Alexithymia in anorexia nervosa: The mediating role of depression

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Abstract

The role of depression in the expression of alexithymia in anorexia nervosa (AN) has been controversially explained and several variables that may mask or increase the presence of emotional difficulties have scant examination in previous studies. This study aims to analyze the associations between alexithymia and state variables, such as age, BMI, illness duration, treatment duration, and medication status in AN participants, and to test the mediating role of depression in emotional difficulties. The Toronto Alexithymia Scale (TAS-20) and the Zung Self-Rating Depression Scale were administrated to 160 females: 80 participants with AN and 80 healthy controls. High levels of alexithymia were not a function of state variables. The mediating role of depression differed by the alexithymia dimension, with total mediation found for the TAS-DDF and partial mediation found for the TAS-DIF. Alexithymia is a relevant feature throughout the spectrum of AN and does not seem to be related to developmental maturation and some clinical features. Depression is probably the variable that best accounts for the variance in alexithymia, but is not a complete explanation for the known cognitive–affective disturbances in AN. Specific emotional competencies require scrutiny during psychiatric treatment.

Keywords: alexithymia; body mass index; eating disorders; emotional difficulties; illness duration; mediation; medication status

1. Introduction

Emotional dysfunctions in anorexia nervosa (AN) were recently synthesized in two systematic reviews (cf. Hatch et al., 2010; Oldershaw et al., 2011). Evidence indicates that a broad range of emotional difficulties exist in AN, including poor emotion recognition and deficits in the processing of emotional information. Historically. a well reported disturbance in cognitive processing and regulation of emotions in AN is that of alexithymia. This cognitive-affective deficit is characterized by (a) a difficulty in identifying feelings and distinguishing emotions from physical sensations (DIF), (b) a difficulty in describing feelings to others (DDF), and (c) a diminution of imagination and an externally oriented thinking (EOT) (Taylor et al., 1997). Due to high levels of alexithymia observed in AN patients (e.g., Corcos et al., 2000; Eizaguirre et al., 2004; Speranza et al., 2005; Torres et al., 2011b), it has been hypothesized that eating-disordered behaviors such as starving, binging or purging, offer a strategy to avoid, suppress, or regulate affect (e.g., Wildes et al., 2010; Haynos and Fruzzetti, 2011; Brockmeyer et al., 2012), thus redirecting attention from negative emotions to the body and eating (e.g., Taylor et al., 1997; Overton et al., 2005). For this reason, it is suggested that alexithymia can affect recovery from eating disorders, because such cognitive limitations in emotion regulation may predispose the use of maladaptive eating behaviors to deal with stressful situations (Speranza et al., 2007). In addition, the reduced ability of reporting emotions to the therapist represents another obstacle for therapeutic efficacy (Jänsch et al., 2009; Haynos and Fruzzetti, 2011).

1.1. Alexithymia and Depression in AN

Considering that alexithymia can perpetuate the clinical expression of ED, several studies in AN have questioned if this emotional deficit is explained by the presence of depressed mood (e.g., Corcos et al., 2000; Eizaguirre et al., 2004; Speranza et al., 2005). Theoretically, is reasonable to assume that there is an association between alexithymia and depression, as

both constructs share many characteristics. In particular, negative affect (Saarijärvi et al., 2001; Mattila et al., 2008), anhedonia (Saarijärvi et al., 2001), less clarity about their feelings (Rude and McCarthy, 2003), decreased ability to communicate affect to other people, use of less adaptive emotion-regulation strategies, such as suppression of affect (Saarijärvi et al., 2001), and problems with interpersonal communication (Mattila et al., 2008). Several studies have also reported a significant, positive correlation between alexithymia and depression in patients with eating disorders (Corcos et al., 2000; Eizaguirre et al., 2004; Bydlowski et al., 2005). However, the role of depression in the relationship between AN and alexithymia continues to be controversial, dividing the authors into two camps: those who consider alexithymia a condition secondary to depressed mood, and those who defend that alexithymia reflects an independent aspect of psychological functioning in AN.

In defense of alexithymia as a secondary diagnosis to depression in patients with AN, Corcos et al. (2000) and Gilboa-Schechtman et al. (2006) found that increased rates of alexithymia in individuals with AN, compared to those with bulimia nervosa (BN), were closely related to depression. The results suggested that after controlling for depression, rates of alexithymia did not vary according to the type of ED. Additionally, Bydlowski et al. (2005), Gilboa-Schechtman et al. (2006) and Montebarocci et al. (2006) found that high alexithymia levels among patients with ED, compared to healthy matched controls, were primarily related to negative affect. Taken together, these studies supported the view that alexithymia is a state-dependent phenomena linked to depression.

Conversely, de Zwaan et al. (1996) found that participants with eating disorders presented higher rates of alexithymia when compared to a control group, even after adjusting for depression. Geller et al. (2000) achieved the same conclusion when they analyzed the DDF component of alexithymia. These studies are consistent with the view of alexithymia being independent from depression.

Incorporating these two perspectives, a third approach has emerged which contends that, depending on the factor being analyzed, alexithymia can to be both state-dependent, strongly related to depression, and a stable trait, not changing in the presence of depressive symptoms. Two studies concluded that AN patients with restricting subtype are less able to describe their emotions when compared to controls, and this difficulty does not seem to be influenced by the level of depression, opposed to the DIF factor, which was associated with depression in both AN subtypes (Sexton et al., 1998; Speranza et al., 2005). With this unified approach, we can also explain the findings by Eizaguirre et al. (2004) in which alexithymic features were closely linked to, and explained as, depression. These features also represented distinct personality traits in some patients with ED.

In conclusion, findings in this area are controversial and the role of depression in the relationship between alexithymia and AN remains unknown. Due to the potential negative impact of alexithymia in recovery from eating disorders, the clarification of this issue is of significant interest, and has been advocated by several authors (Fox and Power, 2009; Hatch et al., 2010; Oldershaw et al., 2011). Therefore, the goal of this study was to analyze the impact of mood disorder symptoms on the expression of alexithymia in individuals with AN.

