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Autism spectrum and disordered eating: the mediating roles of poor sleep quality and mood problems

Hasan Huseyin Ates[1] · **Emily Newman**[2] · **Joanne M. Williams**[2]

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Abstract
Existing literature on the relationship between core autistic traits and disordered eating (DE) in autistic people has limitations in relation to exploring the direction and mechanisms of this relationship. This study aimed to examine the extent to which the relationship between core autistic traits and DE is sequentially mediated through poor sleep quality and mood problems in a community sample of adolescents and adults while controlling for potential covariates. A total of 234 individuals between 16 and 67 years of age participated in the current online anonymous cross-sectional study. Participants were selected based on their response to the self-reported Autism spectrum diagnosis status and completed a battery of standardized scales, including the Abridged Version of the Autism Spectrum Quotient (AQ – Short), Shortened Pittsburgh Sleep Quality Index (sPSQI), Hospital Anxiety and Depression Scale (HADS), Eating Attitudes Test-26 (EAT-26), Inflexible Eating Questionnaire (IEQ) and Nine Item Avoidant/Restrictive Food Intake Disorder Questionnaire (NIAS). The results indicated a direct association between core autistic traits and all outcomes, and an indirect effect of core autistic traits on EAT-26 and NIAS outcomes rather than IEQ outcomes through poor sleep quality and anxious/depressive symptomology. Further exploration of the role of core autistic traits may facilitate the development of novel person-centred treatments for both Autism spectrum and DE, specifically rigid eating behaviours and ARFID symptomology.

Keywords Autism spectrum · Autistic traits · Sleep problems · Mood problems · Disordered eating

Introduction

A robust positive correlation between the number of autistic traits and disordered eating (DE) has been reported in the literature within both clinical and non-clinical samples in research on Autism Spectrum in adolescents and adults (Christensen et al., 2019; Huke et al., 2013). Despite these findings, there remains a gap in understanding the relationship between autistic traits and specific subtypes of DE, as well as the potential underlying mechanisms (Barnett et al., 2021; Galvin et al., 2022; Moseley et al., 2023; Vuillier et al., 2020). Although recent studies have begun to explore both the direct and indirect pathways through which core autistic traits contribute to DE behaviours, particularly via mediating factors such as alexithymia and mood problems, further research is required to fully elucidate the complexity of these mechanisms.

While DE is often examined as a broad construct encompassing a range of problematic eating behaviours, it is crucial to distinguish between its subtypes to better understand their unique associations with core autistic traits (Barnett et al., 2021; Moseley et al., 2023). Global DE refers to general eating pathology, including purging, bingeing, dietary restraint and body dissatisfaction, as measured by standardised questionnaires (Garner et al., 1982; Papini et al., 2022). In contrast, rigid eating behaviours involve inflexible routines around food (e.g., strict eating schedules, limited food variety) and are often characterised by strict adherence to eating rules, food avoidance and a reduced ability to eat intuitively, reflecting psychological rigidity around body image and eating behaviours (Brede et al., 2020; Duarte et al., 2017). Avoidant/Restrictive Food Intake Disorder

✉ Hasan Huseyin Ates
hasanhuseyinates@karatekin.edu.tr

[1] Department of Clinical Psychology, Faculty of Humanities and Social Sciences, Çankırı Karatekin University, Uluyazı Campus, 18100 Çankırı, Türkiye

[2] Department of Clinical Psychology, School of Health in Social Science, The University of Edinburgh, Medical School (Doorway 6), Teviot Place, Edinburgh EH8 9AG, Scotland

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(ARFID) symptomology represents a clinically distinct profile, characterised by extreme food avoidance or restriction that is not driven by concerns about body shape or weight, but by factors such as sensory sensitivity to food textures, fear of choking or vomiting, and a lack of interest in eating (Kinnaird et al., 2019; Zickgraf & Ellis, 2018). These DE constructs (Global DE, rigid eating and ARFID symptomology) are related to different psychological and behavioural mechanisms and are particularly relevant in autistic populations (Barnett et al., 2021; Brede et al., 2020; Kinnaird et al., 2019; Moseley et al., 2023). Therefore, examining these outcomes separately allows for a more precise understanding of the specific pathways linking core autistic traits to DE.

One recent study (Moseley et al., 2023) examined the relationship between core autistic traits and Eating Disorders (EDs) by investigating the serial mediating effects of alexithymia and anxious/depressive symptoms among 463 individuals with EDs. The study found that alexithymia mediated the relationship between core autistic traits and ED psychopathology both directly and indirectly through its impact on anxious/depressive symptoms, which also independently mediated this relationship. According to Moseley and colleagues (2023), autistic people with Anorexia Nervosa (AN), similar to those who are non-autistic, experience emotions such as emotional confusion and anxiety, which they try to suppress or alleviate through restrictive behaviours. Furthermore, recent semi-structured interviews with eleven autistic adults conducted by Healy and colleagues (2021) suggested that autistic people may be affected by anxiety and depression, which in turn influence emotional and binge eating behaviours. Challenges in identifying and managing emotions, coupled with anxiety and a tendency to avoid negative emotions, are considered significant factors contributing to the development and persistence of EDs (Kinnaird et al., 2019).

In contrast, no studies have yet examined the indirect effects of core autistic traits on different types of DE behaviours through mood problems in autistic people. While recent studies (Barnett et al., 2021; Galvin et al., 2022) have used hierarchical multiple regression analyses to investigate whether core autistic traits predict DE even after controlling for anxiety and depression, the inclusion of mood problems significantly increased the explained variance in DE. These findings highlight the importance of both core autistic traits and mood problems in understanding this association. However, the underlying mechanisms remain unclear, highlighting the need for further research to explore how mood problems mediate the relationship between core autistic traits and specific DE behaviours.

Children and adolescents with autistic traits are at heightened risk for developing mood disorders, with symptoms often emerging in late childhood or early childhood (Solmi et al., 2021). Difficulties in social communication, present from early childhood, may contribute to the development of DE during adolescence, particularly as social relationships become more complex and emotionally significant. Challenges informing and maintaining friendships can lead to increased anxiety and low mood, potentially fostering maladaptive coping strategies such as DE (Solmi et al., 2021). This notion is supported by existing literature that indicates pre-existing difficulties with social interactions and cognitive flexibility in individuals with EDs or DE before the onset of their eating pathology (Griffiths et al., 2019; Hoekstra et al., 2011; Mandy & Tchanturia, 2015; Solmi et al., 2021). While mood problems may play a mediating role in this process, further research is needed to clarify the complex interplay between social autistic traits, emotional difficulties and DE.

Beyond mood problems, poor sleep quality has been identified as a potential contributor to both autistic traits and DE behaviours. Autistic individuals frequently experience sleep-related difficulties, including insomnia, delayed sleep phase syndrome, and fragmented sleep patterns (Jovevska et al., 2020; Lugo et al., 2020). These disturbances are associated with increased emotional dysregulation, heightened mood problems and attentional deficits, all of which may contribute to problematic eating behaviours (Bangerter et al., 2020; Goldman et al., 2017; Kaisari et al., 2018; Weir et al., 2021). Additionally, disrupted sleep among autistic individuals is linked to greater social difficulties, cognitive inflexibility, and mood instability (Jovevska et al., 2020; Schreck & Richdale, 2020). Sleep deprivation may also disrupt the regulation of appetite-related hormones, such as ghrelin and leptin, leading to increased craving for high-calorie foods and further exacerbating DE behaviours (Schmid et al., 2008; Van Cauter et al., 2007). These findings highlight the importance of considering the bidirectional relationships between poor sleep quality, mood problems, core autistic traits and DE behaviours, as each may play a distinct role in the mechanisms underlying DE behaviours.

Given the potential interplay among these factors, it is important to recognize that sleep and mood problems may have a reciprocal relationship where poor sleep quality can contribute to mood problems, which can in turn worsen sleep disruptions (Konjarski et al., 2018). This cycle may be important for autistic individuals with DE who may be more vulnerable to the negative effects of poor sleep on mood. Autistic individuals commonly experience difficulties with emotion regulation, resulting in mood problems such as anxiety and depression (Mazefsky et al., 2013). These mood problems might subsequently contribute to the development of DE, including rigid eating and ARFID symptomology, as food might be used as a coping mechanism (Brede et al.,

2020; Kinnaird et al., 2019). Specifically, in autistic individuals, anxiety symptoms may heighten sensitivity to certain food textures, reinforce rigid eating routines, or promote restrictive dietary patterns due to an amplified fear response (Brede et al., 2020; Kinnaird et al., 2019). A qualitative study found that DE in autistic adolescents often persists into adulthood, with sensory sensitivity, medical difficulties, and cognitive rigidity identified as key contributing factors (Kinnaird et al., 2019). Given that sensory sensitivities and cognitive rigidity are core characteristics of autism, these factors may reinforce restrictive eating behaviours, as individuals strictly adhere to food routines to manage distress and maintain predictability. Thus, investigating both rigid eating and ARFID symptomology, alongside global DE behaviours, is essential to capturing distinct aspects of ED risk.

