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Do alexithymic individuals avoid their feelings? Experiential avoidance mediates the association between alexithymia, psychosomatic, and depressive symptoms in a community and a clinical sample

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Abstract

Objective: Alexithymia is defined as the trait associated with difficulty in identifying and describing feelings as well as poor fantasy and imagery. While alexithymia is related to psychopathology in general, it has been associated with increased reporting of medically unexplained symptoms and depression in particular. This study attempts to assess the extent to which alexithymia represents a learned, avoidant coping strategy against unwanted emotions. In this way the study aims to identify a potential mechanism that may elucidate the relationship between alexithymia and psychological symptoms.

Method: Alexithymia is examined in two different samples, students from two universities in Cyprus and intensive outpatients/residents in an American anxiety disorder treatment program. We examine whether alexithymia predicts psychosomatic and depressive symptoms respectively through the mediating role of experiential avoidance, a coping mechanism believed to be reinforced because of the immediate relief it provides.

Results: Experiential avoidance was found to correlate strongly with alexithymia, especially its difficulty in identifying feelings factor, while the mediation hypothesis was supported in all models tested. Furthermore, results from the clinical sample suggest that clinical improvement in depression was associated with a decrease in alexithymia, especially difficulty in identifying feelings, mediated by decreased experiential avoidance.

Conclusions: Alexithymia, and more specifically its difficulty in identifying feelings aspect, may be a learned behavior used to avoid unwanted emotions. This avoidant behavior may form the link between alexithymia and psychopathology. Implications for alexithymia theory and treatment are discussed.

1. Introduction

Alexithymia, a term used by Sifneos [1] to describe patients with a marked difficulty in verbally describing their feelings, has stimulated a plethora of research over the last several decades. It is now viewed as a trait found on a continuum in the population [2,3] associated with difficulties in identifying and describing emotions, poor imagery and fantasy life and externally-oriented thinking [4–6].

Alexithymia was initially described as a characteristic of psychosomatic patients [1], and its link with medically unexplained symptoms has been multiply replicated [e.g. 7–9]. However, subsequent studies [e.g. 8], indicated that alexithymia is a correlate of psychological disorders in general; it is strongly associated with symptoms of depression [10,11], and may share its genetic influences [12]. Furthermore, changes in alexithymia levels predict decreases in depressive symptoms [10,13]. Alexithymia is also associated with anxiety disorders and poor physical health outcomes [5,14], all of which may reflect the difficulties of high alexithymia individuals in processing and expressing emotion. However, despite dozens of studies documenting the association between alexithymia and poor physical and psychological health, little is understood about the

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mechanisms that specifically account for this association [see 15 for a review].

In order to address this mechanism, a clearer understanding of the etiology of alexithymia is required, although this has not been as widely studied as its phenomenology. Extant research suggests that both genetic and environmental factors play a role in the development and maintenance of alexithymia (e.g. [15]; it tends to be encountered more often in certain subgroups, such as middle aged males and individuals low in education, which suggests that it may be influenced by contextual factors [16,17]. Twin heritability studies have also noted that while some aspects of the characteristic may be genetic in nature, other core features, such as difficulty in identifying emotions, seem to be affected by environmental influences [12,18]. Other researchers see alexithymia as a learned behavior and have proposed that certain aspects (e.g. difficulty in identifying and describing feelings) serve as coping strategies that function similarly to suppression and dissociation [19,20] to dampen unpleasant emotions in the face of severe stress or trauma [21–23]. Badura [21] further supports that the trait may be a symptom or correlate of post-traumatic stress. Thus, it appears that more research is warranted to verify whether alexithymia is temperamental in nature or an emotion regulation strategy acquired to cope with stress.

To comprehend alexithymic deficits in emotion processing, an understanding of how emotional information is typically processed may be helpful. According to the Bioinformational Theory of Emotion [24], emotional memories are stored in associative networks comprised of descriptive, meaning-related and response-related information. Activation of any of these components should activate other parts of the network to some degree. Alexithymic individuals appear to have less cohesive memory networks for emotion in comparison to non-alexithymic individuals [25–27]. Specifically, multiple studies have identified a "decoupling" in alexithymia between subjective emotional experience and emotion response systems, such as autonomic physiological reactivity and facial communication [28]. This decoupling may be a hallmark of alexithymia, which accounts for the tendency of these individuals to misattribute interoceptive sensations to illness instead of emotion [e.g., 29,30]. Recent neuro-imaging data support this notion by pointing to poor communication between brain regions involved in processing different aspects of emotion [31–33]. This difficulty could represent an inherent deficit [e.g. in symbolic abstraction; 34] or may indicate a learned behavior to down-regulate intense and unwanted emotions.

The current study addresses the hypothesis that alexithymic difficulties reflect a learned tendency of individuals high in alexithymia to avoid unwanted internal experiences such as particularly unpleasant and highly arousing emotions [25]. We propose that the emotion identification and description deficit of alexithymia is in fact an effort (deliberate or not) to avoid experiencing unwanted affect and that this mechanism is what ultimately predicts the development of mental and physical health problems. This proposition links alexithymia with the construct of experiential avoidance (EA), i.e. the tendency of some individuals to avoid aversive bodily sensations, emotions, thoughts, memories and other internal events by altering their form and frequency and contact with the triggers of these experiences [35,36]. EA, like alexithymia has been found to be particularly pathogenic and may be a common risk factor for a range of disorders including depression, somatization and poor perceived health [37]. The proposed model is a meditational one, in that it is suggested that alexithymia per se has no direct effects on symptoms, but that its effects are indirect and explained by the presence of EA.