1.2. State Variables in Alexithymia

In order to achieve this goal, there are confounding variables that should be considered as they may mask or exacerbate the presence of emotional difficulties. In particular, the importance of controlling for the use of psychotropic medication (Jänsch et al., 2009; Oldershaw et al., 2011) and the effect of other "state variables" such as body mass index (BMI), illness duration, treatment duration, and age on the variation of emotion-processing deficits (Oldershaw et al., 2011). It is possible that some alexithymia dimensions can be less prevalent in less severe ED cases, as suggested by Laquatra and Clopton (1994). For example, how long someone has had an ED may interfere both in DDF and depression levels, as

patients typically become increasingly socially isolated the longer they are ill (Oldershaw et al., 2011). The overall time spent in treatment can account for a substantial symptom reduction (Brauhardt et al., 2014), including impaired emotional functioning. Age can also produce differences in the expression of alexithymia, in that affect development occurs in stages and may be influenced by environmental and developmental variables (Lane et al., 1998).

With these considerations, first we examined whether depression and the aforementioned state variables (use of psychotropic medication, BMI, illness duration, overall treatment duration, and age) were able to predict alexithymia. Then we tested whether and how much depression explains (i.e., mediates) the relationship between AN and alexithymia.

1.3. Methodological Considerations

We addressed some methodological limitations in previous studies that may explain the divergent results. First, we employed the use of a large sample and parsed out individuals with AN from those with BN, as some researches have noted differences in emotion processing between people with these two eating disorders (e.g., Gilboa-Schechtman et al., 2006). In contrast to previous studies which used a total score, we emphasized the multidimensional nature of alexithymia (Taylor et al., 2000) by conducting separate analyses for each facet of the construct (Corcos et al., 2000; Bydlowski et al., 2005; Montebarocci et al., 2006).

Methodologically, the most common assessment used for depression symptoms is the Beck Depression Inventory (BDI; Beck et al., 1961). BDI is well known and widely used, but is limited in scope as it does not assess all potential indicators or symptoms of depression. Considering that there is substantial variability in the symptom areas covered by the BDI and other tests of depression symptomology (Shafer, 2006), we used an alternative depression measure to help clarify the divergent results achieved using the BDI. For this study we chose

the Zung Self-Rating Depression Scale (SDS; Zung, 1965), which is also a common tool in depression assessment, and presents a similar validity and reliability to that of the BDI (Rush et al., 2008). The use of the SDS may have three potential advantages over the BDI. First, the SDS allows for assessment of positive affect (e.g., "feeling happy," "enjoying things," "feel useful and needed") that is a valued component in current models of affect, particularly in combination with depressive symptomology (Reich et al., 2003). While positive and negative affect are inversely related, they can occur simultaneously. Therefore, emotional indicators should be assessed within a model that allows for the co-occurrence of positive and negative affect. Secondly, because the SDS includes depression-absent items it may be more sensitive for assessing low-levels of depression (Spielberger et al., 2003), and consequently provide a more accurate assessment of the effect of the range of depressive symptoms in alexithymia. Third, the SDS has a relatively low literacy level, and low level of complexity in the question phraseology (Shumway et al., 2004), making it an ideal assessment of depression in young samples, as is true with AN samples.

1.4. Study Hypothesis

The current study had two main hypotheses. One, that alexithymia in AN would not vary with age, BMI, illness and treatment duration, or medication status. Two, it was speculated that the link between alexithymia and AN would be partially explained by the presence of depression, and that the mediating role of depression symptoms would vary according to the three alexithymia dimensions. Inherent is the assumption that, although alexithymia may be a tendentious personality trait at the individual level, features of alexithymia may be state-dependent, particularly dependent on the presence of depressive symptoms (Henry et al., 2006). No specific predictions were made for the current study about the mediating role of depression in each alexithymia dimension, as previous studies have highly divergent data on this issue.

2. Methods

2.1. Subjects

Eighty female participants with AN and 80 female healthy control (HC) participants took part in the study. Participants with AN were selected from six public hospitals and two private clinics located in different geographic areas encompassing various socioeconomic strati of Portugal, and at the time of data collection they were all in active treatment, inpatient ($n = 18$; 22.5%) or outpatient ($n = 62$; 67.5%). In both modalities, treatment did not aim therapeutic interventions toward improving emotion regulation. Behaviorally based treatment programs were generically applied as recommend by the American Psychiatric Association (APA, 2006), with nutritional rehabilitation and psychiatric management valences. For eligibility, AN participants were required to have a diagnosis of AN as outlined in the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994), and a minimum illness duration of 6 months. Exclusion criteria were defined as the presence of substance-related and addictive disorders, and schizophrenia spectrum and other psychotic disorders. This information was provided by the subject's doctor. AN patients ranged from 13 to 34 years of age (mean age = 19.21 years; $SD = 5.39$) and their BMI (kg / m^2) varied from 12.4 to 17.5 (mean BMI = 15.27 kg / m^2; $SD = 1.49$). Fifty two patients were diagnosed as having AN restrictive subtype (AN-R) and 28 has having AN binge-purge subtype (AN-B). The length of illness varied between 6 months and 20 years (mean illness duration = 37.90 months; $SD = 45.61$, median 24 months). Sixty-one of the patients (76.3%) were receiving psychotropic medication (antidepressants, anxiolytics, hypnotics, mood stabilizers, and/or atypical antipsychotics) at the time of assessment.

The HC group was matched to the AN participants by age (range = 13 – 34 years; mean age = 19.20 years; $SD = 4.76$), level of education, and socioeconomic status. The great majority of the participants in the HC group were selected from high schools and universities.

The inclusion criteria were as follows: absence of current or past history of an eating disorder and normal weight according to World Health Organization classification (BMI between 18.5 and 24.9 kg / m^2). Within the control group BMI varied from 19.0 to 24.2 (mean BMI = 21.08 kg / m^2; SD = 1.39).

