Longitudinal associations between traits of autism, ADHD, and disordered eating behaviours in adolescence: a UK population-based study.

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DClinPsy Thesis (Volume 1), 2025

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Thesis declaration form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Signature:

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Date: 18/08/2025

Overview

This thesis explores the observed association between traits of autism, attention-deficit/hyperactivity disorder (ADHD), and disordered eating from a longitudinal perspective. The thesis is comprised of three parts which are outlined below.

Part one is a systematic review of 21 studies looking at the longitudinal associations between autism, ADHD, and disordered eating. The review examines the risk of future disordered eating as well as possible causal mechanisms identified by these studies. The results support the known associations from cross-sectional studies with some evidence to suggest there is not an increased risk for those with traits of autism and ADHD. Several possible causal mechanisms have been identified, including co-occurring mental health difficulties.

Part two of the thesis is an empirical paper. The study used data from a population-based cohort study to explore the longitudinal associations between traits of autism, ADHD, and future disordered eating. The study also explored this relationship in terms of co-occurring autism and ADHD which is yet to be established. Latent growth curve modelling is used to estimate developmental trajectories of traits of autism and ADHD with logistic regressions used to ascertain their associations with future disordered eating. The results indicate an association between traits of autism and ADHD with future disordered eating behaviours.

Part three of the thesis is a critical appraisal of the research process across both the systematic review and the empirical paper. This account discusses the challenges that occurred, consideration of the decision-making process, along with a wider reflection on the status of research within the field.

Impact Statement

The systematic review presented in this thesis provided a synthesis of research exploring the longitudinal associations between traits of autism, ADHD, and disordered eating along with possible mediating or moderating factors proposed to underlie this relationship. The systematic review highlighted that there are inconsistencies across the findings of research within this area identifying that this relationship is complex and not well understood. The review also identified that there is a lack of research considering the co-occurrence of autism and ADHD despite the well-known high rates of comorbidity.

The implications of this systematic review will be of importance and benefit to both researchers, clinicians, and those with lived experience. For those within academia, this provides a unique synthesis of the current state of the research base and avenues for further research to improve the understanding of these associations, especially within co-occurring conditions. This directly translates into clinical practice to consider the development of novel interventions or adaptations to current treatment programmes to provide neurodiverse individuals with better outcomes.

The empirical section of this thesis presents original research which aimed to further understand to longitudinal associations between autism, ADHD, and disordered eating from a trait-level perspective. The paper also aimed to explore the consequences of co-occurring traits of autism and ADHD on future disordered eating as well as the gender differences within this population. This research is important given the biases in diagnoses across genders with females less likely to receive a diagnosis of autism or ADHD and males being underdiagnosed with disordered eating. Further, there is evidence to suggest that those who are neurodivergent have poorer outcomes within eating disorder services. Thus, by looking at traits of autism, ADHD and disordered eating we can somewhat account for these biases in

diagnosis and provide a greater understanding of the extent to which this association exists. This will then support the development of evidence-based interventions that are tailored to the needs of neurodivergent individuals offering people the best opportunity for a successful outcome from a psychological perspective.

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Acknowledgements

Firstly, I want to acknowledge the support, guidance and insights of my supervisors Will Mandy and Virginia Carter Leno. Two formidable researchers who through all the trials and tribulations of completing this thesis have shown me unwavering support even when it seemed impossible. Thank you for being incredibly patient with me whilst I attempted to learn this whole new language and set of statistical skills I didn't know existed or was even possible to achieve. Whilst this thesis had not gone to plan, and near the end I was about ready to start all over, you always had trust and faith in me and for that I cannot thank you both enough.

I would also like to thank Francesca Solmi without whom this thesis would not be possible. Your determination and relentlessness at securing this data for me has not gone unnoticed. From completing forms to chasing up on my behalf and even responding to my emails even when you were away, I cannot express my gratitude enough that you were willing to work so hard for a project that is not one of your own.

Thank you to Evie and Emily, my sounding boards and confidants, who I relied on heavily throughout this process. Training would not have been the same without you both. Sharing all our vulnerabilities and triumphs you have kept me going throughout these three years and I would not have been able to complete this course without you both. This was never going to be an easy journey, so I am incredibly grateful to have gone through this process with you both by my side.

Finally, to my family and friends who have been patient and accepting of my absence whilst I dedicated these last few years to reaching this goal I have worked so hard to achieve. I cannot wait to resume some form of normality and make up for every celebration and life moment missed.

Part 1: Literature Review

A systematic review of the association between traits of autism and ADHD with disordered eating from a longitudinal perspective.

Abstract

Aims

Cross-sectional studies have provided evidence for an association between autism, ADHD and eating disorders, however, there continues to be uncertainty around the extent to which neurodevelopmental conditions predict future disordered eating. The present review aimed to synthesise research on the longitudinal associations between traits of autism, ADHD, and future disordered eating. The review also aimed to explore possible causal mechanisms that may underpin these associations.

Method

A systematic review was conducted in line with PRISMA guidelines with. A systematic search of Embase, MEDLINE, PsychInfo (all via OvidSP) and Web of Science was conducted. Peer-reviewed longitudinal studies that measured traits of and/or formal diagnoses of autism or ADHD prior to measuring disordered eating behaviours met criteria for the review. A quality assessment of included studies was conducted using the Newcastle-Ottawa Quality Assessment scale for Cohort Studies.

Results

A total of 21 papers met eligibility criteria to be included in the review. Of these, 13 investigated ADHD, six on autism traits and two on both autism and ADHD. Five of these studies also explored causal factors that could explain the relationship between autism or ADHD and subsequent disordered eating. A range of longitudinal associations were identified across both autism and ADHD and the spectrum of disordered eating. Possible causal mechanisms were also identified including fussy eating, mood and anxiety disorders and impulsivity.

Discussion

This review provides evidence for longitudinal associations between autism and ADHD, and disordered eating persist; autism and ADHD are risk factors for future eating disorders and/or disordered eating behaviours. This association has been shown across ages, genders, and types of disordered eating. Several possible causal factors have also been identified to help understand the mechanisms driving these associations. The review recommends future longitudinal research into co-occurring autism and ADHD and newer eating disorder diagnoses such as ARFID. These findings highlight the need for routine screening of neurodevelopmental conditions in ED populations, as well as the development of appropriate treatment approaches and clinical guidance for those with eating disorders and co-occurring autism and/or ADHD.

Introduction

Eating disorders (EDs) are a group of conditions characterised by persistent and abnormal patterns of eating, dieting and compensatory behaviour such as purging (American Psychiatric Association [APA], 2013). It is widely recognised that EDs pose a risk to individuals social functioning, as well as being associated with significant physical consequences and a high mortality risk (Jenkins et al., 2011). Concerningly, these risks and impact on individuals physical and psychological wellbeing have also been documented in individuals with disordered eating (Pennesi & Wade, 2016) which here refers to the full spectrum of non-normative eating patterns including ED diagnoses and subthreshold behaviours.

The aetiological basis of EDs is also considered complex with both genetic and environmental factors likely to contribute to their onset and development (Culbert et al., 2015). There has also been shown considerable rates of comorbidity across a range of psychiatric and medical disorders (Hambleton et al., 2022). Of particular interest is the associations with neurodevelopmental conditions given their overlapping features (Nickel et al., 2019) and the potential for unfavourable outcomes in treatment (Nimbley et al., 2024).

Despite this, there continue to be difficulties in diagnosing EDs and providing individuals with the specialist services required (Demmler et al., 2020). This has, in part, been attributed to limits in understanding the epidemiological basis of EDs, particularly in males (Mitchison & Mond, 2015). It has been suggested that several challenges contribute to this lack of understanding including the historic use of the residual category “Eating Disorder Not Otherwise Specified” (EDNOS) which has yielded little information on behavioural and cognitive profiles due to its heterogeneity (Mitchison & Mond, 2015). It has been posited the low rates of individuals meeting criteria for a formal diagnosis of an ED and non-response

bias (Mitchison & Mond, 2015) has also contributed to the uncertainty within ED epidemiology research (Mitchison & Mond, 2015). Importantly, it has also been shown that individuals from ethnic minorities are often overlooked within ED studies (Keski-Rahkonen & Mustelin, 2016). EDs have also been shown to be associated with high psychiatric comorbidity further complicating the assessment and treatment of these conditions (Christensen et al., 2019).

In the latest edition of the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM–5; APA, 2013) the category of feeding and eating disorders has been expanded to increase clinical utility (Wilfley et al. 2007). Anorexia nervosa (AN) is described as a disturbance in self-perception of body shape and an intense fear of gaining weight resulting in a persistent restriction of energy intake with or without purging (APA, 2013). Prevalence rates of AN within Europe have been calculated to be between 1-4% of women (Keski-Rahkonen & Mustelin, 2016) but only 0.3% for males (Van Eeden et al., 2021). The second commonly described ED is that of Bulimia nervosa (BN) which is defined by recurrent episodes of binge eating along with compensatory behaviours such as purging or inappropriate laxative use (APA, 2013). The prevalence rates of BN amongst females are around 3% and 1% for males (Van Eeden et al., 2021). Binge eating disorder (BED) on the other hand is frequent episodes of binge eating associated with negative psychological sequalae such as feelings of guilt but does not include such compensatory behaviour (APA, 2013). Similarly, the lifetime prevalence rates of BED amongst females are between 1-4% (Keski-Rahkonen & Mustelin, 2016) and between 0.4-2% in males (Hudson et al., 2007; Udo & Grilo, 2018)

A new diagnosis included within the DSM-5 is that of Avoidant/Restrictive Food Intake Disorder (ARFID) which is characterised by the restriction or avoidance of food to the extent that there is a significant impact on an individual's weight, nutritional intake, or

psychosocial functioning (APA, 2013). However, an important clinical distinction between ARFID and other EDs is that it is not primarily associated with disturbances of body image (Sadler et al., 2024). Further, to meet the diagnostic criteria the restrictive behaviour cannot be explained by certain cultural or religious practices or be due to a lack of food availability (Sadler et al., 2024). The literature on ARFID is still emerging, however studies are showing prevalence rates ranging between 0.3-15.5% in non-clinical populations and up to 32-64% within specialist feeding clinics (Sanchez-Cerezo et al., 2023). Any disordered eating associated with significant distress that does not fit the criteria for a specific ED may meet the threshold for the additional diagnosis of "Other Specified Feeding or Eating Disorder" (OSFED; APA, 2013). Research has shown the lifetime prevalence rates of OSFED to be up to 2.4% across males and females (Lindvall Dahlgren et al., 2017). It has been suggested that these new distinctions across ED diagnoses should help increase the likelihood of individuals meeting the threshold for a formal diagnosis, separate those with eating disturbances from those with an ED and, crucially, better capture the presentation of ED in males (Lindvall Dahlgren et al., 2017).

Autism and disordered eating

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterised by differences in social communication, cognitive thinking styles, and restrictive, repetitive patterns of behaviour (Nickel et al., 2019). Whilst historically considered to have distinct categorical areas of difficulty with clear diagnostic thresholds, current perspectives favour a dimensional view that emphasises how autistic traits are experienced to varying degrees across a broad spectrum of behaviours and levels of functioning (Szatmari et al., 2022).

In line with language preferences from the autism community (Kenny et al., 2016) and by autism researchers (Monk et al., 2022) 'autism' will be used descriptively throughout

this review in replacement of the diagnostic label of 'autism spectrum disorder'. This is based on the view that research within the field should prioritise the perspective of autistic people and that the terminology of ASD can have negative societal consequences and impact the way on which autistic people view themselves (Monk et al., 2022).

Whilst historically thought to have a high male preponderance, a greater understanding of autism has highlighted a likely underdiagnosis amongst females, especially in those of high intellect (Happé & Frith, 2020). Further, it has been suggested that diagnostic overshadowing may contribute to an under recognition of autism in females especially in those with EDs (Happé & Frith, 2020).

The association between autism and EDs was first described by Gillberg in the 1980's through a serious of landmark studies known as the Gothenburg studies, where the authors found a significant association between autism and AN (Gillberg & Råstam, 1992; Nilsson et al., 1999; Råstam, 1992). A substantial amount of research has since replicated these findings (Kerr-Gaffney et al., 2020; Leppanen et al., 2022; Rhind et al., 2014; Westwood et al., 2016) with prevalence rates of co-occurring autism and AN estimated to be 29% (Inal-Kaleli et al., 2025). Consequently, research has attempted to understand the reasons behind these associations with some initial suggestions that the effects of starvation, often seen within enduring restrictive EDs such as AN, can present similarly to the characteristics of autism (Hiller & Pellicano, 2013). Oldershaw et al. (2010) found that weight restoration in AN reduced some traits associated with autism such as difficulties with theory of mind. It has therefore been suggested that autism features in those with AN may be due a combination of trait and state with autism traits being exacerbated by the physiological impacts of being underweight and starvation (Nyuttens et al., 2024).

Whilst there has been a research focus on the association between autism and AN, autism has also been shown to be associated with other disordered eating behaviours. In their pilot study, Gesi et al. (2017) found patients with BN and BED showed greater autistic traits compared to healthy controls. Similarly, another Italian study by Dell'Osso et al. (2018) found elevated autistic traits in those with BN and BED compared to control. As those with BN or BED are typically, yet not exclusively, either of normal weight or overweight (Bulik et al., 2012), these findings suggest that the autistic traits shown in those with EDs are not purely due to starvation effects. However, this association has not been replicated as Iwasaki et al. (2013) did not find a significant association between autism and BN, thus it is not clear the extent to which these associations exist.

The more recently defined diagnosis of ARFID has also garnered attention with its possible associations with autism. Recent studies have shown elevated rates of ARFID in individuals with autism (Koomar et al., 2021; Nygren et al., 2021) and, reciprocally, high rates of autistic traits have been demonstrated in ARFID populations (Inoue et al., 2021; Sanchez-Cerezo et al., 2023). It has been proposed that the patterns of feeding and eating seen in ARFID and autism share common features such as food selectivity and a lack of diversity across food groups along with sensory sensitivities (Christol et al., 2018; Nimbley et al., 2022; Watts et al., 2023).

ADHD and disordered eating

Another area of consideration is that of Attention-deficit/hyperactivity disorder (ADHD) which is another common neurodevelopmental condition characterised by inattention, impulsivity, and hyperactivity (APA, 2013). Like autism, ADHD is considered from a dimensional approach with heterogeneity in symptomatology and impairment (Heidbreder, 2015). As highlighted by a recent scoping review by Makin et al. (2025) there is

currently a lack of literature on language preferences for those with ADHD compared to autistic individuals. Thus, this review will use the language of the studies cited, however it is acknowledged that this may not be optimal and further research into language preferences is warranted.

Prevalence rates of ADHD amongst children have shown to be as high as 15.5% (Rowland et al., 2015) with a 4:1 male-to-female ratio (Ayano et al., 2020). However, it has been identified that this gender discrepancy may be due to missed or misdiagnosis of ADHD in females (Young et al., 2020). Females with ADHD are also, on average, diagnosed later than males (Martin, 2024). This in part may be explained by the suggestion that the female profile of ADHD may be somewhat different to that of males, with typically more inattentive than hyperactive features, resulting in a lack of recognition and consequently reduced referral rates for females (Young et al., 2020).

Similarly to autism, there is an increasing evidence-base highlighting the co-occurrence between ADHD and EDs. Recent studies have shown that up to 20% of individuals with ADHD may present with a co-occurring ED (Ravi & Khan, 2020). In a review by Villa and colleagues (2023) most studies included showed an association between ADHD and EDs, however the cross-sectional nature of these studies means we cannot infer causation. Longitudinal studies are therefore imperative to gain a further understanding of the nature of these associations and whether neurodevelopmental conditions such as ADHD pose a risk to future disordered eating.

It has been posited that the core traits of ADHD may contribute to ED traits such as impulsivity increasing the likelihood of binge eating and inattention contributing to a lack of awareness of hunger or satiety signals (Cortese et al., 2007; Ptacek et al., 2016). As such, ADHD has most commonly been shown to be associated with BN and BED (Ptacek et al.,

2016; Ziobrowski et al., 2018). This finding has been found across cultures with Appolinario et al. (2022) finding those with ADHD had significantly greater rates of BN and BED compared to those without ADHD. Further, the association between binge eating has also been found across gender with a significantly higher prevalence of ADHD in both males and females with BED compared to those without BED (Brewerton & Duncan, 2016).

Some studies have suggested there may also be an association with AN (Fernández-Aranda et al., 2013). Although, it has been shown that the association with ADHD may be more common in binge-eating/purging type AN than restricting type AN (Svedlund et al., 2017). Interestingly, one study exploring the association between ADHD and traits of AN and BN (Bleck et al., 2015) found that those with clinical ADHD were more likely to experience both clinical level bingeing and restrictive behaviours seen suggesting a risk for both BN and AN. The authors did however find that those with subclinical ADHD were more likely to experience bingeing and purging but not restrictive behaviours, suggesting an impact of trait severity on the risk for disordered eating (Bleck et al., 2015). This highlights the complexity of the relationship between ADHD and EDs and the importance of trait or symptom level research.

Co-occurring Autism and ADHD

According to the literature, up to 40% of individuals with autism have co-occurring ADHD (Rong et al., 2021) further complicating the understanding of the association between autism, ADHD, and disordered eating. Thus, it is difficult to ascertain from the existing literature what is associated with autism or ADHD when studies do not adjust for this co-occurrence which has implications for clinical provision. One review by Nickel et al. (2019) aimed to explore the overlap between EDs, autism and ADHD. From the included studies, the authors found that on average 4.7% of individuals with an ED received an autism diagnosis

with this being most common in AN (Nickel et al., 2019). They found that rates of ADHD in individuals with an ED ranged from 1.6-18% with ADHD being least common in the AN restrictive subtype and more common in BN and binge eating/purging subtypes of AN (Nickel et al., 2019). Whilst useful to help understand the patterns and profiles of disordered eating in those with autism or ADHD, the extent to which co-occurring autism and ADHD impacts the associations with EDs remains unclear.

Rationale

Despite the considerable evidence showing associations between neurodivergence and eating disorders, research has often focussed on specific disorders such as the link between autism and AN. Given the high co-morbidity between autism and ADHD (Hours et al., 2022) it is important to understand how this would impact on the risk for and presentation of disordered eating in these individuals. Further, the recent increase in late diagnosis of ADHD for females has highlighted the lack of recognition in how these disorders can present differently across genders (Young et al.,2020). To gain a true understanding of these associations it is therefore imperative to view this from a longitudinal perspective to account for the possibility of missed diagnosis during childhood given females are typically diagnosed later than males (Martin, 2024). To account for the challenges in diagnosis and it may also be beneficial to view this from the level of traits or symptoms. As most existing literature comes from cross-sectional studies this provides limited understanding about directionality which is important for building evidence for causality. A longitudinal approach can also help in understanding real associations, as opposed to being due to artefact or diagnostic overshadowing in EDs (Adams et al., 2024), by exploring traits of autism and ADHD prior to onset of disordered eating. Through gaining a more comprehensive understanding of these associations this may support earlier recognition and diagnosis and thus early or preventative intervention. Despite the increasing evidence based depicting

associations between EDs and neurodevelopmental conditions no clear guidance or recommendations have been developed for this population (Tchanturia et al., 2020). Crucially, a greater understanding of the associations between autism, ADHD and disordered eating will therefore help to support the development of more appropriate treatment pathways and interventions that are neurodiversity affirming.

Aims

This systematic review therefore aimed to answer the following research questions:

1. Is there an association between traits/diagnoses of autism and disordered eating?
2. Is there an association between traits/diagnoses of ADHD and disordered eating?
3. Is there an association between co-occurring autism and ADHD and disordered eating?
4. Are there mediating/moderating factors that could explain the relationships between autism and/or ADHD symptoms and disordered eating?

To my knowledge, this review is the first to consider these questions by looking exclusively at longitudinal studies that incorporates both formal diagnosis and traits or symptoms of autism, ADHD and EDs.

Method

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Page et al., 2021) guidance was used for conducting and presenting the systematic review. The protocol for this systematic review was registered on PROSPERO (registration number: CRD42025613020).

Search strategy

The search was conducted in Ovid MEDLINE®, Embase (via Ovid), APA PsychInfo (Via Ovid) and Web of Science on the 4th of January 2025. No filters or limits were applied at this stage during the search. The full search strategy is presented in table 1. A manual search of Google Scholar and citation chaining of relevant reviews was also conducted to check for additional eligible studies.

Table 1

Search strategy

anorexia OR "anorexia nervosa" OR bulimia OR "bulimia nervosa" OR "eating disorder" OR "disordered eating" OR "feeding and eating disorders" OR "binge eating" OR EDNOS OR "eating disorder not otherwise specified" OR OSFED OR "other specified feeding or eating disorder" OR ARFID OR "avoidant restrictive food intake disorder" OR restrictive eating OR selective eating OR feeding disorder		
AND		
Autism OR Autistic OR ASD OR "Autism Spectrum Disorder" OR "Autistic Disorder" OR Asperger OR "Asperger Syndrome"	OR	"Attention deficit" OR "Attention Deficit with Hyperactivity" OR "attention deficit hyperactivity disorder" OR ADHD
AND		
Longitudinal OR prospective OR retrospective OR cohort		

Eligibility criteria

The following inclusion criteria were used to identify eligible studies: 1) longitudinal studies presenting epidemiological data or causal mechanisms in the association between

autism, ADHD and disordered eating with measurements of autism and ADHD measured prior to disordered eating; 2) studies on children, young people or adults with a formal diagnosis or traits of autism or ADHD and disordered eating; 3) the use of validated instruments and screening tools to confirm diagnosis or identify traits of autism, ADHD and disordered eating or confirmed diagnoses from diagnostic manual codes on patient registers; 4) published in English in a peer-reviewed publication.

The exclusion criteria were: 1) cross-sectional studies; 2) case reports; 3) systematic reviews or meta-analyses; 4) conference abstracts or book chapters 5) studies on obesity, pica, rumination syndrome or dietary behaviour; 5) studies on singular aspects or specific traits of autism, ADHD and disordered eating; 6) lack of clarity if ADHD or autism diagnosis/trait measurement preceding ED diagnosis/trait measurement; 7) retrospective self-report on autism or ADHD traits in childhood

Despite pica and rumination disorder both falling under the category of feeding and eating disorders in the DSM-5, these conditions were not included in this current review due to the complexity surrounding the aetiology and range of possible differential diagnoses (Elkins et al., 2024). Obesity studies were also not included in this review as this is not considered to be a mental health condition and has a range of genetic, physiological, and environmental factors that can contribute to an individual having excess body fat (APA, 2013). Similarly, other dietary behaviours were not included in the review based on the rationale that these could be considered normative differences.

Study selection

The articles identified through the searches were exported into EndNote Reference Management software (Clarivate, 2013) to perform manual de-duplication. The remaining articles were then exported into Rayyan (Ouzzani et al., 2016) for screening against inclusion

and exclusion criteria. Title and abstracts of all identified articles were screened excluding studies that did not meet the eligibility criteria. The full texts of the remaining articles were then screened to identify the studies to be included in the review. Two reviewers (S.A and O.L) independently screened all articles and were blind to each other's decisions. Any disagreements were resolved by further examination and discussion with two additional reviewers who were the project supervisors (W.M and V.C.L).

Quality assessment

All studies included in the review were assessed for their quality using the Newcastle-Ottawa Quality Assessment Scale (NOS) for cohort studies (Wells et al., 2000) (see appendix 1). The NOS is a commonly used tool for assessing longitudinal studies (Ma et al., 2020) and has shown good content validity and inter-rater reliability (Wells et al., 2000). The NOS produces a rating of quality based on the selection of the study groups, comparability of the groups and ascertainment of the outcome of interest. For the current review, the NOS was adapted to account for studies without control groups. When considering the selection of non-exposed cohort, this was adapted to consider studies using secondary data where for example participants with higher and lower scores on measures of autistic traits were included in the analysis rather than compared against each other. Thus, studies will receive a star for this item if their total sample was representative. Similarly, studies would also receive a star for comparability of the exposed and non-exposed cohort if the studies included appropriate confounders in their analysis, compared to samples being matched on these factors in studies with a control group. An adequate follow-up length was agreed to be of one year, but an additional requirement was introduced here that the follow-up length needed to be considered in terms of covering a developmentally appropriate time frame. Thus, a study of five years from birth to age five would not be deemed developmentally appropriate for the onset of

disordered eating, whereas a study of 12 months covering adolescent would receive a star. These amendments were discussed and agreed with W.M and V.C.L.

A maximum score of nine can be given to a study across eight items (two stars for comparability) with a higher number of stars representing greater quality. Quality assessment was independently performed on all included studies by reviewers S.A and E.G with discrepancies discussed with W.M and V.C.L to reach a consensus.

Data extraction and synthesis

Data was extracted into an Excel document by one author (S.A) with dilemmas discussed with the two research supervisors (W.M & V.C.L). Extracted data included author names, year of publication, country, and study design (follow-up duration, number of waves and comparator groups where applicable). Data on sample characteristics (size, age, and gender) and predictor, outcome, and mediator variables (diagnoses or traits measures, time point or age at measurement and measurement tools utilised) were also identified. Finally, relevant results and findings were extracted to allow for examination of the longitudinal associations between autism or ADHD and disordered eating.

