins (De Paepe et al. 2015). These effects on birth weight in monochorionic MZ twins suggests that one or both fetuses is exposed to a deficient maternal blood supply and its intrauterine growth rate slows as a result of the placental insufficiency. One consequence of this type of uterine environment is a lower correlation coefficient between twins in length and weight at birth. This is the case as seen in Tables 6.1 and 6.2.

The values of the correlation coefficients at birth for DZ twins, displayed in Tables 6.1 and 6.2, are higher than expected from a simple genetic model of growth. DZ twins are more likely to have separate placental connections with the mother's vascular system. In this placental condition, it is more likely that each twin receives a relatively equal share of oxygen, nutrients, and other substances from the mother. This may explain, in part, the relatively high concordance in size of the DZ twins at birth. Research and analysis on the genetics of birth weight by Robson (1978) were described in Chapter 2. Not mentioned there is that Robson focused on twins and found that for both DZ twins and singleton newborns up to 66% of the variance in birth weight was due to maternal environment. That environment includes the quality of the mother's diet, smoking habits, alcohol usage, risk for diabetes, physical activity, and other variables of this type that influence the quantity and quality of the maternal blood supply to the fetus. Robson estimated that parental genetic factors accounted for about 24% of the variance in birth weight and only 10% of the variance was due to fetal genotype. A GWAS analysis by Horikoshi and colleagues (2016) raised this to 15%. All these findings indicate that the higher than expected concordance in the birth weight of DZ twins is likely to be due to the shared maternal environment, which sets some common limits to the growth of both twins. After parturition, growth is influenced by the extensive matrix of variables, from the unique genotype of each DZ twin to her or his unique environmental experiences, interacting with each other. These extensive interactions probably explain why the correlation coefficients for DZ twins in the Louisville, Kentucky Twin Study become less concordant as the twins get older.

In some instances, prenatal environment influences on growth have effects that last for many years after birth, and even act transgenerationally (discussed in detail in Chapter 2). These environmental effects may obscure the contribution of genes to the determination of size. Wilson (1979) examined a sub-sample of MZ twins from the Louisville study. This sub-sample consisted of 10 MZ twin pairs with the largest differences in birth weight. The lighter twin averaged 57% of the birth weight of the heavier twin and the average absolute difference between twins in birth weight equaled 1,064 grams. The smaller twin was usually of low birth weight and required

special postnatal care. Wilson found that by six years of age the relatively large initial disparities were progressively reduced but not eliminated. The mean difference for weight at six years was 2.19 kg and for height was 1.85 cm, indicating that the lighter twin at birth was still 10% lighter and 2% shorter, on average. For these 10 pairs of MZ twins the within-pair correlation for height was only 0.72 compared with the correlation of 0.93, or higher, for all MZ twins in the Louisville study (Table 6.1).

Similar long-term differences in growth between MZ twins of markedly different birth weight have been reported by Falkner (1966, 1978). In one case, full-term MZ twin boys had birth weights of 1,460 grams and 2,806 grams and birth lengths of 43 cm and 50 cm respectively. The smaller twin was considered small for gestational age and required special neonatal care. Inspection of the placenta showed that it was monochorionic, with only about 40% of the placenta supplying maternal blood to the smaller twin. During the first year of life, the growth rate of the smaller twin exceeded that of the larger twin, but the catch-up in growth was incomplete. Differences in height, weight, and other physical measurements persisted at all ages to 16 years of age, e.g., heights and weights at age 16 were 161.9 cm and 50.6 kg for the smaller twin and 167.3 cm and 58.5 kg for the larger twin.

The implication of these observations is that twin studies may provide clear indications of the genetic regulation of growth, but only when the environment for growth is, essentially, equal and favorable, meaning that the environment does not inhibit the growth of one or both twins. More than two-thirds of MZ twins share a monochorionic placenta, and many of these twins do not experience equally favorable prenatal environments. Few twin studies systematically account for the effect of placental type or the subtle consequences it may have on growth and development. An additional caveat is that few studies evaluate the effect of assortative mating (e.g., similarity in height, education, religion, diet preferences, political orientation, and other dimensions) of the parents. As mentioned above, assortative mating impacts estimates of heritability, whether estimated by correlations between twins or other biological relatives, by GWAS, or by gene-linkage studies, or by any other method. It is well known that assortative mating correlations between spouses of IQs average about 0.5 and of heights range from 0.1 to 0.6 (mean = 0.23) (Silventoinen et al. 2003; Stulp et al. 2017). The correlations in the richer, more industrialized nations are higher than in the least developed nations. Most of the heritability studies based on twins and GWAS use samples from the richer nations, so assortative mating will most bias these analyses.

Another bias is a factor called to **cultural inheritance**, defined as influences on the development of offspring phenotypes by the interaction of its genotype and her/his parents' phenotypes when the parental phenotypes act via direct vertical cultural transmission (Cavalli-Sforza & Feldman 1973; Feldman & Ramachandran 2018). The proponents of cultural inheritance, Luigi L. Cavalli-Sforza (1922–2018) and Marcus W. Feldman (b. 1942) were/are biologists with specialization in human genetics and demographics. Their conception of cultural inheritance and direct vertical cultural transmission means that the behavior of parents influences the development of their

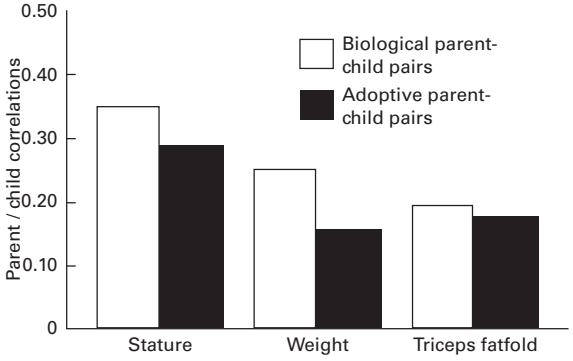


Figure 6.6 Comparison of biological parent-child correlations and adoptive parent-child correlations for stature, weight, and the triceps skinfold using the average correlation value for ages birth to 18 years. The correlations were calculated from age- and sex-appropriate z-scores. Adoptive parent-child correlations are surprisingly close to biological parent-child correlations for length, mass, and outer fatness. Redrawn from Garn et al. (1976)

children's phenotype. For Cavalli-Sforza and Feldman the word "behavior" was focused on teaching, but it may be expanded to include all the SEPE variables that have been discussed in the chapters of this book. Complicating things further, assortative mating interacts with cultural inheritance two ways: (1) via phenotypic homogamy and (2) via social homogamy. The former refers to the similarity between spouses in physical traits, such as height and fatness, and the later refers to similarities in socioeconomic status, class, gender, ethnicity, religion, age, or other sociodemographic traits. The two types of homogamy also interact to enhance the phenotypic similarity between parents and, in turn, multiply the cultural inheritance influence on their offspring. The effect of cultural inheritance from the parental behavioral and SEPE phenotype is often about as powerful as the influence of the offspring genotype on estimates of heritability, namely about 30%, for IQ.

The cultural inheritance for height is also about as strong as that for offspring genotype. Garn and colleagues analyzed a sample of 7,230 parent-child pairs (6,726 biological and 504 adoptive) from a study of European-origin families living in the state of Michigan, United States and reported that " ... adoptive parents and their adopted children tend to resemble each other in height, weight and fatfolds to an extent paralleling height, weight and fatfold resemblances of natural (biological) parents and their children" (Garn et al. 1976, p. 539). The term "fatfold" means "skinfold." The summary of the analysis is given in Figure 6.6. Adoption agencies are known to practice assortative placements based on the height of biological and adoptive parents. In addition, adoptions of infants and children may be by biologically related family members, so these are possible sources of the phenotypic similarity in addition to direct parent-to-child genetic inheritance. Other important sources of cultural inheritance are the ways adopted offspring are treated, in other words, the cultural transmission of height via parental behavior and SEPE characteristics. This is part of the process of generation changes (secular trends) in height – children become

taller than their parents when environments are improving. Such improvement may be in the physical, nutritional, and health environment, but also in the social and emotional environment. Community effects and competitive growth, which were mentioned in Chapter 5, are also factors. The analysis of Maya-American children described in the Introduction shows how rapid the height gain can be when the environment is powerfully improved and the children grow up in a new SEPE environment (Bogin et al. 2018a).

The existence of the genome, the nature of DNA, secular trends, migration effects, community effects, competitive growth, and the power of the SEPE environment were unknown to Galton and other hereditarians of the nineteenth and early twentieth centuries. Secular trends, migration, community effects, competitive growth, and the power of the SEPE environment are not considered in the analyses of heritability by traditional statistical methods or by the new methods of GWAS. Including a mathematical term in GWAS statistical models for just the secular trend in adult height, which has been between 15 and 20 cm over the past 100 years for several European populations (NCD Risk Factor Collaboration (NCD-RisC) 2016) would likely reduce estimates of genetic heritability. Including terms for community effects, competitive growth, and the power of the SEPE environment would likely eliminate a statistically significant effect of the genome on GWAS estimates of phenotypic variance.

The point of all the forgoing discussion is that the magnitudes of parent-child resemblances commonly assessed by "heritability" estimates of MZ twins, DZ twins, and other degrees of biological relatedness reflect more than genotypes. Heritability researches need to consider extensive interacting variables from all sources. But, as Feldman and Ramachandran (2018, p. 7) wrote, "The language used to interpret heritability has not changed much with advances in genomics, despite occasional genuflections towards its inability to assign causes and to gene-environment/gene-culture interactions."

Correlations in Growth between Biological Relatives (Non-Twins)

Studies of familial correlations in growth are another traditional strategy used to clarify the role of genes and the environment. There has been little research in this area since the 1990s. Familial correlations for serial measurements of stature were analyzed by Byard et al. (1983), using data from the Fels Research Institute that included measurements of pairs of relatives, e.g., siblings, parent and offspring, cousins, uncle–nephew, aunt–niece, etc. Each of the pair had been measured once a year from 1 to 18 years of age. Correlations were calculated based on age-matched measurements (e.g., father's height at age 15 and his son's height at age 15). Multivariate analysis of the correlations found that degree of relatedness explained most of the variation in stature. That is, first degree relatives had higher correlations than second- or third-degree relatives. However, the effect of the common environment between first degree relatives, usually living in the same household, could not be separated from their genetic similarity. For example, between the ages of 1 and 15

years, correlations between siblings were always higher than those between parents and offspring. Theoretically, both parents and their offspring and siblings share about 50% more of their genes than the amount shared at random between any two unrelated members of a breeding population. Both full siblings and parents and offspring, then, should have approximately equal correlations in stature. However, the siblings lived together in the same households, which may have resulted in a "commonality of environment" effect, increasing the value of sibling correlations. A similar pattern of correlations in anthropometry was found for rural Guatemalan (Russell 1976) and rural Columbian (Mueller 1977) families. The authors of both studies also interpreted the higher correlations between pre-adult siblings, vs. adult siblings and parent–offspring pairs, as the effect of a more similar environment for growth shared by the young siblings who lived in the same households.

Other studies show how familial correlations, although theoretically a measure of genetic similarity, are equally a measure of the environment. For instance, the power of the environment to influence the value of sibling correlations in size was demonstrated by Mueller and Pollitt (1983). They used data gathered by Dr. Bacon Chow (Chow 1974) from a study of the effects of nutritional supplementation of pregnant women on the subsequent growth of their offspring (Dr. Chow died before analysis of the data could be completed). The study included measures of the prenatal and postnatal growth of siblings, who were living in a rural Taiwan village characterized by high rates of chronic undernutrition. Each woman in the study contributed two infant participants. During pregnancy with her first child the mother was untreated, while during pregnancy with the second child she was given either a food daily supplement of a milk-based formula providing 800 kcal and 40 g of protein daily or a placebo that resembled the supplement in appearance but provided less than 40 kcal per diem. No supplement was given to the infants or children directly, so any nutritional intervention relating to the growth of the offspring was mediated by the mother prenatally or during lactation.

There were 108 pairs of siblings whose mothers received the calorie/protein supplement and 105 pairs of siblings whose mothers received the placebo. Correlations at birth in weight, length, head circumference, subscapular skinfold, and weight/length^3 (also called the ponderal index) between siblings in the placebo group were significant and all were near the value of 0.50. Siblings in the supplemented group had birth size correlations that were "... unusually low and often insignificant" (Mueller & Pollitt 1983, p. 11). The low correlations were due, presumably, to nutritional supplementation of the mother, which produced more favorable prenatal growth in the sibling exposed to the supplement. The differences in sibling correlations between the two groups virtually disappeared by age 2.5 years. Apparently, the maternal mediated effect of the high calorie supplement was limited to prenatal life and infancy. After weaning, the generally adverse nutritional environment of the village was a stronger influence on the growth of all children.

Longitudinal studies, such as the Fels Research Institute and Bacon Chow study, are rare due to the time and expense required to collect data of this type. A more common approach to familial correlation research is to calculate parent–child

correlations in stature. One such study used data from the INCAP "Four Village Study" of malnourished rural Guatemalan families (Martorell et al. 1977). Correlations between mid-parent height and child stature (or length) were obtained for children aged six months, one year, and then yearly up to age seven. The authors hypothesized that in this chronically malnourished sample, the stature of both the parents and the children would have been stunted. Furthermore, different degrees of malnutrition would have been experienced by different individuals. As a result, it was expected that the correlations between mid-parent stature and child stature for this sample would be lower than the values predicted from a simple genetic model, and lower than values from better nourished populations in developing countries. It was found, however, that correlations for the Guatemalan sample did not differ from samples from the United States or Northern Europe.

The researchers considered the notion that variability in stature in the Guatemalan sample is as much a product of genetic influences as it is in the developed nations. However, that notion was rejected because further analysis showed that socioeconomic and nutritional status were correlated across generations. That is, parents who had relatively better living conditions (housing, nutrition) when they were children were more likely to provide a better environment for their own children. This is predicted from cultural inheritance theory. The Guatemala research team reported that the environmental and genetic factors contributing to the parent–child correlations in stature were so interconnected that is was not possible to quantify the unique contributions of either. In other words, human growth is the product of a matrix of biocultural interactions.

Familial correlation and heritability estimates for stature in a West African population were calculated by Roberts and colleagues (1978). The sample studied included the people of two villages in The Gambia, where traditional subsistence agriculture and rural lifestyles were practiced. The authors found that correlations for stature between husbands and wives were low and not statistically significant, indicating that there was no assortative mating for height. Correlations between parents and offspring, and between full siblings, for this sample were lower than those found for European or North American samples of middle to upper socioeconomic status. Moreover, the correlations between full siblings were lower than correlations between parents and children. The heritability for stature was estimated to be about 0.56 (1 being a perfect heritability and 0 indicating no heritability). That heritability estimate is, of course, representative of all sources of variability ranging from the DNA to the cultural transmission.

To help put these African results in perspective, Byard et al. (1983) found, for a United States population, that sibling correlations were higher, generally, than parent–offspring correlations and that the heritability of stature was about 0.68. Roberts and colleagues suggested that their findings reflected a relatively larger environmental influence on stature than in the US or European studies. High rates of infant mortality (up to 50% of newborns died by age 5 years), malaria, droughts and food shortages, and other " ... rigours of the traditional way of life in West Africa ... " (p. 23) all influenced the growth of the villagers. The authors emphasized

Table 6.3 Parent–child correlations at age 16 and 23 years from the National Child Development Study of Great Britain (Lasker & Mascie-Taylor, 1996).

	16 years	23 years
Father–son	0.36	0.41
Father–daughter	0.43	0.41
Mother–son	0.41	0.47
Mother–daughter	0.47	0.46

that family members of different generations, and older and younger siblings, growing up under equally harsh or equally good environmental conditions, would tend to have higher correlations, while those growing up under dissimilar environments would tend to lower familial correlations and heritability estimates.

Similar findings are reported for a sample from rural West Bengal, India (Dasgupta et al. 1997). The sample included 504 individuals, 110 parent pairs (mother and father), 187 of their sons, and 133 of their daughters. The families were of middle economic status Hindu caste, all were farmers, and the mean per capita monthly household expenditure was Rs. 150, or about US$5. For a family with two adults and three children this would equal ~US$25/month. All the coefficients of correlation calculated were less than theoretical expectation of a simple genetic model. Correlations for weight were lower, generally, than those for height. This is expected since weight is known to have a stronger environmental determination than stature. The lowest stature correlations were for brother–brother and father–son, r = 0.14 and 0.17 respectively. The highest values were for the sister–sister and mid-parent–daughter correlation for stature, r = 0.48 and 0.45 respectively. In general, mother–child correlations were higher than father–child correlations. This had been found in several other studies and Dasgupta and colleagues ascribed it to the " . . . persistent effect of the intrauterine environment [and to] greater maternal care of children" (p. 8). Overall, the lower than expected correlation coefficients indicated that factors other than genetic inheritance accounted for the majority of height variation in this rural, low SES population.

Lasker and Mascie Taylor (1996) analyzed parent–child correlations in stature from the National Child Development Study of Great Britain. They calculated the correlations when the offspring were 16 and 23 years old. Their findings are given in Table 6.3. The researchers noted a positive correlation between stature and social class in this sample. Allowance for social class of the father lowered the parent–child correlations, but not by more than five percent in any case. Even so, a 5% reduction in the correlation values when adjusted for father's social class attested to the influence of nongenetic factors, which were not well controlled for in the National Child Development Study. The values of the parent–child correlations were consistently larger in this study than in the studies from Africa and India just discussed. One would expect this, as the standard of living in Britain is much higher than in these other regions. Yet, the correlations are still less than the theoretical value of

0.5 expected from a simple genetic model. Moreover, mother–offspring correlations are higher than father–offspring correlations. This is further evidence, it seems, of the persistent maternal effect due to the prenatal environment and the tendency of mothers to care for children more so than fathers.

Lasker and Mascie-Taylor tried to place their results in the context of parent–offspring correlation studies in general. They pointed out that the correlations reported in the literature, which range from about 0.01 to 0.52, are sample specific, reflecting different degrees of genetic variability within the sample, the effects of different environments, different sampling techniques, and different age ranges among the subjects. It is difficult to come to any concrete conclusions about the meaning of these correlations when the sample may be so ill defined. Even when the sample is very well defined, such as the Fels Research Institute Study, it may be unrepresentative of the larger population both for its ethnic makeup and the high degree of self-selection of the participants (Garn & Rohmann 1966). Family correlation studies, then, are like twin studies in terms of the serious methodological limitations. As with twin studies, great care must be exercised when interpreting the findings of these studies.

The Effects of Genetic Aberrations on Growth

It is usually difficult to separate the genomic, the SEPE, the nutritional, and other environmental influences on growth, development, and maturation. It is not desirable to do so as these form the network of extensive interacting variables that together contribute to the formation of the human phenotype. It is known, however, that genetic aberrations, such as DNA base mutations, may produce various kinds of abnormal growth and development. Understanding what happens when the genetic material is disrupted by these aberrations allows for a better understanding of the role of the genomics in growth and development.

An example is achondroplasia, an aberration that results in short stature due to impaired growth of the legs and arms (there are other growth consequences as well). In 1994 a French research team announced the discovery of the gene defect responsible for achondroplasia (Rousseau et al. 1994). The gene has been mapped to the short arm of chromosome four, and the DNA change is a point mutation that alters the amino acid makeup of a protein called fibroblast growth factor receptor-3 (FGFR-3, Bonaventure et al. 1996). To date, 97% of patients with achondroplasia have the identical DNA mutation. Subsequent research found that other mutations causing aberrations of FGFR-3 protein underlie several other clinically related disorders (e.g., thanatophoric dysplasia, type I and II, and hypochondroplasia), that are collectively called the “achondroplasia family” of disorders (Vajo et al. 2000, provide many photographs). About 1/15,000 live births in European populations are affected, making achondroplasia a frequent cause of very short stature. In non-European populations the occurrence drops to 1/40,000 live births. Why this variation exists is not known. It is known that the risk for achondroplasia rises with parental age.

Otherwise, the FGFR-3 mutation seems to occur at random and does not "run in the family." Why the genomic region for the FGFR-3 protein is so labile to mutation is not known. The high specificity of FGFR-3 mutations for disorders of skeletal growth demonstrates one pathway from DNA sequence to body size and shape, but this is a rare case in medical genetics. In the words of one research group, "These specific genotype-phenotype correlations in the FGFR disorders seem to be unprecedented in the study of human disease" (Vajo et al. 2000, p. 23).

Clinical geneticists group the genomic aberrations of growth into the categories of: (1) numerical chromosome aberrations; (2) structural chromosome aberrations; (3) copy number variations; (4) gene mutations; and (5) epigenetic factors (Mortier & Vanden Berghe 2012). The remainder of this section describes briefly each of these categories.

Numerical Chromosome Aberrations

Studies of people with unusual karyotypes (the number and type of chromosomes inherited by an individual) provide evidence for such genetic effects. Normal human karyotypes are 46,XY for males and 46,XX for females, 46 being the total number of chromosomes, with 23 from each parent, and X or Y being the types of sex chromosomes. Sometimes, during the process of meiosis there is nondisjunction of all the chromosomes and sex cells (sperm or ova) have fewer or greater numbers than 23 chromosomes. The lack of or addition of more than one chromosome is nonviable, meaning that even if fertilization occurs there cannot be development to a live-born infant. The addition of one chromosome can be viable, but damaging, and result in infants born with trisomy 13 (Patau syndrome), trisomy 18 (Edward syndrome), the 47,XXY karyotype (Klinefelter syndrome), and the most common aberration, trisomy 21 (Down syndrome). The only deletion of one chromosome that is viable is Turner syndrome, with the 45,X karyotype.

A classic study by Tanner and colleagues (1959) examined people with sex chromosome anomalies, including individuals with Klinefelter syndrome and Turner syndrome karyotypes. People with the 47,XXY condition are phenotypically males, and taller on average than normal 46,XY males. People with the 45,X condition are phenotypically female, and much shorter, on average, than normal 45,XX females. Tanner and colleagues found that the body proportions (e.g., the ratio of leg length to stature) and rate of skeletal development of 47,XXY boys was like that of normal 46, XY boys. They also found that the rate of skeletal development of 45,X girls, up to puberty, was like that of normal 46,XX girls. Consequently, the authors concluded that genetic factors on the Y chromosome produce the male pattern of growth in body proportions and skeletal development.

An X chromosome effect on growth was proposed by Garn and Rohmann (1962). They used a longitudinal sample of hand-wrist radiographs and dental radiographs to study ossification rate (number of bony centers present), ossification timing (age at the appearance of a center), and tooth **calcification** in siblings. The sample numbered

318 brother–brother, sister–sister, and brother–sister pairs. Garn and Rohmann hypothesized that rates of skeletal and tooth development are genetically controlled and some of these genes are linked to the sex chromosomes. They also proposed that pairs of sisters, who share the same paternal X chromosome, should have greater concordance in rates of development than pairs of brothers, who have only a 50% chance of sharing the same maternal X chromosome, or brother–sister pairs, who share no paternal sex chromosomes. It was found that the correlation between pairs of sisters in skeletal and dental development (averaging about 0.52) was significantly greater than the correlation between pairs of brothers or brother–sister pairs (averaging about 0.35). Garn and Rohmann interpreted these correlations as evidence for X chromosome influence on rates of development.

The growth of Turner syndrome girls is also influenced by family environment factors. In one study, Varrela and colleagues (1984) found that the 45,X women and a comparative sample of 46,XX women were taller and larger in several skeletal dimensions than samples of 45,X and 46,XX women from earlier studies. The authors ascribed the differences to improvements in living conditions since the time of the earlier studies, including higher socioeconomic status of the families, better health care and nutritional status, and smaller family size, all of which are environmental variables that are known to be associated with increased growth.

The effects of extra sex chromosomes on growth have been investigated. Shirley Ratcliffe and her colleagues concentrated on individuals with 47,XXX, 47,XYY, and 47,XXY karyotypes. In a review of her research Ratcliffe (1995) reported that an extra Y chromosome (47,XYY) had no detectable effect on prenatal growth, as measured by weight, length, or head circumference at birth. In contrast, an extra X chromosome resulted in smaller size, and the 47,XXX karyotype significantly reduced all three birth dimensions. During infancy and childhood, 47,XYY and 47,XXY boys grew faster than 46,XY boys, and during adolescence the 47,XYY boys had a greater peak height velocity than 46,XY boys. Boys with either an extra X or Y chromosome end up taller as adults than "normal" 46,XYs, and virtually all of the difference in stature was due to additional leg growth. 47,XXX girls grew less than 46,XX girls at all ages and ended up significantly shorter as adults. Slower growth in sitting height (length of trunk and head) of the 47,XXX girls was largely responsible for the stature difference. Both 47,XXY boys and 47,XXX girls had significantly reduced head circumference at birth and at all later ages. Head circumference reflects growth of the brain, and Ratcliffe found a significant positive correlation between head circumference and scores on the Weschler Intelligence Scale (an "IQ" test) when the subjects of her study were 7–14 years old – those subjects with a smaller head circumference also had a lower "IQ" compared with subjects with larger head circumferences. In summary, Ratcliffe found that a supernumerary Y chromosome increased growth in leg length and stature. An extra X chromosome reduced growth of the brain during fetal life, with no catch-up after birth. In girls an extra X also reduced sitting height and stature.

Structural Chromosome Aberrations

Garn and Rohmann's speculation about X chromosome influence on rates of development was prescient. Today it is known that deletions of one entire X chromosome or of the distal segment of the short arm of an X chromosome, designated the Xp-arm, results in insufficiency of the *short stature homeobox* (called SHOX haploinsufficiency) and usually results in short stature, skeletal abnormalities, and hearing impairments (Oliveira & Alves 2011). The importance of SHOX in growth was mentioned in several previous chapters. Deletion or structural rearrangement of the order of DNA bases of a chromosome often disturbs healthy patterns of growth, development, and maturation. There are too many of these structural aberrations to list here. Interested readers can search for more information online.[5]

Copy Number Variations

A twenty-first-century technology called molecular karyotyping can detect small deletions or duplications of DNA sequences. Copy number variations are associated with several neuropsychiatric conditions including autism and schizophrenia, although there is no evidence of direct causation. In the case of the early adult-onset brain killing condition Huntington's disease there is better evidence that more than 36 multiple repeats of the DNA base sequence CAG (cytosine-adenine-guanine) elevates risk. Multiple duplications of X chromosome region called Xq26 is one cause of pituitary gigantism (Trivellin et al. 2014). Microdeletions of DNA on chromosome 22 cause velocardiofacial syndrome, the most common syndrome associated with cleft palate. Most copy number variation, whether deletions or duplications, seem to have no effect on expected phenotypes. These small changes in DNA content, however, may contribute to the normal range of variation in body size, body shape, and body composition. This may be via direct effects, but more likely via the complex networks of extensive interacting molecular variables.

Gene Mutations

The loss or addition of even one amino acid coding sequence of DNA can have major consequences on human biology as described already for achondroplasia and other FGFR-3 mutations. The Y chromosome carries a DNA region called SRY (an acronym for "sex-determining region Y"), which is known to be involved in formation of the male testis from the undifferentiated embryo gonad. SRY does not do this directly, rather it signals another DNA region located on chromosome 17 called SOX-9, a **transcription factor** for embryonic and fetal skeletal development and sex determination. Some pathologies of sexual and skeletal development are linked to mutations in the SOX-9 gene. The skeletal disorders are of the type related to inadequate cell proliferation. Mutations of other SOX transcription factors cause disorder, such as

[5] www.sciencedirect.com/topics/medicine-and-dentistry/structural-chromosome-aberration

SOX-3 on the X chromosome, associated with X-linked cognitive disability and with growth hormone deficiency, and SOX-5 on chromosome 12, which regulates cell proliferation at the growth plate (the basic biology of the growth plate was reviewed in Chapters 2 and 3). These SOX transcription factors interact within a complex network of intracellular and intercellular signaling that is responsible for greater or lesser amounts, rates, and timing of growth (Baron et al. 2015). The study of mutations of cell signaling function delineates molecular pathways and phenotypic effects. **Cell signaling** is one of the most active areas of research in molecular biology and new research findings are published weekly. One review of research relating to human evolutionary medicine is by Diana Le Duc and Torsten Schöneberg (2019).

Epigenetic Factors

When Boas used the word "plasticity" (Boas 1912, p. 557) to describe ranges of variation in the size and shape of people he opened the way toward research into the epigenetic basis for human growth, development, and maturation. **Epigenetic** means literally "above the level of the gene." In practice, epigenetic refers to changes in DNA expression caused by mechanisms other than changes in the underlying DNA sequence. Conrad H. Waddington (1905–1975) formalized the principles of epigenetics with the publication of *The Strategy of Genes* (Waddington 1957). Waddington wrote about the epigenetic landscape, which is a concept representing embryonic growth and development and the various developmental pathways a cell might take toward differentiation. For a human being the epigenetic landscape describes the range of possible pathways for developmental plasticity of the fertilized ovum and the developmental journey it takes to become the tissues, the organs, and the total physical, behavioral, social, and emotional phenotype of the person.

Different pathways are defined by epigenetic mechanisms, such as DNA methylation, histone acetylation, and micro RNA interference (Figure 6.7), which can affect gene activation and inactivation. Methylation, for example, inactivates or represses gene expression. Epigenetic mechanisms may be activated by exposure to temperature extremes, exposure to disease, excess or lack of dietary factors, and many behavioral practices including physical activity, smoking, and alcohol consumption. More than 100 chemical marks have been identified on DNA and RNA molecules. Segars and Aagaard-Tillery (2009 p. 349) wrote that epigenetic mechanisms " ... are increasingly understood to have a profound effect in altering an individual's appearance, transmission of a specific congenital abnormality ('birth defect'), and even one's lifetime risk of common diseases such as obesity and cancer." The epigenome and its impact on DNA expression explain, in part, how cells with identical DNA derived from a single fertilization can differentiate into the multitude of specialized types that make up different tissues of each person. In the case of twins, the accumulation of different epigenetic marks after fertilization will result in a range of DNA/RNA expression and much potential phenotypic variability, even for MZ twins.

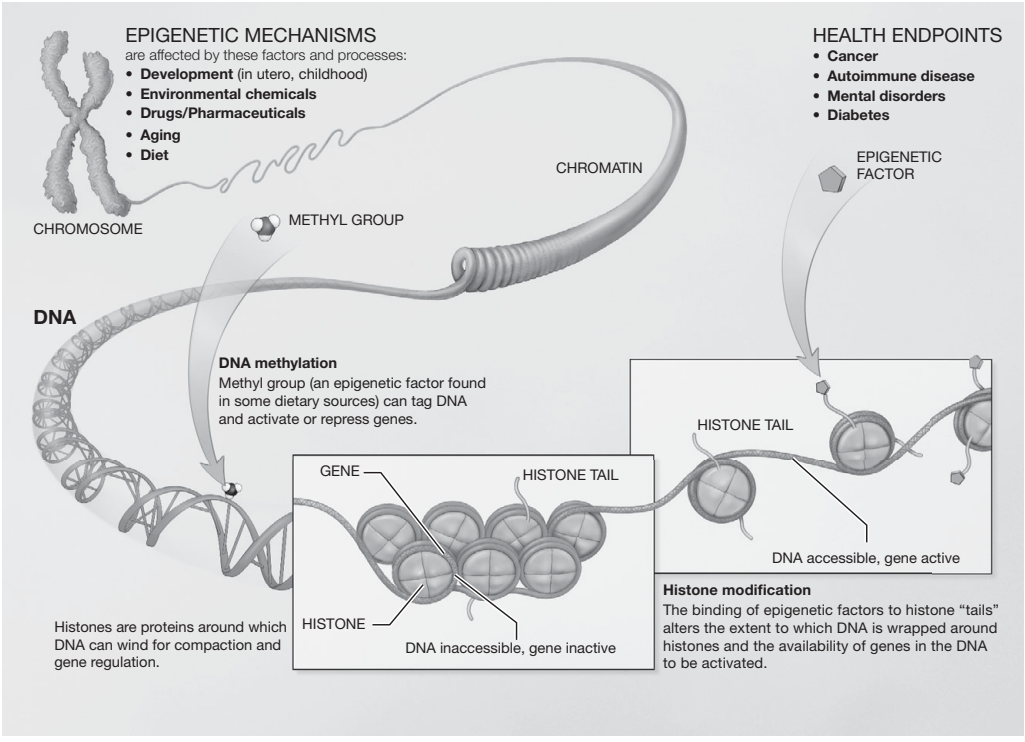


Figure 6.7 Epigenetic mechanisms are affected by several factors and processes including development in utero and in childhood, environmental chemicals, drugs and pharmaceuticals, aging, and diet. DNA methylation is what occurs when methyl groups, an epigenetic factor found in some dietary sources, can tag DNA and activate or repress genes. Histones are proteins around which DNA can wind for compaction and gene regulation. Histone modification occurs when the binding of epigenetic factors to histone "tails" alters the extent to which DNA is wrapped around histones and the availability of genes in the DNA to be activated. All of these factors and processes can have an effect on people's health and influence their health possibly resulting in cancer, autoimmune disease, mental disorders, or diabetes among other illnesses. From the United States National Institutes of Health, public domain (https://commonfund.nih.gov/epigenomics/figure). (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Epigenetic expression in the phenotype may be a heritable change in biology or behavior, but a change that does not alter the DNA sequence of chemical bases. In this sense, epigenetic biology is a departure from the traditional genetic dogma of information flow:

$$DNA \rightarrow amino\ acid \rightarrow polypeptide\ chain \rightarrow protein.$$

The flow of information in epigenetic biology may begin with a social factor, such as the decision of families to migrate from a poorer country to a richer country or the choice by a woman to deliver an infant via caesarian section (c-section). In the first case, second generation Bangladeshi women, that is the daughters of women who migrated to the United Kingdom, have higher levels of salivary progesterone and

higher ovarian function than first generation migrants. The difference in progesterone level is in part due to greater methylation of the progesterone receptor protein in the first-generation migrants, who grew up in Bangladesh (O'Connor et al. 2009). Why this is so is not known, but a consequence of elevated progesterone levels is greater risk for breast cancer in the second-generation women (Núñez-De La Mora et al. 2008). In the second case, women giving birth by c-section deliver infants with greater DNA methylation in general (Schlinzig et al. 2009). Why this is so, and how it influences later health is unknown, but infants delivered by c-section have an " . . . increased risk for allergy, diabetes and leukaemia" (Schlinzig et al. 2009, p. 1096). Several studies reported an increased risk of overweight and obesity in individuals who were born by c-section compared to those born vaginally. Research by our team found that 6–8-year-old Maya girls from Merida, Mexico delivered by c-section had greater amounts body fat than girls delivered vaginally. There were no fatness differences for boys (Azcorra et al. 2019).

These examples and many more require biologists to rethink the traditional "gene-to-protein" dogma. There is considerable environmental control of genomic programming and DNA expression. Several nutrients, such as vitamins A, C, niacin, and D, are known to regulate DNA activity and be related to diseases such as diabetes, atherosclerosis, and cancer. A socioeconomic factor such as poverty can influence the availability of vitamin D_3 due to limited food choices. Vitamin D_3 is found in a small number of foods, such as expensive oily fish such as salmon, in liver, and in eggs. Poor families may not be able to afford to eat these foods on a regular basis. Low socioeconomic status may also lead to a lack of exposure to sunlight due to the need to work at low-paid indoor jobs. Humans get most of their vitamin D_3 from sunlight (ultraviolet radiation) striking the skin and converting cholesterol-based substances into precursors of vitamin D_3.

In this case, the flow of epigenetic information is as follows:

Social-economic-political forces producing poverty → inability to purchase vitamin D_3 containing foods/low sunlight exposure → low bioavailability of vitamin D_3 → low transactivation of DNA expression → low amino acid production → insufficient protein → possible harm to health.

An important human example is risk for the disease multiple sclerosis (MS). Many studies show that people living at northern latitudes, with low exposure to sunlight, low vitamin D intake, and with a specific genetic variant of the Major Histocompatibility Complex (MHC) on chromosome 6, are at greater risk to MS (Ramagopalan & Ebers 2009). People with the same MHC genetic variant, but with adequate vitamin D bioavailability have significantly lower risk for MS.

Other nutrients such as methionine, vitamins B6, B12, and folate are known to influence DNA methylation, and the availability of these nutrients during fetal development may influence susceptibility to complex diseases, such as diabetes and obesity. Via this nutrient route there is a connection between epigenetic events and intergenerational effects, such as the intergenerational persistence of lower birth weight discussed in Chapter 2.

Another epigenetic mechanism is genomic imprinting (also called parental imprinting), a process that restricts gene expression to only the allele inherited from the mother or the father. Some human examples of the deleterious effects of genomic imprinting are the congenital conditions Prader-Willi and Angelman syndromes. Remarkably, both are metabolic disorders due to the same DNA deletions in chromosome 15 (Angulo et al. 2015). But, whereas the features of Prader-Willi syndrome are short stature, mental retardation, poor muscle tone, hyperphagia (overeating), and obesity those of Angleman syndrome are developmental delay, lack of speech, seizures, and walking and balance disorders.[6] The phenotypic expression of these two disorders comes from parental imprinting: Aberrant paternal alleles produce Prader-Willi syndrome and aberrant maternal alleles produce Angelman syndrome. It is important to understand that the phenotypes of these two syndromes arise from the same DNA sequence, modified by two different epigenetic pathways of gene expression. More detail on these syndromes and other aspects of human epigenetics may be found at an educational website on the epigenetics of human development.[7]

Endocrinology of Growth

Hormones are organic substances synthesized in specific body tissues, often called endocrine glands, but many cells throughout the body produce hormones and related substances. Cells and tissues may secrete their hormones into the bloodstream, where they circulate to specific, and distant, sites of action. An example is the hormone cholecalciferol, or vitamin D_3, which begins its synthesis in the deep layers of the skin in response to ultraviolet light from the sun or artificial sources. It travels through the bloodstream to the liver and kidneys, where its biological activity is enhanced, and then to its two primary sites of action: (1) the intestine, where it promotes calcium absorption and (2) bone, where it regulates skeletal metabolism and bone growth. These are just the "classic" actions. There are significant effects on immune responses and on mental health (e.g., depression, bipolar disorder, and schizophrenia) and cognition (Dursun & Durson 2010). The "old" pediatricians of the late nineteenth and early twentieth century knew about these effects and prescribed sun lamp treatments (exposure to artificial ultraviolet light) to improve growth, health, and mood in the wintertime of Europe and North America (Hönigsmann 2013; Nylin 1929).

There are several major hormones with an effect on growth, development, and maturation and these are discussed here. In addition, there are groups of substances known as growth factors, signaling factors, the epigenetic enhancers and repressors, and others that have effects on growth, both independently and interactively with each other and with hormones. Growth factors are those hormones and their "downstream" products that are synthesized by specific cells within a wide variety of body tissues which have specific effects on growth, development, and maturation.

[6] www.angelman.org
[7] https://embryology.med.unsw.edu.au/embryology/index.php/Molecular_Development_-_Epigenetics

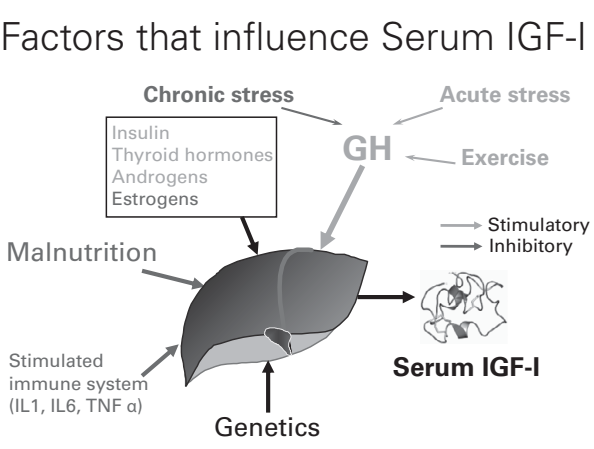


Figure 6.8 Some of the variables that influence levels of insulin-like growth factor-1 in blood serum. Variables in the color red, such as chronic stress, inhibit IGF-1 and variables in the color green stimulate IGF-1. This original figure was prepared by Professor Werner Blum and published in Bogin et al. (2015). It is reprinted here with kind permission of Professor Blum. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

For instance, human liver and **fibroblast cells** produce substances known as the insulin-like growth factors (IGFs), which promote cell division in bone, muscle, and other tissue. IGF synthesis can be stimulated by growth hormone (GH), which is synthesized in the pituitary gland, and both GH and IGFs may need to be present simultaneously at a site of action to have an optimal influence on growth (Blum et al. 2011, Figure 6.8). The IGFs are associated with several binding proteins (IGFBPs). The binding proteins are required to transport the IGFs in the bloodstream to their tissue and cellular sites of action. These IGFBPs may also work independently to regulate cellular growth and other bodily functions. More details of GH, IGFs, and related growth factors are given later in this chapter.

Underwood and colleagues (1986) described how growth factors may be carried by the circulation to their sites of action, the classic *endocrine signaling*, may act directly on the cells that synthesize them, an *autocrine signaling*, or may affect nearby cells in the same tissue, a *paracrine signaling*. Another type of chemical communication is *gap junction signaling* (also called *juxtacrine signaling*) between adjacent cells. These four modes of action are illustrated in Figure 6.9. There are other modes of communication, including *intracrine signaling*, whereby a hormone may act on the nucleus of the cell which manufactures the hormone and *matricrine signaling*, where there is chemical communication between cells and their extracellular matrix.

Some hormones may be peptides, that is protein molecules such as GH and IGFs, manufactured by the transcription of DNA to RNA and then the construction of one or more polypeptide chains of amino acids. Others are steroid hormones, generally manufactured from cholesterol, such as the sex hormones testosterone, progesterone, and estradiol. As already discussed, DNA and RNA do not act directly on the

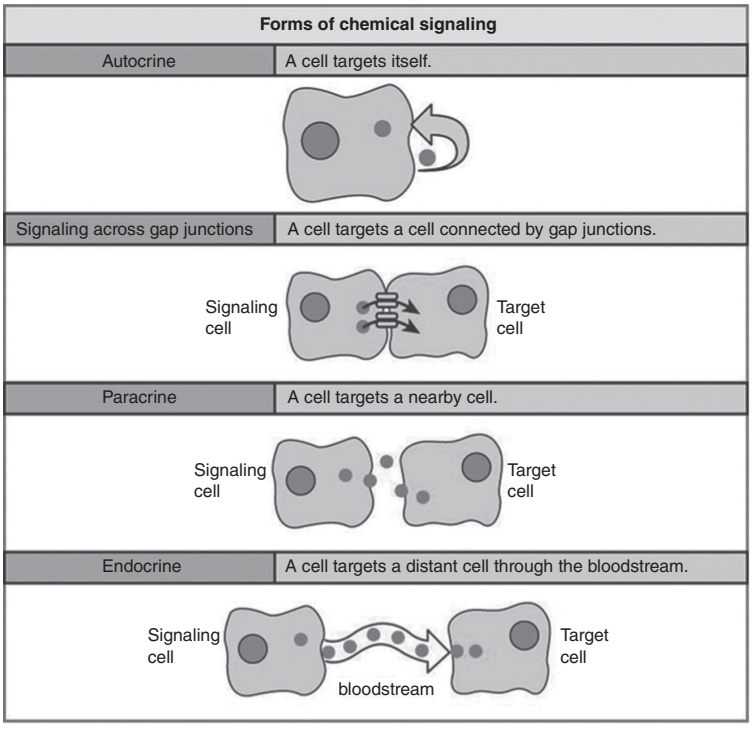


Figure 6.9 Four modes of chemical signaling found in multicellular organisms: paracrine signaling, endocrine signaling, autocrine signaling, and direct signaling across gap junctions. A cell may target itself (autocrine signaling), a cell connected by gap junctions, a nearby cell (paracrine signaling), or a distant cell (endocrine signaling). Paracrine signaling acts on nearby cells, endocrine signaling uses the circulatory system to transport hormones, and autocrine signaling acts on the signaling cell. Signaling via gap junctions involves signaling molecules moving directly between adjacent cells. The main difference between the different categories of signaling is the distance that the signal travels through the organism to reach the target cell. Not all cells are affected by the same signals. Provided by: OpenStax CNX. Located at: http://cnx.org/contents/185cbf87-c72e-48f5-b51e-f14f21b5eabd@10.8. Creative Commons Attribution 4.0 License. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

organism that carries them, rather these genomic constituents provide part of the template to manufacture substances such as hormones that have a biological action. It is important to understand that the production and secretion of a hormone are only part of the process by which endocrine substances affect the body. The target tissues for a hormone must be sensitive to its presence. Tissue sensitivity may be influenced by several factors, including aspects of the environment in which the organism lives, the sex and age of an individual, binding proteins that are needed to carry the hormone to its site of action, the presence of biochemical receptors at the tissue level that recognize the hormone, and the production of "secondary messengers," so called because some hormones do not cross cell membranes and require intermediary

substances to carry their "message" into the target cells. Here we can see how a hugely complicated matrix of factors influences hormone action.

Hormones are part of a larger endocrine system which regulates and coordinates critical developmental processes. The endocrine system is integrated across several other systems, such as the central nervous system and digestive system, and each of these is influenced by nutritional status and infections. Therefore, the endocrine system and its biochemical products provide a mechanism by which "real-time" information about a body's health is communicated to the brain and processed by its regulatory center in the hypothalamus, through which growth is affected accordingly. The hypothalamus plays a central role in many of the hormone feedback loops involved in growth and development. Due to these multiple systems of regulation, growth, development, and maturation may be disrupted in individuals with nutritional deficiencies, disease burden, and hormone abnormalities. Examples are thyroid hormone deficiency and GH insufficiency or insensitivity of cells to available GH. The severity of the growth disruption and the potential for recovery to "normal" growth patterns, or to achieve a particular size, shape, or body composition, depend on many variables, from political systems that create social and economic inequality, to family harmony or discord, to mutations of SNPs.

The following account is limited to the more elementary aspects of the endocrinology of human growth. The reader should be aware that there are additional complexities of the endocrine system. Moreover, endocrinology is a very active area of research, and our current state of knowledge may be subject to substantial revision.

A Hormonal Model of Human Growth

Before reviewing further some of the major growth hormones it is helpful to have a conceptual understanding of the endocrinology of the pattern of human growth. The first formal model to tie hormones together with human growth throughout its different stages was developed by Johan Karlberg (1987, 1989) who proposed that human growth could best be understood as proceeding through three distinct yet overlapping trajectories, each of which had specific hormonal underpinnings (Figure 6.10). This model specified differences in the infancy, childhood, and puberty (ICP) phases of growth. The model also specified that growth of the fetus is driven by hormones that are dependent on, and affected by, maternal nutritional status and after birth by infant nutritional status. Karlberg proposed that the infancy growth stage began, in fact, in mid-gestation, and was driven by insulin and IGF-2 of placental origin. After birth the newborn's own production of insulin and IGF-2, as well as that supplied via breast-feeding, took over. During the early part of infancy IGF-1 becomes more important than IGF-2. The infant's protein intake and its energy balance between nutrition intake and expenditure via physical activity are key regulators of IGF-1 production (Figure 6.8, (Larnkjær et al. 2012). For example, compared to breastfed infants, IGF-1 concentrations are higher in formula-fed infants at 3 months of age because infant formula has higher protein and energy content than breast milk. Formula fed infants are also longer, heavier, and have

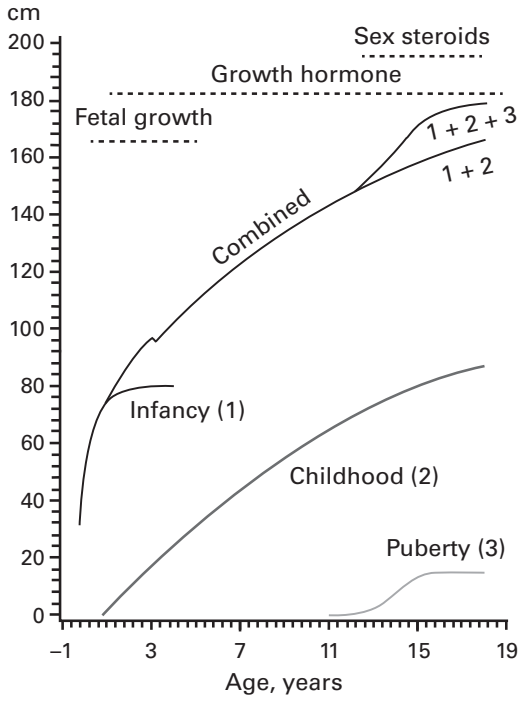


Figure 6.10 The Karlberg Infancy-Childhood-Puberty model of growth in height. See text for explanation. (From Karlberg 1987)

higher BMI values by 1 year of age (Ong et al. 2009). Data from several population studies indicate that IGF-1 appears to increase from birth throughout the first few months of life, followed by a gradual decrease until approximately 9 months of age, at which point levels again increase (Larnkjaer et al. 2009).

At about 9–12 months following birth, Karlberg proposed that the main hormonal regulation of growth switched over from external, nutritional control to endogenous rhythmic hormone secretion. The evidence for increased IGF-1 secretion at about 9 months of age overlaps with this suggestion, indicating a more constant regulatory process governing IGF-1 secretion. Karlberg suggested that the childhood stage of growth was best defined as starting when endogenous GH production takes over as the main driver of somatic growth. Karlberg also proposed that the GH regulation of growth is further modified during puberty (Karlberg's "puberty" stage is described in this book as puberty and adolescence) by both the independent contributions of sex steroids and their additive effect on GH pulsatile secretion.

The full duration of the childhood stage of Karlberg's model includes several of the stages previously described in this book: approximately the last 2 years of infancy plus all of childhood and the juvenile stages. The differences in definition of the growth stages is mostly semantic. Karlberg and I agree that the changes in amount and rate of growth over time are rooted in the physiological regulation of each growth period. Both the stage system of this book and Karlberg's model provide an

understanding of how individual growth strategies can be best matched to available energy and nutrient supply and other environmental cues. Growth researchers have incorporated both the Karlberg and Bogin stage systems of growth and development (Bernstein 2010; Hochberg 2011). Pediatric clinicians have applied these growth systems and models to account for height variation; for example, a delayed infancy to childhood transition has been proposed to feed forward to reduced height, while an accelerated transition is a risk for juvenile and later life overweight and obesity (Hochberg & Albertsson-Wikland 2008).