EA is reinforced and maintained by the immediate relief it provides from unpleasant experiences, even though it ultimately intensifies and sustains them [38]. EA overlaps with other pathogenic constructs such as avoidant coping, thought suppression, stress intolerance and anxiety sensitivity [39]. Previous studies have documented that EA mediates the effects of emotion regulation strategies on mood and distress [38,40], while findings from an inpatient sample suggested mediation by EA of the link between alexithymia and emotion regulation problems [41].

The current study suggests that the difficulty of individuals high in alexithymic traits in recognizing interoceptive cues as signs of emotion and in identifying and describing their feelings, may have become acquired as a learned coping approach involving avoidance of unpleasant affect. This tendency may have become reinforced through its immediate success at relieving distress [35], especially among individuals like males, who are discouraged in many cultures from expressing such "powerless" emotions and seeking support [42]. In the long term, the avoidance of experiencing, describing and processing emotion may inhibit the development of appropriate emotion regulation skills, prevent exposure and extinction of negative affect and increase fear of internal experiences [35,43,44]. Therefore, this study addresses the hypothesis that alexithymia is related to EA and that in fact, EA mediates and explains the association between alexithymia and psychological distress, as manifested by psychosomatic symptoms and depression. To our knowledge, this is the first study to address this hypothesis, which shows promise at providing some explanation (rather than mere description) of the emotion processing difficulties of alexithymic individuals.

1.1. Current investigation

This investigation uses two studies involving a student and a clinical sample. The aim concerns three hypotheses: 1) Experiential avoidance mediates the association between alexithymia and psychosomatic symptoms. 2) EA mediates the association between alexithymia and symptoms of depression. 3) Clinical participants present reductions in alexithymia, EA and depressive symptoms from baseline to post-treatment assessment, and decreased EA mediates the impact of reduced alexithymia on depression improvement.

Study 1 examines the hypothesis that EA mediates the association between alexithymia and psychosomatic symptoms (hypothesis 1) using a student sample. A positive correlation is expected between alexithymia, EA and somatic symptom reporting, along with the predicted mediation effect. Since it is generally believed that it is the difficulty of alexithymic individuals to recognize their somatic sensations as emotion-based that make them vulnerable to psychopathology, stronger associations are expected between somatic symptoms and the difficulty in identifying feelings and difficulty in describing feelings factors of alexithymia. Study 2 replicates and extends the mediating model using depression, as measured by the Beck Depression Inventory-II (BDI-II) as perhaps the most common psychological outcome related to alexithymia and as a frequent common symptom encountered across psychological disorders, either as a primary diagnosis or as a comborbid presentation [45–47]. Study 2, allows for a replication of the hypothesized model among individuals with higher psychopathology and psychological distress (enough to meet a clinical diagnosis). The specific primary diagnosis was not of relevance to this investigation, which aimed primarily on replicating the mediation model on a population suffering from clinical levels of distress and dysfunction, and for whom depressive symptoms were a common problem as they are in most clinical populations. Hypothesis 2 suggests that EA mediates the association between alexithymia and depression.

Furthermore, because the literature suggests that reductions in alexithymia through psychological interventions are associated with symptom improvement [48,49], the current study addresses the question of whether reductions in depression as a result of Cognitive Behavioral Therapy (CBT) are mediated by reductions in EA.

The use of mediation with cross-sectional data has been criticized by some authors on the grounds that the temporal order of variables cannot be easily established, and it has been therefore suggested that temporal order should then be based on when the constructs appear developmentally [47]. In this study, alexithymia is considered to be trait-like, often seen as partially heritable [12] and so it is entered as a predictor. EA instead is conceptualized as a learned pattern of behavior, acquired through reinforcement [39], and is therefore seen as appearing later in life. Finally, psychosomatic symptoms and depression typically present in adolescence and adulthood and are therefore entered last as the dependent variables.

2. Study 1

2.1. Method

2.1.1. Participants

Participants were 205 students (43 male, 162 female; M_{age} = 21, SD = 2.87) from two universities in Cyprus, who received course credit or a small monetary reimbursement and provided informed consent. The study was approved by the Cyprus National Bioethics Committee as part of a larger project on the epidemiology and correlates of anxiety disorders.

2.1.2. Measures

2.1.2.1. The Toronto Alexithymia Scale-20 [TAS-20; 50]. The TAS-20 is a twenty item self-report questionnaire that assesses levels of alexithymia on a 5-point Likert scale. It is comprised of three factors, the means and SDs of which for the current study are as follows: difficulty in identifying feelings (DIF; M = 15.19, SD = 4.65), difficulty in describing feelings (DDF; M = 13.53, SD = 3.87) and externally-oriented thinking (EOT; M = 21.01, SD = 15.82). Higher TAS-20 total scores (M = 52.84, SD = 12.05) indicate higher levels of alexithymia. Mean alexithymia in this study was low and similar to levels found in other studies of student samples [22,51]. The scale was adapted and validated into Greek by Anagnostopoulou and Kossieoglou [see 26]. The Greek TAS-20 showed good reliabilities for most scales (total α = .79, DDF α = .79, DIF α = .74, EOT α = .58) [52]. Internal reliabilities for the present sample are similar to other studies [e.g. 48,53], except that of DDF, which is somewhat lower but acceptable: for TAS-20 α = .83, DDF α = .66, DIF α = .78, EOT α = .73.