2.2. Measures

The Interview for the Diagnosis of Eating Disorders-IV (IDED-IV; Kutlesic et al., 1998) was given to the AN group in order to confirm the diagnosis of AN according to DSM-IV (APA, 1994) criteria. The Portuguese version of IDED-IV has yielded sufficient reliability and validity to be used for establishing a diagnosis of AN (Torres et al., 2008). In the current study, the Cronbach's Alpha coefficient was 0.732. Participants in the HC group completed a screening questionnaire developed to assess (a) current or past diagnosis of eating disorders ("have you ever had an eating disorder, such as anorexia nervosa, bulimia nervosa, or any other eating problem? Please describe it."), (b) current diseases ("do you have any disease, physical or mental?"), (c) weight and height, and (d) medication use.

All participants completed the Toronto Alexithymia Scale (TAS-20; Bagby et al., 1994; for Portuguese validation see Prazeres et al., 2000) and Zung Self Rating Depression Scale (SDS; Zung, 1965; for Portuguese validation see Diegas and Cardoso, 1986). The TAS-20 yields three factors: DIF, difficulty in identifying feelings; DDF, difficulty in describing feelings to others, and EOT, externally oriented thinking style (Bagby et al., 1994; Prazeres et al., 2000). In this study Cronbach's Alpha coefficient for the SDS (α = 0.895), total TAS-20 (α = 0.874), DIF (α = 0.853) and DDF (α = 0.810) indicated good internal consistency. For EOT (α = 0.602) the reliability coefficient was below the generally recommended lower limit of α = 0.700 for Cronbach's Alpha (Hair et al., 1998). Of note, the psychometric properties of the TAS-20 have not been systematically examined in adolescent respondents and the few

existing studies that have used it have not strongly confirmed its validity for use in pre-adult populations (Parker et al., 2010).

2.3. Procedures

The study received approval from relevant ethics boards, and participants completed informed written consent. For participants younger than 18, informed written consent was provided by a parent and assent from the minor. After consent, each participant in the AN group underwent diagnostic confirmation of AN by a trained clinician using the IDED-IV, and completed the TAS-20 and SDS self-report questionnaires. Patients were recruited for the study by their physicians based on inclusion criteria, and subsequently evaluated by research personnel. In the HC group the same self-report measures were given after completion of the screening questionnaire. This material was a group survey administered at the participant's respective educational settings. All participants agreed to participate in the study.

2.4. Data Analysis

The first step was to ensure that the underlying assumption of normality was met with all variables. To test this assumption, we used the Kolmogorov-Smirnov test (KS; with Lilliefors Significance Correction) and the following criteria: absolute skewness (Sk) and kurtosis (K) values lower than 3.0 and 8.0, respectively (Kline, 2005). Based on these criteria the assumption of normality was met for the total sample, and the AN and HC groups independently [significant KS p-values for BMI, age, illness duration, DIF, and SDS, although with acceptable absolute values for kurtosis (varying between -0.088 for DDF and 1.103 for age) and skewness (varying between -1.259 for BMI and 0.814 for age). Since the variables illness duration and treatment duration were extremely positively skewed, our decision was to log-transform these variables to approximate normal distribution. The log-transformed variables revealed acceptable values for skewness and kurtosis: illness duration (Sk= 0.323, K= -0.555) and treatment duration (Sk= 0.368, K= -0.541). Missing scores on

three single items of the TAS-20 and SDS were imputed using factor person-mean substitution. In three cases of the HC group, multiple missing scores on the TAS-20 or the SDS questionnaires required exclusion from the model-testing analyses. Item 6 of the SDS ("I still enjoy sex") had a high number of missing data points both in the HC ($n = 15$; 18.8%) and AN groups ($n = 18$; 22.5%), and was excluded from the SDS total score computing. Given the poor internal consistency of the EOT factor ($\alpha = 0.602$), this alexithymia dimension was not included in data analysis.

Preliminary analyses were conducted to examine relationships among all variables. Differences between groups (AN-R, AN-B and HC) in total alexithymia score, depression, BMI, and age were analyzed using analysis of variance (ANOVA). Multivariate analysis of variance (MANOVA) was used to assess group differences in the two alexithymia factors. In both ANOVA and MANOVA the Scheffé test was applied to post-hoc analyses and the effect size was measured using the eta-squared and partial eta squared coefficients (η^2 and η^2_p). Then, some statistical analyses were specifically conducted with the AN group to compare subgroups with regard to possible confounding factors. Illness and treatment duration mean scores, as well as medication status were compared in the AN-R and AN-B subgroups. Given the sample age range, and the possibility of adolescent participants having specific features that could be considered as confounding effects in reported alexithymia, mean scores of state variables and depression were also compared in adolescent (age between 13-18) and adult (age between 19-34) subgroups. The independent-samples t test was used for subgroup comparisons on continuous variables. The association between subgroups and medication status was analyzed through an independence Chi-square test (with Yates Continuity Correction). Pearson correlation coefficients (r) were also calculated to explore the relationship (and possible multicollinearity) between state variables, alexithymia and depression in the AN group. Multiple regression analysis was used to evaluate the relative

contribution of depression on alexithymia, controlling for patients' age, BMI, illness duration, treatment duration, and medication status. These variables were included in the model based on theoretical reasons.

Subsequently, a mediation analysis was conducted to test the mediating effect of depression on the association between alexithymia (total score and factors tested in each model) and AN diagnosis (AN group vs. HC group). We performed mediation analysis procedures with bootstrap sampling, as recommended by Preacher and Hayes (2004). The bootstrap method has been validated in the literature and is preferred over other methods for assessing possible mediation among variables (Preacher and Hayes, 2008). It estimates indirect effects through one or more mediator variables with bias-corrected bootstrap confidence intervals (Preacher and Hayes, 2004). The analysis includes the estimation of direct, indirect, and total effects. In this study the *direct* path was the effect of alexithymia on AN diagnosis, independent of its effect on depression. The *indirect effect* was the path linking alexithymia to AN via depression. The *total effect* of alexithymia on AN was the sum of the direct and indirect effects. Estimates of all paths were calculated using a set of regressions: ordinary least squares regression (for continuous outcomes) or maximum likelihood logistic regression (for dichotomous outcomes). These analyses were conducted using the PROCESS macro (Hayes, 2013), a computational procedure for SPSS. One thousand bootstrap resamples were used to generate bias-corrected 95% confidence intervals for the indirect effect. As outlined in Preacher and Hayes (2004), mediation is demonstrated when the indirect effect is significant and the confidence intervals do not contain zero (i.e., indicating that the indirect effect is significantly different than zero). All tests were two-tailed and considered statistically significant at $p < 0.05$.