The Major Hormones of Human Growth and Maturation

The actions and interactions of hormones and growth factors provide a system of fine control for the regulation of growth and development. A central feature of this system is the hypothalamic regulation of the pituitary gland. Figure 6.11 illustrates the location of the hypothalamus and pituitary at the base of the brain. Blood vessels

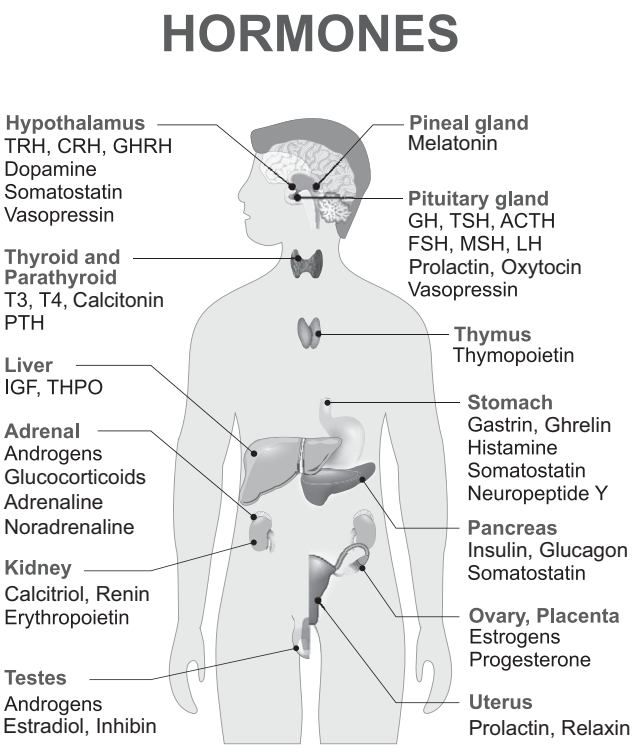


Figure 6.11 Diagram of the location of the hypothalamus, pineal, and pituitary within the brain, and a schematic illustration of some of the target organs and tissues of some of their hormones. Abbreviations are: TRH, thyrotropin-releasing hormone; CRH, corticotropin-releasing hormone; GHRH, growth hormone-releasing hormone; GH, growth hormone; TSH, thyroid stimulating hormone; ACTH, adrenocorticotropic hormone; FSH, follicle stimulating hormone; LH, luteinizing hormone; T3, thyroxin; T4, triiodothyronine; PTH, parathyroid hormone; IGF, insulin-like growth factor; THPO, thrombopoietin. Credit ttsz / iStock / Getty Images Plus.

and nerves directly connect the hypothalamus to the anterior pituitary gland and allow for neurochemical communication from the hypothalamus to the pituitary. The hormones of the hypothalamus stimulate or inhibit the release of the pituitary hormones, and these are released into the general circulation where they act on specific target tissues throughout the body. Figure 6.11 also outlines the major hormones produced and secreted by each endocrine organ and indicates the target tissues of the pituitary hormones.

Several pituitary hormones, such as prolactin, melanocyte-stimulating hormone, vasopressin, oxytocin, and their hypothalamic releasing or inhibiting factors are necessary for normal metabolic activity, the maintenance of the placenta and fetus, birth, and other life-sustaining processes. In a broad sense, therefore, these hormones are essential for growth, but a detailed discussion of them is outside the scope of this book. The following discussion is confined to those hormones involved most directly in growth, development, and maturation of the human body.

Thyroid Hormones

Thyrotropin releasing hormone (TRH), secreted by the hypothalamus, stimulates the release from the pituitary of thyroid stimulating hormone (TSH). The pituitary TSH acts on the thyroid gland to promote the release of two metabolically active hormones, thyroxin and triiodothyronine. A negative feedback relationship controls the release of TSH and the two thyroid hormones into the bloodstream. Negative feedback is a control mechanism that involves self-regulation of a mechanical or biological system inhibition. A mechanical example is temperature regulation by a thermostat. A thermostat is a device, as in a home-heating system, a refrigerator, or an air conditioner, that automatically responds to temperature changes and activates switches controlling the equipment. In a heating system, a drop in temperature results in the closing of a switch that turns the heater on. When the temperature reaches the setting on the thermostat the switch opens, and the heater is turned off. Rising temperature, then, inhibits the production of more heat. Negative feedback works in an analogous manner in biological systems, such as when rising levels of one of the thyroid hormones sends a signal back to the hypothalamus or the pituitary to terminate the release of TRH or TSH.

In Figure 6.12 three negative feedback loops are illustrated showing how thyroid activity is controlled along the hypothalamic-pituitary-thyroid (HPT) axis. This model may be applied, generally, to the other hypothalamic-pituitary target tissue hormones. The ultrashort feedback loop involves **autocrine action** of TRH on the hypothalamus. In this case, rising levels of TRH within the hypothalamus may suppress the activity of TRH secretory cells directly. A second form of **feedback control** involves rising systemic levels of pituitary TSH, which may decrease the release of TRH through a short feedback loop, another example of **paracrine action**. Finally, an increase in the blood levels of thyroxin and triiodothyronine may suppress TRH secretion through a long feedback loop, based on **endocrine action**. All three avenues of feedback control may work simultaneously to "fine-tune" the level of thyroid hormones in the bloodstream.

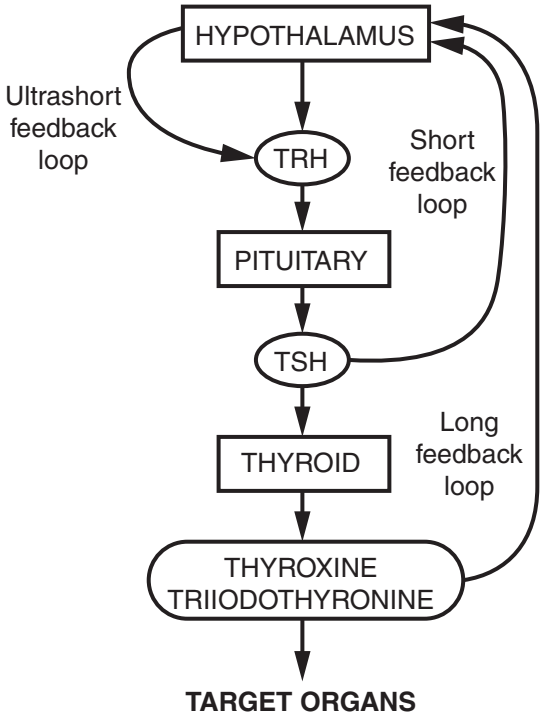


Figure 6.12 Feedback circuits for the control of the hypothalamic-pituitary-thyroid system (original figure).

A fine level of control is needed, since thyroxin and triiodothyronine have powerful metabolic actions. Thyroid hormones are needed for normal growth in stature, the development of normal body proportions, formation of bone from cartilage, and formation of the teeth. A deficiency of these hormones (hypothyroidism) during infancy and childhood results in growth retardation and mental impairment, and in the extreme case, the child suffers from a form of very short stature and cognitive impairment called cretinism. Pituitary TSH also acts directly as growth factor. This is the reason for the enlargement of thyroid tissue in the absence of iodine. Sensing a low level of thyroid hormones, the pituitary gland produces more TSH to stimulate the thyroid, which then causes the gland to enlarge.

Thyroid hormones seem to have an important role in the maturation of brain enzyme systems and myelination, the covering of nerve fibers with a fatty insulation which speeds up the transmission of nerve impulses. These actions account, in part, for why hypothyroidism in infancy results in mental impairment throughout life. The powerful and pervasive effects of thyroid hormones serve to coordinate growth and development. Evolutionary anthropologists speculate that alterations in the regulation of thyroid hormone production, both the life cycle timing and the quantity, may have played a central role in hominin evolution, " ... including the convergent evolution of bipedalism in early hominids, species-specific sexual dimorphism,

coordinated changes in morphology, brain function and gut length over time in hominids, cold adaptation in *Homo neanderthalensis*, the possible independent evolution of *H. sapiens* in Asia, and regional adaptation of hominid populations" (Crockford 2003, p. 105).

Sizonenko and Aubert (1986) reported that thyroid hormones are detected in the human fetus at 78 days of age. Both thyroxine (T4) and triiodothyronine (T3) are essential for normal growth and development of the fetus (Forhead & Fowden 2014). Fetal serum levels rise, generally, throughout the prenatal period. By two weeks postpartum, the infant reaches adult levels of serum thyroid hormone activity. Thyroid hormones have a direct effect on protein synthesis and cell division, and during the pre- and postnatal periods and indirect effects by working along with the catecholamines, GH, and the IGFs to promote growth. "By regulating tissue accretion and differentiation near term, fetal thyroid hormones ensure activation of physiological processes essential for survival at birth such as pulmonary gas exchange, thermogenesis, hepatic glucogenesis, and cardiac adaptations" (Forhead & Fowden 2014, p. R87).

Adult-onset hypothyroidism (a deficiency of the thyroid hormones) is associated with slower metabolic rate, resulting in a decreased activity of involuntary muscles, including heart and gut muscles, in voluntary muscle weakness, and in weight gain. Hyperthyroidism (an excess of the thyroid hormones) speeds up metabolic activity and results in fast heart rate, nervousness, increased appetite, and weight loss.

Gonadal Hormones

The role of the gonadal hormones, also called sex steroids, in human growth and pubertal maturation and human adolescence was discussed in some detail in chapters 2 and 4. An overview of the pituitary-hypothalamic-gonadal (HPG) axis is shown in Figure 6.13 and described in the legend for that figure. During fetal life and early infancy, the hypothalamus produces relatively high levels of gonadotropin-releasing hormone (GnRH). This hormone causes the release of luteinizing hormone (LH) and follicle stimulating hormone (FSH) from the pituitary gland, and these hormones stimulate the ovaries or testes to secrete their estrogen or androgen hormones. The latter promote body growth, regulate reproduction, and regulate reproductive behaviors.

LH and FSH were first discovered to have an influence on the maturation of the female reproductive system, hence their names are derived from ovarian functions. Nonetheless, these same hormones have a significant effect on the male reproductive system as well as the secretion of hormones from the testes that have an influence on growth and maturation. In women, FSH and LH stimulate the growth of the ovaries and the release of ovarian hormones. The ovarian hormones are called, collectively, the estrogens. In men, FSH promotes the development of the seminiferous tubules and initiates the production of spermatozoa. LH stimulates secretion of hormones from the testes, collectively called the androgens. Androgens are required to complete the formation of mature spermatozoa and these hormones also have an influence on

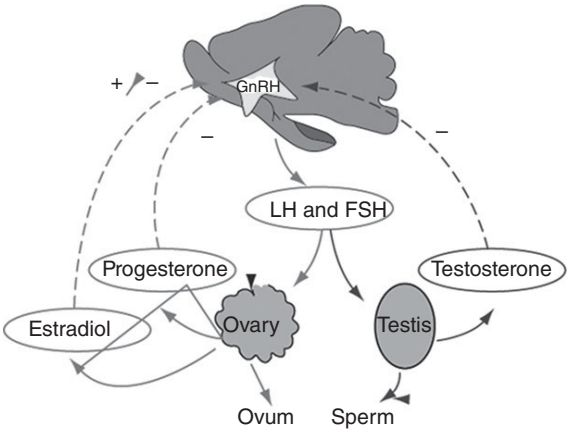


Figure 6.13 The hypothalamic-pituitary-gonadal (HPG) axis. This simplified cartoon indicates the principle tissues and their connections, but does not include the more recently identified gonadotropin-inhibiting hormone (GnIH). The HPG axis is composed of the hypothalamus and its neural connections with the rest of the brain, the pituitary, and the testis (male) or ovary (female). The anterior hypothalamus is responsible for the synthesis of gonadotropin-releasing hormone (GnRH). GnRH reaches the anterior pituitary via neurons and portal veins and stimulates the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) into the general circulation. LH and FSH bind to receptors in the ovary and testis and regulate gonadal function by stimulating sex steroid production and gametogenesis (production of ova and sperm). In the male, LH causes testosterone to be produced from the Leydig cells of the testes. LH in combination with FSH is required for maturation of spermatozoa. FSH stimulates testicular growth and increases production of androgen-binding protein by Sertoli cells. Androgen-binding protein concentrates testosterone near the sperm, enabling normal spermatogenesis. In the female, LH stimulates ovarian production of estrogen and progesterone. An LH surge midway in the cycle causes ovulation, and sustained LH secretion stimulates the corpus luteum to produce progesterone. FSH exerts primary control over development of the ovarian follicle, and FSH and LH are responsible for follicular secretion of estrogen. The solid lines indicate the stimulatory cascade from hypothalamus, to pituitary, to gonads. The broken lines and "–" symbols indicate the inhibitory feedback loops. The "+/-" symbols of the estradiol feedback loop indicate the variation of inhibitory/stimulatory feedback during the menstrual cycle. (With permission of Prof. C. Rivier, unpublished)

the growth of bone, muscle, fat, and other tissues. In men the serum levels of androgens or estrogens regulate the secretion of GnRH, LH, and FSH through a, generally, negative feedback relationship (like that for the thyroid hormones). In women, negative feedback regulates the levels of androgen production while serum estrogen levels exert a positive feedback influence on the hypothalamus and pituitary. Positive feedback works to increase or amplify the levels of activity of a system. In women, positive feedback between the ovaries, the hypothalamus, and the pituitary results in the menstrual cycle. During the pre-ovulatory phase of the menstrual cycle GnRH, LH, and FSH stimulate the ovary to release estrogens, which in turn further stimulate the hypothalamus and pituitary to secrete more GnRH, LH, and

FSH. The progressive amplification of these hormones in the bloodstream leads to an LH surge at mid-cycle that results in ovulation. Negative feedback is restored during the post-ovulatory phase, which ends with menstruation. Then the cycle repeats.

In the year 2000 gonadotropin-inhibiting hormone (GnIH) was discovered. GnIH is the antagonist of GnRH and inhibits gonadotropin synthesis and release. Both GnRH and GnIH are evolutionarily old and highly conserved among vertebrates from agnathans (jawless fish) to humans. These two hypothalamic hormones operate within a complex network of endocrine factors, receptors, and signaling agents to regulate the biology and behaviors for reproduction across vertebrates. This network includes both the endocrine and nervous systems, and these two systems work together so closely that researchers call them the neuroendocrine system. A recommended review of the neuroendocrine system with special emphasis on GnRH and GnIH is by Tsutsui and Ubuka (2018).

One special function of GnIH is to mediate interactions between the HPT-HPG axes during puberty and adolescence. Another puberty factor already discussed is kisspeptin, which was discovered in mammals following the discovery of GnIH. GnIH and kisspeptin are new members of a family of peptides called the RFamides. In the hypothalamus, GnIH neurons connect to kisspeptin neurons. Kisspeptin acts to up-regulate and GnIH acts to down-regulate the reproductive system. Tsutsui and Ubuka (2018, p. 11) suspect that "Further studies may reveal unknown interactions among GnIH, GnRH and kisspeptin." New findings about the stimulation and inhibition of most hormones is published weekly and interested readers should look for open access review articles on PUBMED and other websites.

Gonadotropins, estrogens, and androgens have major influences on human development at all life stages. LH and FSH may be detected in fetal pituitary tissue as early as 10 weeks of age (Sizonenko & Aubert 1986). In the male fetus, LH and FSH probably provoke protein transcription by the SRY gene on the Y chromosome. As mentioned previously, proteins made by the SRY gene eventually direct the undifferentiated gonad to develop into the testis, which will produce and secrete androgens, especially testosterone. However, the source of the prenatal testosterone may not be from fetal tissues entirely. It is known that a chromosomal male embryo lacking the hypothalamus or pituitary develops into a phenotypically male fetus, though with somewhat underdeveloped penis and testes. A placental hormone, human chorionic gonadotropin (hCG), is likely to be capable of stimulating differentiation of the testis and production of testosterone. hCG plays an essential role in the development of the normal fetus as well. In chromosomally normal female embryos, hCG is required, but not estrogen, for the normal development of the female reproductive system during prenatal life. Estrogen is necessary for the final maturation of reproduction following puberty, including the ability to ovulate and lactate (Smith & Korach 1996).

The role of LH and FSH in the development of the female embryo is better understood today than in 1999 when the previous edition of this book was published. The process is regulated by homeodomain proteins (HOXA11 and ISL1), steroid receptors (estrogen receptor alpha and progesterone receptor), transcription factors

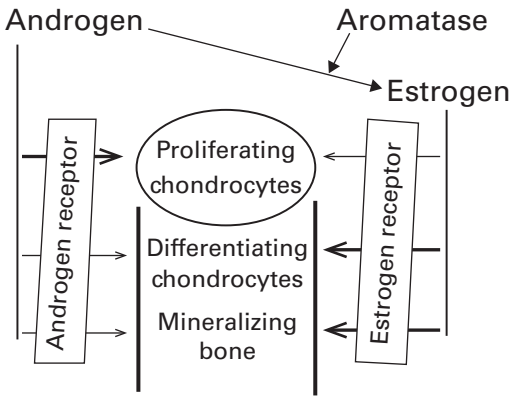


Figure 6.14 A model for the relative actions of androgens and estrogens on the growth of a long bone. Androgen hormones can be converted to estrogens via the action of the aromatase enzyme. The epiphyseal growth plate of the long bone is indicated as a sketch, with the regions of chondrocyte proliferation, chondrocyte differentiation, and bone mineralization labeled. The width of the arrows indicates the relative importance of androgens and estrogens on these regions and processes. Modified after Smith and Korach (1996)

and signaling molecules (TP63 and RUNX1), and keratins which are expressed in a temporally and spatially dynamic fashion (Cunha et al. 2018).

At birth, and up to about two years of age, serum levels of LH, FSH, and gonadal hormones are higher than at any time prior to the onset of puberty (discussed in Chapter 2). The relatively high levels of estrogen and androgens in the bloodstream of the infant are correlated with the rapid velocity of physical growth, neurological development, motor control, and cognitive advancement that take place during infancy.

The rate of production of gonadal hormones and growth velocity fall to relatively low levels by about two years of age, when the negative feedback control becomes highly sensitive. Low levels of gonadal hormones, and a decelerating rate of skeletal growth, are maintained until gonadarche (puberty), when a new positive feedback control develops. The rising levels of testosterone and estrogens have long been associated with adolescence and the growth spurt. A model of the action of androgens and estrogens on skeletal growth is shown in Figure 6.14. Note that the figure indicates that estrogens are the more important steroid hormones. Melvin Grumbach reported that for boys " ... estrogen (not androgen) derived from direct testicular secretion (approximately 20%) and from extragonadal aromatization of testosterone and androstenedione (approximately 80%), is the critical sex hormone in the pubertal growth spurt, skeletal maturation, accrual of peak bone mass, and the maintenance of bone mass in the adult" (Grumbach 2000, p. 1439). The androgen must be converted to estrogen via the enzyme aromatase and more recent investigation reports the absence of the growth spurt if the aromatase SNPs or the estrogen receptor of bone is impaired (Grumbach 2004). The adolescent growth spurt of girls is also estrogen dependent. The onset of gonadarche and the rise of estrogen for girls

occurs, generally, about two years earlier than for boys. Estrogen is important for epiphyseal closure. Lack of estrogen, or inactive estrogen receptors in males result in significant prolongation of long bone growth and eunuchoid, very tall stature. Administration of high levels of estrogen to tall girls results in a stop of lower leg growth within a few weeks (Hannema & Sävendahl 2016; M. Hermanussen, personal communication, see further discussion). The peak velocity of skeletal growth during adolescence for girls is, on average, less than that for boys. Why these sex differences exist is not well understood. Much research by Jeffry Baron and colleagues finds that skeletal growth and maturation of the long bones are both functions of the physiology of the growth plate. In a review of growth plate biology, they report many regulatory factors in addition to estrogen including " ... nutritional, endocrine, inflammatory cytokines, extracellular fluid (for example, oxygen and pH), paracrine, extracellular matrix and intracellular mechanisms" (Baron et al. 2015, p. 4). They also note that there are interactions among many of the regulatory factors, for example, the lack of any essential nutrient or a lack of enough total food intake strongly down-regulates endocrine and other regulators of the growth plate.

The effect of gonadal hormones on personality and behavior has also been an area of much research. It was commonly believed that rising levels of testosterone can increase feelings of aggression and libido in both men and women. On the other hand, rising levels of estrogens were commonly thought to "feminize" (i.e., reduce aggression) the behavior of both men and women. A series of studies by J. Richard Udry and colleagues investigated these popular notions to see if they had scientific validity. The levels of serum testosterone was found to be a strong, positive predictor of sexual motivation and behavior in both adolescent boys and girls (Halpern et al. 1993, 1997; Udry et al. 1985). However, there are also strong effects of social factors that moderate sexual activity. For example, a higher frequency of attendance at religious services significantly delayed age at first coitus. The researchers reported that testosterone was not the primary cause of personality differentiation at puberty – testosterone did not cause male patterns of aggression in the participants of the studies (Udry & Talbert 1988). Rather, as with sexual activity, the measure of "aggressiveness" was moderated by several social factors. The same was true of so-called feminine behaviors in both boys and girls.

Udry (1994, 2000) used these data to develop a model for the development of human genders (as opposed to human sexes). It was a biosocial model and required inputs from both the internal endocrine environment and the external social environment of the individual in order to account for the development of personality and behavior. The model also integrated observation and theory from nonhuman primate behavior. Udry's model was designed to explain changes in the relationship between sex and behavior over time or between groups. The model was one of the first to be trans-primate and biocultural in perspective and to anticipate much current research on human gender development. Scientists and others have long wrestled with the relative importance of hormones, socialization, and cultural ideology as predictors of gender development. An article from 2015 citing Udry reported that the biology and culture of sex and gender are complex, and the direction of causality is not clearly

understood. The conclusion of one analysis reports that, "Prenatal hormones, childhood socialization, and cultural interactionism were all influential factors for gendered selves ... [and that] the social world responds to and reinforces gendered personality" (Davis & Risman 2015, p. 110). This is a mature perspective and one that has come a long way from the racism and sexism of twentieth-century hereditarianism, eugenics, and genetic determinism.

Adrenal Hormones

The role that adrenal hormones play in human growth and development, in relation to adrenarche and the evolution of the childhood period of human development, was discussed in Chapter 4. The adrenal hormones have other effects on growth as well. The cortex of the adrenal produces two classes of hormones, glucocorticoids and androgens. Glucocorticoids, such as cortisol, are involved in the body's ability to maintain homeostasis when faced with physical or emotional stress. Adrenal androgens are produced by the zona reticularis of the adrenal cortex, which is relatively large and active during fetal life, but undergoes involution after birth.

Adrenal androgen levels are low throughout childhood until adrenarche, at about age six to nine years, when secretions begin to increase steadily until a plateau is reached late in the fourth decade of life. The control of adrenal androgen production from its cholesterol precursor involves a complex interplay of many factors (reviewed by Turcu et al. 2014). The adrenal gland contributes only about 1% to the total circulating testosterone in boys and men but up to 30–50% in girls and women. The pituitary stimulating hormone for the adrenal is called adrenocorticotropic hormone (ACTH) and this seems to be the primary mediator of adrenal androgen production (Turcu et al. 2014).

As mentioned in Chapter 4, in otherwise healthy juveniles adrenal hormones do not seem to influence the amount and distribution of body fat. But, some children have an earlier than normal onset of adrenarche and girls with premature adrenarche (<8 years old) are reported to have higher body fat and lower fat-free mass than girls with adrenarche at the expected age of 8 years or older (Cebeci & Taş 2015).

On the other hand, in adults, and possibly in juveniles and adolescents, there are interactions between body fat and the endocrine system. Centralized fat seems to exert the strongest influence. Women with centralized obesity seem to have increased secretion of adrenal androgens, resulting in a more masculinized body in terms of fat patterning and muscularity (Björntorp 1997). In adults with high amounts of abdominal fat there is a lower secretion of pituitary growth hormone and of gonadal androgens and estrogens, due to suppression of hypothalamic GnRH and of pituitary LH and FSH. The cause and effect relationship in all of this is not clear because it seems to take both elevated cortisol levels and lower gonadal hormone and growth hormone levels to direct the fat to be stored at abdominal sites. The placement of fat on the body, in turn, is associated with metabolic syndrome, a cluster of risk factors and diseases, including elevated blood pressure, insulin resistance, diabetes, and heart disease. Centralized body fat, such as that in the abdominal

region, is a "risk factor" for these diseases. Clinical depression and anxiety, as well as alcohol abuse and smoking, also play a role in these hormonal and fat deposit outcomes. Indeed, Björntorp (1997) reported that emotional stress may precede the physiological effects, and that the emotional disorder may cause the disruption in the HPA axis. Experimental studies with monkeys support this interpretation. The emotional stress leads to over-secretion of adrenal cortisol and other hormones, which seems to cause the more generalized hormonal imbalance, the centralized fat deposits, and the metabolic diseases. Study of the social causes of emotional distress, its interactions with the neuroendocrine system, and its effects on growth, development, maturation, and health is currently a very active area of research. In Chapter 7 there is further discussion of recent findings as these relate to community effects, social status competition, and growth.

Growth Hormone and Insulin-Like Growth Factor-1

Some aspects of the actions of growth hormone (GH) and insulin-like growth factor-1 were presented earlier in this chapter. Throughout prenatal and postnatal life the availability of GH and other growth factors are necessary to maintain and promote physical growth and development and also emotional-psychological well-being (Blum et al. 2011). GH is synthesized and secreted by specialized cells in the anterior pituitary gland. A hypothalamic hormone called growth hormone releasing hormone (GHRH), stimulates the synthesis of GH and, along with other agents, causes the release of GH into the bloodstream. Another hypothalamic hormone, growth hormone-release-inhibiting hormone (GHRIH), sometimes also called somatostatin, has an antisecretory effect on GH. At specific locations in the body, GHRIH also inhibits the secretion of TSH, glucagon, insulin, and several digestive acids and enzymes (O'Toole & Sharma 2019).

Experimental and clinical research show that secretion of GHRIH and GHRF are inversely correlated and indicate that the direct feedback control of GH secretion is maintained by **intracrine**, autocrine, and paracrine regulation within the hypothalamus. The overall control of GH secretion is complex, with many inputs from the internal and external environment of the body. Illustrated in Figure 6.15 are some of the more important aspects of GH regulation.

Unlike the other pituitary hormones so far discussed, GH does not seem to affect a single target tissue but appears to have a general growth promoting effect throughout the body. Early research by Donald B. Cheek (1968) found that GH is needed for the body to retain nitrogen, sodium, chlorine, potassium, phosphorous, calcium, and other elements that make up body tissue. Cheek also reported that GH is needed for muscle cell division. Isaksson and colleagues (1982) demonstrated experimentally that GH stimulates long bone growth directly. The authors injected GH into the cartilage growth plate of the proximal tibia of rats, in which the pituitary gland had been surgically removed. Rat tibia receiving the GH injection grew significantly more than rat tibia receiving a saline injection. Green and his colleagues (Green et al. 1985; Morikawa et al. 1982) found that GH promotes the differentiation of pre-

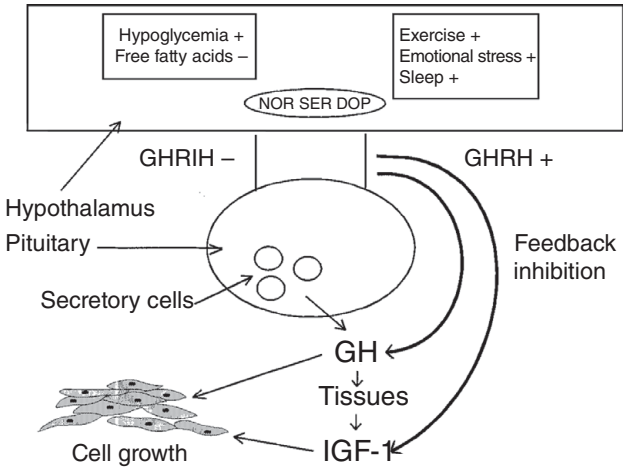


Figure 6.15 Some of the major features of growth hormone (GH) regulation and the hypothalamic–pituitary–GH axis. GH is secreted by specialized cells in the anterior pituitary. GH enters the general circulation and is carried to its target tissues throughout the body. Growth hormone binding proteins (not shown) are needed for GH to have its effect on the target tissues. At the tissues, GH promotes the secretion of insulin-like growth factor 1 (IGF-1) by cells in the tissues. Both GH and IGF-1 may produce cell growth. The regulation of GH secretion is controlled by neurochemical signals from the hypothalamus. GHRIH inhibits GH, as indicated by the minus sign, while GHRH stimulates GH secretion, as indicated by the plus sign. The secretion of both GH and IGF-1 work to inhibit further GH release via feedback inhibition. Several environmental variables may influence GH release. Low food intake leading to hypoglycaemia increases GH release. An increase of free fatty acids in the bloodstream lowers GH release. Exercise, short-term emotional stress, and sleep increase GH release. The impact of these environmental variables on GH is mediated by several neurochemical transmitters in the hypothalamus, including noradrenaline (NOR), serotonin (SER), and dopamine (DOP). Original figure.

adipose stem cells (undifferentiated cells) into adipose cells. Given the widespread action of GH in the body, the name "growth hormone" is an accurate description of the function of this endocrine product.

Another class of growth-promoting substances is called the insulin-like growth factors (IGFs); in older literature they are called somatomedins. The IGFs are similar to insulin in molecular structure and in biological action. Insulin, a hormone produced and secreted by cells of the pancreas, stimulates protein synthesis and the growth of cartilage cells. It is well known that GH acts on the pancreas to increase the synthesis of insulin, and also increases the serum levels of the IGFs. It was once thought that the unique origin of IGFs was from cells located in the liver, but it is now known that IGF-1 is expressed in virtually every tissue of the body. Even so, the liver expression is much greater than that of any other tissue (Ohlsson et al. 2009). Moreover, liver-derived IGF-1 is necessary for the regulation of GH secretion by negative feedback and the regulation of cortical bone mass. Liver derived IGF-1 also plays a central role in the regulation of nonskeletal growth including " . . . kidney

size, prostate size, peripheral vascular resistance, spatial memory, sodium retention, insulin sensitivity, liver size, sexually dimorphic liver functions, and progression of some tumors" (Ohlsson et al. 2009, p. 494).

In humans there are two major types of IGFs: IGF-1, which may be the type that, in concert with GH, regulates postnatal growth; and IGF-2, which appears to be the type that controls some aspects of prenatal and neonatal growth. Production of IGF-2 is stimulated by placental lactogen, a hormone similar to GH and prolactin. The production and signaling actions of GH and the IGFs are also regulated by several epigenetic marks on the genome and a complex variety of epigenetic pathways toward target tissues (see Álvarez-Nava & Lanes 2017 for a review). These epigenetic mechanisms influence carbohydrate and lipid metabolism. One harmful outcome is intrauterine growth retardation and post-natal short stature. Maternal exposure to nutritional, disease, and emotional stressors are a few of the likely causes of epigenetic marking of the genome of the fetus.

Prior to adulthood, a deficiency of GH, IGF-1, their cell receptors, or their signal transducers (JAK2, STAT5b, etc.) results in growth retardation and short stature. Children and adolescents with pituitary gigantism have an excessive production of GH, and IGF-1 levels are elevated. Clinical studies demonstrate the central role of GH and IGF-1 in human growth. Peripubertal children with idiopathic short stature (short height without a known cause) treated with GH show significant increases in adult height in randomized, double-blind, placebo-controlled trials (Leschek et al. 2004). In a randomized, controlled, multi-center clinical trial, children with short stature but not deficient in GH were treated with GH or not treated (Albertsson-Wikland et al. 2008; Kriström et al. 2014). Over a five-year period, the GH recipients showed significant increase in height in a dose-response fashion. The change in IGF-1 levels from baseline explained the largest amount of the variance (28% of the total variance) in greater height compared with untreated children. The authors of this study interpret the findings to indicate that GH treatment stimulated IGF-1 production and this stimulated skeletal growth. In the affluent nations of Europe, cross-sectional surveys of healthy children and adolescents (Alberti et al. 2011) as well as birth cohort studies of healthy children (Rogers et al. 2006) find moderate-to-strong positive associations between serum IGF-1 levels and concurrent measures of height. The authors of the birth cohort study report a positive association of serum IGF-1 at age 5 and 7 years with amount of growth in height at ages 8, 9, and 10 years.

The growth pathologies of Laron syndrome and acromegaly indicate a primary role of IGF-1. One of the features of Laron syndrome is very short stature. Some types of short stature are due to low levels of, or an absence of, GH, but Laron syndrome short stature results from low levels of IGF-1 despite normal or high levels of GH. Adult height is at or below 142 cm for men and 136 cm for women. The cause of Laron syndrome is a genetic mutation that results in a deficiency of the cell receptors for GH.[8]

[8] www.omim.org/entry/262500

Without the GH receptor, cells cannot be stimulated to produce IGF-1, and the result is impaired cell growth.

Gigantism and acromegaly are conditions of abnormal excessive growth, most often due to a pituitary tumor. Gigantism occurs when GH hypersecretion occurs before the fusion of the long bone epiphysis and is characterized by tall stature. Acromegaly occurs when GH hypersecretion occurs after the fusion of the epiphysis leading to slow, but continuous increase in size of the bones of the face, hands, and feet throughout life that often leads to gross disfigurement in adulthood (Bello & Garla 2019). Both may occur in the same person. The professional wrestler and actor Andre the Giant (star of the 1987 film *The Princess Bride*) suffered from these conditions. At the time of his death in 1993, at age 46, he was 224 cm tall and weighed 236 kg, with clear signs of acromegaly in face and hands. Levels of GH and IGF-1 are both above normal in acromegalic patients; however, there is a higher correlation between IGF-1 and the clinical progress of the disease.

IGF-1 and Inflammation

Children with chronic inflammatory diseases, or recurrent infections, often show growth stunting. While factors such as malabsorption have been implicated in their growth failure, it has also been demonstrated that inflammation can down-regulate IGF-1 production. Animal models have shown that this down-regulation can occur independently of effects on GH production, and that markers of systemic inflammation (e.g., C-reactive protein) are inversely correlated with IGF-1 levels (De Benedetti et al. 1997). This down-regulation of IGF-1 may play a role in the high frequency of lower bone mass and high rate of bone fracture in children with chronic inflammatory diseases (Burnham 2012).

The Growth Plate and Its Role in Size Variation

Clearly, GH and the IGFs, along with IGF receptors, IGF binding proteins (proteins that carry IGFs to their target sites), and IGF proteases " ... regulate the fate, localization, and activity of many proteins, modulate protein–protein interactions, create new bioactive molecules, contribute to the processing of cellular information, and generate, transduce, and amplify molecular signals" (López-Otín & Bond 2008, p. 30433). Stated more simply, proteases are involved in DNA transcription and have important roles in normal and pathological growth. In addition, there are many known mutations outside the GH–IGF system that are responsible for tall or short stature, differences in limb proportions, bony breadths of the body, and bone density. Ultimately, any influence of the endocrine system on bone length must act on the growth plate (Figures 2.15 and 3.3) as this is " ... the structure responsible for height gain" (Baron et al. 2015, p. 736).

Growth plate physiology is complicated by the fact that not all growth plates are created equal. As observed by Raimann and colleagues (2017, p. 1680), "The skeleton is not a single functional unit but consists of different, well-organized, and

mineralized compartments with specific functions, developmental aspects, and regulations." Different segments of the skeleton must grow in an independent and coordinated fashion for the maintenance of correct body proportions, support of body mass, locomotion, and other physiological and behavioral functions. The long bones of the skeleton, especially of the leg, contribute to the total body height mainly by the activity of four growth plates of the proximal and distal tibia and femur. In contrast, approximately another 50% of height is contributed by growth of the vertebrae, the head, and to a smaller extent the pelvis. Very little is known about the regulation of growth of the pelvis and head. The head is quite complex because the mandible has growth plates in its condyles, but the facial and skull bones do not have growth plates and grow instead by intramembranous or endochondral ossification.[9]

One study analyzed patterns of growth for skeletal dimensions, weight and body composition, and head circumference of 7,444 boys and 7,375 girls, aged from 0 to 7 years, all of good nutritional status and health, measured in East Germany between 1986 and 1990 (Scheffler et al. 2017). The list of measurements included length/height, leg length, sitting height, biacromial shoulder breadth, thoracic breadth, thoracic depth, thoracic circumference, body weight, head circumference, percentage of body fat, and hip skinfold. The authors used principal component analysis to distinguish patterns of growth associations among these measurements. It was reported that there were strong associations between all the measurements, but despite the general proportionality there were different patterns for skeletal growth, fat acquisition, and head growth. The measurements of skeletal growth loaded predominantly on the first component, which accounted for between 31% and 52% of the total variance in growth depending on age. Body weight and fat deposition loaded on a second component and accounted for between 13% and 19% additional growth variance. After the age of 2 years, head circumference loaded on a separate third component in both sexes, accounting for between 8% and 12% of the remaining variance. The researchers concluded that after the age of 2 years growth of the head is independent from growth of the post-cranial skeleton.

A further complication is that each of the 7 cervical, 12 thoracic, and 5 lumbar vertebrae has growth plates (the 5 sacral and 4 coccygeal vertebrae either fuse early in life or do not contribute to height). Due its multiple vertebrae, the spinal column has both more centers of chondrocyte activity and, perhaps, more actively proliferating cells than the long bones of the legs or arms. Some research has focused on possible differences in the activity and the regulation of the growth plates between spine and long bones. Most of this research relates to growth pathology, such as scoliosis (curvature of the spine, Crijns et al. 2017).

[9] https://embryology.med.unsw.edu.au/embryology/index.php/Musculoskeletal_System_-_Skull_Development

Hypotheses of Long Bone Growth

No matter how the long bones vs. the vertebrae grow, two things must happen at the growth plate: (1) undifferentiated cells in the reserve zone (Figure 3.3) need to be stimulated to begin differentiation toward bone cells. These progenitor cells become committed to clonal expansion, meaning that they will undergo hyperplasia and multiply into a pool of pre-chondrocyte cells in the proliferative zone and (2) the pre-chondrocytes undergo growth in size in the hypertrophic zone and then begin maturation to bone (ossification zone).

It was hypothesized prior to 2019 that there was a limited supply of pre-chondrocyte stem cells in the reserve zone. As elongation of the bone occurred this supply was consumed and eventually exhausted. More, or less, growth in length, resulting in greater or lesser total height or different body proportions of trunk length vs. leg length, would result from a larger or smaller pool of stem cells in the reserve zone of each growth plate. A recent discovery changes this assumption. Using a genetic tracing technique, Newton and colleagues (2019) were able to follow the fate of clonal lines of stem cells in the reserve zone of the growth plate. They found that during the fetal and neonatal stages of growth and development there was a depletion of the pre-chondrocyte stem cell pool. In later infancy, as secondary ossification centers formed and began to mature, the stem cells acquired " . . . the capacity for self-renewal, resulting in the formation of large, stable monoclonal columns of chondrocytes . . . [allowing the growth plate to produce] . . . a continuous supply of chondrocytes over a prolonged period" (p. 234). The Newton et al. article contains several excellent microphotographs and a graphical summary of the findings (see their figure 3, part J, p. 237).

The old "limited supply of pre-chondrocyte stem cells" hypothesis never quite made sense for at least two reasons. The first is that it implied the determinism of adult size by the number of stem cells set very early in life. If this were true, then the tremendous variation that skeletal growth seen in the real world would not be possible. One example of this variation are the Maya refugee children in the United States discussed in Chapter 1 who grow an average of 11 cm taller than their age mates back in Guatemala. A second argument against the "limited supply" hypothesis is the general symmetry of size and shape of the long bones on the left and right sides of the body. This indicated that rather than each growth plate acting independently there is some central regulation. It is now known that in addition to the actions of GH and IGF-1 to promote the differentiation, multiplication, and hypertrophy of the chondrocytes, there are other systemic regulators of the self-renewal of the stem cells of the growth plate. These include the growth signaling pathways called hedgehog and mammalian target of rapamycin complex 1 (mTORC1) as well as inflammatory cytokines, energy balance, and the availability of most of the essential nutrients, epigenetic marks, and biochemical factors present in the extracellular fluid (Baron et al. 2015; Lampl & Schoen 2017). These is evidence that these regulators interact with the mechanical load placed on bone by physical activity and this mechanical load is a major stimulus to produce changes in growth rate (Villemure

& Stokes 2009). Further information about the signaling pathways, cytokines, epigenetic marks, extracellular fluid, mechanical loading is best perused in the primary research literature as these topics are active areas of investigation. The impact of energy balance and essential nutrients on skeletal growth are discussed in Chapter 7.

Another recent discovery is that differential aging of cells in growth plates contributes to the disparities in bone length. The human femur is about 20-times longer than the phalanges in the fingers and toes. The regulation of such differences in bone length are not well understood. It is known that bone elongation occurs rapidly during fetal and early postnatal life and then progressively slows. This process is called "growth plate senescence" (Lui et al. 2018). Research by Lui and colleagues indicates that " . . . senescent changes occur earlier in the growth plates of smaller bones (metacarpals, phalanges) than in the growth plates of larger bones (femurs, tibias)" (p. 1). The researchers also provided evidence that endocrine and signaling factors, including IGF binding protein, bone morphogenetic protein (BMP), and Wingless and Int-1 (WNT), have essential paracrine regulatory effects on rates of both growth in length and senescence. The paracrine action of these signaling factors creates a difference in the balance growth inhibition vs. growth stimulation by changing gene expression in the growth plates. In the growth plates of phalanges, the gene expression of growth inhibitors is enhanced, such as IGF binding protein and WNT Inhibitory Factor 1. In contrast, in the growth plates of the femur and tibia there is upregulation of gene expression of the growth stimulators BMP and WNT5b.

Mini Growth Spurts and Saltations

Long bone growth occurs in cycles, with alternating phases of stem cell differentiation followed by the expansion of newly differentiated cells by hyperplasia and hypertrophy. Growth cycles, or pulses, have been found when growth in length is measured on a weekly or daily basis. Periods of little or no growth, called stasis, are followed by bursts of rapid growth, called "mini-growth spurts" (Hermanussen et al. 1988a; Hermanussen & Burmeister 1993; Hermanussen & Geiger-Benoit 1995) or "saltations" (Lampl et al. 1992). An example of mini-growth spurts is illustrated in Figure 6.16 for growth of the lower leg.

Hermanussen and colleagues (1989) investigated differential growth of the left and right lower legs in 5 boys, 4 girls, and 1 person with Turner syndrome, aged 6.3–14.2 years old. The authors used knemometry, a noninvasive technique of lower leg length measurement with an accuracy (technical error) of 0.09–0.16 mm (Hermanussen et al. 1988b). The children were measured at exactly weekly intervals (same day of the week and at the same time of day) or exactly at 2-week or 3-week intervals in the case of holidays or personal reasons for absence for periods of 11 to 45 weeks. Participants had from 10 to 31 consecutive measurements. Mini growth spurts were detectable in 7 of the children and occurred in both legs. Analysis of the individual series of right vs. left differences of the weekly lower leg length increments provided evidence for alternating periods of overgrowth of one leg compared to the

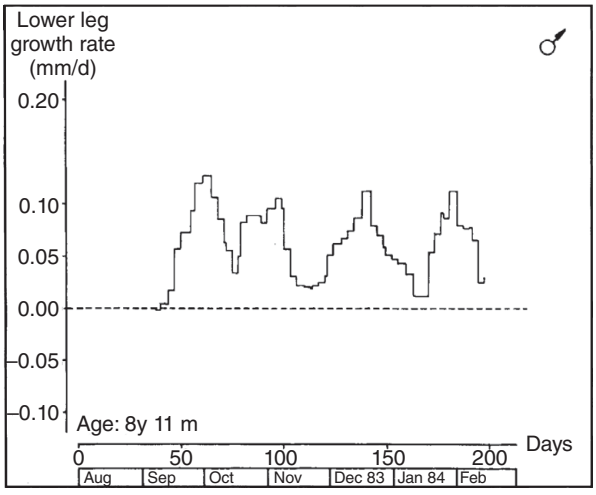


Figure 6.16 Growth velocity data for the lower leg ("knee height") of a boy aged 8 years 11 months, measured once weekly. "Mini growth spurts" occur from week to week, and a longer term cycle of increases and decreases in the size of the "spurts" is also apparent. (After Hermanussen et al. 1988a)

contralateral side in 5 out of the 10 children. The authors suggested that there is evidence of partial independence of lower leg growth in the short term.

Why one leg does not consistently grow more than the other is not known. Some researchers speculate that there is a central regulation of body symmetry. In a personal communication with Professor Hermanussen, he suggested that the reason for leg length symmetry is simply due to mechanical loading, which was mentioned already. If one leg is longer than the other, bipedal locomotion will result in a discrepancy of force on the legs and growth of the shorter leg will be stimulated toward equalization of the bilateral forces. This is for normal growth. In cases of pathology, due to congenital, traumatic, or disease impact on one leg, the regulation of symmetry is complicated by bone age – greater final symmetry may be expected if medical intervention takes place before the pubertal growth spurt. Other important factors affecting the success of medical intervention are the frequency and interval between medical interventions. Clinical experience finds that if more than two lengthening procedures are done over three years, especially during infancy, then there will be growth inhibition. Furthermore, the percentage of lengthening at each intervention should be $<30\%$ of the initial segment length (Journeau et al. 2016).

The discovery of these bursts of growth replaced the traditional belief, based on annual or quarterly measurements, that growth is a smooth and linear process. The discovery also showed that short-term rates of growth cannot be extrapolated to annual rates, an important finding for medical intervention. Also important was the discovery that at any point in time different segments of the body may be growing at different rates. A Japanese study (Ashizawa & Kawabata 1990) of two children (ages 7.5 and 6.6 years at the start of the study) measured daily for 1 year found that all

growth in stature was confined to the lower limbs, except for a seasonal pulse of trunk growth in the Spring (April–May). Saltations and stasis take place at the growth plate of long bones. Growth stasis would occur during the resting and differentiation stage of growth plate activity, and mini spurts/saltations would occur during the expansion stage. Exactly how mini-growth spurts and cycles of growth stasis are controlled is not known. As described earlier, regulation of the growth plate is complex and responds to the entire gamut of genomic, physical environment, and social and emotional circumstances in which humans grow (Lampl 2018).

Other Growth Factors

IGFs are just one class of growth factors. There are many other classes known and more are discovered each year. A classification of some of the known growth factors is given in Table 6.4.

As with the IGFs, the growth factors and cytokines listed in the table are signaling molecules that regulate cellular activities. Each factor is specific to one or more types of cells by means of receptors that bind the growth factor to the cell and activate a cascade of downstream pathways. Ultimately, these pathways usually end in the cell nucleus and influence gene transcription to produce a biological response. A common family of cell membrane receptors are the receptor tyrosine kinases (RTKs). At least 90 unique tyrosine kinase genes in the human genome encode 58 RTKs which are known to be key regulators of normal cell physiology. Mutations and other pathological variations of RTK genes may result in the development and progression of several types of cancer.[10] A schematic example of a typical RTK cascade is shown in Figure 6.17.

Specialized journals, such as *Cytokine and Growth Factor Reviews*, are devoted to this research.[11] These journals should be consulted by interested readers for up-to-date information.

Insulin, Leptin, and Ghrelin

Insulin, leptin, and ghrelin are involved in the regulation of energy metabolism and food intake, playing key roles in signaling hunger, satiety, and satiation in postnatal life. They also have important effects on immune function, inflammation, and growth. Insulin is a peptide hormone secreted by the pancreas and promotes glucose uptake from the food that is digested and absorbed into the bloodstream or from the release of glucose from muscle stores of glycogen. The rate of insulin production is positively associated with energy intake – more food ingested leads to more insulin production. Leptin is a peptide hormone produced by adipose tissue that has effects on many tissues, especially to regulate energy balance. Leptin acts over the long term by signaling its receptor sites within the central nervous system (Gautron & Elmquist

[10] www.abmgood.com/marketing/knowledge_base/growth_factors_cytokines_introduction.php
[11] www.journals.elsevier.com/cytokine-and-growth-factor-reviews

Table 6.4 Growth factor and cytokine classification. Based on structural and functional characteristics, growth factors can be divided into various families and superfamilies. Each family has its own receptors and signaling pathways which typically operate by activating a tyrosine kinase signaling cascade. Listed in this table are some of the major growth factor families. The list is based on information at: www.abmgood.com/marketing/knowledge_base/growth_factors_cytokines_introduction.php. See the website for additional details. Not detailed here are the growth factors Indian hedgehog, PTHrP, mTORC1, and others (see Saxton & Sabatini 2017; van der Eerden et al. 2003 for more on these growth factors).

