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ORIGINAL ARTICLE

Do orthorexia and intolerance of uncertainty mediate the relationship between autism spectrum traits and disordered eating symptoms?

Sarah Giles[1] · Madeline Toohey[1] · Elizabeth K. Hughes[1,2,3] · Matthew Fuller-Tyszkiewicz[4,5] · Isabel Krug[1]

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Abstract

Purpose Autism spectrum disorder traits have been implicated in the psychopathology of eating disorders and may also be relevant for the development of orthorexia symptoms. Further, intolerance of uncertainty (IUS) may indirectly contribute to the development of disordered eating, as the displacement of anxiety onto food may help achieve a sense of control and maximise certainty. We examined a new cognitive model of eating pathology which assessed the role of IU and orthorexia symptoms as potential mediators of the relationship between autistic traits and disordered eating in a community sample.

Methods Three-hundred-and-ninety-six female participants ($M = 20.07$, SD = 4.52 years old) completed an online self-report questionnaire which assessed the variables of interest.

Results Despite finding significant bivariate correlations, our model results showed that autistic traits did not directly predict disordered eating or orthorexia symptoms. Significant indirect relationships were found between autistic traits and eating disorder symptoms through both IU and orthorexia symptoms.

Conclusion The findings provide partial support for our proposed model suggesting that autistic traits may increase the vulnerability for disordered eating, not directly, but through their associations with mechanisms such as IU and the development of problematic eating behaviours typical of orthorexia. Future research should focus on whether targeting IU may assist in preventing the development of disordered eating.

Level of evidence Level V, cross-sectional descriptive study.

Keywords Orthorexia nervosa · Autism · Intolerance of uncertainty · Disordered eating

Introduction

Research investigating the possible link between autism spectrum disorders (henceforth, autism) and eating disorders (ED) has grown substantially in the last ten years. This research has primarily centred on the presence of cognitive processing styles evident in both EDs and autism, such as set-shifting difficulties [1], weak central coherence, defined as a bias towards details at the expense of global meaning [2], and deficits in theory of mind [3]. Further, evidence suggests that autistic traits are overrepresented within EDs with the prevalence of autism in ED populations estimated at approximately 26.50% [4]. With respect to specific ED diagnostic groups, recent evidence [5] suggests that autistic traits are present at higher levels amongst individuals with predominantly restrictive eating patterns [e.g., Anorexia Nervosa (AN) Restrictive subtype] in comparison to individuals who exhibit binge eating behaviours (e.g. Bulimia Nervosa, Binge Eating Disorder and AN-Binge Purge

In accordance with community preferences, this article uses identity-first language to describe individuals with a diagnosis of autism, and traits related to autism (i.e., autistic person; autistic traits).

✉ Sarah Giles
giless1@student.unimelb.edu.au

1 Melbourne School of Psychological Sciences, The University of Melbourne, 12th Floor Redmond Barry Building, Parkville Campus, Melbourne, VIC 3010, Australia

2 Department of Paediatrics, The University of Melbourne, Melbourne, Australia

3 Murdoch Children's Research Institute, Melbourne, Australia

4 Centre for Social and Early Emotional Development, School of Psychology, Deakin University, Melbourne, VIC, Australia

5 School of Psychology, Deakin University, Geelong, VIC, Australia

Subtype). Collectively, the higher prevalence rates and overlapping cognitive and socio-emotional functioning of autism and ED demonstrate the similarities between these groups.

Of interest, interpretation of the similarities between autism and ED differs between researchers. While some conceptualise autistic traits as premorbid difficulties predisposing individuals to the development of ED [6], others have argued that behaviours that resemble "autistic traits" may be entirely attributable to the effects of starvation and irregular eating, rather than neurodevelopmental differences (e.g. [7].). One potential approach to help overcome the state versus trait issue would be through the investigation of autistic traits within community samples. Such an approach provides the possibility to assess both ED symptoms and autistic traits across a continuum of severity.

Within non-clinical populations, a systematic review [8] of the association between "autistic-like" behaviours and disordered eating found significant associations between specific theory of mind deficits, impaired set-shifting, and autistic traits as measured by the Autism-Spectrum Quotient [9] and disordered eating symptoms. Mansour and colleagues [10] study, included in this systematic review [8], investigated the role of emotion regulation difficulties as a potential mediator of the relationship between autistic traits and disordered eating symptoms in a community sample of university students. Results showed a significant indirect effect between autistic traits, body dissatisfaction and disordered eating via emotion dysregulation.

