Received: 26 April 2023 | Revised: 16 September 2023 | Accepted: 17 October 2023

DOI: 10.1002/mhs2.42

RESEARCH ARTICLE

WILEY

Understanding the associations between social and emotional expression, communication, and relationships in individuals with eating pathology

Alyssa Daniels[1] | Talea Cornelius[2] | Amy A. Gorin[1]

[1]Department of Psychological Sciences, University of Connecticut, Storrs, Connecticut, USA

[2]Division of General Medicine, Columbia University Irving Medical Center, Presbyterian Hospital, New York, New York, USA

Correspondence
Alyssa Daniels, Department of Psychological Sciences, University of Connecticut, 406 Babbidge Rd, Storrs, CT 06269-1248, USA.
Email: alyssa.daniels@uconn.edu

Funding information
BOLD Women's Leadership Network

Abstract

Research suggests that a disproportionate number of female individuals being treated for an eating disorder (ED) also have autism spectrum disorder (ASD). Alexithymia, or difficulty identifying and describing emotions, may mediate the relationship between ED and ASD. In this study, we explored the association of autistic traits with symptoms of alexithymia and eating pathology, as well as the potential mediating role of alexithymia. Two hundred and twenty-eight female participants aged 18 and older were recruited from online ED support platforms to complete an anonymous online survey via Qualtrics. The survey included three questionnaires: the Toronto Alexithymia Scale-20, the Autism-Spectrum Quotient (AQ), and the 13-item Eating Disorder Examination Questionnaire. More than half (54.8%) of participants met the clinical threshold on the AQ. Participants with a positive screen on the AQ scale also reported more symptoms of alexithymia (92.6% of individuals with a positive AQ vs. 79.8% of those without), $B = 9.02$, $p < 0.001$. A positive AQ screen was also associated with significantly greater disordered eating symptoms, $B = 4.26$, $p = 0.031$. Alexithymia mediated this association, $a \times b = 1.98$, $p < 0.05$. The results establish a strong positive relationship between autistic traits and alexithymia, supporting previous data and suggesting that autistic female individuals struggle to identify emotions. Additionally, alexithymia served as a mediator between autistic traits and disordered eating. Understanding this relationship may help inform the treatment of autistic female individuals who are also struggling with ED.

KEYWORDS
alexithymia, anorexia nervosa, autism, bulimia nervosa, eating disorders

1 | INTRODUCTION

Recent studies suggest that a disproportionate number of individuals being treated for an eating disorder (ED) are also on the autism spectrum. Evidence indicates that around 23% of individuals with ED endorse traits of autism (Huke et al., 2013; Westwood, Mady, et al., 2017). Conversely, about 1% of the general population meets the clinical cutoff for autism symptomatology (Brugha et al., 2012). This differs by sex/gender, with even lower rates of autism reported in women/female individuals (Brugha et al., 2012). Autistic female individuals are more likely to have a delayed or missed diagnosis than male individuals with autism (Fusar-Poli et al., 2022). It has been

suggested that this could be due to increased internalized autistic traits in females, which may in turn make them more vulnerable to anxiety, depression, and eating disorders (Bargiela et al., 2016; Mandy et al., 2012). The heightened co-occurrence of autism in female individuals with ED has been extensively researched in the context of anorexia nervosa (AN) (Demartini et al., 2021; Westwood et al., 2018). In a sample of 132 female individuals (66 with AN and 66 healthy controls), Tchanturia et al. (2013) found that 25.8% of participants with AN met the clinical cutoff for autism, while only 1.5% of healthy control participants scored within the clinical range. Similarly, in a sample of 30 female ED patients, 23% of them met clinical criteria for autism (Wentz et al., 2005), and a cross-sectional observational study of female individuals with AN in both inpatient and day-patient treatment programs found that 10% of subjects had potentially diagnosable autism spectrum disorder (ASD), as determined by both developmental history and the second edition of the Autism Diagnostic Observation Schedule (ADOS-2), while 40% showed ASD symptomatology (Westwood et al., 2018). A significant relationship between traits of autism and Avoidant/Restrictive Feeding Intake Disorder (ARFID) has also been identified (Fisher et al., 2014; Nicely et al., 2014). Research on the co-occurence of autism with bulimia nervosa (BN) or binge eating disorder (BED) is more limited; however, one study suggests a link between autism and BN/BED as well (Gesi et al., 2017).