Despite growing evidence highlighting the relationships among autistic traits, sleep quality, mood problems, and DE behaviours, no studies have comprehensively examined these variables within an integrated model. Given their potential interplay, it is crucial to investigate whether poor sleep quality and mood problems act as serial mediators in the relationship between core autistic traits and DE, including global DE behaviours, rigid eating patterns and ARFID symptomology, which may inform interventions aimed at reducing DE within this subpopulation. Building on this synthesis of the literature, the current study aimed to address the following research question and hypotheses: To what extent is the relationship between core autistic traits and DE, including global DE, rigid eating and ARFID symptomology uniquely mediated by poor sleep quality and mood problems? (1) Poor sleep quality and mood problems will serially mediate the link between core autistic traits and global DE behaviours, (2) Poor sleep quality and mood problems will serially mediate the link between core autistic traits and rigid eating, (3) Poor sleep quality and mood problems will serially mediate the link between core autistic traits and ARFID symptomology.

Method

Participants

The recruitment strategy for this anonymous online cross-sectional study utilized a combination of opportunity and snowball sampling methods. The target sample included individuals aged 16 years or older who had a good understanding of written English, access to the internet, and, for autistic participants, a self-reported formal diagnosis of autism. Participants could reside anywhere in the world. Sample size calculations were performed using GPower 3.1 software (Faul et al., 2009). For linear multiple regression analyses with 15 predictors, a minimum sample size of 139 was needed, with alpha at 0.05, power of 0.80, and effect size $f^2 = 0.15$. The power calculation was based on prior research (Mansour et al., 2016) that examined the relationship between disordered eating and autistic traits in university students. A systematic effort was made to recruit a sample larger than the minimum requirement to allow for multiple complex analyses.

The study was advertised in the third sector services and centres that provide support and/or advocacy to people affected by mental health issues and specifically Autism Spectrum, Facebook and Reddit forum groups for Autism Spectrum. From June 2021 to October 2021, a total of 1255 individuals entered an anonymous online survey about their autistic traits and DE hosted by Qualtrics. Of these, 502 did not consent to take part and 24 answered all three trap questions incorrectly. From the remaining 729, we included only those who completed the required demographic and screening questionnaires and who had responded '*Yes*' to the self-reported question about receiving a formal Autism Spectrum diagnosis. This resulted in a final sample of 264 respondents. Minor missing data in some questionnaires were handled as outlined in Sect. 2.4.

Measures

Participants were asked to complete information about their demographic characteristics, including age, sex, ethnicity, country of residence, nationality, relationship, employment, education status, estimate of weight and height for calculating BMI, Autism Spectrum diagnosis history, Autism Spectrum treatment history, current diagnosis of any mental health problem, medication status and any health condition that impacts their eating behaviours or diet. Participants completed a number of standardised scales, including the following constructs as predictors, mediators, and dependent variables.

Disordered Eating Behaviours (Global DE, Rigid Eating and ARFID Symptomology)

Global DE symptomology was assessed using the Eating Attitude Test (EAT-26), a widely used self-reported questionnaires for assessing DE within adolescents or adults. It has commonly been employed as an index of the AN symptomology (Garner et al., 1982; Papini et al., 2022). The EAT-26 measures three components of DE behaviours, Bulimia and Food Preoccupation (Factor I- 6 items), Oral Control (Factor II- 7 items), and Dieting (Factor III- 13 items), which are summed to calculate a total score. Six possible responses for each 26 items (with one inverse item) ranging

from "always" to "never" was used (a score of 1, 2, and 3 was given for 'Often', 'Usually' and 'Always', respectively, and 0 for 'Never', 'Rarely' and 'Sometimes'). In this study, the total score of the EAT-26 was used as a continuous measure of DE, as suggested by Papini and colleagues (2022). Additionally, the score derived from the subscales can also serve as a continuous measure of DE. The EAT-26 has been reported to be highly reliable and valid in clinical and non-clinical settings (Papini et al., 2022). Kaisari and colleagues (2018) reported that the Cronbach's alpha in a sample of 227 individuals ranged from 0.82 to 0.88 across subscales. In the current study, the Cronbach's alpha was 0.78, 0.73 and 0.87 for the Bulimia and Food Preoccupation, Oral Control and Dieting subscales, respectively.

Rigid eating behaviours were assessed using the Inflexible Eating Questionnaire (IEQ), a self-assessment tool developed by Duarte and colleagues (2017). This 11-item self-assessment tool measures individuals' beliefs, behaviours and attitudes regarding eating and weight regulation (Duarte et al., 2017). The 11 items are rated using a 1 to 5-point response scale (1 = Fully Disagree, 2 = Disagree, 3 = Neither Agree nor Disagree, 4 = Agree, 5 = Fully Agree), which are summed to calculate a total score. A higher total score on the scale represents a higher level of inflexibility in eating behaviours based on avoidance of specific food, strict adherence to dietary rules, and persistent and rigid patterns of food choices. The IEQ has been reported to be highly reliable and valid, as presented by a Cronbach's alpha value of 0.90 in a sample of 807 women from the community (Duarte et al., 2017). In the current study, the Cronbach's alpha was found to be 0.94.

The Nine Item Avoidant/Restrictive Food Intake Disorder Questionnaire (NIAS), developed by Zickgraf and Ellis (2018), measures the severity of ARFID symptoms through three subscales: Picky Eating (Factor I- 3 items), Appetite/Interest (Factor II- 3 items) and Fear (Factor III- 3 items). Participants respond to nine items using a 6-point Likert type scale ranging from 0 to 5 (0 = Strongly Disagree, 1 = Disagree, 2 = Slightly Disagree, 3 = Slightly Agree, 4 = Agree, 5 = Strongly Agree). The scores of each item are summed to calculate separate subscale scores. The overall score of the scale is then taken by totalling all the subscale scores, with a range of 0 to 45. A higher total score indicates a greater severity of ARFID symptoms (Zickgraf & Ellis, 2018). The scale has been found to be reliable and valid (Zickgraf & Ellis, 2018), with Cronbach's alpha ranging from 0.87 to 0.93 across subscales in a sample of 766 individuals, including participants from Amazon's Mechanical Turk and undergraduate students. In this study, the Cronbach's alpha was found to be 0.90, 0.87 and 0.91 for the 'Picky Eating', 'Appetite' and 'Fear' subscales, respectively.

Autistic Traits

Core autistic traits were assessed using the Abridged Version of the Autism Spectrum Quotient (AQ-Short), a shortened version of the original 50-item self-reported Autism Quotient (AQ) questionnaire. This tool measures autistic traits among autistic and non-autistic individuals (Hoekstra et al., 2011). Four possible responses for each 28 items (with thirteen inverse items) ranging from 1 to 4 are used (1 = Definitely Agree, 2 = Agree, 3 = Disagree, 4 = Definitely Disagree). The AQ-Short measures multifactorial perspectives of autistic traits, Social Skills (Factor I- 7 items), Routine (Factor II- 4 items), Switching (Factor III- 4 items), Imagination (Factor IV- 8 items), Factor Numbers and Patterns (Factor V- 5 items). These factors are summed to calculate a total score ranging between 28 and 112, with higher scores indicating greater levels of core autistic traits. Additionally, Hoekstra and colleagues (2011) reported that the total score of the first four factors (Social Skills, Routine, Switching and Imagination) comprised a higher order of factor representing social behavioural difficulties. Thus, higher scores on all subscales indicates more pronounced autistic traits. The scale has been found to be reliable and valid in a clinical and non-clinical sample (Hoekstra et al., 2011). Kuenssberg and colleagues (2014) reported that the Cronbach's alpha in a sample of 148 autistic adults was 0.84. In this study, the alpha value was found to be 0.86.

Mood Problems, Poor Sleep Quality and ADHD Symptomology

Mood problem was assessed using the Hospital Anxiety and Depression Scale (HADS), a 14-item measure of anxiety and depression symptoms. Items are divided into two subscales of anxiety (Factor I- 7 items) and depression (Factor II- 7 items), and participants respond to items using a 4-point (0, 1, 2, and 3) rating scale. Those items are summed to calculate separate subscale scores which range from 0 to 21, where a total score of 11–21 represents abnormal levels of anxiety or depression symptomology (Bjelland et al., 2002; Zigmond & Snaith, 1983). The two subscales can also be summed to calculate an overall score of mood problems, which was used in the current study. The validity of the questionnaire regarding the factor structure, discriminant validity and internal consistency based on the 747 papers was reported by Bjelland and colleagues (2002). The mean of Cronbach's alpha for anxiety subscale was 0.83 (ranged between 0.68 and 0.93), whereas depression was 0.82 (ranged between 0.67 and 0.82). In the present study, the Cronbach's alpha was found to be 0.83 and 0.79 for the

'Anxiety' and 'Depression' subscale, respectively. Additionally, the total score yielded a Cronbach's alpha of 0.85.