A similar pattern of findings has emerged within autistic populations. García-Villamisar and Rojahn [11] assessed adults with a diagnosis of autism. They found comorbid levels of psychopathology (anxiety, irritability and depression) and stress mediated the relationship between autistic traits and repetitive and restrictive behaviours. Therefore, autistic traits may be indirectly related to eating pathology through depression, anxiety, or difficulties with emotion regulation, and that disordered eating symptoms emerge in an attempt to regulate affective experiences.

Given that a substantial proportion of individuals who exhibit autistic traits do not develop disordered eating or a full threshold ED, further exploration of the mechanisms that may mediate the development of disordered eating in the context of autistic traits is required. Intolerance of uncertainty (IU) is defined as a dispositional characteristic towards a set of negative beliefs about uncertainty and its potential outcomes, and has been implicated as an aetiological process in the maintenance of EDs and other mental health problems such as anxiety and depression [12, 13]. IU is thought to contribute to the manifestation of symptoms such as obsessions, worry, and rituals, which are employed to maximise certainty and gain the perception of control over unknown future outcomes [14]. Within the context of EDs, Brown and Robinson's [13] meta-analysis and systematic review of IU showed women with EDs had significantly higher scores on the Intolerance of Uncertainty Scale (IUS; [15]) compared to healthy controls. Furthermore, depressive and anxiety symptoms are elevated in ED populations [16], and are also likely exacerbated by difficulties tolerating uncertainty [14]. These findings suggest that IU is elevated in ED populations, predisposing these individuals towards negative beliefs about uncertainty (e.g. uncertainty is dangerous), which may account for the ritualised and obsessional dietary practices, typical of EDs, which are employed to maximise certainty [13].

Similarly, emerging evidence suggests autistic individuals also report higher levels of IU when compared to their neurotypical peers [17–19]. Within autism, IU is thought contribute to the manifestation of anxiety and depressive disorders and influence higher-order restricted and repetitive behaviours, such as insistence on sameness and rigid adherence to routines in an attempt to maximise certainty [20]. Collectively, these findings demonstrate associations between IU in both ED and autism and suggest IU may promote the development of rigid routines to maximise certainty. Therefore, in the context of autistic traits, individuals with high levels of IU may engage in ritualised weight and shape control rituals to foster a sense of control.

Dell'Osso and Abelli [5] in their scoping review of Orthorexia Nervosa (ON) hypothesise that individuals with symptoms of ON could share traits of autism, and highlight the overlapping obsessive–compulsive symptom dimensions between ON, AN, and autism. ON, while not formally recognised as an ED diagnosis, is characterised by an extreme fixation on healthy eating in the pursuit of optimal health, and the rigid avoidance of foods perceived as "impure" [21]. ON has been associated with increased dietary restraint, preoccupations with weight and shape, and body dissatisfaction, and has been proposed to serve as a risk factor for future ED diagnoses [5]. Dell'Osso and Abelli [5] argue the intense preoccupations adhering to dietary rules and ritualised patterns of food preparation that characterise ON share behavioural similarities with autism. However, empirical research investigating the links between ON and autistic traits is lacking.

While the literature concerning ON is ambiguous as to whether it constitutes a separate diagnosis, or merely a variant of already existing disorders [22], the symptom overlap between AN and ON, such as the displacement of anxiety onto food to achieve a sense of control, suggests that those factors implicated in the development of AN may help enhance our understanding of ON. Autistic traits have been implicated in the psychopathology of AN [6], and due to the similarities between ON and AN, those factors implicated in AN, may also be relevant for the development of ON symptoms, and subsequent disordered eating.

To summarise, autistic traits have been implicated in the psychopathology of EDs, particularly AN, and due to the similarities between ON and AN, autistic traits may also be relevant for the development of ON symptoms. Further, despite Dell'Osso and Abelli's [5] assertion that autism and ON may be associated, no study to date has investigated the relationship between these two conditions. Moreover, in the context of autistic traits, IU may promote the utilisations of rituals, such as the highly idiosyncratic weight and shape control rituals typical of disordered eating and ON behaviours, in an aim to achieve a sense of control. Therefore, dietary restraint evident within ON may be motivated by an attempt to maximise certainty in one's environment by successfully adhering to highly detailed dietary rules.

