Mood disorders in high-functioning autism:

The importance of alexithymia and emotional regulation

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Abstract

Individuals with autism spectrum disorder (ASD) often have co-morbid anxiety and depression. Alexithymia and emotion regulation difficulties are commonly seen in individuals with ASD and in mood disorders. We constructed a model hypothesizing a relationship between alexithymia and emotional regulation that would mediate the relationship between autistic features and anxiety/depression symptom severity. We collected data about emotional regulation, alexithymia, autistic symptoms and depression/anxiety in a sample of 64 adults with ASD. We constructed two serial multiple mediator models, using autistic features as the independent variable and anxiety/depression symptoms as outcome variables. The serial relationship between alexithymia and emotional regulation mediated associations between autistic features and depression and anxiety, separately. The findings suggest that targeting alexithymia may benefit therapies designed to alleviate mood disorders in ASD.

Keywords: ASD, Autism, Alexithymia, Emotional regulation, mood disorders

Introduction

Autism spectrum disorder (ASD) is characterized by social impairments, including abnormal communication and restricted interests (APA, 2013). ASD has been extensively studied in the context of social functioning and, as is suggested by the name, this disorder can manifest within individuals across a broad spectrum of severity levels (Gotham, Pickles, & Lord, 2009). Individuals with 'less-severe' ASD (e.g., what was previously diagnosed as Asperger's syndrome), can be very high functioning with often well above-average IQs (Helles, Gillberg, Gillberg, & Billstedt, 2017). Despite being capable of great academic and vocational success, these individuals experience mood disorders at higher rates than the general population (Lugnegard, Hallerback, & Gillberg, 2011; Wigham, Barton, Parr, & Rodgers, 2017). In fact, greater cognitive ability is strongly associated with the co-occurrence of depression and/or anxiety in individuals with ASD (Volkmar, Jackson, & Hart, 2017). Some explanations for this association could be increased awareness of impairment (Barnhill, 2001) or even stress accumulated from negative social experiences in the past (Rosbrook, 2010). However, other contributors to mood disorders in ASD remain to be explored. Alexithymia warrants consideration given its relationship with emotional regulation (Swart, Kortekaas, & Aleman, 2009). Here we present and test a model that posits that autistic features will be associated with

alexithymia, which will in turn be associated with emotional regulation difficulties, causing increased severity of anxiety and depression in individuals with ASD.

Alexithymia is common in ASD (Poquerusse, Pastore, Dellantonio, & Esposito, 2018). Translated to "no words for emotions," alexithymia is characterized by difficulty identifying and describing one's emotions and a tendency to focus on external, rather than internal, experiences (Taylor, 1984). Individuals with alexithymia can be described as having a flat, non-emotional affect (Sifneos, 1994), which can be similar to presentations of ASD (Loveland et al., 1994). While several features of alexithymia, including lack of emotional recognition and reduced empathy, overlap with diagnostic features of ASD (Lord et al., 2000), data suggest that alexithymia and ASD are unique. To illustrate, not all individuals with ASD demonstrate emotional deficits characteristic of alexithymia such as impaired emotional face recognition (Harms, Martin, & Wallace, 2010), and evidence suggests that it is alexithymia, not autism, that contributes to emotional face recognition difficulties (Cook, 2013) . There is also neuroimaging evidence that autism and alexithymia disrupt separate brain networks that underlie different socio-cognitive and socio-affective processes (Bernhardt et al., 2014). It is possible that alexithymia is an independent characteristic that stems from autistic symptoms and contributes to emotional impairments, and ultimately to mood disorders, in those with ASD (Bird & Cook, 2013).

Alexithymia is associated with mood disorders even in those without ASD. Prevalence of alexithymia is high in those with anxiety (Karukivi et al., 2010) and depressive disorders (Honkalampi, Hintikka, Tanskanen, Lehtonen, & Viinamaki, 2000). Individuals with alexithymia and co-occurring depression or anxiety disorders, also tend to present with lower emotional intelligence than individuals without these co-occurring disorders (Onur, Alkin,

Sheridan, & Wise, 2013). Individuals with alexithymia also tend to fare more poorly in treatment for many psychiatric disorders, including mood disorders (Grabe et al., 2008). One potential reason for the high prevalence of mood disorders in individuals with alexithymia is alexithymia's link with emotional regulation (Venta, Hart, & Sharp, 2013).

Emotional regulation involves discerning emotions, selecting reactions to that emotion, and implementing strategies to regulate that reaction (Gross, 2015). Common strategies to regulate emotion include suppression, the tendency to try and ignore emotional responses, and emotional reappraisal, in which individuals reframe the meaning of an emotional stimulus or change their thought patterns (Gross & John, 2003).