Sleep quality was assessed using the Shortened Pittsburgh Sleep Quality Index (sPSQI) (Famodu et al., 2018). The Pittsburgh Sleep Quality Index assesses adult sleep latency, sleep quality, sleep efficiency, sleep duration, daytime dysfunction, and use of sleep medications, which group into seven components (Buysse et al., 1989). Famodu and colleagues (2018) reduced the scale to 13 items and subdivided it into five sleep components that are sleep latency, sleep efficiency, sleep duration, sleep disturbances and daytime dysfunction. Nine out of thirteen items use a 4-point Likert scale for each item, ranging from 0 to 3 ('Not during the past month', 'Less than once a week', 'Once or twice a week' and 'Three or more times a week') whereas the remaining four items are open-ended questions that prompt participants to provide information on their actual hours of sleep time, usual bedtime, morning waking time and the time they spent falling asleep. Those open-ended items have their own scoring, which is transformed into a 4-point Likert scale (0–3) to make them consistent with the scoring of the other nine rating items in terms of established guidelines (Famodu et al., 2018). Five components of the scale are summed to calculate an overall score ranging between 0 and 15, which was used in the current study. If participants have a higher score, it means poorer overall quality (Famodu et al., 2018). The sPSQI presents high agreement with the original 19 items questionnaire (Rho = 0.94, $p < .001$), specifically providing similar conclusions in terms of the global scores compared to the original (Famodu et al., 2018). Cronbach's alpha value in a sample of 211 students and professionals was found to be 0.60 for overall sleep quality (Bakul & Heanoy, 2022). The Cronbach's alpha was calculated to be 0.66 for sleep quality in our sample.

The presence of Attention Deficit Hyperactivity Disorder (ADHD) symptoms (inattentiveness and hyperactive-impulsive) was measured using the 6-item Adult ADHD Self-Report Scale (ASRS-v1.1), developed by Kessler and colleagues (2005). Participants respond to six-items using a 5-point scale ranging from 'Never' to 'Very Often'. According to the guidelines, participants receive one point if they mark 'Sometimes', 'Often', 'Very Often' on items 1, 2, 3 and 'Often', 'Very Often' on items 4, 5 and 6. The scores for each item are added together to calculate a total score of the questionnaire ranging between 0 and 6. A higher score represents elevated level of ADHD symptoms. The total score was used as a continuous measure of ADHD symptoms. The reliability and validity of the questionnaire have been well established (Green et al., 2019). The Cronbach's alpha value in the current study was found to be 0.76.

Procedure

This study was reviewed and received ethical approval from the Clinical and Health Psychology Research Ethics Committee at the researchers' institution. A participant information sheet informing participants of the purposes of the research, their rights within the study, and eligibility criteria was stated in the online survey's initial pages. The second page was a consent form where participants confirmed that they had read and understood the study information. All participants provided a consent declaration before proceeding to the survey questions. The anonymous online questionnaire took around 40–50 min to complete. Participants received the opportunity at the end of the survey to enter a prize draw to win a prize of a single £50 Amazon e-voucher gift for their participation, to encourage survey completion and participation. In the study, we followed data quality monitoring methods from Meade and Craig (2012). We used trap questions intermittently (3 in total) to identify participants not following instructions.

Data analysis

To account for occasional missing items in specific questionnaires, a 90% threshold was set. Since there is no standardized procedure for handling missing data across all measures, the researcher manually applied recommendations from existing literature. Guidance for handling missing data was available for the EAT-26, AQ-Short and HADS questionnaires. However, no such guidance was available for the IEQ, NIAS and sPSQI questionnaires. Therefore, based on Dong and Peng (2013), up to 10% missing data (one item) was allowed for these three questionnaires. This procedure ensured that occasional skipped items did not lead to unnecessary data exclusion.

To ensure data quality, several measures were implemented. First, three trap questions were embedded to detect inattentive responding; participants who answered all incorrectly were excluded. Second, only individuals who completed all mandatory sections of the survey were included. Additionally, SPSS Missing Value Analysis (MVA) was used to assess missing data patterns. Little's MCAR test showed non-random missing data for EAT-26 (X^2 (605, $N = 704$), 736.037, $p < .001$), IEQ (X^2 (42, $N = 704$), 69.694, $p = .005$), HADS (X^2 (65, $N = 704$), 120.410, $p < .001$), indicating potential bias. Conversely, the NIAS (X^2 (61, $N = 704$), 52.760, $p = .765$), AQ-Short (X^2 (468, $N = 704$), 514.028, $p = .070$), SPSQI (X^2 (165, $N = 704$), 178.119, $p = .230$) had random missing data. The data were assumed to be Missing at Random (MAR) and managed accordingly.

Participants were included if they: (1) were aged 16 or older, (2) self-reported a formal Autism Spectrum diagnosis

and (3) completed all primary study measures. Exclusion criteria included (1) failure to consent, (2) failing trap questions embedded in the survey and (3) excessive missing data (> 10% per key questionnaire).

All data were analysed using SPSS Version 25.0.0.1. The researcher visually inspected the data to check for any extreme outliers and to assess whether the data were normally distributed. According to George and Mallery (2010), the data collected from all the questionnaires, including their subscales, had acceptable ranges for kurtosis and skewness, except for the EAT-26 questionnaire. To address these non-normal distributions, the Winsorising method was employed.

Descriptive statistics were used to display characteristics of the sample. Pearson's correlation coefficient test was conducted to explore correlations between variables. Prior to conducting the mediation pathways analysis, multicollinearity assumptions were assessed through examining Tolerance and VIF values, in accordance with established guidelines (Coakes, 2013; Hair, 1998). The study employed PROCESS for SPSS to test for mediation effects. Three distinct models were identified, and estimated path coefficients, standard errors, coefficient of determination (R^2), *F* value, *t* value, confidence interval (CI) are presented in both table and figure format for each model. The analysis used Model 6 in PROCESS to examine the relationship between core autistic traits, DE (global DE, rigid eating, and ARFID symptomology), poor sleep quality and mood problems. The indirect effects, or mediating effects, were estimated using bootstrapping techniques with 10,000 bootstrap sample and 95% bias-corrected confidence intervals. The significance of the indirect effect was determined based on whether the confidence interval contained zero, following the guidelines of Hayes (2013).

In this study, variables that could potentially affect the relationship between core autistic traits and DE were identified beforehand based on existing research. These variables, including age, sex, BMI, comorbid psychiatric problems, current psychiatric medication use, other health conditions that could impact diet and ADHD symptoms, were controlled for in the analysis. Sex, comorbid psychiatric problems, current psychiatric medication use, and other health conditions were treated as dummy variables. For example, female participants were assigned a code of '0', while male participants were assigned a code of '1'. The analysis used a multiple serial mediation pathways model to investigate whether the poor sleep quality and/or mood problems such as anxiety and depression might mediate the relationship between core autistic traits and DE, while controlling for the aforementioned confounding variables. Figure 1 presents the model used in this study.

Results

Sample characteristics

The study included 234 participants, with a mean age of 31.8 years (± 11.5) and 58% being female. The majority of the participants were White/White British/White American and were highly educated (45.9%); 26.6% had completed an undergraduate degree, 19.3% were post-graduates, and 23.2% had college/vocational training/Apprenticeship graduates. Among the remaining participants, 23.6% had completed secondary school or equivalent, and 7.3% reported other education levels completed. The largest portion of participants comprised full-time employees, representing just under a quarter (23.9%); 20.9% were students, 21.4% reported currently unemployed, 10.7% were part-time employees, and 10.7% were self-employed. Among the remaining respondents: 2.1% were retired and 10.3% reported other employment status. The participants had a diverse range of BMI values, with a mean of 27.2 kg/m^2 and a standard deviation of 7.49 (SE = 0.50). A large proportion of participants had BMI values within the normal range (39%), while 27% were classified as obese, 20.3% were overweight, and 7.5% were underweight. These classifications were based on the World Health Organization's criteria. Some participants (6.1%) did not provide their weight information.

Relationships between variables

The Pearson's correlation test was used to look at relationships between DE, core autistic traits, mood problems and poor sleep quality, and control variables. All correlations between core autistic traits, DE behaviours, poor sleep quality and mood problems were significant, which are presented in the correlational matrix in Table 1.

Serial mediation effects of core autistic traits on disordered eating (global DE symptomology, rigid eating and ARFID symptomology) through poor sleep quality and mood problems (Hypotheses 1-2-3)

Three serial multiple mediation models were tested. Hypothesis 1 examined the indirect effect of core autistic traits on global DE symptomology, Hypothesis 2 on rigid eating, and Hypothesis 3 on ARFID symptomology. All models tested poor sleep quality and mood problems as sequential mediators using the total scores of AQ-Short, s-SPSQI and HADS questionnaires, as well as individual mediators. This gave rise to three indirect effects for each model.