Due to the heterogeneity in the sample compositions, measures used, and time periods observed it was concluded that a meta-analysis would not be appropriate, and a narrative synthesis has therefore been conducted. Data from the included studies has been synthesised according to the original aims of the review.

Results

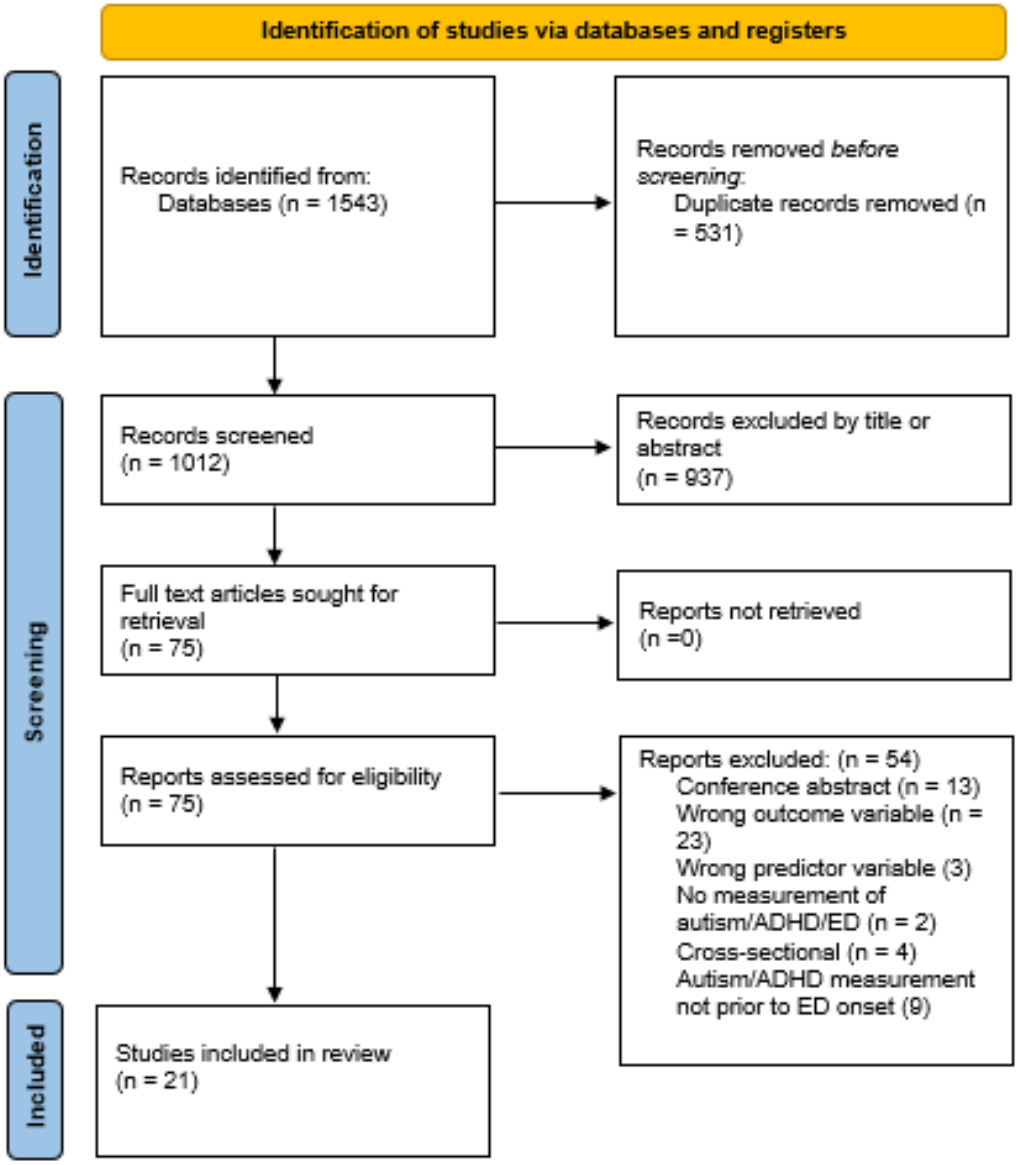
Study selection

Figure 1. illustrates the process of study selection following PRISMA guidelines. No additional studies were identified through citation searches using Google scholar and thus have not been included in the PRISMA flowchart. The search initially yielded 1532 records,

of which 1012 remained after removal of duplicates within EndNote Reference Manager (Clarivate, 2013). A total of 937 records were excluded after screening by title and abstracts resulting in 75 full-text articles to be considered for inclusion. During full-text screening 54 studies were excluded. At the end of the selection process a total of 21 studies met eligibility criteria and were therefore included in the review.

Figure 1

PRISMA 2020 flow diagram of the systematic search (Page et al., 2021)



Study characteristics

The 21 studies included in this review were published between 2006 to 2024 and were conducted in five countries with one additional study reporting on a multi-site study across Europe (see Table 2 for study characteristics). The length of follow-up ranged from six months to 11 years, with two population cohort studies varying in follow-up based on entry to the cohort. As detailed in Table 2. there was a large variation between sample sizes, gender ratios, and age ranges. See Table 3 for results from the included studies and Table 4 for results from additional mediation analyses.

Of the included studies, six investigated the association between autistic traits/diagnoses and future disordered eating with 13 studies exploring traits/diagnoses of ADHD and future disordered eating. Within the subset of studies exploring traits of ADHD, three research groups produced two papers based on different time points during follow-up (Biederman et al., 2007; Biederman et al., 2010; Hinshaw et al., 2006; Hinshaw et al., 2012; Mikami et al., 2008; Mikami et al., 2010). Two studies investigated both traits of autism and ADHD in relation to future disordered eating. Out of these 21 studies, five also reported on possible mediating factors in the association between autism, ADHD and disordered eating across fussy eating, mood disorders, interoception, overeating and strong desire for food.

Across the studies there was a large range of standardised measurement tools used to assess traits or symptoms of autism, ADHD or EDs (see Tables 3 and 4). These included both parent- and self-report measures along with clinician rated questionnaires and structured clinical interview. In those studies that used diagnoses from patient registers, diagnoses had been confirmed against codes in editions of the DSM or Internation Classification of Diseases (ICD).

Table 2

Characteristics of included studies

Study	Country (Cohort)	Study Design	Follow-up length	Sample Size (*N*)	Gender ratio
EDs in Autism					
Carter Leno et al. (2022)	UK (ALSPAC)	Population-based cohort	7 years	8982	Data not available
Dinkler et al. (2021)	Sweden	Prospective	9 years	5987	52.4% Female
Koch et al. (2015)	Denmark	Population-based cohort	From date of birth until the first diagnosis with the disorder of interest, death, emigration from Denmark or 31 December 2012, whichever came first	ASD = 12606 AN = 5006	ASD = 20.6% Female AN = 92.9% Female
Martini et al. (2022)	Sweden	Population-based cohort	9 years	20814	34.2% Female
Schaumberg et al. (2021)	UK (ALSPAC)	Population-based prospective	10 years	4864	Data not available

Solmi et al. (2021)	UK (ALSPAC)	Population-based cohort	9 years	5381	55.2% Female
EDs in ADHD					
Biederman et al. (2007)	USA	Prospective	5 years	Baseline: ADHD = 140, Controls = 122 Follow-up: ADHD = 123, Controls = 106	100% Female
Biederman et al. (2010)	USA	Prospective	11 years	ADHD = 96 Controls = 91	100% Female
Bisset et al. (2019)	Australia (LSAC)	Population-based cohort	2 years	ADHD = 186 Controls = 2486	ADHD = 20.6% Female Controls = 52.3% Female
Bufferd et al. (2022)	USA	Prospective longitudinal	12 years	609	45.5% Female
Hinshaw et al. (2006)	USA	Prospective	5 years	ADHD = 140 Controls = 88	100% Female
Hinshaw et al. (2012)	USA	Prospective	10 years	ADHD group = 140 Control group = 88	100% Female

Martin et al. (2023)	UK	Community-based sample	6 months	345	72.5% Female
Mikami et al. (2008)	USA	prospective	5 years	Baseline: ADHD-C = 93, ADHD-I = 47, Controls = 88 Follow-up: ADHD-C = 86. ADHD-I = 41. Controls = 82	100% Female
Mikami et al. (2010)	USA	prospective	8 years	ADHD = 432 Control group = 264	ADHD = 22.0% Female Control = 20% Female
Robinson et al. (2020)	Europe (IMAGEN)	longitudinal cohort	5 years	1623	51.1% Female
Sonneville et al. (2015)	UK (ALSPAC)	Longitudinal Cohort	9 years	7884	48.8% Females
Viborg et al. (2014)	Sweden	Prospective	1 year	Baseline = 494 Follow-up = 445	100% Female
Yilmaz et al. (2017)	Sweden (TCHAD)	Longitudinal cohort	9 years	2315	50.6% Female
EDs in Autism & ADHD					

Christiansen et al. (2024)	Denmark	population-based cohort	from sixth birthday until death, emigration, ED diagnosis, or December 31, 2016, whichever came first.	1671823	48.7% Female
Lin et al. (2024)	50 US states and 4 countries	retrospective	1 year	ADHD = 2503 ASD group = 1208 ED group = 14,254	ED group = 79.2% Female

ALSPAC, Avon Longitudinal Study of Parents and Children; ADHD, Attention/Deficit Hyperactivity Disorder; ADHD-C, Attention/Deficit Hyperactivity Disorder – Combined type; ADHD-I, Attention/Deficit Hyperactivity Disorder-Inattentive type; AN, Anorexia Nervosa; ASD, Autism Spectrum Disorder; IMAGEN, IMAGEN Consortium; LSAC, Longitudinal Study of Australian Children; TCHAD, The Swedish Twin Study of Child and Adolescent Development.

Table 3

Main findings from the included studies

Study	Predictor Variable			Outcome variable			Results
	Diagnosis (manual) / trait	**Age of trait measurement (years/range)**	**Measurement tools / diagnosis source**	**Diagnosis (Manual) / trait**	**Age of trait measurement (years/range)**	**Measurement tools / diagnosis source**	
EDs in Autism							
Carter Leno et al. (2022)	Autistic social traits	T1 = 7 T2 = 11 T3 = 14	SCDC	Binge eating, Fasting, Purging	14	Self-report	Autistic traits at age 7 were associated with ED behaviours at age 14 (*b*=0.452, 95% CI [0.225-0.679], $p < 0.001$).
Dinkler et al. (2021)	Autistic traits (DSM-IV/DSM-5; ICD-9/ICD-10)	9 + 18	A-TAC ASD module; National Patient Register	AN (ICD-10)	18	National Patient Register	No significant association was found for elevated autistic traits at age 9 with later diagnosed AN

Koch et al. (2015)	ASD (infantile autism, atypical autism or Asperger syndrome; ICD-8/ICD-10)	Age of diagnosis	National Patient Register/ Psychiatric Central Research Register	AN/atypical AN (ICD-8/ICD-10)	Age of diagnosis	National Patient Register/ Psychiatric Central Research Register	Individuals with a diagnosis of ASD had an elevated risk of receiving a later diagnosis of AN (HR = 5.39, 95% CI [4.37, 6.64], $p < .05$).
Martini et al. (2022)	Autism (ICD-9/ICD-10)	Age of diagnosis	National Patient Register	AN (ICD-9/ICD-10), BN (ICD-10)	Age of diagnosis	National Patient Register	Males with ASD had an elevated risk of receiving a diagnosis of AN during follow-up (HR=3.44, 95% CI [1.61-7.32], $p = .001$). Females with ASD had an elevated risk of receiving a diagnosis of AN during follow-up (HR=2.04, 95% CI [1.47, 2.83], $p < .001$). No significant elevated risk for BN was found.
Schaumberg et al. (2021)	Autism -like social communication difficulties	8 +14	SCDC	ED behaviours/AN/BN/BED/PD (DSM-5)	14,16,18	Self-report ED behaviours, DEBQ, IBSS-R, ED diagnosis	Significant associations were found between autistic traits at age 14 and eating pathology at age 16 in females (binge eating: OR = 1.95, 95% CI [1.06, 3.57], $p = .03$; BN: OR = 2.31, 95% CI [1.08, 4.94], $p = .03$). Significant associations were found between autistic traits at age 10 and eating pathology at age 16 in males (binge eating: OR = 3.71, 95% CI [1.50, 9.17], $p = .004$; purging: OR = 4.72, 95%

							CI [1.16, 19.10], p = .029; BN: OR = 6.55, 95% CI [2.02, 21.25], p = .002).
Solmi et al. (2021)	ASD	T1 = 7 T2 = 11 T3 = 14 T4 = 16	SCDC	Disordered eating (DSM-5)	14	Self-report ED behaviours	Adolescents who showed disordered eating behaviours at age 14 had greater overall childhood autistic traits compared to those who did not (RR: 1.23, 95% CI [1.14, 1.32], p < .001). Higher SCDC scores at age 7 were associated with greater odds of reporting any disordered eating at age 14 (OR = 1.18, 95% CI [1.06, 1.32], p = .004).
EDs in ADHD							
Biederman et al. (2007)	ADHD (DSM-III-R/DSM-IV)	6 - 18	Medical records	AN/BN (DSM-III-R/DSM-IV)	10 - 25	K-SADS-E, SCID	ADHD females were 3.6 times more likely to meet criteria for an ED during the follow-up period compared to controls (HR = 3.6, 95% CI [1.4, 9.9], p < .01). ADHD females were 5.6 times more likely to meet criteria for BN compared to controls (HR = 5.6, 95% CI [1.6, 19.0], p < .01).

Biederman et al. (2010)	ADHD (DSM-III-R)	8 - 14	Medical records	AN/BN (DSM-IV)	15 - 30	K-SADS-E, SCID	At follow-up ADHD females 3.5 times more likely to meet criteria for any ED (HR = 3.5, 95% CI [1.6, 7.3], p = .001; AN: HR = 2.2, 95% CI [0.8, 5.7], p = .11; BN: HR = 5.2, 95% CI [2.0, 13.7], p = .001)
Bisset et al. (2019)	ADHD (DSM-5)	12 - 13	SDQ	AN/BN/BED (DSM-5)	14 - 15	BET	Males with ADHD were more likely to experience regular binge eating than controls (OR = 9.4, 95% CI [1.7, 52.8], p = .01)
Bufferd et al. (2022)	ADHD	3 + 6	PAPA	AN symptoms and BN symptoms (DSM-IV), body dissatisfaction	12 + 15	MEBS, K-SADS-PL	No significant associations were found between ADHD at age 3 and AN or BN symptoms at age 15. Greater impulsivity at age 6 was associated with body dissatisfaction at age 12 (p = .05)
Hinshaw et al. (2006)	ADHD (DSM-IV)	6 - 12	SNAP, CBCL, DISC-IV	Eating disorder symptoms	11 - 18	EDI-II, EAT-26	Females with childhood ADHD-combined type scored significantly higher across measures of eating disorder symptomatology at follow-up than controls with effect sizes ranging between .42-.48
Hinshaw et al. (2012)	ADHD (DSM-IV)	6 - 12	SNAP, CBCL, DISC-IV	Eating disorder symptoms	17 - 24	EDI-II, EAT-26	No significant association was found between baseline ADHD future disordered eating

Martin et al. (2023)	ADHD	18 - 60	CAARS:SV	Binge eating, restrictive eating	18 - 60	EAT-26, BES, SCOFF, DEBQ	There was a significant increase in restrictive eating at follow-up ($t(344) = 3.7$, $p < .00$). Inattentive symptoms predicted binge eating and restrictive eating directly (Effect = 0.85, 95% CI [0.60, 1.09], $p < .001$; Effect = 0.30, 95% CI [0.15, 0.55] $p = .006$). Hyperactive/Impulsive symptoms predicted binge eating and restrictive eating directly (Effect = 0.33,95% CI [0.015, 0.64] $p = .04$; Effect = 0.29,95% CI [0.052, 0.53], $p = 0.017$)
Mikami et al. (2008)	ADHD (DSM-IV)	6 - 12	CBCL, TRF, SNAP-IV, DSM-IV, DISC-IV	Eating pathology	11 - 18	EDI-II, EAT, DISC-IV	Childhood ADHD was significantly related to eating pathology ($F(4, 372) = 2.78$, $p < .05$)
Mikami et al. (2010)	ADHD (DSM-IV)	7.0 - 9.9	SNAP-IV, DISC-III	BN	15 - 18	EDI-II, DISC-IV	The association between impulsivity at baseline and parent-report BN symptoms at follow-up was stronger for females than males which was a statistically significant difference (z = 3.79; $p < .001$). Impulsivity at baseline was also associated with self-reported BN symptoms with a stronger association for females than males which was a statistically significant difference (z = 2.49; $p = .01$).

Robinson et al. (2020)	ADHD	14.5	DAWBA, SDQ	Binge eating, purging, dieting	T1 = 16.5 T2 = 19.4	DAWBA section P (Eating Behaviours)	ADHD was associated with purging (OR = 4.03, 95% CI [1.67, 9.68], $p < .05$).
Sonneville et al. (2015)	ADHD	8.1 + 11.6	SDQ	Binge eating	14 + 16	Self-report	No direct effect of ADHD and binge eating during adolescence was found
Viborg et al. (2014)	Hyperactivity-Inattention	School grades 7 - 8	SDQ	Disordered eating	School grades 8 - 9	RiBED-8, BEAA	Hyperactivity-inattention predicted disordered eating at follow-up ($B = .29$, $p < .01$)
Yilmaz et al. (2017)	ADHD (DSM-IV)	8 - 9	Checklist of 14 DSM-IV based item	Disordered eating (bulimia, drive for thinness. body dissatisfaction)	T2 = 13 - 14 T3 = 16 - 17	EDI-II	A combination of higher inattention symptoms and higher hyperactivity/impulsivity during childhood was significantly associated with symptoms of disordered eating during late adolescence; bulimia (.73 [.12]; $p < .01$), drive for thinness (-.14 [.31]; $p < .01$), body dissatisfaction (.43 [.50]; $p < .01$).
EDs in Autism & ADHD							
Christiansen et al. (2024)	ADHD (ICD-10), ASD (ICD-10)	Age of diagnosis	National Patient Register/ Psychiatric Central	AN, BN, EDNOS (ICD-10)	Age of diagnosis	National Patient Register/ Psychiatric Central	There was an increased risk for all EDs in individuals with an initial diagnosis of ASD or ADHD. When adjusting for co-occurring ADHD and ASD in individuals this only slightly attenuated the associations with a later diagnosis of ED.

			Research Register			Research Register	
Lin et al. (2024)	ASD, ADHD	1 year prior to ED diagnosis	Electronic health records	ED/BN/AN/ARFID BED/EDNOS (ICD-10)	Age of diagnosis	Electronic health records	ADHD was more common in those with an ED diagnosis one year later compared to those without an ED diagnosis (17.2% vs 8.4). ASD was more common in those with an ED diagnosis 1 one year later compared to those without an ED diagnosis (8.3% vs 2.3%)

ADHD, Attention-Deficit/Hyperactivity Disorder; AN Anorexia Nervosa; ARFID Avoidant restrictive food intake disorder, ASD, Autism Spectrum Disorder; A-TAC, The Autism-Tics, ADHD and other comorbidities Inventory; BEAA, Body-Esteem Scale for Adolescents and Adults; BED, Binge eating disorder; BET, Branched Eating Disorders Test; BES, The Binge Eating Scale; BN, Bulimia Nervosa; CAARS:SV, The Conners' Adult ADHD Rating Scale: Short Version; CBCL, Child Behavior Checklist; CI, Confidence Interval; DAWBA, The Development and Well-being Assessment; DEBQ, The Dutch Eating Behaviour Questionnaire; DISC-III, Diagnostic Interview Schedule for Children – 3rd ed.; DISC-IV, Diagnostic Interview Schedule for Children – 4th ed.; DSM-III-R, Diagnostic and Statistical Manual of Mental Disorders (3rd ed.) – Revised; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders (4th ed.); DSM-5, Diagnostic and Statistical Manual of Mental Disorders (5th ed.); EAT, Eating Attitudes Test; EAT-26, The eating attitudes Test – 26 Items; ED, Eating Disorder; EDI-II, Eating Disorders Inventory – 3rd ed.; EDNOS, Eating Disorder Not Otherwise Specified; HR, Hazard ratio; ICD-8, International Classification of Diseases – 8th revision; ICD-9, International Classification of Diseases – 9th revision; ICD-10 - International Classification of Diseases 10th revision; IBSS-R, Ideal Body Stereotype Scale-Revised; K-SADS-E, The Kiddie Schedule for Affective Disorders and Schizophrenia – Epidemiological version; K-SADS-PL, The Kiddie Schedule for Affective Disorders and Schizophrenia Present and Lifetime Version; MEBS, Minnesota Eating Behaviour Survey; OR, Odds ratio; PAPA, The Preschool Age Psychiatric Assessment; PD, Purging Disorder; RiBED-8, Risk Behaviour related to Eating Disorders; RR, relative risk; SCID, The Structured Clinical Interview for DSM Disorders; SCDC, Social Communication Disorders Checklist; SCOFF, Sick, Control, One Stone, Fat, Food Questionnaire; SDQ, The Strengths and Difficulties Questionnaire; SNAP, Swanson, Nolan Pelham Rating Scale ; SNAP-IV, Swanson, Nolan Pelham Rating Scale – 4th Edition; TRF, Teacher Report Form

Table 4

Result of mediation and moderation analyses from included studies

Study	Predictor Variable	Outcome Variable	Mediator Variable			Results
	Diagnosis (manual) / trait	Diagnosis (manual) / trait	Diagnosis (manual) / trait	Age of trait measurement (mean years/range)	Measurement tools	
EDs in Autism						
Carter Leno et al. (2022)	Autistic social traits	Binge eating, fasting, purging	fussy eating	T1 = 6.8 T2 = 8.7 T3 = 9.6 T4 = 13.1	Parent-report	An indirect effect of autistic traits to disordered eating behaviours via fussy was found (*b*=0017, 95% CI [0.002-0.032], *p* = .026)
Koch et al. (2015)	ASD (infantile autism, atypical autism or Asperger syndrome; ICD-8/ICD-10)	AN/atypical AN (ICD-8/ICD-10)	major depression (ICD-8/ICD-10)	Age of diagnosis	National Patient Register/ Psychiatric Central Research Register	For those with ASD there was an elevated risk of AN after receiving a diagnosis of major depression (HR = 17.67, 95% CI [16.12–19.36]).
EDs in ADHD						
Martin et al. (2023)	ADHD	Binge eating, restrictive eating	Interoception	18-60	IES, IAS, BPQ-VSF	Inattentive symptoms predicted binge eating (Effect = 0.30, 95% CI [0.15, 0.55], *p* = .006) but not restrictive eating

						indirectly through IAS. No significant effects were found for Hyperactive/Impulsive symptoms and binge eating or restrictive eating via interoception measures
Sonneville et al. (2015)	ADHD	Binge eating	Late childhood overeating and strong desire for food	Late childhood over-eating = 11.7 Strong desire for food = 12.8	CI-BPDUK, DAWBA	A significant indirect effect was found for via late childhood overeating and early adolescent strong desire for food (standardized estimate: 0.085, 95% CI [0.007, 0.128], p = .03) on binge eating during mid adolescence
EDs in Autism & ADHD						
Christiansen et al. (2024)	ADHD (ICD-10), ASD (ICD-10)	AN, BN, EDNOS (ICD-10)	Mood and anxiety	Age of diagnosis	National Patient Register/ Psychiatric Central Research Register	Estimated effects of ASD and ADHD on EDs were small when mood or anxiety disorders were included into the model (mood or anxiety disorders could account for between 44% - 100% of the associations)

ADHD, Attention/Deficit Hyperactivity Disorder, AN, Anorexia Nervosa; ASD, Autism Spectrum Disorder; BN, Bulimia Nervosa; BPQ-VSF, The Body Perception questionnaire – Very Short Form; CI, Confident Interval; CI-BPDUK, Childhood Interview for DSM-IV Borderline Personality Disorder: UK Version; DAWBA, The Development and Well-being Assessment; EDNOS, Eating Disorder Not Otherwise Specified; HR, Hazard Ratio; IAS, The Interoceptive Accuracy Scale; ICD-8, International Classification of Diseases – 8th Revision; ICD-10, International Classification of Diseases – 10th Revision; IES, The Intuitive Eating Scale

Quality assessment

As described above, the quality of each study was assessed across three categories on the NOS including sample selection, comparability, and evaluation of the outcome. Quality assessments were independently reviewed with a percentage agreement of 90%. Whilst the NOS does not provide an overall rating of quality it allows insight into the methodological strengths and limitations of the current literature, in relation to the research question. Importantly, this showed that most studies did not provide information on whether disordered eating traits were present before the measurement on traits of autism and ADHD.