Mental health problems in individuals with alexithymia have been attributed to difficulties in emotional regulation (Pandey, 2011). IAs individuals with ASD and alexithymia get older, deficits in emotional regulation may limit success in school and employment (Ashburner, Ziviani, & Rodger, 2010). Even high-functioning individuals with ASD tend to be less effective at emotional reappraisal than those in the general population (Samson, Huber, & Gross, 2012). Individuals with ASD tend to rely on emotion suppression rather than reappraisal (Samson, Hardan, Lee, Phillips, & Gross, 2015), and also tend to use a pattern of avoidance toward emotionally arousing stimuli (Corden, Chilvers, & Skuse, 2008), similar to patterns seen in alexithymia (Bilotta, Giacomantonio, Leone, Mancini, & Coriale, 2015; Laloyaux, Fantini, Lemaire, Luminet, & Laroi, 2015; Panayiotou et al., 2015; Venta et al., 2013). Poor emotional regulation is associated with poorer overall functioning in ASD (Mazefsky et al., 2013). Elevated alexithymia in ASD may further worsen emotional regulation, and thus worsen mental health (Bird & Cook, 2013).

Given the literature on these three points, we hypothesized that alexithymia would be more prevalent in individuals with more autistic features, and alexithymia would be associated with less healthy patterns of emotional regulation, which would then be associated with increased anxiety and depression. In other words, higher scores on a measure of autistic traits will be associated with higher depression and anxiety symptomology scores and this relationship will be mediated by alexithymia through its impact on emotion regulation. Thus, we tested serial mediation models which posited that autistic features would contribute to anxiety and depression, respectively, in high-functioning individuals with ASD indirectly through alexithymia and emotion regulation.

The theoretical serial multiple mediator model posited that alexithymia and emotional regulation mediate the relationship between the independent variable of autistic symptoms and dependent variables of anxiety and depression symptoms, separately. The models also estimate the indirect effects of autistic symptoms on anxiety and depression through 1) alexithymia alone and 2) emotional regulation alone. Gender, years of education (defined as completion of high school, university or post-graduate degrees) and age were also included as covariates in the model. A significant serial mediation in this model may suggest implications for future therapies which may be able to target alexithymia as a key factor related to difficulties in emotional regulation in individuals with ASD and co-occurring mood disorders.

Methods

Procedure

Participants for this study were recruited through online ASD research websites, community boards, outreach/resource websites, and advertising in an ASD research journal, the Journal of Autism and Developmental Disorders. All assessments were completed online using the Qualtrics online survey system. Informed consent was collected prior to any data being collected, and debriefings were provided to all participants following the completion of the study. For taking part in the study, participants were entered into a raffle to win an Amazon gift card. Ethical approval was received from the University of St Andrews University Teaching and Ethical Committee (Ethical Approval Code: PS10557).

Participants

Sixty-eight adults (age range: 18-65 years) from the UK and the USA with a self-reported clinical ASD diagnosis participated. As diagnosis could not be confirmed by clinical assessment due to the online format of the study, ASD phenotype severity was assessed using the Social Responsiveness Scale, Second Edition; (Constantino, 2012) and participants who produced total T-scores in the 'within normal limits' range of 59 or below (n=2) were excluded from analyses. Additionally, two participants were excluded from analyses due to incomplete responses on at least one measure. As a result, the total sample reported in this study is comprised of 64 adults.

All participants (N=64, 17 male, 47 female) additionally completed a demographics questionnaire providing details regarding their gender, age, ASD diagnosis, depression/anxiety diagnostic history, and level of educational achievement (High School, Some/In-Progress University Study, University Graduate, Post-Graduate Study). Participant education status was as follows: 45.3% completed high school, (n=29), 43.8% completed university or college studies

(n=28) and 10.9% completed post-graduate studies (n=9). Participants reported a mean age of 31.67 years (SD=10.27, range 18-65) and a diagnosis of either Asperger's Syndrome (n=52), ASD (n=10), or PDD-NOS (n=2).

Questionnaire Materials

ASD Phenotype Severity: Social Responsiveness Scale, Second Edition (SRS-2)

The SRS-2 (Constantino, 2012) is a quantitative measure of the traits associated with the ASD phenotype that has been designed to align with the updated ASD symptom criteria outlined in the DSM-5 (APA, 2013). This study utilized the self-report Adult version of the SRS-2, which is comprised of 65 statements. Participants rate how well the statement describes their behavior over the past 6 months on a 4-point Likert-type scale ranging from 1 (not true) to 4 (always true). Items on the SRS-2 are designed to assess ASD-related domains of Social Awareness (related to recognition of social cues), Social Cognition (related to interpretation of social cues), Social Motivation (related to motivation to engage in social-interpersonal behavior), Social Communication (related to reciprocal communication with others), and Restricted Interests and Repetitive Behaviors (related to narrow interests and highly routine behavioral patterns). Higher scores on the SRS-2 represent increased levels of ASD-related deficits.

Total scores on the SRS-2 can be translated into T-scores (μ=50, SD=10) to provide insight into comparative ASD phenotype expression severity levels, with scores of 60 and above representing clinically significant deficiencies in reciprocal social behavior resulting in either mild to moderate (60-65), substantial (66-75), or severe and enduring (76+) interference with daily social interactions. The psychometric properties of the SRS-2 have been found to be good, with

strong internal consistency alphas ranging from .92-.95, test-retest reliability ranging from r=.88 to r=.95, and satisfactory to good convergent validity (r=.35 to r=.58) with established ASD diagnostic tools such as the ADOS, ADI-R, and Childhood Autism Rating Scale (CARS)(Bolte, Poustka, & Constantino, 2008; Reszka, Boyd, McBee, Hume, & Odom, 2014).