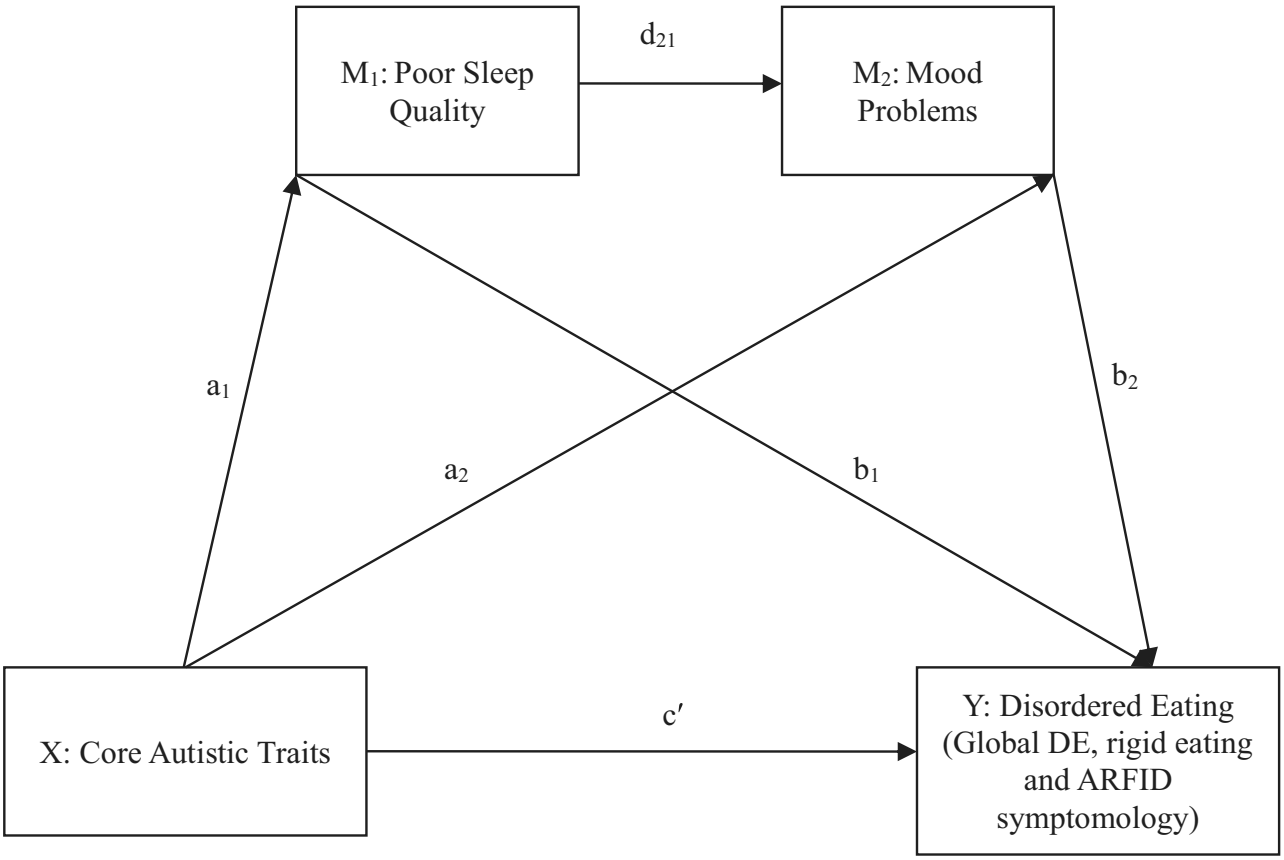

Fig. 1 Model 6 for the serial mediation role of poor sleep quality and mood problems between core autistic traits and DE. Estimates (β) are standardised regression coefficients. All analyses control for age, sex, BMI, other comorbid psychiatric problem, current psychiatric medication use, other health conditions' impact on diet and ADHD symptoms. *p<.05; **p<.01

Hypothesis 1: Serial mediation of the effect of core autistic traits on global DE symptomology

Core autistic traits predicted DE both directly and indirectly. The results (Fig. 2; Table 2) indicated that core autistic traits were associated with increased poor sleep quality and mood problems, which in turn predicted global DE symptomology. The serial indirect effect (core autistic traits → poor sleep quality → mood problems → global DE symptomology) was statistically significant ($a_1d_{21}b_2$=0.019), with a bias-corrected 95% bootstrap confidence interval of 0.002 to 0.042. These findings suggest that core autistic traits contribute to DE through the pathway of poor sleep quality and mood problems. There was also a significant specific indirect effect of core autistic traits on DE through anxiety and depression symptoms (a_2b_2=0.068, bootstrapped 95% CI 0.019–0.130), but not a specific indirect effect of core autistic traits on DE through poor sleep quality only (a_1b_1=0.018, bootstrapped 95% CI −0.012–0.067). The direct effect of core autistic traits on DE remained significant in the presence of the mediators. These findings suggest that core autistic traits influence DE symptomology through poor sleep quality and mood problems, with evidence for both serial and specific indirect pathways.

Hypothesis 2: Serial mediation of the effect of core autistic traits on rigid eating

As shown in Fig. 3; Table 3, core autistic traits predicted rigid eating directly but did not predict rigid eating indirectly through poor sleep quality and mood problems. Specifically, core autistic traits predicted increased poor sleep quality, and increased poor sleep quality predicted mood problems, but mood problems did not predict rigid eating. The serial indirect effect (core autistic traits → poor sleep quality → mood problems → rigid eating) was not statistically significant ($a_1d_{21}b_2$=0.012, bootstrapped 95% CI −0.001–0.031). These results indicate that although there was a sequential association between core autistic traits and the mediators, this pathway did not significantly mediate the relationship between core autistic traits and rigid eating. Thus, no significant serial mediation or specific indirect effects were observed for this model.

Table 1 Descriptive statistics and correlations for study variables

	N	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
1. Age	234	31.78	11.56	1																				
2. Sex	209	0.3541	0.47938	−0.007	1																			
3. BMI	222	27.223	7.4959	0.136*	0.005	1																		
4. Comorbid Psychiatric Problem	231	0.9091	0.2881	−0.099	−0.224**	0.071	1																	
5. Current Psychiatric Medication Usage	230	0.6348	0.48254	0.021	−0.148*	0.168*	0.223**	1																
6. Comorbid Health Conditions' Impact on Dieting	234	0.2821	0.45096	−0.019	−0.019	−0.058	0.1	0.209**	1															
7. ADHD Symptoms[a]	234	3.96	1.648	−0.004	0.023	0.068	0.031	0.131*	0.067	1														
8. Dieting[b]	234	7.71	7.342	−0.114	−0.125	−0.008	0.167*	0.129	0.11	0.127	1													
9. Bulimia and Food Preoccupation[b]	234	2.34	3.107	−0.038	−0.064	0.183**	0.159*	0.11	0.056	0.097	0.642**	1												
10. Oral Control[b]	234	2.66	3.099	−0.194**	−0.055	−0.342**	0.064	−0.014	0.059	0.194**	0.262**	0.053	1											
11. Global Disordered Eating[b]	234	12.7179	10.74268	−0.145*	−0.12	−0.052	0.179**	0.116	0.109	0.171**	0.945**	0.743**	0.483**	1										
12. Rigid Eating[c]	234	33.28	11.433	−0.063	−0.078	−0.106	0.164*	0.051	0.099	0.118	0.694**	0.460**	0.356**	0.710**	1									
13. Picky Eating[d]	234	8.12	4.604	−0.139*	−0.053	0.06	0.087	0.063	−0.002	0.098	0.166*	0.074	0.167*	0.183**	0.249**	1								
14. Appetite[d]	234	5.7	4.692	−0.144*	−0.099	−0.302**	0.116	0.054	0.118	0.091	0.135*	−0.129*	0.527**	0.207**	0.226**	0.379**	1							
15. Fear[d]	234	4.12	4.406	−0.116	−0.1	−0.019	0.188**	0.212**	0.296**	0.086	0.248**	0.109	0.345**	0.301**	0.308**	0.338**	0.398**	1						
16. ARFID Symptomology[d]	234	17.94	10.449	−0.175**	−0.11	−0.119	0.170**	0.141*	0.177**	0.12	0.239**	0.021	0.455**	0.300**	0.341**	0.753**	0.784**	0.750**	1					
17. Core Autistic Traits[e]	234	86.9658	9.76939	−0.005	0.001	0.048	0.113	0.012	−0.032	0.209**	0.200**	0.117	0.199**	0.228**	0.250**	0.231**	0.211**	0.136*	0.254**	1				
18. Anxiety[f]	234	12.21	4.162	−0.079	−0.05	−0.043	0.237**	0.228**	0.076	0.230**	0.309**	0.283**	0.251**	0.365**	0.333**	0.266**	0.225**	0.342**	0.363**	0.289**	1			
19. Depression[f]	234	8.42	4.457	0.059	0.094	0.109	0.218**	0.229**	0.086	0.159*	0.266**	0.339**	0.078	0.302**	0.202**	0.175**	0.182**	0.184**	0.236**	0.265**	0.506**	1		
20. Mood Problems[f]	234	20.63	7.481	−0.009	0.028	0.042	0.262**	0.264**	0.093	0.223**	0.330**	0.359**	0.186**	0.384**	0.306**	0.252**	0.234**	0.300**	0.342**	0.319**	0.858**	0.877**	1	
21. Poor Sleep Quality[g]	234	7.36	2.93	−0.07	−0.072	0.190**	0.102	0.161*	0.053	0.290**	0.243**	0.301**	0.089	0.279**	0.218**	0.123	0.084	0.197**	0.175**	0.210**	0.447**	0.413**	0.495**	1