1. Insulin-like Growth Factors (IGFs). The Insulin-like Growth Factors (IGFs) are proteins with high DNA sequence similarity to Insulin. There are two types of IGF receptor: IGFI-R and IGFII-R. The availability of IGFs can be regulated by IGF Binding Proteins 1–6. The major action of IGFs is on cell growth. Indeed, most of the actions of pituitary growth hormone are mediated by IGFs, primarily IGF-1. Growth hormone stimulates many tissues, particularly the liver, to synthesize and secrete IGF-1, which in turn stimulates both hypertrophy (increase in cell size) and hyperplasia (increase in cell number) in most tissues, including bone. IGFs can also induce neuron survival, protect cartilage cells, and activate osteocytes.
2. The Transforming Growth Factor Beta (TGF-beta) Superfamily. The TGF-beta superfamily includes the TGF-beta proteins, Bone Morphogenetic Proteins (BMPs), Growth Differentiation Factors (GDFs), Glial-derived Neurotrophic Factors (GDNFs), Activins, Inhibins, Nodal, Lefty, and Müllerian Inhibiting Substance (MIS). The TGF-beta superfamily members are multifunctional regulators of various biological processes such as morphogenesis, embryonic development, adult stem cell differentiation, immune regulation, wound healing, inflammation, and cancer.
3. Epidermal Growth Factors (EGFs) Family. The EGF family members include EGF, TGF-α, Neuregulins, Amphiregulin, Betacellulin, and others. The members of the EGF family are best known for their ability to stimulate cell proliferation, differentiation, and survival. Deregulation of the members of this family and their receptors is closely associated with tumorigenesis (cancer).
4. Platelet-Derived Growth Factors (PDGFs). The PDGFs are potent mitogenic and chemotactic proteins. There are currently four known PDGF proteins encoded by four genes (PDGFA, PDGFB, PDGFC, and PDGFD). PDGFs are produced by distinct populations of cells that include activated macrophages, epithelial and endothelial cells, smooth muscle cells, and activated platelets. There are two known PDGF receptors with intrinsic tyrosine kinase activity; PDGFRα and PDGFRβ. Signaling via PDGFRα is important for the development of the facial skeleton, hair follicles, spermatogenesis oligodendrocytes and astrocytes, as well as for the development of the lung and intestinal villi while signaling via PDGFRβ is crucial for the development of blood vessels, kidneys, and white adipocytes. PDGFs are essential for early development, wound healing, and angiogenesis. It is noteworthy that the abnormal regulation and production of PDGF isoforms may cause tumor, vascular disease, and fibrotic disease.
5 Fibroblast Growth Factors (FGFs) Family. In humans, twenty-two members of the FGF family have been identified all of which are heparin-binding proteins. FGFs are pluripotent proteins that are primarily mitogenic but also have regulatory, morphological, and endocrine effects. FGFs are involved in embryonic developmental processes, mature tissues/systems angiogenesis, keratinocyte organization, and wound healing processes.
6. Vascular Endothelial Growth Factors (VEGFs). VEGFs are glycoprotein growth factors that are specific to endothelial cells. They regulate angiogenesis and vascular permeability, especially during embryogenesis, skeleton growth, and reproductive functions. They also play important roles in hematopoiesis. VEGFs signal pathways also stimulate cell survival, proliferation, migration, and/or

Table 6.4 (cont.)

adhesion. Deregulation of VEGFs has been associated with tumors, intraocular neovascular disorders, and other diseases. Placental Growth Factor is one type of VEGF.

7. Hepatocyte Growth Factors (HGFs). HGF is secreted by mesenchymal cells and acts as a multifunctional cytokine on cells that are mainly of epithelial and endothelial origin. It regulates cell growth, cell motility, and morphogenesis. HGF has been shown to have a major role in embryonic organ development, adult organ regeneration, and wound healing. Furthermore, its ability to stimulate mitogenesis, cell motility, and matrix invasion gives it a central role in angiogenesis and tumorigenesis.
8. Tumor necrosis factors (TNFs). Cytokines that were known to be involved in tumor cell apoptosis (programed cell death) were initially classified as Tumor Necrosis Factors (or under the TNF family). To date, 19 TNF superfamily ligands have been identified along with 32 TNF superfamily receptors. While many TNF superfamily members promote or inhibit apoptosis, they also regulate critical functions of both the innate and adaptive immune system including natural killer cell activation, T-cell costimulation, and B-cell homeostasis and activation. In addition, several TNF superfamily members regulate cell type-specific responses such as follicle apoptosis and osteoclast development.
9. Interleukins (ILs). Interleukins are a large group of immunomodulatory proteins that regulate growth, differentiation, and activation of cells in the immune or haematopoietic systems during immune response. ILs can exert pro- and anti-inflammatory effects and are essential for host defense against pathogens.
10. Interferons (IFNs). IFNs are a group of signaling proteins that are made and released by host cells in response to the presence of pathogens such as viruses, bacteria, parasites, or tumor cells. IFNs don't work directly on pathogens but rather stimulate the infected cells and those nearby, to produce proteins that prevent the replication and growth of pathogens. Interferons also have immunoregulatory functions; they inhibit B-cell activation, enhance T-cell activity, and increase the cellular-destruction capability of natural killer cells. More than 20 distinct IFN genes and proteins have been identified in animals, including humans.
11. Colony-Stimulating Factors (CSFs). CSFs are secreted glycoproteins that bind to receptor proteins on the surfaces of hemopoietic stem cells and activate intracellular signaling pathways that can cause the cells to proliferate and differentiate into a specific kind of blood cell (usually white blood cells). There are three types of CSFs. CSF1 promotes the growth and maturation of monocytes and macrophage precursors. It also enhances the phagocytic and tumoricidal activity of human macrophage/monocytes and induces them to secrete a variety of different cytokines. CSF2 is known to stimulate the growth and differentiation of hematopoietic precursor cells while CSF3 is involved in hematopoiesis by controlling the production, differentiation, and function of white cell populations of the blood, the granulocytes, and the monocytes-macrophages.

2011). In addition to exerting its actions through binding to leptin receptors within the brain, leptin likely influences brain development itself, especially in the hypothalamus (Bouret 2010). Many investigations of pediatric leptin concentrations are situated within the context of the development of overweight and obesity (OW/OB, e.g., Granado et al. 2012); however, considering that the OW/OB epidemic is a recent phenomenon, it is more likely that leptin acts as a starvation hormone, signaling an energy-deficient state with downstream consequences on body and brain growth, development, and maturation (Bernstein & Bogin 2019).

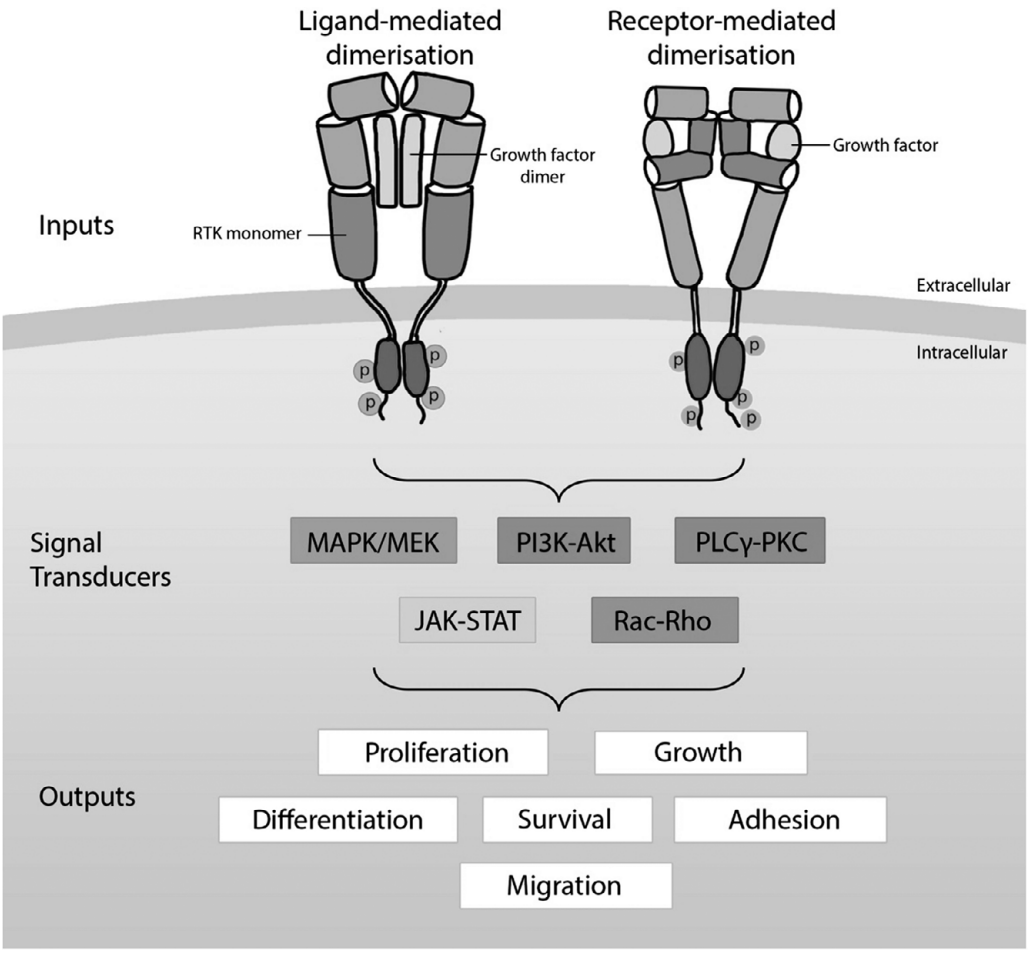


Figure 6.17 General overview of receptor tyrosine kinase activation, signaling, and the cell-fate decisions they influence. The binding of growth factors (inputs) in the extracellular milieu induces conformation changes in the receptor monomer that enables dimerization. Enzymatic autophosphorylation (circled p) by intracellular tyrosine kinase domains in trans results in recruitment of one or more signal transduction cascades. These relay the signal to effectors that determine cell fates (outputs). Abbreviations: MAPK, Mitogen-activated protein kinase; PI3K–Akt, phosphatidylinositol 3-kinase–protein kinase B; PLCy–PKC, phospholipase C gamma–protein kinase C; JAK–STAT, Janus kinase and signal transducer and activator of transcription; Rac-Rho, Ras homologous protein family. From Mele & Johnson (2020, an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (http://creativecommons.org/licenses/by/4.0/). (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Leptin has been proposed to mediate the predisposition of small-for-gestational-age infants to preferentially accumulate fat mass during catch-up growth, via altered adipose tissue metabolism (Lukaszewski et al. 2013). In children with chronic undernutrition, leptin production is suppressed through the combined

effects of decreased fat mass and energy intake, as well as lowered insulin and IGF-1 concentrations – all working to divert energy from growth to maintaining metabolic homeostasis (Soliman et al. 2000).

The energy balance roles of insulin and leptin probably contribute to the timing of puberty and the transition from the juvenile to the adolescent stages of growth. It is possible that both insulin and leptin are part of the hypothalamic signaling pathways that accelerate or retard pubertal timing, but neither substance directly control it. There is evidence that both insulin and leptin interact with kisspeptin neurons in the hypothalamus (mentioned in Chapter 2). Kisspeptin is involved in some of the pathways regulating the hypothalamic–pituitary–gonadal axis and timing of puberty in primates (Plant & Ramaswamy 2009).

Ghrelin, a gastrointestinal peptide, often demonstrates reciprocal effects to those of leptin, that is, ghrelin enhances appetite. Its secretion is regulated by signals integrated by the central nervous system (e.g., food cues, stress, insulin, leptin) that can feed back to inhibit or enhance release of ghrelin. As such, ghrelin production increases not only in the presence of food cues but also in response to chronic or acute stress. Some people feel hungrier when stressed, but other people loose appetite and these states may be due, in part, to more, or less, ghrelin in circulation. In addition to its orexigenic (a good word to look up) effects, ghrelin can stimulate the release of GH, and modulate digestive properties, sleep, and muscle integrity (Müller et al. 2015). In healthy children, ghrelin concentrations peak at approximately two years of age, then gradually decrease until puberty, at which point girls maintain higher concentration than do boys. It is likely that this ensures sustained growth of body fatness in the girls via a relatively greater energy intake (King et al. 2010). In contrast, as discussed in Chapter 2 boys tend to decrease in total body fatness following puberty. The sex differences in patterns of ghrelin secretion and food intake are associated with the greater body fatness that girls will require for successful reproduction (as discussed in previous chapters). Any effect of ghrelin on linear growth is likely to be secondary to the action of GH, IGF-1, and the sex steroids (Chanoine et al. 2009).

Summary of the Neuroendocrinology of Growth

Hormones regulate and coordinate critical developmental processes, integrating across several systems, including the central nervous system, the reproductive system, and the digestive system. Hormones are influenced by nutritional status and infections. Therefore, hormones provide a mechanism by which "real-time" information about a body's health is communicated to the brain and processed by its regulatory centers in the hypothalamus and pituitary, through which growth is affected accordingly. The hypothalamus and pituitary play major roles in many of the central hormone feedback loops involved in growth and development. Growth is disrupted in individuals with congenital hormone abnormalities, such as thyroid hormone deficiency and growth hormone insufficiency or insensitivity, nutritional deficiencies, or disease burden. The severity of the growth disruption and

the potential for recovery to "normal" growth patterns, or to achieve a particular size, depend on the degree of insufficiency or type of disease, the stage of development, interventions to ameliorate the problem, the effectiveness of the intervention, social and emotional support of the affected person, and several other factors. The next chapter explores in more detail nutritional, disease, and other environmental influences on the pattern of human growth.

7 What Makes People Grow?

Love, Hope, Community Effects, and Strategic Growth in the Context of Environmental Factors Influencing Human Development

As a child in the 1950s my mother said to me: "There are starving children in Europe. Eat your supper, otherwise you will not grow." This seemed right to me at the time. During and after World War II Europe did experience starvation in the Netherlands, in Greece, in eastern Europe, the Soviet Union, and defeated Germany. Food rationing was imposed everywhere. In the United States it started in 1942 and most food rationing ended in December 1945, but sugar rationing continued until June 1947. My mother was a teenager during those years and experienced food rationing directly. In victorious Britain food rationing did not end until midnight on 4 July 1954, when restrictions on the sale and purchase of meat and bacon were lifted. That was nine years after the end of the war. Photographs I saw in *Life* magazine[1] and elsewhere in the late 1950s and early 1960s depicted war refugees, holocaust survivors, and US conscientious objectors to military service who "volunteered" for *The Minnesota Starvation Experiment* (Keys et al. 1950).[2] To my child mind these images evoked hunger as the people were thin and seemed small. My mother had to be right – if those people had more food to eat, then they would be bigger.

Young people do need food to grow, but other essential factors are often downplayed or ignored. Young people and pregnant women also need the type of support and care that comes from the healthy biocultural reproduction discussed in Chapter 4. People need the physical and emotional security of knowing that they will have a diet that meets all nutrient requirements, the security of good water, sanitation and protection from infection, and the security of other material safeguards such as adequate housing. These are the minimal requirements for survival. But these alone do not make for a healthy, well-grown person. As explained below, infants lacking social interaction and love die, even when given all their physical necessities. Human biocultural reproduction is successful only with true well-being and this happens when young people have opportunities to participate in healthy social and community environments, when the young are part of systems of informal and formal education, and when the young and their families have hope for the future.

The biocultural needs of all humans were outlined in 2001 by Klaus Toepfer, Executive Director of the United Nations Environment Programme, as the " . . . basic rights to life, health, adequate food and housing, and traditional livelihood and culture" (Shelton n.d.). The UN does not define "livelihood." A narrow definition of

[1] https://www.magnumphotos.com/newsroom/society/david-seymour-children-of-europe/
[2] www.bbc.co.uk/news/magazine-25782294

"livelihood" is the work people do to secure an income and physical necessities. In the social sciences, the concept of livelihood extends to include social, cultural, and emotional needs. One biocultural definition of "livelihood" is " ... the command an individual, family, or other social group has over an income and/or bundles of resources that can be used or exchanged to satisfy its needs. This may involve information, cultural knowledge, social networks and legal rights as well as tools, land and other physical resources" (Wisner et al. 2004). Anthropologists understand that a livelihood is not only a way to make a living – it is a way to make living meaningful. Indian anthropologist Rashmi Rekha Tripathy reviews research on the social science concept of "livelihood" and wrote " ... there is a moral or cultural dimension to livelihood as well as a material dimension: livelihood involves not simply the satisfaction of material needs it also involves the satisfaction of emotional, spiritual and intellectual needs" (Tripathy 2018, p. 1). Tripathy conducted fieldwork with Juang tribal agriculturalists in Odisha State, India and found that changes to their means of financial livelihood due to migration from the village to urban areas not only impacts their economic status but also their socio-cultural life, including their family and kinship networks, religious rituals and festivals, and the marriage system. These are part of the biocultural approach to human growth and development discussed in the Introduction to this book.

Healthy biocultural environments and livelihoods ensure emotional security as much as they provide physical needs. People also need to be loved. Healthy human growth is a "love story." Readers of this book likely have received and given much love during their lives. At the 2009 symposium, "Origins of Altruism and Cooperation" Walter Goldschmidt is quoted as stating, "You talk about cooperation and altruism but what you really mean is LOVE. We shouldn't be afraid to use the word LOVE. That is what makes us truly human" (from the dedication to the book from the symposium, Sussman & Cloninger 2011). "Love" has many meanings and I focus only on those that relate to biocultural reproduction and human growth. Recall from Chapter 4 that Allison Jolly wrote that humans have " ... few, much loved offspring ... " (Jolly 1985, p. 44). "Love" in this context refers to the prolonged care that primate mothers lavish on their offspring. Mentioned in the Introduction was a specifically human expression of love, Robert LeVine's (1977, 1988) proposal that all human parents have a universal evolutionary hierarchy of goals for their offspring. LeVine's parental love manifests as two goals: (1) encourage the survival and the health of offspring and (2) develop offspring into self-supporting adults with culturally specific and acceptable beliefs and behavioral norms. Many anthropologists and others have elaborated on Jolly's and LeVine's version of love (Bogin 2009; Chisholm 1999; Goldschmidt 2006; Hrdy 1999, 2009). Goldschmidt's and Hrdy's type of love manifests in the emotional and physical commitment that many people must make to successfully support a pregnant woman, her infant, and older children, juveniles, and adolescents. Some aspects of the emotional and physical commitment by families and societies were reviewed in Chapter 4. Goldschmidt called the need for this commitment "affect hunger," which is " ... rooted in biology and emerges with culture" (Goldschmidt 2006, p. 141). To satisfy our affect hunger we humans must

have the nurturant love from others to complete our physical development and to bring us to culture. Infants and children deprived of nurturant love often die and those surviving do not grow in body or brain. They never become healthy, cultural persons. A brief account of the twentieth-century history of these discoveries is given in Box 7.1.

Box 7.1 **A lack of love is deadly**

Psychosocial short stature is a well-studied condition that shows how the neuroendocrine system mediates the relationship between psychological-emotional factors and physical growth. Psychosocial short stature is a clinical condition of slow growth or growth failure between birth and adulthood that cannot be ascribed to an organic problem with the child, but rather to behavioral disturbance and emotional stress in the environment in which the child lives. Another symptom is delayed puberty. A diagnosis of psychosocial short statue is confirmed when the child is removed from that environment and growth, development, and maturation are spontaneously restored (Powell et al. 1967a, 1967b). Saenger and colleagues (1977) showed that changes in growth rate of children with psychosocial short stature are not associated with caloric intake. Many behavioral and emotional factors can lead to psychosocial short stature and a history of abuse, neglect, and/or emotional deprivation is common. The key proximate cause is hormonal. In a review of his clinical experience with psychosocial short stature Rappaport (1984, p. 44) stated that " . . . the most consistent biological finding was the decrease of circulating somatomedin [IGF-1] activity . . . " It is also known that there are reductions and derangements in the physiology of other neuroendocrine markers, such as melatonin, serotonin, β-endorphins and ACTH, and in tryptophan metabolism (an essential amino acid) in young people suffering from affective emotional deficiency (Muñoz-Hoyos et al. 2011).

As early as 1701 there was clear evidence of a relationship between the psychosocial environment and human development. This evidence, published in *Gerhardts Handbuch der Kinderkrankheiten* (*Gerhardt's Handbook of Pediatric Diseases*) and reprinted by Peiper (1955), consisted of the percentage of infants admitted to foundling homes who died while in care. The data are reproduced here in Table B7.1.1 in descending order of the percentage of infants dying. In Irkustk, Siberia, Russia 100% of infants entering the foundling home died, but no date was given by Peiper. In Dublin, Ireland for nearly all of the eighteenth century 98% of infants died in the foundling homes. The percentage of deaths decreased, generally, over time, but remains 50% or higher except for the cities of Prague and Bordeaux. Those two cities had foundling homes and also placed some infants with caregivers in the rural countryside. The Prague infants seemed to fare worse there and the Bordeaux

Table B7.1.1 Infants dying in foundling homes in European cities or nations with two cases of deaths to infants placed into care homes in the countryside. Data from Peiper (1955).

City or Country	Year	Percent dying
Irkutsk	No date given	100
Dublin	1701–1797	98
Petersburg	1772–1784	85
Brussels	1811	79
Petersburg	1785–1797	76
Vienna	1811	72
Paris	1780	69
Paris	1817	67
Moscow	1822–1831	66
Gent	1823–1833	62
Dijon	1838–1845	61
Mons	1823–1833	57
Brussels	1817	56
Belgium	1823–1833	54
Petersburg	1830–1833	51
France	1838–1875	50
Prague	1865	20
infants placed in the countryside		35
Bordeaux	1850–1861	18
infants placed in the countryside		15

infants slightly better. Peiper's book contains several comments on the "clean conditions" and "good diet" provided in the foundling homes, nevertheless so many of the infants died. A lack of adequate psychosocial stimulation, to use the WHO phrase, or more simply put, a lack of love, seems to have been a contributing factor to the deaths.

In the United States, one of the first observations of foundling home deaths was made by Harry Chapin in 1915. Chapin, a physician, visited 10 orphanages and hospitals in the New York City region that cared for abandoned infants. These institutions provided an acceptable level of care in terms of hygiene and feeding, yet in 9 of the 10 orphanages all the infants under 2 years old died. In 1942 Harry Bakwin proposed that the cause of this extraordinary mortality was emotional deprivation. In the first half of the twentieth century, the medical community in the United States believed that "excessive" physical contact of an infant and its caregivers was deleterious. In the orphanages and hospitals, where the staff were likely to be overburdened with many infants, physical contact was reduced to a minimum. Bakwin believed that the deprivation of physical contact led to negative emotional state and death.

René Spitz (1887–1974) carefully investigated the causes of poor growth experienced by emotionally disturbed infants and children (Spitz 1945). His

studies focused on orphans confined to foundling homes and other institutions. Spitz quoted a diary entry from 1760 of a Spanish Bishop who wrote, "*En la Casa de Niños Expositos el niño se va poniendo triste y muchos de ellos mueren de tristeza"* [In the Children's Home, the child becomes sad and many of them die of sadness]. Spitz compared the development of infants in a foundling home with infants raised in the nursery of a penal institution for delinquent girls. Inmates of the latter facility were the natural mothers of the infants. Both institutions provided an acceptable standard of housing, sanitation, medical care, and diet for the infants. The children in the penal nursery had more physical and social stimulation, due to their full-time care by their own mothers, or full-time substitutes. In the foundling home, care was provided by one nurse, and infants were confined to their cribs, without human contact, for most of the day. Over the two years of study Spitz found that the foundling home children became progressively delayed in their physical and mental development compared with the nursery infants and a control group of home-reared infants. At about age 3 years the foundling home children had average heights and weights expected for children aged 1.5 years. The developmental status of the nursery infants did not differ significantly from a control group of infants raised at home. Moreover, the mortality rate for infants in the foundling home was 37%, while in the nursery group no child had died. Spitz used the term "hospitalism" to describe the syndrome of poor physical and mental development and high mortality experienced by institutionalized children. It took some time for Spitz's research to make a significant impact, but eventually a new medical paradigm for infant and childcare was proposed by pediatricians. By the 1950s, Benjamin Spock, author of an immensely popular "baby care book," advised parents to hold and cuddle their infants in ways that had been considered indulgent just a few years earlier.

The critical importance of physical contact in early development was the focus of the famous experimental studies conducted by Harry Harlow (1905–1981) and his colleagues (Harlow & Zimmermann 1959; Meyer et al. 1975). Harlow established several types of rearing environments for rhesus monkeys living in a laboratory. Some infants were raised by their mothers, and other infants were separated from their mothers, but given access to inanimate surrogates. One type of surrogate was a wire frame with a bottle and nipple positioned so that the infant monkey could cling to the wire and feed. Another type of surrogate was a wire frame covered with a soft textured cloth. Infant monkeys preferred to cling to the soft cloth surrogate and would even give up feeding for the opportunity to touch and caress the cloth. Behavioral and emotional development was impaired in the monkeys exposed to both types of surrogates, and the infant monkeys had elevated stress hormone levels, but the impairment was more severe in those with wire-only rather than cloth-covered surrogates.

The implications of the research by Spitz and Harlow were applied by Tiffany Field (b. 1942) to the needs of preterm infants. Many preterm infants must be given extraordinary medical care in order to survive. Poor growth and development of those infants that did survive was a common outcome. Often, the medical care required that the infant be isolated from physical contact with the mother or other caregivers. Field conducted a series of experiments which showed that tactile stimulation could ameliorate much of the poor growth and development. The stimulation could be provided by placing an infant confined to an incubator on a sheepskin fur pad or by allowing the infant to be fondled through "glove hole" access into the incubator. As little as 15 minutes of gloved touch resulted in 50% faster rates of growth in the isolated infant (Field 1988, 2007).

What Is More Important, Food or Love?

Green and colleagues (1984) reviewed the literature relating to psychosocial short stature, with an aim toward evaluating the role that nutrition and endocrine factors play in the etiology of the disease. It was found that most cases of growth failure in infants (birth to 36 months) were due to malnutrition; these infants were usually denied food by their emotionally disturbed parents or caregivers. Children over 36 months of age were usually not clinically malnourished. Moreover, it was commonly found that both GH and IGF-1 levels were significantly depressed in these older children. It was known that malnutrition is associated with low levels of serum IGF-1 and abnormally high secretion of GH (reviewed in Chapter 6), so the endocrine profile of the children did not fit with starvation as the cause of their growth failure. Green and colleagues accounted for the growth disturbance in the children with a neuroendocrine hypothesis. They built on the work of Patton and Gardner (1963), who proposed that emotional stress may affect some of the higher brain centers, particularly the amygdala and limbic cortex, which are known to control the emotions. Nerve impulses from these brain centers may pass to the hypothalamus where they are transduced into neuroendocrine messages that may affect the production and release of hypothalamic hormones. In this manner, psychological disturbances in the child might be translated into a cutoff of GHRF in the hypothalamus, a halt in GH secretion from the pituitary, and depressed levels of IGF-1 secretion from the body tissues.

Neuroendocrine mechanisms for the relationship between emotional stimulation, growth, and health were confirmed by both experimental studies with nonhuman animals and in clinical human studies. In a laboratory experiment, Meaney and colleagues (1988) compared infant rats that were licked by their mothers with infant rats who were not licked. The licked infants had lower levels of so called "stress hormones," the glucocorticoids such as cortisol, high levels of GH, high growth rates, and even higher scores on tests of learning.

In later research, Meaney and colleagues (Rostène et al. 1995) reported direct pathways between physical stress, the release of glucocorticoid hormones, and several central nervous system neurotransmitters which regulate the activity of the hypothalamus and pituitary in humans as well as rats.

In clinical human studies, Skuse and colleagues (1996) reported on a group of 29 children with psychosocial short stature and GH insufficiency accompanied with hyperphagia, that is, an excessive intake of food. The researchers found that when these children were removed from their stressful home circumstances the GH levels spontaneously returned to normal and the hyperphagia ended. Clearly, none of these children were denied food, but they were denied proper emotional care and their growth suffered. Studies with infants and children placed into foster care also find little evidence for a nutritional cause for growth retardation. These foster-care studies do find that one of the first physical changes that occurs with placement is an increased rate of growth in height and weight (Wyatt et al. 1997). Wyatt and colleagues point out that few children placed into foster care show signs of clinical psychosocial short stature or sub-nutrition. Their study of 45 apparently healthy and well-nourished infants and children aged 1.5–6 years placed into foster care found more than half experienced clinical catch-up growth following placement. The foster-care study shows that even when stature, weight, and food intake appear to be normal, a stressful home environment may be retarding growth. The catch-up growth experienced by social-upgrading migrants and adoptees described in this chapter are further examples of the effects of stress. There are more recent reports of the harmful growth and development consequences of institutional deprivation and emotional neglect/abuse at home, with the dreadful experiences of Romanian orphans being notable (Kumsta et al. 2017; Rutter et al. 2012; Sonuga-Barke et al. 2017).

A case study, reported by Magner, Rogol and Gordon (1984), provides a final example of the powerful interplay between emotions and growth. The study is of a 12-year-old boy who suffered growth retardation and delayed sexual maturation following an emotional trauma. The trauma was provoked by an argument between the boy and his stepfather, with whom the boy had a warm relationship. After the argument the boy verbalized a wish for his stepfather's death, and the next day the man seriously injured himself falling from a roof. The hospital where the man was recovering sent an erroneous "notice of death" letter to the family's home which the boy received and read while at home alone. Though the man eventually recovered, the boy began a self-imposed period of food refusal and vomiting. He dropped from 34 kg to 25 kg in 5 months. At age 15 years, following periods of hospitalization, drug treatment, and counseling his eating behavior returned to normal. But, his growth did not, and at age 17 years he had the height of a normal child of 11.3 years, a bone age of 13 "years," and was, essentially, prepubertal in physical appearance. He was given treatment with growth hormone at age 19.3 years, and

between ages 20 and 21 years experienced a growth spurt and sexual maturation. Growth in height continued until age 25 years, when the young man reached 171 cm. The authors of this report state that in this patient, an acute "psychic trauma induced a deranged hormonal state that persisted for several years" (p. 741). Though malnutrition and drug treatments in the three years following the trauma may have also upset the hormonal balance, the boy was behaviorally normal and drug-free for about five years before treatment with GH returned his growth and maturation to normal.

This case and the others previously discussed exemplify the intimate and powerful influence that emotional factors, such as love, fear, and guilt, can have on the human neuroendocrine system and the pattern of human growth.

Community Effects and Strategic Growth

Humans are social; social communities rely on signals. Behavioral, linguistic, and nonverbal expressions of love are one type of signal. Human societies are not all about love. Dominance, subordination, and reproductive competition are universal in human societies and underlie social-economic-political-emotional (SEPE) inequalities. Dominance and subordination are linked with physical features such as body size, fatness and fat distribution, muscularity, coloring, distribution of hair and many other physical characteristics. These have been much studied in the nonhuman mammals, including our primate cousins (Clutton-Brock 2016), but are widely neglected in current biomedical studies of human growth and development.

My colleagues Christiane Scheffler, Michael Hermanussen and I have been working for the past few years to understand human anthropometric variation as the outcome of two processes called: (1) community effects and (2) **strategic growth adjustments**. Community effects are part of a hypothesis that there are influences on the attainment of final height, weight, body composition, and body proportions which arise from the social-psychological proximity of members within a social network.

Strategic growth adjustments are observed in nonhuman mammal species where hormonal production and growth rates are associated with position in the social hierarchy. Dominance acquisition often involves changes to achieve larger body size or growth to adulthood at a faster rate. Acquisition or loss of social dominance also is associated with post-adulthood body size changes, body composition changes, and the gain or loss of secondary sexual characteristics in many nonprimate mammals (Clutton-Brock et al. 2006; Huchard et al. 2016; Maggioncalda et al. 2002) and in African monkeys and Orangutans (Emery Thompson et al. 2012; Sapolsky & Spencer 1997; Setchell 2016). Strategic growth adjustments are also known from mammals that shrink and later expand the size of their adult brain and the bones of the skull

encasing the brain in response to temperature and food availability (Dechmann et al. 2017; Lázaro et al. 2017). There is more discussion of body and brain size changes in these species, and in humans, later in this chapter. It is important to emphasize here that until these discoveries it was believed that adult morphology, especially the skeletal size and brain size, of most of mammals were immutable, except for changes related to the deterioration of illness or aging.

Does human growth and development respond to the community effects of social-psychological proximity? Can humans undergo strategic growth adjustments in response to acquisition or loss of dominance? The answers seem to be "yes"!

Height, Width, and Social Class

In 1925, Schlesinger reviewed then current knowledge of a chapter titled "The influence of the social environment on the growth of the children" (*der Einfluss des sozialen Milieus auf das Wachstum der Kinder*, 1925). He explicitly mentioned the shorter but stouter body of children of the lower social class of cities and of rural children compared with children of higher social class, who were taller and thinner. In his 1962 textbook *Growth at Adolescence*, Tanner reviews several German and British studies that report similar differences. Tanner wrote " . . . there is evidence that the upper socio-economic classes have less weight-for-height than the lower ones, and are therefore more linear . . . " (p. 140). None of the studies reviewed by Tanner could find differences in fatness between children of the upper and lower social classes before puberty. Tanner concluded that, "The greater weight for height of the less favoured groups seems to reflect a greater breath of skeleton and perhaps also muscles; or put another way, a lesser growth of the skeleton in length for its breadth" (Tanner 1962, p. 140). Tanner speculated that part of the difference might be nutritional, but he discussed at greater length the impact of social class itself on growth in height vs. width. After all, neither nutrition nor infection can explain why upper-class children grow more in skeletal length, but lower-class children grow more in skeletal width (e.g., shoulder or pelvic breadth) and have more muscle. Could this be a community effect interacting with a classic life history trade-off? Upper-classes grow to a social-psychological norm of tallness, with a trade-off in skeletal breadth, while lower-classes grow to a social-psychological norm of robustness, with a trade-off of shorter stature. Tanner also speculated that the social class differences arose from societal biases in education and occupation that promoted the taller, but thinner, to move up in social class while the shorter, if more robust, moved downward.

In the previous edition of this book I reviewed research showing this is certainly true (Bogin 1999b, pp. 324–328) and I concluded that the concrete result of these biases, just as for the discrimination stemming from racial prejudice, is that individuals or groups of the accepted "type" are more likely to receive better care, in the widest sense, than individuals or groups of the undesired type. A positive feedback relationship between growth and socioeconomic status results from the social bias, better environmental conditions lead to larger size, taller individuals tend to rise in socio-economic status (SES), and higher SES leads to better environmental

conditions. An opposing cycle exists for those from lower SES, or shorter individuals from any social class. The result is that differences in physical size between individuals are both a consequence and a cause of socioeconomic effects on growth.

In the last five years or so important new theoretical modeling and experimental research reports that in addition to "better environmental conditions," community effects and strategic growth adjustments play important roles in the size and shape differences between human social groups.

Dominance Leads to Greater Size in Meerkats

Direct experimentation with humans is often unethical and time inefficient, because people grow slowly. Experiments with nonhuman animals are needed. A recent study with free-living meerkats (*Suricata suricatta*) helps to provide clarity on how social rank relates to growth in body size. The authors report that social dominance itself may be a strong stimulus for growth (Huchard et al. 2016). Meerkats live in hierarchal groups of some 30–50 individuals. The social rank of an individual is based on age and weight. Slowly growing animals may lose their dominant position to faster growing "challengers." Because social position determines access to mates and reproductive success, it may be expected that evolution would favor competitive growth strategies. Such strategies were found in an experiment with a community of wild South Africa meerkats. Pairs of same-sex juvenile (i.e., feeding independent) littermates, brothers and sisters born of the same parents, were divided into a food-supplemented group comprised of the lower body weight individuals, labeled "challengers," and an unsupplemented group comprised of the higher body weight individuals, labeled "challenged." The challenged individuals would normally be more likely to become socially dominant adults. Changes in their body weight were measured over three months. Animals were trained to climb onto a laboratory balance to receive drops of water for all the meerkats, or extra food for the challengers. As the challengers increased in body size due to receiving the food supplements, so did their unsupplemented challenged siblings. The increase in body size required the challenged to eat more, but their growth rate increased first and then food consumption followed.

The growth of newly dominant challenged individuals was followed for the next five months. A secondary phase of accelerated growth occurred and was most " . . . pronounced when the heaviest same-sex subordinate was closer to their own weight at the time of dominance acquisition" (Huchard et al. 2016, p. 534). The authors concluded that meerkat juveniles can strategically adjust their growth in relation to the size of close competitors in their community. Once dominant, meerkats further adjust growth, as if they do not want to be displaced. It is known that dominant, breeding meerkats of both sexes have higher plasma levels of sex steroids and cortisol than subordinates. It is also known that these hormones interact and regulate insulin/IGF-1 pathways and, therefore, may be linked to the changes in growth (Bogin et al. 2015). The authors conclude that a new perspective on social

competition is needed, one that replaces "greater-size-leads-to-dominance" with "dominance-leads to greater-size."

Analogous observations are known from human growth and development. People from socially dominant families, such as European aristocrats, are usually taller than people of lower social status. One example is the analysis by Komlos and colleagues (1992) mentioned in Chapter 1 of the growth of German boarding schoolboys educated in Stuttgart. Recall that the pupils of this school included sons of the nobility (the high aristocracy), the low aristocracy, and the bourgeoisie (lower socio-economic status). Between the ages of 6–23 years the boys were measured at irregular 3–12 monthly intervals. Measurements took place from the year 1771 to 1793. At age 18 years, mean height of all boys was 165.2 cm (min 147.4 cm–max 182.6 cm). As shown in Figure 1.5, the boys of the high aristocracy were some 10 to 15 cm taller and matured 1–2 years earlier than their low aristocratic and bourgeois classmates. All the boys shared lifestyle and the same nutritional conditions. They slept in the same dormitories, so were exposed to the same illnesses and pathogens. More to the point, all the boys knew their own social status and the status of the others. Is it possible that this knowledge produced community effects so that the boys grew in height toward their social status? Is it possible that the social dominance of the higher-class boys imposed stresses on the lower-class boys that repressed their height growth? We will never know from this eighteenth-century example, but supportive evidence does exist from contemporary studies and is presented in the next section.

Can You Shrink and Regrow Your Brain?

Several mammals can do so and also decrease and regrow the size of the bones surrounding the brain. One species is the shrew (*Sorex araneus*), a small molelike mammal native to all parts of the world except Australia and Antarctica. Lázaro and colleagues (2017) captured, measured, released, and later recaptured the same 12 individuals of both sexes over a year's time. Based on repeated X-ray images they found that " . . . individuals decreased the size of their braincases in anticipation of winter by an average of 15.3%. Braincases then partially regrew in spring by 9.3%. Body mass decreased by 17.6% and then dramatically increased by 83.4% in spring" (p. R1106). The changes in body mass were known previously and hypothesized to be a strategy to survive the cold and limited food available during winter, as an alternative to hibernation or migration. The profound increases in brain mass and skull were new discoveries that the researchers suggest are more closely related to reproductive competition in the spring months when shrews of " . . . both sexes expand and aggressively defend their territories" (p. R1107).

Similar changes to brain and skull size were reported for the weasel species *Mustela nivalis* (Dechmann et al. 2017). Using high resolution CT scans the researchers found that the weasels' braincase depth shrinks by 16% and regrows by 8% from winter to spring, but only for males. Shrews and weasels are phylogenetically distinct, with the later in the Order Carnivora, including badgers, otters, ferrets,

martens, minks, and wolverines. The strategic growth of both shrews (Order Eulipotyphla, including hedge hogs, gymnures, and moles) and weasels indicates convergent evolution and opens the possibility for similar biological capacity in other mammalian species, perhaps even humans.

On learning of the adult shrew capacity to shrink and regrow brains, one colleague sent me the following comment:

Think social; be happy with your crews
And shrink your brain like shrews
You'll lack lost hopes
Have not Kings nor Popes
And you'll never be bothered by IQs
Michael Hermanussen

And on to Humans

There is evidence that adolescent and adult humans shrink and regrow brain size. The marked increase in the production of sex steroid hormones, especially estradiol, following puberty is associated with many physical, behavioral, socio-emotional, and cognitive changes. Imaging studies report reductions in grey matter (GM) volume, total brain surface area, and cortical thickness (Hoekzema et al. 2017). Pregnancy also brings about many of these same hormonal changes. Using three-dimensional magnetic resonance imaging, Angela Oatridge and colleagues (2002, p. 19) documented " . . . a reduction in brain size during pregnancy that was maximal at term and that reversed by 6 months after delivery." Elseline Hoekzema and colleagues (2017) confirmed the brain size reduction and noted that it occurred only for primiparous women. As production of sex steroids increased there was a reduction of GM in a symmetrical pattern across both sides of the brain " . . . primarily affecting the anterior and posterior cortical midline and specific sections of the bilateral lateral prefrontal and temporal cortex" (Hoekzema et al. 2017 p. 7). At a two-year follow-up the brain size reductions of pregnancy were still measurable. Nulliparous women with pregnant friends and first-time fathers did not show these brain changes, so it is most likely that hormone changes of pregnancy and not the experience associated with impending parenthood are the cause. The brain regions most affected by the size reductions are known to be involved in important social processes that underline human biocultural reproduction. Hoekzema and colleagues proposed that during both adolescence and pregnancy the changes in size and organization of the brain are a type of strategic growth adjustment for the reorganization of social behavior. The newly pregnant women will be better equipped " . . . to recognize the needs of her highly altricial child, to decode social stimuli that may signal a potential threat, or to promote mother–infant bonding" (p. 8).

My colleagues and I are applying the principles of strategic growth adjustments and community effects to human growth and development. We are reinterpreting the significance of differences in size, body composition, and rates of maturation. The conventional wisdom is that these differences are due primarily to factors such as genetics, nutrition, infection, climate, and migration. Each of these is important, but as was shown in previous chapters the genetic, climatic, and altitude influences on growth and development tend to be much less influential than once thought. In the remainder of the present chapter the associations of human growth and development with some of the conventional factors of nutrition, seasonal variation,

migration, urbanization and modernization, and socioeconomic status will be reviewed. Each topic is evaluated in relation to community effects, strategic growth adjustments, and human life history biology. Some aspects of these topics were discussed in previous chapters in the context of social-economic-political-emotional (SEPE) influence on growth and are treated more formally and intensely in the present chapter.

Environmental Factors Not Discussed

The environmental factors of pollution and sleep, both duration and quality, are important and have effects on human growth, development, and maturation. The chemical pollutants derived from industrial processes, from use of fossil fuels, from use of internal combustion engines of automobiles, trucks, and airplanes, and the noise pollution that these produce are all toxic to humans and interfere with healthy growth and maturation. Pollution and human growth are well reviewed by Lawrence Schell and colleagues and others (Schell 1991; Schell et al. 2009, 2012; Zheng et al. 2016). Most of the research on sleep and growth relates to risks for overweight/obesity (Chaput et al. 2017). There are few studies on growth in length/height. These studies report a " ... small but significant relationship between sleep and growth anthropometric measures in early life [that] might be amplified in later childhood" (Zhou et al. 2015). Pollution, sleep, and other environmental factors, such as climate and altitude which were discussed in Chapter 5, are not treated further in this book.

Nutrients and Food

In the growing human being, the multiplication of cells or their enlargement in size depends upon an adequate supply of nutrients. Nutritional biochemists have determined that the are about 50 essential nutrients required for growth, maintenance, and repair of the body. Essential nutrients are those substances which the body needs but cannot manufacture. There are about 50 because some substances may be essential but a minimally required amount has not been determined. The 50 substances are divided into six classes: protein, carbohydrate, fat, vitamins, minerals, and water. Shown in Figure 7.1 are the essential nutrients in these categories.

Why we have so many essential nutrient requirements is a topic that I have written about elsewhere (Bogin 1998a, 2001, pp. 143–163). Briefly, several sources of data may be considered in the study of the evolution of human nutrition. Archaeological and paleontological evidence provide the only direct data on what our ancestors ate and what effect diet may have had on our physical, behavioral, and emotional evolution. But those data are limited and difficult to interpret. The presence of a food item in the archaeological record does not tell us how, when, and by whom a food was eaten. Extracting food remains from between fossil teeth and from fossilized feces, called coprolites, tells us that the individual in question did use a species, but even then, it is not certain if the species was food, a medicine, or used for some other purpose. Stable isotopes of carbon and nitrogen, which are incorporated into teeth, are very useful indicators of types of plants and the relative quantity of

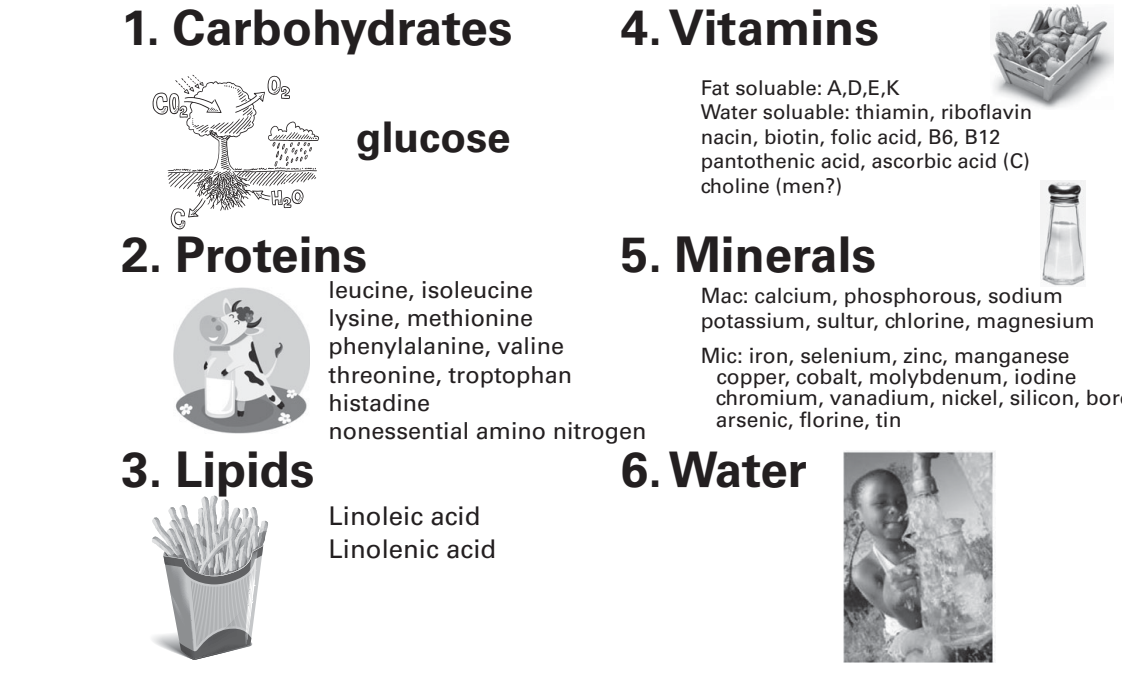


Figure 7.1 Essential nutrients grouped into six categories. The image under "Carbohydrates" is a cartoon of photosynthesis. Under "Proteins" are the nine essential amino acids required during the years of body growth. Under "Lipids" is a container of fried potatoes which due to the absorption of the frying oil provides most of its energy from lipids. The image under "Vitamins" is of fruits and vegetables which are sources of the essential vitamins. The image of a saltshaker indicates that table salt provides calcium and phosphorous. "Mac" is an abbreviation for macromineral and "Mic" is an abbreviation for micromineral. The "Water" image indicates that safe tap water is needed for all people. When safe to drink, tap water is preferred to plastic bottles and is often of higher quality. Credit FrankRamspott / DigitalVision Vectors / Getty Images (Carbohydrates); nafanya241 iStock / Getty Images Plus (Proteins); zbruch / DigitalVision Vectors / Getty Images (Lipids); Peter Dazeley / The Image Bank / Getty Images (Vitamins); Brian Hagiwara / The Image Bank / Getty Images (Minerals); David Malan / Getty Images (Water).

plant vs. animal foods consumed. Analysis of the isotopes also indicates the dental age when weaning took place. Studies of living primates and other mammals, living hunting-gathering societies, and cross-cultural comparisons of cuisines provide indirect evidence that is useful to reconstruct human nutritional history.

One way that nutrients are shown to be essential is via experiments with nonhuman animals. A young rat, pig, or monkey is fed a diet that includes all the known nutrients except the one being tested. If the animal gets sick, stops growing, loses weight, or dies it usually means that the missing nutrient is essential for that animal. Such experiments do not prove that the same nutrient is needed for people, but due to the close biological relationship between monkeys, apes, and people it is likely that nutrient requirements are similar, if not identical. This does not mean that all primate

species should eat the same diet as, for example, a diet as high in animal protein as eaten by many human groups will kill chimpanzees (Finch & Stanford 2004). The primate similarity means only that monkeys, apes, and humans share the same 50 essential nutrients requirements. Another way to discover essential nutrients is that certain medical conditions deprive people of one or more nutrients and the consequence of deficiency helps to prove the essential nature of those nutrients. For example, lactase deficiency leading to lactose intolerance, trauma or surgery, infection, prolonged use of antibiotics, celiac disease, Crohn's disease, chronic pancreatitis, cystic fibrosis, and parasitic diseases can cause malabsorption of one or more nutrients from the intestinal tract. Finally, controlled experiments were done in the twentieth century with humans, such as with children at orphanages, patients at psychiatric hospitals, people incarcerated in prison, and with residents of villages in colonized or low-income nations. Most of these experiments were unethical as they were usually performed without informed consent and often without any understanding or knowledge by the participants.

An experiment of questionable ethics changed the medical classification of pellagra from an infectious disease to a nutritional deficiency. The search for the cause of pellagra was a biocultural detective story (Hung 2018). Pellagra is a disease due to insufficiency of niacin (vitamin B_3). The classic progression of pellagra is dermatitis, diarrhea, dementia, and death (the "four Ds"). The disease was epidemic in the early twentieth century among low-income people in the south of the United States, Italy, and other regions with high levels of poverty. The medical establishment was certain that pellagra was caused by an infectious agent and not related to diet. In fact, it was caused by poverty, which forced people to eat a cheap, monotonous diet based on corn meal, fatback, and molasses. Interventions that added milk, eggs, peas, and beans to the diet of people confined to orphanages, hospitals, and prisons in the United States eradicated pellagra. Those interventions were carried out in 1914–1915 by Joseph Goldberger,[3] but his nutritional cure was rejected by the "experts" and by the institutions where Goldberger had worked, whose administrators reinstated the old diet. It was not until 1937 that niacin was biochemically "discovered" in the laboratory and proved to be the pellagra factor. The pellagra story is one of many that illustrate the ways that social, economic, political, and emotional conditions of life deprive people of food and nutrients.

The history of discovery of other nutrient-deficiency illnesses, such as scurvy (vitamin C), beriberi (thiamine), rickets (vitamin D), and goiter (iodine), is similar to that of pellagra in that the cause is biocultural. The people suffered from the nutrient deficiency, in most cases, because they were dislocated from their traditional way of life and diet, usually by colonization, poverty, social class divides, war, forced migration, and segregation/incarceration into isolated communities. These people could no longer acquire their traditional diet which provided all needed nutrients – if it had not, then the people would have died. In some cases people suffered nutrient

[3] https://history.nih.gov/pages/viewpage.action?pageId=8883184

deficiencies due to more natural causes, such as iodine deficiency experienced by people living in mountainous regions of the Andes, the Alps, the Derbyshire Hills of central England, and highland New Guinea where the soil is leeched of iodine.

