

**Role of Emotion Regulation in Depressive Symptoms and Autism Spectrum Disorder  
Among Adults with Intellectual Disabilities**

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**Abstract**

**Background:** Depressive symptoms in people with autism spectrum disorders (ASD) are common. Transdiagnostic factors as executive functions and emotional regulation strategies can explain the relationship between depressive symptoms and ASD.

**Method:** 121 adults (M = 35.46 years, SD = 9.46) with ASD and intellectual disabilities (ID) were evaluated to determine the predictive and mediating role of executive functioning and emotional regulation strategies.

**Results:** Transdiagnostic variables related to emotion regulation correlated with depressive symptoms; however, executive dysfunction was not associated with depression. Hierarchical linear regression showed emotional regulation strategies were a predictor of depression. A multiple mediation analysis also supported the mediating role of emotional regulation variables between ASD and depression.

**Conclusion:** These findings suggest that emotional regulation (ER) strategies can play an important role in the genesis and development of depressive symptomatology in adults with ASD.

*Keywords:* autism spectrum disorders, depression, transdiagnostic model, emotional dysregulation

### Highlights

- Results supported the relationship between emotional regulation, depressive symptomatology and ASD.
- Transdiagnostic variables had a greater predictive weight for depressive symptomatology than the clinical variables.
- Emotion regulation had a mediating role between ASD and depression symptoms.

## Introduction

Autism spectrum disorder (ASD) is a lifelong neurodevelopmental disorder (DSM-5, APA, 2013) with increasing prevalence. Current prevalence figures indicate that 1 in 59 children receives a diagnosis of ASD (Centers for Disease Control and Prevention [CDC], 2018). ASD is characterized by deficits in communication and social interaction and by repetitive and stereotyped behaviors and interests; however, a significant number of people with ASD now have complex diagnoses (Happé & Frith, 2020). Presentation of other comorbid disorders with ASD is increasing. Several studies propose depression as one of the most common comorbid internalizing disorders in people with ASD (Bruggink et al., 2016; Cassidy et al., 2018). Depressive symptomatology appears more frequently in people with ASD than in both general population and other clinical groups (Gadow et al., 2016; Wigham et al., 2017). Moreover, depressive symptoms in ASD often occur along with other symptoms such as anxiety (Ghaziuddin et al., 2002; Montazeri et al., 2019).

Despite their common comorbidity, research on depressive symptoms in ASD is still emerging since the study of these two conditions is complex. ASD symptomatology and depression often overlap. Symptoms such as withdrawal may be due to ASD or depressive symptomatology (Mayes et al., 2011; Rosen et al., 2018). Furthermore, a high percentage of people with ASD have intellectual disabilities (ID) (Kim et al., 2011; Neece et al., 2015). Diagnosis of depressive symptoms in people with ASD and ID can be extremely difficult as a result of people's verbal and intellectual limitations and professionals having few instruments sensitive to its characteristics (Ghaziuddin et al., 2002; Wigham et al., 2017). In people with ASD and ID, behavioral problems such as tantrums, aggression or self-harm, and changes in the symptoms of ASD are interpreted as warning signs (Ghaziuddin et al., 2002; Jang, 2015).

The relationship and common onset of ASD and depressive symptoms is still not well understood, especially in adulthood. In this regard, a transdiagnostic approach has been used in recent years to study factors underlying the connection to and development of psychopathologies (Ehring et al., 2011). A transdiagnostic approach considers that psychopathological disorders share a set of cognitive and behavioral processes seemingly associated with the origin or maintenance of a group of pathologies (Frank & Davidson, 2014).

Treatment targeting common factors appear to be more beneficial than interventions targeting specific disorders (Egan et al., 2011). Researchers have identified a group of variables as transdiagnostic potentially related to occurrence and maintenance of internalized symptoms in the general population (Aldao et al., 2016; McEvoy & Mahoney, 2012). Many of these transdiagnostic variables have also been linked to the development of anxiety or depression in people with ASD (Bruggink et al., 2016; Hodgson et al., 2016; Mazefsky et al., 2014; Samson et al., 2015). Among these variables, some, such as executive functioning or emotional regulation, are particularly problematic for people with ASD. People with ASD present serious general executive problems in their daily lives, including difficulties with inhibition, flexibility, and generalization of learning (Fletcher-Watson & Happé, 2019). Similarly, researchers identify that people with ASD have serious difficulties with adaptive regulation of their emotions (Bruggink et al., 2016; Cai et al., 2019).