*$p<.05$; **$p<.01$ [a]Adult ADHD Self-Report Scale (ASRS-v.1.1), [b]The Eating Attitudes Test 26 (EAT26) [c]Infilexiable Eating Questionnaire(IEQ), [d]Nine Item Avoidant Restrictive Food Intake Disorder Scale (NIAS), [e]The Abridged Version of the Autism Quotient (AQ-Short), [f]Hospital Anxiety and Depression Scale (HADS) [g]Shortening of the Pittsburgh Sleep Quality Index (SPSQI)

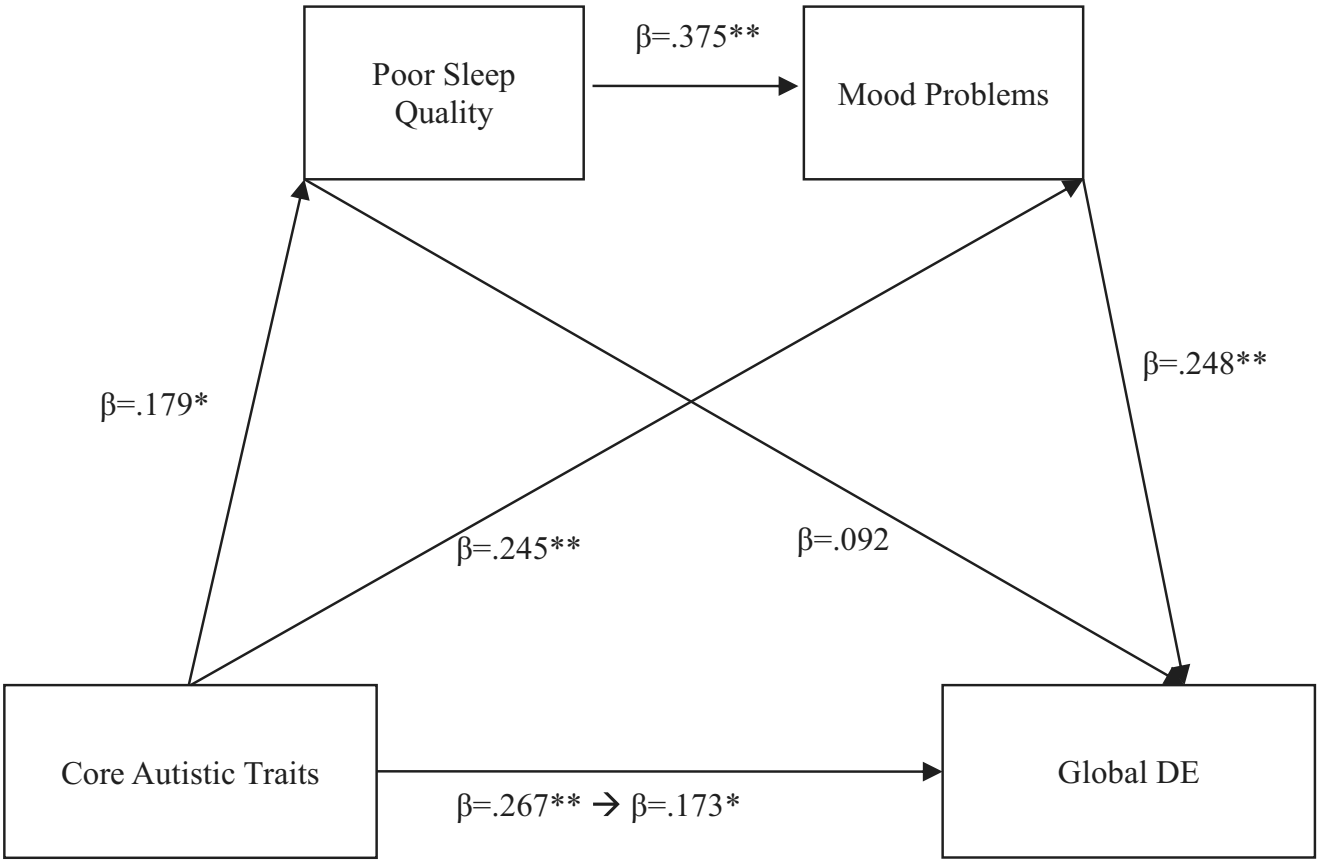

Fig. 2 Model 6 for the serial mediation role of poor sleep quality and mood problems between core autistic traits and global DE. Estimates (β) are standardised regression coefficients. All analyses control for age, sex, BMI, other comorbid psychiatric problems, current psychiatric medication use, other health conditions' impact on diet and ADHD symptoms *p<.05; **p<.01

Table 2 Summary of serial mediation analysis for core autistic traits predicting global disordered eating through poor sleep quality and mood problems

	R	R^2	F	B	B_{SE}	β	t	95%CI
Pathways 1								
Core Autistic Traits[b] → Global DE[a]	0.402	0.162	4.411**	0.300	0.079	0.267	3.788**	0.144-0.456
Pathways 2								
Core Autistic Traits → Poor Sleep Quality[c]	0.422	0.178	4.967**	0.054	0.021	0.179	2.562*	0.012-0.095
Pathways 3								
Core Autistic Traits → Mood Problems[d]	0.598	0.358	11.260**	0.190	0.049	0.245	3.895**	0.094-0.286
Poor Sleep Quality → Mood Problems				0.970	0.169	0.375	5.729**	0.636-1.304
Pathways 4								
Core Autistic Traits → Global DE	0.479	0.229	5.384**	0.194	0.081	0.173	2.405*	0.035-0.354
Poor Sleep Quality → Global DE				0.345	0.293	0.092	1.178	−0.233-0.923
Mood Problems → Global DE				0.359	0.118	0.248	3.043**	0.126-0.592

[a]Eating Attitudes Test (EAT-26), [b]The Abridged Version of the Autism Quotient (AQ-Short), [c]Shortening of the Pittsburgh Sleep Quality Index (SPSQI), [d]Hospital Anxiety and Depression Scale. Sample size (n) is 192. *p<.05; **p<.01.

Hypothesis 3: Serial mediation of the effect of core autistic traits on ARFID symptomology

Core autistic traits predicted ARFID symptomology both directly and indirectly. As shown in Fig. 4; Table 4, increased core autistic traits predicted increased poor sleep quality and mood problems, and increased poor sleep quality and mood problems predicted ARFID symptomology. The serial indirect effect (core autistic traits → poor sleep quality → mood problems → ARFID symptomology) was statistically significant ($a_1d_{21}b_2$=0.022), with a bias-corrected 95% bootstrap confidence interval of 0.002 to 0.048. Among the specific indirect effects, the pathway through mood problems alone (a_2b_2=0.079, bootstrapped 95% CI 0.024–0.153) was