3. Results

3.1. Preliminary Analyses

3.1.1. Group comparisons and correlations between variables.

Table 1 provides results from the total scores of self-report measures (TAS-20 and SDS), BMI, and age in the three diagnostic groups (AN-R, AN-B, and HC). Self-reported levels of alexithymia, $F(2, 157) = 30.66$, $p < 0.001$, $\eta^2 = 0.28$, and depression, $F(2, 157) = 41.92$, $p < 0.001$, $\eta^2 = 0.35$, differed significantly between the groups. Post-hoc analyses demonstrated that in both alexithymia and depression measures the AN-R and AN-B groups did not differ significantly from each other, but both had significantly higher scores than the HC group. As expected, the BMI was lower in the AN-R and AN-B groups, compared to the HC group. Age was the only variable that reached a statistically significant difference between the two AN groups, despite a small effect.

There was also a main effect of group on the two combined alexithymia factors, $F(4, 310) = 14.03$, $p < 0.001$, Wilks' Lambda $= 0.72$, $\eta^2_p = 0.15$. When the results of alexithymia factors were considered separately, we found that differences in each reached statistical significance. Post-hoc comparisons showed that the AN-R and AN-B groups did not present significant differences on the DIF and DDF factors, but both reported higher rates than the HC group (see Table 2).

Additional comparisons between AN-R and AN-B revealed no mean differences in illness duration, $t(78) = 1.48$, $p = 0.142$, $d = 0.35$. However, participants with AN-B presented with a longer length of treatment ($M = 2.32$, $SD = 1.36$) in comparison with AN-R ($M = 1.50$, $SD = 1.18$), $t(75) = -2.77$, $p = 0.007$, $d = 0.64$. The proportion of AN-R taking psychotropic medication was similar in AN-B subgroup, $\chi^2(1, n = 80) = 0.01$, p $= 0.934$, phi $= -0.04$.

Considering the two age groups (adolescents vs. adults) in the AN sample, there was no significant difference between them in BMI, $t_{Welch}(61.7) = 1.19$, $p = 0.241$, $d = 0.27$, illness duration, $t(78) = -0.29$, $p = 0.776$, $d = 0.07$, or depression, $t(77) = -1.78$, $p = 0.080$, $d = 0.40$.

Only treatment duration reached statistically significant difference in the mean scores for the two groups, t (75) = -3.77, $p < 0.001$, $d = 0.85$, with the adults undergoing a longer treatment length ($M = 2.41$, $SD = 1.40$) in comparison with adolescents ($M = 1.36$, $SD = 1.04$). There was no significant association between age group and medication status, χ^2 (1, $n = 80$) = 0.70, $p = 0.403$, phi = 0.12.

The correlations between all study variables in the AN group are shown in Table 3. Depression was the only variable with significant correlations with alexithymia (total score and factors).

3.1.2. Prediction of alexithymia in AN patients.

Results of the multiple regression analyses in the AN group are presented in Table 4. Together, age, BMI, illness duration, treatment duration, medication status, and depression (predictor variables), accounted for a significant proportion of the variance in the total alexithymia score, $F(6, 69) = 7.61$, $p < 0.001$, $R^2 = 0.40$, adjusted $R^2 = 0.35$, in DIF, $F(6, 69) = 9.86$, $p < 0.001$, $R^2 = 0.46$, adjusted $R^2 = 0.42$, and in DDF, $F(6, 69) = 4.70$, $p = 0.001$, $R^2 = 0.29$, adjusted $R^2 = 0.23$. In these three regression models depression was the only significant predictor.

Preliminary analyses suggested that the AN-R, AN-B participants, adolescents, and adults, could be combined into one group, because they did not differ significantly with respect to alexithymia and depression. In addition, the variance of alexithymia (total score, DIF and DDF) in the AN group was significantly related to depression, but not to state variables (age, BMI, illness duration, treatment duration, and medication use). This result suggests that the observed subgroup differences in age (AN-R vs. AN-B) and treatment duration (AN-R vs. AN-B, adolescents vs. adults) may not have a confounding effect in reported alexithymia. As a whole, these results supported the study of the mediating effect of

depression on the relationships between: (a) total alexithymia score and AN diagnosis (Model 1); (b) DIF and AN diagnosis (Model 2); and (c) and DDF and AN diagnosis (Model 3).

3.2. Tests of the Hypothesized Models

Table 5 shows the results of the regression of alexithymia symptoms in depression for the three mediation models. As expected, we found significant associations between the hypothesized mediator and the total TAS-20 score, $F(1, 155) = 135.67$, $p < 0.001$, $R^2 = 0.47$; DIF, $F(1, 157) = 168.73$, $p < 0.001$, $R^2 = 0.52$; and DDF, $F(1, 156) = 88.58$, $p < 0.001$, $R^2 = 0.36$.

The direct, indirect, and total effects of mediational bootstrapping analyses are reported in Table 6. In Model 1, the examination of the *indirect effect* revealed a significant mediation ($B = 0.06$, $SE = 0.02$, $Z = 3.55$, $p = 0.0004$, 95% CI = $0.03 – 0.09$), in which the association between TAS-20 and AN diagnosis was mediated by SDS. However, when SDS was added to the model the *direct effect* of TAS-20 to AN also remained significant ($B = 0.05$, $SE = 0.02$, $Z = 2.65$, $p = 0.008$, 95% CI = $0.01 – 0.09$). As such, we concluded that SDS only partially mediated the link between TAS-20 and AN.