In this sense, numerous studies have proposed how depression is the result of problems people have with regulating emotions (e.g., Aldao et al., 2010; Cai et al., 2018a; Mazefsky, 2015). Emotion regulation (ER) is the process by which people modulate their emotions to appropriately respond to environmental demands (Aldao et al., 2010). People with ASD often use maladaptive regulation strategies thereby increasing their risk of internalized comorbid psychopathology (Bos et al., 2018; Mazefsky & White, 2014; Patel et al., 2017). The most common emotional regulation strategies used by people with ASD are avoidance, victimization (Cervantes & Matson, 2015; Pouw et al., 2013) and rumination (Rieffe et al., 2014). Similarly, people with ID tend to use maladaptive strategies to regulate their emotions (García-Villamizar et al., 2019; Noel, 2018). Therefore, people with ASD and ID are vulnerable to developing depressive symptoms. Despite this vulnerability, research has not addressed in depth the process of emotional regulation in people with ASD and ID.

Research has also proposed that the executive deficit could be a risk factor for developing depression (Gardiner & Iarocci, 2018; Peterson, 2016). Executive functions (EF) refer to a set of cognitive processes involved in physical, cognitive, and emotional self-management, necessary for achieving goals and solving problems (Diamond, 2013). Research identifies a deficit in general executive functions for people with ASD, and although research is unclear about the relationship between EF and depression in ASD, it claims that executive

deficit could put people with ASD at risk of developing depression (e.g., Scult et al., 2017; Stark, 2013). The findings point to a possible link between cognitive flexibility or cognitive rigidity and depressive symptoms, based on evidence linking rumination to depressive symptoms in adults with ASD (Andersen et al., 2015; Crane et al., 2013; Gotham et al., 2015). Moreover, executive problems are related to difficulties of ER, resulting in or maintaining internalized symptoms such as depression (Cai et al., 2018a). Cognitive inflexibility is also related to limited ER and poor coping with situations involving various experiences such as change and novelty leading to or maintaining symptoms such as depression (Kerns et al., 2014).

Considering that recent evidence suggests that EF and emotional dysregulation are important variables in the relationship between ASD and depression, there is value in examining the association of these variables for adults with ASD and ID, a group who have been the focus of limited research to date. Therefore, we implemented a study designed to investigate the role of these transdiagnostic variables (executive functioning and emotional regulation) and their relationship with ASD and depressive symptoms. Results of this study should help to increase understanding of depression in ASD and ID and influence development of models and interventions designed to prevent and reduce symptoms of depression in people with ASD and ID.

Specifically, the following hypothesis will be addressed:

Hypothesis 1. Transdiagnostic variables are expected to be associated with depressive symptomatology in ASD and to be good predictors of that symptomatology (a positive association of executive dysfunction is expected, and a negative association of emotional regulation is expected).

Hypothesis 2. The group of transdiagnostic variables (executive dysfunction and emotional regulation strategies) is expected to have a greater predictive weight than clinical variables for depressive symptomatology in ASD and ID.

Hypothesis 3. Transdiagnostic variables are expected to play a significant mediating role between severity of ASD and depressive symptoms.

## **Method**

### *Participants*

One hundred and twenty-one adults (81 men) aged between 18 and 62 years of age ( $M = 35.46$   $SD = 9.46$ ) with a clinical diagnosis of ASD participated in this study (Table 1). Participants were recruited from health care facilities in the Community of Madrid and Galicia, Spain. Primary inclusion criteria for the participants were being over 18 years old and having a diagnosis of ASD and ID. Prior to the study, psychologists and medical specialists diagnosed participants with ASD and other comorbidities. A chart review was conducted to know this data. However, a screening instrument for ASD traits, DiBAS-R, was included in our research to confirm the diagnosis and assess symptom severity. All participants exceeded the DiBas-R cut-off point. Informed consent was provided by all participants' guardians.

[Insert Table 1]

### *Procedure*

An explanation of the aims of the study was given to all participants and their families who provided informed consent. Participants were recruited from specialized residential health care facilities for people with ASD, consisting of individual homes under 24-hour supervision providing medical, educational, nursing and mental health services. The questionnaires of this research were completed by proxy therapists, who are psychologists or educators and were not included as researchers in the project. For several years, these therapists have been working with the people being evaluated and are their tutors within the center. Each therapist collected information for two or three participants who were evaluated individually at different times with each evaluation session lasting approximately one hour. The instruments selected to be used with this sample have previously been used with people having ASD and ID, some in studies conducted in Spain. Furthermore, each instrument has been back-translated by developers of the original version of the instruments. The project was approved by the ethics committee at the Unit of Psychopathology of the Department of Personality, Evaluation and Clinical Psychology of Complutense University of Madrid (Faculty of Education).

### *Measures*

**Diagnostic Behavioral Assessment for Autism Spectrum Disorder-Revised (DiBas-R; Sappok et al., 2014).** The DiBAS-R is a 20 items other-report scale of ASD traits for adults with ID. Items are rated following the scale: certainly true (3 points), often true (2 points), sometimes true (1 point), and never true (0 points). The cut-off point is 20. Scores higher than

this indicate a possible diagnosis of ASD. Items are distributed in two subscales based on the two symptomatic ASD domains of DSM-5 (APA, 2013). The *Communication and Social Interaction* scale has 12 items and the *Stereotypies, Rigidity and Sensory Abnormalities* scale is comprised of 7 items (Sappok et al., 2014). DiBAS-R has very good psychometric properties. Internal consistency of the total scale is high (.91). For the communication and interaction subscale it is .91 and for the repetitive behavior scale .84 (Sappok et al., 2014). Convergent validity was measured with various ASD diagnostic scales, the highest correlation being achieved with the Autism-Checklist (0.59;  $p < 0.001$ ) (ACL; Sappok, et al., 2014). The sensitivity and specificity of the instrument are 75% according to the work of Mutsaerts et al. (2016). In this study, the total internal consistency was .77, for the communication subscale .83 and for the repetitive behavior subscale .75.