In addition to a co-occurence of autism and ED diagnoses, there appears to be significant behavioral overlap between these diagnoses. Individuals with AN may display traits of autism including high levels of negative affectivity, blunted facial affect, perfectionism, deficits in theory of mind, and challenges interpreting emotional states of others (Bora & Köse, 2016; Leppanen et al., 2018; Zhou et al., 2018). Traits of poor executive functioning typical to autistic individuals such as challenges with planning and organization have also been reported in women with AN (Gillberg et al., 2010; Olde Dubbelink & Geurts, 2017; Råstam, 1992; Tchanturia et al., 2013; Wentz et al., 2001). Although some argue that autistic traits may arise as a result of malnourishment in individuals with eating disorders (Bentz, Jepsen, Kjaersdam Telléus, et al., 2017; Dapelo et al., 2016; Davies et al., 2013; Leppanen et al., 2017; Morris et al., 2014), a 2018 study found that improvement in eating disorder symptoms after 12 months appeared to be similar in individuals both with and without characteristics of autism, but social difficulties remained significant in participants with characteristics of autism, indicating that these autistic traits are not simply due to the cognitive impact of starvation (Nazar et al., 2018).

Autistic individuals also exhibit symptomology reflective of eating pathology. Rituals around food and high food selectivity are common in autism, similar to AN symptoms (Karjalainen et al., 2019; Spek et al., 2020; Vissoker et al., 2015). For example, in a qualitative study by Brede et al. (2020), autistic participants reported challenges with food texture and breaking the cycle of repetitive eating behaviors, which they attributed to their autism. The mechanisms underlying these eating behaviors may differ between autism and AN, however. Rather than cognitions traditionally identified in AN such as body dysmorphia (American Psychiatric Association, 2013), physiological mechanisms play a central role in autism. Decreased interoceptive awareness is a common trait of autism and may pose challenges when identifying hunger versus satiety (DuBois et al., 2016). It has also been suggested that food selectivity may be due to sensory processing challenges in autistic individuals (Spek et al., 2020). Many individuals on the autism spectrum may experience gastrointestinal challenges such as chronic abdominal pain, constipation, chronic diarrhea, and gastroesophageal reflux disease (Vissoker et al., 2015). As such, ED symptoms identified in autistic individuals may be worsened by underlying physiological challenges rather than a cognitive response to body image, suggesting that alternative treatment methods may be required to better serve this population (DuBois et al., 2016; Karjalainen et al., 2019).

ED and autism share an additional commonality that has not been well studied—the co-occurrence of alexithymia. Individuals who endorse traits of alexithymia may experience difficulties identifying emotions, describing emotions to others, and differentiating emotions from physical sensations. They may also show limitations in imagination and externally oriented thinking (Goerlich, 2018; Timoney & Holder, 2013). It is estimated that roughly half of autistic individuals also endorse traits of alexithymia (Cole et al., 2023; Kinnaird et al., 2019; Poquérusse et al., 2018). Prevalence rates of alexithymia in individuals with AN have been reported to be 77.1% (compared to 6.7% in healthy controls) (Bourke et al., 1992). Alexithymia is also more common in individuals with BN and BED (Westwood, Kerr-Gaffney, et al., 2017). In a sample of 93 individuals with BN, Schmidt et al. (1993) found that 50% met the clinical threshold for alexithymia, while only 19% of the 95 healthy controls met this criteria. Similarly, in a sample of 83 individuals with BED, 24.1% met the clinical threshold for alexithymia, exceeding the 11.1% among the control group (de Zwaan et al., 1995). The discrepancy in prevalence of alexithymia in both autism and ED populations as compared to the general population suggests that alexithymia could be a mediating factor between autism and eating pathology (Moseley et al., 2023; Vuillier et al., 2020).

Given that long-term treatment outcomes are significantly worse for individuals with comorbid AN and autism (Morgan & Hayward, 1988; Nielsen et al., 2015), and that research suggesting that traits following ED recovery including social difficulties, decreased empathy/emotional recognition, reduced eye contact, detail processing, and cognitive rigidity appear to persist following treatment (Bentz, Jepsen, Pedersen, et al., 2017; Danner et al., 2012; Harrison et al., 2010, 2019; Lindner et al., 2013; Lopez et al., 2009), it is crucial to further explore whether autism could be a precursor to eating pathology in order that more effective treatment alternatives and mitigation strategies may be implemented. Examining the role of alexithymia as a potential mediator of the relationship between autism and ED is also critical. Though patients can recover from ED, alexithymia symptoms may remain high even following treatment (Pinna

et al., 2014). This is especially problematic when considering the implications associated with alexithymia including increased risk of suicide, self-injury, and substance abuse, as well as decreased response to psychological therapy and drug treatment (Cruise & Becerra, 2018; Hemming et al., 2019; Norman & Borrill, 2015; Ozsahin et al., 2003; Taylor, 1984).