Table 5

Newcastle-Ottawa Quality Assessment Scale for Cohort Studies ratings

Study	Selection					Comparability	Outcomes/Exposure			Quality
	Exposed cohort	Nonexposed cohort	Ascertainment of exposure	Outcome not present at the start of the study		Controls for important factors and additional factors	Assessment of outcome	Adequate follow-up length	Adequate follow-up	Total stars
EDs in Autism										
Carter Leno et al. (2022)	*	*	*			**		*	*	7
Dinkler et al. (2021)	*	*	*	*		**	*	*	*	9
Koch et al. (2015)	*		*	*			*		*	5
Martini et al. (2022)	*	*	*	*		**	*	*	*	9
Schaumberg et al. (2021)	*		*				*	*		4
Solmi et al. (2021)	*	*	*	*		**		*		7
EDs in ADHD										
Biederman et al. (2007)		*	*	*		**	*	*	*	8
Biederman et al. (2010)		*	*			**	*	*		6
Bisset et al. (2019)	*	*	*			**	*	*		7
Bufferd et al. (2022)	*		*				*	*	*	5
Hinshaw et al. (2006)		*	*			*	*	*	*	6
Hinshaw et al. (2012)		*	*			*	*	*	*	6
Martin et al. (2023)	*	*	*			*	*			5
Mikami et al, (2008)		*	*			*	*	*	*	6

Mikami et al. (2010)	*	*	*		*	*	*		6
Robinson et al. (2020)	*	*	*	*	**	*	*		8
Sonneville et al. (2015)	*	*	*		**		*		6
Viborg et al. (2014)		*	*		*	*	*	*	6
Yilmaz et al. (2017)	*	*	*		**	*	*		7
EDs in autism and ADHD									
Christiansen et al. (2024)	*	*	*	*	*	*	*	*	8
Lin et al. (2024)	*	*	*	*	**	*	*	*	9

Study findings

Results from the included studies can be found in Table 3 with results from mediations analyses found in table 4.

Autism and disordered eating

Six studies explored the longitudinal association between traits or diagnoses of autism and future disordered eating. Of these, three studies looked at diagnoses of AN, with one study showing no significant association between autistic traits at age nine and AN at age 18 (Dinkler et al., 2021). In the remaining two studies, Koch et al. (2015) found that individuals with a diagnosis of autism had an elevated risk of receiving a diagnosis of AN at follow-up (HR=5.39). Martini et al. (2022) also found a diagnosis of autism significantly increased the risk of a later diagnosis of AN across males (HR=3.44, p = .001) and females (HR=2.04, p < .001).

Two studies investigated the association between autism and future BN. Martini et al. (2022) did not find a significant longitudinal association between a diagnosis of autism and BN. Schaumberg et al. (2021) however found a significant association between autism traits at age 14 and BN at age 16 in females (OR=2.31, p =.03). The authors also found a significant association for traits of autism at age 10 in males and BN at age 16 (OR=6.55, p = .002).

One study looked specifically at purging behaviours (Schaumberg et al., 2021) finding a significant association between autistic traits at age 10 and purging at age 16 in males (OR=4.72, p = .029). The authors also found a significant association between autistic traits at age 10 and binge eating at age 16 in males (OR=3.71, p = .004).

Two studies (both using the same population-based longitudinal UK cohort) found significant associations between traits of autism and future disordered eating. Solmi et al. (2021) found that autistic traits at age seven were associated with any disordered eating behaviour at age 14 (OR=1.18, p = .004). Similarly, Carter Leno et al. (2022) found that autistic traits at age seven were associated with disordered eating at age 14 (b=0.452, p < .001).

ADHD and disordered eating

Four studies looked at longitudinal associations between ADHD and diagnoses of AN. In their first study at five-year follow-up, Biederman et al. (2007) found that whilst females with ADHD were more likely to receive a diagnosis of an eating disorder (either AN or BN), Cox proportional hazards survival models were not statistically significant for AN (HR=2.7, p =.09). This result was replicated at their 11-year follow-up (Biederman et al., 2010) where females with ADHD were more likely to meet criteria for an ED, but this was not statistically significant for AN (HR=2.2, p =.11). Similarly, Bisset et al. (2019) did not find a significant difference between the prevalence of AN between the ADHD group and controls at follow-up. Further, Bufferd et al. (2022) also found no significant association between ADHD at age three and AN at age 15. One additional study, explored traits of AN through restrictive eating (Martin et al., 2023) showing that restrictive eating at follow-up was directly predicted by inattentive symptoms (Effect = 0.30, S.E. = 0.10, T = 2.9, p = .006, CI = 0.15 - 0.55) and hyperactivity/impulsivity symptoms (Effect = 0.29, S.E. = 0.12, T = 2.4, p = .017, CI =

0.052 - 0.53).

Five studies investigated associations with traits or diagnoses of BN. Biederman et al. (2007) found a significant association between ADHD and future BN compared to controls (HR=5.6, $p < .01$) with this finding being continued in their 11-year follow-up (HR=5.2, $p = .001$; Biederman et al., 2010). Yilmaz et al. (2017) found that a combination of higher inattention and hyperactivity/impulsivity symptoms during childhood was significantly associated with symptoms of bulimia during adolescence (.73 [.12]; $p < .01$). Mikami et al. (2010) also found a significant association between traits of ADHD and future BN with differences across genders. They found that childhood impulsivity in those with ADHD associated with BN at follow-up with this effect significantly stronger for females ($\beta = 0.49$, $p < .001$) than males ($\beta = 0.18$, $p < .001$). This finding was also shown in self-report measures of BN at follow-up (Females: $\beta = 0.49$, $p < .001$; Males: $\beta = 0.18$, $p < .001$). However, Bufferd et al. (2022) did not find a significant association between ADHD at age three and BN symptoms at age 15. One additional study investigated purging (Robinson et al., 2020) finding a significant association between ADHD and future purging (OR=4.03, $p < .05$).

Four studies looked at future symptoms of binge eating in those with ADHD. Bisset et al. (2019) found that boys with ADHD had significantly greater odds of weekly binge eating at follow up compared to controls (OR=9.4, $p < .05$). When looking at traits of ADHD, Martin et al. (2023) found that inattentive symptoms predicted binge eating directly (Effect = 0.85, S.E. = 0.13, T = 6.7, $p < .001$) as well as hyperactivity/impulsivity symptoms (Effect = 0.33, S.E. = 0.16, T = 2.1, $p = .04$). When looking at hyperactivity/inattention during late childhood and binge eating during mid-adolescence, Sonneville et al. (2015) did not find a significant direct association. Robinson et al. (2020) did not find a significant association between ADHD and future binge eating; however, they did find an association at the trait level between hyperactivity/inattention and binge eating (OR=1.25, $p = .014$).

Six studies reported on disordered eating and their associations with ADHD. In their five-year follow-up study, Hinshaw et al. (2006) found that females with childhood ADHD-combined type scored significantly higher on measures of ED symptomatology at follow-up than controls. However, in their 10-year follow-up (Hinshaw et al., 2012) the authors did not find any significant association between childhood ADHD and future disordered eating. Mikami et al. (2008) also found that childhood ADHD was significantly related to eating pathology at five-year follow-up ($F(4, 372) = 2.78$, $p < .05$). Similarly, Viborg et al. (2014) found that hyperactivity/inattention predicted disordered eating at one-year follow-up (β=.13, $p < .01$). Yilmaz et al. (2017) also found that a combination of higher inattention and hyperactivity/impulsivity symptoms during childhood was significantly associated with disordered eating symptomatology at 9-year follow-up (drive for thinness: -.14 [.31], $p < .01$; body dissatisfaction: .43 [.50], $p < .01$). They did not however find significant associations individually between either inattention or hyperactivity/impulsivity and future disordered eating. A significant association between childhood ADHD and body dissatisfaction was also shown by Bufferd et al. (2022) with ADHD at age six being associated with body dissatisfaction at age 12.

Autism, ADHD, and disordered eating

Two studies looked at both traits of autism and ADHD and their associations with future disordered eating. In their population-based cohort study, Christiansen et al. (2024) found an increased risk across all ED diagnoses in individuals with an initial diagnosis of autism or ADHD. When the authors adjusted for co-occurring autism and ADHD this only slightly attenuated the association with future ED diagnoses. The second study (Lin et al., 2024) was retrospective measuring diagnoses of ASD or ADHD one-year prior to ED diagnosis. The authors found that a prior diagnosis of autism was more common in those with an ED diagnosis compared to those without an ED diagnosis (n = 1208 [8.3%] vs 2515

[2.3%]). Similarly, the authors found that a prior ADHD diagnosis was more common in those with an ED diagnosis than individuals without a diagnosis of an ED (n = 2503 [17.2%] vs 9210 [8.4%]).

Possible moderating/mediating pathways

Five studies reported on possible mediating factors in the associations between autism or ADHD and future disordered eating with two studies on autism, two on ADHD and one looking at both autism and ADHD.

In their study on autistic traits at age seven, Carter Leno et al. (2022) found an indirect effect of autistic traits on ED behaviours at age 14 via fussy eating (b=0017, 95% CI=0.002-0.032, p = .026). Koch et al. (2015) investigated the association between diagnoses of autism and future AN. They found that the significant association between autism and AN was even greater when individuals also received a diagnosis of major depression (HR=17.67).

When looking at interoception as a mediating factor in the association between traits of ADHD and disordered eating, Martin et al. (2023) found that inattentive symptoms predicted binge eating indirectly through measures of interoception (Effect = 0.57, Bootstrapped S.E. = 0.29, Bootstrapped CI = 0.06 - 1.09), but this was not found with restrictive eating. The authors found that hyperactivity/impulsivity symptoms did not predict either binge eating or restrictive eating indirectly through interoception as measured by the IAS or BPQ.

Sonneville et al. (2015) also explored traits of ADHD and future binge eating finding hyperactivity/inattention during early childhood indirectly predicted binge eating via late childhood overeating and early adolescent strong desire for food (standardized estimate: 0.085, 95% CI: 0.007, 0.128; p = .03).

In their study investigating associations between diagnoses of both ASD and ADHD with EDs, Christiansen et al. (2024) reported on mood or anxiety disorders as mediating factors. The authors mediation analyses showed that the estimated direct effects of ASD and ADHD on EDs were small when mood or anxiety disorders were included into the model suggesting that mood or anxiety disorders may account for between 44-100% of the associations between ASD, ADHD and EDs.

Discussion

This systematic review aimed to summarise the findings of longitudinal studies exploring associations between traits of autism or ADHD and future disordered eating. The review also aimed to identify any possible mediating factors identified through these longitudinal studies. There have previously been useful systematic reviews on the prevalence of co-occurring autism and EDs (Huke et al., 2013), ADHD and EDs (Kaisari et al., 2017; Levin & Rawana, 2016), and the overlap between autism, ADHD and EDs (Nickel et al., 2019). However, to the best of my knowledge, this is the first systematic review with a focus on longitudinal studies that include traits of autism, ADHD, and disordered eating as well as formal diagnoses. This approach is of particular importance when considering the gender discrepancies in diagnoses with females being more likely to receive a diagnosis of autism or ADHD in later life compared to males or remain undiagnosed (Attoe & Climie, 2023; Gesi et al., 2021) along with needing a greater severity of traits to reach threshold for a diagnosis (Dworzynski et al., 2012). By considering traits of autism and ADHD it is hoped this may capture those who do not reach threshold for a clinical diagnosis with the longitudinal perspective also capturing those who receive a diagnosis later in development. Further, as studies have typically explored associations with a particular ED, such as AN, the inclusion of any disordered eating outcome in this review provides a comprehensive review in the wider profile of these associations. As this review has shown evidence for an association

between autism and disordered eating across the spectrum, this also provides evidence that these associations are not solely due to starvation effects or that high scores on autism measures are inflated by their ED as has been suggested in research on restrictive forms of disordered eating,

Autism and disordered eating

This review has provided evidence for an association between autism and disordered eating behaviours across the spectrum. Longitudinal findings of autistic traits preceding AN provide support for autism being a risk factor for future EDs. Across the three studies exploring the association between autism and AN, two found a significant association between autism and future AN (Koch et al., 2015; Martini et al., 2022). This confirms the previously identified cross-sectional associations of autistic traits in those with AN (Kerr-Gaffney et al., 2020; Rhind et al., 2014; Westwood et al., 2016) and co-occurring AN in those with a diagnosis of autism (Margari et al., 2019). Further this also shows that autistic traits in those with AN are not solely be due to starvation effects, supporting previous findings that autism traits remain after weight restoration and AN recovery (Boltri & Sapuppo, 2021). Whilst one study did not find an association between childhood autistic traits and later AN (Dinkler et al., 2021), the authors speculated whether this may be due to the recognised under-diagnosis of autism in females. To measure the presence of autism in childhood, Dinkler et al. (2021) utilised confirmed diagnoses from a national patient register, it is therefore possible that this approach was not able to capture accurate rates of autism if participants were undiagnosed or misdiagnosed when this data was captured.

Of the two studies looking at the longitudinal associations between autism and BN, there appears to be conflicting findings with Schaumberg et al. (2021) finding a significant association across genders which was not found by Martini et al. (2022). The previous, whilst

limited, research on autism and BN and BED has also shown conflicting results thus it remains unclear to what extend an association exists. However, the prevalence rates of BN in Martin et al.'s (2022) general-population study were reported to be at 0.0-0.1% for males and 0.3-0.1% for females which are below estimated lifetime prevalence rates of 1% for males and 3% for females reported by Van Eeden et al. (2021). Thus, it could be possible that BN was underdiagnosed amongst participants within Martini et al.'s (2022) sample and therefore rates were too low to find an association.

Schaumberg et al. (2021) also looked at specific traits of purging, commonly seen in both AN and BN, and binge eating which is more associated with BN and BED. Using data from the Avon Longitudinal Study of Parents and Children (ALSPAC), the authors found significant associations between autism and purging and binge eating in males but not females, suggesting there may be gender differences in these associations. This finding is interesting when male experiences of EDs have historically been neglected in research (Makin et al., 2025). Again, this highlights the importance of trait level research as when looking at diagnoses of BN a significant association was found in autistic females.

Importantly, there is evidence to suggest that these associations exist across those with traits of disordered eating as opposed to formal diagnoses. Two studies explored the association between autism and future disordered eating behaviour rather than focussing on a specific diagnosis. Both studies (Carter Leno et al., 2022; Solmi et al., 2021) found significant associations between autistic traits at age seven and future disordered eating at age 14. This replication of findings between the two studies is, however, expected as both studies utilised secondary data from ALSPAC. Nonetheless, these findings show that the association between autism and EDs remains present at the trait level. These studies included behaviours on dieting, fasting, purging, and binge-eating which are common features across different ED diagnoses. As most previous research has focussed on a particular ED diagnosis, these

findings therefore highlight the potential utility of a transdiagnostic approach at the trait level to identify risk of disordered eating.

ADHD and disordered eating

When looking at traits of ADHD and AN, no statistically significant associations were found. Whilst Biederman et al. (2007; 2010) found those with ADHD were at a greater risk for an ED this was not significant for AN at either their 5-year or 11-year follow-up. Similarly, neither Bisset et al. (2019) or Bufferd et al. (2022) found any significant longitudinal associations. However, when looking specifically at restrictive eating, indicative of AN, Martin et al. (2013) did find that this was directly predicted by both inattentive symptoms and hyperactivity/impulsivity symptoms. This finding supports those by Bleck et al. (2015) who found that those with ADHD are more likely to experience restrictive eating behaviours than those without ADHD. These mixed findings suggest that whilst individuals with ADHD may not reach threshold for a formal ED diagnosis of AN, there is still a risk of restrictive behaviours. Alternatively, this could point to the gender discrepancies in diagnosis with a tendency for females to be underdiagnosed with ADHD and males to be underdiagnosed with AN (Makin et al., 2025).

The studies exploring the longitudinal associations between ADHD and BN provide a clearer picture; suggesting that ADHD is a risk factor of BN and associated binge/purging behaviours. Four studies showed significant associations between traits of ADHD or ADHD diagnoses and future BN (Biederman et al., 2007; Biederman et al., 2010; Mikami et al., 2010; Yilmaz et al., 2017). These findings support the findings of previous cross-sectional studies (Appolinario et al., 2022; Ptacek et al., 2016; Ziobrowski et al., 2018) and suggest that these associations persist over time. Only one study, that of Bufferd et al. (2022), did not find a significant association between ADHD symptoms at age three and BN symptoms at

age 15. Once possible explanation for this may be due to the age at which ADHD symptoms were measured. Whilst ADHD is more likely to be diagnosed during childhood for males, this is typically much later for females (Martin, 2024), thus this data may not have captured accurate prevalence of ADHD in this cohort. An association was also found between ADHD and future purging behaviours by Robinson et al. (2022). Whilst purging could be indicative of either AN or BN, this finding still proves useful in further identifying associations at the trait level.

The findings on the association between ADHD and binge-eating are somewhat mixed. Two studies found an increased risk of future binge eating across both confirmed diagnoses of ADHD in males (Bisset et al., 2019) and at the trait level (Martin et al., 2023). This supports previous cross-sectional literature identifying an increased prevalence of ADHD amongst those with BED (Appolinario et al., 2022; Brewerton & Duncan, 2016). Whilst Sonneville et al. (2015) did not find a direct effect between childhood traits of ADHD and future binge eating they did find an indirect effect which will be discussed later. Robinson et al. (2020) did not find any association between ADHD and binge eating based on the DAWBA but did find an association at the trait level when using the hyperactivity/inattention subscale of the SDQ. Whilst these findings appear to show a complicated picture they may provide a useful insight into the mechanisms behind this association. The findings on trait level ADHD and future binge eating are compelling but as Sonneville et al. (2015) found this relationship only became significant through an indirect route of late childhood overeating and strong desire for food. We can also infer that severity of ADHD symptomatology may not be the driving force behind this relationship based on Robinson et al.'s findings. This therefore suggests that the association between ADHD and future binge eating is driven by some other factor.

There has also been a substantial number of studies looking at disordered eating in general rather than specific diagnoses. The findings from these studies offer interesting understandings into the relationship between ADHD and disordered eating. Firstly, Hinshaw et al. (2006; 2012) found an association between ADHD-combined type and ED symptomatology at 5-year follow-up but not at their 10-year follow-up in their sample of females. This suggests that the risk of disordered eating may not persist in the longer term. However, on further exploration this picture is complex. Whilst 40% of their participants with ADHD no longer met diagnostic criteria at 10-year follow-up suggesting a reduction in symptoms, their analyses found that longitudinal associations were no longer significant at this time point as the groups were not significantly different from each other in terms of ED symptomatology. Although individuals with ADHD had significantly higher disordered eating scores during adolescence compared to controls, these scores rose across both groups but at greater levels in the control group and therefore became matched during adulthood. Thus, the risk of eating disorders persisted but there was not a significant difference between the two groups (Hinshaw et al., 2012)

Hinshaw et al.'s (2006) initial finding is however supported by the other longitudinal studies showing associations between ADHD or traits of and future disordered eating (Mikami et al., 2008; Viborg et al., 2014). Two other studies found an association between traits of ADHD and drive for thinness (Yilmaz et al., 2017) and body dissatisfaction (Bufferd et al., 2022; Yilmaz et al., 2017). However, Yilmaz et al. (2017) showed that this relationship was only significant when looking at combined inattention and hyperactivity/impulsivity traits and not for inattention and hyperactivity/impulsivity individually. Thus, it could be suggested that it is the combination of ADHD traits that increases the risk for future disordered eating. This may help to understand the findings from Hinshaw et al.'s (2012) 10-year follow-up as it has been purported that symptoms of hyperactivity and impulsivity

decline at a higher rate than inattention with age (Biederman et al., 2000). However, as these results were based on the trajectory of ADHD symptoms in males and not females as is the case in Hinshaw et al.'s (2012) study, this does not provide a comprehensive picture. Nonetheless, a more recent study by Wootton et al. (2022) showed that ADHD scores for males are higher in childhood than females, but over time there is a reduction in symptomatology across both genders, with a steeper decline for males, so that in adulthood average scores for males and females become similar. Although it has been suggested that measures of ADHD are less valid for detecting childhood traits in females, this still provides useful evidence of the developmental course of ADHD into adulthood (Wootton et al., 2022).

Autism, ADHD, and disordered eating

Only two studies looked at both autism and ADHD in their studies, both of which looked at any ED diagnosis. Lin et al.'s (2024) retrospective study measured rates of autism and ADHD one year prior to ED diagnosis. The authors found that both prior autism and prior ADHD diagnoses were more common in those with an ED diagnosis than those without an ED diagnosis. Christiansen et al. (2024) is the only study that specifically investigated the co-occurrence of autism and ADHD in individuals. They found an increased risk of any ED in individuals with either autism or ADHD and when adjusting for co-occurring autism and ADHD they found this only slightly attenuated associations. This finding supports the findings from Karjalainen et al.'s (2016) cross-sectional study on EDs in autism and ADHD. Interestingly they found the rates of any ED diagnosis was lower in the autism and ADHD group (2.2%) compared to the autism group (10.8%) and ADHD group (8.3%). This suggests that those with co-occurring autism and ADHD are still at risk of future disordered eating but importantly the dual diagnosis does not appear to increase the risk, with a possibility that the risk may be lower.

Mediation/Moderation pathways

Across the included studies, several also identified possible mediating factors that may underpin or contribute to observed longitudinal associations. Firstly, one possible factor to consider is that of co-occurring mental health difficulties. In their study, Koch et al. (2015) showed that whilst there was an association between autism and AN, this was strengthened when they included diagnoses of depression in their analysis, suggesting that depression contributes to the risk of future disordered eating in autism. Previous literature has identified an association between depression and AN with one possible explanation for this suggested to be the role of shared genetic risk factors (Wade et al., 2000). Whilst the authors cannot rule out the contribution of shared environmental factors, this finding is interesting given the increasing research on polygenic risk scores in autism and disordered eating. Therefore, these associations and findings from Koch et al.'s (2015) study could be explained by a genetic predisposition. The role of other mental health difficulties as a mediating factor was also shown by Christiansen et al. (2024) who looked at mood and anxiety disorders in those with autism and ADHD. They concluded that mood an anxiety disorders may account between 44% and 100% of the association between autism, ADHD, and disordered eating, suggesting these may play an important role in observed associations. This would support the findings of previous research such as Ziobrowski et al.'s (2018) analysis of associations between ADHD and EDs. The authors found, when adjusting for psychiatric comorbidities, all associations of ADHD and EDs were substantially attenuated with only the association with BN remaining statistically significant. The authors suggest that the associations between ADHD and EDs may be in part due to additional psychiatric diagnoses that are commonly co-occurring in ADHD and disordered eating (Ziobrowski et al., 2018)

The other studies included in the review explored the role of specific cognitive and behavioural factors. Carter Leno et al. (2022) found an association between autistic traits and

ED via fussy eating. This supports previous literature identifying fussy eating or selective eating as prevalent in both autistic populations (Mari-Bauset et al., 2014) and within ED diagnoses (Herle et al., 2020). This provides evidence that fussy eating might be one of the mechanisms underlying the association between autistic traits and future disordered eating.

With ADHD, Sonneville et al. (2015) found that future binge eating was predicted via childhood overeating and strong desire for food and Martin et al. (2023) found inattentive symptoms predicted binge eating via IAS scores, a measure of interoception. These associations may be explained by inattention to internal signs of hunger and satiety (Cortese et al., 2007), deficient inhibitory control (Davis et al., 2006) or shared characteristics of low cognitive control and high impulsivity (Sonneville et al., 2015). This aligns with previous research hypothesising that impulsivity drives the association between ADHD and disordered eating (Ptacek et al., 2016) and diminished interoception accuracy in individuals with ADHD (Bruton et al., 2025).

Strengths and Limitations

A major strength of this review is the focus on longitudinal studies with traits of autism or ADHD presenting before onset or diagnosis of disordered eating. This approach allows us to further our understanding of the previous literature by ruling out the impact of artefact such as starvation effects seen in AN. By including trait level studies this also helps to include those who may be subject to bias in the referral and assessment pathways and thus provides a more accurate representation of the population of interest. The methodology of this review was robust with independent double screening at each stage with minimal disagreements. A thorough quality assessment of the included studies was also conducted with an independent reviewer.

It is important to note that many useful studies were not included as they were outside the scope of this current review. This review focussed on traits of autism and ADHD given the high rates of co-occurrence, however it would be useful to understand such associations across other neurodevelopmental conditions in line with the neurodivergence movement. Whilst the review also aimed to look at possible mediating factors or moderation pathways, this was taken from studies researching longitudinal associations and therefore did restrict the inclusion of studies specifically exploring potential causal mechanisms. These studies are invaluable to give us insight into mechanisms that could be identified and supported within treatment approaches.

There are some limitations that are important to consider when interpreting the results of this review. Firstly, the pre-registration and inclusion criteria of the review stipulated studies must utilise validated standardised measures of traits of autism, ADHD, and disordered eating. Across the included studies there was little consistency in the measures used and it remains unclear what the gold-standard for each construct should be. Some of the included studies used self-report identification of disordered eating behaviours such as those from the ALSPAC studies. Whilst this could not be considered a validated measured these have been well documented and used across a multitude of studies to capture disordered eating behaviours (Bould et al., 2018; Micali et al., 2017), this was agreed to be included on the basis that they are consistently used within research and come from a large population-based cohort study and can therefore be translated across similar research. Similarly, many of the included studies utilised patient health records for confirmation of the diagnosis. An assumption was made here that for participants to receive a formal diagnosis a standardised measure or assessment would have taken place to receive the diagnosis, however, it is recognised that there may be different practices across countries in the assessment of neurodevelopmental conditions. Whilst this study aimed to include trait level research and

one could assume that traits must be present to have received a diagnosis irrespective of the assessment process, the possibility of misdiagnosis cannot be ruled out. Further, the current review included retrospective designs where measures or diagnosis were conducted at the age of interest but did not include retrospective self-report on symptoms at an earlier age time point. This difference is especially pertinent when considering the later age of diagnosis or misdiagnosis for females with ADHD where retrospective self-report may be crucial, and means this review is limited in its capacity to capture the experiences of this population.