2.1.2.2. Acceptance and Action Questionnaire II [AAQ-II; [54]. EA was assessed using the Greek adaptation of the Acceptance and Action Questionnaire II. Its 7 items measure EA on a 7-point Likert scale, yielding scores from 7 to 49 (mean for this study = 22.85, SD = 8.39). The AAQ-II has shown good psychometric properties and is highly reliable, with Cronbach's alphas > .80 in all language translations [55], as well as in this study (α = .87).

2.1.2.3. Patient Health Questionnaire-15 [PHQ-15; [56]. Psychosomatic symptom reporting was assessed with a modified version of the somatic symptoms scale of the PHQ-15, which was designed to assess psychosomatic symptoms typically reported by primary care patients [57]. This scale is composed of the original PHQ-15 items, but was extended to cover 10 additional symptoms, including intestinal gasses and indigestion, vomiting, numbness and tingling, weakness, lump in the throat, dry mouth, inability to lift the feet, flushes or shiver, feelings of sickness and amnesia. These items were drawn from the Brief Symptom Inventory [BSI; 58]. This extended version of the PHQ-15 assesses the severity of 25 symptoms on a 3-point scale ('not disturbed at all' to 'disturbed very much'), during the last month. Possible scores range from 25 to 75 (mean for this study = 23.53, SD = 15.82; Cronbach's α = .90). Factor analysis on this study's sample (available upon request) shows that all items load on a single factor.

2.2. Results

2.2.1. Correlations among measures

Bivariate correlations (Table 1) were carried out in order to identify relationships between alexithymia, EA and somatic symptoms and to rule out multicollinearity between the variables. Results indicate that multicollinearity is not a

Table 1
Pearson correlation coefficients between TAS-20 total and factor scores, EA and somatic symptoms in study 1.

	DDF	DIF	EOT	EA (AAQ-II)	PHQ-15
TAS-20 total	.780**	.792**	.771**	.558**	.265**
DDF		.603**	.375**	.483**	.258**
DIF			.313**	.722**	.410**
EOT				.174*	.015
EA (AAQ-II)					.453**

* $p < .05$.
** $p < .01$.

concern, as all correlations are below .80 and the highest correlations are among the TAS-20 factors. TAS-20 total score and factor scores are positively correlated with EA and somatic symptoms, as expected, while EA shows a particularly strong association with DIF.

2.2.2. Prediction of somatic symptoms from TAS-20 factors and EA

A hierarchical regression (Table 2) was conducted to examine the degree to which alexithymia and EA predict somatic symptom reporting. Each TAS-20 factor was entered as a separate step in the model, in the order of EOT, DDF and DIF. EA was entered in the last step. Parametric assumptions for this model are met, including no multicollinearity, homoscedasticity, and independence and normality of errors. Significant increases in the variance of somatic symptoms explained appear at all steps. In the final step, with all variables entered in the model, the total variance in somatic symptoms explained is 23%, with EA and DIF significantly predicting symptoms, $F(4,200) = 14.942$, $p < .001$.

2.2.3. Mediation model

The proposed mediation model was tested using PROCESS [59], a versatile modeling tool for observed variable mediation and moderation. This approach, based on bootstrapping, is considered as an advancement over previous methods of assessing mediation because of increased reliability of findings [59]. The analyses were based on 5000 bootstrapped samples using bias-corrected 95% confidence intervals. The three TAS factors were also included as predictors in three separate follow-up models in order to identify the sub-dimension of alexithymia that is most predictive of symptoms through EA.

The main model is presented in Fig. 1. The indirect effect of the TAS-20 total score on somatic symptoms through EA suggests complete mediation of the link between alexithymia and somatic symptoms by EA ($B = .32$, $SE = .06$, $\beta = .25$, $p < .001$, BCa *CI* [.21, .45]), and represents a large effect size ($\kappa^2 = .22$, BCa *CI* [.14, .30]) [60].

Focusing on TAS-20 factors, the first follow-up model showed reduced significance of the direct effect of DDF on somatic symptoms compared to the total effect (c: $B = 1.05$, $SE = .28$, $\beta = .26$, $p < .001$) after controlling for the effect of EA as the mediator (c': $B = .21$, $SE = .29$, $\beta = .05$, $p = .48$). The indirect effect of DDF on somatic symptoms through EA ($B = .84$, $SE = .16$, $\beta = .21$, $p < .001$, BCa *CI* [.58, 1.18]), indicates a medium to large effect size ($\kappa^2 = .19$, BCa *CI* [.13, .26]). Similarly, the significance of the direct effect of DIF on somatic symptoms is reduced compared to the total effect (c: $B = 1.40$, $SE = .22$, $\beta = .41$, $p < .001$) after controlling for the effect of EA as the mediator (c': $B = .59$, $SE = .31$, $\beta = .17$, $p = .06$). The indirect effect of DIF on somatic symptoms ($B = .81$, $SE = .23$, $\beta = .24$, $p < .001$, BCa *CI* [.38, 1.27]) is also of medium to large effect size ($\kappa^2 = .18$, BCa *CI* [.08, .28]). The mediating role of EA between EOT and symptoms is not significant. In sum, results support hypothesis 1, that EA fully mediates the association between alexithymia, particularly DIF and DDF, and somatic symptoms.