The results from Model 2 lead to the same conclusion. Both *direct* ($B = 0.09$, $SE = 0.04$, $Z = 2.24$, $p = 0.025$, 95% CI = $0.01 – 0.17$) and *indirect* effects ($B = 0.12$, $SE = 0.03$, $Z = 3.62$, $p = 0.0003$, 95% CI = $0.06 – 0.19$) of DIF on AN were significant, indicating that the SDS partially mediated the relationship between the DIF factor and the AN diagnosis in the current sample.

Concerning Model 3, the *indirect effect* of DDF on AN diagnosis via SDS was significant ($B = 0.17$, $SE = 0.04$, $Z = 4.35$, $p < 0.001$) with a 95% confidence interval between 0.09 and 0.26. After adjusting for the proposed mediator, the *direct effect* of DDF on AN was not significant ($B = 0.06$, $SE = 0.05$, $Z = 1.23$, $p = 0.218$, 95% CI = $-0.04 - 0.15$). These results are consistent with the full mediation hypothesis.

4. Discussion

4.1. The Effect of State Variables in the Expression of Alexithymia

This study allowed for a more comprehensive examination of the role of depression in the expression of alexithymic features in individuals with AN. The first stage aimed to confirm whether variation in alexithymia scores were predicted by age, BMI, illness and treatment duration, and medication status. The nonsignificant associations with state variables were commensurate with previous studies about age (Sexton et al., 1998; Zonnevylle-Bender et al., 2004) and illness duration (Bydlowski et al., 2005), and refuted the confounding effect of age-related differences and AN diagnosis length on high alexithymia levels.

This conclusion is also extended to the time spent in treatment, which reveled to be nonsignificant in alexithymia prediction. As far as we know, this is the first study analyzing this issue and the results suggest that a longer duration of treatment might not be invariably positive in improvement of emotional difficulties, as it seems to happen with other key symptoms of ED (Brauhardt et al., 2014).

Congruent with the first hypothesis of the study, we also found that alexithymia in the AN population has little variation when considering the amount of weight loss.

Furthermore, the finding that medication status did not predict alexithymia scores suggests that individuals with AN have difficulties in processing emotions, even when receiving medication for depression and anxiety and other specific target symptoms (e.g., conditioned fear learning, cognitive rigidity, perfectionism, impulsivity and obsessionality). In a recent meta-analysis Lebow et al. (2013) estimated the influence of atypical antipsychotics on psychiatric symptoms and concluded that these medications, despite having a positive effect on depression, could increase anxiety. Similarly, a Cochrane review of antidepressants for AN (Claudino et al. 2006) was not able to demonstrate any effect of antidepressant drugs, including the class of selective serotonin reuptake inhibitors (SSRI), on

the associated psychopathology. Possibly, the remaining intense emotionality may be one explanation for the nonsignificant effect of psychiatric medication in the variation of alexithymia. On the other hand, this result can also be due to the high proportion of adolescents in the AN sample (57.5%, $n = 46$). The developmental stage at which AN occurs may affect both symptom presentation and medication efficacy. Younger patients may exhibit less specific psychiatric symptoms (Reinblatt et al., 2008) and may be less responsive to some drugs (Balestrieri et al., 2013). Nevertheless, and in addition to these explanations, the possibility of AN being simply one psychopathology which is not responsive to pharmacologic treatment should be considered. In fact, until now, there is no convincing evidence of efficacy for any drug treatment for AN in either adults or adolescents (Holtkamp et al., 2005; Reinblatt et al., 2008; Crow et al., 2009; Balestrieri et al., 2013; Lebow et al., 2013). The ego syntonic symptoms, which are likely to lead to poor medication compliance, and nutritional disturbances, which impede drug response, are two potential reasons for the unproven efficacy (Crow et al., 2009).

In preliminary analyses comparing both AN subtypes by TAS-20 scores and, in agreement with select studies (e.g., Sexton et al., 1998; Bydlowski et al., 2005; Speranza et al., 2005), we did not find significant differences between them. This finding reinforced the notion that impaired emotional functioning is a relevant feature along the whole spectrum of AN. Furthermore, these subtypes have proved to be similar in relation to most state variables, with the exception of age and treatment duration. AN-B participants were older than those with AN-R subtype, and older patients tend to begin treatment earlier. It should be noted that age and longer treatment did not predict alexithymia scores and were unrelated to depression, and for this reason we believe that these subgroup differences may not have had a significant impact on the reported emotional difficulties in the AN sample. However, we cannot exclude

the possibility that age and treatment length had an indirect, confounding effect (through other state variables, such as medication efficacy and BMI) with alexithymia levels.

4.2. The Mediating Role of Depression in the Association between Alexithymia and Diagnostic Status

The second phase of the study was targeted at clarifying whether the relationship between alexithymia and AN was driven by depression. Considering the multidimensional nature of the alexithymia construct we tested the total TAS-20 score and its factors in distinct models. An integrative analysis of these results suggested that the role of depression varied according to the model tested. Specifically, that depression: (a) partially mediated the effect of the total TAS-20 score on AN diagnosis; (b) partially mediated the effect of DIF on AN diagnosis; and (c) mediated the relationship between DDF and AN diagnosis. According to these results, we conclude that depression partially explains the presence of alexithymia in AN, influencing DIF and DDF, with varied levels of effect. This conclusion confirms our initial hypothesis.