The current study aimed to examine a new model, whereby autistic traits were related to disordered eating symptoms through IU and ON (see Fig. 1). We hypothesised that our model would provide a good fit to the data, and that IU and ON would emerge as significant mediators in the relationship between autistic traits and eating pathology. Further, given the significant associations between anxiety and depressive disorders and IU [23] and concerns that autistic traits may be intensified as a consequence of the underweight state [7], we controlled for the effects of age, anxiety, depression, and BMI to ensure the robustness of our model.

Methods

Participants

Participants were 401 females of whom 364 were Psychology students from the University of Melbourne who received course credit for their involvement. The remaining 37 participants were recruited from the community and went into the running to win a voucher for their participation. There were no exclusion criteria for the study. The age of participants ranged from 18 to 57 years ($M = 20.07$, $SD = 4.52$). The majority of participants spoke English as their main language (62.6%), were currently single (76.3%), had a high school degree as their current highest level of education (75.35%), and identified as heterosexual (83.1%). Participants completed an online questionnaire hosted on Qualtrics, at their convenience. Five participants completed less 50% of questions and were excluded, leaving a final sample of 396.

Materials and procedure

Socio-demographic information

Participants answered an online survey which assessed socio-demographic information (e.g. weight, height, and age).

Autism-spectrum quotient (AQ; [9])

The AQ is a 50-item questionnaire which assesses autistic traits. Items are rated using a four-point Likert scale ranging from "disagree" to "agree", with higher scores indicating higher levels of ASD traits. The AQ total score was used, which indicated acceptable internal consistency, omega = 0.72.

Intolerance of uncertainty scale [(IUS; 15)]

The IUS contains 27 items which assess beliefs and reactions around uncertainty, with higher scores indicating greater intolerance of uncertainty. Responses are rated on a five-point Likert scale ranging from 1 = "not at all characteristic of me" to 5 = "entirely characteristic of me". The IUS total score was used, which demonstrated excellent internal consistency, omega = 0.96.

Orthorexia-revised [(ORTO-R; 24, 25)]

The ORTO-R is comprised of 6 items scored on a four-point Likert style scale ranging from "always" to "never" where higher scores indicate higher levels of ON symptoms. The ORTO-R was developed to address the

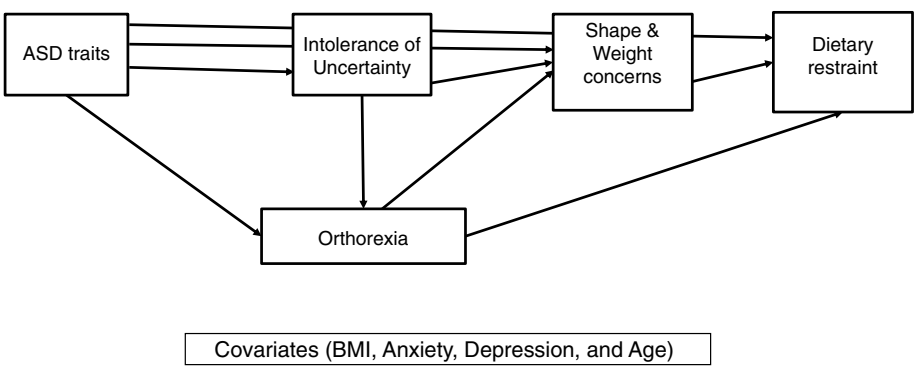

Fig. 1 Conceptual model for the relationship between autistic traits and disordered eating: Intolerance of uncertainty and orthorexia as mediating variables

limitations of the original ORTO-15 [26]. In the current study, the ORTO-R omega was 0.72, while the omega for the ORTO-15 was 0.38, demonstrating that the revised scale significantly improved the internal consistency.

Eating disorder examination questionnaire (EDE-Q; [27])

The EDE-Q is a 36-item, self-report questionnaire examining the severity of disordered eating behaviours and attitudes within the last 28-days. The EDE-Q contains four subscales (restraint, eating concern, weight concern and shape concern). Responses are rated on a seven-point Likert-type frequency scale. In the current study, the shape concern and weight concern subscales were combined to create a latent construct for weight and shape concerns, which has consistently been demonstrated in EDE-Q factor analyses studies [28], including those involving female community samples [29]. The combined weight and shape concerns factor displayed excellent internal consistency, omega = 0.96, and the restraint subscale also demonstrated good internal consistency omega = 0.87.