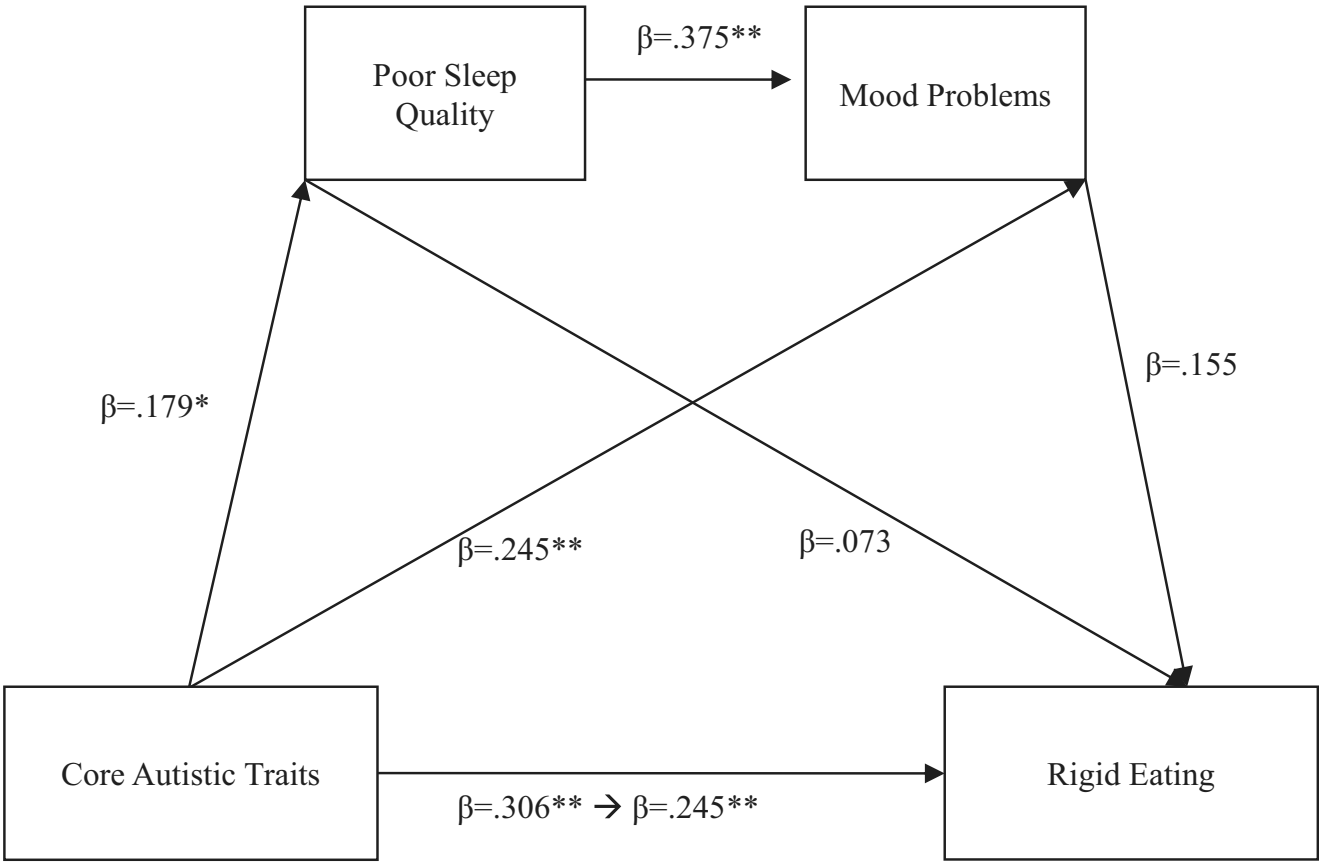

Fig. 3 Model 6 for the serial mediation role of poor sleep quality and mood problems between core autistic traits and rigid eating. Estimates (β) are standardised regression coefficients. All analyses control for age, sex, BMI, other comorbid psychiatric problems, current psychiatric medication use, other health conditions' impact on diet and ADHD symptoms *p<.05; **p<.01

Table 3 Summary of serial mediation analysis for core autistic traits predicting rigid eating through poor sleep quality and mood problems

	R	R^2	F	B	B_{SE}	β	t	95%CI
Pathways 1								
Core Autistic Traits[b] → Rigid Eating[a]	0.411	0.169	4.653**	0.350	0.080	0.306	4.363**	0.192-0.509
Pathways 2								
Core Autistic Traits → Poor Sleep Quality[c]	0.422	0.178	4.967**	0.054	0.021	0.179	2.562*	0.012-0.095
Pathways 3								
Core Autistic Traits → Mood Problems[d]	0.598	0.358	11.260**	0.190	0.049	0.245	3.895**	0.094-0.286
Poor Sleep Quality → Mood Problems				0.970	0.169	0.375	5.729**	0.636-1.304
Pathways 4								
Core Autistic Traits → Rigid Eating	0.446	0.198	4.483**	0.280	0.084	0.245	3.335**	0.114-0.446
Poor Sleep Quality → Rigid Eating				0.278	0.305	0.073	0.914	−0.323-0.879
Mood Problems → Rigid Eating				0.229	0.123	0.155	1.863	−0.014-0.471

[a]Infilexiable Eating Questionnaire (IEQ), [b]The Abridged Version of the Autism Quotient (AQ-Short), [c]Shortening of the Pittsburgh Sleep Quality Index (SPSQI), [d]Hospital Anxiety and Depression Scale. Sample size (n) is 192. *p<.05; **p<.01

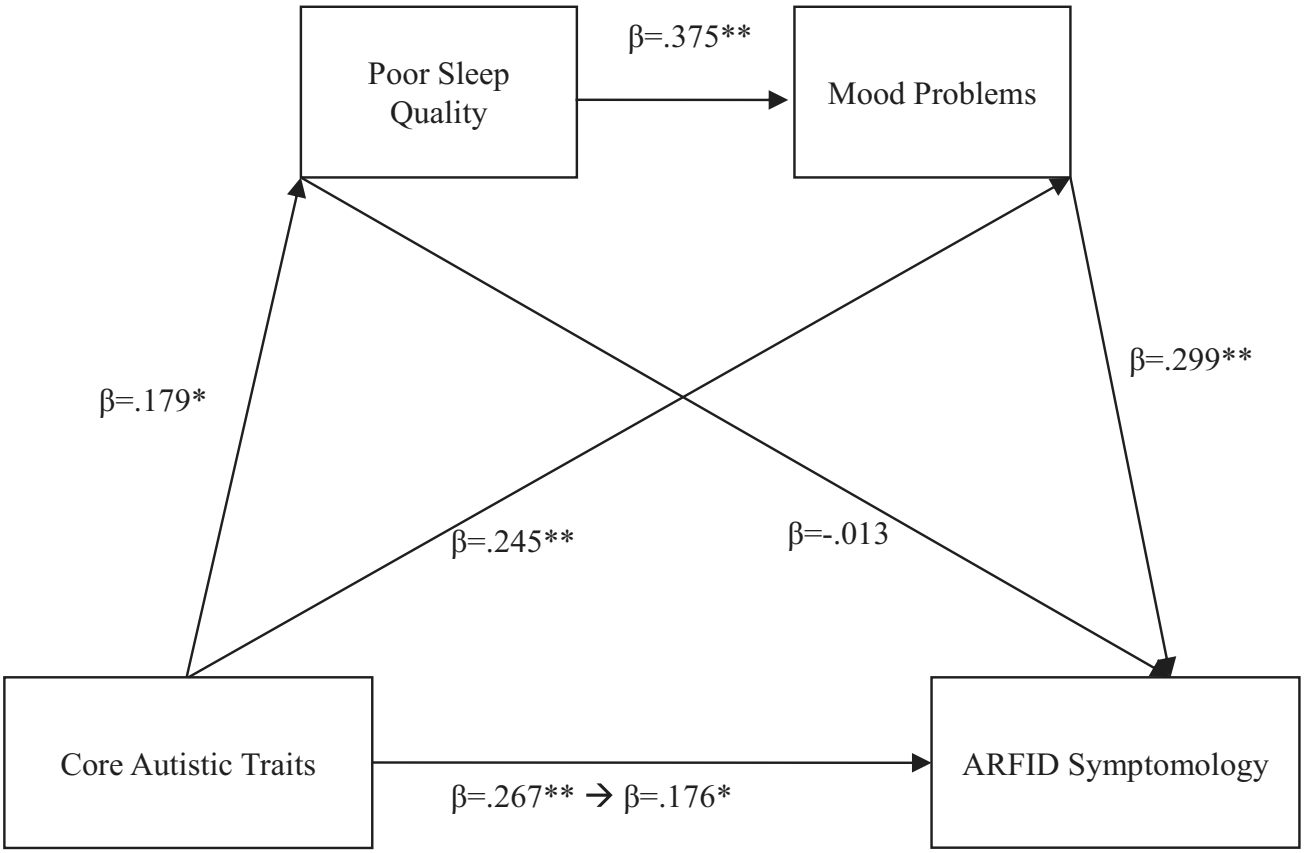

Fig. 4 Model 6 for the serial mediation role of poor sleep quality and mood problems between core autistic traits and ARFID symptomology. Estimates (β) are standardised regression coefficients. All analyses control for age, sex, BMI, other comorbid psychiatric problems, current psychiatric medication use, other health conditions' impact on diet and ADHD symptoms *p<.05; **p<.01

Table 4 Summary of serial mediation analysis for core autistic traits predicting ARFID symptomology through poor sleep quality and mood problems

	R	R^2	F	B	B_{SE}	β	t	95%CI
Pathways 1								
Core Autistic Traits[b] → ARFID Symptomology[a]	0.387	0.150	4.032**	0.287	0.076	0.267	3.756**	0.136-0.438
Pathways 2								
Core Autistic Traits → Poor Sleep Quality[c]	0.422	0.178	4.967**	0.054	0.021	0.179	2.562*	0.012-0.095
Pathways 3								
Core Autistic Traits → Mood Problems[d]	0.598	0.358	11.260**	0.190	0.049	0.245	3.895**	0.094-0.286
Poor Sleep Quality → Mood Problems				0.970	0.169	0.375	5.729**	0.636-1.304
Pathways 4								
Core Autistic Traits → ARFID Symptomology	0.464	0.215	4.963**	0.189	0.078	0.176	2.418*	0.035-0.343
Poor Sleep Quality → ARFID Symptomology				−0.048	0.283	−0.013	−0.168	−0.607-0.511
Mood Problems → ARFID Symptomology				0.415	0.114	0.299	3.636**	0.190-0.641

[a]Nine Item Avoidant Restrictive Food Intake Disorder Scale (NIAS), [b]The Abridged Version of the Autism Quotient (AQ-Short), [c]Shortening of the Pittsburgh Sleep Quality Index (SPSQI), [d]Hospital Anxiety and Depression Scale. Sample size (n) is 192. *p<.05; **p<.01

significant, whereas the pathway through poor sleep quality alone (a_1b_1 = −0.003, bootstrapped 95% CI −0.038–0.34) was not. The direct effect of core autistic traits on ARFID symptomology remained significant in the presence of the mediators. These findings support a mediated serial pathway, suggesting that poor sleep quality and mood problems together contribute to the association between core autistic traits and ARFID symptomology.