These results fit in the above mentioned *third approach*, also held by Sexton et al. (1998) and Speranza et al. (2005), which considers alexithymia to be both linked to, and independent of, depression, depending on the alexithymia factor being analyzed. However, it is notable that our results are substantially different from those found by Speranza et al. (2005), which also used the TAS-20 in a large sample of AN patients. In Speranza et al.'s study, the analysis of covariance performed with depression, BMI, and age as covariates only showed a significant difference between AN-R patients and controls on the DDF factor. It is important to differentiate between statistical procedures used by Speranza et al. and the current study, as analysis of covariance does not provide information about partial mediating effects. Nevertheless, divergent results also might be linked to the specificities of the measures used to assess depression, such as the abridged version of the Beck Depression Inventory (BDI-13) versus the SDS. In addition to the previously mentioned differences between these scales, the

BDI-13 assesses negative affect (Shafer, 2006) and requires a severity or intensity judgment of depressive symptoms (Meites et al., 1980). In contrast, the SDS emphasizes both positive and negative symptoms (Shafer, 2006), and requires a frequency judgment (Meites et al., 1980). Based on these specificities, we hypothesized that the DDF in AN might be related to the low frequency of positive affects (as assessed with the SDS), making the individual less apt to share their feelings with others. The low occurrence of positive emotions is congruent with the inability to experience pleasure from activities that underlie anhedonia symptoms in depression (Loas et al., 1998). Anhedonia refers to a deficit in emotion regulation, and despite closely linked to alexithymia, is a distinct construct (Loas et al., 1997). It is found to be elevated in ED patients (Deborde et al., 2006; Harrison et al., 2014) and, in particular, the higher social anhedonia scores in these patients proved to be linked to alexithymia due to the presence of depression (Deborde et al., 2006). Our data is in line with these findings, and provide some evidence of the significant role of anhedonia in DDF in AN.

In addition, it is possible that DIF in AN patients may be better explained by the severity of depression (as measure by BDI-13), than by the frequency of depressive symptoms. In the same line of reasoning, persistent depressive mood, even if not severe, could be largely responsible for the increase of DDF, as it can promote social fears in interaction and observation situations, and ultimately, lead to social avoidance and withdrawal.

We strongly believe that depression modulates the expression of alexithymia dimensions in AN differently, depending on both the frequency and severity of depressive symptoms, as well as the extent to which each alexithymia dimension changes in response to the presence of depressive symptoms. It should be mentioned that even when controlling for depression, AN patients still present difficulties in the recognition of their own emotions, though at a significantly lower level than if depression is not controlled for (Model 2). This finding suggests that DIF can also have trait-specific features, which is consistent with the more

recent etiological models of AN, which posit that emotional difficulties are premorbid to the development of the eating disorder and can contribute to its development and maintenance. This early vulnerability is characterized by facets of temperament, such as low novelty seeking and high harm avoidance behaviors (Treasure et al., 2012), hyper-sensitivity to negative emotional cues (Hatch et al., 2010; Haynos and Fruzzetti, 2011), and impaired emotional recognition (Connan et al., 2003). Also consistent with results from Model 2, data from more comprehensive emotional processing research on people with AN noted that deficits in emotional awareness exist, even when controlled for depression (Jänsch et al., 2009). In addition, preliminary data on women who recovered from AN suggest that poor emotion recognition may persist following weight gain (Harrison et al., 2010; Oldershaw et al., 2011). Despite this interpretation of results using etiological models, it should be mentioned that the trait features of alexithymia seen in this study do not necessarily prove a causal relationship with AN. The study design does not allow for detection of a causal pathway. For this reason, alternative interpretations are also conceivable, including the possibility of alexithymia resulting from AN.

Unpredictably, the relative stability found in DIF was not extended to DDF (Model 3), which was entirely explained by depression. This means that, even with high DIF scores, AN patients are able to express emotions when they are not depressed. In patients without depressive symptoms we expected that the ability to describe feelings would also be affected because, theoretically, the verbal report of feelings is contingent on the ability to previously identify them (Bagby et al., 1994). We believe that emotional reporting occurred because AN patients are not completely unaware of their emotional state. In a previous study by our team (Torres et al., 2011a), AN patients with alexithymia were able to differentiate between the valence and the intensity of emotions while imagining hypothetical scenes. This finding

suggested that the differentiation between positive and negative emotions was easily perceived even by individuals with alexithymia, according to the proximity of their goals.

Recently, Fox and Power (2009) have also suggested that individuals with eating disorders are not completely unaware of their own emotional states, but these feelings are often not expressed due to an intense fear of dealing with emotions that are perceived to be overwhelming to the self. Our data lend support to this theory, and adds information about the influence of depression on this process. Depression significantly accounts for the increase of DDF, and it likely occurs because mood disturbance amplifies the experience of negative feelings. If depressive symptoms are frequent, negative emotions are potentially appraised as dangerous and, consequently, its expression is inhibited (Ioannou and Fox, 2009). On the other hand, with the persistence of depressive symptoms, social withdrawal tends to take root and creates an unfavorable context for emotional expression. In sum, the marked influence of depression in DDF supports the definition of this dimension as state dependent, and in particular, dependent on depression. As such, DDF may ameliorate following mood disorder recovery, in congruence with a greater ability to manage emotions found in recovered AN patients (Harrison et al., 2010; Oldershaw et al., 2011).

4.3. Implications for Practice

It should be mentioned that the distinct mediating role played by depressive symptoms have important implications for AN treatment, in that some emotional difficulties are more, and others less or not explained by the frequency of a depressive mood. This means that some specific emotional competencies should be addressed, because they will not completely ameliorate with intervention for depression and, in addition, may even reduce an individual's response to antidepressant medication (Özsahin et al., 2003). Specifically, emotional awareness seems to be an important area for treatment, though it is partially mediated by depression. The importance and usefulness of emotions should also be stressed during therapy

in order to promote emotion acceptance. It is expected that in making one's own emotions more acceptable, AN patients become less likely to inhibit them, which naturally promotes the ability to identify emotional states and use a more introspective cognitive style. Recently, promising approaches such as emotion–focused therapy (Dolhanty and Greenberg, 2009) and emotion acceptance behavior therapy (Wildes and Marcus, 2011) may be useful tools to differentiate between, accept, and manage emotions. Assessment of the efficacy and validation of these treatments is of major interest.