Depressive/Anxious Symptomology: Depression Anxiety and Stress Scale (DASS)

The DASS (S. H. Lovibond, & Lovibond, P. F. , 1995) is a standardized self-report measure designed to assess the extent to which an individual expresses the cognitive and behavioral symptoms associated with the negative affective states of depression, anxiety, and stress. In total the DASS consists of 42 statements that participants rate on how much the statement applied to them during the preceding week on a 4-point Likert-type scale ranging from 0 (Did not apply to me at all) to 3 (Applied to me very much or most of the time). The statements are equally divided among those designed to measure symptoms of depression (e.g., "I felt I was pretty worthless."), anxiety (e.g., "I was worried about situations in which I might panic and make a fool of myself."), and stress (e.g. "I found it difficult to relax.").

Scores for each scale within the DASS range from 0-42 with higher scores indicating increased expression of the symptoms associated with either depression, anxiety, or stress. Given the focus of the current study, only the depression and anxiety scales are considered. Established severity levels for scores on each scale are as follows: Normal (depression: 0-9; anxiety: 0-7), Mild (depression: 10-13; anxiety: 8-9), Moderate (depression: 14-20; anxiety: 10-14), Severe (depression: 21-27; anxiety: 15-19), and Very Severe (depression: 28+; anxiety: 20+) (Brown, Chorpita, Korotitsch, & Barlow, 1997). The DASS has produced excellent psychometric properties with reported internal consistency alphas ranging from .91-.97 for depression and .84-.92 for anxiety, and strong concurrent validity with well-established related measures of depression (Beck

Depression Inventory: r=.74-.77) and anxiety (Beck Anxiety Inventory: r=.81-.84) in both clinical (Brown et al., 1997) and non-clinical samples (Antony, 1998; Crawford & Henry, 2003; P. F. Lovibond & Lovibond, 1995). This scale has been demonstrated to be reliable and valid when administered in an online setting (Zlomke, 2009).

Alexithymia: 20-item Toronto Alexithymia Scale (TAS-20)

The TAS-20 (Bagby, Parker, & Taylor, 1994) is a widely utilized instruments in both clinical and research settings for the assessment of alexithymia (J. D. Parker, Taylor, G.J., Bagby, M., 2003). The instrument consists of 20 statements that participants are requested to rate on how much they agree or disagree with the ability of the statement to describe their behavior on a 5-point Likert-type scale ranging from 1 (strongly disagree) to 5 (strongly agree). The statements in the TAS-20 assess three interrelated and fundamental alexithymic features: difficulties with identification of one's feelings through one's bodily sensations (e.g., "When I am upset, I don't know if I am sad, frightened, or angry."), difficulties with describing one's feelings (e.g., "It is difficult for me to find the right words for my feelings."), and a concrete, externally oriented thinking style (e.g., "I prefer talking to people about their daily activities rather than their feelings."). Total scores for the TAS-20 range from 20-100 with higher scores indicating increased alexithymia. Typical cut-off scores for this instrument are as follows: 20-51=typical (non-alexithymic), 52-60=intermediate/moderate, 61-120=high/diagnostic level (alexithymic). This instrument has been found to have strong internal consistency (α= 0.77 – 0.86), satisfactory test-retest reliability (r=0.74), and good convergent, discriminant, and concurrent validity across numerous studies (Bagby et al., 1994; Campbell & McKeen, 2011; Dalbudak et al., 2013).The online version of this scale has shown to be comparable reliable and valid (Bagby, Ayearst, Morariu, Watters, & Taylor, 2014).

Emotion Regulation: Difficulties in Emotion Regulation Scale (DERS)

The DERS (Gratz, 2004) is a 36-item self-report questionnaire designed to assess the extent to which an individual struggles with the regulation of negative emotional states. Participants rate each item (statement) on a 5-point Likert-type scale ranging from 1 (almost never) to 5 (almost always) based on how frequently each statement applies to them. The statements in the DERS assess six domains related to emotion dysregulation: non-acceptance of negative emotional responses (e.g., "When I'm upset, I feel guilty for feeling that way."), difficulties engaging in goal-directed behaviors when experiencing a negative emotion (e.g., "When I'm upset, I have difficulty focusing on other things."), struggles with impulse control during a negative emotional state (e.g., "When I'm upset, I have difficulty controlling my behaviors."), limited emotional awareness (e.g., "When I'm upset, I take time to figure out what I'm really feeling."; reverse scored), inadequate access to emotion regulation strategies (e.g., "When I'm upset, I believe that there is nothing I can do to make myself feel better."), and impaired emotional clarity (e.g., "I have difficulty making sense out of my feelings."). Total scores for the DERS range from 36-180 with higher scores indicating greater difficulties with emotion regulation. Psychometric findings for the DERS have produced results suggestive of good internal consistency (α= 0.86-0.94) and test-retest reliability (r= 0.83-0.88), as well as adequate construct, concurrent, and predictive (associations with related behavioral outcomes of deliberate self-harm and partner abuse) validity across multiple studies (Lougheed, 2012; Ritschel, Tone, Schoemann, & Lim, 2015; Ruganci & Gencoz, 2010). Previous studies using internet-administered versions of this scale have demonstrated that the internet version has good reliability and validity (Rusch, Westermann, & Lincoln, 2012)

Analysis

Questionnaires were scored and the total scores collected. Data analysis was performed in SPSS. Descriptive statistics for gender, age, alexithymia (TAS-20 total score), emotional regulation (DERS total score), autistic traits (SRS total score), and anxiety (DASS-Anxiety) and depression (DASS-Depression) were first used to describe the characteristics of the study population.