**Autism Spectrum Disorders-Comorbidity for Adults (ASD-CA; Matson & Boisjoli, 2008).** The ASD-CA is an other-report scale of psychopathological comorbidity in adults with ASD and ID. Items are rated with 0 (not at all a problem or a disability), 1 (problem or disability) or X (not applicable or not known). High scores indicate significant comorbid symptoms. ASD-CA has good psychometric properties. Test-retest reliability averaged .54. Reliability was also evaluated with the Kuder-Richardson-20, obtaining satisfactory results (.91) (Matson and Boisjoli, 2008). Its factorial composition consists of five dimensions: Anxiety/Repetitive Behavior, Behavior Problems, Irritability/Behavioral Excess, Attention/Hyperactivity/Impulsive and Depressive Symptoms (Matson & Boisjoli, 2008). The internal consistency of the scale was .91, and in our study was .84.

**Glasgow Depression Scale for people with a Learning Disability - Carer Supplement version (GDS-LD; Cuthill et al., 2003).** The caregiver version of the GDS-LD has 16 items that a person who has a close relationship with the person completes (Cuthill et al., 2003). The GDS-LD is an instrument used for evaluating behaviors related to depression in people with a learning disability and ID; however, some studies (e.g., Fung et al., 2015) successfully used this measure to assess adults with ASD and ID. These behaviors are rated on a scale of 0 (Never/No), 1 (Sometimes), and 2 (Always/A lot). A score of 15 or more indicates clinical depressive symptoms. Test-retest reliability of the caregiver version is very high ( $r = .98$ ), as well as the convergent validity between the self-report and other-report

versions (.93). Internal consistency is 0.88 (Cuthill et al., 2003). Convergent validity of this scale was evaluated with the Beck Depression Inventory II, with a correlation of .88 (Cuthill et al., 2003). The sensitivity and specificity values of the version used in this research reached 96% for sensitivity and 90% for specificity (Cuthill et al., 2003). Reliability for our sample was .80.

### **Dysexecutive Questionnaire of the Behavioral Assessment of the Dysexecutive**

**Syndrome (DEX; Wilson et al., 1996).** DEX is a 20-item instrument addressing everyday dysexecutive function. DEX is used as a general measure of executive dysfunction; however, the scale specifically assesses problems in abstract thinking, impulsiveness, planning problems, time sequencing problems, disinhibition, impulse control difficulties, perseverance, decision making, among other executive skills. This measure is part of the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson et al., 1996). In this investigation, the other-report version was used. This version has been used in numerous investigations evaluating people with ASD with and without ID with good psychometric results (e.g., García-Villamizar et al., 2016; Hagberg et al., 2015). Each item is scored on a 5-point scale from 'never'= 0 to 'very often'= 4. High scores mean greater executive deficits in the person being assessed. Internal consistency reliability is good ( $\alpha = .90$ ) (Burgess et al., 1998). The Spanish version shows a Cronbach's alpha of .91 (.79 in a non-clinical subsample and .92 in a clinical subsample) (Pedrero et al., 2009). In our case, Cronbach's alpha was .87 for the sample of adults with ASD and ID.

**Emotional Regulation Checklist (ERC; Shields & Cicchetti, 1997).** The ERC is an other-report 24-item instrument used to assess emotional regulation (Shields & Cicchetti, 1997). The items are rated on a 4-point Likert scale assessing frequency of behaviors. The scale is divided into two subscales: Emotion Regulation and Lability/Negativity. Emotion Regulation (8 items) assesses the ability to modulate the emotional excitement that favors the person's adaptation to the environment, emotional self-awareness, demonstrations of affection and other adaptive traits such as empathy. The Lability/Negativity (15 items) assesses mood instability, emotional inflexibility, emotional dysregulation, negative affection, emotional reactivity and anger regulation. A high score on the first subscale assumes management of adaptive ER skills; high scores on the second subscale indicate use of non-adaptive ER strategies (Kinkead et al.,

2011). Internal consistency is good for the Lability/Negativity scale ( $\alpha = .90$ ) and somewhat lower for the ER scale ( $\alpha = .79$ ) (Molina et al., 2014). In our investigation, the internal consistency was .83 for the ER scale and .82 for the emotional Lability/negativity scale.