Table 2
Predictors of PHQ somatic symptoms in study 1.

	ΔR^2	*B*	*SE*	β	95% *CI*
Step 1	.000				
EOT		.038	.182	.015	−.320, .396
Step 2	.074**				
EOT		−.247	.189	−.096	−.620, .125
DDF		1.200	.298	.294**	.612, 1.788
Step 3	.110**				
EOT		−.356	.179	−.138*	−.709, −.003
DDF		.235	.336	.058	−.428, .899
DIF		1.423	.273	.418**	.884, 1.962
Step 4	.046**				
EOT		−.293	.175	−.113	−.639, .053
DDF		.110	.330	.027	−.540, .760
DIF		.693	.340	.204*	.022, 1.364
EA		.589	.171	.312**	.252, .925

* $p < .05$.
** $p < .01$.

3. Study 2

Study 2 aims to extend these findings in a clinical population, with a different outcome variable known to be associated with alexithymia (i.e. depression symptoms), and in a different culture. Depression was selected as the outcome variable, because it is a common symptom of distress, found in many kinds of psychological disorders, and a very common comorbid diagnosis, reaching a prevalence of up to 50%, for most disorder categories including OCD and other anxiety conditions [e.g. 46,61]. Because data were collected from patients both at the intake and post-treatment, longitudinal effects are taken into consideration (to the degree permitted by the sample size), which help address a limitation of study 1, namely its cross-sectional nature. Furthermore, study 2 examines the effectiveness of CBT in reducing alexithymia and EA and their effects on depressive symptoms.

3.1. Method

3.1.1. Participants

Participants were 163 patients (63% male; $M_{age} = 29.54$, $SD = 1.16$; 81.5% Caucasian) treated in the residential and

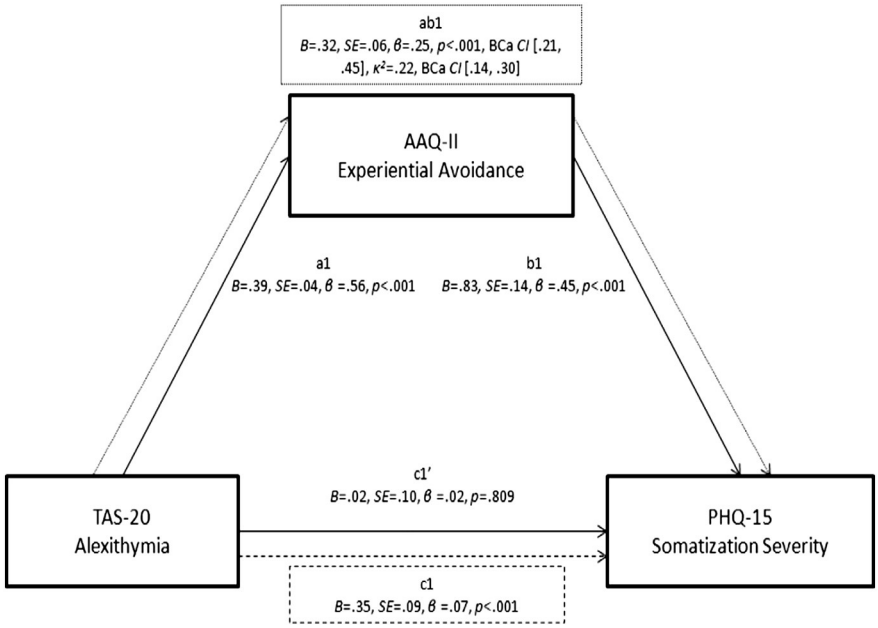

Fig. 1. The mediating effect of EA in the relationship between alexithymia and somatization severity (c1 = total effect, c1' = direct effect, ab1 = indirect effect).

intensive outpatient programs of the Houston OCD Program in Texas, USA. Patients completed an intensive course of CBT involving daily exposure and response prevention (ERP) in addition to psycho-educational groups in a milieu setting. The mean length of stay in the program is 46.43 days (*SD* = 3.58). Most participants have obsessive compulsive disorder (71.8%) as their primary diagnosis, followed by a range of other anxiety and mood disorders including generalized anxiety disorder (7.1%), social anxiety disorder (6.4%), panic disorder with or without agoraphobia (4.5%), body dysmorphic disorder (3.8%). In this patient group the diagnosis of major depression as primary or as one of their secondary diagnoses reached 49.4%, while an additional 12.4% received dysthymic disorder as one of their diagnoses and 12.9% were diagnosed with major depressive disorder in remission. The mean depression score at intake on the Beck Depression Inventory is 24.63, (SD = 12.73) indicating moderate depression. Study 2 received archival approval from Southern Illinois University's Human Subjects Committee as part of a larger project. Consent for participation was obtained at admission, and patients were informed in writing that it could be voluntarily removed at any point.