Expanding on earlier work examining the co-occurence of autism and ED, as well as research by Moseley et al. (2023) and Vuillier et al. (2020), the present study examined the associations of autism, ED, and alexithymia in a sample of female individuals recruited from online eating disorder support platforms. The study hypotheses were as follows. First, it was expected that autism symptomatology in this sample would significantly exceed the population estimate of 1% (Brugha et al., 2012). Second, it was hypothesized that individuals with greater autism symptomatology would have a higher prevalence of alexithymia. Third, it was hypothesized that individuals endorsing traits of autism would be positively associated with ED symptomatology. Finally, it was hypothesized that alexithymia would serve as a mediator of any positive association between autism and disordered eating behaviors.

2 | METHODS

2.1 | Participants

Participants were recruited from online eating disorder support platforms in summer 2022. These online platforms are self-organized within the ED community and serve to support individuals struggling with AN, BN, and Eating Disorders Not Otherwise Specified. Inclusion criteria were as follows: (1) female; (2) aged 18 and older; (3) have been treated for (or are currently being treated for) or have been told by a medical professional that they have an eating disorder; and (4) consent to participation.

2.2 | Procedure

This was an anonymous, cross-sectional, online survey distributed via virtual support platforms. Interested participants clicked a link that directed them to an online Qualtrics survey. Those who consented to participate then completed the survey. All participants provided informed consent, and all study procedures were approved by the University of Connecticut Institutional Review Board. The order of the survey was as follows: Toronto Alexithymia Scale-20 (TAS-20), Autism-Spectrum Quotient (AQ), and 13-item Eating Disorder Examination Questionnaire (EDE-Q-13). On average, the survey took participants approximately 18.2 min to complete. Participants were not made aware of the study goals beyond that this was a study on the emotional and social experiences of female identifying individuals with eating disorders. Participants were made aware at the beginning of the survey that potentially triggering questions could be skipped.

2.3 | Measures

2.3.1 | Alexithymia

Alexithymia was assessed using the TAS-20 (Bagby et al., 1994). The 20-item Toronto Alexithymia Scale is a valid measure of alexithymia symptomology with good internal consistency (Bagby et al., 1994; Parker et al., 2003). Participants were asked to rate each item based on to what extent they agree or disagree with a statement. Response options included: "strongly disagree," "disagree," "neither agree nor disagree," "agree," and "strongly agree" (Bagby et al., 1994). Responses were scored based on a 5-point Likert scale. In the current study, internal reliability was acceptable (Cronbach's $\alpha = 0.82$). A score of 52–60 points indicates possible alexithymia, while a score greater than or equivalent to 61 points meets the clinical cutoff for alexithymia (Taylor et al., 2000).

2.3.2 | Autism symptomatology

Autism symptomatology was assessed using the AQ (Baron-Cohen et al., 2001) questionnaire. The AQ is a well-validated scale with strong psychometric properties that measures the presence of autistic traits (Baron-Cohen et al., 2001). Participants were asked to rate each item based on what extent they agree or disagree with the statement. Response options included: "definitely agree," "slightly agree," "slightly disagree," and "definitely disagree" (Baron-Cohen et al., 2001). Responses are scored using a binary system, where a score of 1 indicates autism characteristics, while a score of 0 does not. Internal reliability was acceptable (Cronbach's $\alpha = 0.83$). An accurate cutoff score is reported as ≥26 (Woodbury-Smith et al., 2005).

2.3.3 | ED symptomatology

ED symptomatology was assessed using the EDE-Q-13 (Lev-Ari et al., 2021). The EDE-Q-13 is a condensed form of the EDE-Q (Fairburn & Beglin, 1994). It measures eating disorder characteristics through five subgroups: eating restraint, shape and weight overvaluation, body dissatisfaction, bingeing, and purging. Participants were asked to rate the frequency of ED-related thoughts and behaviors during the past 28 days. Response options included the following categories: 0 days, 1–5 days, 6–12 days, 13–15 days, 16–22 days, 23–27 days, and every day. Convergent validity is shown between the EDE-Q-13 and original EDE-Q (Lev-Ari et al., 2021). Internal reliability was acceptable with Cronbach's $\alpha = 0.81$.