People do not usually eat the essential nutrients directly as pure chemicals, rather we eat food. This was certainly true for our animal ancestors throughout evolutionary history. Human foods come from five of the traditionally defined six Kingdoms of living organisms:[4] plants, animals, fungi (e.g., mushrooms), protists (e.g., species of algae referred to as "seaweed") and eubacteria (e.g., bacteria used in fermented foods). The sixth Kingdom, archaebacteria, are not eaten directly, but are essential in the diet of other species that people do eat. Herbivores, for example, have archaebacteria in their guts to digest plant cellulose. Some people eat the herbivores, such as cows, horses, sheep, deer, goats, and the like. Furthermore, every human society creates a cuisine, that is, a list of acceptable food items, the style of preparing these items, the rules for serving and sharing food, food taboos against the consumption of certain foods based on age, sex, gender, state of health, religious beliefs, and other culturally defined reasons (Pelto & Pelto 1983). In addition, people use food for nonnutritional purposes, such as for medicine to cure or cause disease and as offerings in ritual or religious activities. In these contexts, food may have physiological functions due to their ideological and symbolic meaning. All people have the same nutrient requirements, but the biocultural nature of human nutrition makes the connection between food, growth, development, and maturation hugely complicated to study.

Energy and Growth

Despite the complexity, the adequacy of the total variety and quantity of food consumed are major determinants of growth. This is so because, in part, nutrients may be widely distributed across many different types of food. An adequate diet in terms of food quantity is important because of the energy (kilocalories) that food provides. Different kinds of food can substitute for each other to produce energy. Carbohydrates and proteins in food supply about 4 kilocalories (kcals) per gram, while fats supply about 9 kcal per gram. During the years from birth to adulthood, the human body requires energy for several processes, which may be summarized by the following formula:

$$\text{Energy required} = \text{Maintenance} + \text{Immune function} + \text{Repair} + \text{Work} + \text{Growth}$$

where maintenance means the energy used in basal metabolism; immune function means defense against viruses, bacteria, parasites such as worms and single-celled pathogens, and other disease causing pathogens; repair means the energy used to restore cells, tissues, or systems following disease or damage; and work means the energy used in voluntary activity such as physical labor or play. After these requirements are met any energy that remains may be used for "Growth" which

[4] Biologists have proposed as few as two and as many as eight Kingdoms.

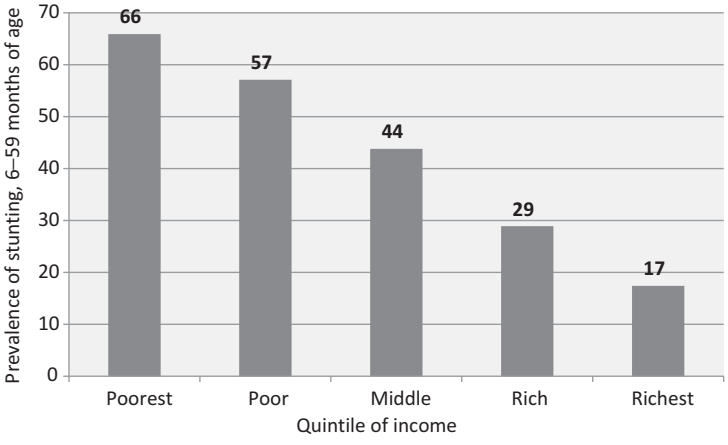


Figure 7.2 Guatemala: prevalence of stunting by quintile of family income. The data come from the Guatemala DHS Key Indicators Report, 2014–15 available at https://dhsprogram.com/what-we-do/survey/survey-display-440.cfm. The figure is based on a World Bank presentation, "Desnutricion en Guatemala" (Malnutrition in Guatemala), January 2016

includes hyperplasia and hypertrophy of cells and tissues, as well as development, and maturation of the person. Energy requirements vary by age, sex, biological maturation status, intensity of physical activity, and health status. A two-year old requires about 1,000 kcals per day, and moderately active 18 year-old women and men need about 2,000 and 2,800, respectively. Energy requirements by age and activity for the US population may be found online.[5]

In populations where food shortages are present growth delays occur, and children are shorter, lighter, and mature later than in populations with adequate or over-abundant supplies of food. Guatemala, where I have worked since 1974, has the one of the highest prevalence of stunting of all nations, affecting 46.7% of all infants and children <5 years old.[6] Recall from the Introduction that "stunting" is the term used to describe very short height, technically a girl or boy whose height-for-age is among the shortest 2.3% (less than 2 standard deviations) of a healthy reference group of children. In rural Maya communities the prevalence of stunting exceeds 60%, as was documented in previous chapters. For adults, the total Guatemalan population ranks 12th shortest for men (mean height = 163.4 cm) and 1st shortest for women (mean height = 149.4 cm) of the 200 nations surveyed by the NCD Risk Factor Collaboration (NCD-RisC 2016). The prevalence of stunting in Guatemalans across all quintiles of family income is illustrated in Figure 7.2. High rates of stunting are present for all economic groups, with up to 17% stunting for infants and children from the highest income quintile families of Guatemalans (i.e., the richest 20%). A deficit of food energy is unlikely for families with incomes in the middle, rich, and richest quintiles

[5] https://health.gov/dietaryguidelines/2015/guidelines/appendix-2/
[6] https://data.worldbank.org/indicator/SH.STA.STNT.ZS?name_desc=false

of earnings. There are many reasons for this very short stature of all Guatemalans unrelated to food. These reasons likely include a variety of SEPE factors, especially the emotional stresses that result from a constant exposure to high levels of violence and insecurity that pervade the entire country. The relationship of emotional stress to stunting is discussed in the following paragraphs.

Food energy shortages, along with infections and psychosocial stress, are more likely for the families in the poor and poorest income quintiles. The World Bank estimated food shortages in a country via a statistic called "the depth of the food deficit." This statistic indicates how many kilocalories would be needed to lift the undernourished to a status of adequately nourished, everything else being constant (such as no infection, no increase in workload, and no psychosocial stress). The average intensity of food deprivation of the undernourished was estimated as the difference between the average dietary energy requirement and the average dietary energy consumption of the undernourished population. The depth of the food deficit in Guatemala was reported at nearly 140 kilocalories per person per day in 2001 and 101 kcal/person/day in 2016, according to the World Bank collection of development indicators.[7] The deficit hits the poor and poorest quintiles of income most strongly. The poor constitute the 56–64% of the Guatemalan population that survive on an income of US$2/day/person. The poorest constitute 21.5% of the population and suffer from an income of US$1/day or less per person (World Health Organization 2012).

In addition to the total energy deficit, the poor in Guatemala suffer micronutrient deficiencies of iron, vitamin A, and iodine.[8] A shortage of any one of these nutrients will reduce body growth and brain development in the growing person from embryo to adolescent. It is estimated that worldwide the most common nutrient deficiencies are iron, iodine, vitamin A, folate, and zinc. Vitamin B_{12} may also be in widespread shortage. Undoubtedly, many infants, children, pregnant women, and others in Guatemala and elsewhere are deficient in one or more of these nutrients. However, a concern about estimates for specific nutrients is that the intake requirements are set at a level above the known biological requirement. The US National Institutes of Health sets the average daily requirement for iron, for example, at 13.7–15.1 mg/day in people aged 2–11 years. The average daily iron intake from foods is 11.5–13.7 mg/day in this US age group. Nearly all these people should be iron deficient, but the prevalence of iron deficiency for this age group is less than 3.5% as assessed by blood tests for anemia (Le 2016). It is unlikely that the other 96.5% take iron supplements. The recommended intakes of nutrients are purposefully set high so if consumed at that level that at least 98% of the human population will be adequately nourished. But, setting the recommendations so high means that deficiency misdiagnosis will also

[7] https://tradingeconomics.com/guatemala/depth-of-the-food-deficit-kilocalories-per-person-per-day-wb-data.html

[8] http://documents.worldbank.org/curated/en/485361468251183980/Guatemala-Nutrition-at-a-glance

be high. This is one reason why nutrient supplementation interventions to prevent or overcome stunting and underweight so often fail – the people were not, in fact, deficient for that nutrient.

An Experimental Study of Nutrition and Growth

Some nutrition interventions do work when the reason for growth failure is a real deficiency of total food or specific nutrient intake. The "Four Village Study" of the Institute of Nutrition for Central America and Panama (INCAP) was described briefly in Chapter 1. Recall that the original study ran from 1969 to 1977 and involved the supplementation of pregnant women and all infants and children with either a high energy drink, with protein, carbohydrates, vitamins, and minerals, or a low energy drink with essentially no protein or carbohydrate, but the same vitamins and minerals. The study took place in a rural region of eastern Guatemala in two large (~900 residents) and two small (~500 residents) *ladino* villages. INCAP personnel screened 300 communities to identify these four villages which were chosen because they met the criteria of " . . . appropriate size, compactness, ethnicity and language, diet, access to health care facilities, demographic characteristics, nutritional status and degree of physical isolation" (Stein et al. 2008, p. 716). The criteria of "appropriate" diet meant that the villagers suffered from a chronic shortage of food intake, especially of protein. In 1969 it was thought that protein deficiency was the most important nutritional problem facing the poor in the developing countries. "Appropriate" nutritional status meant that most infants and children were very thin and stunted. The four villages chosen for the study suffered from extreme economic poverty which caused hunger and growth failure. "Appropriate" ethnicity and language meant that participants be Ladinos speaking Spanish and not Maya speaking one of the many Maya languages.

One large and one small village received the experimental supplement called *atole*, which is the local name of a thick, corn-based beverage, and the other two villages received *fresco*, the local name of a drink made from watered-down fruit juice. The atole used in the intervention was a gruel-like drink made from Incaparina, a proprietary formula developed at the INCAP laboratories. Incaparina is composed of a vegetable protein mixture (a cereal), dry skimmed milk, and sugar. In the other two villages, residents were given an INCAP formulated *fresco*, a drink that contained energy from sugar, but no protein and at the start of the study no other nutrients. From October 1971, both supplements were fortified with micronutrients in equal concentrations. The atole drink provided 163 kilocalories (kcal) per cup (180 ml) from 11.5 g of protein and 27.8 g of carbohydrate. The *fresco* provided 59 kcal per cup from 15.3 g of carbohydrate.

The study participants were pregnant women, infants, and children who were given the drinks twice a day at feeding stations in the villages. The participants could consume all they wished, and intake was carefully monitored. The infants and children participating in this original INCAP study were followed until they

were seven years old. Results of this study showed that pregnant women receiving the atole drink gave birth to newborns with higher birth weights and lower infant mortality. Infants receiving the atole had greater weight and length-for-age until age three years (Martorell 1995). Analysis of these findings showed clearly that it was the total energy (kcal) added by the atole supplement, and not the amino acids of the protein per se, that was associated with the greater weight and length. Since the time of the start of the INCAP study (early 1970s) the recommended amounts of protein intake for infants dropped from more than 3 grams per kilogram body weight per day (g/kg/day) to the current estimated average requirement 1.52 g/kg/day for infants 0–6 months, 1 g/kg/day for infants 7–12 months, and 0.87 g/kg/day for infants 13–36 months old. Even so, the powdered skim milk component of the atole may have added one or more growth factors associated with milk that resulted in greater length and weight. Milk growth factors are discussed later in this chapter.

Several follow-up studies have been conducted with the original INCAP participants. One follow-up took place from 1988 to 1989 to assess participants who were then 11–27 years old (Martorell, 1995). The reason for the follow-up was to see if nutritional supplementation up to age seven years had long-term effects. The answer is yes, there are several long-term effects of the atole drink, including greater stature and fat-free mass (the weight of the skeleton, muscles, and organs of the body), especially for girls and women. Atole users also showed improved work capacity in the males and higher scores on tests of intellectual performance in both males and females. The atole group did not experience a faster rate of maturation, as measured by either skeletal development or age at menarche, than the fresco group. More recent follow-ups reported that the atole group had higher scores on both a measure of reading comprehension and on the Ravens' Progressive matrices, a type of cognitive performance (IQ) test. Participants receiving atole between 0 and 24 months of age had a 46% increase in net wages as adults. There was concern that the enhanced feeding of children might promote obesity and metabolic disease in adults (Stein et al. 2008). Between the years 2015–2017 a sample of 683 women and 456 men, aged 37–54 years, from the original intervention cohort were assessed for anthropometry, fasting and post-challenge glucose, fasting lipid concentrations, and blood pressure (Ford et al. 2019). Those who had received atole from conception to age 2 years had increased fatness, including greater risk for obesity. But reduced risk for diabetes.

Some researchers concluded that the INCAP intervention study showed clear benefits of improved nutrition at all stages of the life cycle from fetal life to adulthood. Another interpretation is that providing more food to the clearly underfed people of the four fastidiously selected villages has a beneficial effect. Would the same atole supplement improve growth and development in the 296 villages not selected? Would the intervention have a similar effect in Maya villages? These questions are not possible to answer, but it is known that the height growth status of the Guatemalan population has changed little in the past 100 years (NCD Risk Factor Collaboration (NCD-RisC) 2016).

Infection and Psychosocial Stress in Guatemala

The WHO states that in addition to malnutrition, repeated infection and inadequate or inappropriate psychosocial stimulation can cause stunting. Between 1960 and 1996 Guatemala suffered from both repeated bouts of infections and an inappropriate psychosocial environment due to civil war. The war resulted in more than 200,000 deaths and millions of displaced people, especially from rural villages that were targeted for destruction by the military (Lovell 2010). In addition, the 1976 earthquake of moment magnitude 7.5 killed at least 23,000 and injured at least 76,000 people. Many thousands were left homeless, especially in rural areas. The civil war and the earthquake forced many rural families to migrate to Guatemala City to live in slums and other informal settlements as there were no other places to live. It is estimated that Guatemala City had a population of 285,000 inhabitants in 1950; 573,000 in 1964; 1,202,536 in 2006; and 2,450,212 in 2018. The total population in Guatemala was estimated at 17.3 million people in 2018. About 42%, or about 7.2 million people, live in the greater metropolitan region. Guatemala City's population continues to increase with the influx of predominantly indigenous migrants from the other departments as well as people from other countries. By the year 2003 the city was characterized by a large horizontal expansion, an inefficient public transportation system, a decrease in state attention to housing needs, and a proliferation of precarious settlements (slums). Of Guatemala City's 2.45 million inhabitants, approximately a third live in precarious settlements.[9]

The civil war was most intense from 1978 to 1985 and led to a decline of the Guatemalan economy, massive urban migration, the growth of urban slums, and an increase in infectious disease. The Pan-American Health Organization (PAHO) country profile for Guatemala in 1991 reported that for the years 1988–1990 the two leading causes of death were respiratory infections and intestinal infections. The death rate from measles rose during the years 1989–1990 and the number of malaria cases rose from 41,711 in 1990, to 57,560 in 1992. There was an "alarming rise" of acute malnutrition for infants and children under 5 years old as assessed by weight-for-height. In 1979 the prevalence of endemic goiter due to iodine deficiency was 8% of the Guatemalan population but by 1989 had increased to 20.4% as a consequence of notable deterioration in the salt iodization program (Bogin & Keep 1999).

The rural, especially rural Maya, population was affected most strongly by these generally negative changes in health and nutrition during the 1980s and 1990s. However, the civil war and the collapse of the Guatemalan economy after the war brought a decline in health and nutrition to the urban population as well. Even the children of the wealthy may have suffered as the safety of the water and food supply of the country was compromised during the most intense periods of social, economic, and political instability during and after the civil war. The quality of urban water and food did decline in Guatemala during the 1980s and was associated with the outbreak

[9] www.ucl.ac.uk/dpu-projects/Global_Report/pdfs/Guatemala.pdf

of cholera in the 1990s. Quoting again from the PAHO country report for Guatemala: "In response to the cholera epidemic that struck the country in 1991, the use of chlorine in municipal water systems has doubled. In 1991, 48 systems were chlorinating their water supplies and by 1992 the number increased to 94; nevertheless, only 45% of the total urban population is served by those 94 systems."

The PAHO report concludes that in addition to cholera contaminated water, the food supply was a source of infection: "With regard to food sold by street vendors, 52% of the samples from the departments [rural areas] and 48% of the samples from the capital [Guatemala City] were found to be microbiologically acceptable in 1992."

Those figures represent an improvement over the 34% level of acceptability found in 1991 (Bogin & Keep 1999). Street vendor food is consumed by all segments of the Guatemalan population, especially school-aged children of all economic levels. Even with some improvement, the 1992 data indicate that about one-half of this food was contaminated as recently as 1992. Thus, Guatemalans of all ages, ethnicities, sexes, and family income levels were subjected to risks for health from food and water. My ethnographic experience in Guatemala provides ample evidence that people know of these risks. This knowledge, as well as the biological contaminants, create a climate of insecurity that compromised growth and resulted in the high prevalence of stunting for the nation.

Following the end of the civil war in 1996 there was a rise in organized crime and corruption related to drugs and human trafficking. The crime and gang violence were, and still are, symptoms of much larger structural problems in the government and society, " ... including deepening economic inequalities, the erosion of political and social infrastructures and disparate access to healthcare and education" (Thomas & Benson 2008, p. 39). The murder rate of women in Guatemala is the third highest in the world with an average of 755 violent deaths/year of women in the years 2014–2016.[10] Between 1991 and 2001 the rate was about 200 murders of women per year. The number of killings increased to just over 600 by 2006, according to a 2007 study by UN Rapporteur Philip Alston. Alston noted that "the death toll is only the beginning of the cost, for a society that lives in fear of killing is unable to get on with its life and business in the ways that it wants." (Sanford 2008, p. 21). Life and business in Guatemala have been insecure for everyone due to crime at the highest levels of government. In 2015 the Guatemalan president and vice-president were arrested for crimes of corruption and money-laundering. The Guatemalan National Postal Service was suspended for two years following its involvement in similar crimes, resuming some services in April 2019.

In addition to the crime and corruption suffered by the poorest to richest in Guatemala, the wealthy live under the threat of kidnappings for ransom. These kidnappings are so common that in my personal experience every richer Guatemalan family I know has had at least one family member kidnapped. Rich and richest

[10] www.theguardian.com/healthcare-network/2018/mar/07/health-workers-stop-thousand-women-killed-guatemala-femicide

families send their children to school in bullet-proof vehicles accompanied by armed guards. The stress of real and threatened violence is pervasive and is part of the lives of people of all ages.

It Is All About Stress

These insecurities from endemic corruption, violence, and kidnappings are forms of inadequate and inappropriate psychosocial stimulation, commonly referred to as toxic emotional stress and adverse childhood experiences (ACEs).[11] This type of stress takes a toll on human health, including the physical growth of people, as much as do food shortages and infection. ACEs and toxic emotional stress are associated with susceptibility to disease (Chrousos & Gold 1998), disregulated gene expression (Slavich & Cole 2013), and low birthweight (Varea et al. 2016). Too often the toxic emotional stress is exacerbated by food shortages, infection, and other health problems, a combination that is especially harmful – even deadly.

Toxic stress and ACEs increase the production of cortisol, the catecholamines dopamine, norepinephrine, and epinephrine (adrenalin), and glucagon. Recent experimental evidence with rodents and clinical observations with human patients indicate that various types of physical and emotional stress also cause a rapid rise in osteocalcin (OC) release into the blood stream and that this OC is needed for the acute stress response (Berger et al. 2019). Higher serum levels of these hormones induce a hypermetabolic state of catabolism – the breakdown body cells to liberate amino acids, fatty acids, and glucose from body cells and tissues for a response to the stress. This hypermetabolic state is called the acute stress response (ASR). In the short term, the catabolism of the ASR may be beneficial for immune response, wound repair, and dieting for weight loss. In the long term the consequences are harmful because a chronic stress response results in permanent loss of tissue and growth stunting (Arlt & Stewart 2005; Christiansen et al. 2007; Matthews & Battezzati 1993). The relationship of toxic stress and ACEs to bone growth is illustrated in Figure 7.3. Also shown in the figure is the impact of stress on adipose tissue and body fatness, which is discussed in more detail in Chapter 8.

The physiology and metabolism of stress are well studied in living people in both the wealthy and poorer nations and have been noted in skeletal and dental remains. Julia Beaumont and colleagues have confirmed the catabolic effects of chronic nutritional, infectious, and emotional stress in medieval archaeological populations and in living people with low SEPE resources, such as low-income families in the United Kingdom (Beaumont et al. 2018). Another analysis of infant and child skeletal remains is based on ~300 victims of the Great Irish Famine (1845–1852). The skeletons were excavated from the mass burial ground of the workhouse in the city of Kilkenny and were deposited between 1847 and 1851 (Geber 2014). It was found that starvation and infectious diseases were likely to have greatly contributed to

[11] https://developingchild.harvard.edu/ACEs

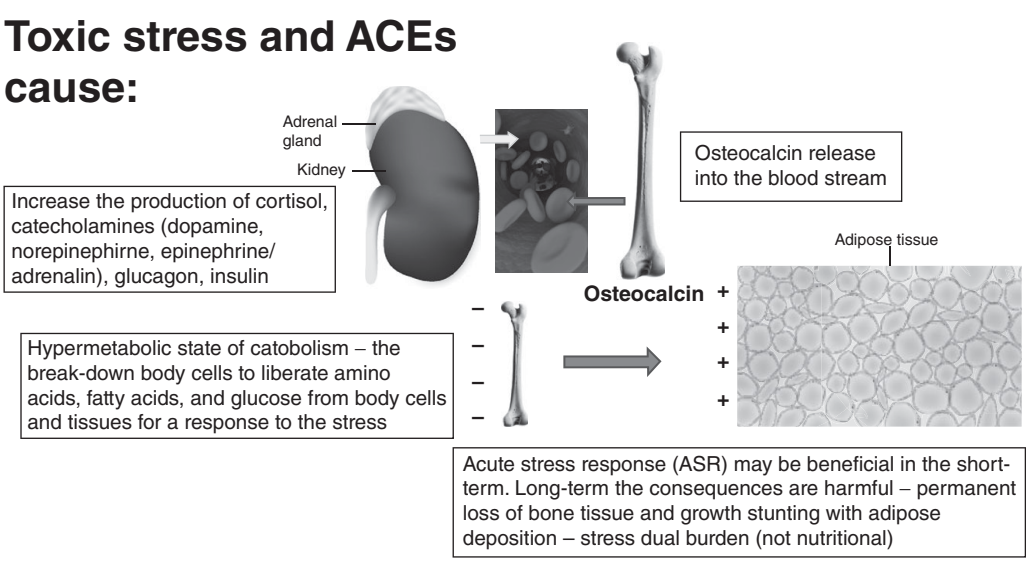


Figure 7.3 The relationship of toxic stress and ACEs to bone growth and body fatness. Credit ttsz / iStock / Getty Images Plus (Kidney); idcde / iStock / Getty Images Plus (Blood vessel); SCIEPRO / Science Photo Library / Getty Images (Femur); Benjamin Toth / DigitalVision Vectors / Getty Images (Fat cells).

skeletal pathology, growth stunting, and death of these 1–5-year-olds. In addition, Geber emphasized that " . . . the psychosocial stress relating to institutionalization in the workhouse should not be underestimated as a substantial causative factor for skeletal stress in this population" (p. 149).

Additional biocultural mechanisms by which social and emotional insecurities inhibit physical growth work through community effects and strategic growth adjustments. Details of these mechanisms are given later in this chapter.

Material and Emotional Security

The recent history of Guatemala is, unfortunately, repeated in many parts of the world. There are daily news reports of civil war, violence, exposure to infection, disruption of food supplies, and toxic emotional stress in dozens of nations. For health, happiness, and hope people require material and emotional security, and these are based on the reliability of an adequate diet, protection from infection, safe housing, protection from forced migration, and a meaningful livelihood. Material and emotional security are the basis of successful human biocultural reproduction, family care, and healthy growth, development, and maturation of young people of all ages before and after birth. The need for these factors has been known for hundreds if not thousands of years. The public health research of René Villermé (1829) with French military conscripts and of Edwin Chadwick (1833) with English factory

children, reviewed in Chapter 1, emphasized the harmful impact of poor social conditions on healthy systems of biocultural reproduction, and not just hunger, as causes of poor growth. In his 1842 *Report on the sanitary conditions of the labouring population of Great Britain*, Chadwick found that there was a link between poor living standards, the spread of disease, feelings of hopelessness, and poor physical growth. He reported that unsanitary conditions in Britain's urban slums, into which rural peoples had been forced to migrate due to evictions from their homesteads, had a demoralizing effect on the families affected. The Health Foundation, a UK charity, explains that Chadwick recommended " ... the government should intervene by providing clean water, improving drainage systems and enabling local councils to clear away refuse from homes and streets." To persuade the government to act, Chadwick argued that the poor conditions endured by impoverished and ailing labourers were preventing them from working efficiently.[12] Chadwick explained that it was in the selfish interest of the wealthy factory owners to eliminate poverty.

Chadwick's advice is as pertinent today as it was 178 years ago. Despite many important historic improvements to public health in Britain and other industrial nations during the past two centuries, poverty, unsanitary conditions, and hunger persist today in the low-income regions of all nations. Residents of impoverished cities and rural areas in the United States suffer persistent food insecurity and unsafe drinking water – the most notorious current example is Flint, Michigan. An estimated 11.8 percent of US households, totaling 40 million people, were food insecure at least some time during the year in 2017, meaning they lacked access to enough food for an active, healthy life for all household members.[13] In the United Kingdom, a nation with universal social support and health care, school children from low-income families come to school hungry. They often have not had a proper meal since their last school lunch. During weekends and school holidays some suffer severe hunger (Holley & Mason 2019). The United Kingdom ranks 4th worst of all nations in Europe for people under 15 years old at risk for hunger, behind Romania, Bulgaria, and Lithuania.[14] People in the US, the UK, and other nations with high prevalence of food insecurity have higher risk for overweight, diabetes, and some form of physical or emotional/psychological disability. These health risks are inter-related in that the food insecurity leads to emotional stress and unhealthy food choices of energy-dense but nutrient poor foods (diets with lots of carbohydrates and fats with few fruits and vegetables).

In the low-income nations of Africa, Asia, and Latin America the quality of life for the poor is even worse. Both rural and urban poor, especially those families living in slums, face insecurities from inadequate water and sanitation, called WASH by the public health researchers and policy makers, and inadequate

[12] https://navigator.health.org.uk/content/edwin-chadwicks-report-sanitary-conditions-labouring-population-great-britain-was-published

[13] www.ers.usda.gov/publications/pub-details/?pubid=90022

[14] 2019 UK Parliamentary Business Report https://publications.parliament.uk/pa/cm201719/cmselect/cmenvaud/1491/149105.htm

amounts, quality, and diversity of foods. Much effort and money has been spent to improve WASH and nutritional status in these low-income areas. Effectiveness is often assessed via measurements of growth, particularly reductions of underweight and stunting.

A Review of Failed Attempts to Overcome Insecurities and Poor Growth

Many attempts have been made to promote health growth in impoverished communities. To assess the impact of these attempts researchers often make use of a systematic review methodology. *Cochrane Database Systematic Reviews* (*Cochrane Reviews*) are systematic reviews and metanalyses of primary research in human health care and health policy and are internationally recognized as the highest standard in evidence-based health care. *Cochrane Reviews* provide a transparent and objective evaluation of the effectiveness of treatments designed to improve health. There are several *Cochrane Reviews* of intervention studies to reduce underweight and stunting available in the literature. Sometimes the interventions were effective to increase weight, especially fatness, but most were ineffective to increase length-for-age of infants or height of children.

In one *Cochrane Review*, Sguassero, de Onis, and Carroli (Sguassero et al. 2005) reported a positive effect on length (cm) in a nutrition-supplemented group compared to controls (mean difference 1.3 cm, CI (0.03–2.57 cm) after 12 months of an intervention conducted in Jamaica, but no similar benefit in growth after 12 months of supplementation in a trial from Indonesia. In 2012, the same authors (Sguassero et al. 2012) updated their *Cochrane Review* of community-based supplementary feeding in children under five years of age in low-income and middle-income countries with a meta-analysis of eight intervention studies. They reported that the mean difference in length or height growth between intervention and control groups was +0.48 cm (95% CI 0.07–0.89) after 3 months of intervention and +1.3 cm (95% CI 0.03–2.57) after 12 months of intervention. The greater average increase of the supplemented groups was statistically significant in some cases but was quite small and highly variable in practical biological terms. Moreover, the studies employed diverse methodologies that were not readily comparable, were poorly designed to eliminate biases of various types, and were under-powered due to small sample sizes. The authors concluded that given the findings and the important limitations, " . . . supplementary feeding has a negligible impact on child growth" (p. 1).

Alan Dangour and colleagues completed a Cochrane review of interventions to improve WASH conditions (Dangour et al. 2013). The authors reported that there was no overall statistically significant improvement in weight-for-age, with a mean difference of +0.05 z-score between samples of infants and children who received a WASH intervention vs. control samples that did not receive an intervention. Height-for-age showed an overall improvement for the intervention samples, but the amount of improvement was only 0.08 z-score which is statistically significant but of negligible biological importance because it is less than

measurement error. Carefully trained technicians can measure an infant's length or a child's standing height to within 0.03 cm (3 mm). A height-for-age z-score of 0.08 for under 5-year-olds equates to no more than 0.2 cm. Worse still, several of the intervention studies reviewed by Dangour's team resulted in decreased weight-for age and height-for-age.

Another *Cochrane Review* set out to assess the effectiveness of supplementary feeding interventions to improve the physical and psychosocial health of disadvantaged children aged three months to five years in low- and middle-income countries (Kristjansson et al. 2015). The authors also assessed the potential of these interventions to reduce socioeconomic inequalities in undernutrition and to determine whether there are any adverse effects of supplementary feeding. There were 32 studies using various methods included in the analysis. The authors assessment was that less than 50% of all the studies had low risk of bias for the methods employed. Most interventions were able to increase weight. For height, meta-analysis of 9 studies with moderate quality evidence found that supplemented infants and children grew an average of 0.27 cm more over 6 months than those who were not supplemented (95% CI 0.07–0.48, 1,463 participants). This is of very little or no biological importance because, again, it is within measurement error. Moreover, refeeding studies of malnourished children after World War I reported average catch-up growth rates more than 10 times greater, typically 3–5 cm, within 8 weeks of supplementary feeding (Hermanussen et al. 2018a; Scheffler et al. 2019).

Another Cochrane review by Visser and colleagues (2018) reported on 95 community-based supplementary feeding interventions meant to reduce food insecurity and improve the nutritional status of vulnerable and malnourished groups of people. The authors defined "supplementary feeding" as providing extra food to people or families over and above their home diet. The majority (74%) of the participants were from low- and middle-income countries (LMICs). The number of participants included in each intervention study varied between 91 and 7,940 adults, and 271 to 12,595 children. The interventions varied in duration from 6 weeks to 2 years. They also varied in the frequency and format of feeding. The authors reported that, "In children under five years of age from low and middle-income countries, supplementary feeding had a small impact on child growth" (p. 3). Based on the studies that measured the change in length for newborns due to supplementary feeding of pregnant women (3 studies) or the change in length or height for <5-year olds fed directly (7 studies), the mean increase was 0.26 cm for those receiving supplementary feeding compared with those not given additional food. This is, once again, a biologically negligible outcome.

Perhaps a reason for the negative, inconsistent, and negligible results of the interventions reviewed above is that the specially created food supplements, WASH interventions, and imposed education programs are too artificial and do not reflect biocultural reality. Biologically, consuming nutrients in supplementary form is often not the same as ingesting the same nutrients as food due to "food synergy." Biological and chemical interrelations occur between nutrients during food preparation (cooking, fermenting, etc.), digestion, and intestinal absorption.

A Cochrane review by Eaton and colleagues (2019, p. 2) noted that, "Animal-source foods, such as eggs, meat, fish, and dairy, are energy dense and contain multiple micronutrients and essential fatty acids with high bioavailability. The benefits of animal-source foods may include higher food synergy relative to fortified foods as well as decreasing dependence on external suppliers of fortified foods." The review is titled, "Effectiveness of provision of animal-source foods for supporting optimal growth and development in children 6 to 59 months of age." The authors' review included " . . . randomized controlled trials and quasi-randomized controlled trials of any duration, where children between 5 months and 59 months (6 years) of age were provided with an animal-source food (e.g., consumption of milk, meat, or eggs), prepared with any cooking method, compared with any intervention or no intervention" (p. 2). Five studies were identified which measured the change in length-for-age or height-for-age z-scores (LHZ or HAZ) of infants and children (n = 2,972). Three of these studies (n = 592) reported a statistically significant increase in LAZ and HAZ in the animal food group compared to the control group. The other two studies (n = 2,380) reported a decline in LAZ of infants in both groups. Based on the authors' rating, the quality of the evidence for all studies was very low. Problems with the study methods included imbalances in important physical and social characteristics of the participants of the intervention and control groups at the start of the study and imprecision due to wide confidence intervals and inconsistent direction of effects. The authors' concluded that, "We have little confidence in the results . . . Given the limited quality of the evidence, we are uncertain of the effects of the provision of animal-source food versus cereal products or no intervention on the growth or development of children" (p. 2).

My colleagues and I completed a Cochrane review, "Nutritional interventions for preventing stunting in children (0 to 5 years) living in urban slums" (Goudet et al. 2019). Our systematic review included 15 studies, of which 14 were randomized controlled trials (RCTs). An RCT is considered the most accurate and reliable scientific method to assess the efficacy of a treatment. In an RCT the participants are randomly assigned to a treatment group vs. control group (no treatment or a sham treatment such as an inert substance that looks like a drug). The randomization reduces the likelihood of biases, such as age or sex, in the allocation of treatment. The studies included in our review took place in recognized slums or poor urban or peri-urban areas. Urban slums are defined by the United Nations as low-income informal settlements lacking one or more indicators of basic services or infrastructure, such as electricity or treated water supply. The study locations were mainly Bangladesh, India, and Peru. The participants included 9,261 infants and children and 3,664 pregnant women. There were no dietary intervention studies in which women were given food to prepare and serve to their infants and children as they saw fit. All the studies identified were nutrient supplementation and educational interventions. The interventions included zinc supplementation in pregnant women (three studies), micronutrient or macronutrient supplementation in children (eight studies), nutrition education for pregnant women (two studies), and nutrition systems strengthening targeting children (two studies) intervention. System strengthening refers to changes

in one or more of the following areas: policies and governance, infrastructures and markets, inputs and services, information and communication, financing, household resources, and the sociocultural environment. Six interventions were adapted to the urban context and seven targeted household, community, or "service delivery" via systems strengthening.

The primary review outcomes were that there was no effect of giving pregnant women nutrient supplementation on the birth weight and length of their newborns and there were inconclusive or negligible results for nutrient supplementation of infants and children on improving their length, height, or stunting status. The two studies of zinc supplementation for pregnant women, for example, found an increase in newborn body length of 0.13 cm. Supplementation with vitamins and minerals for <5-year-olds resulted in 0.02 cm greater length or height. These results were about equal or just slightly greater than measurement error, meaning that there was likely no effect. There was a positive impact on birth weight of maternal education interventions with birth weight increases averaging 478 g in infants exposed to the intervention. Improving health systems that support nutrition had inconclusive or negligible results on children's stunting status. The certainty of all the evidence was very low to moderate overall. This was due to methodologies that introduced possible biases, such as not truly randomizing the allocation of treatment, or allowing participants or researchers to know who was receiving an intervention and who was in a control group. Many of these types of methodological issues are impossible to overcome within the traditional RCT model when it is applied to real people living in their home neighborhoods in slums.

Our review showed the need to better understand urban slum environments and their people. The types of interventions that failed in urban slums were moderately successful in some low-income rural areas and other locations outside of urban slums. In a review of eight of these other studies Sally Grantham-McGregor and colleagues summarized that, "Five benefited child development, but one did not, and two showed deficits. There was generally little benefit of at-scale programs to nutritional status" (Grantham-McGregor et al. 2014, p. 11). Only three of the eight interventions reassessed the children more than a year later. There were no benefits on weight or height in these follow-up assessments.

Why Don't These Interventions Work?

Villermé and Chadwick would not be surprised that giving impoverished people a bit more food or some specific nutrients does not make them grow in height and may even make them grow less. These two pioneers of public health knew that the problems of the poor are due to more than nutrition and that multi-sectorial interventions were needed that combine the provision of food, nutrition education, safe housing, sanitation, clean water, and secure employment. Both Villermé and Chadwick worked tirelessly to influence their national governments to recognize and to do something about the problems. Things have not changed. Our *Cochrane Review* (Goudet et al. 2019) recommended that more evidence is needed on the effects of

"up-stream" practices and policies of governmental, nongovernmental organizations (NGOs), and the business sector to improve low birth weight and stunting in poor urban environments.

The findings of the systematic reviews are surprising and frustrating to many nutritionists, public health, and anthropological researchers because we have been trained to believe the admonition of my mother – "eat or you will not grow." Textbooks in human auxology and nutrition state, essentially, the same words. Researchers and clinical practitioners are wedded to the idea that growth is, first and foremost, nutrition dependent. Many articles and reports declare that poor growth is prima facie proof of malnutrition. Professional publications often begin with a sentence such as, "Chronic malnutrition in children remains an important global problem, with an estimated 165 million children under five being stunted" (Leroy et al. 2015, p. 2). Note that without any evidence or explanation the authors assume that a stunted infant or child is malnourished. Later in the same article the authors repeat the assumption by stating, "Child linear growth is the best available summary measure of chronic malnutrition" (p. 10). I must point out that aside from these statements on stunting, the Leroy et al. article is an excellent and important analysis of the use of measured height-for-age changes as opposed to height-for-age z-scores to assess infant and child growth. Their analysis finds that height-for-age z-scores obscure the increasing age-to-age decline from the World Health Organization (WHO) growth standards of measured height for under-five-year-olds in low- and middle-income countries.

Another example of the conflation of stunting with malnutrition comes from the Executive Summary of the authoritative series of reports on Maternal and Childhood Nutrition published by *Lancet* which states, "Stunted linear growth has become the main indicator of childhood undernutrition . . . It should replace underweight as the main anthropometric indicator for children."[15] A third example is the declaration, "There is increasing agreement among the nutrition community about the use of length/height-for-age as the indicator to monitor the long-term impact of chronic nutritional deficiencies" (Lartey 2015, p. 449). These three examples, and many more similar ones, make the case for the equation "stunting = malnutrition" but this equation is wrong. It is wrong because malnourished infants and children may not be stunted and because stunting can be due to nonnutrition causes. The WHO defines height stunting as " . . . impaired growth and development that children experience from poor nutrition, repeated infection, and inadequate psychosocial stimulation."[16]

Stunting does not equal malnutrition, at least for many cases of stunting. Christiane Scheffler, Michael Hermanussen and I, along with 25 Indonesian colleagues, showed that stunting is not a synonym for malnutrition (Scheffler et al. 2019). Lest any reader misunderstand, we accept the equation "malnutrition = stunting" when it can be shown that a growing person does not receive enough total

[15] www.thelancet.com/pb/assets/raw/Lancet/stories/series/nutrition-eng.pdf (p. 3)
[16] www.who.int/nutrition/healthygrowthproj_stunted_videos/en/

food (caloric energy) or any one of the 50 or so essential nutrients that must be supplied by the diet. We question the reverse equation, that stunting is prima facie evidence of malnutrition.

We published a critical analysis of variation in height and body composition of 1,716 children, juveniles, and adolescents aged 6–13.2 years from some of the poorest and wealthiest regions of Indonesia. We worked with Indonesian colleagues who were mostly pediatricians and they clinically assessed the participants for signs of malnutrition and skin infections. We defined nutritional status in two ways; (1) the mean value of the triceps + subscapular skinfold ($\bar{x}$SF) and (2) by mid-upper arm circumference (MUAC). Both of these are standard methods for the assessment of nutritional status as advocated by the WHO and other public health organizations (Bates et al. 2017). We used the mathematical methods of correlation and linear mixed effects models to assess statistical significance between $\bar{x}$SF, MUAC, and height-for-age z-score (zHT).

The distributions of zHT against $\bar{x}$SF may be seen in Figure 7.4. In the poorest region sampled, rural Soe in West-Timor (~40,000 inhabitants), a total 53% of boys, and 46% girls were stunted. There were statistically significant correlations between height, skinfold thickness, and MUAC that ranged from r = 0.12–0.37. The square of these values (r^2), indicates the amount of variance explained by the bi-variate correlation. With a maximum r^2 of 0.14 there was no meaningful association between zHT and either $\bar{x}$SF or MUAC. In fact, the mean skinfold thickness of these impoverished rural children and youths was close to German values. Furthermore, the shortest and tallest participants did not differ relevantly in skinfold thickness. The same applied for the association between zHT and MUAC. Kupang is a city in West-Timor with 330,000 inhabitants, a university, an airport, and a harbor. A total of 28.8% of boys, and 17.8% girls were stunted. There were no biologically meaningful correlations between zHT and either $\bar{x}$SF or MUAC. There were no clinical signs of malnutrition or chronic infection in the stunted children examined in either Soe or Kupang.

We also studied elementary school children of the city of Ubud, Bali. In total only 5.4% of boys, and 4% girls were stunted, but with mean zHT = −0.3 these school students were still short. The Kupang samples had less than 15% overweight and about 11% obese children and youth, whereas the Soe sample had only 2% overweight and no obese children. In contrast, in wealthier, urban Ubud 35.6% boys and 29.2% girls were overweight and 21.4% boys and 12.4% girls obese. The correlations between zHT, $\bar{x}$SF, and MUAC were highest among these Ubud children confirming that growth is accelerated in overweight and obese children. We concluded from our analysis that the Indonesian data seriously question the concept of stunting as prima facie evidence of malnutrition and chronic infection.

It is very important to state that we are not claiming that these Indonesian children are “small-but-healthy” in the sense of discredited proposals by some economists (see Pelto & Pelto 1989 for reviews). Clinical stunting and very short stature for a large segment of a population indicate problems exist, but these may not be, primarily, nutritional problems.

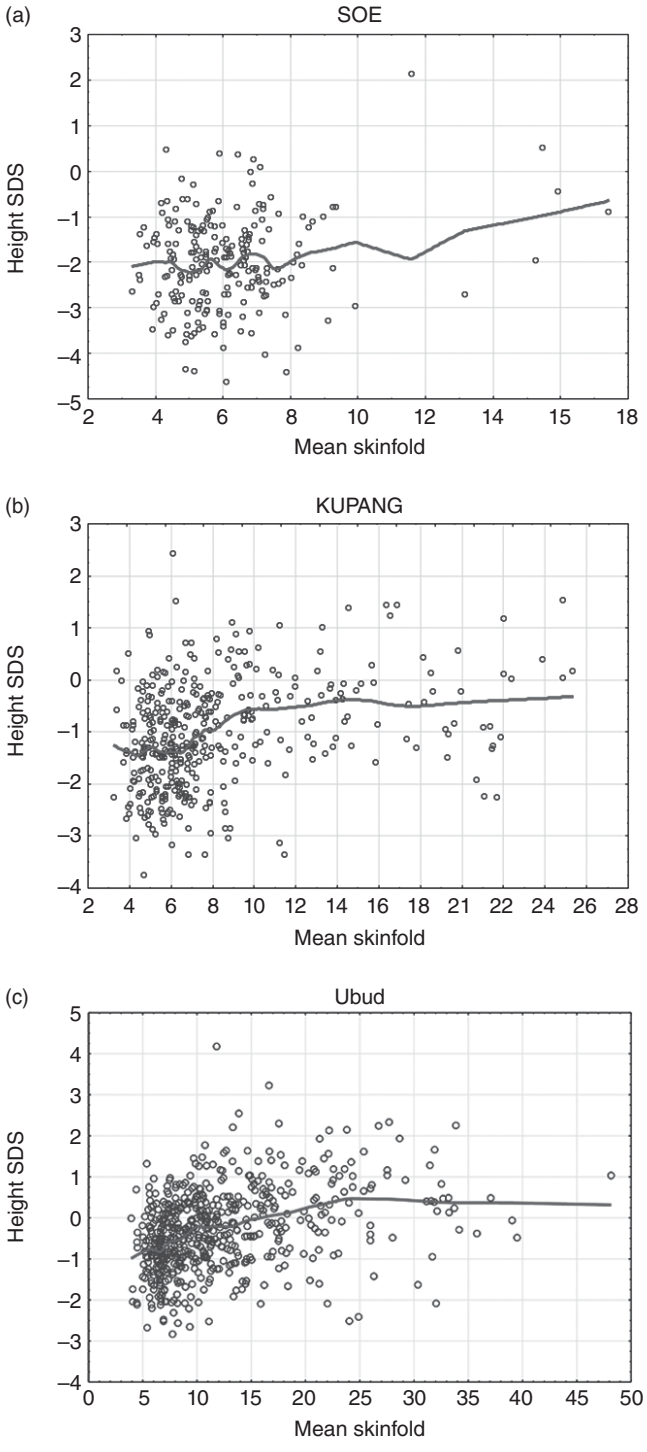


Figure 7.4 Height z-scores (zHT) and mean skinfold thickness [(subscapular + triceps)/2, $\bar{x}$SF] of: (a) 107 school students from rural Soe, West-Timor; (b) 206 students from urban Kupang, West-Timor, Indonesia, Indonesia; and (c) 591 students from urban Ubud, Bali, Indonesia. zHT and $\bar{x}$SF were plotted and fitted with LOWESS to better highlight the overall shape of the relationship between the x and y variables. Please note that the scale of the Ubud children differs due to the exuberant prevalence of obesity.

We proposed instead that psychosocial and emotional factors, associated with community effects and strategic growth adjustments, are the primary cause of the stunting and, generally, short stature of the Indonesian school students. Rural Soe has some 40,000 inhabitants. The region of Soe was the most isolated geographically and socially from the more "modern-westernized" regions of Indonesia. Soe had very little ecotourism, no remarkable industry, and the parents of the Soe children had the lowest number of years of formal education. Kupang is only 110 km from Soe by the one asphalted road, but it takes about 3 hours by private car to drive that distance. Kupang has some 330,000 inhabitants, a university, an airport, and a harbor. Kupang parents had more years of formal education compared with Soe parents. The town of Ubud is in the uplands of Bali, Indonesia. Ubud is considered a "traditional" town, known as a center for traditional crafts and dance. The population of Ubud is 74,320 people, but the local population is visited by the more than 3 million foreign tourists every year. Ubud is part of the "tourist island" of Bali, which is densely populated and economically more prosperous than Soe or Kupang. Balinese people are the tallest Indonesians as were the Ubud, Bali children in our sample. Ubud parents had the highest number of years of formal education. "Modern-western" material culture is ubiquitous and Ubud children are exposed to a material and social world that is unavailable in Soe or Kupang. Ubud children and their families see and interact with "tall foreigners" every day and see tallness as an indicator of higher social, economic, and political status. These differences between the three localities translate into community effects and strategic growth adjustments in height toward the social norm or toward the desired "type."

We proposed that in Soe and parts of Kupang, the negative emotional impact of living in poverty with food and housing insecurity and other insults are some of the stress factors that explain the comparably short stature of those children. In addition, we proposed that the children may be short because they lacked those social, economic, political, and psychological infrastructures that in the modern western world have stimulated growth beyond all previously recorded limits for height. Our explanation emphasized the bilateral links between height and social position (Hermanussen & Scheffler 2016). Moreover, our hypothesis includes growth modulation and strategic height adjustment by feelings of hopefulness for the future that come from parental education and family involvement with the modern western world. A subsequent analysis of the Indonesian data reported that parental education is especially important and, again, nutritional status unimportant in the Indonesian samples (Groth et al. 2019). Intensity of hope for the future rose from Soe, to Kupang, to Ubud – and so did average height-for-age.

A New Perspective on Stunting and Nutrition

Stunting is not simply due to malnutrition and even the WHO definition of stunting recognizes that infection and inadequate psychosocial stimulation are also culprits. But questions remain. Why are infection and psychosocial stimulation ignored so often in the literature on stunting? Why does the WHO definition of stunting only

focus on inadequate psychosocial stimulation and ignore the impact of toxic emotional stress and adverse childhood experiences due to constant feelings of insecurity from the lack of a physically and socially safe community environment? These insecurities are psychosocial stimulants but are harmful to growth. Why is there no, or little, mention of the absence of hope for the future? Hopelessness is known to harmfully impact growth in height, as is explained further on. Each of these non-nutritional causes of stunting is a community effect and each requires a strategic growth adjustment if the individual is to survive. This new perspective on the regulation of growth by social, emotional, and neuroendocrine factors was presented in our article "The impact of social identity and social dominance on the regulation of human growth: A viewpoint" (Hermanussen et al. 2019).

Famines and Starvation

The impact of the famines and starvation people experienced during the World Wars and social-political upheavals of the twentieth century have been much studied by growth, nutrition, and medical researchers. Malnutrition is clearly one consequence of famine and starvation. Chronically hungry people are also at greater risk for infection, in part due to a compromised immune system, and may feel hopeless.

In a review of research prior to 1950 Keys and colleagues (1950) summarized that: (1) starvation of pregnant women in the Netherlands and Leningrad, Russia during World War II reduced the birthweight of their newborns and (2) the effect was most pronounced if the starvation was experienced during the last trimester of pregnancy. This makes sense because, as explained in Chapter 2, growth in fetal weight is fastest during the last three months of pregnancy.

Better information is available from World War II Japan. In a review of Japanese studies Kimura (1984) found that compared with pre-war levels, the mean stature of age-matched children decreased between 1939 and 1949. Average heights returned to pre-war levels in 1953 for girls, but not until 1956 for boys. Kimura also found that the growth of infants, children, and juveniles, between the ages of birth and 12 years during the war, was affected more than the growth of adolescents. Why this was so is not known. Finally, Kimura noted that the post-war recovery in height and weight " ... occurred most rapidly in large cities, followed by small cities, and then rural mountain villages" (p. 200). Since improvements of social-economic-political-emotional factors, including diet, also followed the same path it is likely that the growth recovery was, in large part, due to a complex matrix of interacting factors changing simultaneously.