3.1.2. Measures

At both time 1 (intake; T1) and time 2 (post-treatment; T2) participants completed a questionnaire package, which included the TAS-20, BDI-II, and AAQ-II. The TAS-20 and AAQ-II were used in study 1 and were described earlier in this paper. To ensure that changes in the composition of the sample due to dropout did not affect the mediation model, this was tested both at T1 and T2 based on the existing participants at each phase.[1] Levels of alexithymia at T1 were: TAS-20 total score *M* = 55.53, *SD* = 10.60; DDF *M* = 13.78, *SD* = 3.62; DIF *M* = 18.64, *SD* = 6.37; EOT *M* = 23.11, *SD* = 4.61, and were similar to those found in other studies on clinical populations with depression and anxiety diagnoses [e.g. 62].

3.1.2.1. Beck Depression Inventory-II (BDI-II; [63]).

Depression symptoms were assessed with the Beck Depression Inventory-II, which measures the frequency of 21 depression symptoms over the last two weeks on a 3-point scale. The BDI-II has good reliability, construct, discriminant, criterion and convergent validity [e.g. 63–66]. The internal consistency of the scale as provided in the manual [63] was similar to that of the current study (α = .92). Means, SDs and Cronbach's α for *T*1 and T2 are presented in Table 3.

3.2. Results

3.2.1. Change from time 1 to time 2

Repeated measures ANOVAs were conducted to examine changes in alexithymia, EA and depression symptoms from

[1] The attrition noted from T1 to T2 is due to the fact that this was a naturalistic archival study therefore data were collected on all patients who consented to take part. At T2, the rate of completion was lower either because of treatment non-completion or because patients declined to undergo the voluntary process of questionnaire completion at discharge. Due to the change in sample composition the mediation model was tested and verified separately at T1 and T2 and on those who completed both phases. Furthermore, ANOVAs compared participants who completed both phases to those who dropped out at T2 on the measures of interest to this study and found that the two groups did not differ significantly in depression, EA or alexithymia.

Table 3
Means, SDs, reliabilities and change scores for time 1 to time 2 for sample completing both assessment points in study 2.

	N	T1 *M* (*SD*)	T2 *M* (*SD*)	*ΔM* (*SD*)	α[1]
TAS total	77	55.12 (10.69)	51.35 (11.69)	−3.77 (9.20)	.79
DDF	77	13.42 (3.46)	12.79 (4.05)	−0.62 (3.40)	.58
DIF	77	17.92 (6.57)	15.87 (6.71)	−2.05 (5.48)	.81
EOT	77	23.77 (4.78)	22.68 (5.15)	−1.09 (4.78)	.40
EA	79	29.78 (7.85)	23.67 (7.27)	−6.11 (7.11)	.83
BDI-II	83	23.94 (13.45)	12.79 (11.82)	−11.14 (13.05)	.95

T1 = time 1 assessment; T2 = time 2 assessment.
[1] Cronbach's alpha from time 1 assessment.

T1 to T2, hypothesized to be the result of CBT. As shown in Table 3, significant reductions are found in TAS-20 total score $F(1,76) = 12.91$, $p < .001$, $\eta^2 = .15$, DIF, $F(1,76) = 10.80$, $p < .01$, $\eta^2 = .12$ and EOT, $F(1,76) = 4.02$, $p < .05$, $\eta^2 = .05$ with the greatest reduction shown in DIF. The change in DDF is not significant. Significant improvements after treatment are also observed in EA, $F(1, 78) = 58.40$, $p < .001$, $\eta^2 = .43$ and depression, $F(1, 82) = 60.55$, $p < .001$, $\eta^2 = .43$.

3.2.2. Correlations among measures at time 1

Pearson correlations were conducted at T1 to verify the associations between the constructs found in study 1. Correlations among the TAS-20 factors and between these factors, EA and depression scores on the BDI-II are shown in Table 4. As expected, alexithymia and EA are significantly correlated and both these constructs are related to psychopathology (depression scores). TAS-20 factors show significant inter-correlations, especially DDF with DIF, while the correlations of these two factors with EOT are not significant, indicating that the factors measure different constructs: DDF and DIF signal mostly emotion difficulties while EOT signals mostly cognitive difficulties (limited fantasy, imagery, etc.).

3.2.3. Mediation models

In study 2, the mediation of EA in the relationship between alexithymia and depression symptoms was tested in two cross-sectional models (for T1 and T2) using PROCESS, with 5000 bootstrapped samples and bias-corrected 95% confidence intervals.

Table 4
Pearson correlations between TAS-20 total and factor scores, EA and depression in study 2.

	DDF	DIF	EOT	EA (AAQ-II)	BDI-II
TAS-20 total	.747**	.827**	.569**	.438**	.294**
DDF		.525**	.206*	.257**	.224**
DIF			.106	.317**	.375**
EOT				.367**	−.013
EA (AAQ-II)					.466**

* $p < .05$.
** $p < .001$.

Table 5
Predictors of change in depression scores in study 2.

	ΔR^2	*B*	*SE*	*β*	95% *CI*
Step 1	.019				
*Δ*EOT		.387	.323	.139	−.256, 1.031
Step 2	.060*				
*Δ*EOT		.441	.316	.158	−.189, 1.071
*Δ*DDF		.958	.444	.245*	.074, 1.843
Step 3	.163**				
*Δ*EOT		.291	.291	.104	−.290, .872
*Δ*DDF		.024	.471	.006	−.915, .962
*Δ*DIF		1.135	.290	.470**	.556, 1.713
Step 4	.189**				
*Δ*EOT		.317	.254	.114	−.190, .824
*Δ*DDF		.039	.411	.010	−.780, .858
*Δ*DIF		.676	.270	.280*	.137, 1.216
*Δ*EA		.883	.183	.473**	.518, 1.248

* $p < .05$.
** $p < .01$.

