Autism and anorexia nervosa: longitudinal prediction of eating disorder outcomes

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Research Article

Keywords: anorexia nervosa, autism, autistic traits, eating disorder symptoms, depression, anxiety, BMI

Posted Date: July 5th, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1791109/v1

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Abstract

Background: Recently, elevated levels of autistic traits and autism diagnoses have been reported among people with anorexia nervosa (AN). In clinical settings high levels of autistic traits have been linked to more complex, highly comorbid illness presentation and poorer outcome. This study aimed to examine whether autistic traits predict AN symptom profile in long term.

Methods: Altogether 118 women with lived experience of AN completed two autism assessments at time 1, the Autism Diagnostic Observation Schedule (ADOS) and the short version of the Autism Quotient (AQ10). Measures assessing AN symptom profile, including eating disorders symptoms, anxiety, depression, OCD symptoms, and BMI, were also recorded. The symptom profile measures were administered again 6 months and 2 years later. We conducted two analyses to examine the extent to which the ADOS and AQ10 scores predicted broad AN symptom profile at each three time points.

Results: Overall, high levels of autistic traits were consistently associated with worse psychological symptoms, but not BMI, across all time points. Both the analysis using baseline ADOS scores and self-reported AQ10 scores showed similar pattern.

Limitations: The sample consisted of young women only and it is therefore, difficult to ascertain whether these findings extend to other genders. Additionally, 70% of the participants who completed the self-report questionnaires at Time 1 also agreed to undergo ADOS assessment. This may have limited the power of the analysis involving ADOS scores compared to the analysis using AQ10 scores.

Conclusions: The present findings solidify previously reported associations between autistic traits and worse psychological outcome among people with AN. The findings also suggest that self-report measures may be sufficient for assessing autistic traits among people with AN. Importantly, the study highlights the need for development and further investigation of neurodiversity accommodations in the treatment of AN.

Background

Anorexia nervosa (AN) is a complex psychiatric disorder, characterised by intense fear of gaining weight and subsequent malnutrition (1, 2). It has been suggested that approximately 45–97% of people with AN have at least one comorbid psychiatric disorder (3). The most prevalent comorbid disorders are depression, anxiety disorders and obsessive-compulsive disorder (OCD) (4–6). Although adolescents with AN with shorter duration of illness have been found to have lower rate of comorbid psychiatric disorders than adults, it has been estimated that up to 60% of adolescents with AN also had mood disorders, while up to 16% also had comorbid anxiety disorder (7, 8). Importantly, highly comorbid presentation of AN has been linked to greater severity and longer duration of illness (6), which may go some way to explain the age-related differences in the prevalence of comorbid diagnoses. Together these findings suggest that AN frequently has complex psychopathology and symptom profile.
Previous research has documented that in addition to high rates of comorbid psychiatric disorders, somewhere between 4 and 53% of people with AN also meet criteria for autism diagnosis either based on self-report or clinician assessment (9–11). Autism is a neurodevelopmental condition present from early childhood, but it is often missed among girls and women resulting in late diagnosis during adolescence or early adulthood (12–14). Autism is typically associated with differences in cognitive style, including greater detail focus and difficulties making top-down predictions about the world resulting in intolerance of uncertainty, cognitive rigidity and greater desire for sameness (15). In addition to cognitive differences, autism is often associated with alterations in social communication and interaction (16, 17). Autistic people typically communicate and experience the world in a different way compared to neurotypical people (17, 18). This results in a gap in communication expectations, also known as the double empathy problem, such that both autistic and neurotypical people struggle to relate to and empathise with one another (16–18). Similar differences in cognitive style and social functioning have been documented in AN, particularly among those with long duration of illness (19).