The effect of European wartime famine on older infants, children, and juveniles is less clear. One reason for this is that English language publications were based on growth measurements taken before the wars began and then a year or more after the wars ended. What happened during and immediately after the wars was unknown. Comprehensive collections of literature are available on starved and refed early twentieth-century European populations. Most of the articles were published at the

end of the nineteenth and in the beginning of the twentieth century and focused on the nutritional situation before and during the period of starvation, and subsequent refeeding after World War I. But this literature is published in German medical journals, in the German language, and is not available electronically. Few scholars of human growth can understand the language and are willing to find this literature in German libraries.

Fortunately, two German colleagues, Christiane Scheffler and Michael Hermanussen were willing to find this "lost" literature. Scheffler is a human biologist and anthropologist and Hermanussen is a pediatrician, endocrinologist, and auxologist. They first identified two seminal reviews, in German, on child growth and development published after World War I by E. Schlesinger in 1925 and E. W. Koch in 1935 (the citations to these German language publications are given in Hermanussen et al. 2018b). Generated from these reviews was a list of journals most frequently mentioned as publication outlets for manuscripts on child growth and development, especially manuscripts on child nutrition. Scheffler and Hermanussen then performed a search for this literature at the *Staatsbibliothek zu Berlin* (State Library of Berlin, Germany), the third largest European library with an almost complete collection of the German medical journals. My colleagues spent 3 days working in the library and systematically browsed 15 bookshelf meters in search of relevant articles.

In all, about 25,000 pages in 16 pediatric and school doctors' journals were examined. My colleagues detected 66 publications, published between 1858 and 1932, of primary interest. Twenty-six studies contained forgotten details on child growth, including individual growth patterns, seasonality of growth, social influences on growth and patterns of growth during starvation and refeeding.

The important findings of this search through the "lost" literature may be summarized in four key notes:

(1) Newborn and infant nutrition and growth was the priority among the early auxological contributions both in number of publications and their size in pages. Detailed case histories, often including daily measures of breastmilk quantities, were frequently published. In those days, infant growth was remarkably poor, even most of the breastfed infants of the upper social class grew below modern WHO standards.
(2) Late nineteenth- and early twentieth-century children, juveniles, and adolescents measured in Boston, USA and German schools, independent of social strata, grew far below WHO standards in height but close to the standards in weight. Given their weight these students would be judged as healthy and well nourished, but 15–30% of Boston students measured in 1877, aged 5–18 years old, would be classified as stunted by the WHO standards. In Germany, 25–30% of the apparently healthy children were stunted at age 6.
(3) The historic literature lacks evidence of a strong association between food, child growth, and adult height. Even in view of mass starvation during and shortly after World Wars. Undernutrition led to weight loss that was relatively greater than loss in height. German pediatricians concluded that the shorter stature of

those children affected was an inhibition, that is a delay in growth tempo. Even prolonged starvation during childhood did not affect adult height as the formerly hungry children and juveniles were able to catch up after the starvation ended. With on average 3 cm within 6–8 weeks, catch-up growth in height of severely undernourished school children during refeeding was significantly greater than any catch-up reported in modern nutrition intervention studies.

(4) Also reported in the historic literature were observations focused on the impact of chronic illness on child growth. School children who recovered from rickets, a deficiency of vitamin D, were not shorter than unaffected children. Infections such as measles, scarlet fever, tuberculosis, and pneumonia were listed among those illnesses that after recovery promoted, rather than impaired, overall growth, contrary to what the modern pediatrician would expect and what the WHO lists as a cause of stunting.

Consideration of the historic literature is indispensable to understand why modern nutrition and WASH interventions often fail to prevent stunting. The historic studies concluded that supplying more food to truly starving children would allow for catch-up growth, but not more. Few of the modern nutrition interventions focus on starving populations. Most are described as "moderately malnourished" and that assessment is based on the prevalence of stunting which as we now know is not a synonym for malnutrition! The modern studies interpret the, often, negligible effects on height growth of food supplementation as either a small success or as a failure of the intervention. But this is, in fact, the normal biology of human growth in short, but not starving populations. If scientists, clinicians, policy makers, and politicians understood better the normal biology, as revealed from the old literature, there would need to be a major change in the approach to the causes of growth stunting.

This shift of theory and application would be along the lines proposed by Subramanian and colleagues (2016), who recommend that we look to the upstream, structural factors of the social-economic-political-emotional (SEPE) systems that systematically deprive lower class families of hope, dignity, and a belief that they can better themselves economically and socially. Subramanian and colleagues propose that we move away from nutrition and WASH intervention strategies to prevent stunting and move toward support-led strategies that offer integrated policies to reduce SEPE risks for stunting. Support-led strategies are a type of intervention that creates " ... equitable public policies and provisions that matter for nutrition" (p. 233). Greater equity in social, economic, and political conditions creates greater emotional security and these associate with taller average adult height across 169 countries (Bogin et al. 2017).

To implement the recommendations of Subramanian and colleagues requires a multifactorial approach such as combining a strong food distribution system with investments in water and sanitation infrastructure, health services, and quality education leading to meaningful employment. The currently rich, industrialized nations implemented support-led strategies in the past 150 years to overcome some,

but not all, of the inequalities, extreme poverty, and high prevalence of stunting evidenced in the historic studies. Some of these nations, such as Germany, Switzerland, and the Netherlands experienced secular increases in average height of up to 20 cm in the past century (NCD Risk Factor Collaboration (NCD-RisC) 2016). This is achievable for short-stature populations today, as shown by my research with Maya refugee children in the United States who sprouted an average of 11 cm within one generation. Part of that increase was due to more total food and better WASH, but another part was due to greater emotional security from opportunities for education, meaningful livelihoods, and protection from violence. There is every reason to expect similar changes in growth in the low- and middle-income nations, if they are given similar opportunities.

Long-Term Effects of Starvation

In addition to the World War II famines in the Netherlands and Leningrad mentioned earlier there are other examples available with sufficient sample size and historical records for analysis of effects on human growth and development. These include nineteenth-century crop failures in Sweden and Finland, seasonal famines in the Gambia between 1949 and 1994, the Chinese Great Leap Forward famine of 1959–1961, and recent seasonal famines in Bangladesh. The Ukraine famine of 1931–1933, severe undernutrition in Greece and the Channel Islands during German Occupation in World War II, and in Germany itself at the end of World War II and in the early postwar period also may be used for systematic follow-up of the people exposed (Lumey et al. 2011).

The Dutch Hunger Winter of 1944–1945, near the end of World War II, is the most studied and some of its findings are explained here. The information is derived from comparative review of several famines by Lumey and colleagues (2011). A combination of food blockades by the Germans and by the Allies, as well as a particularly severe winter, caused the people of the Netherlands to be in famine conditions for seven months. Food allocation registers indicate that residents of cities in the western region of the Netherlands subsisted on official food rations of 1,500–1,800 kcal/day from June 1942 to June 1944, then rations dropped rapidly to ~500 kcal/day by January 1945 and then rose steadily to ~2,000 kcal/day by May 1945. There was hunger throughout the entire period as the minimal requirement for adult women is 2,200 kcal/day and for pregnant women an additional 90 kcal/day are needed during the first trimester, increasing to 287 kcal/day in the second trimester, and 466 kcal/day in the third trimester (Butte & King 2005). There was severe famine from the late 1944 to early 1945 period. It is important to note that the Dutch famine took place near the end of World War II and exposure to many years of war and German occupation may have had SEPE effects that were interacting with famine. During the famine some people resorted to eating food substitutes, such as tulip bulbs which can be toxic. The very cold winter temperatures may have had effects independent of, or interactive with, the famine and the other exposures. The

point is that rarely, if ever, is it possible to isolate the effects of nutrients on human development from other factors. People consume food and food is always part of a complex matrix of interacting variables, set within a SEPE context.

At 18 years of age, Dutch military recruits with prenatal famine exposure in early pregnancy had a 2.8% prevalence of obesity compared with 1.7% for the entire cohort of recruits. At older ages, up to 50 years old, the women who were exposed to famine during prenatal and early post-natal life had higher body weight, BMI, and waist circumference than nonexposed women. No effect of exposure was found in older men. In men and women up to 59 years old with famine exposure during fetal growth and development there were the following health risks: (1) higher impaired glucose tolerance; (2) weakly elevated total cholesterol and triglycerides in women, but not in men; (3) weak evidence for increased anti-social behavior and mood disorders; (4) more asthma, chronic bronchitis, emphysema, or chronic nonspecific lung disease compared with unexposed controls; and (5) weak or no evidence of lower birth weight in the next generation. No meaningful effects of famine exposure have been reported for elevated blood pressure effect, cardiovascular disease, or cognition (IQ), but performance on a task of selective attention declined faster than in those not exposed (de Rooij et al. 2010). The presence of these health problems varied by the timing of famine exposure during fetal development. Laura Schulz (2010, p. 16757) noted that, "Those who were exposed to the famine only during late gestation were born small and continued to be small throughout their lives, with lower rates of obesity as adults than in those born before and after the famine. However ... those exposed during early gestation experienced elevated rates of obesity, altered lipid profiles, and cardiovascular disease."

Research by economists reported significant negative effects of the Dutch famine exposure during the first trimester of gestation on employment outcomes 53 or more years after birth (Scholte et al. 2015). The exposed people were less likely to be employed. People exposed to the famine in the second and third trimesters showed evidence of more pre-retirement hospitalizations. The risk for early retirement or employment termination and for ill health requiring hospitalization was ascribed to lower cognitive performance and emotional effects, such as the slightly increased risk for anti-social behavior and mood disorders in those exposed to the famine during gestation. As mentioned, the emotional sequelae of famine exposure were weak, meaning they were statistically significant but of small biological and behavioral importance. Other studies reviewed by Scholte and colleagues reported good evidence for more neural tube defects and a two-fold increase in risk for schizophrenia at young adult ages after exposure to famine early in gestation. It is curious that at age 18 years there were no significant cognitive differences between those exposed to the Dutch famine and those not exposed. Scholte and colleagues reported that at age 59 years the cognitive evidence is mixed, with one study reporting no effects and another reporting an effect on a selective attention task but not on other measures. It was noted that the post-18-year-old effect could be the result of something that happened after age 18 years, which may mean that famine exposure had no direct effect.

Germany was devastated at the end of World War II with millions of people homeless and suffering a hunger that lasted longer than the Dutch famine. Hermanussen and colleagues wondered what effect this post-war period had on lifetime earnings (Hermanussen et al. 2017). The research noted that about 2.25 million homes had been destroyed and at least that many had been seriously damaged. "People lived in cellars, ruins and barracks and life was dominated by a daily struggle for survival, with most Germans living in utter poverty . . . The average daily calorie intake per capita in Germany had progressively fallen to 2,010 kcal by the spring of 1945 and continued to fall down to 1,451 kcal in 1946, with some regions recording even lower values . . . It is estimated that in 1946/47 German agriculture only provided about 35% of the population's needs, as the war had reduced the amount harvested to 50–60% of the normal level. The lack of animal protein and fat was a critical factor . . . " (p. 2). Post-war surveys found that the disastrous living conditions in Germany led to significant growth impairment in school children with reductions in height of up to 7 cm in early adolescence when compared with prewar cohorts of the same age. Infant mortality increased from 69.7/1,000 in 1939, to 105.4/1,000 in 1945, and was 81.7/1,000 in 1948. This is higher than in most low-income nations today.

Despite this evidence for suffering, hunger, and growth reduction, infants born in 1945–1948 who survived to work retirement age had only slightly impaired labor market outcomes compared with cohorts born before the war (1935–1938) and 10 years after the end of war (1955–1958). The effect was measured in terms of monthly old-age pensions, which are valued in relation to the amount of money earned during one's working life. Men born in the 1930s received the highest monthly old-age pensions, peaking at an average of €1,181 per month in the 1934 cohort. Payments were slightly lower in men born during and after the war, with a minimum average monthly pension of €1,023 in the 1947 born cohort and an average pension of €1,052 per month in the 1949 born cohort, the youngest cohort that had reached regular retirement age. The researchers noted that the variation in " . . . payments indicate a minute reduction in the lifetime earnings of cohorts born shortly after World War II" (p. 3). The pension payment system was not constant between birth cohorts due to political changes and this may have biased the findings. As an alternative assessment of such bias the researchers calculated the risk-of-poverty rates for the three birth cohorts. There was no evidence that the 1945–1948 birth cohort was at higher risk of poverty than the 1935–1938 or 1955–1958 birth cohorts. These findings challenge a wide-spread belief that infant and early childhood malnutrition and deprivation reduce future lifetime earnings.

The Milk Hypothesis Rejected

To what extent can a specific food make an impact on human growth and development? There has been considerable interest in this question and of all the foods studied milk has generated the most attention. Milk is the first food received by mammals after birth and milk supports the high velocity of growth during infancy.

For humans, mother's milk is the only food needed during the first few months of post-natal growth and development. The WHO and pediatric medical societies in the United States, United Kingdom, and elsewhere recommend exclusive breast-feeding for the first 6 months after birth with continued breast-feeding along with appropriate complementary foods for 1–2 years of age. Reviewed in Chapter 3 was the evolution of lactation. Milk has important impacts on mammalian nutrition, immunology, behavior, social organization, and psychology. Milk is essential for the growth and health of all mammalian infants, including human infants. No mammal other than humans regularly consumes milk after infancy. So, the question here is, will milk consumption by human children, juveniles, and adolescents result in greater height or body mass?

I reviewed the twentieth-century evidence in favor of milk as a growth stimulant in my article "Milk and human development: An essay on the 'milk hypothesis'" (Bogin 1998b) and in the previous edition of this book. In those reviews I was generally in favor of milk as a height stimulant. Today I am less sure that milk has any special properties that, in and of themselves, promote growth in height.

The "milk hypothesis" proposes that a greater consumption of milk during infancy, childhood, and adolescence will result in taller adult stature. Much of the evidence I reviewed in the past were anecdotal reports or associations studies. Examples are pastoralists living in traditional, non-Western cultures whose diet included animal milk vs. agriculturalists whose diet was usually devoid of milk and milk products. The pastoralists of Central Asia (peoples of the Gobi, Takola Makan, and Kavil Deserts) and the pastoralists of East Africa (the Maasai, Samburu, and Datoga) were found to be taller than their rice- or grain-growing counterparts (Little et al. 1983; Takahashi 1984). Since these studies of East African pastoralists, the situation has changed in tragic ways and the growth of the people has suffered. A brief account of these changes in given in Box 7.2.

An historical study of heights of conscripts in the Bavarian army in the nineteenth century reported that average heights were greatest for conscripts from the dairy herding regions, and milk consumption was the single most important variable associated with stature variation (Baten 1998). Conscripts from grain- or potato-producing regions, and from weaving districts where workers had money to purchase food, were significantly shorter than conscripts from the milk regions. These association studies are suggestive, but were the height differences due to some factor in milk or was it one or more SEPE factors related to different lifestyles between pastoralists and dairy farmers vs. grain-potato farmers and weavers? It is impossible to decide based on the available data.

Other evidence I gathered came from milk supplementation studies of poorly nourished and impoverished children in Europe and the United States between the years 1920–1960. In the 1920s, Scottish schoolchildren were given an extra pint of milk per day for seven months. Some children received whole milk and some skimmed milk. Both groups increased faster in height and weight than two control groups of children of the same ages, one group given no supplement and the other given a supplement of biscuits, equaling the milk in total calories. Since the biscuit

Box 7.2 **A milk tragedy: The Pastoralists of East Africa**

A deficiency of any one of the nutrients contained in milk will slow or stop growth in height. Research with nutritionally deficient groups of infants, children, and juveniles shows that milk supplements can promote height growth. In one study with Samburu pastoralists in Kenya, Lora Iannotti and Carolyn Lesorogol (2014) supplemented the infant and child diets with animal milk and found that the milk provided the deficient vitamin and mineral micronutrients and promoted a healthier growth in height, weight, and body composition. The Samburu were mentioned earlier in this chapter as one of the "tall" milk-drinking peoples of the world. The plight of the Samburu and other East African pastoralists from tall, milk drinkers to short and malnourished underlies an important case study of the role that social, economic, political, and emotional (SEPE) factors have on humans.

The Samburu are part of the larger Maasai culture, a group of people whose traditional and historical homelands spread from southern Sudan, to Uganda, through Kenya, and into northern Tanzania. References for the ethnographic background of Turkana, Samburu, Karimojong, and related culture groups may be found in Gray et al. 2003; Gray & Sundal 2017; Little et al. 1983; Little & Gray 1990, and online.[17] The Samburu reside mostly in the north-central region of Kenya and their homeland covers a large area that is shared with other pastoralists such as the Pokot, Karimojong, and Turkana. All are semi-nomadic pastoralists whose traditional livelihood is based on maintaining herds of cattle, sheep, goats, and camels. These animals provide the dietary staples of meat, milk, and blood. Above all, cattle are the center of social, political, economic, and emotional life. The size of a person's or family's cattle herd indicates social status in the community. Accumulating animals, rather than consuming or selling them, leads to social, economic, political, and emotional security and power.

The Samburu, Turkana, Karimojong, and Pokot eat meat, but the staples of their traditional diet are milk and blood. Drunk raw, or soured, drunk in tea, or turned into butter or ghee (which are especially important as a food for infants), milk is a part of almost every meal for these pastoralists. Blood is obtained by piercing the jugular vein of a cow precisely, allowing for blood-letting that doesn't kill the animal. The blood may be consumed raw, after cooking, or in the form of several blood-milk mixtures. Blood-milk mixtures are consumed not only as food, but also as ritual drinks in social and religious celebrations or given to the sick as a medicine.

Blood and milk are dietary staples. Complete nutritional status is achieved with additional foods including meat, from domestic livestock and foraged

[17] www.africa.upenn.edu/NEH/kethnic.htm

wild animals, tubers, honey, plants, berries, and seeds. These are often prepared as soups and stews. The Samburu further supplement their diet with sorghum and millet that they grow and grains and maize-meal that they acquire by trade with agricultural neighbors or receive as government subsidies. Even with these "foreign foods" milk plays a role. A popular maize-based porridge is *ugali* and is generally served with milk in Samburu households. The primacy of milk in the diet was found for all other East African pastoral groups surveyed by Kathleen Galvin and Michael Little (1999). They compared 10 African pastoralist groups studied prior to 1995 and all groups consumed at least 16% of total daily energy intake as milk and not less than 21% of daily intake as animal-based foods. For the Turkana pastoralists it was reported that milk consumption per household in some years peaked at between 24 and 29 liters or about 4 liters per person/day. Across the 2-year period of observation with the Turkana 62% of food consumed per day was milk and 18% was other animal-based food, mainly blood, fat, and meat.

Traditional pastoralists such as the Turkana and Samburu had milk as a central part of their diet and biocultural existence. Why then, did the Samburu need the milk supplement provided in the Iannotti and Lesorogol study? The reasons began with colonial era "pacification," which to the early twentieth century British colonial officers meant settlement of the people on agriculture-based farms. Additional reasons are colonial and post-colonial land appropriation, warfare, droughts, a rise in inter-tribal violence, and political corruption (Gray et al. 2003; Gray & Sundal 2017).[18] Colonial era policy failed because the land was not suitable for sustained agriculture. Pastoralism was the correct ecological adaptive strategy. Resistance to "pacification" by the pastoralists was often met with military force from the British and later by the post-colonial national governments. British policy resulted in degradation of the pastoral lands. During the 1960s and mid-1970s there were devastating droughts with livestock losses, and crop failures. In addition, armed violence and civil war swept the former colonies after liberation. Another drought from 1979–1981 brought widespread famine which the government then ignored. The weakened state of the pastoralists and their animal herds allowed human and livestock epidemics to become more frequent after 1980. Climate instability brought more droughts and food insecurity became the norm. To avoid mass starvation, local governments and international agencies instituted emergency food relief in every year between the 1980 famine and 2004.

The Maasai, Samburu, and Turkana had always practiced cattle rustling. Prior to the 1980 famine this theft had been carried out at the person and family level and was largely nonviolent. Law enforcement officials tended to ignore the rustling and left disputes arising between and within groups to be settled by elders. Often the guilty person was fined cattle, goats, camels, or

[18] www.culturalsurvival.org/publications/cultural-survival-quarterly/land-and-pastoralists

sheep. As cattle herds declined from drought and disease and food insecurity became critical, armed violence escalated. By the later 1980s large-scale predatory cattle raiding emerged and was carried out mainly by young men who no longer consulted or respected the elders. By undermining the structure of elder authority, the traditional system of herd management and distribution collapsed which decreased breeding, which exacerbated the decline of animal populations and further destabilized the food supply.

By 2004 the diet intake of many East African pastoralists changed from primarily meat to grains. In some groups the diet became more than 90% plant based and less than 1% milk based. The brewing of beer for sale became an important economic activity for women. The type of beer produced loses its alcohol content after 24 hours, so any beer not sold was consumed by the family, including the infants and children. Sandra Gray and Mary B. Sundal (2017, p. 673) reported that, "Not uncommonly, beer constituted the entire evening meal, and children's only food during the day was dregs" from beer making. To survive the people practiced nutritional buffering as a household strategy for accommodation to food scarcity. "The greatest portion of food was fed to the youngest children; older children received smaller portions. If milk was available, it was consumed exclusively by the youngest children. Adults often went hungry or satisfied themselves with a cup of beer. The weakest elderly were the least likely to eat on any given day. Indeed, in our experience, death from 'old age,' commonly reported as a cause of death, was actually culturally sanctioned voluntary starvation or neglect of the very old" (p. 675).

Even though mothers recognized that even a little milk improved the health and growth of their children the dire economic conditions forced women to see cows for their exchange value to purchase sorghum or maize. Women and their families were chronically stressed by their lack of economic alternatives. Without the animals that had been the basis of their livelihoods and culture the people became physically and emotionally immobilized. Quoting one pastoralist mother, "Where does this hunger want me to go? I don't know where to go. I am not thinking of migrating because I don't have any money. Where can I go?" (Gray & Sundal 2017, p. 679).

Today malnutrition is the norm and as the pastoralists say milk has gone, "The baby cried, 'Milk! Milk!'. . . We told him, 'Milk has gone! Milk has gone!'" (from the fieldnotes of Gray & Sundal 2017, p. 677). The tall and imposing East African pastoralists who once appeared in Hollywood films such as the 1950 production of *King Solomon's Mines* and the 1953 film *Mogambo*, directed by John Ford and starring Clark Gable, Grace Kelly, and Ava Gardner, have been reduced to a people with nearly 60% severe stunting for infants and children. Jumping contests and dancing are still performed by those few Samburu groups that make a tolerable living from tourism, but the majority of the populations' worries are mostly about AK-47 attacks from neighboring tribes and the military and starvation (Gray et al. 2003; Gray & Sundal 2017).

Addendum: Strategic Growth Adjustments?

East African pastoralists once prided themselves on their tallness and the linearity of their physique. The biocultural life history stage of adolescence was socially extended to about age 30 years. This was so, in part, because older men controlled the cattle and cattle were needed to secure a young woman in marriage. Extended adolescence was also due to a delayed rate of maturation in boys, with height growth continuing until to age 23 years and beyond. It is possible that this pattern of development was influenced by strategic growth adjustment. The social dominance of older men may have repressed the adolescents who were unable to marry until they were about 30 years old and had accumulated enough cattle. Marriage marked the major transition from adolescence to adulthood. Unmarried young men formed groups similar to adolescent gangs in the United States and Europe. These were well known for their solidarity and bravado. Before the nutritional crisis of 1980 these groups were highly competitive, but nonviolent. Samburu "gangs" would face off in "height contests." One of these contests involved jumping to see who could lift his long and lithe body higher. The jumping was done in the presence of adolescent girls who tapped out a rhythmic beat for the show. The highest jumpers received the most attention from the girls and were considered the most virile. The social solidarity and the height contests of the "gangs" were accompanied with much positive emotional pressure toward greater stature, especially longer legs for more effective jumping. This Samburu example suggests how the adolescent community effects might modify biological life history development and influence strategic growth adjustments.

supplement had no effect on growth, it was concluded that some factor in milk, either whole or skimmed, accelerated growth. However, a reanalysis of the Scottish studies by Celia Petty (1989) found that the milk supplemented groups included " ... a disproportionate number of children who were stunted (i.e., whose height for age was below the third centile) ... " (p. 106), compared with either the group receiving the biscuit supplement or the unsupplemented group. This indicates that there was selection bias in the allocation of children to the different groups. The children who were seen to be in greatest need were allocated to the milk groups. Petty proposed that catch-up growth following a period of severe undernutrition is why the milk groups grew significantly faster than the nonmilk groups. Any other food with similar nutritional qualities to milk in terms of protein, fats, vitamins, and minerals may have produced the same catch-up.

Eiji Takahashi (1984) proposed a central role of milk for the secular trend of increasing height in Japan. Rice is the dietary staple of Japan. Until 1950, fish and shellfish were the major sources of animal protein, although most dietary protein was of plant origin, from soybean products. Post-war changes in Japan, including greater contact with Western cultures and economic development, altered the traditional

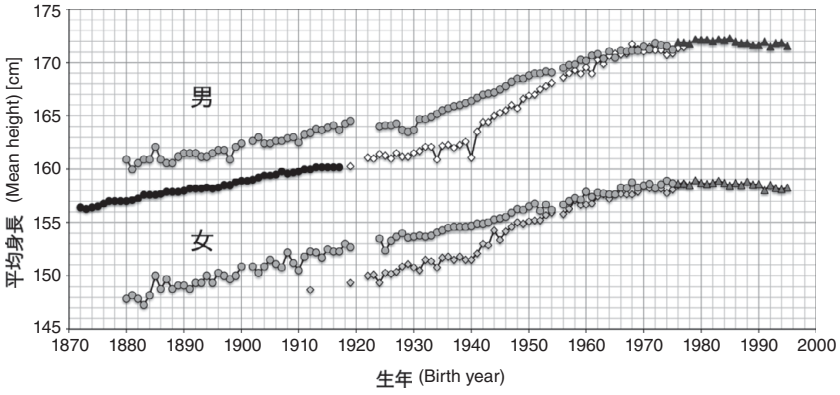


Figure 7.5 The relationship between the year of birth and average adult height for Japanese born after 1870, based on government statistics. The data for students for the years 1924–1927 and for the general population for the years 1919–1926 are for height measured at ages 21–35 years. All other data are for height measured at age 20-24 years. "The average adult height of Japanese young men and women increased with time, especially in the generations of the 1940s and 1950s after World War II. However, the rate of change has slowed in generations born after the mid-1960s and has stopped in generations born after 1980" (translated from the original Japanese, Kouchi 1996). The upper two plots are for men and lower two plots for women. The symbols indicate: ● students, ◉ conscripts, ◇ general population, ▲ post-1975 students and general population. Source: Dr. Makiko Kouchi, National Institute of Advanced Industrial Science and Technology, reprinted with kind permission of the author.

diet. These diet changes began in the late 1950s but became pronounced in the mid-1960s. From 1966 to 1976, rice consumption decreased from about 350 to 225 grams per person per day. During the same time, meat consumption rose from about 35 to 60 grams per person per day and milk consumption rose from about 55 to 100 grams per person per day. The height of schoolboys, aged 6 to 17 years, rose by an average of 4.1 cm between 1930 and 1960, a period of relatively great social and economic change that included World War II and the deprivations and hunger of the immediate post-war period. Average height rose even faster, by an average of 5.3 cm, between 1960 and 1975 so that by the end of this period the average height of 17-year-old Japanese boys was 168 cm. Takahashi attributed almost all the increase in the 1960 to 1975 period to changes in diet, especially the increased consumption of milk. The 9.4 cm increase in 45 years is remarkable but how much of it was due directly to milk consumption and how much to other factors? Reductions in infant and childhood infectious disease and reduced family size likely contributed to some of the height increase. Social and political changes due to democratization, especially the breakdown of the power of the monarchy and aristocracy after the war, rural-to-urban migration, universal education, and reduction of social inequalities all led to more opportunities and hope for the lower social classes. And it was this group that experienced the greatest increases in height, as may be seen in Figure 7.5.

Shown in this figure are height at adulthood by birth year in Japan. The data for "students" were based on measurements of university students, who are the upper social class of Japan. "Conscripts" and "general population" are the lower social

classes. The data for students for the years 1924–1927 and for the general population for the years 1919–1926 are for height measured at ages 21–35 years. All other data are for height measured at age 20 years (Kouchi 1996). The steepest increase in average height for the general population occurred from the late 1930s to the early 1950s. These are people born 20 years earlier, growing up from the late 1910s to 1920s. This was decades before the increased consumption of milk that followed World War II. A more likely reason for the strong positive height trend of the general population was the early twentieth-century industrialization and urbanization of Japan that brought about major changes in SEPE factors, especially for the lower social classes.

The Search for Milk's "Special Property"

Hoppe, Mølgaard, and Michaelsen (2006) suggested that milk promotes growth in height due to the stimulation of IGF-1 production in the human body by some factor in cow's milk. Hoppe and colleagues noted that cow's milk is a complex of bioactive substances. Milk is energy-dense from fats, proteins, and carbohydrates and these sources of energy also offer essential and nonessential lipids and amino acids. Cow's milk also offers a variety of micronutrients including the minerals calcium, magnesium, sodium, potassium, phosphorous, chlorine, iron, copper, zinc, and selenium. Cow's milk includes the fat-soluble vitamins A, D, E, and K and water-soluble vitamins thiamine (B1), riboflavin (B2), niacin (B3), pantothenic acid (B5), pyridoxine (B6), biotin (B7), folate (B9), cobalamin (B12), and ascorbic acid (C). The availability of this variety and concentration of nutrients is unique to milk and is not found in any other single food (Gaucheron 2011).

There is no convincing evidence that excesses of the vitamin or mineral nutrients contained in milk will, on their own, promote greater height growth and some excesses may be toxic. So, most research on the "special property" growth factor in milk centers on IGF-1. In addition to a possible IGF-1 stimulating property, cow's milk contains IGF-1 synthesized by the cow. Cow IGF-1 is structurally identical to human IGF-1 (Hoppe et al. 2006). But, it is not known if IGF-1 in cow's milk has bioactivity in humans. Hoppe and colleagues hypothesize that human IGF-1, stimulated by cow's milk, is key and cite findings from the Avon Longitudinal Study of Parents and Children (ALSPAC). ALSPAC is following a cohort of British infants born in the early 1990s. One analysis investigated associations between diet, IGF-1, and IGFBP-3 levels in a sample of 521 juveniles, aged 7- to 8-years-old. ALSPAC collects routine data on dietary intake, IGF levels, and many family demographic and socioeconomic variables. The study reported that protein intake from animal, but not vegetable, sources was associated strongly with both IGF-1 and IGFBP-3. Further analysis of the animal protein intake found that the IGF-1 effect was due to dairy, but not meat, consumption. Dairy products, including milk, cheeses, butter, and yogurt, accounted for approximately 25% of total protein intake in this sample of juveniles.

Other observational studies reviewed by Hoppe and colleagues reported no association between milk intake and IGF-1. Despite the mixed results, the research

team concluded " . . . that increased intakes of cow's milk are able to increase the concentration of IGF-1 in the circulation" (2006, p. 146). My assessment of the evidence that Hoppe and colleagues review is that their conclusion appears to be wishful and magical thinking. Only two of the three carefully controlled investigations they review reported an IGF-1 effect and the statistical significance of the effect is questionable with some of the reported "p" values greater than 0.05. Endocrine and pharmacological research generally reports significance when "p" values are less than 0.01, because there are many possible confounding variables. Hoppe and colleagues note some of these confounders in their review. Only the ALSPAC study associated greater IGF-1 with increased height and even that association does not prove causality. Cow's milk consumption may increase IGF-1 production, but there is no known mechanism by which greater IGF-1 in an otherwise healthy, well-nourished person with their own adequate production of IGF-1 production will result in greater height. Children deficient in their IGF-1 will respond to human IGF-1 injections by growing taller, but the effect is limited to the first year of treatment and by the second year growth rates decline even with equal or greater amounts of supplied IGF-1 (Laron 2001; Lui et al. 2019). It is unethical to inject healthy children with IGF-1, so it is not known if they would respond differently compared with IGF-1 deficient children.

Since the review by Hoppe and colleagues an association study by Andrea Wiley (2012) reported that greater milk consumption was associated with greater height in childhood and adolescence, but not during the juvenile growth stage. Wiley analyzed data from the United States National Health and Nutrition Examination Survey (NHANES) collected from 1999 to 2004. Adjusting for the available social and economic indicators in the NHANES sample, she found that the association was limited to milk and not dairy foods or meat. Wiley also proposed that IGF-1 is the likely bioactive substance linking milk consumption to greater height growth, but she made clear that the mechanism for this effect is unknown.

Taking a slightly different perspective, a study by Hrolfsdottir and colleagues (2013) analyzed the association between maternal milk consumption, the size at birth of their offspring, and the adult height of offspring. This was a prospective cohort study of 809 Danish pregnant women, recruited in 1988–1989. The adult offspring (n = 685) were measured at ~20 years of age. The mothers were divided into two groups: (1) milk consumption of $\geq$150 ml/day and (2) milk consumption $<$150 ml/day. Milk consumption by the offspring was not measured. There were no statistically significant differences in birth length or adult height for the offspring of these women.

The effect of milk proteins on linear growth and IGF variables in overweight adolescents was investigated by Larnkjær and colleagues (2014). The sample included 193 overweight adolescents aged 12–15 years. They were randomized into four groups to drink 1-liter (L) per day of: (1) skimmed milk, (2) whey, (3) casein, or (4) water for 12 weeks. Whey and casein are the two major proteins in cow's milk.

All milk-based drinks contained 35 g protein/L. A pre-test control group of 32 same-aged adolescents was examined 12 weeks before the main study began. Skimmed milk was associated with increased IGF-1, but there was no difference in rate of height or height z-score change for any of the milk-based test drink groups compared to water or compared to the pre-test control group. In fact, height z-score decreased within the whey group. Perhaps 12 weeks was not long enough for the IGF-1 increase to be translated into greater height. Perhaps the overweight status of participants interacted with the drink treatments. Or perhaps the milk effect hypothesis on height is just not valid.

Are Milk Drinkers Really Taller?

The answer is, probably not. For this book I compiled data on milk consumption and average adult height for 27 nations. The findings may be seen in Figure 7.6. Most of the data are for the wealthier nations of Europe, North America, Australia, and New Zealand. The exceptions are India and Uruguay. This is not surprising because the biological capacity to drink milk is diminished or eliminated in more than 65% of the human population over the age of 7 years due to the genetic down-regulation of lactase synthesis. That down-regulation may begin with the infancy to childhood life

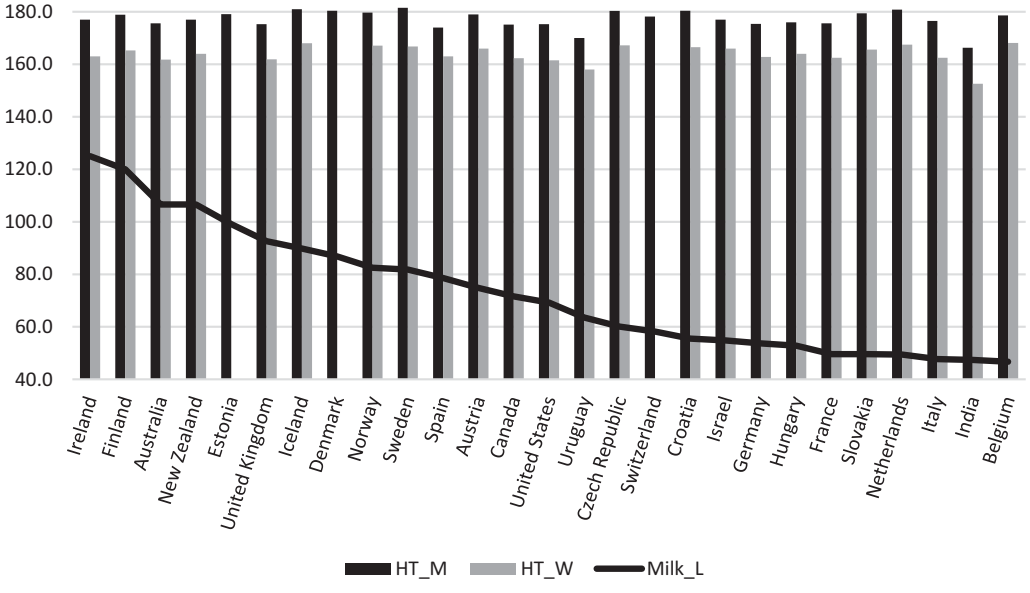


Figure 7.6 Mean adult heights for men (HT_M) and women (HT_W) for 27 countries and per capita fluid milk consumption. The Y-axis indicates average height in cm and per capita fluid milk consumption in liters per year. Height data from http://worldpopulationreview.com/countries/average-height-by-country/#dataTable, milk consumption data from www.statista.com/statistics/535806/consumption-of-fluid-milk-per-capita-worldwide-country/. Original figure.

history stage transition and is typically complete by the end of childhood. The enzyme lactase is needed to digest the milk sugar lactose. Without lactase people suffer digestive distress ranging from abdominal bloating and cramps to flatulence, diarrhea, nausea, and vomiting. In European people (the population of Uruguay is predominately of European origin), a few African populations, and much of India the adult population typically has lactase persistence and the prevalence of adult lactose deficiency is less than 10%. It is estimated that at least four different genetic mutations allow for lactase persistence. These genetic mutations arose at different times in different human populations. All human groups with the mutations include dairy herding and the consumption of milk and dairy foods as part of their historical and current livelihood. All these societies have a cultural view of milk as a "good food" in terms of its health benefits, including healthy growth and development (Bogin 1998b, 1998a; Gerbault et al. 2011). "Milk makes tired men cheerful" was an advertising slogan of the West German dairy industry, developed in the 1950s. "Nature's perfect food" was a slogan of the US dairy industry (Dupuis 2002). Lactase persistence is a powerful human example of genetic natural selection and convergent evolution in response to the biocultural practice of dairy herding interacting with emotional and ideological attitudes toward milk.

The amount of fluid milk consumed per person varies quite a bit between groups with lactase persistence biology. The data available in Figure 7.6 show that the highest amount is 125 liters/year/person for Ireland and the lowest is 46.7 liters/year/person for Belgium. The Belgians are 12th tallest and the Irish are 14th tallest. The heights of the other national samples do not appear to have a relationship with milk consumption. Formal statistical analysis found no significant, or even marginally significant, correlation between fluid milk consumption per person and adult height for men ($r = 0.14$) or women ($r = 0.06$). The tallest countries in the world rank lower than predicted by the milk hypothesis – Iceland is at 7th place in milk consumption, Denmark is 8th, Sweden is 10th, but relatively "short" Spain ranks 11th. Netherlands men are sometimes ranked as the tallest in the world, but in terms of fluid milk consumption Dutch men and women rank 24th, just above Italy, India, and Belgium. I located another set of data on milk consumption, which included 12 of the European nations shown in Figure 7.6 as well as Greece, China, and Brazil and analyzed these data using the estimates for height at age 18 years from the NCD Risk Factor Collaboration (NCD-RisC 2016). The data may be seen in Table 7.1. The correlation between fluid milk consumption per capita and height for men was $r = 0.44$ and for women was $r = 0.42$. These correlation coefficients are larger than for the previous analysis, but neither is statistically significant at $p < 0.05$. Included in Table 7.1 are data on the annual per capita consumption of cheeses and butter. There are statistically significant correlations between milk and cheese consumption, $r = -0.61$, and cheese consumption and height for men, $r = -0.77$, and for women, $r = -0.76$. The negative sign of these correlations means that greater cheese consumption is associated with less fluid milk intake and with shorter height. A simple-minded interpretation would be that cheese inhibits height

Table 7.1 Per capita consumption of milk (L) and milk products (kg) in various countries, 2011 data (www.uoguelph.ca/foodscience/table-1-0) and height (cm) at age 18 years for men and women (NCD Risk Factor Collaboration (NCD-RisC) 2016).

Country	Fluid milk	Cheeses	Butter	Height men	Height women
Netherlands	47.5	19.4	3.3	182.54	168.72
Germany	51.8	22.9	5.9	179.88	165.86
France	55.5	26.3	7.5	179.74	164.88
Sweden	90.1	19.1	1.7	179.74	165.7
Finland	127	22.5	4.1	179.59	165.9
Australia	105.3	11.7	4	179.2	165.86
Ireland	135.6	6.7	2.4	178.93	165.11
Canada	78.4	12.3	2.5	178.09	163.91
Italy	54.2	21.8	2.3	177.77	164.61
UK	105.9	10.9	3	177.49	164.4
Greece	49.1	23.4	0.7	177.32	164.87
USA	75.8	15.1	2.8	177.13	163.54
Brazil	55.7	3.6	0.4	173.55	160.86
China	9.1		0.1	171.83	159.71
India	39.5		3.5	164.95	152.59

growth, but this is spurious and silly. It does show that all types of association analysis need to be interpreted with caution. A cautious interpretation of the two analyses presented here is that average adult height in a country is unrelated to the annual per person consumption of fluid milk.

Vitamin D_3: The Effect of a Specific Nutrient

For more than a century one nutrient, vitamin D, has been touted as a growth stimulant. Even before vitamin D was biochemically isolated in 1932, products were recommended for consumption that may have contained vitamin D – such as cod liver oil and various "snake oils" sold by dodgy "health promotors."[19] Vitamin D is fat-soluble and can be supplied in the diet by fatty foods, such as the flesh of fatty fish (e.g., tuna, mackerel, salmon, and cod – hence cod liver oil), beef liver, and egg yolks. However, these foods do not contain enough vitamin D, are expensive and not eaten in sufficient quantity, or have about 50% of their vitamin D content destroyed when cooked. So, dietary intake is usually not enough to achieve optimal status. For optimal nutrient status people must either ingest vitamin D supplements or have adequate exposure to sunlight.

[19] Snake oil refers to the petroleum-based mineral oil that was sold as a cure-all elixir for many kinds of physiological problems. The oil may have been mixed with various active and inactive household herbs, spices, and compounds. The phrase "snake oil" is a euphemism for deceptive marketing and a "snake oil" salesperson is someone who knowingly sells fraudulent goods or who is himself or herself a fraud, quack, or charlatan (https://en.wikipedia.org/wiki/Snake_oil).

A relation between light and growth has been known since 1921, when it was shown that ultraviolet light could cure rickets, a disease of bone growth. Two years earlier it was demonstrated that cod liver oil could cure rickets even when the vitamin A in the oil was destroyed. This led to the proposal of the exitance of a new vitamin – D. Vitamin D_2, ergocalciferol was chemically isolated from plant sources in 1932 and cholecalciferol, D_3 was isolated in 1937. A few years later the relationship between vitamin D and normal bone growth was demonstrated (Deluca 2014). It is now known that when people are exposed to ultraviolet light the vitamin D_3 precursor cholecalciferol is synthesized by the skin, then carried by the blood stream to the liver and finally to the kidneys where the cholecalciferol is modified to the active form of vitamin D_3, called 1α, 25-dihydroxyvitamin D (1α,25-$(OH)_2D_3$). The sites of action of 1α,25-$(OH)_2D_3$, the intestine and the skeleton, are where the physiologically active form of vitamin D_3 increases the intestinal absorption of calcium and controls the rate of skeletal remodeling and the mineralization of new bone tissue (Basit 2013). Given these physiological characteristics it is more accurate to classify Vitamin D_3 as a steroid hormone as much or more than an essential nutrient. Like many hormones, vitamin D_3 is produced by a body organ, the skin, then enhanced by the liver and kidneys, and is transported to its sites of action via the blood stream. There is a vitamin D binding protein that functions to transport all forms of vitamin D and its metabolites between skin, liver, and kidney, and then on to the various target tissues. At the cellular level of the target tissues there is a vitamin D receptor that activates nuclear DNA to promote tissue activity required for growth, development, and maturation. Vitamin D interacts with two other hormones, calcitonin and parathyroid hormone, to regulate serum calcium balance, which is essential for muscle contraction, oocyte activation, tooth enamel formation, blood clotting, nerve impulse transmission, regulating heartbeat, and fluid balance within cells, and bone formation.

In sum, vitamin D is a hormone that is essential for calcium metabolism and normal bone growth, and thus growth in height. The endocrine physiology of vitamin D is essentially the same for the D_2 and D_3 forms. In the past, plant derived vitamin D_2 was commonly added to milk and other foods in the belief that had the same general properties of D_3. However, D_3 is many times more potent in action (Tripkovic et al. 2012; Wurtman 1975) and is the preferred form for humans.

Seasonal Growth and Vitamin D_3

Annual variation in daylength, and thus sunlight, as well as opportunities to be exposed to sunlight due to sociocultural practices, such as style of clothing and indoor schooling vs. holiday time could influence vitamin D_3 synthesis and skeletal growth in young people. Buffon (1777) published in *Histoire Naturelle*, his encyclopedia of living things, the first data suggesting the existence of seasonal variation in the growth rates of healthy children. Studying the growth records of the son of Count Montbeillard, Buffon noted that most of the boy's height increase during the year took place in the spring and summer months. Scammon (Miller 2018; Scammon

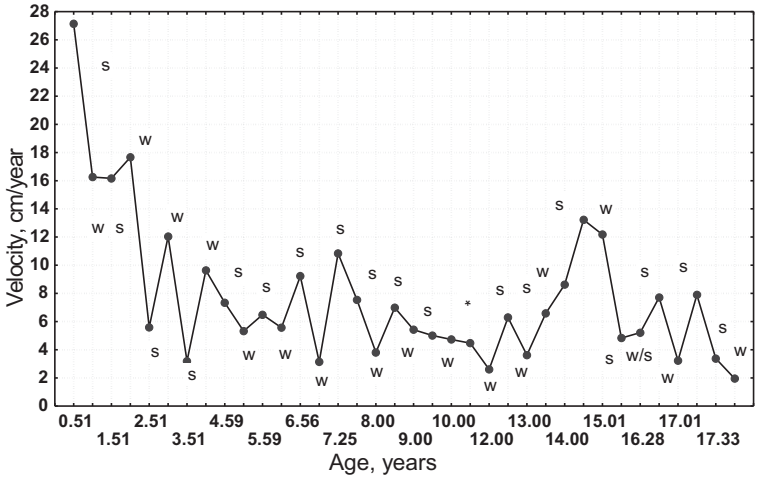


Figure 7.7 Seasonal variation in the velocity of growth of Montbeillard's son from age 6 months to 18 years. S indicates the "summer" measurement, which was taken in October, meaning that it followed six months of growth over the spring and summer months. W indicates the "winter" measurement, which was taken in April, meaning that it followed 6 months of growth over the fall and winter months. The * indicates an interval of 1 year between successive measurements, which precludes a seasonal effect. The W/S symbol indicates a time period from October to July, which includes some of the summer months.

1927) converted the old French metric units used by Buffon into modern metric values. These data are used to construct the seasonal curve of growth for Montbeillard's son shown in Figure 7.7. The boy was born on April 11, 1759 and his father measured his length or height at six-month intervals, with a few exceptions. The boy's growth from April to October took place during the seasons of spring and summer in France, while the period of growth from October to April covered the seasons of fall and winter. Until about age four years, the boy grew faster during the fall and winter, but after that age grew faster during the spring and summer. During his adolescent height spurt, from ages 13 to 15.6 years, the seasonal effect seems to be obliterated.

Since Buffon's time, dozens of investigations of seasonal growth, using larger sample sizes, have generally confirmed that at temperate latitudes, healthy well-nourished children grow more quickly in height during the spring and summer than they do during the fall and winter (Bogin 1977, 1978; Satake 1994). The summer vs. winter difference in height velocity is even found in boys and girls, ages 3–14 years old, with idiopathic growth hormone deficiency who were receiving recombinant human growth hormone (rhGH) therapy (Shulman et al. 2013). Data for this study came from medical clinics in the United States at different latitudes. Because latitude influences several climate variables, "season" was analyzed by hours of daylength per month at the latitude of the clinic. The difference between the summer and winter rates of growth was " ... greatest in the first year of therapy 0.146 cm/yr/daylight hour; $P < 0.0001$) but persisted in subsequent years (0.121 cm/yr/daylight hr;

$P < 0.0001$)" (p. 1). The difference in height gain between summer and winter increased with distance from the equator, but at all latitudes a statistically significant difference was noted. At the Anchorage, Alaska clinic (61° N latitude, 17.9 hours summer daylength) the seasonal height velocity difference was 1.7 cm/yr. At the Honolulu, Hawaii clinic (21° North latitude, 13.1 hours summer daylength) the difference was 0.32 cm/yr.

Similar results were found in another study of pre-pubertal juveniles with GH deficiency receiving rhGH therapy (Land et al. 2005). In this German study the patients were divided into two groups, with Group 1 starting therapy during the spring and summer (greater daylength) and Group 2 starting therapy in the autumn or winter (shorter daylength). The juveniles of Group 1 had a significantly greater bone formation and height growth response. In sum, these findings suggest that season of the year acting through daylength and sunlight exposure influence skeletal growth velocity and, even, the efficacy of treatments to promote height growth. Other factors might also play a role, including physical activity, diet preferences, social activity, and emotional status at different times of the year.

How to Explain Seasonal Variation in Height?

The mechanism for seasonal variation in height growth rate is not completely understood; however, the bulk of the evidence points to seasonal periodicity in sunlight and vitamin D_3 synthesis as the cause. Vitamin D_3 has several effects on the human endocrine system. The effect of seasonal variation in sunlight, especially the ultraviolet radiation of sunlight, seems to be one that synchronizes the body's natural fluctuations in growth-regulating hormone activity so that all the necessary hormones are working simultaneously to speed up or slow down the rate of skeletal growth. An experimental study of the sunlight effect published by Karl Gustav Nylin (1892–1961) was probably the first to test this hypothesis. Nylin (1929) tested the sunlight effect on two groups of Swedish boys. One group of boys (n = 45) was treated during the winter months in Stockholm (from winter solstice to spring equinox) with "sunlamp" exposure using a lamp that produced both visible and ultraviolet light. These treated boys were compared with a control group of boys (n = 292) not receiving treatment. Situated at about 59° N latitude, Stockholm has only 6 hours of daylight at winter solstice. During the 3-month period of treatment, the experimental group averaged 1.5 cm more growth in height than the control group. During the summer, the control group grew at a faster rate than the experimental group, so that over the entire year there was no difference between groups in total height gain. Only the time of year when the maximum gain occurred differed between groups. One interpretation is that during the years of growth there is a set amount of height increase, that is a target height, for the year. The ultraviolet light from the sunlamps allowed for synthesis of vitamin D_3 and "forced" the experimental group to have an early maximum growth velocity and reach their target for the year. The control group also grew to target later in the year when more natural ultraviolet light was available during the summer months.