2.3.4 | Demographics

All participants self-reported their sex, whether they had an ED, autism, or alexithymia diagnosis, and whether or not they had

previously received treatment for ED. Participants previously treated for ED reported the type(s) of treatment they received. No other demographic information was collected.

2.4 | Data analysis strategy

Data analyses were conducted in SPSS version 28. For missing data at the item level, scale scores were imputed as the mean of answered items for those participants who answered >50% of items.

To compare the frequency of positive scores on the AQ scale (defined as a score ≥26) in a sample recruited from ED support platforms, frequency tables with 95% bootstrap confidence intervals were computed and compared to frequencies established in the general population (Brugha et al., 2012). Next, the bivariate association of positive AQ score with alexithymia symptoms was examined. This was done in two ways. First, categorical associations were examined using a cutoff of TAS-20 score ≥52 and a χ^2 analysis. Second, continuous TAS-20 scores were examined using an independent-sample *t*-test.

It was hypothesized that a positive AQ score would be associated with elevated alexithymia symptoms and greater disordered eating symptoms, as measured by the EDE-Q-13. It was additionally hypothesized that alexithymia symptoms would partially mediate the association of a positive AQ score with disordered eating. To test this hypothesis, three regression analyses were implemented. First, TAS-20 was regressed on the binary AQ score (*a* path). Next, EDE-Q-13 scores were regressed on AQ (*c* path). Finally, EDE-Q-13 scores were regressed on both the AQ score and the TAS-20 score (*b* path, *c′* path). The mediated effect was computed as $a \times b$, and significance was determined based on the joint significance test, which provides a balance between Type I and Type II errors (MacKinnon et al., 2002).

Anonymous data and code, including scoring syntax and annotated analyses, are available on the Open Science Framework (https://osf.io/zrpuq/?view_only=cd3717549b33495dbcf969b5c48efa15).

3 | RESULTS

A total of 228 individuals began the survey, 214 of whom finished the survey. Of these, 197 provided complete data for analysis. Of these 197, most ($n = 173$, 87.8%) participants reported previous ED treatment, 195 participants reported history of an ED diagnosis (99.0%), 31 reported an ASD diagnosis (15.7%), and 14 reported traits of alexithymia (7.1%). Descriptive statistics and correlations of focal variables can be found in Table 1.

Hypothesis 1. Frequency of positive AQ scores.

More than half of participants had a positive screen on the AQ scale ($n = 108$, 54.8%, 95% confidence interval [CI]: 47.7, 61.7).

Hypothesis 2. Bivariate association of AQ scores and TAS-20.

As hypothesized, participants who screened positive for autism symptoms on the AQ scale also reported more symptoms of alexithymia. As shown in Figure 1, when examining clinical cutoffs, 92.6% of individuals with a positive AQ score also had a positive TAS-20 score, as compared to 79.8% of those who did not have a positive AQ score ($\chi^2(1) = 7.00$, $p = 0.008$). The mean TAS-20 symptoms score for individuals with a positive AQ score was 67.89 (SD = 9.90); for those without a positive AQ score, the mean was 58.88 (SD = 10.68; $t(195) = 6.14$, $p < 0.001$).

Hypotheses 3 and 4. Alexithymia as a mediator of the association between a positive AQ score and disordered eating symptoms.

As shown in Figure 2, in the first model, individuals with a positive AQ score had significantly greater alexithymia symptoms (*a* path; $B = 9.02$, SE = 1.47, 95% CI: 6.12, 11.91, $p < 0.001$). In the second model, individuals with a positive AQ score also demonstrated significantly greater disordered eating symptoms (*c* path;

TABLE 1 Descriptive statistics and correlations of focal variables.