3.2.3.1. Cross sectional mediation model based on time 1 scores. Mediation analysis for T1 included 151 participants and shows that although alexithymia has a significant total effect on depression scores (c: $B = .35$, $SE = .09$, $\beta = .29$, $p < .001$), after controlling for the effect of EA as the mediator (a: $B = .34$, $SE = .06$, $\beta = .44$, $p < .001$; $B = .68$, $SE = .12$, $\beta = .47$, $p < .001$) the direct effect of alexithymia on depression scores is not significant. The indirect effect of alexithymia on depression scores through EA indicates complete mediation by EA ($B = .23$, $SE = .06$, $\beta = .19$, $p = .001$, BCa *CI* [.13, .36]), and represents a medium to large effect size ($\kappa^2 = .18$, BCa *CI* [.11, .27]) [60]. Subsequent analyses, with the 3 factors of TAS-20 as predictors, indicate complete and partial mediation by EA for the association between DDF and depression (c: $B = .79$, $SE = .28$, $\beta = .22$, $p < .01$; c': $B = .39$, $SE = .26$, $\beta = .11$, $p = .13$; indirect effect: $B = .39$, $SE = .14$, $\beta = .11$, $p < .01$ BCa *CI* [.14, .70], $\kappa^2 = .12$, BCa *CI* [.04, .20]) and DIF and depression (c: $B = .75$, $SE = .15$, $\beta = .36$, $p < .001$; c': $B = .51$, $SE = .15$, $\beta = .25$, $p = .001$; indirect effect: $B = .25$, $SE = .08$, $\beta = .12$, $p < .01$, BCa *CI* [.12, .42], $\kappa^2 = .13$, BCa *CI* [.06, .21]) respectively, while the mediating role of EA between EOT and depression symptoms is not significant.

3.2.3.2. Cross sectional mediation model based on time 2 scores. In the same model for T2, mediation analysis includes 79 participants and shows that although alexithymia has a significant total effect on depression scores (c: $B = .50$, $SE = .10$, $\beta = .48$, $p < .001$), after controlling for the effect of EA as the mediator (a: $B = .30$, $SE = .06$, $\beta = .48$, $p < .001$; b: $B = .67$, $SE = .17$, $\beta = .55$, $p < .001$) the direct effect of alexithymia on depression scores shows reduced significance (c': $B = .29$, $SE = .11$, $\beta = .28$, $p < .01$). The indirect effect of alexithymia on depression through EA indicates partial mediation ($B = .20$, $SE = .07$, $\beta = .20$, $p < .01$, BCa *CI* [.09, .38]) and represents a moderate to large effect size ($\kappa^2 = .20$, BCa *CI* [.10, .33]) [60].

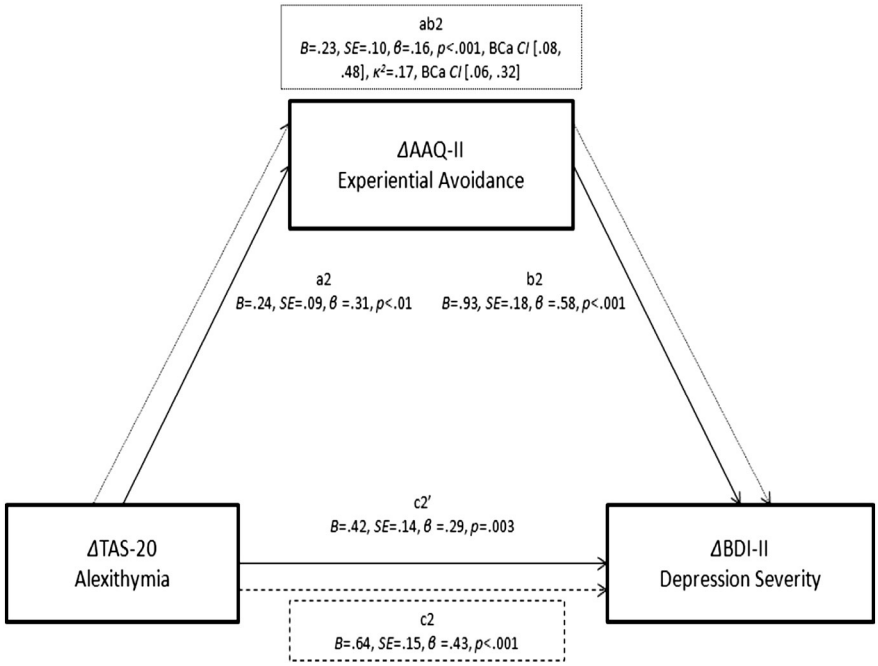

Fig. 2. The mediating effect of EA change score in the relationship between alexithymia change score and depression severity change score (c2 = total effect, c2' = direct effect, ab2 = indirect effect).