Another consideration important to discuss is the decision to exclude studies that solely looked at a single trait of autism or ADHD such as social communication difficulties or impulsivity. The rationale for this was that these individual traits may not be exclusive to autism or ADHD and would therefore limit the validity of these studies and interpretability of findings. Mikami et al.'s (2010) study was included as participants had received a diagnosis of ADHD and thus represented traits across the full criteria despite their main finding reported being on impulsivity. This does however show the utility of inclusion of single trait studies that may further our understanding of the causal mechanisms driving these associations. Further, it has been suggested that these individual traits are highly predictive of later diagnosed neurodevelopmental conditions such as autism and ADHD (Dinkler et al., 2022).

Whilst the large sample sizes and age ranges of the included studies are strengths of this review, there are several factors that limit their generalisability and interpretability. Three of the included studies had short follow-up lengths ranging from six months to one year (Lin et al., 2024; Martin et al., 2023; Viborg et al., 2014) thus conclusions on the direction of association must be interpreted with caution as a reciprocal association may be possible. Across the studies there was also variation in gender rations, with several studies including females only (Biederman et al., 2007; Biederman et al., 2010; Hinshaw et al, 2006; Hinshaw

et al., 2012; Mikami et al., 2008; Viborg et al., 2014) limiting their generalisability across genders. Nonetheless, the inclusion of the Biderman et al. (2007; 2010) and Hinshaw et al. (2006; 2012) studies provided a unique opportunity to understand how these associations may change over time at different follow-up lengths. It is also important to consider gender biases in such diagnoses with Koch et al. (2015) a prime example of this with almost 80% of their autism sample being male and around 93% of the AN sample being female. This reflects the well-documented issues surrounding underdiagnosis of neurodevelopmental conditions in females and a lack of recognition of disordered eating in males. Another significant limitation of the studies samples is the lack of diversity across ethnicities with all included studies coming from western cultures. For example, around 94% of Biederman et al.'s (2007) sample identified as White. Lastly, despite a range of disordered eating being explored across the included studies there were no studies looking at newer diagnoses such as ARFID and thus highlights a gap in the literature.

Clinical Implications

Historically, research has focussed on the relationship between autism and AN, however, through the increasing evidence base and this current review, it is apparent that the relationship between neurodivergence and disordered eating is complex and not limited to restrictive patterns or autism alone. This is particularly important for clinical practice where existing treatment methods that are often modelled around AN may not meet the needs of those with neurodivergence or other forms of disordered eating (Herle et al., 2025). The greater understanding into the complexities of these associations helps to identify more appropriate treatment pathways which in turn will support better outcomes for these individuals.

The documented longitudinal associations show that autism and ADHD may predict or increase the risk for future disordered eating. It is therefore crucial that individuals within ED services are being screened for both these neurodevelopmental conditions. Whilst there is an impact of starvation on cognitive functioning that can appear consistent with autistic traits it should not be assumed that these are due to being underweight, and a thorough assessment of autism should be considered once individuals have reached weight restoration should the traits or difficulties persist. If traits of autism or ADHD are present, treatment should be appropriately tailored to meet the needs of the individuals based on their personal strengths and challenges in lieu of specific adapted treatment protocols. Consideration of how their neurodivergence may be contributing to the development of their disordered eating needs to be addressed within treatment as specific traits may be acting as maintaining or causal factors. An individual's formulation should therefore consider the range of possible causal factors that are emerging within the research such as specific cognitive or behavioural traits and co-occurring mental health conditions that can guide the intervention.

Avenues for future research

This review identified associations between autism and ADHD across the spectrum of disordered eating, however it is clear there are existing gaps within this area, particularly a lack of research on ARFID. Given its relatively recent addition to diagnostic manuals it is understandable that there is a paucity of longitudinal studies on ARFID, however this is a pertinent area of research to be furthered due to the shared traits with autism and its consideration of being a disorder of childhood which may result in misdiagnosis in adults. As long-term outcomes of ARFID and associations with neurodevelopmental conditions remain largely unknown, further longitudinal research would therefore be essential to provide further clarity.

This study also revealed a distinct lack of research which considered the co-occurrence of autism and ADHD. We are therefore limited in our understanding of the complex interplay between these and their impact on future disordered eating. Most of the included studies in this review did not report on co-occurring conditions or controlled for them, and given the high rates of comorbidity between the two, one could reasonably conclude that many of these studies may be inadvertently looking at associations of co-occurring autism and ADHD on disordered eating rather than individual diagnoses. This may in part explain the wide ranging and complex associations identified and further research on co-occurring autism and ADHD is therefore warranted to understand this profile better. There has also been a lack of research into other neurodevelopmental conditions which was outside the scope of this review but would be pertinent to explore to ensure inclusivity across neurodivergence.

The focus on longitudinal associations within this review has helped to disentangle artefact from true associations, such as the impact of starvation on cognitive function in AN, and identified possible causal mechanisms. Previous research has suggested certain features characteristic of autism and ADHD may contribute to the development of disordered eating behaviours. Several features have also been suggested to be common across neurodivergence and disordered eating such inflexible cognitive styles, emotion dysregulation and differences in sensory processing, interoception and alexithymia (El Archi et al., 2020; Kinnaird & Tchanturia, 2021; Westwood et al., 2025). It has been suggested that these characteristics may act as mediating or moderating factors in the development of disordered eating. Hypothesised mechanisms include the role of sensory differences presenting as food selectivity and sensitivity to smell and textures leading to restrictive eating behaviours (Karaca et al., 2025). Further, interoception has been suggested to contribute to the development of disordered

eating in those with ADHD due to differences in awareness of hunger and satiety cues (Martin et al., 2023).

This review has begun to evidence that these associated process may act as causal mechanisms for disordered eating such as the role of interoception. However, further longitudinal studies are required to understand which features of neurodivergence may contribute to the development of disordered eating behaviours and the mechanisms behind this This would help the understanding of the cognitive and behavioural presentation of disordered eating in neurodivergent people which could be distinguished from the underlying drive for thinness or preoccupation with body image as in traditional understandings of eating disorders. Whilst these individuals may still experience such cognitions, this would impact on current approaches to therapeutic support for this population with a focus on individual underlying characteristics as opposed to beliefs about body image and weight-control behaviours.. A greater understanding of these possible mechanisms underlying the association between neurodivergence and disordered eating would therefore be essential for adapting therapeutic intervention for this cohort.

Lastly, but perhaps most importantly, it is imperative that future research should be conducted on the development of effective interventions for EDs that are rooted in the understanding that the key mechanisms driving disordered eating may differ from those whose are neurotypical. In turn this will support the development of evidence-based guidance for clinicians working with neurodivergent individuals in ED services that is crucially lacking. Co-production of these interventions with individuals with lived experience is recommended to ensure that treatment is not only accessible and affirming for neurodivergent people but is also effective.

Conclusions

This review provides a comprehensive synthesis of current literature pertaining to longitudinal associations between autism and ADHD, and disordered eating. Whilst these associations are complex and further understanding of the mechanisms underlying these relationships is needed, this review provides evidence that these associations exist across both autism and ADHD and the spectrum of disordered eating. This review supports previous research suggesting that autism and autistic traits are likely a risk factor for AN; and ADHD and ADHD traits are likely a risk factor for BN with emotional distress likely mediating the relationship between neurodevelopmental conditions and future disordered eating. Further, the longitudinal approach has clarified that neurodevelopmental conditions can predate the onset of disordered eating and are not solely due to artefact or shared traits. The review advocates the need for further insight into causal mechanisms and development of appropriately adapted interventions. Importantly, these implications for clinical practice and recommendations for future research are in line with the priorities identified by individuals with lived experience of autism, ADHD, and disordered eating (Keller et al., 2024).

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Part 2: Empirical Paper

Longitudinal associations between traits of autism, ADHD, and disordered eating behaviours in adolescence: a UK population-based study

Abstract

Aims

This study aimed to estimate developmental trajectories for traits of autism and ADHD in childhood, and test associations between these trajectories and disordered eating behaviours in late adolescence. The study also aimed to test whether any gender differences were present in the links between neurodivergent traits and eating disorder outcomes.

Methods

Data was used from the Avon Longitudinal Study of Parents and Children (N = 3014). Traits of ADHD and autism were assessed across childhood from age seven to age 16 using the hyperactivity/inattention subscale of the Strengths and Difficulties Questionnaire and the Social and Communication Disorders checklist, respectively. Disordered eating behaviours (fasting, purging and binge eating) were self-reported at age 18 years. Covariates included gender, ethnicity, maternal level of education and maternal age at birth of child. Latent growth curve models were used to estimate developmental trajectories of traits of autism and ADHD. Logistic regressions were conducted to evaluate the associations of trajectories of ADHD and autism with future disordered eating behaviours.

Results

Of the 3014 young adults included in the sample, 402 reported any disordered eating behaviours at age 18. Higher levels of traits of ADHD at age seven were associated with increased weekly binge eating behaviours at age 18 years. Higher levels of traits of autism at age 7 were associated with increased fasting and binge eating behaviours at age 18, with a steeper increase in traits between the ages of seven and 16 years being associated with a greater likelihood of experiencing purging behaviours. In models that included both traits of autism and ADHD as simultaneous independent variables, associations between trajectories

of autism traits and later disordered eating outcomes were replicated. No significant gender differences were found within this study.

Conclusions

Traits of ADHD and autism in childhood may be a risk factor for the development of future disordered eating behaviours. Gender differences within these associations are yet to be elucidated. Further research is required to understand the causal mechanisms behind these associations.

Introduction

Eating disorders (EDs) are serious and enduring mental health conditions that have a significant impact on individual's quality of life, making effective treatment options imperative (Van Hoeken & Hoek, 2020). However, despite advances in intervention approaches in EDs the mortality rate remains high (Van Hoeken & Hoek, 2020). It is therefore crucial to advance our understanding of these complex disorders and understand the factors that contribute to these poor outcomes.

Within the current version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association [APA], 2013), the category of "Feeding and Eating Disorders" contains diagnostic criteria for several conditions that are typically characterised by restriction of calorific intake, weight and shape concerns, binge eating and compensatory behaviours. Two EDs that have been well documented historically are those of anorexia nervosa (AN) and bulimia nervosa (BN). Features of AN include disturbances in the perception of body shape and an intense fear of gaining weight which results in persistent restrictions of calorific intake with or without purging (APA, 2013). BN, on the other hand, is characterised by recurrent episodes of binge eating co-occurring with compensatory behaviours of purging or excessive laxative use (APA, 2013). A similar, yet distinct, condition to BN is that of binge eating disorder (BED) which is described as frequent episodes of binge eating and negative cognitions such as feelings of guilt without the associated compensatory behaviour (APA, 2013). BED was introduced into the DSM-5 following research on the validity and clinical utility of this being a separate disorder (Attia et al., 2013). Another more recently described condition is Avoidant/Restrictive Food Intake Disorder (ARFID) which is understood to be a restriction or avoidance of food that has a significant impact on one's weight, nutritional intake, and psychosocial functioning without the typical disturbances of body image (APA, 2013). When an individual shows traits of disordered eating with

significant distress that does not meet criteria for one of the disorders described above, the individual may fulfil criteria for the diagnosis of "Other Specified Feeding or Eating Disorder" (OSFED) or the term used within the previous edition of the DSM "Eating Disorder Not Otherwise Specified" (EDNOS).

AN has historically been shown to be more common in females with a lifetime prevalence rate of around 4% in females and 0.3% in males (Van Eeden et al., 2021). Similarly, BN is thought to have prevalence rates of around 3% in females and 1% for males (Van Eeden et al., 2021). The gender difference in BED is somewhat smaller with prevalence rates estimated at 1.4% for females and 0.4% in males (Erskine & Whiteford, 2018). As ARFID is comparatively a more recent diagnosis, research is still in its infancy. Nonetheless, a systematic review by Sanchez-Cerezo et al. (2023) has shown that prevalence rates are between five and 22.5% within specialist feeding clinics and 0.3 to 15.5% within non-clinical populations. However, the authors interestingly found that the gender discrepancy appears reversed in ARFID with more males receiving a diagnosis compared to females (Sanchez-Cerezo et al., 2023). Lifetime prevalence rates of OSFED have been reported to be 0.6% in females and 0.3% for males (Lindvall Dahlgren et al., 2017). Further, discrepancies in reported prevalence rates are suggested to be due to inconsistent use of measurement tools, study designs, and non-representative samples (Lindvall Dahlgren et al., 2017).

Eating Disorder comorbidity and outcomes

Treatment outcomes for EDs remains suboptimal despite individuals often receiving intensive intervention (Vall & Wade, 2015). It has been suggested that predictors of ineffective treatment adverse outcomes include co-occurring conditions (Vall & Wade, 2015). In AN, co-occurring physical and mental health conditions are common (O'Brien & Vincent, 2003), with disturbances in cognitive and emotional functioning resulting from starvation and low body weight being well documented (Brockmeyer et al., 2012). However, despite those

with BN often having a normal body weight, medical complications still occur due to bingeing or purging (Mehler & Rylander, 2015), with co-occurring mental health conditions remaining more prevalent in those with BN than the general population (O'Brien & Vincent, 2003). Similarly, physical, and mental health conditions also commonly co-occur in those with BED (Udo & Grilo, 2020), suggesting that the profound outcomes of EDs goes beyond the impact of starvation.

Thus, comorbidity has been proposed to arise for several reasons including being two manifestations of the same disorder, two stages of the same underlying condition, the same or correlated risk factors, one condition predisposing to the other, a result of artefact or finally to two conditions being nosologically distinct (Adams et al., 2024). One area of research that has begun to gain traction is the association of eating disorders and neurodivergence including autism and attention-deficit/hyperactivity disorder (ADHD).

Autism and Disordered Eating

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition characterised by wide-ranging traits across areas of social communication differences, sensory differences and restricted and repetitive behaviours and interests (APA, 2013). As such, the presentation of ASD varies greatly and is considered to exist across a spectrum (Rea et al., 2018). Research on terminology by the autism community has identified that "autism" and "autistic people" are preferrable and as such these terms will be used henceforth in replacement of the diagnostic label of ASD (Kenny et al., 2006; Monk et al., 2022).

Prevalence rates for autism are estimated at around 3% of the population (O'Nions, et al., 2023), with a ratio of 3:1 of males to females (Loomes et al., 2017). This greater preponderance of autism in males has historically resulted in a gender bias in research leading to a lack of understanding of female presentations (Van Wijngaarden-Cremers et al., 2014)

with diagnostic criteria and assessment tools being based on the male phenotype (Lai et al., 2015). This has contributed to females requiring greater severity of autistic features to meet thresholds for a diagnosis (Dworzynski et al., 2012) and being more likely to receive a diagnosis in later life compared to their male counterparts who are more likely to be diagnosed during early childhood (Gesi et al., 2021). Nonetheless, rates of autism diagnoses are rising suggesting there is an increased understanding of the female presentation of autism (Arvidsson et al., 2018) along with more individuals able to receive a diagnosis due to changes in diagnostic criteria (Kulage et al., 2014).

The conceptualisation of autism has evolved over time with it historically considered in terms of deficits and impairments (Pellicano & den Houting, 2022). The neurodiversity movement, proposing that cognitive diversity is normal, has supported an alternative understanding of autism where differences in cognition and communication are relational (Pellicano & den Houting, 2022). Thus, the challenges and distress than autistic people encounter are purported to be caused by norms and expectations of a neurotypical society and social barriers (Chapman, 2021). Along with the impact of social barriers on psychological distress, autistic people are shown to be at a greater risk for physical and mental health conditions compared to non-autistic individuals (Lai & Baron-Cohen, 2015; Tye et al., 2019). In their meta-analysis, Lai et al. (2019) found that in those autistic people, 20% met criteria for an anxiety disorder, 11% for depressive disorders and 9% for obsessive-compulsive disorders.

A substantial body of research has also focussed on the association between autism and EDs due to the complexities in the management and treatment of those with co-occurring autism and disordered eating. Amongst ED populations, those with autistic traits have been shown to have longer illness duration (Saure et al., 2020), poorer psychosocial outcomes (Nielsen et al., 2022) and typically worse treatment outcomes (Kinnaird et al., 2019). Despite

these adverse outcomes, there remains no clinical guidelines for supporting those with co-occurring autism and EDs (Tchanturia et al., 2020) and little research interesting into improving treatments (Field et al., 2023). It is therefore unsurprising that ED services have been shown to often fail in adapting interventions to meet the needs of those with co-occurring autism (Kinnaird et al., 2019), with clinicians lacking confidence and knowledge in how to best support autistic people with EDs in lieu of evidence-based therapeutic approaches (Kinnaird et al., 2017). Critically, autistic individuals with EDs have also reported difficulties in accessing and engaging in treatment (Kinnaird et al., 2019). This is further complicated by the gender discrepancies within autism diagnoses with females reporting a lack of recognition and diagnosis of their autism leading to misdiagnosis and mistreatment within ED services (Bargiela et al., 2016). An understanding of the association between autism and EDs, and subsequently research into the development of novel of adapted treatment options is therefore imperative.

Autism and AN

One area that has received most attention is that of the well documented association between autism and AN. This association was first observed by Gillberg (1983) and has been replicated across numerous studies (Boltri & Sapuppo, 2021; Huke et al., 2013; Koch et al., 2015; Westwood & Tchanturia, 2017). Whilst less than one percent of females in the general population are autistic (Loomes et al., 2017), up to 35% of females with AN meet the criteria for a diagnosis of autism (Westwood & Tchanturia, 2017). For those with anorexia and autism, a high proportion of individuals only received their autism diagnosis once accessing eating disorders services (Kinnaird et al., 2017).

One possible reason for the relationship between autism and AN is an artefactual association with the effects of starvation producing similar traits to those in autism (Pooni et

al., 2012; Westwood et al., 2016). This suggests that such apparent autistic traits are caused by starvation and therefore do not precede the onset of the eating disorder. In line with this possible causal mechanism, some research has shown that autistic traits are lower in those who have reached weight restoration in AN compared to those who have not (Kerr-Gaffney et al., 2021). In contrast, other studies have shown that autistic traits persist in individuals with AN who have reached weight restoration (Bentz et al., 2017; Nazar et al., 2018). This highlights the importance of longitudinal studies to understand if autistic traits precede the onset of disordered eating and thus whether autism is a risk factor for or an artefactual consequence of AN.

Alternatively, the association may be understood in terms of a conceptual overlap with common traits between autism and AN across social and communicative, sensory and emotion regulation domains (Kinnaird & Tchanturia, 2021). Similarities in cognitive styles has also been proposed to explain the association such as difficulties with set-shifting and weak central coherence (Vagni et al., 2016). Other explanations have considered mechanisms of a genuine association such as AN being a female manifestation of autism (Carpita et al., 2000; Odent, 2010). However, this interpretation is not well supported by the research as it has been shown that autistic males are also at a greater risk of experiencing AN and have lower body-mass index scores compared to neurotypical males (Courty et al., 2013; Sobanski et al., 1999) thus it cannot be concluded that this association is exclusive to females. Perhaps a more credible explanation of a genuine association is that both disorders have common or correlated causal factors including shared genetic and neurobiological factors (Adams et al., 2024).

Autism and other disordered eating

Less research has investigated autistic traits in relation to BN or BED (Halls et al., 2022), although there is some evidence to suggest that these individuals also have greater autistic traits than non-ED comparisons (Gesi et al., 2017). As those with BN and BED typically do not reach the underweight status of AN and therefore do not experience the same effects of starvation, this shows the association is not limited to restrictive EDs or starvation effects. Given that BN and BED account for almost half of ED prevalence rates it is crucial to understand their association with autism and the implications this has for treatment (Hay et al., 2017).

Of the limited research on BN and BED, two studies have found elevated rates of autistic traits in individuals with BN and BED compared to controls in Italian populations (Dell'Osso et al., 2018; Gesi et al., 2017). Two further longitudinal studies have shown conflicting results with Schaumberg et al. (2021) finding an association between autistic traits and BN in females, whereas Martini et al. (2022) did not find a significant association between diagnoses of autism and future BN. Whilst there has been two systematic reviews and a meta-analysis on the association between autism and EDs (Huke et al., 2013; Nickel et al., 2019; Sader et al., 2025), none have examined BN and BED specifically. Given the paucity of research on prevalence rates of co-occurring autism and BN or BED, there is little understanding of what might be driving this association if it exists.

Despite ARFID being a relatively new diagnosis, there is already a clear association with neurodevelopmental conditions described in the literature, particularly with co-occurring autism (Kambanis et al., 2020). In addition to high comorbidity rates, similar traits can be seen across the two conditions including cognitive inflexibility, rigidity relating to food consumption, a need for control and preferences for routine (Bourne et al., 2020). Further, it

has been suggested that sensory sensitivities in autistic children may perpetuate ARFID (Farag et al., 2022).

ADHD and Disordered eating

ADHD has also received attention in the literature for its potential association with disordered eating. ADHD is a neurodevelopmental condition that is characterised by hyperactivity, inattention, and impulsivity (APA, 2013). ADHD has also been shown to be associated with significant physical and mental health difficulties including anxiety, low self-esteem, sleep disorders, and respiratory diseases (Du Rietz et al., 2021; Schatz & Rostain et al., 2006; Travell & Visser, 2016). As discussed below, the gender discrepancies and challenges in achieving a formal diagnosis in autistic people is also experienced with ADHD, thus traits of ADHD, probable ADHD and formal diagnoses of ADHD shall be considered here.

Historically thought to be a disorder of childhood, it is increasingly recognised that ADHD persists throughout adulthood (Agnew-Blais et al., 2016). Whilst some have conceptualised ADHD in adulthood as a continuation of childhood ADHD, recent evidence provides an alternative account of ADHD arising in adolescent or adulthood (Moffitt et al., 2015). Further complicating the understanding of the trajectory of ADHD is the long-standing gender discrepancies and recent increase in ADHD referrals for adult women. Whilst ADHD has historically been thought to be condition that predominately affects males, it has been suggested that these prevalence rates are based on systematic underdiagnosis of female (Young et al., 2020). It has been suggested that this may, in part, be explained by a somewhat different profile of ADHD in females that is less recognised and thus females are often diagnosed with ADHD later in life than males (Martin, 2024). It has been shown that the ratio of male to female diagnosis of ADHD in childhood is around 4:1 and by adulthood becomes

nearer to 2:1 (Martin, 2024). Despite the potential underdiagnosis in females, prevalence rates of ADHD diagnoses have already been shown to be up to 15.5% (Rowland et al., 2015) representing a significant proportion of young people. This, therefore, highlights the importance of longitudinal and trait level research in ADHD, given the risk of underdiagnosis and late diagnosis in females, to fully understand the implications of this condition.

ADHD has also received attention in the ED field as it is suggested to impact on risk of developing disordered eating, presentation, and treatment outcomes (Cortese et al., 2007; El Archi et al., 2020). Studies have shown that up to 20% of people with ADHD may experience disordered eating (Ravi & Khan, 2020). In their systematic review, Nickel et al. (2019) found seven studies exploring the association between ADHD and disordered eating with prevalence rates ranging between 1.6% and 18%, however has many of these studies did not include control groups it is unclear to what extent this differs from those without ADHD. A recent review by Villa et al. (2023) found that most included studies showed associations between disordered eating and ADHD, however as most included studies were cross-sectional it is unclear whether ADHD predates the ED symptoms and thus whether ADHD represents a risk factor for disordered eating.

ADHD and BN/BED

Unlike with autism, there is substantial literature investigating the association between ADHD and BN and BED. Systematic reviews have provided clear evidence of an association between ADHD traits and BN or BED. In their review, Nazar et al. (2016) found that an elevated risk of ADHD in individuals who exhibited binge eating behaviours compared to controls. Similarly, Kaisari et al. (2017) found that ADHD traits are associated with disordered eating behaviours, with impulsivity in particular driving bulimic symptoms. Importantly, this association has been found across genders with both males and females with

BED showing elevated rates of ADHD compared to those without BED (Brewerton & Duncan, 2016).

Whilst these studies provide useful evidence for the association between ADHD and BN and BED, it is important to examine this association from a longitudinal perspective to understand the mechanisms behind this interaction. Several longitudinal studies have provided evidence for ADHD representing a risk factor for future disordered eating by showing this association persisting over time with ADHD predating ED symptoms. ADHD has been shown to increase the risk for future BN (Biderman et al., 2007; Mikami et al., 2010; Yilmaz et al., 2017) with some evidence for an elevated risk of future BED in those with ADHD (Bisset et al., 2019; Martin et al., 2023).

It has been suggested that the core characteristics of ADHD may directly contribute to ED behaviours such as impulsivity driving purging or binge eating behaviours and inattention hindering hunger or satiety cues (Cortese et al., 2007; Ptacek et al., 2016). Further, it has been proposed that emotional dysregulation and executive function difficulties may increase emotional eating as a coping mechanism and contribute to irregular eating patterns (El Archi et al., 2020). Given traits of ADHD may directly influence the development of or presentation of disordered eating (Makin et al., 2025), this shows the importance of understanding this association further to support the development of treatment pathways that do not currently exist.