	Mean (SD)	AQ	AQ (imputed)	TAS-20	TAS-20 (imputed)	EDE-Q-13	EDE-Q-13 (imputed)
AQ	26.22 (7.71)	—	1.00**	0.47**	0.48**	0.18*	0.17*
AQ (imputed)	26.51 (7.60)	1.00**	—	0.46**	0.46**	0.17**	0.16**
TAS-20	63.76 (11.14)	0.47**	0.46**	—	1.00**	0.23**	0.22*
TAS-20 (imputed)	63.82 (11.18)	0.48**	0.46**	1.00**	—	0.21**	0.21**
EDE-Q-13	43.12 (13.94)	0.18*	0.17**	0.23**	0.21**	—	1.00**
EDE-Q-13 (imputed)	43.30 (13.86)	0.17**	0.16**	0.22*	0.21**	1.00**	—

Abbreviations: AQ, Autism-Spectrum Quotient; EDE-Q-13, 13-item Eating Disorder Examination Questionnaire; TAS, Toronto Alexithymia Scale.
*$p < 0.05$; **$p < 0.01$.

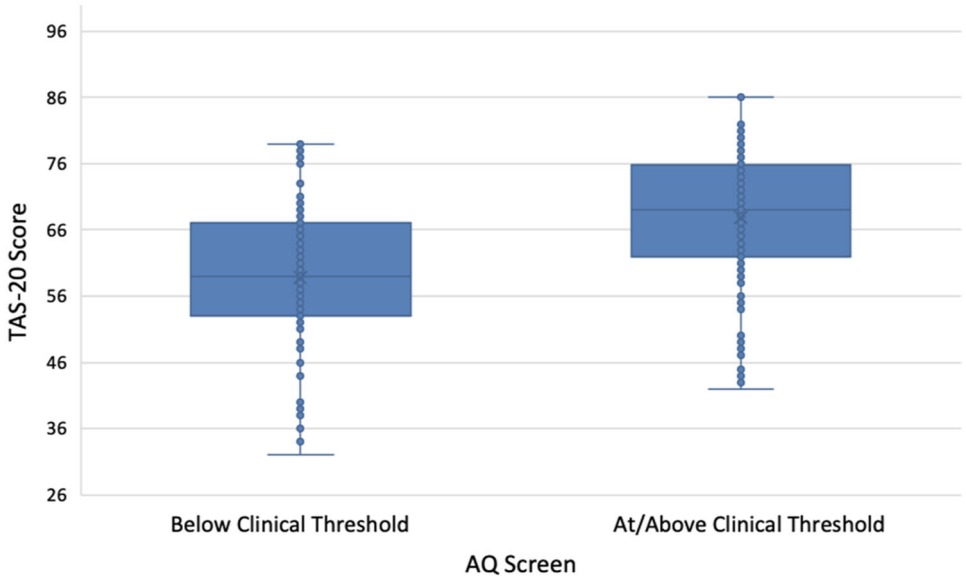

FIGURE 1 TAS-20 scores of individuals scoring below clinical Autism-Spectrum Quotient (AQ) threshold versus at/above clinical AQ threshold. TAS-20, Toronto Alexithymia Scale-20.

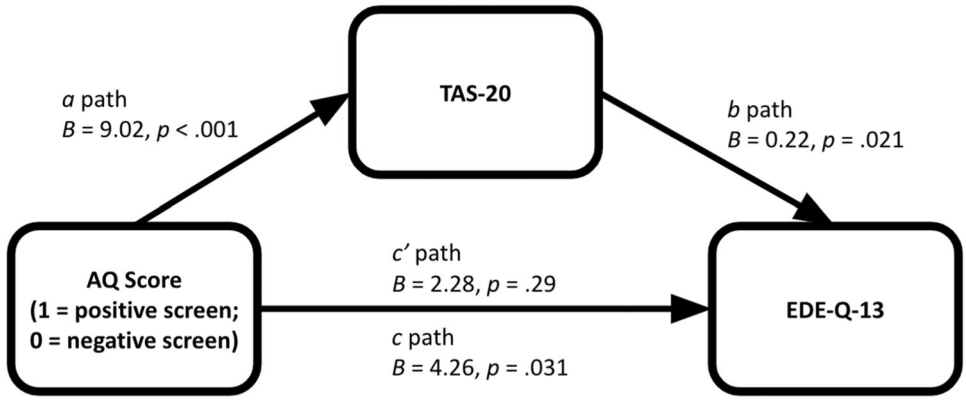

FIGURE 2 Alexithymia as a mediator of the association between a positive AQ score and disordered eating symptoms. AQ, Autism-Spectrum Quotient; EDE-Q-13, 13-item Eating Disorder Examination Questionnaire; TAS-20, Toronto Alexithymia Scale-20.