The theoretical model is illustrated in Figure 1. Models were tested separately for depression and anxiety as the dependent variable using SPSS PROCESS by Hayes (Andrew F. Hayes, 2013) which utilizes bootstrapping. This is a robust method for serial multiple mediation models, and generates an approximation of the distribution of a statistic by repeated random sampling, which is effective in handling limited sample sizes. Models were tested with 5000 resamples and 95% confidence intervals (95%CI) were calculated. Significance was set at $p = .05$ and when 95% CI did not include zero.

SPSS PROCESS utilizes ordinary least squares regression analysis, a widely used method that minimizes the summed squared differences between observed and expected values for fitting linear statistical models (A. F. Hayes & Matthes, 2009). Our analyses examined the direct path between autistic symptoms and anxiety or depression, as well as their indirect paths through alexithymia and emotion regulation levels. The analyses utilized the default model 6 embedded within SPSS PROCESS. We did not modify for additional paths in the mediation model. The model 6 is a serial mediation model which included DASS-Depression or anxiety scores as the Y variable, SRS scores as the X variable, and TAS-Total scores and DERS scores as potential mediators of interest. Age, gender, and years of education were included as covariates. The data

met the assumptions of linearity, homogeneity of error variance, and multicollinearity. Skewness for all variables was within ±.9 and kurtosis for all variables was within ± 1.5.

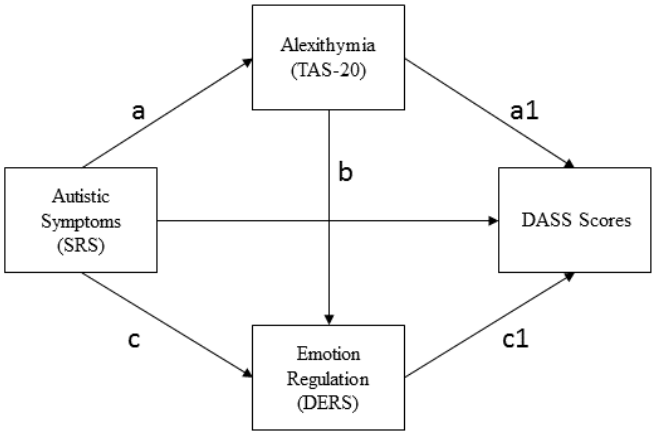

Figure 1: Theoretical Model of Indirect Relationships between Autistic Symptoms, Alexithymia, Emotion Regulation and Mood Outcomes. Indirect Effects of autistic symptom on mood through alexithymia (A-A1); through emotion regulation (C-C1); through alexithymia and emotion regulation (A-B-C1).

Results

Scores on the measures outlined above were collected. 64 individuals completed the SRS, DASS, TAS-20 and DERS. Table 1 illustrates the means and standard deviations for our variables of interest.

Table 1: Means and standard deviations for our variables of interest, alongside the clinical cut-off ranges for certain measures.

Means	Mean	St. Dev	N	Clinical range/cutoff
SRS	114.33	22.73	64	Higher scores indicate more social impairment
DASS Depression	15.09	9.6	64	0-9 (normal) 10-13(mild) 14-20 (moderate) 21-27 (severe)
DASS Anxiety	11.38	6.94	64	0-7(normal) 8-9(mild) 10-14 (moderate) 15-19 (severe)

TAS-20	61.92	14.19	64	<50 (none) 51-61(possible alexithymia) >61 (alexithymia)
DERS	108.00	23.32	64	Higher scores indicate worse regulation

SRS = Social Responsiveness Scale, DASS = Depression and Anxiety Stress Scales, TAS-20 = Torontro Alexithymia Scale, DERS = Difficulties in Emotional Regulation Scale; GP = General population

In our sample of adults with ASD, 20.5% reported no history of depression, 41.1% reported a current diagnosis of depression and 38.4% reported a past diagnosis of depression. For anxiety, 27.4% reported no current or past history of anxiety disorders, 45.2% reported a current diagnosis and 27.4% reported a past diagnosis. When we compared means of our questionnaires with presence of a current or past diagnosis, we found indications that presence of past or present anxiety disorder was associated with higher scores on the DASS-anxiety scale ($t = 3.7$, $p < .04$), but not with TAS-20 scores, DERS scores, or SRS scores (all $p > .5$). Similarly, presence of past or present depressive disorder was not associated with differences in DASS-Depression scores, TAS-20 scores, DERS scores, or SRS scores (all $p > .3$). Means for our questionnaires assessing mental health and related features are presented in table 2.