### *Statistical Analysis*

All analyses were completed using SPSS, version 22 (IBM Corp. Released, 2013). A p value  $<0.05$  was considered significant. Descriptive statistics of the demographic and psychological variables were indicated with mean, standard deviation (SD), and number (N) and percentage (%) as appropriate. Pearson's correlation was used to examine correlations among ASD symptoms, depressive symptoms, and other clinical and transdiagnostic variables (Executive Dysfunction, Emotional Regulation and Lability/Negativity).

Multiple hierarchical regression analysis was used to identify predictors of depression in participants with ASD. We tested two different sets of variables: the power of clinical scores and the predictive strength of transdiagnostic variables. Standardized estimate ( $\beta$ ), F,  $R^2$  and  $R^2$ -changes ( $\Delta R^2$ ) for each step were provided. Tolerance and variance inflation factors were used to check for multicollinearity.

Finally, a multiple mediational analysis was performed with the Hayes (2018) macro Process for SPSS (version 3. 2). This analysis is based on regression and path-analysis. Mediation calculates the indirect effect and bootstrapping confidence intervals. An indirect effect is considered significant when in its confidence interval is different from zero (Field, 2013). A parallel model of multiple mediation was executed for the relationship between depressive symptomatology and autistic symptomatology with Executive Dysfunction (M1), Emotional Regulation strategies (M2) Lability/Negativity (M3) as mediating variables. This analysis was performed with 10,000 bootstrapping samples (Hayes, 2018).

## Results

### *Pearson Correlations*

Pearson's bivariate correlations are shown in Table 2. Results revealed that all variables were significantly correlated with each other and were in the expected direction. Specifically, we expected executive dysfunction to be positively associated with depressive symptoms. Results support this association between executive dysfunction and depressive symptomatology ( $r = .297$ ;  $p = .001$ ). We were also expected a positive and significant association between Regulation Emotion strategies and depressive symptoms ( $r = -.497$ ;  $p = .000$ ). Similarly, depressive symptomatology correlated significantly with Lability/Negativity (LN) ( $r = .340$ ;  $p = .000$ ).

[Insert Table 2]

### *Hierarchical Regression Analysis*

Results of the multiple hierarchical regression analyses used to explore predictors of depressive symptoms are shown in Table 3. A multiple hierarchical linear regression analysis was performed for Depressive Symptomatology in ASD following the method of introducing variables by blocks. The independent variables included were those that had the strongest correlation with the Depressive Symptomatology variable. Preliminary analyses were conducted to ensure no violation of the assumptions of normality, linearity, and homoscedasticity. There was no collinearity between variables entered, tolerance and PIV statistics were satisfactory.

[Insert Table 3]

Table 3 shows the regression model for depressive symptomatology. This model explained 35.7% of the variance. In step one we introduced the set of clinical variables (ASD symptomatology -DiBAS-R- and behavioral excesses and irritability -ASDCA-EXC-). In the second step, the group of transdiagnostic (executive dysfunction -DEX-, lability/negativity -LN-, and emotional regulation -ER-) variables was entered.

In step one, ASD symptomatology ( $\beta = .208$ ,  $t = 2.382$ ;  $p = .019$ ) and behavioral excesses ( $\beta = .313$ ,  $t = 3.590$ ;  $p = .000$ ) were significant predictors. In step two, emotional regulation was a predictor statistically significant ( $\beta = -.487$ ,  $t = -6.051$ ;  $p = .000$ ). According to these results, hypothesis 1 is partially confirmed. Transdiagnostic variables were associated with depressive symptomatology, but only emotional regulation strategies were good predictors.

Although not expected, behavioral excesses ( $\beta = .203$ ,  $t = 2.027$ ;  $p = .045$ ) are also a significant predictor of depressive symptoms in ASD.

The change in  $R^2$  for the clinical variables -step 1- was lower ( $\Delta R^2 = .181$ ,  $p = .000$ ) than that offered by the transdiagnostic variables -step 2- ( $\Delta R^2 = .203$ ,  $p = .000$ ). Thus, hypothesis 2 regarding the greater predictive power of transdiagnostic variables over clinical variables was confirmed.

#### *Multiple Mediation Analysis*

A Parallel Model (Model 4) was used to determine whether mediating effects of the transdiagnostic variables on the ASD severity-depression relationship were significant (see Figure 1). The total model explained 60.1% of variation in Depressive Symptomatology.

[Insert Figure 1]

As shown in Table 4, the mediating role of Executive Dysfunction between ASD Symptomatology and Depressive Symptomatology was not statistically significant ( $F_{[1,119]} = 44.392$ ,  $p = .000$ ). Its bootstrapping confidence interval contained zero ( $a_1b_1 = .203$ ,  $SE = .032$ ,  $[-.044, .084]$ ). However, we found that the mediating role of Emotional Regulation between ASD Symptomatology and Depressive Symptomatology was statistically significant ( $F_{[1,119]} = 15.174$ ,  $p = .000$ ;  $a_2b_2 = .108$ ,  $SE = .030$ ,  $[.053, .170]$ ). In addition, Lability/Negativity emerged as a significant mediating variable between ASD Symptoms and Depressive Symptomatology ( $F_{[3,119]} = 18.715$ ,  $p = .000$ ;  $a_3b_3 = 0.064$ ,  $SE = 0.025$ ,  $[.020, .119]$ ). Hypothesis 3 is partially confirmed.