Moving from Sweden to the tropics, Vincent and Dierickx (1960) found that healthy children living near the equator in Kinshasa (then Leopoldville), Zaire, grew more rapidly in height in the dry season than in the rainy season. Diet, temperature, humidity, and sunlight variation were considered as possible influences. No evidence in favor of the first three was found. Although day length was slightly longer during the rainy season, there were far more hours of insolation (bright sunshine), and more opportunity for children to be exposed to the sun, during the dry season. The authors concluded that exposure to sunlight could regulate growth rate.

In the tropics, where variation in day length is minimal, the amount of ultraviolet light reaching the ground is a function of the amount of cloud cover blocking the sun's radiation. Physicists call the process whereby light energy is reduced when passing through any medium "extinction." The extinction of ultraviolet radiation occurs when sunlight entering a cloud is scattered by collisions with water droplets or suspended particles. It is estimated that a thick white cloud will extinguish 90% of the ultraviolet light entering it (Van de Hulst 1957). Thus, at tropical latitudes, the measurement of hours of insolation provides an accurate indicator of available ultraviolet light and rate of vitamin D_3 synthesis in the body.

Moving back to the northern latitudes, Marshall and Swan (1971) compared the monthly growth rates over a year's time of blind and normally sighted children living in southern England. Both groups showed rhythmic changes in growth rate of equal magnitude. The months of maximum growth of individual blind children were distributed evenly throughout the year. The maximum growth rates of the normally sighted children virtually all occurred between the months of January and June. The authors suggested that seasonal variation in day length might have entrained the growth rates of normally sighted children, but no mechanism was known to explain this. Nevertheless, the results do add support to the hypothesis that month-to-month rates of growth vary within an individual and this results in a natural endogenous rhythm to growth. To further investigate this hypothesis, Marshall (1975) analyzed monthly measurements of height, taken over a 2 year period, for 300 healthy children living on the Orkney Islands (59° N latitude). Marshall also recorded changes in day length, hours of insolation (hours during the day when the sun is not obscured by clouds or haze), rainfall, and temperature. Relatively small, negative, but statistically significant correlations were found between monthly increments of height and the meteorological variables. The strongest correlation between monthly height increment was with the mean of the maximal daily temperatures (-0.32) for the boys and with hours of bright sunshine (-0.29) for the girls. The children's mean growth rates reached maxima at midwinter and minima at midsummer. This is the opposite of the prediction of the daylength or hours of insolation hypotheses. Marshall concluded that there was no support for the climate hypothesis and that, "The influence of other factors, as yet unidentified, is greater" (p. 243).

The use of correlation analysis may have biased the results of Marshall's studies because correlation alone cannot detect period (seasonal) patterns in the growth data of the same people measured repeatedly. Month-to-month growth of an individual is

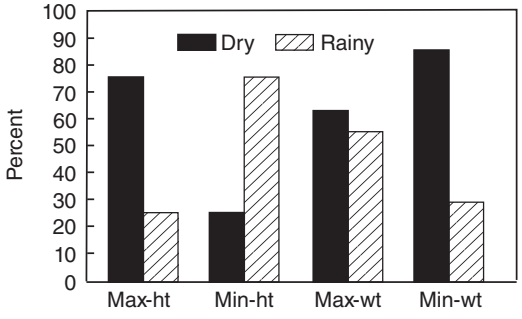


Figure 7.8 Seasonal variation in height and weight growth of healthy, high SES Guatemalan children. The sample includes 27 boys and 21 girls, aged 5–6.9 years old. The timing of maximum and minimum rates of growth in height follow a true seasonal trend. About 75% of the children have maximum rates of height growth in the dry season and minimum rates of height growth in the rainy season. Rate of weight growth does not follow a seasonal trend. About equal numbers of children have maximum weight gains in both the dry and rainy season. A significantly greater percentage of children have minimum weight increments, including weight loss, in the dry season.

auto-correlated, meaning that the preceding and subsequent measurements influence the current measurement of height, weight, or other variables.

For my own research into seasonal growth variation I used periodic regression and harmonic analysis, which accounts for the autocorrelation and provides a less biased estimate of the annual rhythm of growth (Bogin 1977, 1978). Starting in September 1974, I measured monthly increments of height growth over a 14-month period for a sample of 164 healthy children, juveniles, and adolescents of high socioeconomic status living in Guatemala City (14° N latitude). The participants in my study included 3 groups of school students: (1) 27 boys and 21 girls, aged 5–6.9 years; (2) 28 boys and 25 girls, aged 11–13.9 years; and (3) 30 boys and 33 girls aged 14–17.9 years. The periodic regression and harmonic analysis results indicated a seasonal rhythm in height growth for boys and girls of Group 1 and girls of Group 3. Group 1, boys and girls in the childhood stage of growth, showed the clearest evidence of a true seasonal pattern of growth in height. About 75% of the children grew at a significantly faster rate during the dry season than during the rainy season (Figure 7.8). Conversely, about 25% of these children grew at their fastest rate during the rainy season and their slowest rate in the dry season. Results such as these are found in most seasonal growth studies, but the reason why a minority of children follow a pattern that is just the opposite to that of the majority has never been explained.

Why the growth of the Group 2 girls and boys and the boys of Group 3 did not follow a seasonal rhythm is not known. One possibility is that these participants were experiencing their adolescent growth phase and that the hormonal regulation of the adolescent growth spurt was overriding the seasonal effect. The impacts of social and emotional stress during adolescence, which have hormonal consequences, may also have been stronger than seasonal effects. The Group 3 girls may have been near or at

the end of their adolescent growth spurt and have returned to a greater susceptibility to seasonal effects.

A possible explanation for the seasonal pattern followed by most of students is linked to hours of insolation. As in the African study of Vincent and Dierickx, day length in Guatemala City was longer during the rainy season, by about 1.5 hours, but there were significantly more hours of insolation during the dry season (1,662 hours from October to April) than during the rainy season (962.8 hours from May to September). Both in equatorial Africa, and in Guatemala, therefore, the strongest association between sunlight and growth rate is not for day length, rather it is for hours of insolation. More hours of insolation would increase the opportunity for human bodies to synthesize more vitamin D_3.

Other research reports significant month-to-month variation in the circulatory levels of vitamin D_3 and its metabolites. The amount of vitamin D_3 in the blood rises and falls in direct proportion with the availability of ultraviolet radiation from the sun. The consensus of research is the major source of vitamin D_3 is the body's own synthesis of the D_3 form, and not dietary ingestion by food or supplements of D_3 or ingestion of foods fortified with vitamin D_2. At the latitude of London, the amount of ultraviolet light penetrating the atmosphere and reaching the ground at noon is 15 times greater in June than in December (Wurtman 1975). At northern and southern latitudes, variation in day length, outdoor exposure, and clothing requirements increase the difference between seasons in the exposure of people to ultraviolet light.

While many reports propose that dietary vitamin D in either of its forms may be of little importance, the amount of vitamin D_3 in cow's milk could be. Samuel Brody (1945, p. 211), whose work on growth theory was reviewed in Chapter 3, noted a strong seasonal variation in the concentration of vitamin D_3, as well as vitamin A, other vitamins, several minerals, and fatty acids, in cow's milk. Brody reported that in the northern hemisphere, the July levels of vitamin D_3 were at 200% of the yearly average, while the December levels were about 35% of the yearly average. Today, processed milk sold at food markets, at least those in the United States, has uniform levels of vitamin D_3 throughout the year. Such uniformity did not exist 45 or 50 years ago, when much processed milk in the United States was fortified with the less efficacious vitamin D_2 form. It is possible, therefore, that the seasonal variation in the concentration of vitamin D_3 in cow's milk may have contributed to bioavailability of this vitamin in the diet of boys and girls studied prior to 1970, and their seasonal pattern of growth in height. Perhaps this explains, in part, the positive milk hypothesis findings of nineteenth- and early-to-middle twentieth-century research.

There is much ongoing research into the importance of vitamin D_3 in health and disease. Much of this relates to adult metabolic health and bone density in pre-adults. This research is not reviewed here. Other research focuses on vitamin D_3 supplementation of pregnant women and birth outcomes. One study is described here, by Daniel Roth and colleagues (2013). This was a randomized double-blind trial that took place in Dhaka, Bangladesh. The trial provided vitamin D_3 at 35,000 IU/wk or a placebo to

women in their third trimester of pregnancy. The researchers reported that there were no differences in newborn length-for-age z-score (LAZ) between groups at birth, but LAZ was 0.44 (95% CI, 0.06–0.82) higher in vitamin D group vs. placebo group at 12 months. The average absolute difference in one-year-old length was an increase of 1.1 cm (95% CI, 0.06–2) in the treatment group. The significant difference in LAZ was established by 1 month of age, with no significant additional divergence thereafter. The incidence of stunting was lower in the infants whose mothers received the vitamin D (17% vs. 31% of the placebo infants). Other anthropometric measurements, such as weight and head circumference, were similar between groups. The women participating in this study were known to have low-to-deficient serum vitamin D_3 levels, so it is not surprising that the intervention had a positive effect.

In human groups with adequate diets, the availability of ultraviolet light, and the opportunity to be exposed to it, is the mostly likely explanation for seasonal variation in height growth. Greater amounts and exposure to ultraviolet light increase the rate of vitamin D_3 synthesis by the human body. This promotes both calcium absorption by the intestines and skeletal remodeling and bone mineralization. The outcome is likely to be a maximization of the rate of growth in height toward its annual target amount. Direct studies of these relationships have yet to be done.

Seasonal Growth in Weight

Studies of seasonal variation in weight gain and loss find that for healthy individuals, fall or winter are the seasons of maximum weight gain and summer tends to be the season of minimum weight gain for children, and the time of maximum weight loss for adults. Several researchers also find that minimum weight gains, or even weight losses, occur simultaneously with maximum height gains (Bogin 1979; Tobe et al. 1994). For my PhD thesis, I reviewed the literature on seasonal growth and found that 22 of the 29 studies located reported that maximum increments in height and weight do not occur at the same time of the year (Bogin, 1977). The authors of some of these studies speculated that children have a natural, endogenous rhythm for growth in weight that is independent of both the seasonal rhythm of growth in height and of seasonal variation in climate.

In my own research (Bogin, 1979), I measured monthly increments of weight change of 48 healthy well-nourished children and juveniles, 27 boys and 21 girls aged 5–6.9 years, living in Guatemala. Most of the participants were from very high SES families. I found that minimum weight gains, and even weight loss, occurred for most individuals (90% of the boys and 80% of the girls) during the dry season, the time of maximum increments in height (Figure 7.8). The number of participants losing, or not gaining, weight showed a tendency to increase from month to month during the dry season. In the last three months of the dry season (March, April, and May), the percentage of boys and girls losing or not gaining weight rose from 30.5%, to 37%, and finally to 57.5%. The pattern of weight

change could not be explained by observable behavior related to diet, exercise, or disease. There may have been more physical activity during the dry season, after school and on weekends, that I did not observe. Perhaps the increased rate of metabolism required to grow in height at the fastest rate for the year required more energy than was supplied by the diet of the these, otherwise, healthy and wellnourished children. The children and juveniles may have lost body fat to supply the energy needed by the skeleton for growth. Unfortunately, I was young and inexperienced at the time of the study, and I did not take any measures of body composition. To my knowledge, monthly measurements of body composition have never been taken on a statistically reliable sample of healthy children, juveniles, or adolescents.

The Obesity Epidemic Changes the Seasonal Pattern

Maximal weight gain in winter and minimal weight gain in summer was the most commonly reported seasonal pattern prior to the onset of the pediatric overweight/obesity epidemic of the late twentieth and twenty-first centuries. Today, for some children the seasonal pattern is reversed, likely due to social, economic, political, and emotional (SEPE) factors. Baranowski, Moreno, Chen and colleagues have been studying body mass changes in a sample of 1,597 southeast Texas elementary school pupils, aged 5–12 years old, followed from kindergarten to 5th grade (Baranowski et al. 2014; Chen et al. 2016; Moreno et al. 2015). The researchers used the BMI to assign students to "healthy weight," "overweight," and "obese" groups – there were too few "underweight" children for statistical analysis. They reported that age and sex standardized BMI scores (zBMI) tended to increase during the summer school vacation period and decline during the school year for the children in all BMI groups, and for all four ethnicities included in the study (white, black, Hispanic, Asian). This pattern of weight gain and loss was consistent for all years of elementary schooling.

Due to the many problems of using BMI to assess fatness, especially in children and juveniles, it is not certain that the summer increase is due to fat gain. As summertime may be the season of faster height growth, the vacation-time increase in BMI may be due to greater skeletal and muscle mass. Moreover, these Texas studies measured height and weight only two times during the year, usually at the start of the term and about six months later or at the end of the school year, so they are not valid seasonal studies which require at least monthly measurements. Despite these limitations, it is likely that the summer holiday increase in BMI represents, in part, greater fatness.

What factors might account for the school vacation effect? The researchers suggest that it is due, primarily to a decrease in levels of physical activity (PA) during summer months. This may be due to the lack of the structured PA provided by schools, or by summer childcare arrangements that do not provide for PA, or changes in eating and in sleep, or some combination of these factors. The hot summer temperatures of Texas limit PA, especially for lower-income school students who cannot afford air-conditioned indoor play opportunities. Children may eat more or more energy-dense

food during the summer vacation. They may spend more time at sedentary activities such as TV viewing and small-screen gaming and social networking. These activities may not only lower energy expenditure but compete with PA and sleep. Low PA during the school holiday may translate into greater fat gain. Sleep duration has been inversely related to child fatness and summer holidays may result in less sleep per day. But, these were not measured in the Texas studies and seasonal patterns in PA and sleep need to be clearly established and then associated with changes in adiposity.

SEPE factors are also likely to be associated with the summer vacation gain of BMI/fatness. At least 40% of students were from low SES families based on the characterization of their schools as Title 1. A school receives Title 1 category when at least 40% of the families are low-income. Title 1 schools are given additional US Federal funding to help students perform better in school. The extra money is for academic programs, PA, and free or reduced-cost breakfasts and lunches. In the United States there is a significant and strong negative relationship between SES and overweight/obesity. In southeast Texas black and Hispanic families have the highest rates of poverty and low income. The Title 1 schools in the study had a higher zBMI than non-Title 1 schools ($p < 0.001$). Black and Hispanic children in the studies of summer BMI gain had a higher zBMI at all times of the year compared with the White and Asian ethnic groups ($p < 0.01$). Families of poverty and low-income status in the United States are strongly associated with lower educational attainment of the parents and their children as well as poorer and less secure health care, housing, amount and quality of the diet, opportunities for leisure activities including PA, and highly insecure livelihoods. Poverty and low-income families often have little political power in their communities and suffer discrimination and exclusion by more socially, economically, and politically powerful people and groups.

The social, economic, and political insecurities suffered by low-income families create adverse childhood experiences and toxic emotional stress. When this negative stress is chronic or when it is poorly managed it may lead to elevated cortisol levels, which are associated with poor sleep, enhanced appetite, food cravings, and decreased motivation for PA (Geiker et al. 2018). Singly or in combination these factors can contribute to weight gain and obesity. Cortisol is a growth hormone/IGF-1 axis inhibitor which can reduce growth in height (Bogin et al. 2015). For low-income families the summer school vacation period can be a time of increased stress due to problems of childcare, including the burden of food insecurity due to the need to provide meals at home that were provided at school – recall the discussion of school holiday hunger in the UK from Chapter 5. These SEPE stresses experienced by low-income families during the school vacation may easily translate in a gain of body fatness.

The authors of the Texas studies also propose that " ... the clear and consistent pattern of increases in zBMI during the summer and decreases in zBMI during the school year suggests that entrance into elementary school has a significant impact on the development of a seasonal pattern of weight gain that is observed in children regardless of race/ethnicity, gender, or SES" (Moreno et al. 2015, p. 495). The

researchers cite evidence of community effects on body weight and fatness. It is well known that social friendship networks have effects on human biology. Adults and children with more overweight friends are more likely to be overweight themselves. Conversely, having more healthy-weight friends reduces one's own risk of overweight (Christakis & Fowler 2007). More evidence of the biology of social networks is given in the section about migration, social networks, competitive, and strategic growth.

Month of Birth Effect

Seasonal variation in the rate of height growth is now a well-established phenomenon, and its causes are probably known. A month-of-birth effect on the height of both young people and adults has been reported in several studies. The validity of this phenomenon is less well established, and its causes are not understood. Weber and colleagues (1998) analyzed a sample of Austrian military conscripts of 507,125 men aged 18 years old. They reported that over the 10 years of data examined there was a clear seasonal variation in adult height with a maxima for men born in the spring months and a minima for men born in the autumn months. The average difference in height at age 18 years was 0.6 cm. Other studies report equally small differences, usually less than one centimeter (Douros et al. 2019; Henneberg & Louw 1990, 1993).

Weber and colleagues speculated that the month-of-birth effect may be caused by sunlight influencing the pattern of human growth during the late fetal and early postnatal period. The exact mechanism by which sunlight could do this is unknown. Weber's team speculated that the mechanism may involve the pineal gland, which is sensitive to periodicity in light and darkness, and its hormone melatonin. It is more likely that the month-of-birth effect relates to the serum vitamin D_3 levels of the mother during the last trimester of pregnancy. It is known that growth in length is faster during the last trimester and first few months after birth than at any later time of life. The stimulus of greater amount and intensity of sunlight and vitamin D during the spring and summer months may act on both the mother and the fetus/neonate to program a pattern of growth that is different than that for individuals born during the fall and winter months. Henneberg and Louw (1990) pointed out that there is no evidence of a seasonal effect on birth weight or birth length. So, the month of birth effect on growth must act on growth after birth, and possibly accumulate over time. The findings reported by Roth and colleagues from their Bangladesh vitamin D study are very similar to the proposal by Henneberg and Louw – just a coincidence? Infants born during the spring and summer may grow a bit more in the first year or so following birth than infants born in the fall and winter. By 6 years of age the birth month effect is detectable, significant statistically, and seems to persist to age 18.

The birth month effect is small in biological terms and may have little practical impact on an otherwise healthy individual. Nevertheless, the discovery of this effect and the eventual understanding of its causes, may help researchers to better

understand the control of human growth and may offer clinicians new treatments for infants and children with growth disorders.

Migration and Urbanization

Throughout human history people have moved from place to place. There are several definitions of the word **migrant** in the human biology literature. The definition used here is that migrants are people who move from one place of residence to another, especially from their place of birth to a geographically and socially new place of residence. This includes people who have migrated within the same country, such as rural-to-urban migrants. When all humans lived as hunters and gatherers, migration redistributed the population and exposed people to different climate biomes. There were consequences for human biology due to variation in temperature, altitude, food availability, predators, parasites, and the like. The impact of migration on SEPE factors was relatively low, since social, economic, and political organization of forager societies are, basically, similar from place to place and emotional stressors are shared similarly as well. Foragers in the past were not all the same size or shape. Based on skeletal remains, body heights varied as much as they do today in the wealthy, democratic nations of northwest Europe (Bogin 2001). Indeed, people in these modern nations seem to have returned to the average height and height variation that our species displayed in our history as foragers – the 99% of our history prior to the advent of animal and plant domestication in the past 20,000 years or so. Domestication led to horticulture, pastoralism, and agriculture. These new means of livelihood changed the effect of migration on human biology. The discussion in Chapter 5 relating to the 8,000 years of secular trend in Latin America, demonstrated that horticulture and agriculture, along with the concentration of people into state-level societies, resulted in a decline in human stature and human health. The formation of concentrated human populations and eventually cities, both in the time of the agricultural revolution and later in the time of the industrial revolution, required new types of migration, new types of social organization, new categories of social, economic, and political status, and new types of emotional stress. These changes imposed novel and powerful forces shaping both human biology and culture.

Throughout written history, social, economic, political, and biological events encouraged the development of cities and the migration of rural people to urban areas. The ancient Sumerians, Egyptians, Greeks, and Romans wrote of the virtues of the rural, farming livelihoods precisely because these were disappearing with the rise of cities. The word "urban" is derived from the 5,000 year old Sumerian term *uru.bar.ra*, referencing the "outer-city" with its concentration of farming areas around the "inner-city" where elites and their supporting bureaucrats lived (Oppenheim 1974). The "pull" of the city were its opportunities for employment, education, health, religious power. Each of these was more available, generally, in or near the city than in the countryside, and each provided a form of physical and emotional security. Given this, it is not surprising that rural-to-urban migration is the most common

type of migration that has occurred during recorded history (Bogin 2001), and this type of human migration is occurring more rapidly today than ever before, especially in the least economically developed nations of the world (Henning 2017).

There are many excellent reviews of the human biology of rural-to-urban migration (e.g., Bogin 2001; Dufour & Piperata 2004; Eckert & Kohler 2014; Mascie-Taylor & Krzyżanowska 2017). It is clear that rural-to-urban migration has impacts on human growth, development, maturation, fertility, rates of aging, longevity, mortality, and emotional well-being. The focus of discussion in this book is on the nongenetic and nonnutritional determinants of adult body height in **migrant people**. These determinants align with the influence of SEPE factors, community effects, and strategic growth adjustments.

Migration, Social Networks, Competitive, and Strategic Growth

How does the growth status of migrant children, juveniles, and adolescents compare with nonmigrants? Of special interest is the comparison with sedentes, that is the people living in their geographic region of birth. How similar or different are migrants to their peer group of sedentes, meaning young people of similar age and developmental status of the migrant? How is the growth of the migrant affected by the fact or perception of being a dominant or a subordinate member within its new social network?

Migration usually is directed by "Push-Pull" factors (Ravenstein 1885). "Push-Pull" factors can be economic, political, cultural, and environmentally-based forces. These "Push-Pull" factors either induce people to move to a new location or oblige them to leave old residences. Migrants usually leave their original social network and enter new social networks, they may arrive alone, or in groups (Gratton et al. 2007). Migrants tend to identify with their home country and culture, often maintaining cross-border ties to their origin communities. They often keep up with traditional values, cultural, ethnic-religious, ethnic-juristic and ethnic-political concepts, as well as traditional lifestyle and eating habits. Migration is an arduous process and staying connected to the homeland is one coping strategy. Adding to the difficulties of the migration process is the fact that many migrants are marginalized and socially disadvantaged in their new communities. Integrating into the local social networks is often difficult and prejudice against the newcomers may lead to ethnic conflicts (Landis & Albert 2012). Nevertheless, most migrants tend to assimilate to some degree in the long run. Assimilation refers to the harmonization of different social groups (up to their fusion) with cultural and structural changes. Cultural assimilation is the process by which the migrant person's or group's culture comes to resemble that of the host. Structural assimilation is a process in which a social or ethnic group gains the same access to structures such as education/law/jobs, as the ruling social or ethnic group. In summary, migration usually exposes individuals or groups to different cultural conditions, in that the biological and socioeconomic environment differs from that of their place of origin. This transition can produce behavioral and psychological responses, as well as physical changes in the amount and tempo of

growth, development, and maturation resulting in new degrees of variation in attained adult height.

The Nature of Networks

Human societies can be viewed as networks that can be characterized by nodes (individuals or groups of people), and edges connecting the nodes. Migration changes the structure of human networks. The connectivity between people has been studied and not only shows social clustering on emotional traits, on tastes in movies, music, and books, but also on food consumption, obesity, smoking, likelihood of health screening, and other behavioral characteristics (Christakis & Fowler 2007, 2013). Human social networks are ancient as they are well described in forager populations and in nonhuman primates and other mammals (Apicella et al. 2012; Clutton-Brock 2016). In human societies these network associations are social in nature – they do not depend on genetic relatedness between people. The nongenetic nature of human kinship networks was described in Chapter 4, in the section "The nature of human biocultural reproduction." Their nongenetic basis notwithstanding, human social networks are of biological importance in that they produce effects that lead to impact on nutritional status, physical fitness, timing of fertility, morbidity, and mortality (Holt-Lunstad et al. 2010). More recent research finds that social networks influence hormonal activity, including oxytocin and serotonin (Dölen et al. 2013; Kanat et al. 2014) as well as cortisol and testosterone (Ketay et al. 2017; Kornienko et al. 2014, 2016; Ponzi et al. 2016). How social networks may influence growth hormone and insulin-like growth factor-1 is not well researched, but in view of recent work on serum IGF-1 and body size as expressions of sport and social dominance (Bogin et al.

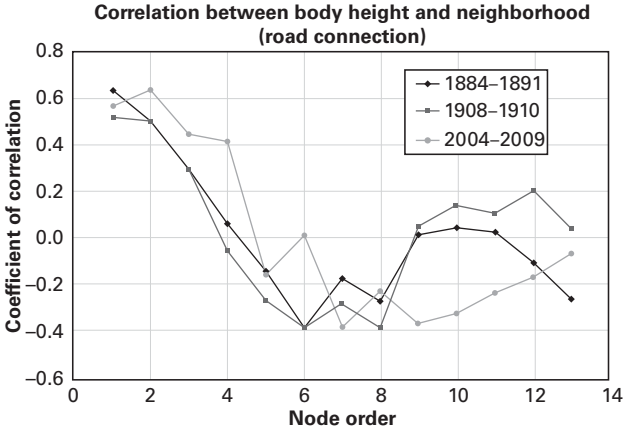


Figure 7.9 The correlation in height between neighboring districts. Significant height correlations ($p < 0.01$) exist between 1st, 2nd, and 3rd order neighbors in 1884–1891, in 1908–1910, and in 2004–2009 ($p < 0.01$); in the 2004–2009 cohort significant correlations in height were found up to 4th order neighbors. Figure adapted from Hermanussen et al. 2014a, with permission of the author

2015; Hermanussen & Scheffler 2016) there is evidence supporting the hypothesis that migrant children can adjust in height toward the average height of their recipient social network, or surpass the height of their host and donor populations in cases of violent, oppressive or military, and colonial migration.

Another source of evidence about the impact of social networks is statistical. One analysis was based on Monte Carlo simulations of body height in an artificial network to show that spatial connectedness can affect height clustering, similar to that seen in natural societies (Hermanussen et al. 2014a, 2016). These simulations were done using empirical data on Swiss conscript height and the results further strengthened the concept of connectedness, in this case measured as distance travelled by road between nodes, being involved in the regulation of human height. The researchers considered Switzerland as a geographic network (Figure 7.9) with 169 nodes (district capitals) and 335 edges (connecting roads) and studied effects of connectedness on height in Swiss conscripts from 1884–1891, 1908–1910, and 2004–2009. The significant associations were explained by road distance between districts. There were no significant correlations between mean height in a district and body mass index (nutrition), population density (rural/urban), or altitude above sea level (oxygen saturation). Height in a district depended on height of physically connected neighboring districts. The association decreased with greater road distance in the network. The researchers suggested that people can be short because their neighbors are short, or tall because their neighbors are tall due to community effects on growth mediated by emotional and neuroendocrinological growth regulators.

Testing the Impact of Social Networks

The following discussion highlights the growth patterns that parallel the cultural transition of migrant children and adolescents. Of particular interest are migrants who escape from poor or dangerous living conditions to find more prosperous general living and socio-economic conditions, usually with improvements in nutrition, health, housing, water, and sanitation (Ngure et al. 2014). These migrants may be classified as "social upgraders" when successfully integrating into the new host population. Adoptees and refugees from lower-income nations to the high-income nations belong to this group. In contrast, there are "social downgraders," such as war refugees who are forced into camps or slums. The living conditions of these social downgraders is often terrible and the growth and development of their offspring suffers (Lutfy et al. 2014), often due to gross malnutrition and rampant infections. Social downgraders suffer from social-emotional abuse that leads to mental health difficulties, including post-traumatic stress disorder, depression, self-harm, sleep disturbance and behavioral difficulties (Flood & Coyne 2019). The daily living conditions in camps and slums impose psychological stressors, such as lack of space and control, violence, feelings of inadequacy and hopelessness. Social downgraders in the context of migration are not further discussed here, but the harmful consequences of hopelessness on human physical growth are detailed later in this chapter.

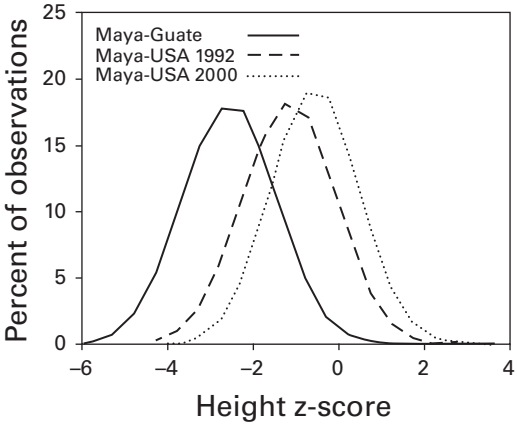


Figure 7.10 Height distribution in z-scores of Maya children and juveniles aged 5–12 years old raised in Guatemala (N = 1,897) in 1998, and in two US locations in 1992 (N = 245) and in 2000 (N = 444). The width of the three distributions is almost identical despite the much shorter height of the Guatemala-raised sample.

Another group of interest that is considered here consists of migrants who arrive in a new place as invaders, colonial rulers, and other types of political, economic, or psychological oppressors who dominate their host populations. These migrants may be classified as "conquerors."

To compare the effects of being a social upgrader or conqueror it is useful to test two hypotheses:

1. Cultural and structural assimilation by "social upgrading" migrants into their new host population is accompanied by adjustments in height growth of their growing offspring toward the median height of the hosts.
2. Migrants using violent, oppressive or military, and colonial conquest is accompanied by height growth of their offspring that significantly surpasses the median height of both the conquered population and the population of origin.

Readers are already familiar with the first case to test these hypotheses which is the Maya refugees from Guatemala to the United States. These Maya were described in the Introduction and earlier in this chapter. A community effect on height predicted by hypothesis 1 may be seen in Figure 7.10. The figure illustrates the marked improvements in average height of children who migrated to the United States. Mean height of children and adolescents aged 5–17 years (n = 1,897), raised in Guatemala, measured in 1998, was −2.54 z-scores below WHO standard/reference (WHO Multicentre Growth Reference Study Group 2006). Mean height of children and adolescents aged 5–12.99 years (n = 245), raised in Florida and in Los Angeles, California, measured in 1992 was –1.15 z-scores. And mean height of children and adolescents aged 5–12.99 years (n = 444), raised in Florida and Los Angeles, measured in 2000 was only −0.53 z-scores below WHO standard/reference. The figure not only shows that migration affects the mean values of height, but also that migration

fails to affect the width of the height distribution. The US-born group increased in height by more than 10 cm within a single generation, but the height difference between the tallest and the shortest within the group remained unchanged. Better nutrition, WASH, and medical care certainly account for some of the height increase of US-born Maya. But, if these were the only explanations, then we would expect that the lower tail of the distribution, those most suffering from malnutrition, etc., would show the greatest increase in height. This is not the case. Height distributions remain virtually scale invariant, they simply shift to the right, as a whole.

Despite the civil war and forced migration out of Guatemala, most adult Maya migrants to the United States aspired to maintain cultural identity rooted in their formative experiences in rural Guatemala, such as Maya language. Maya refugee children, on the other hand, were born or raised in the United States and most learned both English and Spanish simultaneously, fewer of the refugee children learned their Maya language. While Maya values were still strongly emphasized at home, children acquired non-Maya cultural values and behaviors on the streets and in the schools. The increase in height of these socially upgrading immigrants moved them closer to the height distribution of their hosts, that is, European-American and Africa-Americans. But the first-generation of Maya-Americans were still part of a migrant community with its own SEPE ecology and still tended to cluster in height.

Future generations of Maya-Americans are predicted to grow even closer to their host's height distribution. A secular tend in this direction has been observed for the offspring of Mexican immigrants to the United States who have become taller, on average, with each generation since the 1930s (Bogin 1989). The most recent analysis of US-born Mexican-American boys, aged 2–14 years old, reports that they average 1.7 cm less in height compared with European-American and Africa-Americans of the same age (Delajara & Rodríguez-Segura 2010). Two generations previously the difference was closer to 7 cm. The proximate reason for the increase is greater maternal height, which accounts for 38% of the gain in height of the boys. The ultimate reason for greater height in mothers and their offspring may be the secular trend toward greater SEPE integration into the host society, a reorientation of growth toward the mean height of the host community, and the strategic growth adjustments that follow from increasing social status, both real and perceived.

Very similar changes in height distributions to the Maya-Americans were published by Houghton and colleagues (2014) on Bangladeshis in Bangladesh and London. The study found catch-up growth in height and an acceleration of developmental tempo, measured as age at adrenarche, of first-generation migrants, a short final height of first-, but not of second-generation migrants, and the propensity for overweight. But even though height increased from −1.2 z-scores in Sylhet, Bangladesh to −0.8 z-scores in the first, and to −0.2 z-scores in the second generation, the standard deviation for height remained almost invariant with 1.3 SD in the Sylheti, 1 SD in the first-, and 1.3 SD in the second-generation indicating that also these girls tend to cluster in height.

A final example is the famous study by Harry L. Shapiro (1902–1990) on Japanese sedentes and migrants (Shapiro 1939). In this work Shapiro compared the growth of

Hawaiian-born Japanese of Japanese immigrant parentage, Japan-born Japanese who migrated to Hawaii, and Japanese sedentes living in the same villages from which the migrants originated. The sedentes were mostly farmers and laborers in rural villages. The first-generation immigrants did similar work as laborers on sugar plantations, but some were employed in sedentary occupations (e.g., store clerks). In contrast, 76% of the Hawaiian-born were either students or sedentary workers, including office workers. The immigrants appear to have been developing an urban lifestyle.

Shapiro reported that the Japanese sedentes and the recent Japanese immigrants differed in a few anthropometric measurements, some increasing and some decreasing with migration. The largest differences were between the immigrants and the Hawaiian-born. Most of the immigrants came to Hawaii as adults, so had grown up under biocultural conditions in Japan. This makes my study of Maya in the United States similar to Shapiro's Japanese study – both studies include measurements of offspring who were born and grew up in the new biocultural environment. A student of Shapiro's, Fredrick Hulse (1906–1990) who became a prominent anthropologist in his own right, assisted with the data collection. Hulse located the villages of origin in Japan of the Hawaiian immigrants and measured the close relatives (siblings, cousins, etc.) of the immigrants. Hulse described his efforts to locate family in the origin villages and the findings of the study by writing that (Hulse 1981, p. 497),

> Thus it was possible to compare the Japanese in Hawaii to their closest genetic relatives rather than to Japanese in general, and to find out just who became a migrant and who remained a sedent. Among males, younger sons of landholding families were most likely to leave, and they tended to be just a little bit taller than their stay-at-home brothers. Their Hawaiian-born sons were, on average, distinctly taller than the migrants, and obviously more brachycephalic [round-headed] as well. They also had narrower noses and faces. Female differences were similar but less pronounced. It is of interest to note that a generation later Froelich (1970) found in Hawaii that females of Japanese ancestry grew to a larger size than they had during the first generation. He suspected that the traditional Japanese custom of feeding boys better than girls might at last be giving way to a less sexist division of food resources.

Shapiro argued that with migration there were improvements in diet, health care, and socioeconomic status and that these conditions, associated with an American-esque, urban lifestyle, were responsible for the growth changes. Hulse did not appear to accept Shapiro's conclusion as his 1981 review of the study continues:

> It flatters us to suppose that this is due to better living conditions . . . but it would be naive to accept this explanation uncritically. Better in what way? Was life healthier on Hawaiian sugar plantations than in Japanese villages? Was the food more plentiful or nutritious, the climate more salubrious? Was the work less onerous? These questions have not been answered, with any scientific rigor.

There is evidence that changes in body size and shape of Hawaii-born could be due to community effects and strategic growth adjustments within social networks. During the time of Shapiro's study, Hawaii was an administered territory of the

Table 7.2 The population of Hawaii by ethnicity, 1900–1940. The European ethnicity is comprised mostly of "whites" from the United States. "NA" indicates that data are not available. Data from Schmitt (1968) and www.ohadatabook.com/T01-03-11u.pdf.

Ethnicity	1900	1910	1920	1930	1940
Japanese	61,111	79,675	109,274	139,631	157,905
Native Hawaiian	37,656	38,547	41,750	50,860	64,310
European	28,819	44,048	54,742	80,373	112,087
Chinese	25,767	21,674	23,507	27,179	28,774
Other Groups	415	376	310	217	579
African-American	233	695	348	563	255
Filipino	(NA)	2,361	21,031	63,052	52,569
Korean	(NA)	4,533	4,950	6,461	6,851
Total	154,001	191,909	255,912	368,336	423,330

United States. People with Japanese or Japanese-American ethnicity were the largest group, making up about 38% of the total population of Hawaii in the 1930 decade (Table 7.2). Native Hawaiians and Europeans, predominately "whites" from the US mainland, were the next two largest ethnic groups. Many of the "whites" were serving in the military and in the years prior to the US entry into World War II there was an impressive military buildup in Hawaii and a US military "culture." Despite being third in population size, the United States "whites" were the dominant economic-political group and imposed "American" cultural norms of behavior on the urban areas of the main islands, including a US-style racism against "nonwhite" ethnic groups.[20] The Japanese in Hawaii suffered under the "white" American domination but may still be considered as "social upgrading" in that the Hawaii-born Japanese were becoming better educated, in US-style schools. The biocultural target for young Japanese born in Hawaii was to integrate into the social network of the "white" US and their growth in height moved toward that target network.

Studies of American-born Japanese living in California by Greulich (1976) found that from childhood to adulthood both boys and girls were taller than same-aged people in Japan. They grew away from the old Japanese target and toward the new US target. In a review of many studies of growth following migration Roche (1979) reported that the growth in height of each generation of the children and grandchildren of migrants continued to increase until it converged on that of the host population. Each generation integrated to a greater extent into the hosts' social networks. In sum, these findings lend much support to accepting the first hypothesis of "social upgrading" and height.

[20] www.nytimes.com/2005/04/18/arts/television/racism-mayhem-and-madness-in-paradise.html; www.pbs.org/wgbh/americanexperience/features/island-murder-1930s-honolulu/

Adoptees

A relatively new type of migrant are adoptees from low income countries, adopted by families in high income countries. Most of these international adoptees (IA) have delays in physical growth, motor development, and cognitive skills, but most show rapid catch-up of the delays. Some part of the catch-up is certainly due to better nutrition and health care. Another part of the catch-up, equal to or greater than the material conditions of life, may be associated with the new social networks of improved emotional care and the host society's target for average height and psychological competences.

Many, if not most, of the IAs were institutionalized or placed in foster care prior to adoption. They may have been stunted, wasted, and emotionally scarred by their experience with maltreating families or neglecting orphanages (van Ijzendoorn & Juffer 2006). Severe psychosocial deprivation prior to adoption, such as the infamous case of Romanian orphans during the regime of dictator Nicolae Ceauşescu between 1965 to 1989, invariably has negative physical, behavioral, and cognitive impacts on arrival and through childhood. Edmund Sonuga-Barke and colleagues have shown that these impacts last to adulthood and include derangements of the hypothalamic-pituitary-adrenal (HPA) axis and stress hormone activity (Kumsta et al. 2017). The association of physical growth with the HPA axis and emotional stress were mentioned briefly in Chapters 5 and 6. Later in this chapter the negative effects of HPA derangements on growth in height are described in greater detail.

Another case of long-lasting negative effects are the more than 6,800 infants and children from India who were adopted by Swedish families from 1970 to 2009 (Proos 2009). Upon arrival, most of them were very short, severely undernourished and suffered from infectious illnesses. Many showed a catch-up in height growth. As discussed in previous chapters, catch-up growth is a physiological condition of temporary over-growth, and is indicative of previous growth impairment. It is defined as a height velocity above the statistical limits of normality for age and/or maturity during a defined period of time, following a transient period of growth inhibition (Wit & Boersma 2002). Catch-up growth occurs after illness, starvation, and psychosocial deprivation, and takes the child back onto their original pre-insult centile or z-score position. Catch-up growth in adoptees is usually accompanied by long lasting developmental acceleration. Particularly, adopted girls tend to prematurely start puberty. Proos (2009) studied 107 Indian girls, adopted in Sweden, over a period of 2 years. The girls' menarche (first menstruation) was at 11.6 (range of 7.3–14.6) years which was earlier than Swedish (13 years) and earlier than wealthy Indian girls in India (12.4–12.9 years). Also, the adolescent growth spurt of the adoptees was, on average, 1.5 years earlier. The premature onset of pubertal development increased the pace of bone maturation, and in many cases, led to clinically significant reductions in adult height. Final height in these girls was 154 cm (–1.4 z-score), final weight 46.9 kg (–1.1 z-score). Eight percent of the girls remained 145 cm or shorter. This acceleration in maturation is difficult to explain. Those who were most stunted at arrival, and had the fastest catch-up growth, had the earliest

menarche. This suggests that there was a fundamental derangement of the girls' developmental physiology that in the new Swedish bio-social network resulted in an excessive growth adjustment toward the target height of the Swedish hosts.

Adoptees who were treated better than the Romanian orphans and Indian adoptees in Sweden migrate into new families and social networks that, in general, provide a high standard of nutritional and medical care, attachment security, positive emotional and cognitive stimulation, and love. A small and important focus of research has been the assessment of physical growth, development, school achievement, self-esteem, and behavior outcomes in the IAs (Jacobs et al. 2010; Miller & Hendrie 2000; Rutter et al. 2012; Woodhouse et al. 2018). The most recent **meta-analysis** (a type of systematic review of research) reported that the more time spent in institutional care prior to adoption the more the infants and children lagged in physical growth (Van Ijzendoorn et al. 2007). The statistical importance of institutionalization was measured by its **effect size** using a statistic called "Cohen's d." A large d = 0.60 and a medium d = 0.40. The value of institutionalization on physical growth was d = 1.71, a very large effect (95% CI: 0.82–2.60, n = 893). At the time of adoption, the infants and children had large delays in height, weight, and head circumference (d = −2.39 to −2.60; n = 1,331–3,753). Follow-up measurements after adoption showed " . . . almost complete catch-up of height (d = −0.57, 95% CI: −0.87 to −0.27, n = 3,437 adoptees) and weight (d = −0.72, 95% CI: −1.04 to −0.39, n = 3,259 adoptees), catch-up of head circumference seemed slower and remained incomplete (d = −1.56, 95% CI: −2.27 to −0.85, n = 527). Later age at arrival was related to less complete catch-up of height and weight" (p. 334). The authors also evaluated social, emotional, and cognitive domains and conclude that their meta-analysis provides " . . . evidence for massive catch-up and plasticity in physical, socio-emotional, and cognitive development" (van Ijzendoorn & Juffer 2006, p. 1228).

In the view of their review of the "massive catch-up" the authors state that adoption is one of the most effective interventions to overcome previous growth stunting and developmental delays. Adoption is certainly more effective than any of the nutrition supplementation, WASH, or education interventions tried in the countries of origin of the adoptees. International adoption, just like the migration of Maya from Guatemala to the United States, places the migrants in new social networks and leads, in general, to positive community effects and strategic growth adjustments on height, cognition, and emotional well-being.

Conquerors

Military conquests are a form of migration. Conquerors form the dominant social strata, and they usually take care to ensure that their offspring maintain social dominance. They do not integrate, but rather impose their social networks on their subjects. Colonial Europeans of the nineteenth and early twentieth centuries were not only taller than their native subjects, but also taller than their continental relatives in Europe. White US Americans of the early-to-mid nineteenth century surpassed white Europeans by several centimeters (Komlos & Cuff 1998). By 1960, American white

Table 7.3 Mean height (cm) and standard deviation (SD) of Dutch children raised in Brastagi, Indonesia boarding school (1926–1928), compared with the mean height of boys raised in Amsterdam, Netherlands (1916), in German cities, and boys from upper SES families of Frankfurt, Germany in 1922.

Boys					
Age	Brastagi	SD	Amsterdam	German cities	Frankfurt, upper SES
6	120.9	5.5	109.1	121	
7	126.2	4.6	112.2	115.6	124.5
8	131.5	5.4	117.4	120.7	129
9	136.5	5	123.5	125.5	135.5
10	141.3	5.7	127.7	130.3	138
11	147.2	5.7	133.5	134.7	142.5
12	153.3	7.8	139.2	138.8	148
Girls					
6	116.9	3.6	108.9		
7	123.7	3.7	112.5	114.7	
8	129.4	4.8	118.4	119.8	
9	136.2	6.6	123.4	124.7	
10	143.4	7.8	128.5	129.6	
11	148.7	6.6	132.9	134.6	
12	155.8	6.9	137.8	139.7	

men averaged 174.1 cm, while adult men in England averaged 165.6 cm and in Sweden and Norway averaged 168.6 cm (Komlos & Baur 2004). Greater height for colonists was also true for Dutch white settlers of nineteenth-century South Africa (Henneberg 2001) and Australia (Boyd 1980).

Tall stature also prevailed in the children of colonialists during the early twentieth century. De Haas (1931) and Gorter and de Haas (1947) measured Dutch children raised in a boarding school at Brastagi, Indonesia (1926–1928). They were similar in height compared with "tall" US American children measured in 1924 and up to 15 cm taller than German children measured in 1922 or children in Amsterdam measured in 1916 (Table 7.3). The Dutch colonial children were, of course, very much taller than indigenous Indonesian children. It is unlikely that the greater stature of the European colonists and their children was due to better nutrition and health compared with their counterparts in Europe. The wealthy of Europe could acquire good diets. The burden of tropical infections from microorganisms and parasites that are considered responsible for poor growth in many indigenous populations likely also affected the offspring of the new colonists, but these infections do not exist in Europe.

Instead, it is proposed that these examples are evidence in support of hypothesis 2. The greater height of the colonial Europeans was in large part due to their self-perception as the dominant social class with a consequent competitive growth and strategic growth adjustment toward greater average height. As detailed in

Chapter 5, the height advantage of the South African colonists over their Dutch sedente counterparts was lost as the Apartheid system crippled the SEPE ecology of South Africa. Feelings of superiority cannot overcome the reality of inequality and injustice.

What Makes Migrants Grow?

The likelihood of selective migration, "are migrants taller than sedentes," has been discussed in the literature, and case studies of both selective and random migration are reported (Krzyżanowska & Mascie-Taylor 2011). The push factors, such as war and poverty, usually affect people without regard to their height. An analysis of rural-to-urban migration in Poland reported some selection for greater height when the rural migrants were of very low SES, but no selection when SES was higher (Zielińska 1991). Reviews of rural-to-urban migration (Bogin 2001; Dufour & Piperata 2004) found some selection for higher SES that may associate with taller height, but overall, there is not much evidence that migrants are selected for physical traits.

The intuitive link between food and growth has been prevalent in the literature for more than a century and resulted in the longstanding tradition of claiming changes in nutrition responsible for the bodily changes observed in migrants. As detailed earlier in this chapter there is little evidence that nutrition interventions increase height and there is less evidence that nutrition explains height increases in most migrants. Relevant here is another older and sometimes forgotten study of nutrition and growth by (Garrow & Pike 1967). They also questioned the primary role of nutrition as the cause of growth failure. The authors examined 65 Jamaican children 2–8 years after they had been treated for severe malnutrition in a hospital. The former patients were found to be small by North American standards, but not when they were compared with Jamaican children of similar genetical and economic background. A major strength of this study is that Garrow and Pike compared the former patients with their own siblings raised in the same household. This is a good control for genetic and environmental variation. The authors failed to find convincing evidence that a period of severe malnutrition in infancy per se causes stunting of growth in children. In fact, they found some evidence for just the opposite, that is, the children who had been hospitalized tended to be taller than their siblings (using sex-age z-scores). The former patients also tended to be faster growing than their siblings and this may be why they were susceptible to severe malnutrition. Garrow and Pike wrote that " . . . the children who were admitted to hospital were genetically bigger than their siblings. We would suggest that the child whose genetic make-up is such that he would grow very rapidly if well fed, will suffer more on a restricted diet than one with more modest demands. This hypothesis could explain both the fact that in a given family on a restricted diet some children suffer much less harm than others, and also the tendency of the child who has been successfully treated for malnutrition to outgrow his siblings very slightly" (p. 4). Garrow and Pike's hypothesis has not been tested and likely cannot be tested as there is no genetic test for tallness or more

rapid growth. The reasons for the more rapid growth of the former patients were not known, but it was certainly not due to greater food intake. In light of the studies of competitive growth of meerkat siblings discussed earlier, it is possible to hypothesize that the effects of social-emotional competition between the Jamaican siblings led to more rapid growth in some of the children. This was disadvantageous in low-income families that could not provide the food needed to support that growth. The result was that the taller, faster-growing sibling suffered severe malnutrition.

Height is a Social Signal for Migrants and for All People

Even if statistical, biological, and practical effects are relatively small in many studies; genetics, nutrition, health, and WASH are important determinants of growth and adult body height of migrants and all human beings. These factors interact with wealth, socioeconomic circumstances as well as with social strata, education and professional career, and last but not least, with choice of partners and the genetic make-up of the next generation. This is the conventional interpretation of "what makes children grow". The conventional interpretation provides the basis for nutrition, WASH, and education interventions, but these have had limited success or outright failure to improve human growth and development.