Hospital anxiety and depression scale (HADS; [30])

The HADS is a 14-item questionnaire comprised of two subscales which assess depression and anxiety symptoms within the last week. Items on the HADS are rated on 4-point phrases. Good internal consistency was demonstrated for the depression subscale (omega = 0.87) and the anxiety subscale (omega = 0.85).

Data analyses

Bivariate correlations were conducted to assess the relationship between autistic traits, ON symptoms, IU, and ED symptoms using IBM SPSS 26.0. Inspection of remaining missing data showed less than 0.5% missing per variable and were distributed in a pattern consistent with missing completely at random, $\chi^2(46) = 49.08$, $p = 0.35$. Expectation Maximisation was used to impute the missing values. Path analysis using Mplus version 8 [31] were conducted to evaluate the proposed theoretical model (see Fig. 1), controlling for age, BMI, anxiety, and depression. All paths were tested for significance at $p < 0.05$ (two-tailed). Mediated effects were tested using bias-corrected bootstrapping with 5000 samples. A mediation effect was deemed significant if the 95% confidence intervals for the indirect effect parameter estimate did not include 0.

To assess model fit, the comparative fit index (CFI), Tucker–Lewis index (TLI), root mean square error of approximation (RMSEA), and standardised root mean square (SRMR) were chosen. Indicators for acceptable model fit were CFI, and TFI values ≥ 0.90, RMSEA values ≤ 0.06 with a 90% CI where the lower limit contains or is close to 0, and the upper limit is less than 0.08, and SRMR values < 0.08 [32].

Results

Associations between autism, ON , IU and DE

Means, standard deviations and correlations between all variables of interest are presented in Table 1. Autistic traits were associated with higher levels of IU, ON symptoms, weight and shape concerns, and dietary restraint. IU was positively correlated with weight/shape concerns and dietary restraint.

Table 1 Means, standard deviations, and bivariate correlations between the variables of interest

	Mean [3]	1	2	3	4	5	6	7	8
1. AQ Global	20.07 (6.38)	–							
2. IUS Scale	70.99 (22.10)	0.58***	–						
3. ORTO-R	14.06 (2.71)	0.15*	0.31***	–					
4. EDE-Q Weight and Shape Concern	5.17 (3.09)	0.16**	0.40***	0.59***	–				
5. EDE-Q Dietary Restraint	1.55 (1.41)	0.12*	0.26***	0.64***	0.63***	–			
6. HADS Anxiety	8.87 (4.34)	0.32***	0.59***	0.35***	0.41***	0.27***	–		
7. HADS Depression	5.04 (3.63)	0.39***	0.52***	0.26***	0.37***	0.29***	0.62***	–	
8. BMI	21.97 (4.74)	− 0.14**	− 0.06	− 0.07	0.24***	0.10	0.02	− 0.01	–
9. Age	20.07 (4.53)	− 0.10*	− 0.10	− 0.02	− 0.01	− 0.01	0.04	0.12*	0.20***

AQ = Autism Quotient; ORTO-R = Orthorexia Revised; HADS = Hospital Anxiety and Depression Scale; BMI = body mass index. *$p < 0.05$, **$p < 0.01$. ***$p < 0.001$

ON was positively correlated with IU, dietary restraint, and weight and shape concerns. As anticipated, anxiety and depression were significantly correlated with all disordered eating variables, autistic traits, and IU. Interestingly, BMI was only significantly associated with autistic traits.

Path analyses

Overall model fit

The results from our initial model revealed no significant direct effects for ASD to weight and shape concerns or dietary restraint. In the interest of maximising model parsimony, these non-significant paths were iteratively removed. The resulting model is shown in Fig. 2. This parsimonious model provided a good fit to the data, $\chi^2(9) = 12.70$, $p = 0.118$, CFI = 0.996, TLI = 0.988, RMSEA = 0.032 (CI: 0.000, 0.070), SRMR = 0.021. In total, the model accounted for 53% of the variance in IU, 15% of the variance in ON, 46% of the variance in weight/shape concerns, and 51% of the variance in dietary restraint (all $p < 0.001$).

Pathways from autistic traits to DE

As previously stated, our initial model revealed no significant direct effects for ASD on weight and shape concerns or dietary restraint. However, as shown in Fig. 2, autistic traits significantly predicted IU ($\beta = 0.41$, $p < 0.001$), but did not significantly predict ON symptoms ($\beta = 0.10$, $p = 0.09$).