4.4. Limitations

Some limitations of our study must be acknowledged. By definition, a mediator occurs after that which it mediates and before the outcome (Kraemer et al., 2001). The timing of the presence of alexithymia in this study was assumed on the basis of theory, and not by the use of experimental or longitudinal data. This temporal relationship cannot be established with a cross-sectional design. For this reason, the current study does not prove the pattern of causation between these variables. We can only assume that what we observe is consistent with what we would expect to see if a causal path leading from alexithymia to depression to AN diagnosis were demonstrable.

Some drawbacks linked to the use of self-report measures should also be pointed out. Individuals with low emotional awareness likely have difficulties completing self-report measures about their own emotional status (Fox and Power, 2009; Brockmeyer et al., 2012; Oldershaw et al., 2012). For this reason, complementary measures, especially for the assessment of the DIF and DDF factors, relying less on self-report would have been beneficial. Considering that EOT does not require participants to make judgments about their own deficits with regard to the experience of emotions (Lane et al., 1998; Henry et al., 2006), some authors argue that EOT is the most pure (Meganck et al., 2009), objective (Lane et al., 1998), and accurate (Henry et al., 2006) alexithymia subscale. However, the low internal

consistency found in some studies (e.g., Prazeres et al., 2000; Loas et al., 2001), including ours, document the instability of the EOT factor solution (Kooiman et al., 2002) and limits a reliable analysis of this alexithymia dimension. More attention needs to be given to psychometric properties of the EOT subscale.

Despite the aforementioned advantages of the SDS, this scale also presented two limitations for the use in this study. We cannot discount the possibility that somatic items on the SDS may have contributed to the increase in the total SDS score, as they are simultaneously affected by depression and representative of core symptoms (e.g., weight, eating, and constipation) in AN. It is notable that Item 6 ("I still enjoy sex") was not answered by a significant number of participants in both groups (AN, $n = 18$; HC, $n = 15$). The high percentage of missing, but applicable, responses suggests that Item 6 may not be appropriate for adolescent respondents. Furthermore, the appropriateness of the TAS-20 for use in adolescents between ages 13-14 is still being investigated. Although psychometric studies with adolescents provide preliminary support for the generalizability of the three-factor model of alexithymia to adolescent populations, the TAS-20 may need further adaptation to increase reliability and validity with young adolescents (Parker et al., 2010). The EOT subscale was found to present a high degree of syntactic and semantic complexity in young samples (Parker et al., 2010), suggesting the need to review the wording and the content of some items. For this reason, we believe that the suboptimal level of internal consistency of the EOT subscale in this study was due to the high number of adolescent participants.

Important limitations related to sample characteristics also need to be mentioned. In the present study, the exclusion criteria among the HC sample included ruling out eating disorders. Current and past eating problem histories, as well as weight and height, were self-reported by participants. It is possible that participants with a history of eating disorder symptoms who had not been formerly diagnosed would have responded negatively to this

question, and may have resulted in systematic response distortions. In the AN sample, the presence of psychotic and substance-related disorders were considered as exclusion criteria, but no other psychiatric symptoms were taken into account. For this reason, other psychiatric comorbidities underlying psychotropic medication use, such as anxiety and bipolar disorders, could have impacted the report of alexithymia.

We should also take a critical look at the effect of treatment on the level of alexithymia in AN participants. Despite that neither inpatient nor outpatient treatment explicitly included an emotion regulation component, we cannot exclude the possibility that some aspects of psychiatric intervention have focused on emotional awareness and expression. Even controlling for the treatment length, we cannot ensure that the treatment may not have influenced the levels of alexitimia in some AN participants. Lastly, the fact that these results are based on a treatment seeking sample also limits generalizability to a general community sample.

4.5. Conclusions

The current study represents further progress in the accumulation of knowledge about the effect of depression in the expression of alexithymic features in AN. This research question has been considered in previous works, but with inconsistent results. To our knowledge, no studies to date have investigated this question controlling for, in parallel, the confounding effect of age-related differences and some clinical state features such as BMI, illness and treatment length, and psychotropic medication use. The results added information about the low influence of a set of state variables and the differential impact of depression in the expression of DIF and DDF alexithymia facets. We believe that, with the use of a distinct measure to assess depression, this study explained inconsistent results in literature. We found evidence that the effect of depression in alexithymia may differ depending on the frequency

or severity of depressive symptoms. Possibly, DIF is better explained by the severity and DDF by the persistence of depression.

While this study does not prove causal relationships, it does contribute important information about how alexithymia and depression may interact in AN. Future research should address state/trait status of emotional deficits in this eating disorder population. Longitudinal research, assessing the variation of these co-occurring symptoms, along the disease process, is essential.

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Table 1. Alexithymia, depression, BMI and age in AN and HC groups

Measure	AN-R ($n = 52$)	AN-B ($n = 28$)	HC ($n = 80$)	$F(2, 157)$	η^2	Scheffé Post-Hoc
	M (SD)	M (SD)	M (SD)			
TAS-20	63.81 (10.86)	63.07 (12.28)	48.84 (12.23)	30.66***	0.28	AN-R, AN-B > HC
SDS	48.76 (10.60)	53.18 (9.80)	37.48 (7.68)	41.92***	0.35	AN-R, AN-B > HC
BMI	15.41 (1.55)	14.99 (1.39)	21.08 (1.39)	326.21***	0.08	AN-R, AN-B > HC
Age	18.19 (5.01)	21.11 (5.65)	19.20 (4.76)	3.09*	0.04	AN-R < AN-B

Note. AN-R – Restricting anorexia nervosa; AN-B – Bingeing/purging anorexia nervosa; HC – Healthy Control group; TAS-20 – Toronto Alexithymia Scale (20 items); SDS - Zung Self Rating Depression Scale; BMI – Body Mass Index. Pairwise comparisons listed were significant at least at $p < 0.05$.