It is obvious that adequate nutrition, health, and living conditions are required for healthy growth and development, but in contrast to the conventional view that these factors are the only regulators of growth, newer research finds that these factors are only prerequisites for successful growth regulation. Understanding stature as a social signal moves the focus of growth regulation from the prerequisites to social-economic-political-emotional (SEPE) mechanisms. More simply stated, growth is a process that allows for adjusting individual height toward its relative optimum within the group. This social, community-based growth adjustment takes place within a wide genetic frame. Historic evidence suggests that populations may vary in average height by up to 20 cm within short historic periods of a few generations such as the Dutch secular trend reviewed previously. Strong support for growth adjustment within the group is given by the observation that even during periods of marked changes in the mean value for height, the distribution of height remains scale invariant. This was shown in Figure 6.5 for cohorts of Swiss conscripts born between 1872 and 1989 and Figure 7.10 for Maya born in Guatemala and the United States. This is amazing. In a changing world with changing living conditions, it is expected that conditions do not change equally for everybody. In addition, responsiveness to the living conditions is expected to be unequal among different individuals due to everything from genomic/epigenomic traits to socioeconomic/psychological factors. Those who respond well to improvements should grow better than those who do not. This is not the case.

The idea of some additional social regulation in height that narrows height variation toward the mean, appears straightforward. Social interactions and group behavior have long been studied. Turner and Oakes (1986) discussed social self-concept in their social identity theory and showed that the system of cognitive

representations of self is based upon comparisons with other people and relevant to social interaction. Group membership correlates with emotional, evaluative, and other psychological parameters. Turner and Oakes employed the concept of a "self-categorization theory" to explain the social-psychological basis of group phenomena, and to identify the mechanisms by which individuals become unified into a psychological group. An important chapter by Tajfel and Turner on social identity theory, originally published in 1986, was reprinted in the book *Political Psychology* (Tajfel & Turner 2004). The authors' goal was to develop a theory of intergroup conflict and it is noteworthy that the focus of this theory has shifted over time from the social psychology of individuals to a new field of political psychology of groups. The focus on SEPE factors in the present discussion builds on Tajfel and Turner's pathbreaking ideas. Tajfel and Turner emphasized "group identification" and "identity signaling" as mechanisms to facilitate in-group favoritism, and shape common goals and social norms. Conflicts of group interests not only create antagonistic intergroup relations, but also heighten identification with, and positive attachment to, the in-group. In relevant intergroup situations, individuals will not interact as individuals, on the basis of their individual characteristics or interpersonal relationships, but as members of their groups. Tajfel and Turner brought our attention to the process whereby people derive a sense of self-worth and social belongingness from their memberships in groups. People will then see their group and themselves favorably and see other groups as superior, inferior, or competitive to their own group depending on the SEPE resources and social position that each group controls.

These observations are grounded today in Social Network Theory, which was reviewed earlier. The connectivity between people has been shown to produce clustering of social-behavioral properties such as happiness, loneliness, depression, sleep, drug use, divorce, sexuality and sexual orientation, political orientation, and tastes in movies, music, and books. These types of networks are not surprising, but less expected are findings of networks of social contagion in the "spread" of food consumption, obesity, smoking, alcohol consumption, likelihood of health screening, cooperative behavior, influenza (Christakis & Fowler 2013). All of which can have biological importance in that they produce phenotypic variation, including height variation. Observational studies and statistical modeling support the view that an individual's position within the group is a regulator and potential stimulus for growth. Aßmann and Hermanussen (2013) used a Bayesian statistical approach to model data from a longitudinal study of school children and adolescents from Zurich, Switzerland, and found evidence for a growth regulation that operates during adolescence and adjusts individual height toward the average height of their peers, i.e., their immediate community. They reported that the smaller the adolescent was compared with past mean average height (of the community), the more the adolescent grew during puberty. Conversely, taller than average adolescents grew less. The net outcome was that the distribution of heights of members of the community, which was the definition of a social network in this analysis, will be narrow and will cluster toward the mean value. The first use of the phrase "community effect on height" appeared in this article. Aßmann and Hermanussen's view was that people

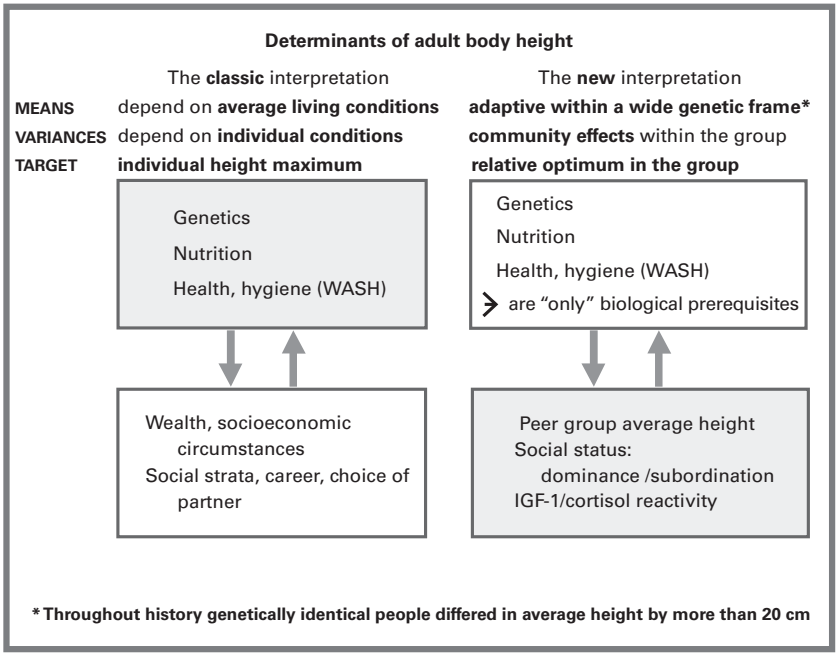


Figure 7.11 The determinants of adult body height, comparing the conventional interpretation considering genetics nutrition, and health vs. the new interpretation, considering community effects and strategic growth adjustments within the group.

may simply be short because their friends and neighbors are short, or tall because their friends and neighbors are tall. This view may be a bit overstated, but it captures the essence of the community effect in the sense that all the SEPE factors of a community are regulating height toward a group mean.

An analysis of Polish school children further supported this idea. Koziel and Gomula (2017) sampled 1,810 juveniles and adolescents aged 14 years and showed that the variance in height and BMI was smaller within groups of students of the same classroom and larger between classrooms. There are many possible reasons for this "shared classroom" effect, but the important focus is the common social network. Hermanussen and Scheffler (2016) further elaborated on these findings by providing historic evidence that stature may serve as a social signal. From this literature developed the hypothesis that social upgrading migrant children adjust their height growth toward the mean of the recipient social network. They start perceiving themselves as members among peers within their new social networks and they grow toward the average height of their hosts via the processes of competitive growth and strategic growth adjustments. Also derived from the literature was the second hypothesis that conqueror migrants grow to a greater height than the native population of the host population and a greater height than their genetic relatives in their country of origin.

The new interpretation of human growth regulation considering community effects and strategic growth adjustments within the group, is contrasted with the older, conventional interpretation in Figure 7.11. The lower right-hand box in the figure suggests some mechanisms for this community effect. It is important to people to be the right height, which is the average height of the peer group. This is because size signals status. Being short signals subordination, tallness signals dominance. Infants, children, juveniles, and adolescents learn about their social position in society as they grow, develop, and mature. Wanting to be of higher status may sometimes be possible, but just as often that desire is met with hostility and derision from members of the family and the peer group. A great deal of art, prose, and poetry exists about people of "knowing their place" in society. Ancient and modern art, and popular television shows and films, almost always depict people of higher social status as taller than the people of lower status. Stop reading this book and watch a film about social class or political power. The very popular *Downton Abbey* television series is one place to start.

The physiological pathways for a direct stimulatory effect of dominance and subordination on growth remain unclear. The existing data on IGF-1 in social hierarchies of male primates (Sapolsky 2005; Sapolsky & Spencer 1997) and on the effect of sport success in human women and men (Bogin et al. 2015) suggest involvement of the IGF-1-growth hormone axis to play a role in this regulation. In wild baboons social subordinance was associated with suppressed IGF-1 concentrations, but not concentrations of IGF-2 or IGF-binding protein. After eliminating other possible reasons, such as age, hypercortisol levels, testosterone, and food, Sapolsky and Spencer concluded that the social status differences in IGF-1 profiles were a consequence, rather than a cause, of the rank difference. In related research, Sapolsky documents a host of pernicious health consequences of social subordination in several primate species including humans. The severity of the health deficits arises from psychosocial factors and follows the social status gradient. Qualitative studies report that feeling poor predicts poor health. Quantitative economic research finds that greater income inequality predicts poorer health. The feelings and the reality of poverty work synergistically to disrupt social networks and create decreased levels of trust and increased senses of alienation and disenfranchisement leading to a downward emotional spiral, depression, and hopelessness with negative consequences on health and height growth.

Sex, Sport, and the Community Effect in Height

With several colleagues, I tested the hypothesis that differences in social status between groups of people within a population induce variation in insulin-like growth factor-1(IGF-1) levels and, by extension, growth in height (Bogin et al. 2015). The relationship between IGF-1, assessed via finger-prick dried blood spot, and elite level sport competition outcomes were analyzed for a sample of 116 undergraduate men and women. Blood spots were taken 24 hours before a competition and 24 hours after the competition. There were no meaningful differences between the sexes or between

Table 7.4 Mean (SD) IGF-1 levels (μg/L) 24 hours before and 24 hours after the game by win or lose.

	Win (n = 55)	Lose (n = 46)	
Pre	47.8 (14.8)	43.1 (14.9)	
Post	46.8 (16.5)	42.8 (14.3)	
Mean IGF-1	46.2 (13.8)	41.6 (12.4)	p = 0.04

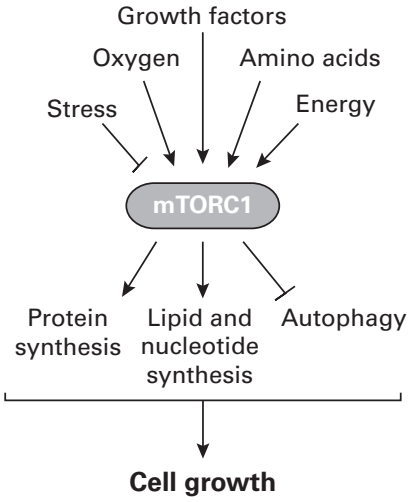


Figure 7.12 Basic elements of the mTORC1 signaling pathway. Hypertrophy and hyperplasia of cells requires the production of the cellular "building-blocks" (anabolic constituents) of proteins, lipids, and nucleotides while also suppressing catabolic pathways such as autophagy (destruction of cells). mTORC1 plays a central role in regulating all of these processes and therefore controls the balance between anabolism and catabolism in response to environmental conditions such as the supply of essential nutrients, oxygen, IGF-1, and other growth factors. Various forms of stress, including emotional stress that increases cortisol and other stress hormones, blocks the action of mTORC1. Adapted from Saxton & Sabatini (2017)

the sport practiced (lacrosse, handball, rugby, football (soccer), netball, and volleyball). There was a statistically significant difference between winners and losers of a competition. Winners, as a group, had higher average pre-game and post-game IGF-1 levels than losers (Table 7.4). Both winners and losers decreased in IGF-1 levels from the pre- to post-competition time points. Members of the same team were not more similar in IGF-1 levels than they were to players from other teams. We proposed the pre-game differences in IGF-1 may serve as a proxy for feelings of social dominance, that is, the winning team had an overall feeing of dominance and this raised IGF-1 levels. Our findings provide some support toward the community effect in height hypothesis.

We discussed our findings in relation to the action of the growth hormone/IGF-1 axis as a transducer of multiple bio-social influences into a coherent signal which

allows the growing human to adjust and adapt to local ecological conditions. IGF-1, as one of the most potent regulators of human growth, closely interacts with the mechanistic Target of Rapamycin Complex 1 (mTORC1) signaling pathway (Figure 7.12). Research on this metabolic pathway, mentioned briefly in Chapter 6, was reviewed by Saxton and Sabatini (2017). They reported that mTORC1 is a master growth regulation pathway that is sensitive to the requirements for cell growth, both hypertrophy and hyperplasia. IGF-1 and other growth promoting factors stimulate mTORC1 activity. The result is greater size of body cells, tissues, organs, and height. Various forms of stress, including nutrient and oxygen deficits as well as cortisol and other stress hormones, block the action of mTORC1 and inhibit cell enlargement and division. Moreover, active mTORC1 blocks cell autophagy, the self-destruction of body cells. Under stress mTORC1 is inactive and there is an increase in catabolism, including autophagy.

The extensive interacting matrix of variables that mTORC1 regulates throws additional light onto the mechanisms that allow for growth adjustments and competitive growth. In social upgrading of migrant infants, children, and youth the complex interplay of changes in nutrition and energy expenditure patterns, along with the escape from the stresses of their homeland, the improvements in social-economic-political-emotional (SEPE) conditions, and incorporation into new social networks, all stimulate height catch-up toward their new hosts. In contrast, the offspring of conquered peoples suffer the violent and oppressive regimes of their military and colonial masters and it is obvious that these people will grow less. The conquerors, in opposition, create new social networks of SEPE dominance and diverge from both the conquered and sedentes in their homeland toward greater mean height.

You Can't Be Too Rich or Too Tall

The heading for this section is a paraphrase of Wallis Simpson, Duchess of Windsor (1896–1986) who was quoted as stating, "You can never be too rich or too thin." It is bad to be "too thin", especially due to hunger, or "too tall," especially when due to growth hormone over-production. Those pathologies notwithstanding, the rich do tend to be thinner (less fat) and taller than the poor. Imagine richer and poorer families in any part of the world. The rich have a greater array and depth of SEPE resources. The rich have effective methods to help assure that they will keep and grow these resources, for example financial instruments, such as stocks, bonds, and property, that require very high monetary starting points for investment. The rich and politically dominant class of society also benefit from tax incentives that lower taxes paid to a point below that of the middle and lower classes. The rich and dominant classes have social exclusion strategies, such as schools, clubs, and professional societies, that restrict membership to a narrow social network. The poor have less of these and must live with the insecurity that they can lose what little they have. The richer know that they are better-off, they believe that they deserve their wealth

and status, and they "view the lower classes as subhuman."[21] The rich know that they are dominant in terms of SEPE resources and that feeling is enhanced when the rich are taller than the poor and even the middle-class, because stature signals status (Hermanussen & Scheffler 2016). While rich individuals are not always the tallest people in a society, the historical and contemporary data of representative population samples, discussed in Chapters 1 and 5, and earlier in this chapter, show a clear gradient of increasing height from poorer to richer.

How the Rich Stay Rich and Tall

The rich form social networks to preserve their elevated SEPE status. One way these networks form is through access to education. Private schooling that demands a high fee is one way to exclude access to the social network. School segregation by income may begin in infancy with nurseries, creches, and kindergartens. This economic and social segregation intensifies in primary and secondary schools and reaches its maximum in colleges and universities. This can be shown by using family surnames as markers of social status. In many societies and nations, surnames are inherited as a paternally-linked trait – children inherit their father's surname. Sometimes surnames are maternally linked as well – in many Spanish-speaking countries both the father's and mother's surname are used for official records and identification. Surname inheritance is similar in many ways to genetic inheritance (Lasker 1985). In a study that examined associations between surnames and the genetic markers of Y chromosomes, haplotypes, and haplogroups, the authors reported that, "Our analysis of 40 British surnames demonstrates a remarkably strong relationship between these patrilinearly inherited cultural markers and Y-chromosomal haplotypes" (King & Jobling 2009, p.1100).

A study by economists Gregory Clark and Neil Cummins (2014) used surnames to track educational status and social mobility in England from the years AD 1170 to AD 2012. The authors noted that past studies of the association of surnames and social status reported parent–child correlations that range between r = 0.2–0.6. These correlations are for the general population, but not representative of the elites and are likely misleading about rates of social mobility into and within the elite class. Clark and Cummins " . . . tracked families that sent their children to study at Oxford and Cambridge – the two most prestigious and elitist Universities in the world since 1096 . . . [and] the researchers found that students were more likely to inherit their parent's social status than their height" (Puiu 2014). The same elite-class surnames were highly likely to appear generation after generation in the student registers of Oxford and Cambridge. Clark and Cummins reported correlation coefficients of r = 0.7–0.9 for intergeneration surname continuity at the elite universities. The correlation coefficient for height between generations is less than r = 0.7 for parents and offspring of upper-middle class social status in the rich nations (shown in

[21] www.theguardian.com/society/2014/sep/15/how-super-rich-got-richer-10-shocking-facts-inequality

Chapter 6). Clark and Cummins concluded that the social status " . . . correlation is unchanged over centuries. Social mobility in England in 2012 was little greater than in preindustrial times. Thus there are indications of an underlying social physics surprisingly immune to government intervention" (p. 517).

How the Tall Stay Dominant

Social, economic, and political dominance of the wealthy elites is maintained by cultural inheritance, which was defined in Chapter 6. Cultural inheritance occurs in all human beings. It is the process by which parental phenotypes interact with offspring genomes to shape offspring phenotypes. Wealthy elites maintain a psychological and behavioral phenotype of prestige (Cattell 1942, Box I.1) which gives rise to an emotional attitude of entitlement. The psychological-emotional phenotype of the parents with high prestige is culturally transmitted to their offspring and interacts with offspring physiology to reduce negative stress and its growth inhibiting hormones and stimulate production of growth-promoting hormones. The parental status to offspring growth pathway is not a hypothesis, it is a fact and has been empirically documented in several nonhuman species such as baboons (Sapolsky & Spencer 1997), mandrills (Setchell 2016), orangutans (Emery Thompson et al. 2012; Maggioncalda et al. 2000, 2002), mole rats (Young & Bennett 2010), meerkats (Dubuc & Clutton-Brock 2019; Huchard et al. 2016; Russell et al. 2004), and other species (Clutton-Brock 2016). The human difference is culture, especially the ideological justifications for the dominance of wealthy elites that maintain their social position and their taller height. These ideological justifications are today enshrined in the hereditary aristocracies, constitutional monarchies, parliamentary monarchies, and other forms of anti-egalitarian status differentials practiced in many of the wealthier and European and Asian nations. The elites know that they are superior and are treated as such by the nonelites. The physical and emotional stimulation through these social networks maintain the gradients in height between elites and nonelites (Floud et al. 2011).

An extensive research literature documents height bias in education, earnings, marriage, and many other socioeconomic realms that predict social dominance (Blaker et al. 2013; Ellis 1994; Hensley 1993; Stulp et al. 2015, 2017). The way that social dominance is played out in society is usually in terms of both overt and subtle preferences for people who have characteristics of the desired type, that is the stereotype that a society defines as attractive, healthy, intelligent, and successful. In the WEIRD (Western, Educated, Industrialized, Rich, Democratic) nations these stereotypical preferences are often measured by height. Based on results from a questionnaire survey, Keyes (1979) found that men under 175 cm in stature, just under the average height for men in the United States, " . . . invariably wished they were taller." Keyes reviewed other research and found that job status and economic rewards could account for this desire. In 2 studies, conducted 3 years apart, of graduates from the University of Pittsburgh, there was a 12.4% difference in initial starting salary favoring men 188 cm tall vs. men 180 cm tall. The salary difference

favoring cum laude graduates was only 4%. Additional research on 5,000 Army recruits measured in 1943 found that in 1968, those over 183 cm earned 8% more than those below 168 cm, even after the influence of IQ, educational level, marital status, and occupation were statistically controlled. In another study, 140 personnel officers of companies involved in retail sales were asked which of two equally qualified job applicants would they choose – one who is 185 cm tall or one who is 165 cm. The taller applicant was preferred by 72% of the personnel officers, 27% had no preference, and 1 respondent chose the shorter applicant.

Since Keyes' 1979 study and review little has changed. A report in *The Economist* (December 23, 1995, pp. 19–22) cited a survey of American Fortune 500 companies reporting that more than 50% of chief executives were over 183 cm tall, but only 3% were less than 170 cm tall. Current research finds that height is positively associated with income, authority status in the workplace, the likelihood of holding a managerial position vs. a nonmanagerial position, and military rank (Blaker et al. 2013).

Another example of how social bias, social dominance, and height seem to be related came from a study titled "Height as a measure of success in academe" (Hensley 1993). The author of this study analyzed a random sample of 83 male faculty and 52 male heads of department in the United States. Hensley reported that, overall, academics are taller than the average American of the same age and sex, but the degree of tallness varies by academic rank. The mean difference in stature from lower to higher rank are: assistant professors, 3.2 cm taller than the average male; associate professors, 3.8 cm taller; and full professors, 5 cm taller. The sample of department chairmen averaged 5.4 cm taller than American men of the same age. A satirical perspective of the height bias in academia was offered by the British comedy troupe "Monty Python" in their television sketch called "Archeology Today."[22]

Another arena of bias is political contests. In the United States the taller of the two major-party candidates from 1789 to 2012 won 58% of presidential elections and received the majority of the popular vote in 67% of those elections (Stulp et al. 2013). The 2016 election pitted a man against a woman, so the height comparison was not the only bias, but Donald Trump's height of 188–191 cm (according to various "official" documents) vs. Hillary Clinton's 165 cm did not help Mrs. Clinton. Stulp and colleagues also reported that, "Taller presidents were also more likely to be reelected. In addition, presidents were, on average, much taller than men from the same birth cohort" (p. 159). The advantage of taller candidates was explained by perceptions associated with height. The researchers concluded that taller political candidates are perceived as having more and better leadership and communication skills. Other research reports that winners of political contests are perceived as being taller than they actually are, and conversely, political losers also "lose" height. A Canadian study interviewed 177 voters about their perceptions of the heights of several candidates for the 1988 Canadian Federal elections (Higham & Carment

[22] https://www.imdb.com/video/vi1636154649

1992). The subjects were asked to estimate the heights of the candidates both before and after the election. The losers were judged to be shorter after the election, while the winner was considered to be taller than he was before the election. The authors of this study stated that their results confirm previous research showing that people in higher social status positions are perceived as being taller than they really are. In addition, their research shows that the perception of social status and height is dynamic, in that election outcomes can rapidly alter judgments about stature.

It is possible to speculate about the evolutionary, biological, historical, psychological, and cultural reasons why the "bigger is better" bias exists in many societies. There is considerable evidence from research on nonhuman social animals, including the primates, that larger individuals enjoy higher status in their social hierarchies (Clutton-Brock 2016; Dubuc & Clutton-Brock 2019; Ellis 1994; Emery Thompson et al. 2012; Maggioncalda et al. 2002). There is human experimental and observational evidence that taller stature is directly and positively related to feeling and actions of dominance in interpersonal relationships. A few of these experimental and observational studies are described in Box 7.3. For nonhuman animals the rewards of higher status are very tangible, as higher-ranking males and females have preference to desirable foods, more security from predation, less harassment from other members of their social group, and often have greater reproductive success. The reproductive advantage would have been a powerful force of natural selection for strategic adjustments toward greater body size. Recent evidence indicates that strategic growth operates in more subtle and complex ways. Constance Dubuc and Tim Clutton-Brock (2019, p. 1127) reported that " ... some vertebrates can adjust their growth rate in relation to changes in the social context that affect their probability of breeding ... we show that, in meerkats (*Suricata suricatta*), which are singular cooperative breeders, subordinate adult females increase in body mass after their father is replaced as the dominant male in their natal group by an immigrant male, giving them regular access to an unfamiliar and unrelated mating partner ... " The growth increase occurred before increases in food intake. The subordinate brothers of these females did not increase in size and neither did subordinate females in neighboring groups, so the effect was specific to only the subordinate females who now had a chance to breed if they could out-grow their competitors. Adult mammals are not expected to be capable of rapid increase in adult body size. Dubuc and Clutton-Brock (p. 1133) point-out that " ... a number of recent studies now show that the transition from non-breeding to breeding status is associated with a renewal of growth in either or both sexes ... [in orangutans and naked mole rats] ... and these effects may be less rare than has been supposed. If so, they offer unusual opportunities for research into the mechanisms that control."

The social advantages of larger physical size may predate the evolution of *Homo sapiens*. But, the human species adds some further complications to the advantages of larger size. A social bias for desirable types, in terms of stature and body composition, exists in virtually all human cultures. Taller individuals display more self-confidence, even overconfidence, in social interactions (Belmi et al. 2019; Stulp et al. 2012, 2015). The increased self-esteem of the taller or more massive person may

Box 7.3 **Height, dominance, and social mobility**

The three articles reviewed here provide recent evidence that taller stature is directly and positively related to feelings and actions of dominance in interpersonal relationships. The three articles are based on psychology, human ethology, and biological anthropology. Their different academic perspectives complement each other, and the references cited within each article provide an extensive review of the literature. Consult the articles for details of methodologies, analysis, and possible limitations.

Study 1. Human height is positively related to interpersonal dominance in dyadic interactions (Stulp et al. 2015).

The researchers conducted two naturalistic observations to test the hypothesis that height predicts interpersonal dominance during brief interactions between a taller and shorter person. Study 1 investigated the likelihood of giving way in a narrow passage. They observed pedestrians entering and leaving a supermarket through a narrow passageway that was too narrow for two individuals to pass through simultaneously. When approaching from opposite directions one individual was required to give way. In 50 observations of same-sex pairs the shorter person gave way 67% of the time. There was no effect of the sex of the pairs. Study 2 investigated giving way in a busy shopping street, plus the likelihood of colliding with another individual. In this study several members of the research team, with heights varying from 160–183 cm for women and 170–200 cm for men, walked along a crowded shopping street in a straight line while gazing at shop windows. A total of 1,108 encounters between a team member and an unknown pedestrian were observed. Height of the team member was positively related to the likelihood of pedestrian giving way. The effect was stronger for men, that is, pedestrians of shorter height were more likely to give way when the team members was a man. Even when the team member was taller than the pedestrian a collision, such as bumping shoulders, was more likely when the team member's height was at the lower end of the range of variation. The researchers concluded that " . . . in the absence of overt physical aggression, height influences the outcome of nonverbal confrontations between individuals. Thus, the increased social status and upward social mobility of taller individuals in modern society, usually attributed to variables such as improved health and nutrition, may occur, at least in part, as a consequence of their increased interpersonal dominance" (p. 15).

Study 2. The social advantage of miscalibrated individuals: The relationship between social class and overconfidence and its implications for class-based inequality (Belmi et al. 2019).

The power of social dominance derived from high self-esteem and overconfidence to drive interpersonal and group differences in behavior and biology was tested by Belmi and colleagues (2019) with data from 4 large databases

with a combined sample of 152,661 individuals. The first analysis was based on a large field study of small-business owners from Mexico, and found evidence that individuals with relatively high social class are more overconfident compared with their lower-class counterparts. The second analysis was based on measurements of the same individuals at three time points to assess their current social status, their desire to achieve higher social status, and their overconfidence. The findings were that individuals with relatively high vs. low social class have a stronger desire to achieve high social rank and tend to be more overconfident. The third analysis was performed on an independent sample of people to replicate the previous findings. Again, individuals with relatively high social class were more overconfident, even in a task in which they had no performance advantages. Finally, the fourth analysis was based on a mock job interview conducted at a university psychology laboratory. Higher social class individuals were more overconfident; overconfidence, in turn, made them appear more competent and more likely to maintain their current social rank and move to a higher rank. The researchers did not include height or body size in their analyses.

Study 3. Social mobility of the father influences child growth: A three-generation study (Koziel et al. 2019).

Building on the known associations between body height and social status, the researchers tested the effect of intergenerational changes in social status on height. Body height was measured in 2,008 paternal grandfather-father-son and 1,803 paternal grandfather-father-daughter triplets in Poland. The intergenerational triplets were dichotomized according to grandfathers' occupation, into a lower or upper grandparental social class and according to paternal education, into a lower or upper paternal class. Intergenerational comparisons allowed for two nonmobile groups in which grandfathers and fathers stayed in the same social class (both remained in the lower class or both in the higher class), and two mobile groups in which social class of fathers and grandfathers differed (lower class grandfather to higher father or higher grandfather to lower father). It was found that upper social class fathers were taller than lower class fathers. This class effect on height persisted into the third generation. Upward social mobility of lower-class fathers was achieved by them receiving secondary or university education. This was associated with taller stature both in the fathers, when they were children, juveniles, and adolescents, and in their daughters and sons. The opposite applied for downward social mobility. Upper class fathers with only basic or vocational education lost the social advantage of the grandfathers. Both these downwardly mobile fathers and their daughters and sons were shorter. The researchers acknowledge that there was a complex interplay between social class and height, but also that there was good evidence that investment in higher education acted as major regulator of body height.

These three examples of associations between height, social dominance, and confidence, along with dozens of independent studies in the literature, allow for following this possible pathway:

social class ———— over-competence ———— perceived competence ———— height

There would be many biocultural interactions between all four components. Growing girls and boys become increasingly aware of their biological and social assets or deficiencies. Those with the positive assets actively develop positive self-concepts, greater self-confidence, assertiveness, and height.

be a consequence of a lifetime of experience. Children as young as 10-months-old mentally recognize that size plays a role in dominance contests and by age 3 years children have an efficient mental calculus to ascribe dominance in social interactions (Lourenco et al. 2016; Thomsen et al. 2011).

Throughout the world there is a general bias in favor of taller men by women in courtship and mating. This bias is not confined to industrial or even agricultural nations or societies. It operates among the Mehinaku people of Brazil, a traditional culture based on subsistence farmers and fishing. Anthropologist Thomas Gregor (1979) found that taller Mehinaku men had more wives and lovers, more wealth, and more social prestige than shorter men, and the latter were often the objects of social and sexual ridicule. It is interesting to note that the range of stature for Mehinaku men observed by Gregor was 151.8–175.9 cm, meaning that the tallest men would be considered about average in height if US values of tallness and shortness were applied. Gregor reviewed ethnographic data from several other cultures and found that only the Crow of North America and the Mbuti pygmies of Central Africa specifically mention tallness as a disadvantage.

While most societies today and in the historical past favor tallness, the preference is not universal. One such group are the Tsimane', a foraging–farming society of indigenous Amazonians in Bolivia. In one analysis, Ricardo Godoy and colleagues assessed 248 Tsimane' women and 255 Tsimane' men aged 22 years and older (Godoy et al. 2010). The participants were measured and interviewed annually during five consecutive years from 2002 to 2006. The researchers analyzed the association between height and nine outcomes of well-being; including wealth, monetary income, illness, access to credit, mirth, schooling, math skills, plant knowledge, and amount of forest land cleared for farming. Using a statistical regression model, with control for possible confounding variables, they found no significant association between any of the indicators of own well-being with adult stunting or tallness. Another Tsimane' study (Undurraga et al. 2012) performed an experiment in which pairs of photographs were shown to 40 Tsimane' women and 40 men, 16 years old or older. The photographs depicted people of the same sex and age but were manipulated so that one person was shorter. The researchers reported that, "Tsimane' women

and men attributed greater strength, dominance, and knowledge to taller girls and boys, but they did not attribute most positive traits to taller adults, except for strength, and more social concern only when women assessed other women in the photographs" (p. 1). It seems clear that for Tsimane' adults there are no height premiums and stunting penalties. But, the findings for children are less clear. The researchers proposed three possible explanations: (1) height is a signal of good child health; (2) greater height is equated with greater bio-social maturation; and (3) the Tsimane' are in the process of integration into the global market economy and adults' expectations about their children's future success is in the process of accommodating the height bias of the modern, industrialized world.

You Can Be Too Tall

People judged to have excessive height are often at a social disadvantage. In the United States, for example, the ideal height for men is in the range of 182–188 cm and for women from 166 to 177 cm.[23] There is little evidence for greater economic, social, and educational rewards for men above that ideal. Male heights above 193 cm, and female height above 175 cm are incompatible with standard sizes of clothing, the design of most furniture, and the space provided in many automobiles and commercial airlines. People with these "excessive heights" may be required to spend more money in order to compensate. Men, and especially women, who are deemed "too tall" face both overt and subtle types of discrimination in employment and social life. School-age boys and girls who are taller than average may be expected by their teachers to act more mature, be more responsible, and have higher academic performance than their shorter classmates. Teasing and outright physical abuse may be heaped on the "too tall" child. The bias against excessive tallness for girls also comes from parents, who take their "too tall" daughters to the medical doctor for treatments to stop their growth. Estrogen administration is the most common form of treatment, given its role in closure of the growth plates. The use of estrogen comes with immediate and long-term risks for reproductive system pathology and cancer, but the societal and political beliefs about what it means to be a tall girl provide ideological justification for the risks (Lee & Howell 2006). The effects of these types of stresses on taller than average boys and girls have not been well studied, but there is an emerging literature (Booth et al. 2019; Cohen 2009).

Bringing It All Together – Evolution, Ecology, SEPE, Biocultural Reproduction, Community Effects, Strategic Growth, and Human Life History

The conventional wisdom that human growth is optimal when adequate amounts of all nutrients, minimal infection, and adequate psychosocial stimulation are available

[23] https://yougov.co.uk/topics/politics/articles-reports/2014/07/11/ideal-height-56-woman-511-man

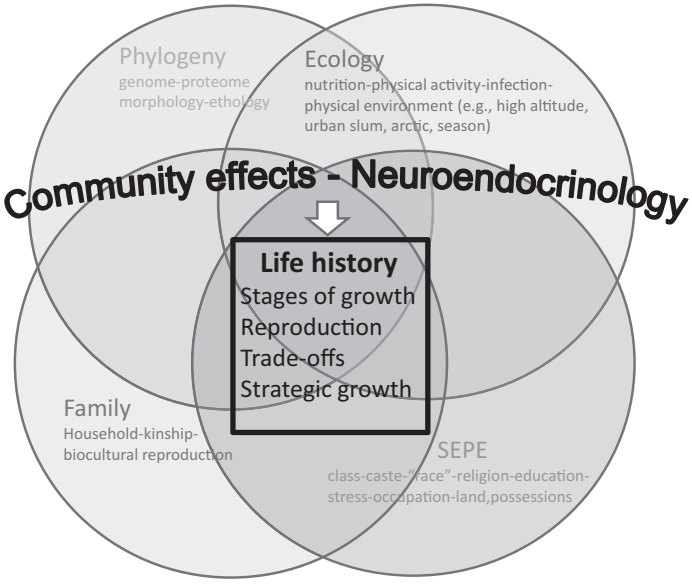


Figure 7.13 Biocultural domains of influence on human life history. The relative spatial distribution of each domain indicates their influences on each other. This version of the figure builds on the information presented in Figure 6.1. The boxes around domains are removed to indicate that all domains are part of a hugely complicated matrix of factors. The "Phylogeny" domain has the most direct impact on genome, on the limits of the functional proteome, and on some aspects of human morphology and behaviors (such as the capacity for bipedalism). The influence of "Phylogeny" is mediated through the domains of "Ecology," "Social–Economic–Political-Emotional," (SEPE) and "Family." All human beings share a highly similar "Phylogeny" domain, but live within highly variable ecologies, SEPE, and families. The unevenness in these last three domains interacts with the phylogenetic factors to produce a range of variation in reaction norms via community effects and neuroendocrine activity. Community effects and their interactions with the neuroendocrine system have the most direct influence on the regulation of human life history, directing development through the stages of growth, adjusting the timing and frequency of reproduction, and modulating trade-offs in biology and behavior that ultimately regulate human strategic growth (original figure).

is too simplistic. The extensive interacting networks of material, biological, social, and ideological variables that comprise human life give rise to a hugely complicated matrix of factors that shape human phenotypes. There is no single optimal pattern of growth. There are ranges of possibilities with a multitude of local optima within the developmental matrix. An outline of the developmental matrix is given in Figure 7.13. This figure builds on the information shown in Figure 6.1 by adding a few more domains of influence on human life history. Figure 7.13 is a graphical abstract of Chapters 4–7. The network of interactions between the biocultural domains shown in Figure 7.13 and the myriad factors within each domain, both shown and not shown, is what makes people grow, develop.

8 A Biocultural View of Human Growth

As this whole volume is one long argument, it may be convenient to the reader to have the leading facts and inferences briefly recapitulated. Charles Darwin, *The Origin of Species*, 1859

Like Darwin, my intent in this final chapter is to recapitulate some of the material from the preceding chapters. This review will take the form of a synthesis of the many strands of my "one long argument" for a biocultural view of human growth. There are two essential messages of this synthesis. First, human growth, development, and maturation are best understood via an evolutionary life history perspective. Throughout this book the life history perspective was applied to the study of the stages of life of modern people, living nonhuman primates, and other mammals. The life history perspective was also applied to better understand the deep biological history – the phylogeny – of the pattern of growth of human ancestors. The second essential message relates to a newer way to conceptualize the biocultural nature of human development. Since the late nineteenth century, anthropologists have used biocultural models to explain human growth and development. Investigators such Bowditch and Boas (Chapter 1) used biocultural models to dispel the unscientific notions of the biological determinists, eugenicists, and racists, who believed that human phenotypes were fixed by heredity and not amenable to environmental influences. Despite the elegant work of Boas and others, prejudice and racism allowed the eugenicists to dominate the scientific and political environment. Even after the atrocities of Nazi extermination camps during World War II, Britain, the United States, and other countries continued to enforce policies of incarceration into "care-homes" or institutions for so-called deficient types, who were mostly from poor families and disabled. In Britain this policy continued until the 1970s.[1]

In the 1960s, with the discovery of the nature of DNA and other fundamentals of developmental biology, growth theory and research focused on human development as, basically, a biological process toward a genetically programmed target size, shape, and body composition. Culture was ignored or downgraded as a regulator of growth, which could be influenced but in no way determined by the social and cultural environment. The concept was that "optimum" nutrition, "optimum" health, and other environmental optima would allow the individual to reach their predestined genetic target. The "optimum" was never specified and, in fact, notions of growth

[1] www.theguardian.com/commentisfree/2019/oct/03/eugenics-francis-galton-science-ideas; if possible, watch the 2019 BBC series on Eugenics www.bbc.co.uk/programmes/m0008zc7.

"optima" have changed since the 1960s and 1970s. One example is the change from recommendations for formula-feeding of infants to breast-feeding. The propaganda in favor of formula-feeding fueled increases in infant morbidity and mortality in lower-income nations where families unable to afford sufficient formula, and unable to sterilize bottle and nipples, fed their infants watered-down formula from filthy bottles (Muller 1974).

Since the 1990s there has grown a revised and expanded biocultural view that there is a recurring and seamless interaction between the biology of human development and the sociocultural environment. Not only does the latter influence the former, but human developmental biology modifies social and cultural processes as well. It is now understood that environmental forces, including many social, economic, political, and emotional (SEPE) factors, regulate the expression of the genome as much, or more so, than any single nucleotide polymorphism (SNP) of DNA regulates the growth process. Some researchers held on to the old notion of genetic determinism and articles published even in 2019 claim that about 80% of height is determined by genes. These claims and the search for the "missing heritability" from genome-wide association studies (GWAS) analysis, discussed in Chapter 6, are astonishing examples of the persistence of mid-twentieth-century traditional notions even in the presence of newer, profound knowledge of the complex nature of interactions between genome, epigenome, physical environment, and SEPE factors.

Today we understand that growth, development, and maturation take place within a wide frame, and are not directed toward a narrow target. A hugely complex matrix of phylogenetic, ecological, familial, and SEPE factors form a "lens" of biocultural plasticity that refracts growth trajectories to create many possible outcomes across the landscape of the frame. The differences between the older "growth toward a target" and "growth within a frame" concepts are illustrated in Figure 8.1. This chapter is dedicated to recent progress and future applications of life history theory to human growth and the expansion of the biocultural perspective toward human development to a fully interactional model.

Biocultural Interactions in Contemporary Populations

Infant, child, juvenile, adolescent, adult, post-menopause grandmother, and the generally but not always post-reproductive grandfather are universal biological stages of human postnatal growth and development. There is much cultural variation, however, in the social and behavioral response to each of these stages. Moreover, the evolution of the new human life history stages of childhood, adolescence, and grandparenthood bring about many biosocial benefits, but also incur new risks. The evolution of any new characteristic incurs both benefits and risks. Bipedalism, for example, is the method of human locomotion unique among the primates. Often considered to be one of the crucial feeding and reproductive adaptations of our species, bipedalism also brings about many physical ailments, including lower back pain, fallen arches, and inguinal hernias. In a similar fashion, the benefits of new life history stages need to be tempered against their hazards.

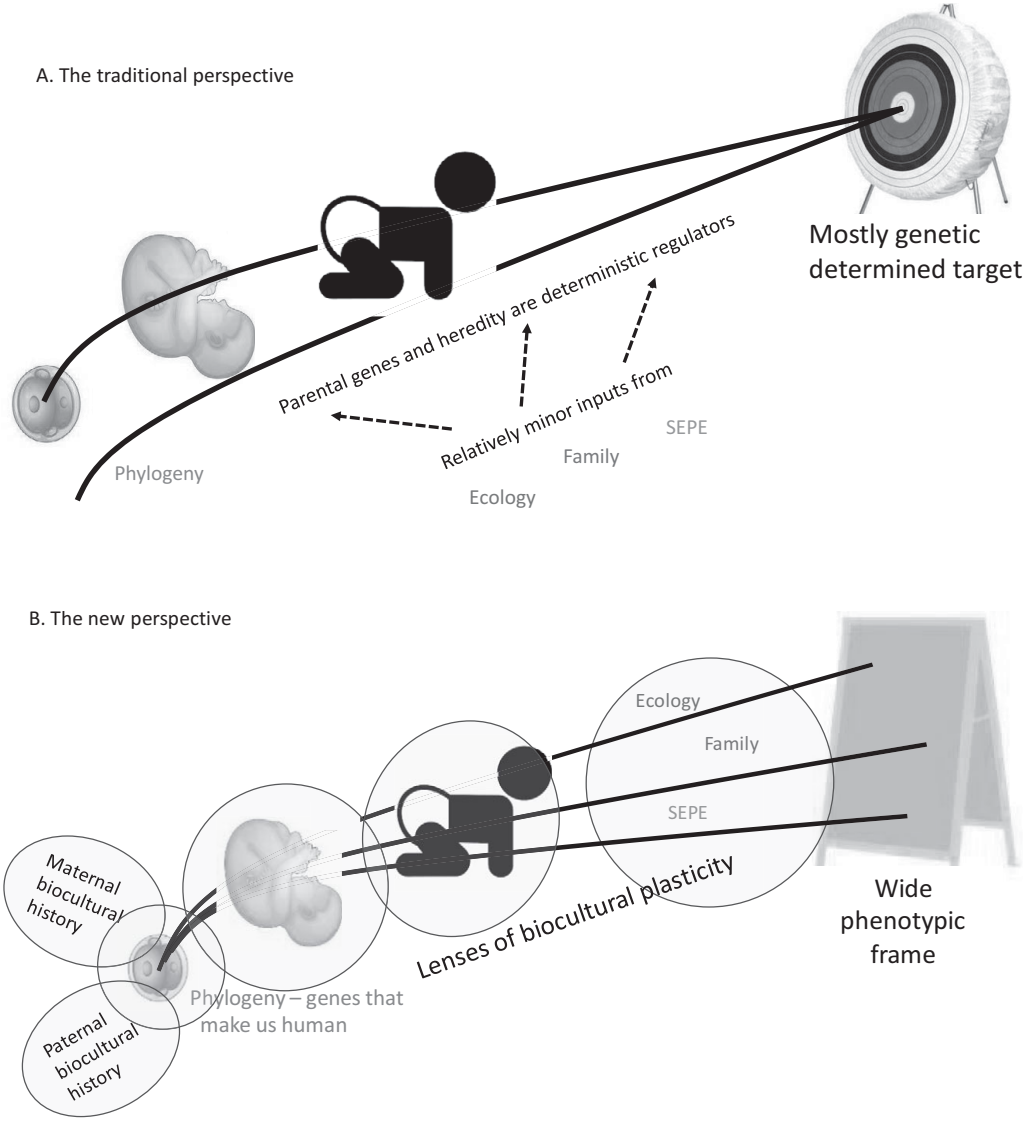


Figure 8.1 Part A represents the traditional perspective of "growth toward a target." From the dividing cells of the zygote to the fetus, to the infant and beyond, growth is directed by deterministic regulation of genetic inheritance from Phylogeny and parental genes and other types of imagined inheritance. The influence of these causes growth to converge toward the adult target for size, shape, rate of maturation, etc., with only minor inputs from the Ecology, Family, and SEPE factors. Part B represents the newer perspective adopted in this book of "growth within a frame." The border of the frame sets the limits, which are quite large, for normal variation in size, shape, rate of maturation, etc. The domains of Phylogeny, Ecology, Family, and SEPE factors now form "lenses" of biocultural plasticity. These biocultural lenses are at work during all stages of growth and development of the individual and even operate between generations, via maternal and paternal biocultural history, grandparental history, and so on. These biocultural lenses refract growth trajectories toward many possible outcomes across the landscape of the frame. Original figure, based on a suggestion from Michael Hermanussen. (A black and white version of this figure will appear in some formats. For the color version, please refer to the plate section.)

Risks for Young People

Dependency on older individuals for food and protection, small body size, slow rate of growth, and delayed reproductive maturation each entail liabilities to the infant, child, juvenile, and adolescent. The "charms of youth" do not provide for total security. To illustrate this point, we may examine traditional societies of both historic and prehistoric eras. In such societies, including hunter-gatherers and horticulturists, about 35% of live born humans die by age 7, that is, by the end of childhood (Bogin & Smith 2012). Even if two-thirds of these deaths occur during the infancy stage, the childhood period still has an appreciable risk of death. In hunter-gatherer societies, starvation, accidents, and predation account for most childhood deaths. !Kung parents, in an effort to minimize these risks, protect their children by confining them to camp and the watchfulness of adults (Draper 1976). Mbuti parents (hunter-gatherers of central African forests) allow children and juveniles to form age-graded play groups, also located near the camp. This method of grouping children with older juveniles and adolescents demands less parental supervision (Turnbull 1983). However, the risks of hunter-gather childhood can, perhaps, best be illustrated by Hadza society. Hadza children, aged three to eight years, form age-graded work/play groups that may wander far from camp, often out of sight of older individuals, and carry out costly and dangerous tasks including significant foraging for food (Blurton-Jones 2002; Hawkes et al. 2018). One study found that of 301 Hadza infants born to 75 women aged 20–50 years old, 115 (39%) of the offspring had died by the time of the survey (Blurton Jones et al. 1992). Demographic modeling indicates that most of the deaths occurred during infancy and childhood. The living Hadza may not be the best models for the human evolutionary past, because the Hadza have been pushed into less secure and productive lands by more numerous and powerful agriculturalists, pastoralists, Colonial-era conquerors, and post-Colonial governments. Even so, Volk and Atkinson (2013) analyzed data from 20 contemporary forager societies and 43 historical societies, from Greece in 400 BCE to China, Japan, and Western Europe in the year AD 1900. The researchers estimated an average infant mortality rate of 26.9% for all societies, with no significant statistical difference between the foragers and the historic past. Mortality prior to age 15 years was estimated to average 47.5% for all societies. Volk and Atkinson stated that, "These rates represent a serious selective pressure faced by humanity . . . " (p. 182) and may have helped to shape human parenting, **attachment behavior**, theory of mind, alloparenting, language, kinship, and other aspects of human biocultural reproduction.

Today, hunter-gatherer and traditional horticultural societies account for less than one percent of human cultures, so it may be more instructive to examine current risks to children in post-Colonial and industrialized societies. The United Nations reports that globally, the under-5-year-old mortality rate was 39 deaths per 1,000 live births (3.9%) in 2017. Between the ages of 5–17 years of age the global mortality rate is 0.7%. The world is a much better place today in terms of mortality between birth and adulthood. But, the world could be an even better place. The most common risks to

pre-adults today are inadequate nutrition, infection, heavy workloads, neglect, physical/emotional abuse, and violence. Most of these risks were assessed and discussed in previous chapters. Viewed in historical perspective, inadequate nutrition, infection, and unreasonable workloads for children, juveniles, and adolescents are products of the agricultural and industrial revolutions. Agriculture, for example, increased social stratification and reduced the variety and quality of foods consumed by people of the lower social classes (Bogin 1998a, 2001). Industrialization further increased social disparities and resulted in the forced labor of children in mines and factories (Chapter 1).

Despite the efforts to enact programs of legal protection and welfare for children and youth since the nineteenth century, there remain many risks for children in the contemporary world. Underage labor is rampant in all nations and is associated with risks for abuse and neglect. The International Labour Organization (ILO 2017) estimates that 152 million children – 64 million girls and 88 million boys – are in daily child labor globally. The ILO defines "child" as a person less than 18 years old. The ILO estimates that 48% of these young people are children or juveniles between 5–11 years old. About 71% are working in agriculture, and 12% in industry, and 17% in services (e.g., cleaning). Nearly half of all those in child labor – 73 million under 18s in absolute terms – are in hazardous work that directly endangers their health, safety, and moral development. The total number of people under 18 years old in employment, a broader measure comprising both illegal child labor and permitted forms of employment involving young people of legal working age, number 218 million.

Abuse, neglect, and violence are also global problems. The World Health Organization data for abuse and neglect are shown in Table 8.1. The numbers are percentages of people under age 18 and the evidence strongly indicates that children and juveniles, due to their greater dependence on adults, are most affected. The sexual abuse data are given separately for boys and girls, as girls suffer 2.4 times the amount of sexual abuse than boys.

Susan Hillis and colleagues (2016) published a systematic review the global prevalence of violence against children (defined as people aged 2–17 years old) for the year 2015. The team reviewed 38 reports providing quality data for 96 countries of violence directed at the children or witnessed by them. The researchers estimated that " . . . a minimum of 50% or more of children in Asia, Africa, and Northern

Table 8.1 World Health Organization global estimates of maltreatment of people under 18 years old. All numbers are percentages of the world population experiencing one or more episodes of maltreatment. Source: http://apps.who.int/gho/data/view.main.VIOLENCECHILDMALTREATMENTv

Indicator	Age Group	Both sexes	Male	Female
Lifetime prevalence of child emotional abuse (%)	<18 years	36.3		
Lifetime prevalence of child physical abuse (%)	<18 years	22.6		
Lifetime prevalence of child physical neglect (%)	<18 years	16.3		
Lifetime prevalence of child sexual abuse (%)	<18 years		7.6	18

America experienced past-year violence, and that globally over half of all children – 1 billion children, ages 2–17 years – experienced such violence" (p. 1).