Comparisons with mental illness history

Anxiety disorder

		SRS total		DASS-Anxiety		TAS-Total		DERS Total	
	N	**Mean**	**Std. Dev**	**Mean**	**Std. Dev**	**Mean**	**Std. Dev**	**Mean**	**Std. Dev**
Never	19	116.05	19.352	9.84	4.729	62.42	13.525	105.4	20.726
Current	27	115.07	22.506	13.67	7.815	63.59	12.401	113.3	21.803
Previous	18	111.39	27.038	9.56	6.853	58.89	17.432	102.9	27.467
Total	64	114.33	22.736	11.38	6.941	61.92	14.2	108	23.322

Depressive disorder

	SRS total	DASS-Depression	TAS-Total	DERS Total

	N	Mean	Std. Dev	Mean	Std. Dev	Mean	Std. Dev	Mean	Std. Dev
Never	14	113.57	15.931	9	8.602	61.36	12.695	102.7	24.222
Current	22	112.18	21.973	20.82	11.342	61.23	12.862	113.6	20.839
Previous	28	116.39	26.457	13.64	5.807	62.75	16.215	106.3	24.623
Total	64	114.33	22.736	15.09	9.656	61.92	14.2	108	23.322

It should be noted that a surprisingly large proportion of our sample (84%) was female. As ASD is more common in males (Lyall et al., 2017), we performed a gender comparison for our variables of interest. However, contrary to our expectations, males (M = 106.6, SD = 23.8) demonstrated fewer autistic features than females (M = 119.1, SD = 22.3) ($F_{1,71} = 4.3$, $p < .05$) according to scores on the SRS. However, there were no other gender differences found in our variables of interest (all p values > .1).

Mediation Analysis

Depression: Results of serial multiple mediation analysis of the pathway between autistic symptoms and depression scores through alexithymia and emotion regulation, while controlling for gender, education level and age, are shown in Figure 2. Gender showed no direct effects with any of our variables (all p > .5), age showed no direct effects with any of our variables (all p > .2), and education level showed no direct effects with any of our variables (all p > .1). Autistic symptoms were associated with alexithymia (b=.26, se=.07, p<.001) and accounted for significant variance in alexithymia (R^2=.19, F(2,61)=4.69, p=.002). Alexithymia (b=1.07, se=.17, p<.001), but not autistic symptoms (b=.06, se=.11, p=.59), was associated with emotion regulation, and accounted for significant variance in emotion regulation (R^2=.47, F(3,60)=13.4, p<.001). Autistic symptoms (b=.1, se=.05, p=.8) were not associated with depression, but

emotion regulation (b=.24, se=.06, p<.01) and alexithymia (b=-.23, se=.11, p< .03) was. The effect of emotional regulation explained significant variance in depression (R^2=.27, F(4,59)=4.3, p=.001). The indirect effects of autistic symptoms on depression, individually through alexithymia (b=-.03 se=.10, 95%CI=-.112, .028) or through emotion regulation (b=.02, se=.03, 95%CI=-.04, .09) were not significant. However, the serial indirect effect of autistic symptoms, through alexithymia that contributes to emotion regulation, on depression was significant (b=.06, se=.03, 95%CI=.02,.15). Together, these results indicate a serial mediation of autism symptoms and depression through alexithymia and its contribution to emotion regulation.

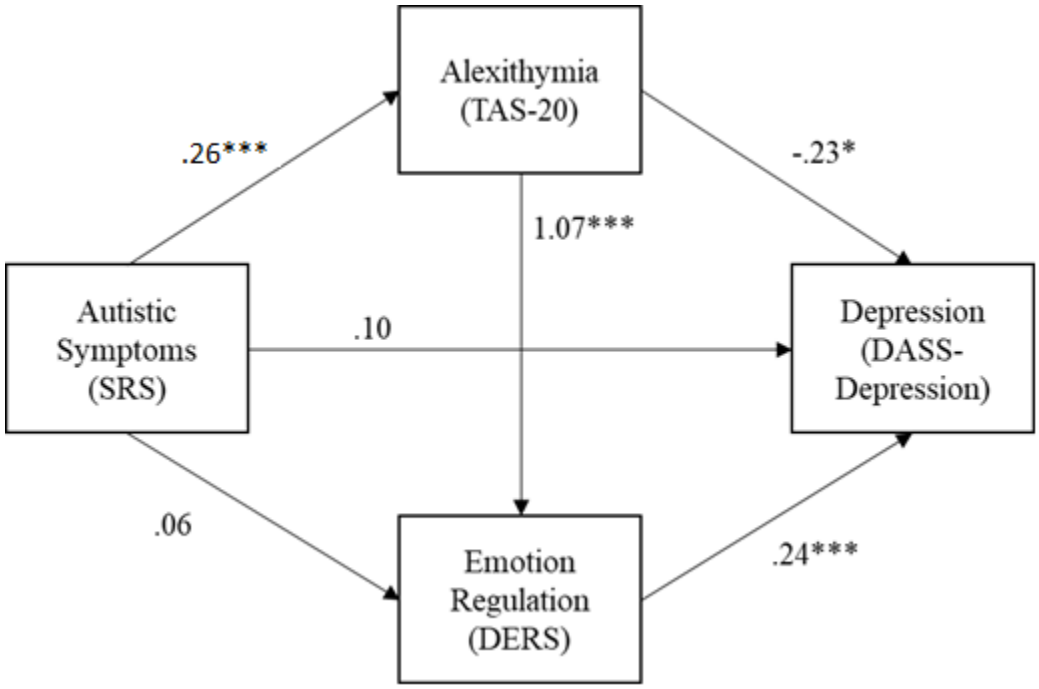

Figure 2: Serial multiple mediation results of the relationship between autism symptoms, alexithymia, emotion regulation, and depression. *p≤.05, **p≤.01, ***p≤.001.