ADHD and other disordered eating

In contrast to autism research there is little on AN and other restrictive EDs. Whilst some researchers have suggested there may be an association between ADHD and AN (Fernández-Aranda et al., 2013), others have shown that this association is more likely to exist with binge/purging types of AN rather than the restricting type (Svedlund et al., 2017).

Similarly, in their longitudinal studies, Biederman et al. (2007; 2010) found that whilst ADHD increased the risk for a future ED, this was not statistically significant for AN. However, when looking specifically at traits of restrictive eating, Martin et al. (2023) found that inattentive symptoms and hyperactivity/impulsivity traits directly predicted restrictive eating at follow-up. There is a paucity of research on the associations between ADHD and ARFID, however, a recent study by Thomas et al. (2025) has provided initial evidence with their findings of higher levels of ADHD traits in those with higher levels of ARFID symptoms in their gender diverse sample. These mixed findings therefore highlight the possibility of an association between ADHD traits and restrictive EDs warranting further research to fully understand this profile and causal mechanisms.

Intersection of autism, ADHD and EDs

When considering the association between autism or ADHD and disordered eating, it is important to note the high co-occurrence of autism and ADHD as this further complicates current understandings of the relationship between neurodivergence and EDs. It has been suggested that up to approximately 40% of autistic people also have ADHD (Rong et al., 2021), thus when examining the literature on associations of neurodivergence and disordered eating it becomes unclear if we are looking at autism and ADHD exclusively or whether we are in fact looking at co-occurring autism and ADHD. As the previous literature has focused on restrictive EDs in autism and BN/BED in ADHD it remains somewhat unclear what the risk of EDs might be in co-occurring autism and ADHD. There are a small number of studies that have begun to explore this. Karjalainen et al. (2016) found that rates of ED diagnoses were lower in the autism and ADHD group compared to the autism only and ADHD only groups. This finding is interesting as it suggests that the risk of ED may in fact be lower for those with co-occurring autism and ADHD, however, as this was a cross-sectional design it limits our interpretation of these findings. Nonetheless, a more recent longitudinal study by

Christiansen et al. (2024) supported these findings showing that the risk of future disordered eating in those with autism or ADHD was slightly attenuated when adjusting for co-occurring autism and ADHD.

This intersection also needs to be considered in terms of gender differences with each condition manifesting differently across the different genders (Breton et al., 2023; Cruz et al., 2024; Martin, 2024). As noted previously, autism and ADHD are often misdiagnosed or underdiagnosed in females with a lack of recognition of EDs in males. It has been suggested that both autistic males and females are at a greater risk of experiencing disordered eating (Martini et al., 2022), whilst others have found some evidence of gender differences for those with ADHD, Grabarek & Cooper (2008) found that men with ADHD are particularly at risk for restrictive eating disorders whilst females may be more likely to experience symptoms of BN (Mikami et al., 2010). Thus, it is important to gain a further understanding of these associations with disordered eating across different genders.

Rationale and aims

Despite the documented relationship between neurodivergence and disordered eating, progress is still to be made in this field to advance the understanding of these associations. Whilst there is a relative abundance of cross-sectional studies, more longitudinal studies are required to understand the extent to which autism and ADHD predate disordered eating and therefore pose a risk, or whether these associations are better understood by common or correlated causal factors. Furthermore, the literature has focussed on formal diagnoses of neurodevelopmental disorders and eating disorders, rather than on a dimensional approach that reflects contemporary understandings of these conditions (Curzio et al., 2020). In addition, the focus on AN and autism research and BN and BED in ADHD highlights the need for further research across the spectrum of disordered eating in neurodivergence to

ensure that possible associations are not missed. Similarly, research has primarily focussed on individual diagnoses of autism and ADHD and as there is high co-occurrence between the two it will be beneficial to include traits of both to understand specificity and whether there are shared or distinct associations. Further, given the bias against diagnoses across genders in both neurodivergence and EDs, trait level research is imperative to help account for this by ensuring research is not reliant on formal diagnoses when exploring such associations to capture traits in individuals who may not come to clinical attention.

By furthering the understanding of these associations and therefore possible causal mechanisms, this will support the development of more appropriately tailored evidence-based interventions that are sorely needed within the field. Whilst advancements have been made by the PEACE pathway (https://www.peacepathway.org/) to support clinicians in adapting ED interventions to those with autism, further initiatives are clearly warranted across the spectrum of neurodivergence and disordered eating.

Finally, and perhaps most importantly, individuals with lived experience of autism, ADHD and disordered eating have identified priorities for future research including identifying causal mechanisms and improving outcomes (Keller et al., 2024). This this research could support these needs identified by enhancing the current understand of the longitudinal associations between trait level autism, ADHD, and disordered eating. This study therefore aims to address the following questions:

- Are the trajectories of Autism traits (aged seven to 16 years) predictive of disordered eating behaviours at age 18 years?
- Are the trajectories of ADHD traits (aged seven to 16 years) predictive of disordered eating behaviours in adolescence?

- What are the independent contributions of overlapping autistic and ADHD traits to risk of disordered eating behaviours at age 18?
- Are there sex-specific effects in the association between disordered eating and autistic and ADHD traits?

Method

Sample

The present study utilised data from the Avon Longitudinal Study of Parents and Children (ALSPAC) a large population birth cohort of children born in South-West England (Bristol, UK). To participate in ALSPAC individuals were required to have a pregnancy with an expected delivery date falling between 1st April 1991 and 31st December 1992. A total of 14,541 individuals were recruited in the study, with 14,062 live births and 13.988 children alive at one year of age (Boyd et al., 2013; Fraser et al., 2013). The participants have been followed regularly since birth with the use of self-administered questionnaires for the mothers, children, and teachers as well as face-to-face clinic assessments. The cohort is generally considered to be representative of the UK population when the data was collected, a largely White population with diverse socioeconomic backgrounds, with a small proportion of participants (5%) from minority ethnic backgrounds (Washbrook et al., 2013).

In the present study, participants included a subsample from the core ALSPAC sample who were alive at one year of age. Participants whose consent was withdrawn either by the young person, or the mother were removed from the dataset. Participants were required to have at least one measurement of autistic traits and one measurement of Attention Deficit/Hyperactivity Disorder (ADHD) traits between the ages of seven and 16 years old. Participants were also required to have at least one measurement of disordered eating (across fasting, purging and binge eating) at age 18 years. In line with previous research (Bruckauf & Chzen, 2016; Russell et al., 2019), for each set of twins in the sample (n=178), one child was

excluded to guarantee independence, reducing the impact of shared environmental and genetic effects. The study was thus compromised of predictor variables of ADHD traits at age seven, 11, 13 and 16 years, autistic traits at seven, 11, 14, and 16 years with outcome variables of disordered eating assessed at 18 years.

Ethical approval was granted by the ALSPAC Law and Ethics Committee and the Local Research Ethics committee. Further details on the ALSPAC cohort and methodology can be found online (www.bristol.ac.uk/alspac), which also provides details of all the data available through their searchable data dictionary (http://www.bris.ac.uk/alspac/researchers/data-access.data-dictionary/.).

Measures

Autistic traits

Autistic traits were measured using the Social and Communication Disorders Checklist (SCDC), through parent report, at approximate ages of seven, 11, 14, 16 years of age. The SCDC measures traits across social reciprocity and social communication difficulties in the both the general population and in clinical settings (Skuse et al., 2005). Containing 12 items (Likert scale 0 to 2) across communication and behavioural difficulties, parents rate the extent to which these apply to their child on a three-point scale: “not true”, “quite or sometimes true” or “very often true”. A total SCDC score is yielded by summing all items, a maximum score of 24, with higher numbers indicating a greater number of traits present.

The SCDC has consistently been used in literature as a measure of autistic traits and is validated against other clinical measures of autistic traits and diagnosis of autism (Bolte et al., 2011; Carter Leno et al., 2022; Pickard et al., 2017; Solmi et al., 2021). The SCDC is shown to be predictive of autism with sensitivity and specificity scores of 0.88 and 0.91,

respectively, when using a cut of score of ≥9 out of 24 to indicate a probable autism diagnosis (Skuse et al., 2009). The use of gender neutrality across the items helps to minimise gender bias and increase the sensitivity (Schaumberg et al., 2021). The SCDC has been shown to have strong internal consistency (α=.93) and test-retest reliability (*r*=.81), and high heritability in both genders (0.74) (Skuse et al., 2005).

ADHD traits

Traits of ADHD were measured using the Strengths and Difficulties Questionnaire (SDQ; Goodman, 2001) which is comprised of five subscales measuring emotional problems, conduct problems, hyperactivity/inattention, peer relations, and prosocial behaviour. Different formats of the SDQ include parent and teacher report children aged between two and 17 years old and a self-report for young people between the ages of 11 and 17 years old. The SDQ has been shown to have good concurrent and discriminant validity (Lundh et al., 2008; Muris et al., 2003) and moderate test-retest reliability (Yao et al., 2009). Research on the internal consistency of the SDQ has been mixed and is therefore suggested to be utilised for screening purposes only (Mieloo et al., 2012).

In the present study, parent-reported SDQ scores on the hyperactivity/inattention (H/I) subscale were used to measure traits of ADHD at approximately seven, 11, 13 and 16 years of age. The scale is comprised of five questions (Likert scale 0 to 2) with possible answers of "Not true", "Somewhat true" and Certainty true". A total subscale score is calculated on a scale of 0-10 with higher scores indicating a greater level of traits and severity.

Disordered eating behaviours at age 18

Disordered eating behaviours were measured at approximately 18 years of age through self-report of the presence of fasting or purging for weight loss, and binge eating in the previous 12 months. The questions included in the ALSPAC self-report questionnaire are

considered to capture behaviours representative of eating disorders and have been used in numerous previous studies allowing for comparison across research (Carter Leno et al., 2022; Bould et al., 2018; Micali et al., 2017; Solmi et al., 2021).

To identify the presence of disordered eating behaviour, adolescents rated the frequency of fasting ("During the past year, how often did you fast [not eat for at least a day] to lose weight or avoid gaining weight?") or purging ("During the past year, how often did you make yourself throw up [vomit] to lose weight or avoid gaining weight" and "During the past year how often did you take laxatives to lose weight or avoid gaining weight?"). Young people were also asked about binge eating behaviour ("Sometime people will go on an 'eating binge', where they eat an amount of food that most people would consider to be very large, in a short period of time. During the past year, how often did you go on an eating binge?"). If episodes of binge eating were reported, the individual was directed to a follow-up question probing a sense of lack of control during the episode with possible responses of: "Never", "Yes, sometimes, "Yes, usually". Response options for the primary questions on frequency of fasting, purging and binge eating were as follows: "Never", "Less than once a month", "1–3 times a month", "Once a week", "2–6 times a week" and "Everyday". Disordered eating is considered present if these behaviours have occurred at least monthly.

Covariates

The covariates included in the analysis were categorical variables of the child's sex and child ethnicity. Maternal highest level of education was also included which was dichotomised as compulsory education obtained at 16 years of age (CSE, Vocational or O Levels) and non-compulsory education (A-levels and Degree). The analyses were also adjusted for maternal age at birth used as a continuous variable. These covariates were chosen

due to their association with traits of autism, ADHD, and disordered eating (see Appendix 2 for rationale).

Additional sensitivity analyses were also adjusted for the child's cognitive function measured at approximately age 8 years of age using the Wechsler Intelligence Scale for Children total IQ score (WISC-III ^{UK}; Wechsler et al., 1992).

Data processing

Predictor Variables

Autistic traits were transformed into a continuous variable of total score at each time point from the individual SCDC questions. Answers on all 12 items of the SCDC were recoded to allow for a maximum total score of 24. A sum score was calculated across the 12 items along with the number of item-level missing data at each wave. If fewer than six items (i.e. <50% of items) were missing at a time-point, an average score across completed items was calculated and imputed into each missing item to create a total prorated score. If six or more items were missing across the measure the total prorated score would be recoded as missing. If a participant was missing a total prorated score at each of the four time points, they were removed from the sample.

The continuous variables of ADHD traits from the SDQ five-item hyperactivity/inattention scale were already prorated with their average score imputed where two or fewer items were missing. Participants were removed from the sample if they had missing data at each of the four time points.

Outcome variables

For disordered eating, several variables were created for the final analyses, based on the methods used by Solmi et al. (2021). Firstly, an overall binary indicator of whether the

individual did or did not report any disordered eating was created (i.e. any fasting, purging or binge eating occurring at least monthly). Disordered eating was considered present if participants reported these behaviours at least monthly across any measure of fasting, purging or binge eating. A measure of frequency was then created with a three-level categorical variable indicating whether the young adult had (1) no disordered eating behaviour (2) episodes of fasting and/or purging and/or binge eating at least monthly (but less than weekly) and (3) experienced at least weekly episodes of fasting, purging or binge eating. The frequency definitions were modelled based on severity definitions within the DSM-5 criteria of weekly episodes of binge eating, purging, or fasting meeting criteria for BED and BN diagnoses (Solmi et al., 2021).

Additional analyses considered disordered eating behaviours in respect to the type of disordered eating. Initially, binary variables were created for presence or absence of at least monthly fasting, purging (compensatory behaviours of vomiting and laxative use) and binge eating (episodes of binge eating and loss of control during binge eating episodes). For each of these three variables, they were again reduced into three levels of severity with (a) no fasting, purging and binge eating, (2) monthly episodes of fasting, purging and binge eating and (3) at least weekly episodes of fasting, purging and binge eating. A complete breakdown of the disordered eating variables can be found in appendix 3. Participants were required to have data for at least one disordered eating variable to be included in the analysis.

All data processing was conducted in Stata 19 (StataCorp, 2025).

Data analysis

All analyses were conducted in Stata 19 (StataCorp, 2025).

The sample is described in relation to baseline covariates and measures of autistic traits, ADHD traits, and disordered eating. Chi-square tests of independence and independent

sample *t*-tests were completed on missing data to assess whether those who completed the measures of interest differed on baseline characteristics compared to those who had missing data.

Trajectories of autistic traits and ADHD traits over time were modelled using latent growth curve modelling (LGCM), a multivariate structural equation modelling technique which can estimate individuals change across time on one or more outcome variable (Duncan & Duncan, 2004). Latent factors of intercept (baseline) and slope (trajectory) were estimated for the repeated measures of autistic traits and ADHD traits separately. A quadratic term was included into the model for ADHD traits with goodness of fit assessed using structural equation modelling fit indices. These included the Comparative Fit Index (CFI; Bentler, 1990) and Tucker-Lewis Index (TLI, Tucker & Lewis, 1973), the Standardized Root Mean Square Residual (SRMR; Byrne, 1998) and Root Mean Square Error of Approximation (RMSEA; Steiger, 1990). It is suggested that a satisfactorily fitting model should have a CFI and TLI >0.90, a RMSEA ⩽0.05, and <.08 on the SRMR (Hu & Bentler, 1999).

Due to the dispersion of autistic traits and the nature of the data (a count of total traits), a Poisson model was used to estimate latent intercept and slope. This model does not provide indices of goodness of fit so the correlations between actual values and those predicted by the growth curve model were used as an indicator of whether the model specified within the latent growth curve structure is a good approximation of the true data structure.

Factor loadings of time points were specified as time relative from baseline, with the baseline at 7 years of age for autistic traits (0, 1, 1.75, 2.25) and ADHD traits (0, 1, 1.5, 2.25). Residual variances of these observed measures were constrained to be fixed at each time point for ADHD traits (residual variances cannot be constrained in Poisson models).

Once, latent growth curves had been separately estimated for autistic traits and ADHD traits, individual factor scores were extracted and saved. These estimated factor scores were then entered as predictors in logistic regression models to assess their association with disordered eating variables at age 18. Each regression was run as an unadjusted model and then as an adjusted model with covariates entered the model. Binomial logistic regressions were used for all binary disordered eating outcomes presented as Odds ratios (*OR*) with multinomial logistic regressions used for frequency of disordered eating presented as relative risk ratios (*RRR*). First, associations with ADHD and autistic trait factor scores were tested in independent models, then they were included in the same model to test for specificity of effects.

To test for sex specific effects (i.e., moderation), multi-group structural equation models were run, recreating the logistic regressions above, but now grouped by gender. The models were run with estimates constrained to be the same between gender and where coefficients can vary. Testing for significant differences in model fit between the two models shows whether there are significant gender differences in the association between the disordered eating variable and autistic and ADHD traits.

Sensitivity analyses were then run to see if the inclusion of WISC-III^{UK} total IQ scores as an additional covariate to see if the coefficient of effect for autistic and ADHD traits changed.

Results

Sample and missing data

Of the total dataset (*N*=15,645), 13,744 (87.9%) participants remained in the sample who were in the core ALSPAC sample, were alive at one year and, when multiple births occurred, with one twin/triplet retained. Of these, 9,208 (67.0%) had at least one SCDC

measurement. A total of 4,322 (46.9%) had SCDC data across all four time points, 1,796 (19.5%) at three time points, 1,492 (16.2%) at two time points and 1,598 (17.4%) at one time point. Chi-square tests of independence showed there were no significant differences across gender in those with and without any SCDC measurements with 4,526 females (49.0%) with at least one SCDC measurement and 2145 (47.3%) females without any SCDC measurement, χ^2 (1, N = 13,744) = 3.75, p = .053. There was significant difference in ethnicity between those who had SCDC measurements (7.5% non-white participants) and those without any SCDC measurements (4.1% non-white participants), χ^2 (1, N = 11,878) = 55.69, p<.001. The two groups also significantly differed on level of maternal education, χ^2 (1, N = 12,288) = 346.50, p<.001, with 22.0% of Mothers of those without SCDC measurements completing non-compulsory education compared to 40.1% in those with an SCDC measurement. An independent samples *t*-tests showed that there was a significant difference in maternal age at the birth of their cohort child between those who completed a SCDC measurement (M = 28.8, SD = 4.7) and those who did not (M = 26.3, SD = 5.1), t(13,742) = -28.36, p<.001. The two groups also significantly differed on mean WISC total IQ score with those completing the measures having a higher mean WISC IQ score (M = 104.8, SD = 16.4) compared to those without any SCDC measurements (M = 95.42, SD = 15.7), t(6924) = -11.50, p<.001.

For the SDQ H/I measurements, a total of 9,370 (68.2%) had at least one measurement. Of these, 4,226 (45.1%) had completed the SDQ at all four time points, 1,915 (20.4%) at three time points, 1,345 (14.4%) at two time points and 1,884 (20.1%) at one time point. There was a significant difference of gender for those who had an SDQ measurement (49.2% female) and those who did not have any SDQ measurements (46.8% female), χ^2 (1, N = 13,744) = 7.13, p = .008. There was also a significant difference in ethnicity between those who had SDQ measurements (4.0% non-white participants) and those without any SDQ measurements (8.0% non-white participants), χ^2 (1, N = 11,878) = 73.20, p<.001. The two

groups also significantly differed on level of maternal education, χ^2 (1, N = 12,288) = 328.60, p < .001, with 22.0% of Mothers of those without SDQ measurements completing non-compulsory education compared to 39.8% in those with an SDQ measurement. There was a significant difference in maternal age between those who completed a SDQ measurement (M = 28.8, SD = 4.7) and those who did not (M = 26.2, SD = 5.2), t(13,742) = -28.71, p < .001. The two groups also significantly differed on mean WISC total IQ score with those completing the measures having a higher mean WISC IQ score (M = 104.7, SD = 16.4) compared to those without any SDQ measurements (M = 96.98, SD = 15.9), t(6924) = -9.53, p < .001.

There was a total of 3,179 (23.1%) participants with at least one measurement of disordered eating. Those with and without any disordered eating measures differed significantly on gender with 43.8% female in those without any measurements compared to 64.1% female in those with any completed measurement, χ^2 (1, N = 13,744) = 406.83, p < .001. The two groups were also significantly different on ethnicity for those without completed measurements (5.5% non-white) and those with completed measurements (3.9% non-white), χ^2 (1, N = 11,878) = 11.89, p = .001. There was also a significant difference for maternal education levels with 30.0% of those without disordered eating measurements having mothers who completed non-compulsory education compared to 50.2% of those with completed measurements, χ^2 (1, N = 12,288) = 418.05, p < .001. There was a significant difference in maternal age between those who had completed disordered eating measurements (M = 29.6, SD = 4.6) and those who did not (M = 27.5, SD = 5.0), t(13,742) = -20.95, p<.001. The two groups also significantly differed on mean WISC total IQ score with those completing the measures having a higher mean WISC IQ score (M = 108.5, SD = 16.3) compared to those without any disordered eating measurements (M = 101.7, SD = 16.1), t(6924) = -16.86, p < .001.

A total of 8,676 (63.1%) had at least one SCDC measurement and one SDQ measurement. Of these, 3,014 (34.7%) participants had data available on at least one measurement of disordered eating at age 18 years which compromised the final sample and were thus included in the analyses (See Appendix 4 for flowchart of participation). Of those with at least one SDQ and one SCDC measure, 99.7% (*n* = 3,005) had data on fasting. Across the two questions relating to purging behaviours, 99.7% (*n* = 3,004) had data on vomiting for weight loss with 4.4% (*n* = 131) having data available for medication use. In response to the questions around binge eating, 99.8% (*n* = 3,009) had data on the frequency of binge eating episodes with 39.6% (*n* = 1,193) having data available on whether they felt out of control during a binge eating episode.

Descriptive statistics

Within the final sample, almost 64% were female with approximately 94% of the sample being of white ethnicity (see Table 1). Just over half of the participants had mothers who completed non-compulsory education (50.5%) with the greatest number of mothers being between the ages of 26 and 35 (72.9%). Total WISC IQ scores (see Table 2) were higher for males (*M* = 109.82, *SD* = 17.25) compared to females (*M* = 107.90, *SD* = 15.57) at eight years of age.

Table 1

Sample characteristics (N = 3014)

Baseline characteristics	***N***	**%**
Child's sex		
Female	1916	63.6
Male	1098	36.4
Child's ethnicity		
White	2829	93.9
Ethnic minority	104	3.5

Not known	81	2.7
Maternal education		
Compulsory	1448	48.0
Non-compulsory	1521	50.5
Not known	45	1.5
Maternal age at delivery		
16 - 19	35	1.2
20 - 25	481	16.0
26 - 35	2196	72.9
36 - >43	302	10.0

Table 2

Mean total IQ scores from the WISC-III^{UK}

	Age (years)			WISC-III^{UK} Total IQ score		
	N (%)	**M (SD)**	**Range**	**N (%)**	**M (SD)**	**Range**
Female	1605 (53.3%)	8.59 (.23)	8.08 – 10.33	1585 (52.6%)	107.90 (15.57)	57 – 145
Male	945 (31.4%)	8.57 (.23)	8.17 – 10.33	936 (31.1%)	109.82 (17.25)	45 - 159

WISC-III^{UK}, Weschler Intelligence Scale for Children – Third Edition UK version

Males had higher SCDC scores at age seven years (M = 2.99, SD = 3.83) compared to females (M = 2.16, SD = 2.81). For males there was a decrease in mean SCDC scores over time before rising again at age 16. Females mean SCDC scores also appeared to reduce at age 11 before rising again at 14 and 16 years of age (see Table 3). For SDQ hyperactivity/inattention scores, males also had a higher mean score at age seven (M = 3.46, SD = 2.37) compared to females (M = 2.78, SD = 2.10). For both males and females, mean SDQ scores decreased at age 11, increasing at age 14 before decreasing again at age 16. (see Appendix 5 for box plots of median SCDC and SDQ scores).

Table 3

Mean SCDC and SDQ scores

	Total		Female		Male	
	n (%)	*M (SD)*	*n* (%)	*M (SD)*	*n* (%)	*M (SD)*
SCDC measurements						
Age 7	2,772 (92.0%)	2.46 (3.25)	1,739 (57.7%)	2.16 (2.81)	1,033 (34.3%)	2.99 (3.83)
Age 11	2,753 (91.3%)	2.04 (3.17)	1,718 (57.0%)	1.81 (2.75)	1,035 (34.3%)	2.41 (3.75)
Age 14	2,708 (89.9%)	2.15 (3.22)	1,689 (56.0%)	2.10 (3.03)	1,019 (33.8%)	2.24 (3.50)
Age 16	2,576 (85.5%)	2.54 (3.53)	1,593 (52.9%)	2.59 (3.49)	983 (32.6%)	2.46 (3.60)
SDQ H/I measurements						
Age 7	2,786 (92.4%)	3.04 (2.23)	1,753 (58.2%)	2.78 (2.10)	1,033 (34.3%)	3.46 (2.37)
Age 11	2,723 (90.4%)	2.40 (2.02)	1,714 (56.9%)	2.14 (1.90)	1,009 (33.5%)	2.84 (2.14)
Age 13	2,700 (89.6%)	2.50 (2.04)	1,678 (55.7%)	2.24 (1.92)	1,022 (33.9%)	2.93 (2.15)
Age 16	2,568 (85.2%)	2.25 (1.99)	1,591 (52.8%)	2.09 (1.90)	977 (32.4%)	2.51 (2.11)

SCDC, Social and Communication Disorders Checklist; SDQ H/I, Strengths and Difficulties Questionnaire – Hyperactivity/Inattention subscale.