As can be seen in Figure 1 and Table 4, the total effect ( $c = .178$ ,  $SE = .051$ ,  $p = .000$ ) was significant. The direct effect between ASD Symptomatology and Depressive Symptoms was not significant ( $c' = -.015$ ,  $SE = .055$ ,  $p = .783$ ).

[Insert Table 4]

### **Discussion and implications**

The objective of this research was to study depressive symptoms in people with ASD and ID and examine their relationship with a group of clinical and transdiagnostic variables: emotional regulation strategies and executive functions. The results of our study supported the relationship between transdiagnostic variables (emotional regulation), depressive symptomatology and ASD. Other research has also found similar results (e. g., Cai et al.,

2018a,b; Cotrena et al., 2016; Samson et al., 2015). It should be noted, however, that results of this study cannot be fully compared to research on ASD and depression because it was conducted with a very specific and under-researched sample. Furthermore, due to the complex characteristics of the sample, it is difficult to determine directionality of relationships. Other works, such as Cotrena et al. (2016), describe how the symptoms studied here are related in a bidirectional manner.

Specifically, depression symptomatology correlated with the emotional dysregulation variables. Maladaptive emotional strategies were associated with development of depressive symptomatology. Meanwhile, adaptive strategies had an inverse relationship with depressive symptoms. These associations have been described in other studies, with similar findings (Bruggink et al., 2016; Hodgson et al., 2016; Mazefsky et al., 2014; Samson et al., 2015). Several findings identify how adaptive ER is negatively associated with internalized symptoms (Samson et al., 2015), suggesting an important link between them that needs further investigation (Cai et al., 2018b). Their results identified a relationship between self-blame or avoidance, among others, and development of internalized pathologies.

As expected, the group of transdiagnostic variables had a greater predictive weight for depressive symptomatology than the clinical variables. ER strategies were good predictors of depression symptoms in ASD, thus confirming Hypothesis 2. This may indicate that use of adaptive emotional regulation strategies protects against development of depressive symptoms. Similar findings can be found in other studies with people with ASD (Aldao et al., 2010; Cai et al., 2018b; Bos et al., 2018; Mazefsky & White, 2014). For example, Mazefsky et al. (2014) described how use of non-adaptive ER strategies was related to development of internalized symptoms, such as anxiety or depression in this population. As mentioned previously, studies such as that of Cai et al. (2018b) investigated how each of the separate regulatory strategies is related to internalized symptoms. This is interesting and has important clinical implications; however, this detailed analysis could not be performed due to characteristics of measurements used in this study.

Externalized symptoms also were identified as having a positive relationship with depressive symptoms. Specifically, behavioral excesses and irritability (i.e., anger, temper tantrums, rages) proved to be good predictors of depressive symptoms in people with ASD.

These types of maladaptive behaviors have traditionally been associated with depression in people with ASD (Matson & Boisjoli, 2008; Mazzone et al., 2013; Saéz-Suanes et al., 2020). In line with these results, the literature proposes a series of ways of expressing depression in populations with low functioning ASD related to vegetative, somatic and behavioral signs, because of their verbal and expression limitations (Ghaziuddin et al., 2002; Jang, 2015). These include behavioral problems, increased irritability (Gotham et al., 2015) and aggressive behavior (Adams & Matson, 2015). Thus, a significant change in a person's behavior could be interpreted as a sign of internalized problems.

In this study executive dysfunction did not play a predictive role for depression symptoms. The results offered by research are mixed. Most studies show executive deficits in people with depression, indicating them as risk factors for development of depressive disorders (Andersen et al., 2015; Crane et al., 2013; Gotham et al., 2015). In contrast, other research identifies no significant alterations in the EF of people with a diagnosis of depression (Watkins & Brown, 2002). Besides this, in this study we used an ecological and observer's measure to assess executive functioning. Findings from this study may be different and not totally comparable with others in the literature. Most of the work on executive function in ASD uses laboratory instruments, which are difficult to apply in a clinical setting. In addition, the sample in this study had multiple comorbidities, such as epilepsy or ID, which affect executive functioning.

Furthermore, the emotion regulation variables had a mediating role between ASD and depression symptoms. According to these results, the association between two sets of symptoms is better explained by the indirect effect of the ER strategies. Several studies identify these variables as factors of vulnerability to development and maintenance of depression symptoms (Gardiner & Iarocci, 2017; Peterson, 2016). In our literature review, we found research that identify use of maladaptive ER strategies as a transdiagnostic factor to internalize problems and for behavioral alterations in people with ASD (Bos et al., 2018; Patel et al., 2017). In this regard, Conner et al. (2019) have begun work studying comorbid psychopathologies from a transdiagnostic perspective by implementing interventions designed to reduce internalized symptoms. Similarly, Factor et al. (2019) developed a program to improve ER in children with ASD. These studies show that development of such programs is at an early stage and require further scientific examination; however, the researchers claim that initial results are promising.