*$p < 0.05$; ***$p < 0.001$

Table 2. Alexithymia factors in AN and HC groups

Measure	AN-R (*n*=52)	AN-B (*n*=28)	HC (*n*=78)	$F(2, 156)$	η^2_p	Scheffé Post Hoc
	M (*SD*)	*M* (*SD*)	*M* (*SD*)			
DIF	24.94 (6.64)	25.32 (5.27)	17.72 (5.73)	29.99***	0.28	AN-R, AN-B > HC
DDF	17.75 (4.36)	17.29 (5.06)	13.39 (4.60)	16.54***	0.18	AN-R, AN-B > HC

Note. AN-R – Restricting anorexia nervosa; AN-B – Bingeing/purging anorexia nervosa; HC – Healthy Control group; DIF– Difficulty in identifying feelings; DDF– Difficulty in describing feelings to others. Pairwise comparisons listed were significant at least at $p < 0.05$

***$p < 0.001$

Table 3. Correlations between alexithymia (total scores and factors), depression, BMI, illness and treatment duration, and medication use in AN group

	TAS-20	DIF	DDF	SDS	Age	BMI	Illness Dur	Treat Dur
DIF	0.864***							
DDF	0.804***	0.589***						
SDS	0.604***	0.650***	0.518***					
Age	0.034	0.078	0.053	0.149				
BMI	-0.083	-0.120	-0.161	-0.188	-0.232*			
Illness Dur	0.111	0.042	0.196	0.178	-0.062	-0.095		
Treat Dur	-0.167	-0.110	-0.076	0.006	0.488***	-0.094	-0.017	
Medicat use	0.124	0.218	0.098	0.203	0.115	-0.098	0.109	0.295*

Note. TAS-20 – Toronto Alexithymia Scale (20 items); DIF– Difficulty in identifying feelings; DDF– Difficulty in describing feelings to others; SDS - Zung Self Rating Depression Scale; BMI – Body Mass Index; Illness Dur – Illness duration; Treat Dur – Treatment duration; Medicat use – Medication use

*p < 0.05; ***p<0.001

Table 4. Multiple Linear Regressions analysis predicting alexithymia (total score and factors) as a function of age, BMI, illness duration, treatment duration, medication status, and depression in AN group

Outcome	Predictor	B	SE	β	95% CI
TAS-20	Age	0.10	0.23	0.05	[-0.37, 0.57]
	BMI	0.20	0.73	0.03	[-1.28, 1.67]
	Illness Dur	0.01	1.06	0.01	[-2.10, 2.12]
	Treat Dur	-1.83	0.98	-0.21	[-3.77, 0.12]
	Medicat use	1.68	2.66	0.06	[-3.62, 6.98]
	SDS	0.64	0.11	0.59***	[0.42, 0.85]
DIF	Age	0.06	0.12	0.05	[-0.18, 0.30]
	BMI	-0.01	0.38	-0.01	[-0.76, 0.76]
	Illness Dur	-0.52	0.55	-0.09	[-1.61, 0.57]
	Treat Dur	-0.87	0.50	-0.19	[-1.88, 0.13]
	Medicat use	2.13	1.37	0.15	[-0.60, 4.87]
	SDS	0.37	0.06	0.63***	[0.26, 0.48]
DDF	Age	-0.02	0.10	0.02	[-0.19, 0.22]
	BMI	-0.20	0.33	-0.06	[-0.84, 0.45]
	Illness Dur	0.46	0.47	0.10	[-0.47, 1.39]
	Treat Dur	-0.33	0.43	-0.09	[-1.19, 0.53]
	Medicat use	0.83	1.17	0.01	[-2.25, 2.42]
	SDS	0.21	0.05	0.48***	[0.12, 0.31]

Note. TAS-20 – Toronto Alexithymia Scale (20 items); DIF– Difficulty in identifying feelings; DDF– Difficulty in describing feelings to others; BMI – Body Mass Index; Illness Dur – Illness duration; Treat Dur – Treatment duration; Medicat use – Medication use; SDS - Zung Self Rating Depression Scale

***$p < 0.001$

Table 5. Regression analysis predicting alexithymia as a function of mediator (depression)

Outcome	Predictor	*B*	*SE*	t	95% CIs
Model 1					
SDS	TAS-20	0.55	0.05	11.65***	[0.46, 0.65]
Model 2					
SDS	DIF	1.16	0.09	12.99***	[0.98, 1.33]
Model 3					
SDS	DDF	1.34	0.14	9.41***	[1.06, 1.62]

Note. SDS - Zung Self Rating Depression Scale; TAS-20 – Toronto Alexithymia Scale; DIF – Difficulty in identifying feelings; DDF – Difficulty in describing feelings to others

***$p < 0.001$

Table 6. Direct, indirect and total effects of alexithymia (total score and factors) on AN diagnosis

	B	SE	Z	95% CI
Model 1 (TAS-20 → AN)				
Direct effect	0.05	0.02	2.65**	[0.01, 0.09]
Indirect effect (via SDS)	0.06	0.02	3.55***	[0.03, 0.09]
Total effect	0.10	0.02	5.81***	[0.07, 0.14]
Model 2 (DIF → AN)				
Direct effect	0.09	0.04	2.24*	[0.01, 0.17]
Indirect effect (via SDS)	0.12	0.03	3.62***	[0.06, 0.19]
Total effect	0.19	0.03	5.93***	[0.13, 0.25]
Model 3 (DDF → AN)				
Direct effect	0.06	0.05	1.23	[-0.04, 0.15]
Indirect effect (via SDS)	0.17	0.04	4.35***	[0.09, 0.26]
Total effect	0.19	0.04	4.84***	[0.11, 0.27]

Note. AN diagnosis (0- HC group; 1- AN group); SDS - Zung Self Rating Depression Scale; TAS-20 – Toronto Alexithymia Scale; DIF– Difficulty in identifying feelings; DDF – Difficulty in describing feelings to others; Direct effect - the effect of alexithymia on AN independent of its effect on depression; Indirect effect – the effect of alexithymia on AN through depression; Total effect - the sum of the direct and indirect effects.

*p < 0.05; **p < 0.01; ***p < 0.001