Anxiety: Results of serial multiple mediation analysis of the pathway between autistic symptoms and anxiety scores through alexithymia and emotion regulation, while controlling for gender, are shown in Figure 2. Gender showed no direct effects with any of the variables (all $p > .5$), and age additionally showed no direct effects with any of our variables (all $p > .2$). Similar relationships between autistic symptoms, alexithymia, and emotional regulation were present as in the model including depression. Alexithymia (b=-.13, se=.08, p=.19) was not associated with anxiety, but greater autistic symptoms (b=.08, se=.03, $p < .04$) and emotion regulation (b=.15, se=.04, $p<.001$) was. The effect of emotional regulation explained significant variance in anxiety (R^2=.24, F(4,59)=3.7, p=.001). The indirect effects of autistic symptoms on anxiety, individually through alexithymia (b=-.03 se=.0, 95%CI=-.07, .02) or through emotion regulation (b=.01, se=.02, 95%CI=-.03, .06), were not significant. However, the serial indirect effect of autistic symptoms through alexithymia that contributes to emotion regulation on anxiety was significant (b=.06, se=.02, 95%CI=.01,.1). Together, these results indicate a serial mediation of autism symptoms and anxiety through alexithymia and its contribution to emotion regulation.

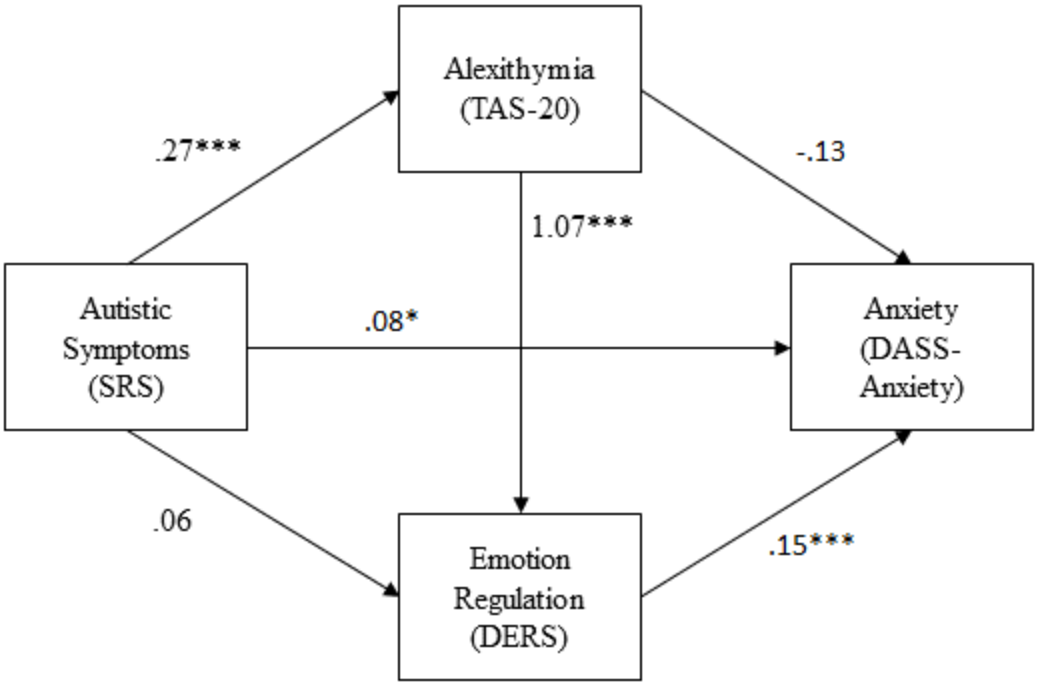

Figure 3: Serial multiple mediation results of the relationship between autism symptoms, alexithymia, emotion regulation, and anxiety. *p≤.05, **p≤.01, ***p≤.001.

Correlations

Intercorrelations between autism symptoms, alexithymia, emotion regulation, depression and anxiety are shown in Table 2. For both models, autism symptoms were significantly associated with proposed mediators of alexithymia ($r = .43$, $p < .01$) and emotion regulation ($r = .352$, $p < .01$). Furthermore, alexithymia was associated with the mediator of emotion regulation ($r = .68$, $p < .001$).