Although such research focuses on the effects of transdiagnostic treatments on children, it is interesting for our research as it uses similar measures to ours and provides preliminary results on transdiagnostic interventions. According to authors such as Chandrasekhar (2015), to date treatment of depressive symptoms in people with ASD occurs with medication or psychotherapy; however, more research is needed examining effects of other types of interventions designed to manage depressive symptoms. Our findings add to transdiagnostic research by supporting the relationship between ER, ASD and depressive symptoms. These findings increase understanding of depression in adults with ASD and ID and identify the need for interventions and prevention of depression through emotion regulation.

For people with ASD and moderate and severe ID, research has shown that some strategies such as external regulation, behavior alternatives or self-monitoring could be implemented using visual tools such as social stories, especially to regulate negative-value emotions. Provision of spaces for relaxation, recreation activities, and physical activity are also useful in promoting emotional well-being of people with ASD (Bouvet & Coulet, 2015; García-Villamizar & Dattilo, 2010; McClure, Halpern, & Wolper, 2009; McWilliams, de Terte, Leathem, Malcolm, & Watson, 2014).

Several limitations of the present study must be acknowledged. First, it is difficult to assess depressive symptomatology in ASD due to the lack of instruments, especially for people with ASD who also have ID. In addition, the assessment was observational, based on behavior, which could introduce response bias. This type of assessment was conducted because the people who participated in this study lacked enough functional language skills to express their feelings.

Second, although the size of the sample is quite large in comparison with other similar studies, generalization of results should be taken with caution. In addition, the sample used in this study is very specific since they are people who have ASD, ID, and other comorbidities, such as epilepsy. Although this limits our ability to generalize findings to all people with ASD, and complicates interpretation of directionality of identified relationships, studying such a sample is valuable since these individuals are members of an under-researched group. Further studies, which account for these variables will need to be undertaken, to increase generalization of results. Thirdly, although some socio-demographic factors were included in the study, other

factors, such as family history of depression, were not considered. Collecting additional socio-demographic variable will assist researchers in learning more about the relevance of such factors.

In conclusion, results of this study suggest that ER strategies can play an important role in the development of depressive symptomatology in adults with ASD; therefore, these ER strategies could be included in depression reduction therapy of people with ASD. In addition, future research may examine whether rehabilitation designed to alleviate depressive symptoms could lead to significant improvements in other disorders associated with ASDs, such as depression, anxiety, or stress.

**Conflict of Interest:** The authors declare that they have no conflicts of interest. The ethics commission of the Psychopathology Teaching Unit of the Complutense University of Madrid, Spain, reviewed and approved this study.

**Informed consent** was provided by all participants' guardians.

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Table 1.

*Demographic and clinical characteristics of the sample*

Variables	Sample N = 121		
	<i>M</i>	<i>SD</i>	<i>N (%)</i>
Chronological Age (years)	35.46	9.46	
Gender			
Males			81 (66.9%)
Females			40 (33.1%)
ID level			
Mild ID <sup>1</sup>			19 (15.7%)
Moderate ID <sup>2</sup>			38 (31.4%)
Severe ID <sup>3</sup>			29 (24%)
Profund ID <sup>4</sup>			25 (20.7%)
ID not specified <sup>5</sup>			10 (8.3%)
Epilepsy			
Yes			50 (41.3%)
No			71 (58.7%)

Note: Mild ID<sup>1</sup>: IQ range of 50 to 69; Moderate ID<sup>2</sup>: IQ range of 35 to 49; Severe ID<sup>3</sup>: IQ range of 20 to 34; Profund ID<sup>4</sup>: IQ less than 20; ID not specified<sup>5</sup>: characteristics of people prevented a proper assessment of their intellectual level.

Table 2.

*Pearson R correlations between autistic symptomatology, comorbidity and transdiagnostic variables*

	2	3	4	5	6	7	8	9	M	SD	Range
Depression <sup>1</sup>	.302**	.177	.376**	.093	.137	.297**	-.497**	.340**	8.626	3.471	1-16
ASD <sup>2</sup>		.211*	.299**	.147	.186*	.521**	-.373**	.336**	43.314	5.885	25-30
Behavioral Problems <sup>3</sup>			.514**	.524**	.286**	.541**	.225*	.768**	4.396	3.187	0-12
Behavioral Excesses <sup>4</sup>				.251**	.305**	.439**	-.067	.675**	4.975	1.846	0-7
ADHD <sup>5</sup>					.190*	.429**	.321**	.425**	4.775	1.547	0-8
Anxiety <sup>6</sup>						.199*	.098	.320**	2.297	1.388	0-6
Executive Dysfunction <sup>7</sup>							-.093	.648**	57.223	10.126	21-53
ER <sup>8</sup>								.015	17.562	5.108	7-23
Lability/Negativity <sup>9</sup>									38.264	8.496	22-33