Discussion

This study aimed to examine the extent to which the relationship between core autistic traits and DE is mediated through poor sleep quality and mood problems in a community sample of adolescents and adults while controlling for potential covariates. To our knowledge, this is the first study to assess serial mediation with these specific mediators between core autistic traits and different types of DE, including global DE, rigid eating behaviours and ARFID symptomology. The results indicated both direct and indirect effect of core autistic traits on global DE and ARFID symptomology. Specifically, core autistic traits were associated with poor sleep quality and mood problems, which in turn predicted higher levels of global DE and ARFID symptomology. These results provide evidence for serial mediation, suggesting that the pathway from core autistic traits to certain DE outcomes operates through a sequence of poor sleep quality followed by mood problems. However, core autistic traits were found to be a significant direct predictor of rigid eating behaviours, but no significant indirect effects were observed through poor sleep quality and mood problems. This suggests that rigid eating may be more directly associated with core autistic traits rather than being explained through the proposed mediation pathway.

A main theme emerging from our findings is that both poor sleep quality and mood problems appear to play a key role in the association between Autism Spectrum and global DE and ARFID symptomology. These findings align with the majority of previous clinical ED, Autism Spectrum and DE literature on the association between pathological eating, autistic traits and mood problems (Moseley et al., 2023; Weir et al., 2021). Firstly, our findings indicate core autistic traits predict poor sleep quality, which in turn predicts mood problems (anxiety and depression). Similarly, Moseley and colleagues (2023) investigated whether alexithymia and mood problems mediated the relationship between core autistic traits and pathological eating among individuals with formally diagnosed and suspected ED and found that core autistic traits had a significant total effect on ED psychopathology, and this effect was underpinned by three indirect effects, including through alexithymia alone, mood problems alone, and through alexithymia and then mood problems sequentially (Moseley et al., 2023). Similarly, our study demonstrates a significant total effect of core autistic traits on global DE, rigid eating behaviours and ARFID symptomology. Two distinct indirect pathways emerged for global DE and ARFID symptomology: one through mood problems alone and another through serial mediation, where poor sleep quality led to increased mood problems, which in turn predicted DE outcomes. Importantly, no significant indirect effects – either specific or serial – were observed in the relationship between core autistic traits and rigid eating behaviours. This suggests that rigid eating may be primarily influenced through direct pathways rather than through sleep or mood-related difficulties. Our findings highlight the complex interplay between core autistic traits, mood problems, sleep quality and eating behaviours and suggest that further investigation into these relationships is warranted.

While no previous studies have explored the mediating role of poor sleep quality in the link between autistic traits and various forms of DE, there is evidence suggesting that sleep issues are prevalent in autistic individuals. For example, Weir and colleagues (2021) examined the diet, sleep, BMI and exercise behaviours of 2364 individuals, including 1882 autistic individuals, ranging in age from 16 to 90 years. The study found that autistic individuals, particularly women, exhibit unhealthy sleep patterns and dietary habits and are more likely to be underweight or obese. Additionally, existing studies on sleep and DE in autistic individuals have predominantly focused on children, with limited research on adolescents and adults. For example, studies have shown that sleep disturbances in autistic children is associated with challenges in daily living skills, social communication and intellectual development (Yang et al., 2018). Similarly, Güller and Yaylacı (2022) found that a significant proportion of autistic children (83.3%) experience sleep and eating problems, with a positive correlation between autistic traits, sleep disturbances and eating difficulties. Our study's findings align with these investigations of the relationship between core autistic traits, sleep and eating problems. We found that poor sleep quality did not significantly mediate the relationship between core autistic traits and DE. However, evidence of serial mediation emerged, indicating that poor sleep quality indirectly influenced DE through its impact on mood problems. These findings highlight the complexity of the relationship and suggest that mood-related difficulties may be a key mechanism linking sleep disturbances associated with core autistic traits to DE outcomes. Taken together, the findings from the current study and previous research emphasize the importance of developing targeted treatments to address sleep and mood problems among autistic adolescents and adults with concurrent EDs or DE.

Understanding the causal links or mechanisms by which core autistic traits increase vulnerability to DE behaviours in autistic individuals is crucial. Both autistic individuals and those with high levels of autistic traits share common features with individuals with EDs or DE (Barnett et al., 2021), including poor sleep quality and symptoms of anxiety and depression (Moseley et al., 2023; Weir et al., 2021). Moreover, autistic individuals with AN, like their non-autistic counterparts (Cooper et al., 2020), also report experiencing poor sleep quality and anxiety, which they attempt to alleviate through DE (Brede et al., 2020; Schreck & Richdale, 2020). The cognitive-interpersonal research approach, along with insights from individuals with EDs or DE, emphasizes the crucial role of cognitive processes such as attention to detail and switching, as well as interpersonal relationships characterised by difficulties in social interactions, emotion regulation and understanding social cues (Huke et al., 2013). These factors contribute to the understanding and management of DE in both autistic and non-autistic individuals (Huke et al., 2013) and also relate to poor sleep quality and mood problems (Brede et al., 2020; Schreck & Richdale, 2020). However, the specific indirect pathways through which core autistic traits influence DE, including ARFID symptomology, via poor sleep quality and mood problems in serial order are not yet fully understood. There is some evidence to suggest that poor sleep quality and mood problems may play a role in this relationship, such as social communication and interaction difficulties, psychological stress, sensory processing issues and executive function differences (Boyd et al., 2010; Brede et al., 2020; Gernert et al., 2024; Huke et al., 2013).

Research has linked physiological stress to sensory sensitivity in autism, yet the mechanisms connecting sensory over-responsivity, stress-related autonomic arousal and the severity of autistic and anxiety traits remain unclear (Gernert et al., 2024). Beyond autism, sensory processing patterns significantly affect sleep quality, and sleep disturbances are bidirectionally associated with anxiety and depression (Gernert et al., 2024). Sleep difficulties are highly prevalent among autistic individuals, with 85.6% reporting at least one sleep-related concern (Weir et al., 2021), and are positively correlated with autistic trait severity (Schreck & Richdale, 2020). Meta-analyses reveal reduced sleep efficiency and increased light sleep in autistic individuals across the lifespan (Morgan et al., 2020). Given that sensory over-responsivity can trigger chronic psychological stress, which disrupts autonomic regulation, these stress responses may contribute to difficulties in falling and staying asleep. Poor sleep not only exacerbates psychological difficulties, adaptive functioning challenges and physical health concerns, but also heightens vulnerability to circadian rhythm disruptions (Gernert et al., 2024; Schreck & Richdale, 2020).

Given the strong associations between sensory sensitivity, physiological stress, and sleep disturbances in autism, these disruptions may have downstream effects on emotion regulation and eating behaviours, which could contribute to DE. Sleep disturbances, for instance, are common among autistic individuals and may exacerbate mood disturbances such as anxiety and depression, which in turn may lead to DE, including rigid eating and ARFID symptomology (Brede et al., 2020; Kinnaird et al., 2019). The serial mediation model suggests that sleep problems may first contribute to mood problems, such as anxiety and depression, which then increase the likelihood of DE, including ARFID symptomology. In particular, anxiety symptoms may amplify food-related avoidance behaviours by increasing intolerance to certain food textures, rigid adherence to eating routines or restrictive dietary patterns driven by heightened fear response (Brede et al., 2020; Kinnaird et al., 2019). Individuals may engage in restrictive eating as a coping mechanism to regain a sense of control and emotional stability, mitigating the risk of overwhelming emotional distress (Brede et al., 2020). Furthermore, poor sleep quality, influenced by core autistic traits such as social difficulties, can heighten stress and anxiety in social situations (Richdale & Schreck, 2009; Schreck & Richdale, 2020), further contributing to DE.

Sensory processing issues, including hypersensitivity and hyposensitivity, are prevalent in autism and can disrupt sleep by making it difficult to fall or stay asleep (Boyd et al., 2010; Schreck & Richdale, 2020). These sleep disturbances, in turn, exacerbate mood problems such as anxiety and depression (Richdale & Schreck, 2009; Schreck & Richdale, 2020), increasing vulnerability to DE, including ARFID symptomology (Brede et al., 2020). Moreover, altered executive function abilities, such as difficulties with planning, organization, and time management, are common among autistic individuals (Demetriou et al., 2018) and may contribute to DE (Ghiotto et al., 2022). Impaired executive function also affects emotional and behavioural regulation (Godefroy et al., 2008), further worsening sleep quality, anxiety, and depression (Olaithe et al., 2018). By disrupting self-regulation processes, executive function deficits may increase susceptibility to both sleep disturbances and stress-related eating behaviours. Consequently, difficulties in social communication, sensory over-responsivity and executive function deficits contribute to poor sleep quality, exacerbating anxiety and depression, which in turn increase the likelihood of DE such as rigid eating and ARFID symptomology.