Autistic social and cognitive styles clashing with the neurotypical world has been proposed to underlie high levels of mental health problems in autism, which are further compounded by other factors including stigma, trauma, and prejudice (15, 18, 20). In the same vein, in the field of eating disorders (ED), cross-sectional studies have documented that those who report more autistic traits present more severe ED psychopathology and complex illness profiles, with more comorbid anxiety, depression, and OCD symptoms (21, 22). Furthermore, previous longitudinal, naturalistic studies of AN patients admitted to inpatient ED services found that those who self-reported higher levels of autistic traits also reported more ED symptoms, depression and anxiety, and poorer social functioning at both admission and discharge (23–25). Although the average duration inpatient treatment was 16 weeks, these findings suggest that those at the intersection of autism and AN may at higher risk poor illness and treatment outcome. These findings have led to interest in examining the extent to which autistic traits predict complex illness presentation in AN in long term.

The present study aims to build on the previous literature by examining whether autistic traits measured at baseline can predict ED symptom profile among those with AN at later time points. We additionally aimed to explore whether a short, self-report screening measure could be used to predict ED symptom profile over time in a similar fashion as a gold standard diagnostic tool. Based on the previous work outlined above, we hypothesised that autistic traits would consistently predict worse illness presentation over time and that this relationship would not vary as function of time.

**Methods**

**3.1 Participants**

One hundred and eighteen participants completed the assessments across three time points. All participants were female aged 12–27 with lived experience of AN ranging from acute illness to fully recovered. The average duration of illness was 3.7 years (standard deviation = 2.8). All participants were
recruited through local NHS eating disorder services in London and online through BEAT eating disorders charity. Participants provided written, informed consent prior to taking part in the study. The study procedures were all completed in accordance with the latest version of the Declaration of Helsinki (2013) and the study was approved by The London-Surrey National Research Ethics Committee (17/LO/2071 & 19/SC/0367).

### 3.2 Assessments

Autistic traits were assessed using the Autism Diagnostic Observation Schedule (ADOS, (26, 27)) and the short version of the Autism Quotient (AQ10, (28)). The ADOS is a gold standard observational module used to in the diagnosis of autism and the revised algorithm was used to calculate a total score (27). The AQ10, on the other hand, is a brief self-report screening measure for autism.

ED symptom profile was assessed by collecting participants height and weight to calculate BMI and by using the following self-report questionnaires: The Eating Disorder Examination Questionnaire (EDEQ, (29)), Hospital Anxiety and Depression Scale (HADS, GL Assessments ref. 629398237, (30)), Obsessive-Compulsive Inventory (OCI, (31)), and Work and Social Adjustment Scale (WSAS, (32)).

### 3.3 Procedures

At the first time point, Time 1, participants completed the AQ10 and ED symptom profile self-report questionnaires. A subset of the participants also agreed to complete the ADOS assessment (N = 73, 70% of Time 1 sample) at Time 1. ADOS was delivered by a trained examiner and took approximately 45–60 minutes to complete. Participants’ height and weight were measured after the ADOS assessment and were used to calculate body mass index (BMI). The Time 1 assessments were a part of a larger study conducted between June 2017 and February 2019, which examined neural underpinnings of AN (33–36).

At Time 2, 6 months after the Time 1 assessments, participants were asked to complete the ED symptom profile self-report questionnaires. Participants were also asked to report their current height and weight for BMI calculation. At Time 3, 2 years after the initial Time 1 assessments, participants were asked to again complete the ED symptom profile questionnaires and report their current height and weight.

### 3.4 Data analysis

All data analysis was conducted in R (37). We first conducted a principal component analysis to reduce dimensionality of the outcome dataset. This was done by applying the function `prcomp` to the measures used to assess ED symptom profile, which included BMI, EDEQ total score, HADS anxiety score, HADS depression score, OCI total score, and WSAS score. We used the elbow method to determine the number of components that should be taken forward to further analysis. The retained principal components were then entered as outcome variables to two multivariate linear mixed effects models (LMERs) with the following predictors: Model (1) Time 1 ADOS total score and time point, Model (2) Time 1 AQ10 total score and time point. The multivariate LMERs were conducted using the `lme4` package (38). Threshold for statistical significance was set at $p < 0.05$. The data and code used in the analyses are available at https://osf.io/tqysn/?view_only=69a0f2183df14f5e82f4b728fba50f34.
Results

The elbow method indicated that the first two principal components, which explained 71.4% of the variance in the data, should be retained (Supplementary Table 1, Supplementary Fig. 1). The first principal component, PC1, was negatively associated with the WSAS score, EDEQ total score, HADS depression, HADS anxiety, and OCI score (Supplementary Table 1). The second principal component, PC2, was primarily positively associated with BMI.