\*p <.05 \*\*p <.01

Depression<sup>1</sup> = Glasgow Depression Scale for people with a Learning Disability - Carer Supplement (Cuthill, Espie, & Cooper, 2003). ASD<sup>2</sup> = *Diagnostic Behavioral Assessment for Autism Spectrum Disorder-Revised* Sappok et al., 2014). Behavioral Problems<sup>3</sup> = Behavioral problems subscale of *Autism Spectrum Disorders-Comorbidity for Adults* (Matson & Boisjoli, 2008). Behavioral Excesses<sup>4</sup> = Behavioral excesses and irritability subscale of *Autism Spectrum Disorders-Comorbidity for Adults* (Matson & Boisjoli, 2008). ADHD<sup>5</sup> = ADHD subscale of *Autism Spectrum Disorders-Comorbidity for Adults* (Matson & Boisjoli, 2008). Anxiety<sup>6</sup> = Anxious symptomatology subscale of the ASD-CA of *Autism Spectrum Disorders-Comorbidity for Adults* (Matson & Boisjoli, 2008). Executive Dysfunction<sup>7</sup> = *Dysexecutive Questionnaire of the Behavioral Assessment of the Dysexecutive Syndrome* (Wilson, Alderman, Burgess, Emslie, & Evans, 1996). ER<sup>8</sup> = Regulation subscale of the Emotional Regulation Checklist (Shields & Cicchetti, 1997). Lability/Negativity<sup>9</sup> = Lability/Negativity subscale of the Emotional Regulation Checklist (Shields & Cicchetti, 1997).

Table 3.

*Multiple hierarchical regression analysis for depressive symptomatology in ASD*

	B	T	Sig.	R <sup>2</sup>	ΔR <sup>2</sup>	F
<b>Step 1</b>						
ASD <sup>1</sup>	<b>.208</b>	2.382	.019	.167	.181	13,000***
Behavioral excesses <sup>2</sup>	<b>.313</b>	3.590	.000			
<b>Step 2</b>						
ASD <sup>1</sup>	-.041	-.433	.666	.357	.203	14,328***
Behavioral excesses <sup>2</sup>	<b>.203</b>	2.027	.045			
ER <sup>3</sup>	<b>-.487</b>	-6.051	.000			
Lability/Negativity <sup>4</sup>	.187	1.584	.116			
Executive Dysfunction <sup>5</sup>	.075	.704	.483			

\*\*\*p &lt; .001.

ASD<sup>1</sup> = *Diagnostic Behavioral Assessment for Autism Spectrum Disorder-Revised* (Sappok et al., 2014). Behavioral excesses<sup>2</sup> = Behavioral excesses and irritability subscale of *Autism Spectrum Disorders-Comorbidity for Adults* (Matson & Boisjoli, 2008). ER<sup>3</sup> = Emotional Regulation subscale of the Emotional Regulation Checklist (Shields & Cicchetti, 1997). Lability/Negativity<sup>4</sup> = Lability/Negativity subscale of the Emotional Regulation Checklist (Shields & Cicchetti, 1997). Executive Dysfunction<sup>5</sup> = *Dysexecutive Questionnaire of the Behavioral Assessment of the Dysexecutive Syndrome* (Wilson et al., 1996).

Table 4.

Multiple mediation model of transdiagnostic variables for autistic and depressive symptoms.

Constant																
Antecedent	M <sub>1</sub> (Exec Dysfunction)			M <sub>2</sub> (Emotional Regulation)			M <sub>3</sub> (Lability/Negativity)			Y (Depressive Symptomatology)						
	Coef.	SE	p	Coef.	SE	p	Coef.	SE	p	Coef.	SE	p				
X (ASD Symptom)	a <sub>1</sub>	.896***	.134	<	a <sub>2</sub>	-.310***	.071	<	a <sub>3</sub>	.485***	.124	<	c'	-.015	.055	>.05
M <sub>1</sub> (Exec Dysfunc)	-	-	-	-	-	-	-	-	-	-	-	-	b <sub>1</sub>	.022	.036	>.05
M <sub>2</sub> (ER)	-	-	-	-	-	-	-	-	-	-	-	-	b <sub>2</sub>	-.349***	.057	< .000
M <sub>3</sub> (Lability/Negativity)	-	-	-	-	-	-	-	-	-	-	-	-	b <sub>3</sub>	.133**	.040	< .01
Constant	i <sub>M1</sub>	18.375**	5.883	< .01	i <sub>M2</sub>	31.118***	3.137	<	i <sub>M3</sub>	9.379**	2.708	< .01	i <sub>Y</sub>	9.379***	2.708	< .000
			R <sup>2</sup> = .521			R <sup>2</sup> = .336			R <sup>2</sup> = .368			R <sup>2</sup> = .601				
			F(1,119)=44.392			F(1,119)=15.174			F(3,119)=18.715			F(4,119)=16.442				

Note: i<sub>M1</sub>, i<sub>M2</sub> and i<sub>Y</sub> are regression interceptions, SE = Standard Error.