For the disordered eating variables at age 18 years, 342 (11.4.%) of females reported at least one disordered eating behaviour with 155 (5.1%) reporting monthly behaviours and 187 (6.2%) reporting any weekly disordered eating behaviour. In comparison, 59 (2.0%) males reported any disordered eating with 27 (0.9%) reporting monthly behaviours and 32 (1.1%) reporting weekly disordered eating behaviours in the previous year. Across individual

types of disordered eating, females had a greater frequency of fasting, purging and binge eating episodes compared to males (see Table 4). A full breakdown of disordered eating by type can be found in appendix 6.

Table 4

Proportion of any disordered eating within the total sample

	Binary		**Frequency**		
	Absence	**Presence**	**Absence**	**Monthly**	**Weekly**
	***n* (%)**	***n* (%)**	***n* (%)**	***n* (%)**	***n* (%)**
Total	2,613	401	2,613	182	219
	(86.7%)	(13.3%)	(86.7%)	(6.0%)	(7.3%)
Female	1,574	342	1,574	155	187
	(52.2%)	(11.4%)	(52.2%)	(5.1%)	(6.2%)
Male	1,039	59	1,039	27	32
	(34.5%)	(2.0%)	(34.5%)	(0.9%)	(1.1%)

Latent trajectories of ADHD traits

The LGCM of ADHD traits included an intercept, linear and quadratic slope terms. The RMSEA score was 0.056 with a CFI of 0.995 and TLI of 0.990 which indicates a satisfactory fit. The SRMR value was 0.017 again indicating a good fit of the model. The overall trajectory of ADHD traits remained relatively stable across time (Mean linear slope = .009). See appendix 7 for further results of the trajectory.

Latent trajectories of Autistic traits

As the autistic trait data was a count of symptoms and then heavily skewed, a Poisson model was used to estimate developmental trajectories. A negative binomial model was also

specified to account for any additional overdispersion, but this led to issues with model convergence, so the Poisson model was used in the final model. Similarly, it was attempted to include a quadratic term, but this meant the model did not convergence, so just the intercept and linear slope were estimated. As this model does not provide goodness of fit statistics the correlation between actual values and the values predicted by the growth curve model can be used as an indicator whether the model specified within the latent growth curve structure is a good approximation of the data structure in real settings. For this model, as correlations are high and the residuals are low (near zero) this would indicate a good approximation. The correlations ranged from 0.88 to 0.95 with residuals ranging from 0.11 to 0.23. Like the trajectory of ADHD traits, autistic traits remained relatively stable over time (Mean linear slope = .0001). See appendix 7 for further results of the trajectory.

Logistic regression models: ADHD traits

Any Disordered eating

In the unadjusted model, there was no association between the binary disordered eating behaviour variable at age 18 and intercept, slope, or quadratic factors of ADHD traits. There remained no significant associations when adjusting for possible covariates. See table 5 for all regression results for any disordered eating at age 18 years.

For the multinomial regression of the frequency of disordered eating, there were no significant associations found in the unadjusted model (see table 5). Within the adjusted model with the possible covariates included, the intercept was associated increased risk of weekly disordered eating (RRR = 1.14, 95% CI [1.03, 1.25], p = .010) but not monthly disordered eating behaviours (RRR = 0.96, 95% CI [0.86, 1.08], p = .524) at age 18. There were no significant associations between slope and quadratic factors with monthly or weekly disordered eating in the adjusted model (see Table 5).

Table 5

Binomial and Multinomial logistic regression results for ADHD growth curve parameters and any Disordered eating at age 18

ADHD traits trajectory factor	Any disordered eating at 18 years		Frequency of any disordered eating at 18 years			
	Any Unadjusted model OR	Any adjusted model OR	Monthly Unadjusted model RRR	Weekly Unadjusted model RRR	Monthly adjusted model RRR	Weekly adjusted model RRR
Intercept	1.01 (0.94, 1.09), *p* = .773	1.06 (0.98, 1.14), *p* = .148	0.91 (0.82, 1.02), *p* = .109	1.09 (0.99, 1.19), *p* = .066	0.96 (0.86, 1.08), *p* = .524	1.14 (1.03, 1.25), *p* = .010
linear slope	0.93 (0.70, 1.23), *p* = .620	0.91 (0.68, 1.22), *p* = .527	0.80 (0.53, 1.22), *p* = .312	1.03 (0.72, 1.48), *p* = .856	0.81 (0.53, 1.25), *p* = .346	0.99 (0.68, 1.43), *p* = .944
quadratic slope	0.85 (0.39, 1.87), *p* = .681	00.72 (0.31, 1.63), *p* = .428	0.58 (0.18, 1.88), *p* = .365	1.12 (0.41, 3.06), *p* = .832	0.51 (0.15, 1.71), *p* = .273	0.92 (0.32, 2.64), *p* = .880

OR = Odds ratio, RRR = relative risk ratio, Multivariate model including covariates of gender, ethnicity, maternal education and maternal age.

Disordered eating variables by type

For the absence or presence of fasting at age 18 years there was no significant association between ADHD traits in the unadjusted models or adjusted models (see Table 6). There were also no significant associations with frequency of fasting behaviours at age 18 in any of the unadjusted or adjusted multinomial regression analyses.

Similarly to fasting, there was no significant associations between latent factors of ADHD traits and any purging behaviours at age 18 years (see Table 6). There were also no significant associations with frequency of purging behaviours at age 18 across both the unadjusted and adjusted models.

For binge eating, there was no significant effect of ADHD trait trajectory terms for any binge eating behaviours at age 18 for both the unadjusted and adjusted models. When looking at frequency of binge eating behaviours at age 18, the intercept was associated with weekly binge eating (RRR = 1.17, 95% CI [1.04, 1.32], p = .012) behaviours but not monthly behaviours. This significant association remained when adjusting for covariates (RRR = 1.21, 95% CI [1.07, 1.38], p = .003).

Table 6

Binomial and Multinomial logistic regression results for ADHD growth curve factors and Disordered eating by type

ADHD traits trajectory factor	**Any Fasting at 18 years**		**Frequency of fasting at 18 years**			
	Any unadjusted model OR	**Any adjusted model OR**	**Monthly Unadjusted model RRR**	**Weekly Unadjusted model RRR**	**Monthly adjusted model RRR**	**Weekly adjusted model RRR**
Latent intercept	1.04 (0.93, 1.16), p = .501	1.10 (0.98, 1.24), p = .103	0.98 (0.84, 1.15), p = .830	1.08 (0.94, 1.25), p = .263	1.05 (0.88, 1.24), p = .610	1.15 (0.99, 1.33), p = .077
Latent slope	0.96 (0.63, 1.46), p = .831	0.99 (0.64, 1.55), p = .995	0.88 (0.47, 1.65), p = .691	1.08 (0.58, 1.79), p = .950	0.95 (0.50, 1.80), p = .864	1.04 (0.58, 1.86), p = .895
Latent quadratic	0.91 (0.28, 2.98), p = .874	0.86 (0.25, 2.97), p = .816	0.84 (0.15, 4.82), p = .842	0.97 (0.20, 4.67), p = .968	0.82 (0.14, 5.02), p = 0.833	0.90 (0.18, 4.58), p = .894
	Any purging at 18 years		**Frequency of purging at 18 years**			
Latent intercept	0.98 (0.86, 1.12), p = .784	1.03 (0.90, 1.19), p = .658	0.98 (0.77, 1.24), p = .831	0.98 (0.84, 1.15), p = .836	1.00 (0.78, 1.29), p = .998	1.05 (0.89, 1.23), p = .603
Latent slope	1.01 (0.61, 1.67), p = .976	0.99 (0.59, 1.68), p = .978	1.06 (0.43, 2.59), p = .905	0.99 (0.54, 1.80), p = .962	1.01 (0.40, 2.54), p = .987	0.98 (0.53, 1.84), p = .960
Latent quadratic	1.30 (0.32, 5.31), p = .712	1.12 (0.26, 4.89), p = .877	2.06 (0.17, 24.81), p = .570	1.07 (0.20, 5.73), p = .939	1.78 (0.13, 23.69), p = .663	0.92 (0.16, 5.34), p = .929
	Any binge eating at 18 years		**Frequency of binge eating at 18 years**			
Latent intercept	1.05 (0.96, 1.15), p = .276	1.09 (0.99, 1.19), p = .076	0.95 (0.85, 1.08), p = .449	1.17 (1.04, 1.32), p = .012	0.99 (0.87, 1.12), p = .862	1.21 (1.07, 1.38), p = .003

Latent slope	1.02 (0.72, 1.44), p = .927	0.97 (0.67, 1.38), p = .858	0.91 (0.57, 1.45), p = .690	1.14 (0.70, 1.86), p = .588	0.86 (0.53, 1.40), p = .554	1.10 (0.66, 1.81), p = .725
Latent quadratic	1.07 (0.41, 2.81), p = .888	0.90 (0.33, 2.), 45p = .831	0.83 (0.22, 3.06), p = .774	1.42 (0.36, 5.59), p = 614	0.64 (0.17, 2.49), p = .521	1.30 (0.31, 5.40), p = .720

OR = Odds ratio, RRR = relative risk ratio, Multivariate model including covariates of gender, ethnicity, maternal education and maternal age.

Logistic regression models: Autistic traits

Any Disordered eating

In the unadjusted model, there were significant associations between the binary variable (absence or presence) of any disordered eating behaviours at age 18 and intercept (OR = 1.18, 95% CI [1.08, 1.30], p = .001) and slope (OR = 1.73, 95% CI [1.28, 2.35], p < .001) of autistic traits. This association remained significant when adjusting for possible covariates. See Table 7 for all regression results.

For the multinomial regression of the frequency of disordered eating at age 18 years with autistic trajectory factors, the intercept (RRR = 1.26, 95% CI [1.11, 1.44], p < .001) and linear slope (RRR = 2.06, 95% CI [1.39, 3.05], p < .001) were associated with weekly disordered eating but not monthly behaviours in the unadjusted model. The significant associations remained when covariates were entered into the model.

Table 7

Binomial and Multinomial logistic regression results for latent Autism growth curve factors and any Disordered eating at age 18

Autism traits trajectory factor	**Any disordered eating at 18 years**		**Frequency of any disordered eating at 18 years**			
	Any Unadjusted model OR	**Any adjusted model OR**	**Monthly Unadjusted model RRR**	**Weekly Unadjusted model RRR**	**Monthly adjusted model RRR**	**Weekly adjusted model RRR**

Latent intercept	1.18 (1.08, 1.30), p = .001	1.21 (1.09, 1.34), p < .001	1.09 (0.96, 1.25), p = .193	1.26 (1.11, 1.44), p < .001	1.11 (0.96, 1.28), p = .145	1.30 (1.14, 1.48), p < .001
Latent slope	1.73 (1.28, 2.35), p < .001	1.52 (1.11, 2.08), p = .009	1.40 (0.90, 2.17), p = .136	2.06 (1.39, 3.05), p < .001	1.22 (0.78, 1.93), p = .376	1.81 (1.21, 2.71), p = .004

OR = Odds ratio, RRR = relative risk ratio, Multivariate model including covariates of gender, ethnicity, maternal education, and maternal age.

Disordered eating variables by type

For the absence or presence of fasting at age 18 years there was a significant association between intercept (OR = 1.22, 95% CI [1.05, 1.41], p = .008) and linear slope (OR = 1.70, 95% CI [1.07, 2.67], p = .025) of autistic traits trajectory in the unadjusted model (see Table 8). The significant association of latent intercept persisted in the adjusted model (OR = 1.25, 95% CI [1.07, 1.45], p = .005), however the latent slope became not significant. The intercept was also associated with weekly fasting behaviours at 18 years, but not monthly behaviours, in the unadjusted (RRR = 1.29, 95% CI [1.06, 1.56], p = .012) and adjusted (RRR = 1.32, 95% CI [1.08, 1.62], p = .008) multinomial analyses.

For any purging behaviours at 18 years, there was a significant association between the linear slope and fasting in both the unadjusted (OR = 2.23, 95% CI [1.31, 3.81], p = .003) and adjusted models (OR = 2.06, 95% CI [1.19, 3.55], p = .010) (see Table 8). This association of slope was also found with weekly purging in both the unadjusted (RRR = 2.64, 95% CI [1.41, 4.95], p = .001) and adjusted (RRR = 2.48, 95% CI [1.30, 4,71], p = .006) models, but no significant association was found for monthly purging behaviours.

For binge eating, there was a significant association between intercept and any binge eating behaviours at 18 years in both the unadjusted (OR = 1.18, 95% CI [1.05, 1.33], p = .005) and adjusted models (OR = 1.20, 95% CI [1.06, 1.36], p = .004) (see Table 8). For the frequency of binge eating behaviours, the intercept was associated with weekly, but not

monthly, binge eating in the unadjusted model (RRR = 1.35, 95% CI [1.14, 1.62], p = .001) which persisted in the adjusted model (RRR = 1.36, 95% CI [1.13, 1.64], p = .001).

Table 8

Binomial and Multinomial logistic regression results for latent Autism growth curve factors and Disordered eating by type

ADHD traits trajectory factor	Any Fasting at 18 years		Frequency of fasting at 18 years			
	Any unadjusted model OR	Any adjusted model OR	Monthly Unadjusted model RRR	Weekly Unadjusted model RRR	Monthly adjusted model RRR	Weekly adjusted model RRR
Latent intercept	1.22 (1.05, 1.41), p = .008	1.25 (1.07, 1.45), p = .005	1.15 (0.93, 1.41), p = .206	1.29 (1.06, 1.56), p = .012	1.16 (0.93, 1.45), p = .182	1.32 (1.08, 1.62), p = .008
Latent slope	1.70 (1.07, 2.67), p = .024	1.51 (0.94, 2.41), p = .087	1.73 (0.89, 3.36), p = .104	1.66 (0.90, 3.07), p = .104	1.53 (0.77, 3.01), p = .223	1.50 (0.80, 2.81), p = .207
	Any purging at 18 years		**Frequency of purging at 18 years**			
Latent intercept	1.14 (0.96, 1.35), p = .138	1.16 (0.97, 1.39), p = .119	1.19 (0.88, 1.61), p = .269	1.12 (0.91, 1.37), p = .298	1.17 (0.86, 1.61), p = .323	1.14 (0.92, 1.42), p = .228
Latent slope	2.23 (1.31, 3.81), p = .003	2.06 (1.19, 3.55), p = .010	1.51 (0.57, 3.99), p = .410	2.64 (1.41, 4.95), p = .002	1.33 (0.49, 3.61), p = .578	2.48 (1.30, 4.71), p = .006
	Any binge eating at 18 years		**Frequency of binge eating at 18 years**			
Latent intercept	1.18 (1.05, 1.33), p = .005	1.20 (1.06, 1.36), p = .004	1.07 (0.91, 1.25), p = .410	1.35 (1.14, 1.62), p = .001	1.09 (0.93, 1.28), p = .293	1.36 (1.13, 1.64), p = .001
Latent slope	1.40 (0.95, 2.04), p = .086	1.24 90.84, 1.82), p = .287	1.25 (0.76, 2.06), p = .388	1.62 (0.93, 2.80), p = .089	1.09 (0.66, 1.83), p = .733	1.45 (1.13, 2.55), p = .198

OR = Odds ratio, RRR = relative risk ratio, Multivariate model including covariates of gender, ethnicity, maternal education, and maternal age.

Multivariate logistic regression models: ADHD and Autism

Any Disordered eating.

When variables for the trajectories of autism traits and ADHD were entered into the regression model with covariates included, there was a significant association of autistic traits

intercept (OR = 1.22, 95% CI [1.09, 1.38], p = .001) and linear slope (OR = 1.70, 95% CI [1,21, 2.39], p = .002) with any disordered eating at 18 years (see Table 9). All adjusted models will be presented here, for results of the unadjusted regression results please see appendix 8. There was some evidence to suggest the ADHD traits slope was negatively associated any disordered eating behaviours (OR = 0.73, 95% CI [0.54, 1.01], p = .055). In the multinomial regression there was a significant association of autistic traits intercept (RRR = 1.26, 95% CI [1.08, 1.48], p = .004) and slope (RRR = 1.97, 95% CI [1.28, 3.05], p = .002) on weekly disordered eating behaviour at age 18. There was some evidence to suggest the intercept of autistic traits was associated with monthly disordered eating behaviours (RRR = 1.18, 95% CI [1.00, 1.40], p = .051).

Table 9

Binomial and Multinomial adjusted logistic regression results for latent ADHD and Autism growth curve factors and any Disordered eating at age 18

Latent Trajectory factor	**Any disordered eating at 18 years**	**Frequency of any disordered eating at 18 years**	
	Any OR	**Monthly RRR**	**Weekly RRR**
ADHD intercept	0.96 (0.88, 1.06), p = .436	0.89 (0.78, 1.02), p = .102	1.02 (0.91, 1.15), p = .730
ADHD slope	0.73 (0.54, 1.01), p = .055	0.70 (0.44, 1.11), p = .124	0.76 (0.51, 1.13), p = .177
ADHD quadratic	0.46 (0.19, 1.08), p = .075	0.37 (0.10, 1.30), p = .122	0.53 (0.18, 1.58), p = .252
Autism Intercept	1.22 (1.09, 1.38), p = .001	1.18 (1.00, 1.40), p = .051	1.26 (1.08, 1.48), p = .004
Autism Slope	1.70 (1.21, 2.39), p = .002	1.42 (0.88, 2.31), p = 0.153	1.97 (1.28, 3.05), p = .002

OR = Odds ratio, RRR = relative risk ratio, Multivariate model including covariates of gender, ethnicity, maternal education, and maternal age.

Disordered eating variables by type.

When entering latent factors for trajectories of both autistic traits and ADHD traits, there was some evidence to suggest there is an association with fasting at age 18 years with the autistic traits intercept predicting fasting behaviours (OR = 1.23, 95% CI [1.02, 1.47], p = .028) (see table 10). When looking at frequency of fasting behaviours there was no evidence to suggest an association between autistic and ADHD traits with monthly fasting at 18 years. There was some evidence of weekly fasting with the autistic traits intercept somewhat associated with weekly fasting (RRR = 1.27, 95% CI [0.99, 1.62], p = .058).

For purging behaviours there was a significant association between the autistic traits slope and future purging (OR = 2.36, 95% CI [1.31, 4.24], p = .004). There was no significant association between trajectories of autistic traits and ADHD traits with monthly purging at 18 years. There was some evidence to suggest an association with weekly purging with a significant association between the autistic traits slope and future purging behaviours (RRR = 2.86, 95% CI [1.43, 5.71], p = .003).

There was some evidence to suggest an association with future binge with the intercept of autistic traits associated with binge eating at age 18 years (OR = 1.18, 95% CI [1.02, 1.36], p = .029). When looking at frequency of binge eating behaviours, there were no significant associations with monthly binge eating. The autistic traits intercept was however associated with weekly binge eating (RRR = 1.25, 95% CI [1.00, 1.56], p = .046).

Table 10

Binomial and Multinomial adjusted logistic regression results for latent ADHD and Autism growth curve factors and Disordered eating by type

Latent Trajectory factor	**Any Fasting at 18 years**	**Frequency of Fasting at 18 years**	
	Any	**Monthly**	**Weekly**

	OR	RRR	RRR
ADHD intercept	1.01 (0.88, 1.15), $p = .948$	0.97 (0.79, 1.18), $p = .729$	1.04 (0.86, 1.24), $p = .702$
ADHD slope	0.82 (0.51, 1.31), $p = .400$	0.77 (0.38, 1.53), $p = .454$	0.86 (0.46, 1.60), $p = .624$
ADHD quadratic	0.57 (0.16, 2.08), $p = .395$	0.52 (0.80, 3.46), $p = .502$	0.61 (0.11, 3.38), $p = .573$
Autism Intercept	1.23 (1.02, 1.47), $p = .028$	1.18 (0.91, 1.53), $p = .214$	1.27 (0.99, 1.62), $p = .058$
Autism Slope	1.61 (0.97, 2.67), $p = .068$	1.68 (0.81, 3.50), $p = .162$	1.55 (0.78, 3.06), $p = .209$
	Any purging at 18 years	**Frequency of purging at 18 years**	
ADHD intercept	0.93 (0.79, 1.10), $p = .401$	0.89 (0.66, 1.21), $p = .464$	0.95 (0.78, 1.15), $p = .594$
ADHD slope	0.72 (0.41, 1.26), $p = .255$	0.82 (0.30, 2.23), $p = .702$	0.68 (0.35, 1.32), $p = .261$
ADHD quadratic	0.55 (0.12, 2.53), $p = .442$	1.19 (0.8, 17.98), $p = .900$	0.40 (0.06, 2.43), $p = .317$
Autism Intercept	1.21 (0.98, 1.50), $p = .082$	1.29 (0.88, 1.89), $p = .187$	1.17 (0.91, 1.51), $p = .231$
Autism Slope	2.36 (1.31, 4.24), $p = .004$	1.51 (0.52, 4.38). $p = .447$	2.86 (1.43, 5.71), $p = .003$
	Any binge eating at 18 years	**Frequency of binge eating at 18 years**	
ADHD intercept	1.02 (0.91, 1.14), $p = .787$	0.94 (0.81, 1.09), $p = .415$	1.11 (0.95, 1.29), $p = .206$
ADHD slope	0.86 (0.59, 1.26), $p = .440$	0.79 (0.47, 1.33), $p = .373$	0.94 (0.55, 1.61), $p = .812$
ADHD quadratic	0.71 (0.25, 2.04), $p = .526$	0.55 (0.13, 2.22), $p = .394$	0.95 (0.21, 4.23), $p = .950$
Autism Intercept	1.18 (1.02, 1.36), $p = .029$	1.12 (0.93, 1.36), $p = .217$	1.25 (1.00, 1.56), $p = .046$
Autism Slope	1.29 (0.85, 1.96), $p = .231$	1.20 (0.69, 2.08), $p = .513$	1.43 (0.78, 2.63), $p = .250$

OR = Odds ratio, RRR = relative risk ratio, Multivariate model including covariates of gender, ethnicity, maternal education, and maternal age.

Gender differences

Gender differences were assessed within a multi-group structural equation model framework, specifying autistic traits and ADHD traits intercept and slope terms as predictors of each disordered eating outcome. When the models were compared against coefficients constrained to be equal across genders and then allowed to vary, there were no significant differences in model fit. This was true for all disordered eating outcome models (see Table 11). Thus, there was no evidence of moderation of associations by gender.

Table 11

Significance levels for sex-specific effects across multi-group structural equation models

Disordered eating variable	Significance level
Any disordered eating	$p = .8481$
Fasting	$p = .4139$
Purging	$p = .2908$
Binge eating	$p = .6539$

Sensitivity analyses

The WISC-III^{UK} total IQ score completed at age 8 years of age was entered into the analyses for both autistic traits and ADHD traits. When entering the total IQ score there were no changes in the associations between autism and ADHD traits and any disordered eating across the binomial and multinomial analyses. There were also no changes across all analyses for fasting and purging behaviours. For binge eating, the significant association between autistic traits intercept and any binge eating behaviours became not significant. Similarly, the association between autistic traits intercept and weekly binge eating behaviours also become not significant.

Discussion

This study aimed to construct developmental trajectories of traits of ADHD and autism across childhood and evaluate their association with disordered eating behaviours at 18 years of age. By modelling trajectories of ADHD and autistic traits across childhood and adolescence it was possible to show how these levels of traits predate the onset of disordered eating and thus may present as a risk factor future disordered eating. This provides some evidence of the direction of causality between childhood ADHD and autistic traits and later disordered eating. Whilst one cannot fully exclude the possibility of artefactual associations or reverse causation, it is unlikely as eating disordered behaviours are rare at age seven (Solmi et al., 2021). There was also evidence to suggest a dose-response relationship with an association between higher trait levels and more frequent disordered eating behaviours.

Summary of main findings

The LGCM for ADHD traits showed that these remain relatively stable over childhood and adolescence suggesting trait levels at age 7 are an indication of trait levels at age 16. Whilst controlling for possible covariates (gender, ethnicity, maternal level of education, maternal age at birth of child), higher levels of ADHD traits at age seven were associated with an increased risk of reporting weekly disordered eating behaviours at age 18 years. When looking at individual types of disordered eating behaviours, no evidence was found for an association between the level of ADHD traits and fasting or purging behaviours. This result is somewhat surprising as there has been an increasing evidence base to suggest that traits of ADHD are associated with restrictive eating disorders (Bleck et al., 2015; Kaisari et al., 2018). There was however an association with weekly binge eating with baseline traits of ADHD increasing the risk for more frequent binge eating at age 18 years. This supports the existing evidence base that has consistently shown a link between ADHD and binge eating (Cortese et al., 2007; Appolinario et al., 2024).

The LGCM for autistic traits also showed that these traits remain relatively stable from age seven to 16. There was an association between autistic traits and all disordered eating behaviours captured in this study. There is an overall increased risk of any disordered eating and evidence to suggest that both higher baseline (age 7 years) levels of autistic traits and a steeper increase in traits between age 7 and 16 years were associated with more frequent disordered eating at age 18 years. This fits with previous research suggesting there is an association between autism and all forms of disordered eating when these are measured at age 16 (Solmi et al., 2021). When considering the different types of disordered eating and their frequency, greater autistic trait levels at baseline were associated with a greater risk of any fasting at age 18 years as well as more frequent fasting. This supports the abundance of research evidencing an association between autism and restrictive forms of disordered eating like AN of which fasting can be a characteristic of (Vagni et al., 2016; Westwood et al., 2016; Westwood and Tchanturia, 2017). The linear slope of autistic traits was associated with the presence and frequency of purging behaviours with a steeper increase in traits between the ages of seven and 16 being associated with a higher likelihood of experiencing weekly disordered eating at age 18. Whilst often seen across forms of AN, purging behaviours are representative of BN which has previously been shown to be associated with autistic traits (Makin et al., 2025). Higher levels of autistic traits at age seven were also associated with a higher likelihood and frequency of binge eating behaviours at age 18. Again, this fits with previous research that has shown significantly elevated autistic traits in individuals with BN and BED of which binge eating can be characteristic of (Gesi et al., 2017).