Regarding our serial mediation model, bootstrapped analyses revealed several significant indirect effects from autistic traits to restraint, (1) Autism traits IU weight/shape restraint ($\beta = 0.025$, 95% CIs 0.093, 0.046),

(2) Autism traits → IU→ ON → weight/shape → restraint ($\beta = 0.017$, 95% CIs 0.007, 0.032), and (3) Autism traits → IU → ON → restraint ($\beta = 0.039$, 95% CIs 0.016, 0.068). However, several paths, both of which did not include IU, were not significant (4) Autism traits → ON → restraint ($\beta = -0.040$, 95% CIs − 0.093, 0.006) and (5) Autism traits → ON → weight/shape → restraint ($\beta = -0.018$, 95% CIs − 0.040, 0.003).

There were also several significant indirect effects from autism traits to weight and shape concerns, (1) Autism traits → IU → weight/shape ($\beta = 0.064$, 95% CIs 0.022, 0.110), and (2) Autism traits → IU → ON → weight/shape ($\beta = 0.044$, 95% CIs 0.018, 0.077). The indirect effect from (3) Autism traits → ON → weight/shape ($\beta = -0.045$, 95% CIs − 0.099, 0.007) was not significant.

Discussion

This study explored the potential relationships between autistic traits and disordered eating, and whether IU and ON mediated this relationship. In our path analyses, we did not find any significant direct effects between autistic traits to ON, weight and shape concerns, or dietary restraint. However, as per our predictions, we found several significant direct and indirect effects in our model. Specifically, autistic traits were directly related to IU, IU was directly related to ON symptoms, and IU and ON were both directly related to greater weight and shape concerns and dietary restraint. Second, autistic traits were indirectly related to greater weight and shape concerns and dietary restraint, through higher levels of IU and ON symptoms. These findings were significant while controlling for the potentially confounding effects of anxiety and depression symptoms, BMI, and age.

Investigation of our first aim demonstrated that autistic traits were significantly associated with disordered eating symptoms. Our findings support the accumulating clinical evidence, which suggests that autistic traits are associated with ED symptoms across the continuum of their expression [8]. Our findings also provide partial support for Dell'Osso et al.'s [5] argument that ON and autistic traits may be associated as evidenced by the significant positive bivariate association between autistic traits and ON. This represents a unique finding as no studies to date have examined whether

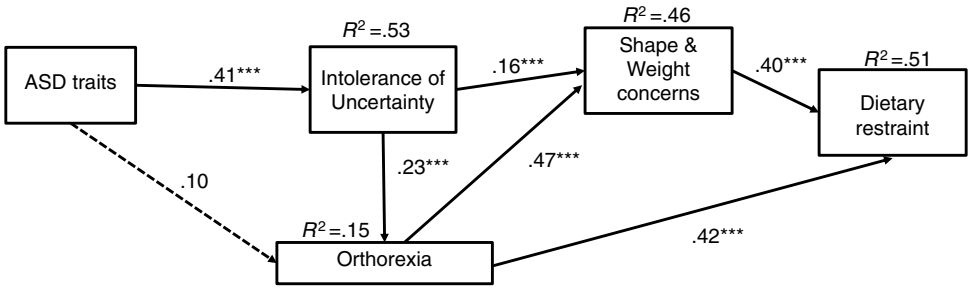

Fig. 2 Findings for the relationships between autistic traits and disordered eating: Intolerance of uncertainty and orthorexia as mediating variables. * < 0.5; ** < 0.01; *** < 0.001. Note: Covariates (age, BMI and depression and anxiety) were included in analyses but omitted from the figure due to complexity

ON symptoms are associated with autistic traits. Interestingly, despite finding significant bivariate correlations, autistic traits were not directly related to ED symptoms in our path analyses. This suggests that the associations between autistic traits and ED symptoms may change once other variables are taken into consideration (e.g. controlling for the effects of anxiety and depression). Moreover, the significant indirect effects may suggest that autism does not directly increase the likelihood of presenting with disordered eating, but autistic traits may indirectly contribute to the risk of developing disordered eating. However, it must be acknowledged that our findings are correlational, and therefore, the temporal relationships between autistic traits and disordered eating cannot be established.