Subsequent analyses, including the 3 factors of TAS as predictors, indicate the partial mediating role of EA between DDF and depression (c: $B = 1.44$, $SE = .29$, $\beta = .48$, $p < .001$; c': $B = .94$, $\beta = .32$, $SE = .29$, $p < .01$; indirect effect: $B = .50$, $SE = .19$, $\beta = .17$, $p < .01$, BCa CI [.21, .96], $\kappa^2 = .18$, BCa CI [.08, .31]) and DIF and depression (c: $B = 1.03$, $SE = .17$, $\beta = .58$, $p < .001$; c': $B = .72$, $SE = .18$, $\beta = .40$, $p = .001$; indirect effect: $B = .31$, $SE = .13$, $\beta = .17$, $p < .01$, BCa CI [.10, .61], $\kappa^2 = .18$, BCa CI [.07, .32]). Mediation by EA between EOT and depression symptoms is not significant. The results of all cross sectional models replicate the findings of study 1 and support hypothesis 2, that EA mediates the association between alexithymia and depression symptoms, primarily explaining the link between DIF/DDF and depression.[2]

3.2.3.3. Mediation model accounting for change after treatment. To examine the longitudinal course of this meditational relationship, an additional model was examined based on the change scores from T1 to T2 for TAS total score, EA and depression symptoms. First, the TAS-20 total change score was used as the predictor and subsequently the TAS-20 factor change scores (i.e. DDF, DIF and EOT) as the predictors, with EA change score as the mediator and depression change score as the dependent variable.

The model, based on a sample of 75 participants who completed both assessment times is presented in Fig. 2. The indirect effect of alexithymia change score on depression change score through EA change score is $B = .23$, $SE = .10$, $\beta = .16$, $p < .05$, BCa CI [.08, .48]. Although the difference in the significance levels between the total and the direct effect model is not large, results indicate partial mediation by EA change scores and represent a medium to large effect size ($\kappa^2 = .17$, BCa CI [.06, .32]) [60]. Results suggest that decreases in EA mediate the effects of alexithymia improvement in depression symptom reduction. Subsequent analyses including the 3 TAS factors as predictors indicate the partial mediating role of EA between DIF and depression change (c: $B = 1.16$, $SE = .25$, $\beta = .48$, $p < .001$; c': $B = .71$, $SE = .24$, $\beta = .30$, $p < .01$; indirect effect: $B = .45$, $SE = .188$, $\beta = .19$, $p < .01$, BCa CI [.17, .95], $\kappa^2 = .20$, BCa CI [.08, .36]). The mediating role of EA between DDF and depression, and between EOT and depression is not significant.

3.2.4. Prediction of change in depression severity by change in TAS factors and EA

A hierarchical regression was carried out in order to examine the amount of variance of change in depression symptoms explained by change in each TAS factor and EA, as a result of treatment. Each of the TAS factors change scores was entered as a separate step in the model, starting from EOT, DDF and DIF (see Table 5). EA change score

[2] To verify that the model is not restricted to depression but also pertains to general distress and negative affect (including anxiety, another common emotional symptom in this population and much other psychopathology), it was also tested using scores of negative affectivity as measured in the Positive Affectivity Negative Affectivity Scale (PANAS) as the outcome variable. Mediation of the effects of alexithymia by EA on negative affectivity was supported both at T1 and T2. Application of the model to other symptoms and populations is beyond the scope of this paper.

was entered in the last step. Parametric assumptions for this model (no multicollinearity, homoscedasticity, independence and normality of errors) were met. Significant changes in the variance explained are found in step 2, where DDF was added to the model, in step 3, where DIF was added to the model and in step 4, where EA was added. In the final step, with all predictors entered, the total variance in depression improvement explained is 43.1%. Significant predictors of depression improvement are changes in EA and DIF, $F(4,70) = 13.26$, $p < .001$).

4. Discussion

Alexithymia is a known correlate of psychopathology [8], especially depression and medically unexplained symptoms. This association has been attributed to the difficulty of alexithymic individuals to identify and describe their emotions and to differentiate the interoceptive experiences associated with emotion from the symptoms of a somatic illness [5,30]. Although alexithymic deficits in processing emotions have been repeatedly examined, neither their exact nature, nor the mechanisms that link them to psychological and health problems are well-understood [67]. This investigation examined the hypothesis that alexithymia is associated with EA, and that alexithymic deficits in emotion processing are associated with psychological symptoms through the effects of EA. Two studies were conducted, representing a community and a clinical sample, two different cultures (Cyprus and USA), a cross-sectional and a longitudinal approach, and were in respect to two outcome variables, namely psychosomatic symptoms and depression. The studies contribute new findings, which advance the understanding of alexithymia and its role in psychopathology.

In both samples, alexithymia, and in particular DIF, are strongly related to EA [41]. Individuals who present with difficulties in identifying, labeling and describing emotions, may in essence be trying to avoid experiencing them, and may have learned over time to use this avoidance strategy quite efficiently. EA is typically believed to be reinforced through the immediate relief it procures [44], which, however, ultimately amplifies unwanted experiences [68]. The observed correlations cannot foster an etiological interpretation but provide a basis for longitudinal and experimental studies to examine if the practice of EA leads to the increase of alexithymic traits in some individuals.