In the full multivariate model which included both ADHD and autistic traits as independent variables (and thus accounting for the likely overlap in the two domains of traits), both the starting level of autistic traits and change across time were informative on disordered eating outcomes. When looking at any disordered eating at age 18, both the

baseline autistic traits and change in traits across time were associated with any disordered eating behaviours being reported. There was also an effect on the frequency of disordered eating with autistic traits at age seven being associated with a higher likelihood of experiencing both monthly and weekly disordered eating behaviours. The linear slope also provided evidence that a steeper increase in traits up to age 16 years is associated with future weekly disordered eating behaviours. The is some evidence from the ADHD linear slope that there is a decreased risk of disordered eating behaviours, however as this was on the threshold level of significant it is not conclusive and would warrant further research. Nonetheless as this is a scarcity of prior research evaluating the impact on disordered eating across traits of autism and ADHD this provides some useful initial evidence.

Significant associations with future disordered eating were also found across the presence of different types of disordered eating behaviours and their frequency. Higher baseline autistic traits were shown to associated with a greater likelihood of fasting behaviours at age 18 years with some evidence to suggest that this is also associated with weekly behaviours. Like the findings of purging when looking at autistic traits in isolation there was an association between the change in traits over time with a steeper increase in traits associated with a greater likelihood of any purging and weekly frequency. Finally, greater autistic traits at age seven are also associated with any binge eating behaviours and weekly occurrence at age years. These findings are consistent with those reported by Christiansen et al. (2024) that co-occurring autism and ADHD are associated with an increased risk across the spectrum of disordered eating. The results for autistic traits suggest a lack of specificity in effects regarding the type of disordered eating as associations were found with the domains of fasting, purging and binge eating, Whilst for ADHD traits only an effect on binge eating was found when looking at individual types of disordered eating behaviours which was then not found within the multivariate analysis. This suggests that

unmeasured autistic traits might in part account for observed associations between ADHD and disordered eating. This has important implications for the field as it highlights the importance of recognising co-occurring autistic traits when trying to find effects specific to ADHD. Further as this study has shown an association with more frequent disordered eating behaviours this may in part fit with prior research that has suggested that those with autism and/or ADHD often present to eating disorder services with increased illness severity (Makin et al., 2025; Zhang et al., 2022)

The multi-group models that sought to understand if any neurodivergent-disordered eating associations were moderated by gender did not find evidence of significant moderation effects. This suggests that within this sample these associations exist across both genders, although as there was only a small proportion of males who reported disordered eating behaviours at age 18 it is possible this study was underpowered to detect any effects of gender. This would however fit with previous research that has shown both autistic males and females have an elevated risk of experiencing AN (Martini et al., 2022). Conversely, other research has shown that females with ADHD are more likely to experience symptoms of BN compared to males (Mikami et al., 2010) with an associated between ADHD and restrictive eating disorders found particularly in males (Grabarek & Cooper, 2008). Thus, there is still a lack of understanding how these associations may exist across genders especially for those with co-occurring autism and ADHD. However, it could be argued that more recent cohorts may have a better gender ratio as the recognition of ADHD and autistic traits in females has increased since the commencement of ALSPAC.

Strengths, limitations, and future research directions

To my knowledge this is one of the first studies exploring the longitudinal associations of disordered eating behaviours with traits of both autism and ADHD. Whilst

there is a substantial evidence base on the association between autism and disordered eating, in particular restrictive eating disorders, and a growing evidence base on the association of ADHD and disordered eating, there has been a scarcity of research considering the impact of co-occurring autism and ADHD on disordered eating behaviours, despite the well high documented rates of co-occurrence (Rong et al., 2021). Further compared to previous cross-sectional or case-control studies, the longitudinal nature of this study provides some insight into the temporality of these associations which is an essential step in understanding causal mechanisms.

A strength of this study is the use of trait level scores across all variables of autism, ADHD, and disordered eating. Whilst it is suggested that diagnoses of eating disorders are relatively uncommon within the community and thus difficult to capture within cohort-based studies due to their complexity in presentation (Solmi et al., 2021), this did however provide opportunity to evaluated disordered eating behaviours that are more prevalent within community settings. These behaviours measured map onto behaviours commonly seen across formal diagnoses of eating disordered and thus provide a broader perspective on associations with autism and ADHD in individuals who may not reach clinical services.

Despite these strengths there are several important limitations that warrant discussion. Although the study had a large sample size there was a low prevalence of individuals with high traits scores for both the autism and ADHD measures. This may have limited statistical power to detect effects of these predictors on future disordered eating. This overdispersion also impacted on the approach taken to analyse the autism trajectories as it was not possible to include a quadratic slope of change for autistic traits which may have more accurately captured the shape of change over time. It would therefore be useful to replicate this work with the inclusion of a greater number of individuals reporting higher scores across measures of autism and ADHD traits or alternatively with measures that may better capture population-

level variability in traits rather than focusing on the more severe manifestations of autistic traits. A comparison of these associations between those with low and high scores would help to understand the extent to which these associations exist across different levels of traits and therefore whether the risk of disordered eating increases as the numbers of traits increases.

Similarly, as is typical in any longitudinal cohort study, the sample was subject to high levels of attrition which was compounded by extending the measurements of disordered eating behaviours to age 18 building on the previous work by Solmi et al. (2021). Whilst efforts were taken to reduce the impact of attrition through the inclusion of participants who had at least one measurement across all variables, as opposed to complete data sets, and imputation of missing data, it is possible this may have impacted the construction of the latent trajectories and thus the results from the regression analyses. As identified in the analyses of missing data there were significant differences across baseline covariates between those who did or did not complete the measures of interest. Thus, this may limit the generalisability of the results as it is possible attrition was selective which would bias the results.

This study was also not able to explore whether there are specific traits or characteristics of autism and ADHD that predict disordered eating. This may help to understand the underlying causal mechanisms behind these associations which would be important for clinical applications. Across neurodivergence and disordered eating there are several shared characteristics such as cognitive flexibility, impulsivity, and sensory differences (Kinnaird & Tchanturia, 2021). Thus, incorporating measures of disordered eating behaviours that can assess these concepts would be helpful to consider whether there are particular traits that underlie these associations rather than broadly looking at overall traits levels when there is a great diversity in the spectrum of traits neurodivergent individuals may experience. This approach may help to ascertain whether certain neurodivergent are more at risk of disordered eating.

Further, it would be important to utilise tools that are able to capture other forms of disordered eating, such as with the relatively new diagnosis of Avoidant/Restrictive Food Intake Disorder, that were less researched and/or understood at the time of the development of the ALSPAC study design and thus not captured within this study. Given the documented association between autism and ARFID (Sader et al., 2025) it would be imperative to utilise psychometric tools such as that by Bryant-Waugh et al. (2019) to evaluated longitudinal associations in this population.

This study is also limited in its ability to understand gender differences amongst these associations as in comparison to the females, there was a relatively small proportion of males who reported disordered eating behaviours at age 18 years. This will have limited the statistical power to detect associations in this group. Just as there are reported to be differences in the way that females present with autism and ADHD (Brown & Stokes, 2020; Young et al., 2020), it has also been suggested that there are differences in how disordered eating may present in males. For example, it has been reported that males may have different body image concerns with greater focus around muscularity driving different forms of weight-controlling behaviours compared to females (Neumark-Sztainer et al., 2014). It is therefore possible that the measures of disordered eating in this current study could not capture the extent of possible disordered eating behaviours amongst males with their focus on fasting and purging for weight loss. It would therefore be important for future research to incorporate measures that are able to capture other forms of disordered eating behaviours not examined in this study and therefore increase the understanding of the gender differences within the associations this study has attempted to explore.

Finally, one must consider the generalisability of this research. This study was comprised of a predominately White British sample and thus limits its application across different ethnicities and cultures. This is of particular importance as ethnic minorities are

historically underrepresented across eating disorder research (Egbert et al., 2024). Further to the historical lack of cultural understanding and racial bias in the recognition of disordered eating, this has also been the case in neurodivergence research. It has been reported that individuals from minority ethnic and racial backgrounds are often underrepresented within such research contributing to a lack of awareness of cultural views and understandings of neurodivergence and related constructs (Martin et al.,2025; Merrill et al., 2024). Whilst misunderstandings of the nature of neurodivergence or unfamiliarity with such clinical diagnoses have been suggested to contribute to discrepancies in diagnosis rates across cultures (Kooij et al., 2010), it is also important to consider non-Western understandings in the methodological development of research to ensure alternative perspectives are accounted for. A greater understanding of different cultural views on neurodivergence would support the use of culturally sensitive screening tools which would be essential for future research with more diverse samples (Huda et al., 2024).

Clinical implications

The trait level approach taken in this study has allowed for an exploration of disordered eating behaviours that may commonly be seen within the community. As these individuals may not reach thresholds for clinical services this may support in increasing the awareness of the risk of disordered eating behaviours, particularly for autistic children, in community settings. Specifically, children with high autistic traits in childhood and those with increasing traits into adolescence might be vulnerable to experiencing disordered eating. Thus, early screening for disordered eating behaviours may be appropriate in those identified as exhibiting traits of autism and possibly ADHD. As neurodivergence, particularly autism, has been associated with adverse treatment outcomes of eating disorders (Nielsen et al., 2015) early screening for disordered eating behaviours in individuals with traits of autism

and/or ADHD and thus early intervention may in part contribute to a positive shift in outcome for this cohort.

It will also be pertinent for professionals within clinical services for eating disorders to not overlook the possible presence of traits of autism and ADHD. Whilst it has been suggested that the effects of starvation, often seen within restrictive disordered eating, can produce features reminiscent of autistic traits (Pooni et al., 2012; Westwood et al., 2016) this study provides further evidence that autistic traits can predate the onset of disordered eating. These associations therefore cannot be assumed to be artefactual and may warrant further investigation to ensure clinicians are able to distinguish between autism and disordered eating related behaviours to provide appropriate therapeutic intervention (Field et al., 2023).

Similarly, for ADHD traits, an exploration of the presence of such traits and the impact this may have on the development of disordered eating behaviours such as binge eating would be crucial in clinical services. Understanding how an individual's hyperactivity/inattention contributes to their disordered eating would be essential to facilitate the process of regaining control within treatment (Cortese et al., 2007). Further, when an individual has co-occurring traits of autism, this research provides evidence to suggest that the risk of disordered eating may extend to fasting and purging behaviours. As such clinicians should consider a thorough assessment across the spectrum of feeding and eating related difficulties.

Together this provides further indication that the development of guidance for the treatment of disordered eating in the context of neurodivergence would be beneficial for clinicians and individuals within this cohort. There have been useful advances in the development of guidance for the treatment of AN for autistic people by The Pathway for Eating Disorders and Autism developed from Clinical Experience (PEACE pathway;

Tchanturia, 2021; Tchanturia et al., 2020). However, it would be imperative for further developments in this area to ensure there are clear guidelines for supporting those with autism and ADHD across the spectrum of disordered eating.

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Part 3: Critical Appraisal

Introduction

This critical appraisal outlines my reflections on the research process. It begins with a consideration of the influences drawn from my clinical practice before moving to my reflections on the decision-making and challenges within the systematic review, and a discussion of the difficulties encountered in conducting the empirical study. I then reflect on common themes that arose across both parts. My thoughts on implications for future research and clinical practice are integrated throughout the appraisal.

Influences from clinical practice

Having worked with children with neurodevelopmental conditions prior to training, I witnessed the significant challenges faced by these young people and their needs going unmet by services. Thus, I shared the frustrations of other clinicians and services in feeling deskilled and lacking the resources needed for optimal care. I observed these children being stuck between services, with neurodevelopmental teams unable to meet their mental health needs, and vice versa. My experience led me to appreciate the importance of understanding the co-occurring mental health needs of young people with neurodevelopmental conditions to provide appropriate screening and early intervention to mitigate against potential future risks to their wellbeing. This has fuelled a desire to focus on the development of my leadership skills during training to enable me to improve access to holistic support for this cohort.

Whilst completing the research project I worked in a specialist inpatient eating disorder (ED) service for adolescents and young adults. Witnessing these associations I was researching in a clinical setting, this provided valuable insights into how this research would impact the care and treatment of these young people. Most patients had either a suspected or confirmed diagnosis of autism or attention-deficit/hyperactivity disorder (ADHD) meaning I was able to consider the impact of this on the development and maintenance of their ED,

formulating their difficulties within the context of neurodivergence. I observed how differences in the cognitive and behavioural characteristics of their ED had made traditional evidence-based therapies ineffective, leading to multiple unsuccessful inpatient admissions. This experience informed my approach to the research, ensuring that I grounded the study in the needs of these young people.

Reflections on the systematic review

Designing the review and research questions

The first challenge within the research process was deciding the systematic review topic. I looked to reviews on the association between disordered eating with autism (Huke et al., 2013; Inal-Kaleli et al., 2025) and ADHD (Kaisari et al., 2017) to help me generate ideas and identify gaps in the literature. Whilst these reviews found increased prevalence rates of autism and ADHD in ED populations, most included studies were cross-sectional and thus do not evidence how these associations exist over time. These reviews also highlighted a focus on the association between autism and anorexia nervosa (AN), with other EDs overlooked. Further, despite the high co-occurrence between autism and ADHD (Hours et al., 2022) they are often considered in isolation. Whilst two reviews (Makin et al., 2025; Nickel et al., 2019) did consider both autism and ADHD, the included studies focussed on either autism *or* ADHD, highlighting the need for a review positioning these as co-occurring conditions. Another area of consideration was the gender disparities in diagnoses which is discussed later in the critical appraisal. It therefore felt important to explore trait-level research to account for the possibility of gender-based underdiagnosis, differing from previous research and reviews that required a formal diagnosis of such disorders. A longitudinal approach also felt important to understand these associations over time and mechanisms driving these associations.

Although the rationale for my systematic review was based on the gaps identified in previous reviews, the scope I initially proposed was overly broad, and hence potentially outside the remits of a doctoral thesis. On reflection, two separate reviews may have been more appropriate, with one focusing on longitudinal associations and another on the causal mechanisms underlying these processes. However, it felt important to include both aspects within the review as although it is an understanding of the causal mechanisms that will aid our approach to treatment, this becomes less relevant if we still lack a comprehensive understanding of the extent to which these associations exist.

The next challenge, therefore, was deciding on the review's inclusion and exclusion criteria. Whilst my research question aimed to be broad and inclusive to gain a representative understanding of the associations and underlying causal mechanisms, I had to narrow the focus to keep within the remit possible for a thesis. To include causal mechanisms, I therefore decided to only include studies meeting all the criteria for the initial research questions regarding longitudinal associations. This reduced the number of causal mechanism-focused studies in the review but felt necessary to ensure that the overall focus of the paper remained on the longitudinal nature of these associations.

The quality assessment

The next challenge of the systematic review was deciding on the quality assessment tool. Whilst this would not directly influence the studies selected for the narrative synthesis, a study's quality does impact the interpretation of its results and potential conclusions. For this review, it was important to seek a quality assessment tool that could evaluate the reliability and validity of longitudinal studies. However, when scoping the different tools, it became apparent that many have criteria on the use of a control group within longitudinal study designs which I had not required to be present within the inclusion criteria of the current

review. This led me to consider whether studies examining traits of autism and ADHD could be seen as including a kind of "control" group, since they often include participants with a full range of scores on the measurement tools. In other words, these studies typically include individuals with no neurodivergent traits as well as those whose scores exceed the thresholds for autism and/or ADHD diagnoses. Although these groups may not be directly compared against one another, the studies still effectively capture both the presence and absence of these traits.

Previous systematic reviews on longitudinal associations used a vast array of tools, either adapting a known tool to fit the research question, developing their own, or not reporting on quality assessment at all. This led me to conclude that an existing tool may be sufficient with adaptations allowing me to tailor it to the review aims, whilst also allowing for some consistency across future research using the same tool. Two potentially useful tools identified were the Critical Appraisal Skills Programme (CASP) for cohort studies checklist (CASP, 2025) and the Newcastle-Ottawa Scale (NOS) for cohort studies (Wells et al., 2012). Although the CASP provides a thorough list of questions to evaluate each study, this results in an appraisal of "positive/methodologically-sound" or "negative/relatively poor" methodology. This felt quite difficult to present in the review, as some studies were likely to have a combination of positive and negative features. Thus, discussing the quality of each study within the review would require a comprehensive breakdown of each study's evaluation to highlight the pertinent parts when interpreting the results. On the other hand, the NOS is a briefer tool that uses a star system with criteria on when and how to apply a star to each question. This felt less subjective and clearer to present, which would allow readers to see the full breakdown and thus enable a broader look at the independent elements of the appraisal rather than an overall judgement of the quality. I therefore decided to tailor the NOS to fit with the reflections I raised earlier. This experience allowed me to reflect on the role of the

researcher during the research process, the different perspectives that may influence decision-making, and the importance of minimising researcher bias. To aid these decisions I found it invaluable to discuss my thought processes with my thesis supervisors, allowing for alternative perspectives.

Reflections on the empirical paper

Data access and transformation

One of the major challenges faced during the thesis process was accessing the dataset. To address the research questions, I felt that using data from the Avon Longitudinal Study of Parents and Children (ALSPAC) would be preferable. However, gaining access to this dataset proved difficult and lengthy.

Unlike projects involving data recruitment, secondary data analysis presents different challenges, namely being reliant on others' timelines and uncertainty concerning variables available to access. Due to unforeseen circumstances, access to the dataset was gained late in the thesis process which limited the thesis to the variables provided with little flexibility on accessing additional variables due to the timeframe.

Another challenge when using secondary data is the lack of control over the format in which variables are received. This resulted in a time-consuming process of cleaning and transforming the data and creating new variables for the analysis. Using unfamiliar software added the challenge of learning and understanding new commands and writing code. Since this project aimed to further the work of Solmi et al. (2021), the construction of these variables needed to align with their methodology. For the autistic traits measure, I therefore had to create a total prorated score from individual questions on the Social Communication Disorders Checklist (SCDC; Skuse et al., 2005). Within the dataset, the SCDC score at age 7 was provided as a total prorated score, thus, the challenge was to ensure consistency across

the development of the variable at other ages ranges. Specifically, it was important to manage missing data in the same way and develop syntax to complete this, which proved complicated.

Creating binary and categorical disordered eating variables was also challenging. Fortunately, the fasting variable consisted of one question, making it simpler to recode into a binary variable (presence or absence of fasting) and a second three-level variable reflecting none, monthly or weekly fasting. However, the purging and binge-eating variables were harder to create, with each construct measured across two questions of differing formats. Here, I deviated from Solmi et al.'s (2021) approach. The creation of variables in my project needed to allow for the exploration of the distinct types of disordered eating. This created a dilemma between simplifying variable creation to align with the project's aims and following previous work's methodology to facilitate cross-research comparison. As this project used data from age 18 and had greater attrition rates, it felt best to create variables in a way that maximised data inclusion, even if this required a deviation in approach.

Data analysis

When planning the analysis, I had to consider how best to appropriately investigate the impact of autism, ADHD, and their co-occurrence in research. Firstly, as the prevalence of co-occurring autism and ADHD is suggested to be as high as 70% (Hours et al., 2022), I had to consider how studies examining autism or ADHD in isolation disentangle this overlap. Whilst some studies on the association between autism or ADHD and disordered eating have considered the rates of the other diagnosis in their demographics, others have not. Similarly, some studies include the other as a covariate in their analysis but, again, this is inconsistent across research. This complicates the ways in which we can explore their association with future disordered eating, making it difficult to determine how much autism or ADHD each

contribute and whether this differs for those with both. Feasible options included treating each as covariates within the analysis or, as attempted in this paper, examining each individually and then assessing how the model changes when both are included. Given these high co-occurrence rates, one could however argue that separating these into distinct diagnoses is not essential and taking a transdiagnostic approach to analyses is an appropriate alternative, especially since neurodevelopmental conditions share traits and features (Asaria, 2025). As this research aims to deepen our understanding of the associations with EDs and inform clinical interventions, both approaches offer meaningful insight into individual experiences and risk of disordered eating across the neurodivergence spectrum.

As is often the case in research, there was unfortunately a series of challenges within the analysis of the data due to overdispersion of autistic traits. Whilst all attempts were made to follow the proposed design and methodology of this thesis, the research needed to be led by the data rather than fitting the data to a predefined design. This resulted in a last-minute change in approach and methodology to find an appropriate way of exploring the aims of the study without compromising the validity of the research. Whilst this was an incredibly difficult position to be in to complete the thesis for submission it did provide me an opportunity to think about the utility of different statistical approaches and the importance of research integrity.

Common themes across the thesis

The spectrum of feeding and eating related difficulties

A challenge across both the systematic review and empirical paper was deciding how broadly to go within the spectrum of disordered eating. While this thesis aimed to be as inclusive as possible regarding autism, ADHD and disordered eating, certain aspects within

each area felt contradictory to my aim and there also existed limitations posed by the data available for the empirical study.

Although pica and rumination disorder are included in the DMS-5 chapter of feeding and eating disorders (American Psychiatric Association; APA, 2013), I excluded these from the systematic review search. Whilst there is a known association between pica and autism (Fields et al., 2021), its causes, features and prevalence remain unknown (Delaney et al., 2015). Some evidence suggests that pica may be caused by biological factors including iron- and micronutrient-deficiencies, with evidence of its occurrence among pregnant women (Borgna-Pignatti & Zanella, 2016). Similarly, there is a dearth of research on the epidemiology of rumination disorder as well as complexity surrounding the diagnosis given its similarities with conditions such as gastroparesis, gastroesophageal reflux disease, and bulimia nervosa (BN; Delaney et al., 2015). It therefore felt unhelpful to include these diagnoses in the review as clinically they appear different to the other included forms of disordered eating. The decision to include avoidant/restrictive food intake disorder (ARFID) within the present review may be challenged based on this rationale, as its behavioural and cognitive aspects also differ from other forms of disordered eating, with less focus placed on weight and body image. On reflection, if completing this research again, I would consider including these disorders to maximise inclusivity across disordered eating. However, stringent criteria during full-text screening would be necessary to ensure that studies on the potential biological or health correlates of pica and rumination disorder are excluded during the screening process.

I also decided to exclude obesity studies from the systematic review. Since obesity is not included in the feeding and eating disorders category in the DSM-5 (APA, 2013), this was an easier decision. However, it has been suggested that obesity and EDs are intrinsically linked, with shared risk and maintenance factors documented (Camacho-Barcia et al., 2024).

Further, there is growing evidence of increased ED prevalence in people living with obesity, particularly across individuals with BN or binge eating disorder (BED; Camacho-Barcia et al., 2024). Nonetheless, obesity can arise from a complex interplay of biological and behavioural determinants (Masood & Moorthy, 2023), meaning that the potential benefits and implications of the inclusion of obesity studies within ED research must be considered. On one hand, given the distinct similarities in appetite dysregulation and body image concerns across obesity and EDs, including obesity studies in ED research could be relevant. Conversely, other contributing factors for obesity must not be ignored as co-occurrence with EDs does not necessarily mean these conditions are related. Overall, as this review focussed on the association between neurodivergence and disordered eating, it was deemed unnecessary to include studies on obesity as it is not considered an ED. An alternative approach could have been to include obesity studies where participants also exhibit ED traits, to ensure that EDs in individuals living with obesity are not overlooked.

Regarding the empirical paper, deciding which disordered eating behaviours to include was restricted by the available ALSPAC data. The data related to possible disordered eating behaviours included questions on fasting, exercise frequency, dieting, purging, and binge eating episodes. As this empirical paper aimed to continue the work of Solmi et al. (2021), it felt important to use the same variables for continuity. However, some differences in the variables used and how to include these were made. Firstly, I decided to exclude variables on dieting despite its association with future weight-control and eating pathology (Liechty & Lee, 2013; Neumark-Sztainer et al., 2007). Like my reflections regarding the inclusion of obesity, dieting behaviours could have several possible explanations unrelated to disordered eating, such as health or performance-related reasons (Ashton et al., 2015). Whilst it is important to recognise behaviours that may indicate underlying disordered eating, the responses to dieting questions in such a large cohort study do not provide any contextual

understanding, thus I decided to exclude these variables. For similar reasons, questions regarding the use of exercise with the aim to lose weight were also excluded. Again, though these could also indicate disordered eating-related behaviours, the lack of context meant it did not feel appropriate to include them in the current study. Future research would benefit from examining these variables as mediating or causal mechanisms of disordered eating. However, this was outside the current study's remit.