$B = 4.26$, SE = 1.97, 95% CI: 0.38, 8.14, $p = 0.031$). In the third and final model, the association of AQ score with disordered eating symptoms became nonsignificant (c' path; $B = 2.28$, SE = 2.12, 95% CI: −1.91, 6.46, $p = 0.29$); however, the association of alexithymia symptoms with disordered eating was positive and significant (b path; $B = 0.22$, SE = 2.32, 95% CI: 0.03, 0.41, $p = 0.021$). The mediated effect was computed as $a \times b = 9.015 \times 0.220 = 1.98$, and was statistically significant at $p < 0.05$ (as determined by the joint significance test (MacKinnon et al., 2002).

4 | DISCUSSION

There is a disproportionate prevalence rate of autism in ED populations (Brugha et al., 2012; Demartini et al., 2021; Doris et al., 2014; Gesi et al., 2017; Huke et al., 2013; Westwood, Mandy, et al., 2017; Westwood et al., 2018; Zucker et al., 2007) and poorer treatment outcomes and prognosis are associated in individuals with both autism and ED (Bentz, Jepsen, Pedersen, et al., 2017; Cashin et al., 2018; Chesney et al., 2014; Fichter & Quadflieg, 2016; Hirvikoski et al., 2016; Huke et al., 2014; Meczekalski et al., 2013; Morgan & Hayward, 1988; Nazar et al., 2018; Nielsen et al., 2015; Stewart et al., 2017; Tchanturia et al., 2016, 2019). High prevalence rates of alexithymia among both autistic populations and ED populations (Conway et al., 2018; Kinnaird et al., 2019; Poquérusse et al., 2018) suggest that alexithymia may have a mediating effect between autism and ED. The goals of the present study were thus to further define the overlap between autism and ED, to examine alexithymia symptomatology in individuals with ED and autistic traits versus neurotypical individuals with ED, and to further explore the mediating effect of alexithymia on the relationship between autism and ED.

4.1 | Overlap between autism and ED

Study results reinforce previous findings of a significant over-representation of autistic traits in ED populations. Over half (54.8%) of the participants in this sample, comprised of female individuals recruited from online ED support groups, screened at or above the clinical threshold for autism. Not only does this observed rate of autistic traits substantially surpass autism prevalence rates within the general population, which is estimated to be around 0.3% in women (Brugha et al., 2012), it also exceeds previous estimates of the prevalence rate of autistic individuals with ED (Tchanturia et al., 2013; Wentz et al., 2005; Westwood et al., 2018; Westwood, Mandy, et al., 2017). This is likely due to our utilization of a self-report measure rather than an interview formatted questionnaire (Wentz et al., 2005) like the semistructured interview style of the ADOS-2 (Westwood et al., 2018; Westwood, Mandy, et al., 2017). Using a clinical interview to determine autism diagnosis may have resulted in a lower overlap in this population.

4.2 | Alexithymia prevalence in autistic versus neurotypical individuals in ED populations

In line with prior literature documenting an overlap between autism and alexithymia (Bourke et al., 1992; Kinnaird et al., 2019; Poquérusse et al., 2018; Schmidt et al., 1993; Westwood, Kerr-Gaffney, et al., 2017), findings from the current study suggest a higher rate of alexithymia among individuals with ED meeting the clinical cutoff on the AQ than individuals with ED who did not meet this cutoff. In fact, nearly all (96.2%) of those individuals who screened within the clinical range on the AQ also had high TAS-20 scores, indicating traits of alexithymia. As with the estimate for the overlap of autism and ED, the estimated rate for overlap of autism and alexithymia is higher than those previously reported (e.g., Kinnaird et al. (2019) suggested an overlap between alexithymia and autism of 49.93%). This discrepancy is likely due to the fact that the present study recruited women from online ED support forums, whereas Kinnaird et al. (2019) focused on autism alone, such that the present sample reflected comorbid autism and ED. This discrepancy further emphasizes the unique psychological risk profile associated with co-occurring autism and ED.

4.3 | Alexithymia as a mediating factor between autism and ED

Based on previous research documenting overlap between alexithymia with both autism and ED separately (Bargiela et al., 2016; Bourke et al., 1992; Conway et al., 2018; Kinnaird et al., 2019; Poquérusse et al., 2018; Westwood, Kerr-Gaffney, et al., 2017), as well as previous research investigating alexithymia as a potential mediating factor in a general population (Bargiela et al., 2016), it was expected that alexithymia would mediate the association of autism with ED symptomatology in a sample of female individuals with ED. As hypothesized, alexithymia was a significant mediator of this association. These results corroborate those of Vuillier et al. (2020) and align with research by Hobson et al. (2020) who found that individuals with AN have an increased likelihood of reaching the diagnostic threshold for autism if they experienced comorbid alexithymia (Conway et al., 2018).