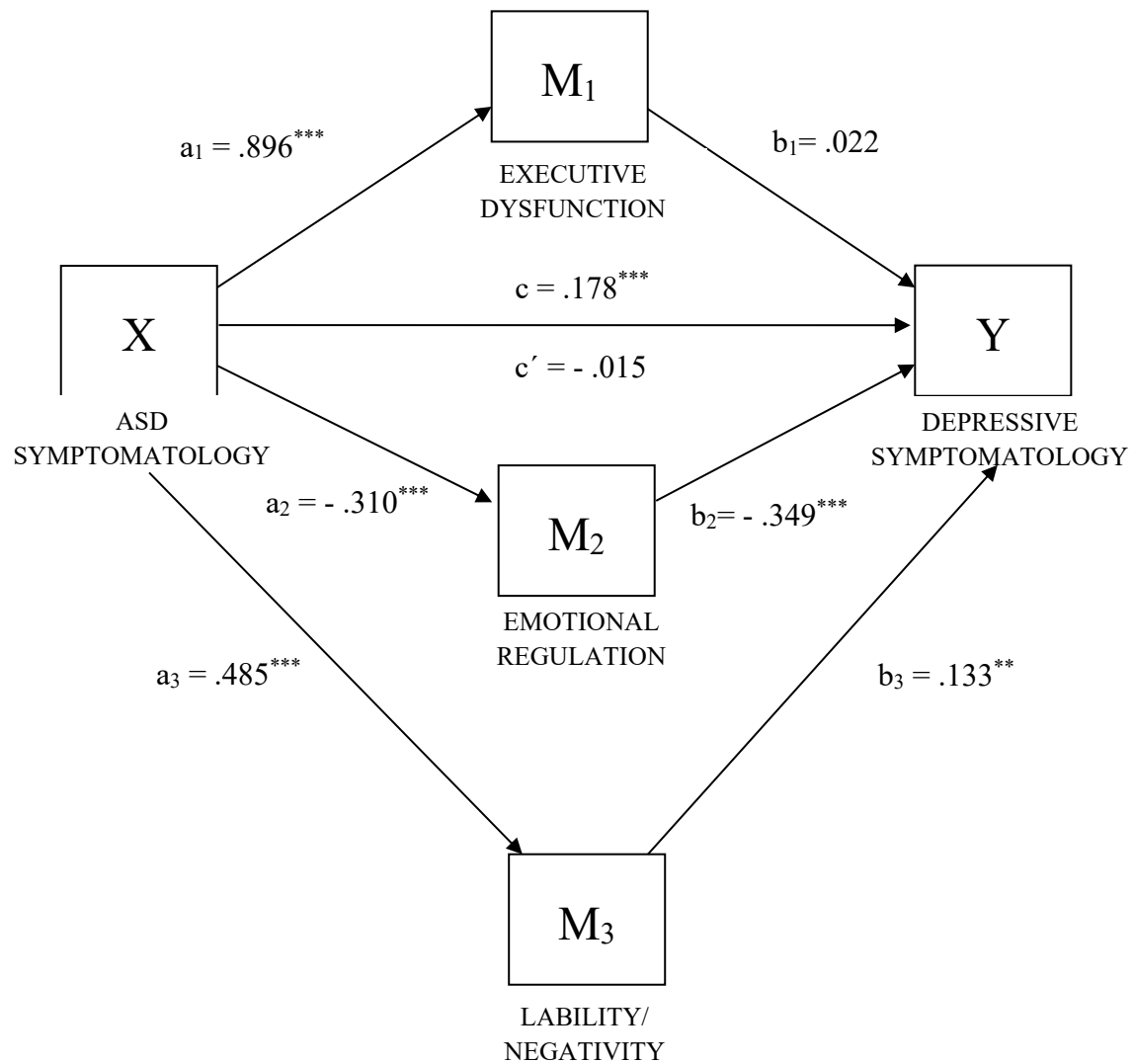


Figure 1. Graphic representation of the multiple mediation model of transdiagnostic variables on autistic and depressive symptoms.

Note:  $a_1$  = direct effect of ASD symptomatology on executive dysfunction;  $b_1$  = direct effect of executive dysfunction on depressive symptomatology;  $a_2$  = direct effect of ASD symptomatology on ER;  $b_2$  = direct effect of ER on depressive symptomatology;  $a_3$  = direct effect of ASD symptomatology on lability/negativity;  $b_3$  = direct effect of lability/negativity on depressive symptomatology,  $c'$  = direct effect of ASD symptomatology on depressive symptomatology;  $c$  = total effect.

\* $p < .05$ ; \*\*\* $p < .00$ .