Our results also showed significant correlations between ON and both dietary restraint and weight and shape concerns. This finding contributes to a growing literature which demonstrates associations between body image concerns and ON [33]. Therefore, the preoccupations with "healthy" eating evident in ON may be motivated by underlying weight and shape concerns rather than the pursuit of health. However, examining the associations between ON and other ED symptoms is undermined by a lack of well-validated questionnaires which assess ON. We chose to use the ORTO-R [25, 26] as it was developed to address the modest psychometric properties of the original ORTO-15. Specifically, the ORTO-15 has been criticised for having poor content validity, low internal consistency, and highly variable prevalence estimates of ON (ranging from 41.9 to 88.7%), which has led some to question the validity of this measure [34]. While the ORTO-R was revised with these criticisms in mind, the recentness of this measure means firm conclusions regarding its validity cannot be drawn. Therefore, our findings should be contextualised within the limitations of existing measures of ON.

Concerning our second aim, we found that both IU and ON were significant mediators in our serial mediation paths. Results showed significant indirect effects between autistic traits through IU and ON symptoms to both dietary restraint and weight and shape concerns. The findings also provide support for our line of reasoning that IU may motivate preoccupations with weight and shape control which drives dietary restraint, as well as IU directly contributing to the development of dietary restraint. Such findings echo the description of 'motivated eating restraint' in the cognitive-interpersonal maintenance model for AN [35] wherein weight and shape concerns are seen as one possible motivation for dietary restraint. However, dietary restraint may arise for reasons unrelated to weight and shape concerns, such as to achieve a sense of emotional numbing or control. Therefore, clinicians must be diligent in formulating the function that disordered eating and ON behaviours may serve.

Our results have implications for the treatment of disordered eating. Fear of losing control and fear of change has been shown to represent significant treatment barriers for individuals experiencing disordered eating [36] and may represent a central aeitological process in disordered eating. Our findings suggest IU may in part motivate the formation of rigid eating patterns, as dietary rules provide a sense of achievement and control. Enhancing an individuals' tolerance for uncertainty through cognitive behavioural protocols, which expose individuals to novelty and ambiguity, may be a potentially effective method to address IU, and consequently disordered eating. These protocols have effectively targeted IU across a range of disorders (e.g. anxiety [37] and autism [38]) suggesting IU is a malleable process. Therefore, directly targeting the role of IU in the development of disordered eating may be an important avenue for future research. The finding that ON symptoms acted as a mediator to ED symptoms highlights the importance of providing psychoeducation about ON behaviours (e.g. preoccupations with "healthful" eating), which may serve as a precursor to future disordered eating [39, 40].

However, the results must be interpreted within the context of certain limitations. Chiefly, our study is cross-sectional, and as such, precludes any conclusions about causality. Despite using widely used self-report measures to assess our constructs of interest, self-report may not be the most appropriate method to assess constructs such as autism spectrum disorder traits. Finally, the majority of participants were university students and were all female. Future research should examine this study's model within more diverse samples using longitudinal designs, and incorporate diagnostic measures such as the Autism Diagnostic Observational Schedule [41] and the Eating Disorder Examination [42].

Withstanding these limitations, the current study provides new insights into the relationship between autistic traits, ED symptoms, IU, and ON. Our findings provided partial support for Dell'Osso et al.'s [5] argument that ON and autistic traits may be associated, suggesting further research is required to verify whether ON is associated with autistic traits. Finally, our results show that autistic traits were indirectly related to ED symptoms, with both IU and ON emerging as significant mediators.

What is already known on this subject?

Research suggests autistic traits are frequently associated with ED symptoms. Numerous studies have also reported that IU is elevated within ED and autistic samples. However, no studies have examined whether ON is associated

with autistic traits, and whether IU or ON may mediate the development of disordered eating symptoms.

What this study adds?

This is the first study to examine the relationships between autistic traits and disordered eating, and whether IU and ON mediated this relationship. Significant correlations were found between autistic traits and disordered eating, and ON. However, we did not find a significant direct relationship between autistic traits and disordered eating, and ON. Of interest, autistic traits were indirectly related to ED symptoms, such that high levels of autistic traits predicted higher levels of IU, which in turn predicted higher levels of ON symptoms, weight and shape concerns and dietary restraint. These findings suggest that autistic traits may increase the vulnerability for disordered eating, not directly, but through their associations with cognitive mechanisms such as IU and the development of problematic eating behaviours typical of ON.

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Data availability Data is available upon request from the corresponding author (Email: giless1@student.unimelb.edu.au).

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval Ethical approval for the current study was obtained from the University of Melbourne Ethics Committee (HREC 1851160.3).

Informed consent Informed consent was obtained from all participants included in the study.

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