However, while these factors provide insight into the potential mechanisms at play, the exact indirect pathways through which core autistic traits influence DE, including ARFID symptomology, via poor sleep quality and mood problems remain unclear. Our findings indicate that the relationship between core autistic traits and DE, including

ARFID symptomology is mediated through a serial pathway involving poor sleep quality and mood problems. This suggests that there may be complex underlying mechanisms at play. It should also be noted that sleep and mood problems may have a reciprocal relationship where poor sleep quality can contribute to mood problems, which can in turn worsen sleep disruptions (Konjarski et al., 2018). This cycle may be important for autistic individuals with DE who may be more vulnerable to the negative effects of poor sleep on mood. For instance, autistic individuals commonly experience difficulties with emotion regulation, resulting in mood problems such as anxiety and depression (Mazefsky et al., 2013). These mood problems might subsequently contribute to the development of DE, including ARFID symptomology, as food might be used as a coping mechanism (Brede et al., 2020; Kinnaird et al., 2019). Furthermore, mood problems may disrupt sleep patterns, which can further exacerbate difficulties with emotional regulation (Konjarski et al., 2018). Therefore, further research is necessary to fully understand these mechanisms. While we did not find significant indirect effects of core autistic traits on rigid eating behaviours through poor sleep quality and mood problems, either as individual mediators or serially, there is consistent evidence of the relationship between anxiety and rigid eating behaviours (Arlt et al., 2016). Therefore, it is necessary to investigate specific mood problems such as anxiety and depression as potential mediators between core autistic traits and rigid eating behaviours among autistic adolescents and adults.

Strengths, limitations and implications

The sample includes male and female adolescents and adults, allowing us to attain adequate statistical power to identify small to medium sized effects with confidence. This enhances the generalizability of the findings to the wider population. Missing data on covariate variables, such as age, sex and BMI, limited the inclusion of certain participants in our regression models. This may have introduced biases, particularly in understanding subgroup differences. Future research should consider implementing stricter data completeness criteria or imputation techniques to address missing data. Our data collection strategy allowed participants to skip questions, reducing pressure to answer, and offered options like 'other' or 'would rather not to say' for the sex question, leading to missing data in these variables. As a result, approximately 192 autistic participants were included in our final sample size for analysis. Despite excluding some participants from the analyses due to missing data, the sample size in our study remains adequality powered for the analyses presented.

Furthermore, we utilized validated measures and adjusted for various potential confounding factors, including age, sex, BMI, other concurrent psychiatric condition, current psychiatric medication use, other health conditions' influence on diet and ADHD characteristics in all analyses. However, the current study did not control for intellectual functioning, a crucial factor that significantly influences the development and manifestation of Autism Spectrum. Intellectual functioning has a broad impact on various aspects of an individual's life, including emotional regulation, communication skills and adaptive behaviours (Kraper et al., 2017). By not considering this important variable, the study findings may limit a comprehensive understanding of the relationship between Autism Spectrum and DE, hindering the ability to discern the specific mechanisms underlying this association. Future studies should incorporate standardized cognitive assessments (e.g. WASI-II) to ensure a more comprehensive understanding of these relationships.

While web-based recruitment allowed access to a diverse and broad sample, we acknowledge that self-selection bias and reliance on self-reported diagnoses may limit generalizability. These factors, along with missing data concerns, should be considered when interpreting the findings, as they may introduce biases affecting subgroup comparisons and overall representativeness.

Although our study found that poor sleep quality was not a significant individual mediator between core autistic traits and DE, we did find that both poor sleep quality and mood problems were significant mediators between core autistic traits and DE, including ARFID symptomology. It is important to include objective sleep measures like actigraphy or polysomnography in combination with self-reported sleep measure to minimize recall bias and obtain a more comprehensive assessment of sleep quality. For example, actigraphy could be used to track sleep duration and movement patterns over multiple nights while polysomnography could provide detailed data on sleep architecture. These allows researchers to differentiate between various types of sleep disturbances. Future studies should consider incorporating a multi-method assessment approach, where self-reported sleep quality is supplemented with objective measures to better understand the role of poor sleep quality as an individual mediator between core autistic traits and DE. While we propose that core autistic traits are linked with DE and ARFID symptomology through mood problems, longitudinal studies using objective sleep tracking across different time points are necessary to investigate the relationships between these variables over time. However, the present study is a preliminary effort to elucidate the variables that could potentially account for the complex association between core autistic traits and DE.

The present findings highlight the significance of mood problems in the manifestation of various DE behaviours among autistic individuals. Previous studies have established that anxiety and depression significantly impact the

association between core autistic traits and DE (Barnett et al., 2021; Galvin et al., 2022). Expanding on this, our study identifies poor sleep quality as an underlying factor contributing to mood problems in autistic individuals. We found that more severe poor sleep quality exacerbates mood problems, which in turn, may increase the severity of DE behaviours, including ARFID symptomology. This underscores the importance of targeting poor sleep quality as part of interventions for autistic adolescents and adults with co-occurring mood and eating difficulties.

Given the strong link between sleep disturbances, mood dysregulation and DE behaviours, our findings emphasize the need to develop and adapt prevention and intervention programs that address autistic traits, mood problems and sleep quality. Numerous studies have focused on Cognitive Behavioural Therapy (CBT) as the primary intervention for individuals with chronic AN, consistently demonstrating greater efficacy compared to specialist supportive clinical management (Starzomska et al., 2020). Similarly, CBT for Insomnia (CBT-I) has demonstrated long-lasting improvements in sleep quality and has been adapted to meet the needs of individuals with various mental health conditions (Hertenstein et al., 2022). For example, Jernelöv and colleagues (2019) examined the effectiveness of CBT-I individuals with ADHD and sleep problems, demonstrating significant reductions insomnia severity immediately after treatment and at a three-month follow-up. These findings suggest that tailored CBT-I protocols for autistic individuals with DE symptoms may be beneficial. Potential adaptations could include sensory-friendly modifications, structured routines and psychoeducation on how sleep quality influence mood and eating behaviours.

Beyond CBT-I, other behavioural sleep interventions may also be effective. Hunter and colleagues (2020) conducted a systematic review on how behavioural sleep interventions in autistic children and adolescents affected their daytime functioning and wellbeing and found improvements in their symptoms of anxiety and depression, externalizing difficulties, emotional functioning and reduction in stereotypic and restricted behaviours. To address these issues, sleep interventions should be incorporated into the therapeutic approach for autistic individuals who have mood and DE problems. Additionally, recent reviews support Metacognitive Therapy and Rumination-Focused Cognitive Behavioural Therapy as effective interventions for reducing rumination and negative thinking, anxiety and depression, potentially preventing EDs and showing promise in reducing binge eating behaviours and improving cognitions in individuals with BED (Leppanen et al., 2022). Future research should explore the integration of these interventions with sleep-focused treatments to address the interrelated challenges of poor sleep, mood dysregulation and DE behaviours in autistic individuals.

Conclusion

Core autistic traits are a predictor of DE, including ARFID symptomology through higher levels of poor sleep quality, anxiety and depression symptoms. Importantly, core autistic traits directly predict rigid eating behaviours, with no evidence of indirect effects through poor sleep quality and mood problems. Further exploration of the role of core autistic traits may facilitate the development of novel person-centred managements for both Autism Spectrum and DE, specifically rigid eating behaviours and ARFID symptomology. Additional research is needed to better comprehend the underlying mechanisms of the relationship between Autism Spectrum and DE through negative affect since various factors may be associated with mood problems among autistic individuals, such as alexithymia.

Authors' contribution All authors have approved the final manuscript. HHA, EN, JMW contributed to the study conception and design. Material preparation, data collection and analysis were performed by HHA. The manuscript was drafted by HHA. EN and JMW provided revisions to the manuscript.

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Data availability Data is available upon request from the authors.

Code availability Code availability was not applicable.

Declarations

This article is based on the first author's doctoral dissertation, which is available at: https://era.ed.ac.uk/handle/1842/41746.
The authors confirm that this review is an entirely original work, and work and/or words of others have been appropriately cited or quoted.
This study did not receive any specific grant from funding agencies in the public, commercial, or not for profit section.
I, Hasan Huseyin Ates, can confirm that this manuscript is not under consideration elsewhere.

Ethics approval This study was reviewed and received ethical approval from the Clinical Psychology Research Ethics Committee at the School of Health in Social Science in the University of Edinburgh on 3rd June 2021 (Reference No: CLPS025).All procedures carried out in studies involving human participants followed the ethical guidelines set by the institutional and/or national research committee, as well as complied with the 1964 Helsinki declaration and its later revisions, or equivalent ethical standards.

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

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

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