4.4 | Clinical implications

Based on these findings, it is apparent that there is an over-representation of individuals with autism characteristics within the ED population. Given that poorer treatment outcomes and increased comorbid mental illnesses (such as eating pathology) are associated with delayed autism diagnosis in women (Bargiela et al., 2016; Leedham et al., 2020), autism symptomatology may present an obstacle to ED recovery.

The overlap identified between autism, ED, and delayed diagnosis in women indicates that autism should be universally screened for upon ED diagnosis or treatment. The mediating effect of alexithymia between autism and greater ED symptomatology suggests that these individuals may additionally benefit from treatment targeted toward improving the social and emotional deficits autistic individuals may face due to alexithymia (Aaron et al., 2015; Bird & Cook, 2013; Cook et al., 2013; Courty et al., 2015; Milosavljevic et al., 2016). New therapeutic approaches and interventions that incorporate emotional awareness merit further research, with the potential to mitigate ED symptomatology and improve ED recovery rates within the autistic population.

4.5 | Limitations and conclusions

Conclusions drawn from the data are limited due to some methodological decisions. Participants were recruited from online ED support platforms, which may not capture the full range of individuals experiencing ED. Only female individuals were recruited for this study and provided limited demographic information, so the generalizability of the data can not be established. Like previous studies on this topic as highlighted in a systematic review by Miniati et al. (2022), the cross-sectional nature of this study limits any conclusions about causal pathways. It is also important to note that, although the TAS-20 is a common self-report measure of alexithymia, it requires a level of emotional awareness that individuals with alexithymia, or on the autism spectrum may lack (Huang et al., 2017; Lane et al., 1998). Another limitation is that self-report measures were used rather than clinical interviews, which may have impacted the accuracy of the results. Additionally, there is a chance that unmeasured variables may have skewed the proportional outcome of our mediating variable (Richiardi et al., 2013). Finally, there is evidence to believe that the overlap between traits of autism is transdiagnostic across a multitude of eating disorders including AN, BN, BED, and ARFID, but this study

was limited in its ability to investigate the differences between eating pathologies individually (Demartini et al., 2021; Fisher et al., 2014; Gesi et al., 2017; Nicely et al., 2014; Tchanturia et al., 2013; Wentz et al., 2005; Westwood et al., 2018). Future research should both investigate the relationships between autism and different eating disorders individually and also control for other variables such as comorbid depression and anxiety to distinguish the scale of alexithymia's mediatory impact on autism versus ED using longitudinal designs (Espina Eizaguirre et al., 2004; Sexton et al., 1998; Speranza et al., 2005; Torres et al., 2015).

Despite the listed limitations, this study also had multiple strengths including a sample entirely of female individuals with eating disorders and a large sample size. Additionally, this study gives evidence of alexithymia as a mediating factor between ED and autism ultimately building on existing literature exploring alexithymia's mediatory role (Moseley et al., 2023; Vuillier et al., 2020).

This study is an important preliminary step in examining the relationship between autism, ED, and alexithymia; additional research is required to further delineate these pathways. In particular, longitudinal observational studies and interventional research would be invaluable for determining causal pathways between these variables. A better understanding of these relationships may lead to better treatment techniques and, ultimately, outcomes for individuals with elevated autistic traits who are also struggling with life-threatening eating pathologies.

ACKNOWLEDGMENTS

Funding for this study was provided by the BOLD Women's Leadership Network.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Anonymous data and code, including scoring syntax and annotated analyses, are available on the Open Science Framework (https://osf.io/zrpuq/?view_only=cd3717549b33495dbcf969b5c48efa15).

ORCID

Alyssa Daniels http://orcid.org/0000-0002-0175-5347

PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1002/mhs2.42.

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How to cite this article: Daniels, A., Cornelius, T., & Gorin, A. A. (2024). Understanding the associations between social and emotional expression, communication, and relationships in individuals with eating pathology. *Mental Health Science*, *2*, 4–13. https://doi.org/10.1002/mhs2.42