There was a significant interaction between the principal components and ADOS scores in the first multivariate LMER (Table 1). There was a stronger negative association between ADOS scores and PC1 ($b = -0.19$, 95% CI [-0.26, -0.11]) than between ADOS scores and PC2 ($b = -0.04$, 95% CI [-0.11, 0.04]) ($t(267) = -3.58$, d = -0.11 95% CI [-0.18, -0.05], p < 0.001). These results together with visual inspection of the associations between ADOS scores and the ED symptom profile measures presented in Fig. 1, show higher level of autistic traits generally predicted worse presentation across all three time points, with the exception of anxiety and OCD symptoms. The first multivariate LMER also revealed significant main effects of principal component and ADOS total score. There were no other significant main effects or interactions, but the three-way interaction between Time 1 ADOS scores, principal components and time points approached significance.

Table 1

<table>
<thead>
<tr>
<th>Measure</th>
<th>Time point</th>
<th>F(DF)-statistic, p-value</th>
<th>ADOS (N = 65)</th>
<th>AQ10 (N = 105)</th>
</tr>
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<tbody>
<tr>
<td>ADOS total</td>
<td>5.27 (3.07)</td>
<td>PC: F(1,298) = 11.51, p = 0.001</td>
<td>PC: F(1,403) = 52.98, p &lt; 0.001</td>
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<tr>
<td>AQ10</td>
<td>3.83 (2.24)</td>
<td>Time point: F(2,328) = 0.79, p = 0.454</td>
<td>Time point: F(2,434) = 1.04, p = 0.354</td>
<td></td>
</tr>
<tr>
<td>PC1</td>
<td>-0.12 (1.58)</td>
<td>PC x ADOS: F(1,298) = 12.79, p &lt; 0.001</td>
<td>PC x AQ10: F(1,403) = 67.55, p &lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>PC2</td>
<td>-0.06 (0.96)</td>
<td>ADOS x Time point: F(2,239) = 0.54, p = 0.583</td>
<td>AQ10 x Time point: F(2,438) = 1.05, p = 0.349</td>
<td></td>
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</table>

ADOS = Autism Diagnostic Observation Schedule; PC = principal component; DF = degrees of freedom.
The second multivariate LMER similarly showed a significant interaction between principal component and Time 1 AQ10 scores (Table 1). The AQ10 scores were differently associated with PC1 and PC2 ($t(400) = -8.22, d = -0.33$, 95% CI [-0.41, -0.25], $p < 0.001$), such that there was a negative association between AQ10 scores and PC1 ($b = -0.32$, 95% CI [-0.41, -0.23]) and only a slight positive association between AQ10 scores and PC2 across the time points ($b = 0.06$, 95% CI [-0.04, 0.14]). Together with a visual inspection of the associations between Time 1 AQ10 scores and the ED symptom profile measures presented in Fig. 2, the results suggest that higher level of autistic traits predicted more psychological symptoms across time points but was not associated with BMI. As above there were also significant main effects of principal component and Time 1 AQ10 scores, and the three-way interaction between Time 1 AQ10 scores, principal components, and time points approach significance. There were no other significant main effects or interactions.

**Discussion**

The present study investigated whether baseline autistic traits can predict ED symptom profile at a later date in a diverse sample of women with lived experience of AN. The findings revealed that high baseline autistic traits were consistently associated with worse psychological symptoms even two years later. Interestingly, our exploratory comparison of a brief self-report screening measure of autism and a diagnostic tool showed that both methods similarly and significantly predicted psychological ED symptoms, but not BMI, which is usually one of the key outcome measures in AN research and treatment. Further visual examination of each symptom measure showed some differences between the two methods.