For measures of anxiety, the independent variable of autism symptoms was significantly associated with the primary dependent variable of anxiety scores ($r = .28$, $p < .03$). Alexithymia was associated with anxiety ($r = .316$, $p < .02$), and emotion regulation was associated with

anxiety (r= .575, p= .001). In addition, correlation analyses were conducted between DASS anxiety scores and the bivariate variable of any current or past anxiety disorder, as reported by the participants. However, results showed no significant correlation between DASS scores and presence of anxiety disorder (r = .145, p = .25).

For measures of depression, the independent variable of autism symptoms was not significantly associated with the primary dependent variable of depression scores (r = .18, p = .13). However, alexithymia was associated with depression (r = .29, p < .02), and emotion regulation was associated with depression (r= .54, p < .001). In addition, correlation analyses were conducted between DASS depression scores and the bivariate variable of any reports of current or past depressive disorder, as reported by the participants. This correlation was significant (r = .337, p < .01), where individuals with higher DASS depression scores were more likely to report past or present depressive disorder.

Table 3: Correlations between the variables of interest:

Correlations (r)	1	2	3	4
1. Autism Symptoms (SRS)				
2. Alexithymia (TAS-20)	0.43***			
3. Emotion Regulation (DERS)	0.35**	0.68***		
4. Anxiety (DASS-Anxiety)	0.28*	0.31*	0.57***	
5. Depression (DASS-Depression)	0.18	0.29*	0.54**	0.55***

*p≤.05, **p≤.01, ***p≤.001;

Discussion

We constructed serial mediation models to test our hypotheses that autistic symptoms would be associated with anxiety and depression symptoms through alexithymia's relationship with emotional regulation. Considering emotions that one may not identify well would be very difficult to regulate, we hypothesized that alexithymia would worsen emotional regulation, thus

contributing to worse anxiety and depression symptomatology in individuals with ASD. Our mediation analyses supported our hypotheses. There were strong associations between alexithymia and emotional regulation, and this relationship serially mediated the association between autism symptoms and symptoms of anxiety and depression. Our findings suggest important implications for alexthymia in mental health in individuals with ASD. Results may be particularly relevant for females with ASD, as our sample was predominantly female.

Despite demonstrating relatively high functioning, individuals in the sample showed high levels of alexithymia, with an average TAS-20 score of 61, which is the threshold suggested for a classification of alexithymia (Bagby et al., 1994). This is consistent with previous reports of increased prevalence of alexithymia in samples of individuals with ASD (Berthoz & Hill, 2005). Participants also demonstrated relatively high scores for depression (15) and anxiety (12) on the DASS subscales, with averages in the moderate range for depression and anxiety symptoms (S. H. Lovibond, & Lovibond, P. F. , 1995). A large portion of our sample also reported lifetime diagnoses of depression or anxiety, with roughly 30% reporting depression and nearly half reporting anxiety disorders. In addition, total emotion-regulation scores were comparable to those in another sample of individuals with ASD (Swain, Scarpa, White, & Laugeson, 2015), and relatively high in contrast to a sample of healthy comparison subjects (Becerra, 2013). These findings suggest that the current sample demonstrated difficulties in emotional regulation similar to that observed in other samples of individuals with ASD. In addition, our sample reported high prevalence of autistic symptoms as indicated by high scores on the SRS. Interestingly, the females in our sample showed higher scores on the SRS than the males, and the large proportion of females for ASD samples may in part underlie the high SRS scores.

The results of the serial mediation analyses support our hypotheses regarding the contribution of autism symptoms on anxiety and depression through its effects on alexithymia and emotion regulation. The relationship we identified in this population of adults with ASD indicates that alexithymia, which encompasses impaired emotional identification and labeling, may disrupt regulation of emotion and ultimately increase severity of symptoms for anxiety and depression. Better capabilities or tendencies with respect to labeling emotions are important aspects of emotional intelligence (J. D. A. Parker, Taylor, & Bagby, 2001). Increased alexithymia during a stress task has been linked to higher reports of negative affect despite identical autonomic measures of stress (Connelly & Denney, 2007), suggesting that alexithymia may lead to poorer regulation of negative affect.

Our data suggest the possibility that alexithymia, through its relationship with emotional regulation, contributes to greater symptoms of anxiety and depression in ASD, and this may occur in relation with the social deficits in ASD. The link between ASD and alexithymia may be complex, whereby an initial autistic symptoms may include poor social communication that may impede the development of emotional intelligence and increase alexithymia (Poquerusse et al., 2018). Of course, many other factors may affect emotional intelligence and awareness, including gender, age, and socio-economic status (Mankus, Boden, & Thompson, 2016). Gender is particularly relevant as our sample was heavily skewed toward females. Autistic individuals in particular may develop avoidance of social or emotional situations due to negative experiences in their past (Ainslie Rosbrook, 2010). In addition, evidence suggests that emotional intelligence in particular may have a strong biological basis (Petrides et al., 2016).

Emotional intelligence, which may be considered as being inverse to alexithymia (J. D. A. Parker et al., 2001), is an important construct, and is associated with better well-being and

resilience (Armstrong, Galligan, & Critchley, 2011), including reduced anxiety and depression in younger people (Fernandez-Berrocal, 2006). Alexithymia, by negatively impacting emotional regulation, may contribute to poorer development of overall emotional intelligence (Naghavi & Redzuan, 2012) and lead to mood disorder. Targeting alexithymia in ASD, as part of a combined therapy that also targets other complicating factors such as past negative experiences and coming to terms with and accepting impairment, may thus help those individuals, especially with regard to decreasing the incidence, prevalence and severity of mood disorders.