The key finding of this study is that in both a clinical and a community sample EA mediated the effects of alexithymia, and primarily its DIF factor, on symptomatology. This effect, obtained through the highly reliable bootstrapping approach, was true for both somatic symptom reporting and depressive symptoms and held for both a relatively healthy, high functioning community sample and a sample of residential patients with anxiety disorders and clinical depression. Full mediation was supported in both studies, while in the cross-sectional T2 analysis in study 2 partial mediation was supported. The latter effect is probably due to the smaller sample size at T2, due to attrition. Although alexithymia and EA have been known to have pathogenic effects on mental health and have both been associated with emotion regulation difficulties [5,69–72], few attempts have previously been made to examine their association and how they operate synergistically to exert their negative consequences (with the exception of the Venta et al.'s study [41]).

Results from the present investigation show that both alexithymia and EA are highly predictive of psychopathology and psychological distress, psychosomatic symptom reporting and depression respectively, and suggest a mechanism through which this relation holds. This mechanism is proposed to be the use of EA, understood as a learned coping strategy, to avoid coming into contact with unwanted experiences. This has implications for the understanding of alexithymia. Although alexithymia is believed to be a personality trait, and therefore fairly stable, it has recently been found to decrease in response to therapy [48] and interventions that teach high alexithymic individuals how to process emotion more effectively [25]. Given that alexithymia is amenable to therapeutic change, that it is higher in certain SES groups, and the current findings showing that its effects are mediated by EA, an emotion regulation strategy that increases through reinforcement, one can suggest that alexithymic deficits may be to some degree temperamental and inherent in nature but are also acquired through experience [16,17]. It is not, it appears, that alexithymic individuals lack the ability to process emotion, but may not have learned to do so functionally, especially when emotion is unpleasant or highly arousing [25]. This assertion needs validation through genetic studies and longitudinal approaches that can best clarify the temporal order in which these characteristics and behaviors appear in an individual´s repertoir and lifespan.

Clinically, the current findings have important implications. Although previous investigations have suggested that alexithymic deficits can be mitigated through targeted interventions such as guidance in deep emotion processing and imagery training [25,73], the current study suggests that a general CBT program, not specifically targeted at alexithymia, can have therapeutic effects by decreasing alexithymia itself (especially DIF and DDF), and by decreasing EA, which appears to be an important mediator through which alexithymia is linked with psychopathology. Despite the need for further research identifying specific mechanisms of change, some initial hypotheses can be made. For example, the intensive treatment program described in study 2 focused on OCD pathology and is heavily based on ERP skills [74]. Exposure increases tolerance for negative affect and is believed to improve perceptions of controllability and ability to accept the aversive experience of anxiety [44,75,76]. The newly acquired confidence of these patients in handling or tolerating unpleasant affect can be hypothesized to have played a role in the decrease of their EA and in turn their willingness to experience unpleasant affect and

increased physiological arousal. Their increased CBT skills probably also operated in improving their ability to identify emotions and distinguish them from other bodily symptoms, and to better describe their experiences.

Some strengths and limitations of the current studies should be noted. Firstly, although relatively good health could be assumed among the participants in study 1 due to their young age and the low somatic symptoms scores, participants were not asked whether they had any actual medical diagnoses, which may have inflated some PHQ-15 scores. Also, study 1 was cross-sectional in nature, which allows only for cautious interpretation of results, as alternative models could also be possible [77]. Study 2 remediated this in part by providing two data collection points, that permitted the examination of the impact of therapy-related change on the proposed model. In this case too, however, Structural Equation Modeling would be most appropriate to capture the longitudinal course of the hypothesized model, which was not permitted by the small sample size. The change scores approach was an alternative solution that provided preliminary evidence about the longitudinal nature of these effects. Future research should also look into the therapeutic mechanisms of CBT that led to the promising improvements in alexithymia and EA in study 2, and it should also test the proposed model in clinical samples where depression is the primary diagnosis, as well as on anxiety and other negative emotions that have been linked to alexithymia. Finally, it is also important to note that the internal consistencies of two of the TAS-20 factors, DDF and EOT, especially in study 2, were lower than most other studies. However, this does not influence the interpretation of findings as TAS-20 total score and DIF, which were the strongest correlates of EA and psychopathology, showed good internal consistencies.

In spite of these limitations, the study has some noteworthy strengths. Models were tested in two different samples, two countries and both a clinical and a non-clinical population. The samples differed in age and gender composition, which along with the two different outcome variables examined, allow for generalization of results to both depression and somatic symptoms as well as both low and high levels of alexithymia and psychopathology across adult age groups. Furthermore, the PROCESS approach used produces highly reliable results due to the multiple iterations to which the data are subjected.

In sum, the present investigation provides evidence that alexithymia is related to psychopathology, and specifically to depression and somatic symptom reporting through EA. In fact, EA is highly related to alexithymia's core characteristic of DIF and should be further examined as the explanatory mechanism for alexithymic emotion processing deficits. Alexithymic individuals may not lack the ability to experience emotion appropriately, but may have learned to avoid it, potentially because they do not possess appropriate emotion regulation skills. To them, intense and unpleasant emotion, as noted by others, may appear overwhelming [10], and they may have learned to blunt their emotions and avoid them to the degree that they cannot recognize them or distinguish them from other bodily sensations. Therapeutic interventions like CBT that foster emotion regulation, emotion tolerance, psychological flexibility and a sense of efficacy in dealing with one's experiences may be valuable in remediating alexithymic difficulties and their impact on mental health. It seems that a decrease in EA is the vehicle through which this change can be achieved.

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