The present findings lend further credence to previous work documenting that people with AN who report high levels of autistic traits have more severe psychological presentation (24, 25, 39). Furthermore, the present findings add to the literature by highlighting that high levels of autistic traits predict poorer psychological outcome in long term. Although 15.3% of participants in the present study were recovered and no longer in treatment for AN, this is in line with qualitative findings that many autistic people with EDs report that currently available treatments do not adequately meet their needs forming a further barrier to recovery (40, 41). Additionally, many clinicians working ED services lack the experience and confidence needed to identify autism and effectively meet the needs of autistic ED patients, despite acknowledging that treatment adaptations are needed (42). Together, these findings highlight the importance of additional training for clinicians and neurodiversity-focused treatment adaptations to improve ED outcomes among autistic people and those with high levels of autistic traits.

The present findings show that high levels of autistic traits have strong association with complex psychological ED symptom profile, but not BMI. Similarly previous studies have documented that autistic traits were not associated with BMI, but were strongly associated with psychological symptoms and general level of social functioning, among people with lived experience of AN (22, 25). A recent examination of clinical audit data showed that AN patients reporting high levels of autistic traits had significantly higher BMI upon admission to inpatient care (39). The focus on BMI as the measure of
recovery, in particular, has been highlighted as less helpful for autistic people with AN, who discuss the psychological elements of recovery as being more difficult for them to manage (43). Furthermore, qualitative work exploring the experiences of autistic people with EDs has found that body image difficulties and drive for thinness are less relevant for this group of patients (40, 41). This may explain why autism symptomatology in this sample predicts psychological ED symptoms two years later, but not BMI and physical recovery. These findings highlight the need for a shift in thinking about the illness presentation among autistic ED patients and those with high levels of autistic traits with greater focus on psychological wellbeing and outcomes.

Interestingly, both the AQ10 and the ADOS robustly predicted psychological illness profile long term suggesting that self-report and observational autism measures have sufficient predictive power. This is of interest considering that previous work has criticised the use of measures that solely focus on current features, tendencies, and behaviours, suggesting that they may not be beneficial in detecting autistic traits in people with acute ED due to lack of exploration of the person's developmental history (11). Additionally, self-reported autistic traits have been reported to change over time among clinical populations, with one study reporting small but significant reduction in AQ10 scores following an average 16-week inpatient treatment for AN (25, 44). As autism is a neurodevelopmental condition, these findings raise questions about the explanatory power of these measures and their ability to accurately identify autistic traits. It may be worth exploring whether including retrospective questions exploring developmental history would help improve the robustness of self-report measures.

Thus far, few treatment adaptations have been introduced to better serve autistic ED patients (45–47). A recent review synthesised findings from nine studies exploring various ways to adapt interventions based on cognitive remediation therapy (CRT), emotion skills training and cognitive behavioural therapy (CBT) to better suit the needs of autistic ED patients (48). The authors also reported that autistic ED patients, or those with high levels of autistic traits, may be less likely to standard interventions, which frequently focus on altering the autistic social and cognitive processing styles. Additionally, across the studies it was apparent that autistic ED patients or those with high levels of autistic traits are unlikely to benefit from group interventions and may favour individual therapy. Only two of the studies reviewed explored the impact of proposed treatment adaptations: one presented data from a one-time sensory wellbeing workshop (46) and the other presented a case example of adaptations to the Maudsley Anorexia Nervosa Treatment for Adults (MANTRA; (47)). Although both studies resulted in some improvements among AN patients with high levels of autistic traits, there is relative paucity of research exploring the impact of neurodiversity accommodations on ED treatment outcomes and more work is needed.

**Limitations**

Although the findings from the present study are important, there are also some limitations. Firstly, only 70% of the participants who completed the self-report questionnaires at Time 1 also agreed to undergo ADOS assessment. This reduced the power of the LMER involving ADOS scores when compared to the analysis involving AQ10 scores. Additionally, the sample consisted of only young women, and it is
therefore, difficult to ascertain whether these findings extend to other genders and older age groups. Finally, the outcome measures were self-reported and participants height and weight could only be measured by the researchers at Time 1 because subsequent assessments were completed online. This may have impacted the results, particularly regarding BMI.