Poor social responsiveness may make alexithymia worse, as learning how to label emotions and demonstrate social reciprocity may be challenging for individuals with ASD. Poor social responsiveness may also worsen overall mental health, especially in treatment which may rely on communication between a patient and a therapist. While our model suggests that alexithymia in ASD leads to emotional regulation difficulties and then worse anxiety and depression, the underlying basis of alexithymia in ASD is still unclear. One theory posits that alexithymia is developed early by a lack of social interaction in childhood and a failure to learn proper emotional labeling, which is supported by findings of alexithymia in those individuals who suffered childhood neglect (Aust, Hartwig, Heuser, & Bajbouj, 2013). For those with ASD who do not seek out or resist social interaction, increased symptoms of autism may worsen alexithymia, underscoring our finding of an association. There is also evidence of a biological basis to alexithymia, as it can be acquired following damage to the anterior insula (Hogeveen, Bird, Chau, Krueger, & Grafman, 2016). In addition, individuals with alexithymia may not seek, or perform as well in, mental health treatment (Grabe et al., 2008), leading to worse outcomes in depression and anxiety, and this may be true in individuals with ASD. However, with the

knowledge that alexithymia may worsen emotional regulation and mental health, future treatments should be examined that target alexithymia in ASD.

Limitations of the current study include the small sample, which was alleviated somewhat by the use of bootstrapping in our models. Additionally, while there was no significant association found between gender and any of the variables of interest in this study, the large proportion of females also makes the findings less generalizable to the general population of those with ASD, which is skewed towards males (Lyall et al., 2017). The age range of our sample is also very broad, at 18-65 years, and while age did not play a role in our findings, future studies with greater power should consider age as a potentially important factor in the severity of mental illness. We included covariates of age, gender and education, but other unmeasured confounders (such as SES) may play a role. Another limitation is the lack of longitudinal data, as all data were collected at one time point. Collection and analysis of longitudinal data may examine whether alexithymia predated temporally difficulties in emotional regulation, anxiety and depression to provide further understanding of their relationships. It is possible that emotional regulation or depression may in fact contribute to alexithymia. However, given the strong theoretical link presented between alexithymia and emotional regulation and the small number of studies examining potential mediation effects through alexithymia and emotion regulation, the current results provide initial support of a pathway between autism and mood disorders such as anxiety and depression. Future data should examine alexithymia and emotional regulation in larger samples and over longer time periods. It should also be acknowledged that difficulty in identifying emotions in those with alexithymia may complicate accurate reporting of anxiety and depression. However, investigators (e.g. Berthoz & Hill, 2005; Hill, Berthoz, & Frith, 2004; Cederlund et al., 2010) have noted that adults with ASD without co-occurring intellectual

disability are not only aware of any emotional problems with which they may be struggling, but are fully capable of accurately reflecting on that awareness in commonly utilized self-report measures of depressive and anxious symptomology (in comparison to both clinical interviews and caregiver/parent reports). An additional concern is that recruitment specifically of individuals with depression or anxiety may have biased our results. However, the levels of depressive symptomology and emotion dysregulation found in our sample align with what has been published in other studies examining these topics in adults with ASD (Wigham et al., 2017).

Finally, while conducting the study in an online format allowed for broader participant recruitment, and was potentially a more comfortable research format for individuals with HFASD who might be discouraged by the idea of coming into a research lab and having to directly interact with investigators, some research control was lost as a result. Specifically, this format did not allow for confirmation of diagnosis through clinical assessment, and researchers were not able to control the environment in which the study was conducted, thus being unable to ensure the participant was not multi-tasking or being distracted by their surrounding environment while completing the study components.

In sum, our results indicate that alexithymia may relate importantly to mental health in individuals with ASD, specifically through its relationship with emotional regulation. As alexithymia is common in individuals with ASD, therapies that address anxiety and depression in individuals with ASD should consider alexithymic characteristics.

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Contributors

Dr. Morie wrote the first draft of the paper and worked with co-authors on subsequent drafts. All authors contributed to the editorial process and have approved the final submitted version of the manuscript.

Conflicts of Interest

The authors report no conflict of interest with respect to the content of this manuscript.

Dr. Potenza has consulted for and advised Shire, Rivermend Health, Opiant/Lightlake Therapeutics and Jazz Pharmaceuticals; received research support (to Yale) from the Mohegan Sun Casino and the National Center for Responsible Gaming; participated in surveys, mailings, or telephone consultations related to drug addiction, impulse control disorders or other health topics; consulted for and/or advised legal and gambling entities on issues related to impulse control and addiction; provided clinical care in the Connecticut Department of Mental Health and Addiction Services Problem Gambling Services Program; performed grant reviews for the National Institutes of Health and other agencies; has guest-edited journal sections; given academic lectures in grand rounds, CME events and other clinical/scientific venues; and generated books or chapters for publishers of mental health texts. The other authors report no financial relationships with commercial interests.

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