As explained in Chapter 7, these adverse childhood experiences (encompassing infants, children, juveniles, and adolescents) have immediate and long-term consequences for biocultural growth and development. Some cases of abuse, neglect, and violence may result from a severance of the biology of human infant, childhood, juvenile, and adolescent stages from the rapid pace of technological, social, and ideological change relating to families and their offspring. With the advent of safe infant feeding formulas and sanitary delivery devices (bottles, nipples, etc.) it became technologically possible to nourish infants without breast-feeding. This allowed parents (mothers) of all socioeconomic statuses an opportunity to pursue other socioeconomic activities or have another baby. In the United States, nearly 29% of mothers who had a second or higher-order birth in 2014 had a short birth interval of less than 18 months (Thoma et al. 2016). Short interpregnancy intervals are associated with adverse birth outcomes, such as preterm birth, even in wealthy nations. Among the poor populations of the lower-income countries, short birth intervals (less than 23 months) compromise the health of both the infant and the mother. A major negative effect on the infant is low birth weight, which is known to impair both physical growth and cognitive development during the years of growth and later life stages.

In the United States for the year 2016, about 84% of infants were "ever-breastfed" meaning breastfed at least one time during their first 12 months after birth.[2] The US government recommendation is exclusive breastfeeding until age 6 months. In the United States in 2016, exclusive breastfeeding at 3 months was 48% and at 6 months it was 25%. Other data indicate that the weaning process (from breast or bottle) to other foods may begin by three months of age. This severely curtails the infant stage and prolongs childhood. These "premature children" present a problem for care, often solved by sequestering them to restraining devices such as highchairs, playpens, and cribs (cots) and segregating them from the family by placement in creches or preschools. When the infants react poorly to these arrangements, the frustrated parents or care givers may respond with abusive or neglectful behavior.

Older children, juveniles, and adolescents are age-segregated in schools. The evolutionarily derived learning needs of these stages of development are often ignored by formal school curricula. There are calls for a new science of learning, based on insights from evolutionary biology, psychology, neuroscience, instructional technology, and education (Meltzoff et al. 2009). Other concerns relate to fertility rates in the richer nations of less than two offspring per woman, on average. Such low fertility denies juveniles of opportunities to participate in childcare. Due to schooling laws, job market realities, and moral codes, adolescents are unable or strongly discouraged from participation in adult economic and sexual activities. Young adults increasingly require many years of post-secondary education before

[2] www.cdc.gov/breastfeeding/data/nis_data/results.html

attaining the social, economic, political, and emotional status required to marry and begin a family.

The Western model of behavior stands in sharp contrast to that of many non-Western societies. Only a few of the older, "classic" ethnographic reports are reviewed here, but many, many newer publications are available (see Lancy 2014). Thomas Weisner (1987) studied sibling child care as an institutionalized method to prepare juvenile girls for the demands of motherhood. Weisner worked with the Abaluyia tribe of Kenya, with Hawaiians, and surveyed other published research on this topic, including nonmiddle-class subcultures in the United States. He found that, "In most non-Western societies, training for competent, culturally appropriate child-care is an active apprenticeship experience for [juveniles], usually completed by adolescence and learned along with the performance of domestic tasks essential for family, and often community, survival" (p. 238). The importance of this apprenticeship in relation to human evolution was explained in Chapter 4. To reiterate, the juvenile girl as sibling caretaker allows the mother, and father, to do work that provides food, shelter, and other necessities for the whole family. One may also note that juvenile caretakers allow the mother to have another infant after a healthy birth interval that includes at least 3 years of infant feeding via breast milk and complementary foods. Other research focuses on adolescence. Carol Worthman's biocultural research with the Kikuyu tribe of Kenya was highlighted in Chapter 4. Worthman found that adolescence is a well defined and prolonged stage of development. As with most rural African cultures, the traditional Kikuyu do not use the concept of chronological age, rather they use the biological markers of sexual maturation to define adolescence. Kikuyu girls officially enter adolescence following circumcision, which Worthman described as removing "the very tip of the clitoris," but other sources described as radical clitorectomy or worse (Boddy 2007). This rite of passage was timed by the development of breasts, body hair, and growth in height to take place just before menarche. Following the circumcision ritual, the girls began receiving explicit teaching in reproductive function and sociosexual behavior. Girls also were expected to work with the older women of their household in domestic chores; learning about cooking, tending fields, and childcare.

Adolescents in non-Western societies learn these skills in practical work settings, not in school settings. Thus, as discussed in Chapter 4 the work that adolescents perform results in real productive contributions to the welfare of the family and the social group. The contributions of adolescents often have positive impacts on the physical growth of infants, children, and juveniles in these communities. Working in a rural agricultural village of Aymara people in highland Bolivia, Sara Stinson (1980) found that the number of productive adolescents living in a household was related to the growth of children in the same household. After accounting for differences in SES between families, the children living in households with more adolescents were taller than children in households with few or no adolescents. In contrast, heights of infants and children were reduced in households with many young, nonproducing children and few or no adolescents.

Prior to Western acculturation, Copper Eskimos (Canadian Inuit), a hunting-based society, followed a course of adolescent social development that was closely tied to their biological development. Richard Condon (1990) reported that adolescent girls married at or just prior to menarche. The timing of such marriages was, most likely, predicted by the regular series of puberty events, such as breast development and the growth spurt, that always precede menarche. The new bride could not bear children for three to four years, due to her adolescent subfecundity (discussed in Chapter 4), so she had time to improve her economic and social skills, on-the-job as it were, prior to motherhood which took place at an average age of 19 years. Young men had to prove their abilities as hunters and providers prior to marriage. Until age 17 or 18 adolescent males were not strong enough (pre-muscle spurt as explained in Chapter 4) to hunt large game or to build a snow house. At that age the late adolescent male might be accepted by a prospective father-in-law but years of bride service prior to marriage was required. Thus, the groom finished growing and was a young adult (25+ years old), with years of experience at important adult economic and social activities, at the time of marriage.

These few examples (see the works cited here for additional cases) show that, "In many societies around the world training for parenthood is an apprenticeship experience, learned along with the performance of domestic and subsistence tasks within shared-function family systems. In such shared caretaking families, child-care skills are acquired first, followed only gradually by autonomy from parents and siblings, and then by full managerial control of a household" (Weisner 1987, p. 265). This behavioral sequence is in harmony with the normal succession of stages in human biological development and promotes good physical growth.

The influence of contemporary Western culture may introduce considerable discord into the normally harmonious relationship between human biology and behavior. Weisner reported that Kenyan mothers with more formal (European-style) education believed that sibling care responsibilities teach juveniles to be passive and that domestic work, including childcare, is menial. Condon (1990) also reported that due to the loss of their hunting lifestyle, the Inuit were forced to acculturate to settled life, the economics of wage labor, and the social values of television. The biocultural definition of Inuit life stages was also transformed. The biggest change was lengthening of the adolescent stage due to earlier sexual maturation, that is, menarche occurred at an earlier age than in the past. Condon reported that a longer adolescence affects girls especially, as they married several years after menarche. That is not a bad thing and better aligns motherhood with the completion of growth and education. Another modification is that during the juvenile and adolescent stages both sexes have much autonomy from parental control, and no longer participate in traditional social learning. A likely consequence of these shifts in values and behavior for future generations of Kenyans and Inuit will be a delay in the acquisition of parental knowledge of human growth and development until after the birth of the first infant. The consequences of these changes in social learning for the physical growth and development of the next generation are less clear.

Risks for Grandparents

Finally, the evolution of a significant period of life after women experience menopause is associated with several risks for older women. The hormonal changes and bone loss that occur with the cessation of ovarian function, reviewed in detail in Chapter 4, are one type of risk. These biological changes may bring about several degenerative diseases, such as osteoporosis, heart disease, and dementia. Men may also be at risk for these same diseases due to a decline in the production of several hormones, including IGF-1 and the gonadal hormones. Post-menopausal women, and older men, must often assume new social and economic roles for which they need adequate training and social support. However, in some "modern" societies, grandmothers and grandfathers, just like their adolescent grandchildren, may no longer receive appropriate training for post-reproductive sexual, social, and economic expectations. In these societies the elderly may also be denied a productive social role, and may even be segregated away from productive society – in "retirement communities" or "assisted living facilities" (another name for an old age home) for those who can afford them, or poverty-level housing for those of limited means. The social isolation that these sequestered elderly people may experience is known to exacerbate the normal degenerative process of aging. Moreover, research shows that children living in households with little or no contact with grandparents suffer more abuse and neglect than children in multigenerational households – another testament to the value of grandmothers and grandfathers.

The Most Important Discoveries about Human Growth, Development, and Maturation

When I began preparations for this new edition of *Patterns of Human Growth* it occurred to me that a list of the most important discoveries in the field might be a good way to start. I prepared a list of on September 7, 2016 and saved it as I had other commitments to complete before working on the book. In November of 2017 I started work on this new edition, remembered my list and sent it to a few colleagues asking them to add, delete, or modify my list as needed. By December I received a few replies. I thank Michael Hermanussen, Christiane Scheffler, Chris Kuzawa, Carlos Varea, Luis Rios, and Gillian Bentley for their suggestions. The following is my distillation of the suggestions into one list, which emphasizes the biocultural discoveries more than technical methods of measurement or data analysis. Details of these discoveries were discussed in previous chapters and more of their history may be found online. Here the list includes the discovery, its importance to practical applications, and its connection to later discoveries, some of which are yet to be discovered!

1. Embryogenesis, rather than preformation – William Harvey, 1651 and sexual fertilization – Oscar Hertwig, 1875. Eventually leads to the importance of DNA.
2. Phenotype plasticity – Boas, 1912. Derived from the study of migrants, the concept of development plasticity eventually leads to Waddington's vision of

the epigenetic landscape, 1942,1957, the field of epigenetics, and the realization of "Growth as a mirror of the condition of society" by Tanner, 1986, the importance of social-economic-political-emotional (SEPE) factors and "growth within a frame" as described in this book.

3. X-rays and radiography – Wilhelm Conrad Röntgen, on January 23 1896 Röntgen used X-rays to produce a radiographic image of the bones of the hand of 78-year-old Professor von Kölliger and showed the image to members of the medical community. When applied to still-growing individuals, X-rays led to discovery of the growth plate and the concept and methods for skeletal age estimation.
4. Speciation by changed growth trajectories and allometry – D'Arcy Thompson, 1917, Huxley and Tessier, 1936. Their mathematical and conceptual discoveries were fundamental to the creation of the field of Evolutionary Developmental Biology.
5. Documentation of the substantial independence of dietary quantity and longitudinal growth, Schlesinger, 1919. By the 1960s this and related work was largely forgotten until rediscovery by Hermanussen and Scheffler, 2018.
6. Essential nutrient deficiency can stop growth – many people were involved with the discovery of the ~50 essential nutrients, most of which were proposed and then chemically isolated in the years 1900–1950.
7. Tooth development and eruption are markers of life history stages – Krogman, 1930, Schultz, 1935. Dental maturation underlies all of mammalian life history biology. Today, dental microstructure and periodicity in growth of dental tissues are linked to life history and behavioral traits such as weaning, diet, migration, age at reproduction, and other variables. Schultz's 1960 book on primate life history evolution led to my discoveries about the evolution of the pattern human growth presented in the first edition (1988) of this book.
8. Fleming describes the adolescent growth spurt and sex differences in maturational timing and intensity, 1933. The reality of human adolescence as a biological stage of development is established. Widdowson, 1951, shows that nutrition and emotion interact. Both show the need for physically and emotionally stable environments for adequate nutrition and growth.
9. Growth is target seeking – Tanner, 1963. Then and today there is little agreement on the target. But the concept allowed for an understanding of the biology of catch-up and catch-down growth, which have widespread clinical utility.
10. Li, 1966 discovered that GH is a protein. This led to discoveries of other endocrine growth factors, such as IGF-1, IGF-2, their binding hormones, cell receptors, etc.
11. Linear growth does not occur continuously but in short-term, even overnight, saltational bursts – Hermanussen and colleagues, 1988; Lampl and colleagues, 1992. These discoveries required precision measurements and were aided by the invention of knemometry, Valk and colleagues, 1983 and mini-knemometry, Hermanussen and Seele, 1997. Both are high precision techniques for lower leg length measurements in infancy and early childhood. Saltational growth and precision measurement of body segments are applied to understanding the

development of human body shape in response to SEPE stressors and are applied in the clinical treatment of several disorders of human growth and development.
12. Fetal growth, and fetal nutrition, predict later-life adult phenotypes and risk for chronic disease – Barker and colleagues 1989. The aging process and many diseases of aging have partial origins in the earliest stages of in utero development.
13. Strategic growth – dominance vs. subordinate status influences IGF-1 secretion and body size, Sapolsky and colleagues, 1997, Huchard 2016.
14. Community effects – social-economic-political-emotional networks regulate body size, shape, and composition – many people contributed to this. Aßmann and Hermanussen, 2013, published the first formal model of evidence for a community-based target in height.

Unsolved Problems for Future Research

Building on a few of the discoveries listed here, I propose that the following current "problems" of human growth, development, and maturation are solvable by better research that incorporates the newer hypotheses based on SEPE factors, community effects, and strategic growth adjustments presented in this book. I leave it to you, dear reader, to pursue research on these and other unsolved problems.

Sibling Effects on Height

Birth order is an important determinant of height. Later-born siblings are, on average, shorter than earlier-borns (Lawson & Mace 2008, but see Hermanussen et al. 1988c for a counter example). The effect is usually ascribed to resource dilution, meaning that the parents have fewer and/or low-quality resources to invest in each subsequent offspring. The resources may be material (food, health care, education) or may be time and affection. In his 1962 book, Tanner (1962, p. 138) illustrated the sibling effect using data from the 1947 Scottish Mental Health Survey. The heights of the total population of 11-year-olds in Scotland were measured along with their father's, or primary caregiver's, occupation. The occupations were used to create 7 social classes: Classes 1–3 were professionals, employers, and salaried staff; Class 4 nonmanual wage-earners; Classes 5 and 6 were skilled and semi-skilled manual laborers; and Class 7 were unskilled laborers. Tanner's figure for the relationship of height to social class and size of the family is reproduced here as Figure 8.2. "Family size" was defined in the Scottish Mental Health Survey as the number of siblings in the family and the birth order of each sibling. With a few exceptions there is a monotonic decrease in height from higher to lower social class and with increasing family size. A dilution of material resources may account for the shorter height of later-born 11-year-olds of social classes 4–7, but is less feasible for social classes 1–3, whose caregivers would have been able to afford all needed food, health care,

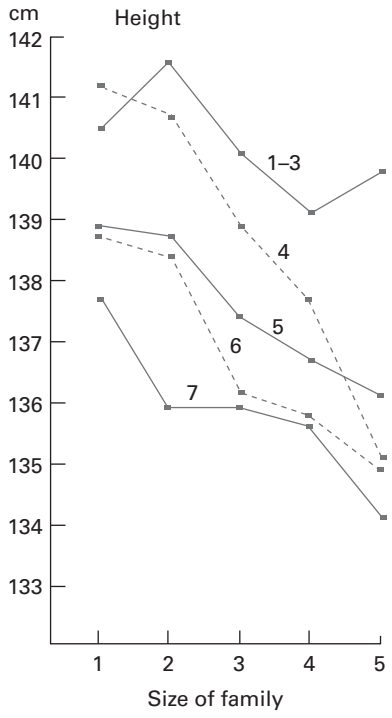


Figure 8.2 Relation between height of 11-year-old juveniles and size of the family in different social classes in Scotland, 1947. Social classes are labeled 1–7 and are described in the text. After Tanner (1963)

and education. In fact, the pattern for social classes 1–3 is different from the others, with 2nd and 5th borns taller than predicted.

Taking a meerkat perspective, there is another explanation, namely the community effects of the family and strategic growth adjustments. Like meerkats, human siblings compete for social status and affection within their families. One study of human sibship size and height is especially relevant. The data came from the Avon Longitudinal Study of Parents and Children (ALSPAC), an ongoing birth cohort study of British children and their families. The participants are mostly white, native British and of varying SES, similar to the SES variation of the UK as a whole. Growth measurements of the study children were taken regularly from birth onward and families were interviewed and observed up to three times per year. Lawson and Mace (2008) analyzed more than 12,000 records for the associations of sibling number, birth order, and birth spacing with growth of the study child from birth to age 10 years. Similar to Tanner's analysis of 1947 Scottish data, the ALSPAC data show a near monotonic decrease in amount and rate of height growth with more siblings in the family. It is especially important to note that later-born siblings are not just shorter, adjusted for their age and sex, they grow more slowly. Adjusting for family SES did not change the findings. When compared with children without siblings the

researchers wrote that " . . . children with four siblings had a reduced rate of growth by 2.3 mm per year (95% CI: 3.8 to 0.8), leading to a deficit of 31.5 mm by age 10" (p. 1408). Older siblings were associated with larger deficits of height in the study child than were younger siblings. The sex of the older sibling did not influence the findings.

The authors interpreted their findings in terms of the conventional explanation of a dilution of parental investment and resource allocation with more siblings. The ALSPAC families are, however, relatively wealthy, the children are well fed, and British society has universal health care and education, with extra benefits to those in need. It is difficult to understand how the sibling effect on height can be explained by resource dilution. Especially puzzling is the greater effect of an older sibling vs. a younger sibling. Social competition may be a better explanation. With or without intentional malice, older siblings are known to inflict physical and emotional stress on younger brothers and sisters (Buist et al. 2013). The presence of an older, and bigger, sibling likely has impacts on the hypothalamic–pituitary–adrenal (HPA) axis, cortisol reactivity, and GH/IGF-1 levels, which can all impact growth and final size.

The Nutritional "Dual Burden," or Stress, Short Stature, and Over-Fatness?

The combination of very short stature with excess body fatness in the same population, family, or individual is often called the nutritional dual-burden. But, it is not simply a problem of nutrition or diet; rather it is a burden of overweight and obesity (excessive fatness) regardless of stature. There is no doubt that overweight/obesity places people at increased risk for both physical and mental illness. Very short stature also entails some of these same risks, so the combination may be a "dual-burden." Many people in the lower income nations of the world are known to suffer from very short stature with overweight/obesity and the Maya children, juveniles, adolescents, and adults discussed in this book are an extreme example. Twenty years ago, the phrase "nutritional dual-burden" did not exist. Human biologists thought it was physically impossible to suffer from both short stature due to diet inadequacy and over-fatness due to excess energy intake. A new globalized culture, with novel forms of technology, social organization, and ideology, were required to create food environments that never existed in human history to produce people, families, and communities that are characterized by very short stature and overweight/obesity (Bogin et al. 2014a). Biologically, socially, and ideologically the human species is not adapted to the globalized food environment.

Public and private health agencies usually ascribe the "dual-burden" to poor diet and inadequate physical activity and ask that the people affected change their behavior. This type of blaming the victim intervention has proved useless and the worldwide prevalence of overweight/obesity has increased steadily, especially since 2008, the year of the global banking and economic crisis. Increases in stature have occurred much more slowly than increases in fatness, so it appears that there is a new health problem – the dual-burden – when in fact it is an increase in fatness of most people, including the very short. As discussed in Chapter 7, a more productive

approach is to consider the major causes of very short stature and overweight/obesity to be due to the emotional stress imposed by harmful SEPE factors and community effects. These include the insecurity imposed by economic inequality, the persistence of preventable infectious diseases and parasites, unsafe water, globalized production of low-quality foods, and the unbearable emotional stress of being segregated into communities that experience constant violence and inequitable political abuse. The physiological response to these chronic stressors is upregulation of glucocorticoids and calcitonin, and down-regulation of GH/IGF-1 activity.

The impact of stress on the most vulnerable in our societies, poor people, is rarely considered in research carried out by nutritionists and public health specialists. Stress gets into the skeleton (Berger et al. 2019) and into the adipose tissue (de Kloet & Herman 2018) and the cross-communication between these tissues and the brain results in bone loss and fat deposition. The effects of stress on bone were reviewed in Chapter 7. It is well established that chronic elevations of serum glucocorticoids enhance the storage of lipids within the adipocytes (fat cells), especially the visceral adipocytes (as opposed to the subcutaneous fat tissue). "Clinical evidence for this is perhaps most robust in the setting of Cushing's Syndrome, which is characterized by high endogenous levels of glucocorticoids that lead to elevated central obesity, muscles weakness, hyperlipidemia, glucose intolerance and many more physiological abnormalities" (de Kloet & Herman 2018, p. 51). For growing young people, the outcome of chronic stress is likely to be short stature and overweight (Figure 7.3). These are serious problems, but the cause is not, primarily, nutritional and nonnutritional interventions are needed.

Father's Absence and Age at Menarche

A provocative hypothesis about early socialization and later reproductive strategy was proposed by Jay Belsky, Laurence Steinberg, and Patricia Draper (Belsky et al. 1991). They described two alternative developmental pathways of reproductive maturation and parental behavior. "One is characterized, in childhood, by a stressful rearing environment and the development of insecure attachments to parents and subsequent behavior problems; in adolescence by early pubertal development and precocious sexuality; and in adulthood by unstable pair bonds and limited investment in child rearing" (p. 647). The other pathway " ... is characterized by the opposite" (p. 647). Presumably, this means that childhood is nonstressful, with secure attachments and few behavior problems; pubertal development and adolescent sexual behavior occur at average or late ages, and in adulthood stable pair bonds and intensive investment in child-rearing occur.

This hypothesis is a good example of the power of life history theory and a biocultural interaction model to integrate many seemingly disparate developmental events into a unified pattern. There have been several tests of the hypothesis with conflicting results. One of the most consistent findings is that the absence of the father from the family, due to separation, divorce, or death, is associated with earlier menarche in his presumed biological daughter. This finding generated a good deal of

theoretical modeling to explain the association in terms of an evolutionary reproductive strategy – essentially, early life adversity predicts shortened life expectancy, and this stimulates a trade-off (a strategic growth adjustment) between early sexual maturation and reproduction but with less investment in offspring.

The evolutionary model is attractive but may not be needed. The cause of earlier menarche may be due to sexual and emotional abuse experienced by the girls and the immediate effects of these stresses on their sexual maturation. There is an extensive sociological literature on father's absence and its consequences for the daughters and sons. Much of this literature deals with the physical, economic, and emotional stress on the offspring imposed by the loss of the father. In many settings around the world, both in wealthier and poorer families, father's absence leads to a decline in material standard of living and a series of new sexual partners for the mother. This situation often causes many adverse childhood experiences (ACEs), including sexual abuse of the children by the new partner, by their associates, and by family members who take advantage of the children. A systematic review and meta-analysis of 43 studies by Zhang and colleagues (2019) found that the total number of ACEs was not associated with early pubertal timing as measured by secondary sexual development or menarche. However, specific types of ACEs had small to medium statistical associations. These were father absence, sexual abuse, and family dysfunction. Much sociological, medical, and legal literature reports that childhood sexual abuse and early pubertal development are tightly correlated. Whether one is causative of the other or both are the consequence of other factors is not known. To understand the causes of the associations requires an interactive biocultural model of sexual maturation and sexual abuse, but not necessarily an evolutionary explanation.

Climate Change, Uncertainty, and Allostatic Load

Allostatic load is defined as the cost of chronic exposure to elevated or fluctuating endocrine or neural responses resulting from chronic or repeated challenges that the individual experiences as stressful (Edes & Crews 2017; Kudielka & Kirschbaum 2001; McEwen & Stellar 1993). As I write this last chapter the United Nations released its 2019 report, *The Heat Is on Taking Stock of Global Climate Ambition*.[3] The report concludes that climate uncertainty, rising sea levels, and ocean acidification are all critical threats to the human population. The report recommends that " ... business as usual simply isn't good enough anymore. We must do more – much more ... Climate change is fast outpacing us and needs an urgent response by all segments of society." Climate uncertainty imposes material and emotional stress on all the world's population. Even those people who deny the scientific evidence are exposed to the allostatic load imposed by climate uncertainty. What might the effects of this stress be on human growth, development, and maturation?

[3] www.undp.org/content/undp/en/home/librarypage/environment-energy/climate_change/ndc-global-outlook-report-2019.html

The title of this section is borrowed from the article by Douglas Crews and colleagues (2019) that assesses likely effects. The researchers explain that, "Allostasis, an evolved neuroendocrine/physiological stressor response system, is our main pathway for doing so. Effective allostasis returns somatic systems to their current optima; over a lifetime of stressor responses, related systems fail, effectiveness declines, and physiological dysregulation (i.e., allostatic load) increases. Global Climate Change (GCC) multiplies environmental stressors on human populations and is likely to increase allostatic load" (p. 3).

GCC increases risks for food and water insecurity, sociocultural instability, economic and political volatility, and motivates migration. Crews and colleagues reviewed the literature, with an emphasis on ethnographic case studies of human groups dealing with climate uncertainty. They find consistent evidence that GCC multiplies stressors on local populations. The maximum impact of these stressors will, of course, be felt by people who are growing, developing, and maturing. From embryo to adolescent, those exposed to GCC are likely to show the effects of lifelong stress and elevated allostatic load. Via the intergenerational effects of epigenetics and cultural inheritance this stress and allostatic load may be passed to their descendants. There is an alternative that comes from research leading to ECCSEP – an Emotional Commitment to Change Society, Economics, and Politics – toward sustainable action.

Coda

It is my hope that this book will stimulate some readers to enter the field of human growth, development, and maturation research. Important new understandings about the nature of human biology, society, and culture await you. Human knowledge of how our bodies work, and how we relate to the biocultural world in which we live, builds incrementally, and in small steps. The need for this heavily revised third edition of this book is just one obvious example of such change in a twenty-year period. It is my hope that in another decade or so this book will, again, be hopelessly out of date. Perhaps some of my readers will help to hasten that day.

Isaac Newton said, "If I have seen further, it is by standing on the shoulders of giants." The research and ideas presented in this book stand on the shoulders of many "giants" of scientific research, the humanities, and philosophy. Newton may have adapted his phrase from a line in Robert Burton's 1621 chrestomathy *The Anatomy of Melancholy* which states, "Pygmies placed on the shoulders of giants see more than the giants themselves." I can think of no better way to end this book than by referring to giants and pygmies, two extremes of the plasticity and wondrous diversity of the patterns of human growth.

Glossary

adolescent growth spurt the rapid and intense increase in the rate of growth in height and weight that occurs during the adolescent stage of the human life cycle.

adolescent stage a phase in the human life cycle, covering the five to eight years after the onset of puberty. The adolescent phase is characterized by a growth spurt in height and weight, near completion of permanent tooth eruption, development of secondary sexual characteristics, sociosexual maturation, intensification of interest and practice in adult social, economic, and sexual activities.

adolescent sterility a physiologic state of pubertal girls that begins after menarche and lasts until ovulatory cycles are established.

adrenarche the onset of secretion of androgen hormones from the adrenal gland, usually occurring at about six to eight years of age in most children.

adulthood the stage of the human life cycle that begins at about age 20 years. The prime of adulthood lasts until the end of child-bearing years (late forties) and is a time of homeostasis in physiology, behavior, and cognition. Old age and senescence mark the period of decline in the function of many body tissues or systems during adulthood. This later phase lasts from about the end of child-bearing years to death.

affect hunger defined by Goldschmidt (2006) as " . . . the urge to get expressions of affection from others." It is the motivating force for human sociality.

age-graded play group a social group of children and juveniles in which the older individuals provide basic caretaker behavior and enculturation for the younger individuals. The play group frees adults for subsistence activities and other adult behaviors.

allantosis a membranous sac that develops from the posterior part of the alimentary canal and which serves as a repository for wastes in the embryos of mammals, birds, and reptiles. It is important in the formation of the umbilical cord and placenta in mammals.

allometry differential rates of growth of parts of the body relative to the growth of the body as a whole.

allostatic load the cost of chronic exposure to elevated or fluctuating endocrine or neural responses resulting from chronic or repeated challenges that the individual experiences as stressful.

anthropogeny the investigation of the origin of man (humans) Oxford English Dictionary, 2006. First used in 1839 edition of Hooper's Medical Dictionary and defined as "the study of the generation of man."

anthropometry the scientific measurement of the human body.

apoptosis programmed cell death, a mechanism that allows cells to self-destruct when stimulated by the appropriate trigger. Cell deaths appear to be programmed by gene-environment interactions, and are initiated for various reasons, such as when a cell is no longer needed within the body or when it becomes a threat to the health of the organism.

attachment behavior the set of physical and psychological cues and responses that bond an infant with one of its caretakers.

Australopithecus genus of early hominids from eastern and southern Africa, dating to between four to one million years before present.

autocrine action a process by which a hormone-like substance has its biological effect on the cells that produce the substance.

auxology the study of biological growth.

biacromial breadth the linear distance between the shoulders, measured between the most lateral points of the acromial processes of the scapula.

biocultural referring to a recurring interaction between the biology of human development and the sociocultural environment. Not only does the latter influence the former, but human developmental biology modifies social and cultural processes as well.

biocultural reproduction the set of marriage and kinship-based rules for extra-maternal cooperation in the production, feeding, and care of offspring.

biological evolution is the continuous process of genetic adaptation of organisms to their environments.

biological standard of living a measure based on health, nutritional status, and longevity of living and past populations. Often summarized by the mean height of a population or group of people within a population.

body composition the make-up of the body in terms of the absolute and relative amounts of adipose tissue, muscle mass, skeletal mass, internal organs, and other tissues.

body mass index (BMI) a mathematical indicator of the relative weight-for-height of a person. BMI is calculated as [weight in kilograms / (height in meters)^2]. Higher BMI scores indicate that an individual has relatively more weight-for-height than a person with a lower score. In the general population higher BMI scores usually indicate more body fatness.

calcification the process of mineral deposition, usually calcium, in tissues of the body. Regular sites of calcification are the skeleton and the dentition.

catch-up growth the rapid increase in growth velocity following recovery from disease or refeeding after short-term starvation.

cell signaling biochemical interactions within and between cells that provide information about environmental conditions, such as energy balance, and nutrient availability, and temperature. See entries for autocrine, paracrine, and endocrine action for different categories of cell signaling.

childhood phase a stage in the human life cycle that occurs between the end of infancy and the start of the juvenile growth period (about the ages 2–10 years). Children are weaned from breast-feeding (or bottle feeding) but must be provided specially prepared foods and require intensive care by older individuals. Childhood is characterized by relatively rapid neurological development and slow physical growth and development.

chorionic placenta a type of placenta in which there is a direct connection between the chorion and the fetus via the umbilical cord. The chorionic placenta is found in rodents, monkeys, apes, and humans.

chorionic-allantoic placenta a type of placenta in which parts of the surface of the allantosis fuse with the chorion (a membrane surrounding the fetus composed of maternal and embryonic tissues). The chorionic-allantoic placenta is found in most orders of mammals, including Artiodactyla, Proboscidians, Carnivora, and others.

community effects on growth an hypothesis that there are influences on the attainment of final height, weight, body composition, and body proportions which arise from the bio-social-psychological proximity of members within a social network. Two of the major factors regulating community effects are socioeconomic status and ego motivation which are transduced in the brain and influence hormonal production.

complementary feeding the provision of nutritionally rich and relatively sterile combinations of foods acquired and processed by caregivers and fed to breastfed infants and toddlers after about six months of age.

congenital states or conditions that exist in the individual at or prior to birth. The term is usually used to refer to hereditary or inborn medical conditions that are most often harmful.

cooperative breeders species of vertebrates, especially birds and mammals, in which only one or a few adults of each sex breed and all the nonbreeding adults contribute time and effort toward the feeding and care of the breeders and their offspring.

cross-sectional analysis measurement on a single occasion of individuals grouped by age and sex, and sometimes other characteristics.

cultural animal a phrase describing the human species, meaning that humans possess all of the potentials and limitations of any living creature, but also add a cultural trilogy of: (1) dependence on technology; (2) codified social institutions, such as kinship and marriage; and (3) ideology.

cultural inheritance influences on the development of offspring phenotypes by the interaction of its genotype and the physical and behavioral phenotypes of family and social group members.

cultural transmission development of offspring phenotypes by interaction with the behavioral phenotypes of its parents and others of an older generation (vertical cultural transmission) and behavioral phenotypes of siblings, friends, and others of the same generation (horizontal cultural transmission). "Behavioral" is defined in a wide sense, as the ways in which an individual responds to a situation or stimulus and the way in which an individual acts or conducts themself, especially towards others.

deoxyribonucleic acid (DNA) one of the class of complex molecules called nucleic acids. DNA is found in the nucleus of virtually all living cells. DNA contains the genetic code needed for a cell to produce the proteins it requires to perform its function.

development progression of changes from undifferentiated or immature state to a highly organized, specialized, or mature state.

diaphysis the shaft of a long bone.

differential fertility one of the two fundamental mechanisms of natural selection (the other being differential mortality), referring to variation in the reproductive success of mature organisms.

differential mortality one of the two fundamental mechanisms of natural selection (the other being differential mortality), referring to the death of some individuals of a population prior to their reproductive maturation.

distance curve a graphic representation of the amount of growth achieved by an individual over time.

dizygotic twins co-gestated siblings that result from two independent fertilizations. Such twins are no more alike genetically than ordinary brothers and sisters. Also referred to as "fraternal twins."

ecology the relationship that an individual organism, or group of individuals of a species, has with its physical, biological, and social environment.

effect size indicates the standardized difference between two means. It is widely used in meta-analysis and expressed as Cohen's **d** for the comparison between two means. Large effects sizes – those of biological importance – are **d** = 0.60 or greater.

embryo stage of pre-natal development lasting from the 2nd to 10th week following fertilization, characterized by the rapid differentiation of tissues and the formation of organs.

endocast a mold, or cast, of the interior of the skull which may be used to estimate brain size and shape.

endocrine action the secretion of a hormone from its tissue of origin and its distribution throughout the body via the blood stream to its site of action.

endocrinology the study of hormones, their origins and actions.

endosteal the thin layer of cells lining the medullary cavity (inner surface) of a bone.

endotheliochorial placenta a sub-type of the chorionic placenta in which the uterine-epithelium barrier is eliminated. Found in rodents.

epidemiology the study of the causes and transmission of disease.

epigenetics the study of changes in organisms caused by modification of gene expression (see entry for transcription) rather than alteration of the genetic code itself.

epiphysis an ossification center of a long bone, separated from the shaft of the bone by cartilage.

epitheliochorial placenta a sub-type of the chorionic placenta in which a separation between the tissues of the chorion and the epithelial lining of the uterus is maintained. Found in the lemurs and lorises (the strepsirhine primates).

essential nutrient a nutrient that cannot be manufactured by the human body from simpler elements and thus must be supplied from the diet.

expanding tissues those tissues or cells that retain their mitotic potential even in the differentiated state. These tissues can increase in size and mass by cell division of all their cells. Examples include the liver, kidney, and the endocrine glands.

feedback control a method used for the regulation of biological activity in an organism based on the flow of information between parts of the organism. An example is negative feedback control in the endocrine system, in which rising levels of a hormone in the blood stream result in a lowering of the level of stimulation of cells that produce that hormone.

fetus stage of pre-natal development lasting from 10th week following fertilization to birth.

fibroblast cell a type of undifferentiated cell which may differentiate into bone, adipose, or other types of tissue.

fictive kin a term used by anthropologists to describe forms of kinship or family ties that are based on neither consanguineal ties ("blood" – i.e., genetic relationship) nor affinal ties ("by marriage").

genetic potential a popular, but incorrect, concept that every human being has genetically determined limits to many aspects of their phenotype.

genome the genetic material of an organism.

genotype the genetic constitution of an individual.

germinative cells undifferentiated cells, usually sequestered in well-defined regions within tissues, that give rise to the differentiated, specialized cells of mature tissues, organs, and systems in the body.

gonadarche maturation of the gonads (testes or ovary) resulting in the secretion of gonadal hormones (androgens or estrogens).

growth quantitative increase in size or mass.

growth factors biochemical substances synthesized by specific cells within a wide variety of body tissues that have effects on growth, both independently and interactively with each other and with hormones.

growth plate region the site of formation of bone tissue in a growing long bone. The growth plate consists of highly ordered rows of cartilage cells; the row farthest removed from the bony shaft is a germinatve layer; it is responsible for cell replication and cartilage growth at the bone shaft. Over time the cartilage will be reformed into true bone tissue.

hemochorial placenta a sub-type of the chorionic placenta in which the cell walls of the maternal blood vessels break down and invade the chorion so that the chorion, and the fetal circulation, are directly bathed by maternal blood. Found in tarsiers, New World monkeys, Old World monkeys, apes, and humans (the haplorhine primates).

heritable/heritability a concept and method to hypothesize the relative contributions of the genome and the environment to the phenotypic expression of a physical or behavioral characteristic. For humans, heritability is influenced strongly by cultural transmission (see glossary entry).

heterochrony referring to several processes in biology that bring about evolutionary change between ancestral and descendant species by modifying characters present in the ancestor via changes in developmental timing.

homeobox genes a highly conserved sequence of 180 DNA base pairs that codes for a 60 amino acid segment of a protein. The homeobox DNA sequence is found in all eukaryote organisms so far examined.

homeostasis any self-regulating process by which biological systems tend to maintain stability while adjusting to conditions that are optimal for survival.

hominin living human beings and their extinct fossil ancestors characterized by habitual bipedal locomotion.

hominoid the group of the Primates that includes human beings, the apes, and their extinct ancestors.

homoiothermy self-regulation of a relatively constant body temperature.

homology likeness in the anatomy or behavior of different organisms due to an evolutionary differentiation from the same or a corresponding anatomical part or behavior of a remote ancestor.

hormone chemical substance secreted from a specific tissue, usually into the general blood circulation, where it travels to its site of action.

HOX genes a category of homeobox genes that encode transcription, that is, proteins that initiate and regulate the conversion of the DNA code to the RNA sequence that is used to make amino acid polypeptide chains.

hypermorphosis a type of heterochrony that extends one or more stages of growth and development of the descendant species beyond that of the ancestral species.

hyperplasia cellular growth by cell division (mitosis).

hypertrophy cellular growth by an increase of material within each cell.

hypothalamus an evolutionarily ancient midbrain structure, which in mammals provides a connection between the nervous system and the endocrine system – it is a neuroendocrine transducer. The human hypothalamus secretes hormones that stimulate or inhibit the production of other hormones by the pituitary which regulate growth and development.

hypoxia the lack of sufficient oxygen supplied to the tissues of the body. May be the result of disease or may be due to residence at high altitude (3,000 meters or more above sea level).

infant phase or infancy (of growth and development) a stage in the life cycle of all mammals. For human beings it lasts from the 2nd month after birth to end of lactation, usually by age 36. Human infancy is characterized by: (1) rapid growth velocity with a steep deceleration in velocity with time; (2) feeding by lactation; (3) deciduous tooth eruption; and (4) many developmental milestones in physiology, behavior, and cognition.

intensity of growth the amplitude and duration of a growth or development events.

intergenerational effect hypothesis the causal association between growth, development, and risk for disease of the current generation of people and the living conditions experienced by their maternal grandmothers and their mothers during the prenatal and postnal life of those women.

intracrine (action) the process whereby a hormone may act on the nucleus of the cell which manufactures the hormone.

juvenile phase (of growth and development) a stage in the life cycle of most social mammals, including all of the higher primates. The juvenile stage is defined as the time of life when an individual is no longer dependent on adults (parents) for survival, and prior to that individual's sexual maturation.

karyotype a description of the chromosomes of an organism in terms of their number, shape, and size.

kwashiorkor a disease of protein deficiency, characterized by failure to grow, wasting of the muscles, loss of appetite, irritability, changes in the hair and skin, and anemia.

lactation the secretion or formation of milk by the mammary glands, as well as the period during which the milk is the major food provided to the infant.

Ladino one of the major ethnic groups of Guatemala, defined as the cultural descendants of the Spanish conquerors of Guatemala, speak Spanish as their primary language, wear western-style clothing, and deny any Maya ancestry or heritage.

life cycle the stages of growth, development, and maturation from conception to death of any organism.

life history theory a field of biology concerned with the strategy an organism uses to allocate its energy toward growth, maintenance, reproduction, raising offspring to independence, and avoiding death. For a mammal, it is the strategy of when to be born, when to be weaned, how many and what type of pre-reproductive stages of development to pass through, when to reproduce, and when to die.

longitudinal data measurement of the same individual or group of individuals, repeated at regular intervals.

low birth weight a weight at birth of 2,500 grams or less for a neonate of normal gestation length (i.e., 37–40 weeks).

maturation the process and the state of reaching functional capacity in terms of biological, behaviroal, and cognitive capacities.

Maya one of the major ethnic groups of Guatemala, defined as the cultural descendants of the pre-Conquest Maya, speak a Maya language and Spanish, wear traditional non-Western clothing (especially during social and religious rituals), and claim Maya ancestry or heritage.

menarche the first menstrual period.

menopause the sudden or gradual cessation of the menstrual cycle subsequent to the loss of ovarian function.

meta-analysis a systematic review of a focused topic in the literature that provides a quantitative estimate for the effect of a treatment intervention or exposure. The results of a meta-analysis can be used to form treatment recommendations or to provide guidance in the design of future clinical trials.

mid-growth spurt a relatively small increase in the rate of growth in height that occurs in many children between the ages of six to eight years.

mid-parent height the average of the stature of the mother and the father. Used in formulas to predict the adult stature of offspring.

migrant people who move from one place of residence to another, especially from their place of birth to a geographically and socially new place of residence. This includes people who have migrated within the same country, such as rural-to-urban migrants.

migration the movement of people from place to place. In recent years, much of this migration has been from rural to urban areas, requiring substantial changes in the environment and lifestyle of the migrants.

mitosis cell division resulting in two "daughter" cells with the same genetic constitution as the original "parent" cell.

model a representation that displays the pattern, mode of structure, or formation of an object or organism.

monozygotic twins co-gestated siblings that result from a single fertilization and, therefore, share the same genotype. Also referred to as "identical twins."

multilevel selection model an explanation for the evolution of social behavior that posits that natural and sexual selection not only act on individuals but can act (simultaneously) on multiple levels of biological organization, including cells, tissues, individuals, and groups of individuals. In the last case, this is also called group selection theory.

natural selection the process by which environmental constraints lead to varying degrees of reproductive success among individuals of a population of organisms. The individuals must vary in terms of genetically inherited characteristics. Natural selection determines the course of evolutionary change by maintaining favorable genotypes and phenotypes in a constant environment (stabilizing selection) or improving adaptation in a direction appropriate to environmental changes (directional selection). Charles Darwin and Alfred Wallace first proposed this concept in 1858.

negative secular trend a decrease in the mean size (height, weight, leg length, etc.) of the individuals of a population from one generation to the next. Negative secular trends usually indicate a deterioration in the quality of the biocultural environment for human development.

neocortex region of the mammalian brain associated with "higher" level motor and sensory activities and the integration of these activities into complex patterns of behavior.

neonate and neonatal phase a stage in the human life cycle lasting from birth to 28 days after birth.

neoteny a type of heterochrony that results in the retention of infantile or juvenile traits into adulthood. This is achieved by having sexual maturation take place while the individual is still in a pre-adult stage of phenotypic development.

norm of reaction in ecology and genetics, describes the pattern of phenotypic expression of a single genotype across a range of environments, also called a reaction norm.

ontogeny the process of growth and development of individuals of a species.

organogenesis the formation of body organs and systems during the first trimester of pre-natal life.

ossification the process of bone formation in skeletal tissue.

paleoecology the study of extinct forms of life and their relations with the environment (e.g., types of foods eaten, requirements for reproduction).

paleontology the study of extinct forms of life, usually as represented by fossilized remains.

paracrine action the process by which a hormonelike substance has its biological effect on nearby cells within the same tissue as the cells that produce the substance.

parental investment the allocation of resources, such as time or energy, to offspring that occurs at some cost to the parents.

peak growth velocity the maximum rate of growth in height, weight, etc. achieved during the adolescent growth spurt.

pelvic inlet the bony opening of the birth canal through which the fetus must pass during parturition.

periosteal the dense fibrous membrane covering the outer surface of bones, except at the joints, and serving as an attachment for muscles and tendons.

phenotype the physical or behavioral appearance of an individual, resulting from the interaction of the genes inherited at the moment of conception and the environment during growth and development.

photogrammetry a method of measuring the human body by taking one or more photographs of an individual posed in particular positions. Photogammetry is often used in somatotyping.

phylogeny the evolutionary history of a species.

pituitary an endocrine gland of vertebrate animals located at the base of the brain, below the hypothalamus to which it is connected via blood vessels and nerves. The hormones secreted by the pituitary stimulate and control the functioning of many other endocrine glands in the body. (y)

placenta an organ of some mammals composed of fetal and maternal tissues that transfers nutrients and oxygen from the mother's blood circulation to the fetus and fetal wastes to the mother's blood circulation for disposal.

plastic the ability of an organism to modify its biology or behavior to respond to changes in the environment, particularly when these changes are stressful.

plasticity the concept and process of modifiable change during the development of the phenotype in response to variations in the quality and quantity of environmental factors required for life. Such variations produce many of the differences in growth observed between individuals or groups of people.

preformation erroneous notion that the pre-natal human body is essentially adultlike in form.

prematurity a state at birth for human neonates who are born prior to 37 weeks of gestation.

psychosocial short stature a type of growth retardation produced by a negative physical and/or emotional environment for growth.

puberty an event of short duration (days or a few weeks) that marks the reactivation of the central nervous system regulation of sexual development. The onset of puberty is accompanied by a dramatic increase in secretion of sex hormones. In social mammals, including humans, puberty occurs at end of the juvenile stage.

regulatory genome noncoding DNA, micro-RNAs and other elements of the genome that initiate and terminate the action of structural genes. Regulatory genes are important for the control of growth in terms of size, shape, and the timing and duration of each stage of the life cycle, see HOX genes.

remodeling of bone the process that maintains the characteristic shape and function of a bone as that bone grows in size. Remodeling is achieved by destroying or creating new bone cells as a bone grows.

renewing tissues those tissues, or cells, of the body that are replaced by a two-step process: (1) the mitotic division of germinative cells; and (2) the differentiation of some of these newly divided cells into mature tissues. Examples of renewing tissues are the blood and the skin cells of the epidermis.

reserve capacity hypothesis those somatic, cognitive, and emotional resources that exceed the minimum required for sustaining life and allowing reproduction.

secondary sexual characteristics as opposed to the primary sexual characteristics of ovaries or testes, these are physical traits associated with the onset of sexual maturation, including the development of facial hair and muscularity in boys and the development of the breast and adult fat distribution in girls. Secondary sexual characteristics may also be behavioral, relating to culture-specific ways of acting and speaking that are defined as "female" or "male."

secular trend the process that results in a change in the mean size or shape of individuals of a population from one generation to the next. Such trends can be positive (increasing size) or negative (decreasing size).

sedente a person living in their geographic region of birth; a non-migrating individual.

senescence the period of the adult phase of the life cycle characterized by a decline in the function of many body tissues or systems. Senescence usually begins after the end of child-bearing years and lasts until death.

sexual dimorphism differences between the sexes in physical appearance, behavioral performance, and psychological characteristics.

skeletal age and skeletal maturation a measure of biological maturation (as distinguished from chronological age) based on stages of formation of the bones.

socioeconomic status (SES) an indicator, often defined by measures of occupation, education, and prestige of an adult, a young person's parents, or the head of household, used as a proxy for the general quality of the environment for growth, health, and well-being of an individual, family, or social group.

somatotyping a method of classifying the human physique based on external, usually visual, assessment of body shape. The goal of somatotyping is to find associations between types of physiques and physiological function, habitual behavior, or risk for disease.

static tissues those tissues or cells that are incapable of growth by hyperplasia once they have differentiated from precursor germinative cells. Examples are nerve cells and striated muscle.

statistical hack a mathematical technique to simplify and often misuse data analysis to find patterns in data that can be presented as statistically significant when in fact there is no real underlying effect.

strategic growth adjustments to body size or rate of growth that are associated with position in the social hierarchy.

strategic growth adjustments changes in growth rates to achieve larger body size or grow to adulthood at a faster rate to achieve social and reproductive dominance. Most often observed in cooperatively breeding mammal species where reproduction is virtually limited to the most dominant female and male individuals. Also observed in human social groups as part of biocultural reproduction.

structural genes elements of the genome that code for specific sequences of amino acids (polypeptide chains) produced by the cells of the body.

subcutaneous fat the layer, or compartment, of adipose tissue that lies just under the skin.

synergistic (action) the interaction of two or more agents or forces so that their combined effect is greater than the sum of their individual effects.

tempo of growth based on a metaphor from classical music, this phrase refers to the pace at which individuals pass through the stages of growth and development. Some humans grow rapidly

and/or mature early (*allegro*), while others grow slowly or are late maturers (*lento*). The tempo of growth is, generally, unrelated to amount of growth that an individual will achieve.

theory of mind or shared intentionality the ability to infer the intentions of others and the disposition to align these intentions with one's own physical and emotional states.

traditional societies an anthropological phrase referring to pre-industrialized and, non-Western societies, such as hunters and gatherers, horticulturists, pastoralists and farmers depending on animal power.

transcription factor genomic transcription is the process whereby DNA sequences are translated (transcribed) into RNA molecules. Transcription factors are proteins that help turn specific genes "on" or "off" by binding to nearby DNA. Activator proteins boost a gene's transcription, repressor proteins decrease transcription (see entry for cell signaling).

transformation grids a method developed by D'Arcy Thompson (based on drawings of the artist Albrecht Düerer) to describe two dimensional changes in growth and form, both within and between species of organisms.

trimesters (of pregnancy) the division of the nine calendar months of human pregnancy into three, three-month periods. Usually called first, second, and third trimester.

velocity curve a graphic representation of the rate of growth of an individual over time.

viviparity giving birth to living offspring that develop within the mother's body.

weaning the termination of breast-feeding.

yolk sac a membranous sac attached to an embryo, providing early nourishment in the form of yolk in bony fishes, sharks, reptiles, birds, and primitive mammals and functioning as the circulatory system of the human embryo before internal circulation begins.

yolk sac placenta a type of placenta found in some marsupials and rabbits, in which blood vessels connect the yolk sac with the uterine wall.

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