**Conclusions**

High levels of autistic traits were associated with poorer psychological ED symptom profile in long term regardless of the method used to assess autistic features. Interestingly, autistic traits were not associated BMI, which is supported by previous work reporting that body image and drive for thinness are less relevant for autistic people with EDs. Finally, the present study highlights the need for the development and investigation of neurodiversity focused treatment adaptations.

**List Of Abbreviations**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ADOS</td>
<td>Autism Diagnostic Observation Schedule</td>
</tr>
<tr>
<td>AN</td>
<td>Anorexia nervosa</td>
</tr>
<tr>
<td>AQ10</td>
<td>Autism Quotient, short version</td>
</tr>
<tr>
<td>BEAT</td>
<td>Beat Eating Disorders Charity</td>
</tr>
<tr>
<td>BMI</td>
<td>Body mass index</td>
</tr>
<tr>
<td>CBT</td>
<td>Cognitive behavioural therapy</td>
</tr>
<tr>
<td>CRT</td>
<td>Cognitive remediation therapy</td>
</tr>
<tr>
<td>ED(s)</td>
<td>Eating disorder(s)</td>
</tr>
<tr>
<td>EDEQ</td>
<td>Eating Disorder Examination Questionnaire</td>
</tr>
<tr>
<td>HADS</td>
<td>Hospital Anxiety and Depression Scale</td>
</tr>
<tr>
<td>LMER</td>
<td>Linear mixed effects model</td>
</tr>
<tr>
<td>MANTRA</td>
<td>Maudsley Anorexia Nervosa Treatment for Adults</td>
</tr>
<tr>
<td>NHS</td>
<td>National Health Service</td>
</tr>
<tr>
<td>OCD</td>
<td>Obsessive-compulsive disorder</td>
</tr>
<tr>
<td>OCI</td>
<td>Obsessive-Compulsive Inventory</td>
</tr>
<tr>
<td>PC</td>
<td>Principal component</td>
</tr>
<tr>
<td>WSAS</td>
<td>Work and Social Adjustment Scale</td>
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</table>

**Declarations**
9.1 Ethics approval and consent to participate

All participants provided written, informed consent prior to taking part in the study. The study was approved by The London-Surrey National Research Ethics Committee (17/LO/2071 & 19/SC/0367).

9.2 Consent for publication

Not applicable.

9.3 Availability of data and materials

The datasets generated and/or analysed during the current study are available in the OSF repository https://osf.io/tqysn/?view_only=69a0f2183df14f5e82f4b728fba50f34. This manuscript has been pre-posted on PsyArXiv: 10.31234/osf.io/rvxdk

9.4 Competing interests

The authors declare that they have no competing interests.

9.5 Funding

This research was funded in whole, or in part, by the Wellcome Trust [213578/Z/18/Z]. For the purpose of open access, the author has applied a CC BY public copyright licence to any Author Accepted Manuscript version arising from this submission. The research was further supported by MRC-MRF Fund [MR/R004595/1]. The funding bodies did not play an active role in the design of this study, nor in data collection or analysis, nor in writing the manuscript.

9.6 Authors' contributions

JL, FS, and DH collected the data used in the manuscript, interpreted the results, and made major contributions to writing the manuscript. KT and JL obtained funding for the study. KT additionally interpreted the results regarding clinical implications and made major contributions to the writeup. All authors read and approved the final manuscript.

9.7 Acknowledgements

Not applicable.
References


Figures

Figure 1

Associations between ADOS scores and ED symptom profile measures at each time point

BMI = body mass index, EDEQ = Eating Disorder Examination Questionnaire; HADS = Hospital Anxiety and Depression Scale; OCI = Obsessive-Compulsive Inventory; WSAS = Work and Social Adjustment Scale
Figure 2

 Associations between AQ10 scores and ED symptom profile measures at each time point

BMI = body mass index, EDEQ = Eating Disorder Examination Questionnaire; HADS = Hospital Anxiety and Depression Scale; OCI = Obsessive-Compulsive Inventory; WSAS = Work and Social Adjustment Scale

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryTable1.docx
- SupplementaryFig1.tiff