The spectrum of neurodivergence

This thesis focused on autism and ADHD, specifically aiming to examine their co-occurrence and how this influenced the presence of disordered eating. However, there are numerous other forms of neurodivergence, including Tourette Syndrome, dyslexia, dyspraxia, and dyscalculia (Cobbaert et al., 2024). Further, it has been suggested that the neurodivergence umbrella could also constitute other neurocognitive variations, including psychotic disorders and obsessive-compulsive disorder due to similar sensory, social-emotional processing, inner speech, and movement differences (Cobbaert et al., 2024). Though there is a greater pool of research on autism and ADHD, some initial studies have shown associations between Tourette syndrome and disordered eating (Bamigbade et al., 2022; Smith & Ludlow et al., 2022). The paucity of research on other forms of neurodivergence and EDs means the possible mechanisms underlying these associations remains unknown, highlighting the need for further research in this area. As it has been shown that autistic people and those with ADHD have poorer ED treatment outcomes (Cobbaert et al., 2024), it is important to understand whether those with other forms of neurodivergence experience the same adverse outcomes. It is therefore imperative for services to understand the impact of numerous types of neurodivergence within their interventions, ensuring that these are tailored to those with needs different to autism and ADHD.

Whilst it was outside the scope of this thesis to consider all forms of neurodiversity, it does bring into question the benefits and drawbacks of looking at a transdiagnostic view of neurodivergence rather than distinct areas of difference. A transdiagnostic method may endorse neurodiversity-affirming practice and research (Fletcher-Watson, 2022) with questions around how well diagnostic taxonomies of neurodevelopmental conditions support our understanding of these experiences and their distinction from mental health conditions (Morris et al., 2025). Although neurodivergent individuals can experience elevated rates of mental health conditions, this approach is based on the concept that neurodiversity is a part of human diversity and may manifest to varying degrees (Morris et al., 2025). Thus, the association between neurodivergence and mental health is complex with significant variation in the severity, presentation, and impact. Therefore, rather than having distinct diagnostic categories, this approach captures the shared features and co-occurrence between neurodevelopmental conditions, allowing for structure across the dimensions and underlying characteristics that may contribute to the development of challenges, such as disordered eating (Morris et al., 2025).

On the contrary, there have been interesting developments in disorder-specific research and intervention pathways. For example, the Pathway for Eating Disorders and Autism developed from Clinical Experience (PEACE) has produced guidelines and treatment for AN, tailored for autistic people (Tchanturia et al., 2020). This pathway focusses on the individualised strengths and challenges in respect to autistic traits to ensure that adjustments are made to ED interventions. Initial evaluations of this approach have shown reduced treatment durations (Tchanturia et al., 2021) and increased understanding of the effectiveness of specific interventions for this cohort (Dandil et al., 2020; Tchanturia et al., 2016).

Thus, whilst moving towards a transdiagnostic model may be optimal for neurodivergent-affirmative practices, there may also be benefits to exploring distinct

neurodevelopmental conditions to gain a comprehensive understanding of their contribution to EDs and resulting appropriate treatment options. I hope this thesis contributes to the understanding of the impact of autism and ADHD, and their co-occurrence, on the development of disordered eating. However, it would be favourable to consider other neurodevelopmental conditions within future ED research as well as transdiagnostic approaches to neurodivergence within the field.

Ethnicity

During initial scoping of the existing research relevant to this thesis, a paucity of literature on the associations between neurodivergence and EDs amongst minority ethnic groups became apparent. This led me to consider the impact of ethnicity and how this is considered in my own research and the wider field. Our research cannot be considered generalisable if we cannot show that these associations exist across populations nor can its impact on individuals from other ethnic groups or cultures be determined. Whilst individuals from minority ethnic groups also experience disordered eating, it has been suggested that these individuals are more likely to be misdiagnosed or underdiagnosed (Asari, 2025) and are often overlooked within research (Keski-Rahkonen & Mustelin, 2016). Furthermore, as definitions of typical and atypical behaviour varies across cultures, and as certain constructs including neurodivergence may be interpreted differently across societies, any consideration of ethnicity needs to be rooted in an understanding of culture. Thus, as a White British researcher examining associations in a UK population from a western understanding of neurodivergence and disordered eating, it could be considered inappropriate for me to make conclusions about the impact of ethnicity on these associations.

Nonetheless, it is still important to discuss the different ways in which ethnicity was considered throughout the research process and how this could be improved in the future.

Firstly, during the systematic review, I decided to only include studies published in English due to not having resources for translating research published in other languages. Without these resources, there is a risk of misinterpreting results and making inappropriate conclusions, hence impacting the review's usefulness. However, this limits opportunities to explore distinct cultural understandings of the associations between neurodivergence and EDs as most included studies were from western populations. Thus, it would be beneficial for this research to be replicated in the future with the inclusion of non-English language papers to allow us to better understand the cross-cultural translation of these concepts. Applied to clinical practice, this would increase cultural awareness and improve the support provided to individuals from different backgrounds, especially in services serving diverse local communities.

A consideration when completing the systematic review was the use of diagnostic tools that are typically developed in western countries and may not have been validated across cultures. This may contribute to the underdiagnosis of neurodivergence and EDs in practice and impact on how research evaluates these traits. Hopefully, including any validated measurement tool of autism, ADHD, and disordered eating, and focussing on trait-level symptoms rather than formal diagnoses helped to minimise the risk of bias against minority ethnic groups. However, it is important to acknowledge that it may have been helpful to explore the application of these measures across cultures to understand their appropriateness in this research.

Another challenge to consider was the use of the ethnicity variable within the empirical dataset as ethnicity was reduced to a binary variable of either White or non-White. Whilst I do not wish to endorse this reductive division of ethnicity, I could not control this variable without having access to the original answers to this question. Based on the local population of the ALSPAC study, which is largely White, one can only assume that this

distinction was made due to significantly small numbers of participants from different ethnic groups. Indeed, to be able to use ethnicity as a covariate it may have only been possible to use these wider categories to ensure enough power for detecting significant effects. Nonetheless, this impacts on the inferences we can make on the impact of ethnicity in these associations and thus compromises the study's generalisability.

Gender differences

Another theme that occurred throughout the project was challenges in understanding gender diversity within the field. Understanding the associations between autism, ADHD and disordered eating is complicated by the gender biases in diagnosis. Autistic females are less likely to be diagnosed with autism than males (Loomes et al., 2017) and are typically referred for an assessment later in life (Giarelli et al., 2010). It has been purported that females with ADHD have often been underdiagnosed due to a lack of recognition and, thus, referral for assessment (Young et al., 2020). These studies highlight inaccuracy of the view that both autism and ADHD have a greater male preponderance. Some explanations for this have been that the diagnostic criteria and measurement tools are based on male presentations of these differences and as such do not capture the female phenotype (Brown & Stokes, 2020; Young et al., 2020). Conversely, research has shown that males are often overlooked within ED research (Murray et al., 2017), resulting in a misrepresentation of prevalence rates of disordered eating across genders. This affects research in this area as one could suggest that we are not able to fully understand the associations between neurodevelopmental conditions and disordered eating with such gender biases. This thesis has attempted to begin overcoming this by focussing on trait-level research rather than formal diagnoses, as well as longitudinal data, to capture individuals who may not have been able to reach services and achieve a formal diagnosis during childhood.

It is also important to consider the current conceptualisation of gender as a non-binary construct. Historically within research, and within this thesis, gender is dichotomised into a binary construct of male or female. However, this does not reflect current societal understandings of gender. Within this thesis, the term gender is used to refer to sex assigned at birth. However, it is acknowledged that this does not accurately portray a person's gender identity. Across both the systematic review and empirical paper, I was limited by using the variables denoted within the individual datasets and could not deconstruct this into a more representative form of individuals' preferred gender identity. This raises concern around how we, as researchers, are representing our participants. Whilst one could understand the use of a binary construct within research to provide a universal understanding that can be translated across studies, this is not affirmative of individuals who do not align with their sex assigned at birth. Thus, this brings into question the utility of research exploring gender differences that do not account for the reality of participants' gender preferences. Furthermore, as there is an emerging evidence-base to suggest higher rates of gender diversity amongst neurodivergent people (Warrier et al., 2020), it is pertinent to consider the impact of this within research. It would therefore be beneficial to replicate previous research surrounding the associations between autism, ADHD and disordered eating considering the entire gender spectrum, rather than using binary variables.

A missing voice: the role of experts by experience in research

One aspect of this thesis that felt particularly missing was the voice of experts by experience (EbE). Though an initial aim of the thesis, it was unrealistic to incorporate co-production due to time limits. Despite the call for increased involvement of EbEs in the production of research, this has rarely been used within studies on EDs (Musić et al., 2022). As neurodivergent people experience the world differently across thinking and learning styles, sensory processing, and executive functioning (Cobbaert et al., 2024), it would be

invaluable to have EbEs insights in the research process to support the understanding of the associations with disordered eating.

Although it would have been optimal to incorporate co-production, this thesis is rooted in the needs and wishes of the community for future research. In consultation with the neurodivergent community, several recommendations have been identified as priorities for ED research clustered into two main themes: identifying causal mechanisms and improving outcomes (Keller et al., 2024). The need for further research into the associations between ADHD and disordered eating has been identified and this thesis has contributed to this priority. As there is a wider existing literature base on AN, it has also been recommended that research broadens its scope to others forms of disordered eating (Herle et al., 2025), which this thesis also addresses. Whilst the exploration of possible causal mechanisms has been a small part of the literature review, some clear avenues for future research have been identified. To conclude, this thesis has attempted to take a broad and inclusive approach to understanding the associations between autism and ADHD with disordered eating establishing a more comprehensive picture of the current evidence-base. It is hoped that this can pave the way for future research on producing clinical guidelines and tailored treatment approaches to optimise the outcomes for this population.

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Appendices

Appendix 1: **Newcastle-Ottawa Quality Assessment Scale (NOS) for Cohort Studies**

NEWCASTLE - OTTAWA QUALITY ASSESSMENT SCALE

COHORT STUDIES

Note: A study can be awarded a maximum of one star for each numbered item within the Selection and Outcome categories. A maximum of two stars can be given for Comparability

Selection

1) Representativeness of the exposed cohort

a) truly representative of the average autistic person/person with ADHD in the community *****

b) somewhat representative of the average autistic person/person with ADHD in the community *****

c) selected group of patients e.g. hospitalised individuals

d) no description of the derivation of the cohort

2) Selection of the non-exposed cohort

a) drawn from the same community as the exposed cohort *****

b) drawn from a different source

c) no description of the derivation of the non-exposed cohort

3) Ascertainment of exposure

a) secure record (e.g. health records) *****

b) structured interview or standardised measure *****

c) self-report

d) no description

4) Demonstration that outcome of interest was not present at start of study

a) yes *****

b) no

Comparability

1) Comparability of cohorts on the basis of the design or analysis

a) study controls for age, gender assigned at birth *****

b) study controls for any additional factor *****

Outcome

1) Assessment of outcome

a) independent blind assessment/structured interview or validated measure *****

b) record linkage *****

c) self-report

d) no description

2) Was follow-up long enough for outcomes to occur

a) yes (≥1 year and measured during a developmentally appropriate period) *****

b) no (<1 year)

3) Adequacy of follow up of cohorts

a) complete follow up - all subjects accounted for *****

b) subjects lost to follow up unlikely to introduce bias: number lost ≤ 20 % to follow up, or description provided of those lost suggesting no different from those followed *****

c) follow up rate < 80% and no description of those lost

d) no statement

Appendix 2: Choice of covariates

The analyses were adjusted for the child's sex as traits of autism and ADHD are purported to differ across males and females as well as across time (Mandy et al., 2018; Murray et al., 2018) with disordered eating being more prevalent in females (Micali et al., 2017). Analyses were also adjusted for maternal age and highest level of education as proxies for socio-economic status which is evidenced to be associated with autistic traits (Russell et al., 2014), ADHD traits (Spencer et al., 2022) and disordered eating (Larsen et al., 2018). Ethnicity was also entered into the analyses given the identified disparities in the diagnosis of autism (Roman-Urrestarazu et al., 2021), ADHD (Shi et al., 2021), and eating disorders (Cheng et al., 2019).

Participants total IQ score on the WISC-III^{uk} was included within sensitivity analyses due to the associations with autistic traits. Higher IQ scores have been shown to be associated with less severe autistic traits, better adaptive functioning, and a greater ability to compensate for cognitive differences (Marinopoulou et al., 2025). Further, IQ is suggested to have a complex interplay with ADHD traits and associated difficulties (Milioni et al., 2017).

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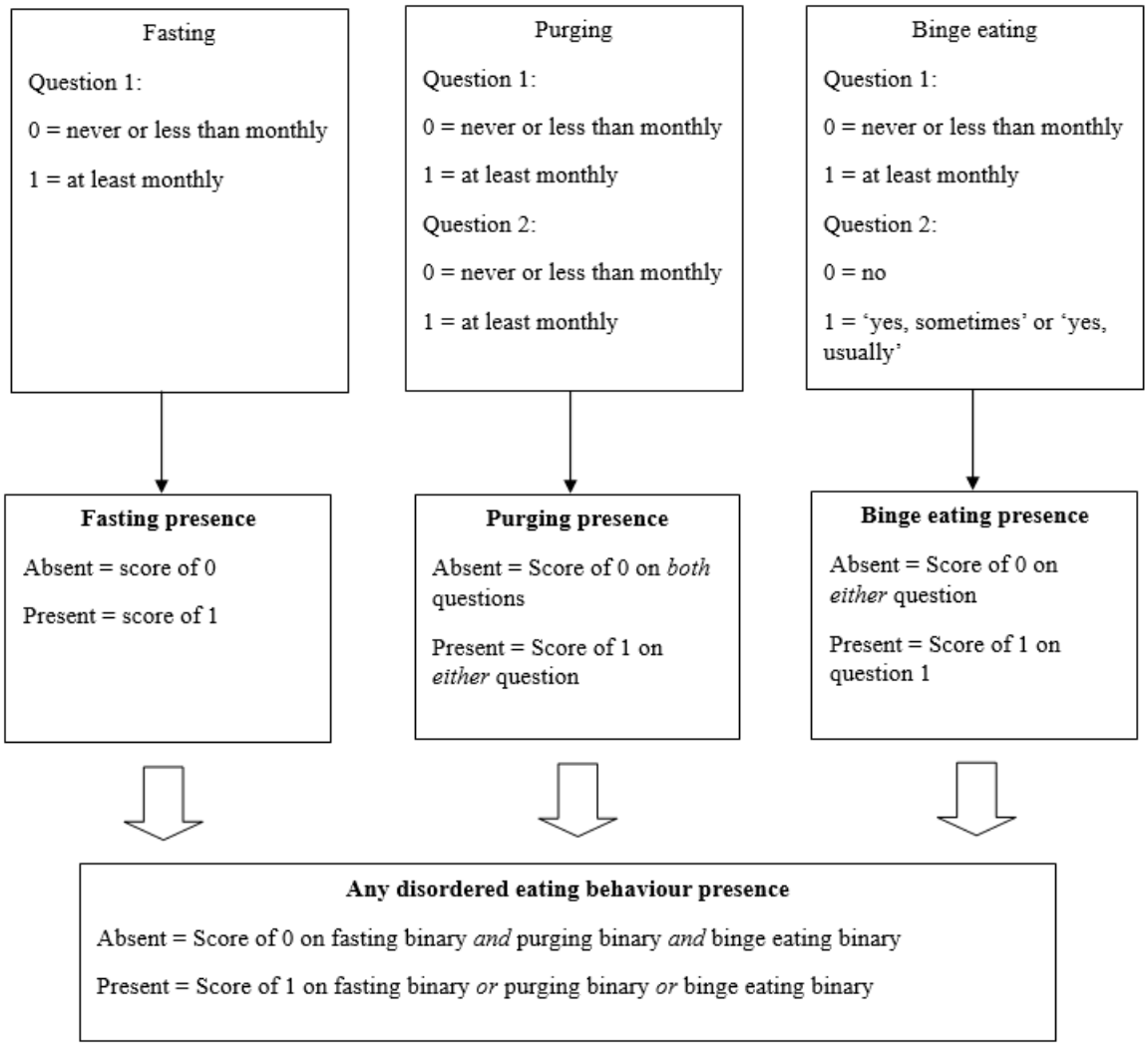
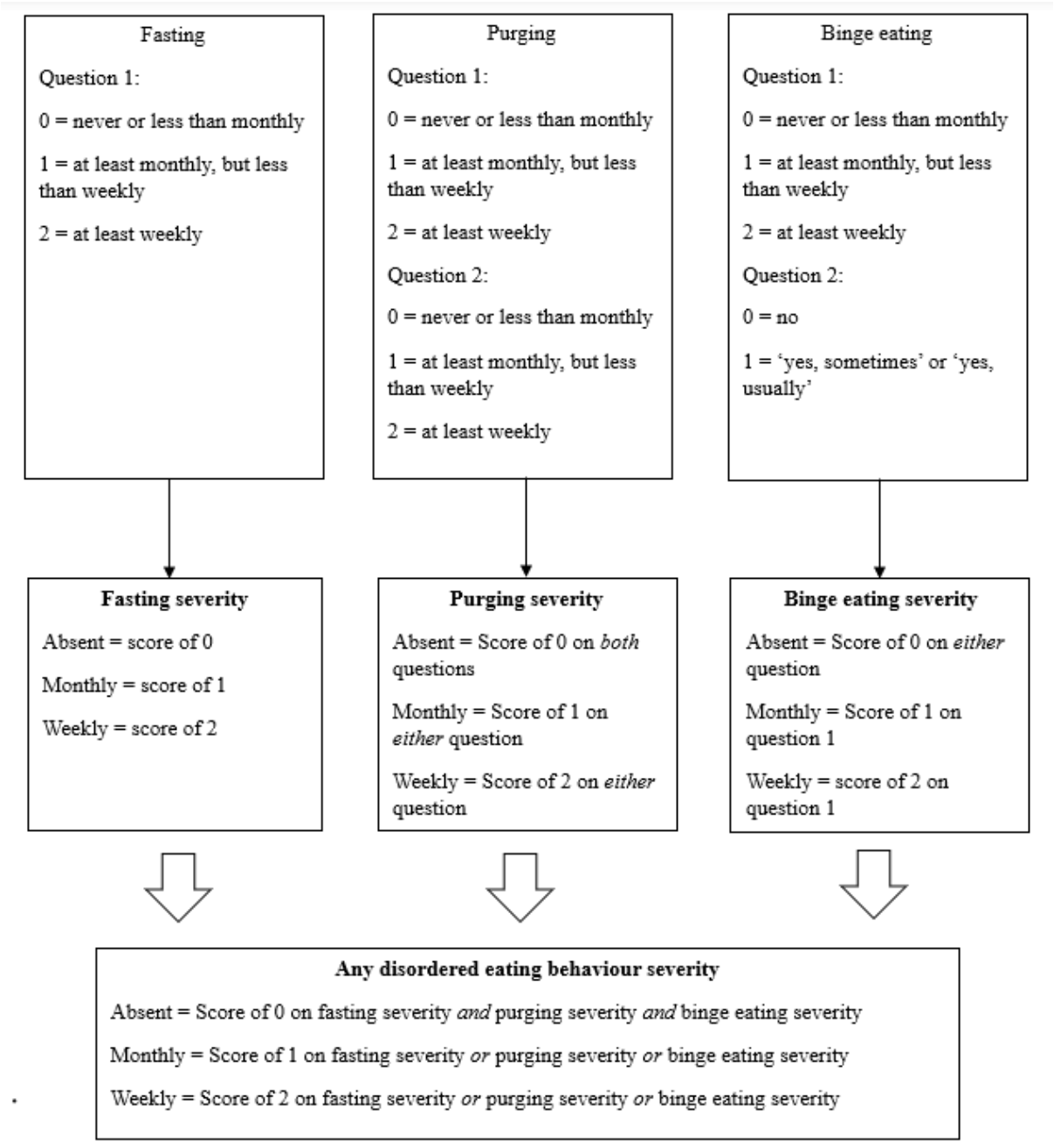
Appendix 3: Construction of disordered eating variables diagrams

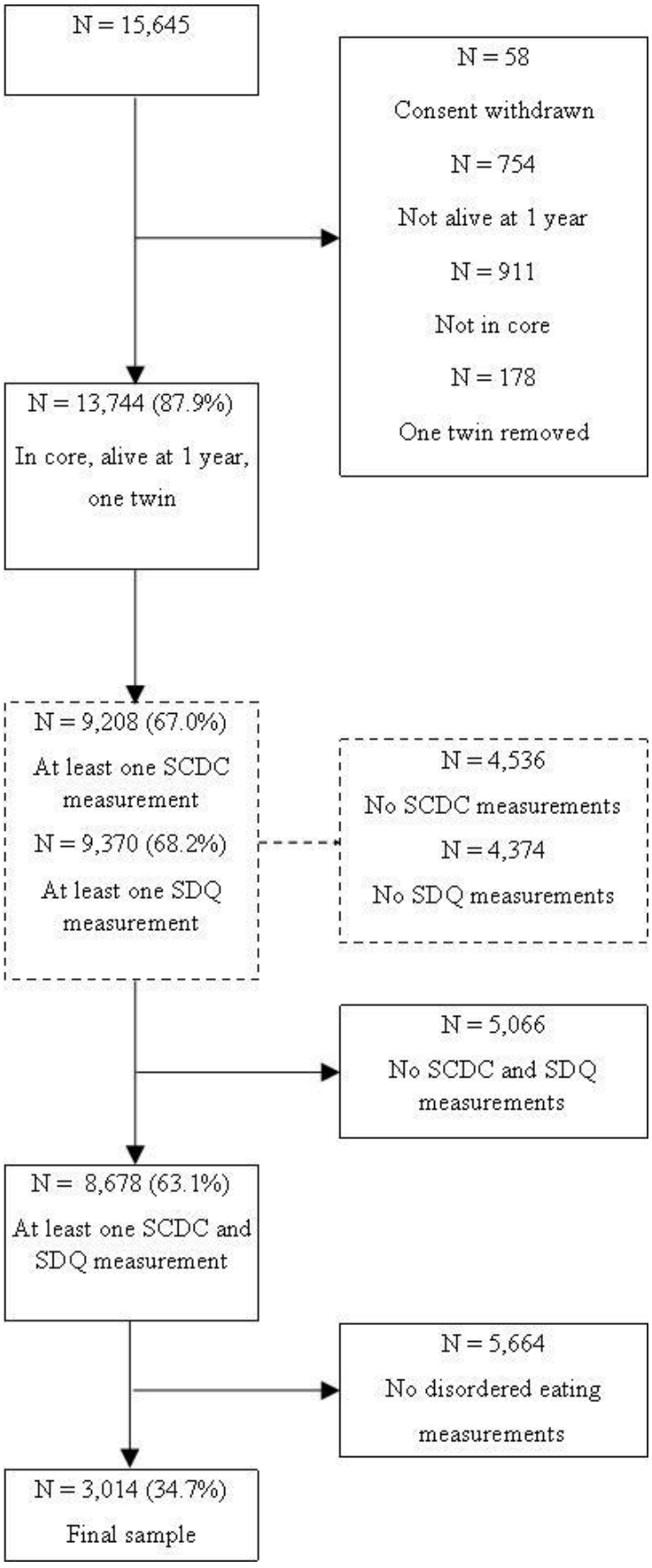
Binary Variables



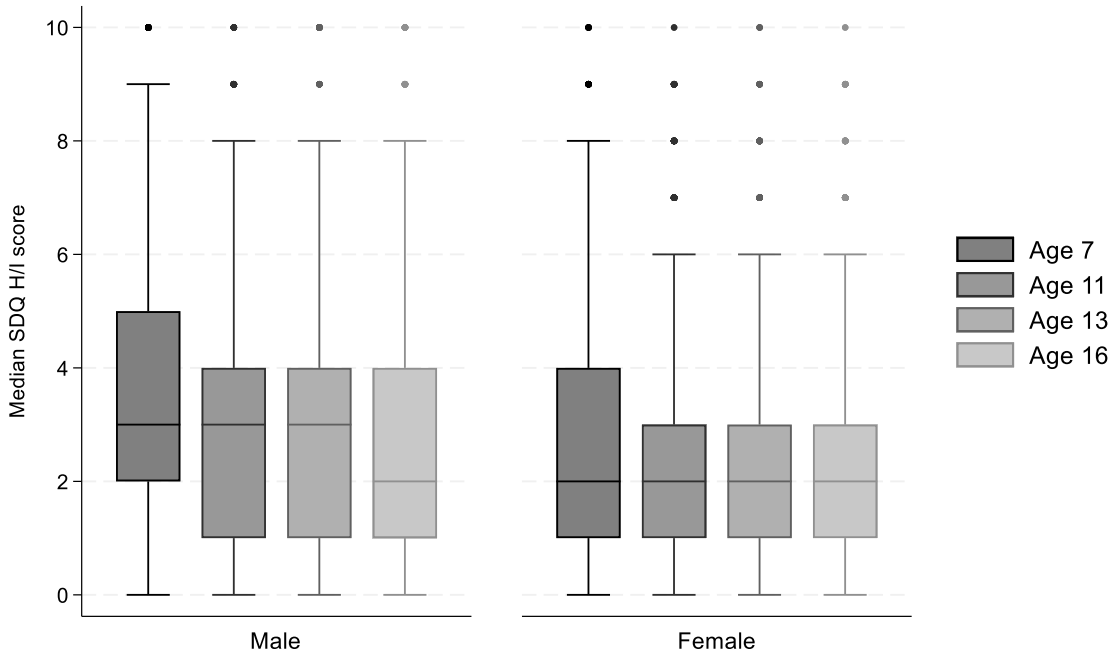
Frequency variables

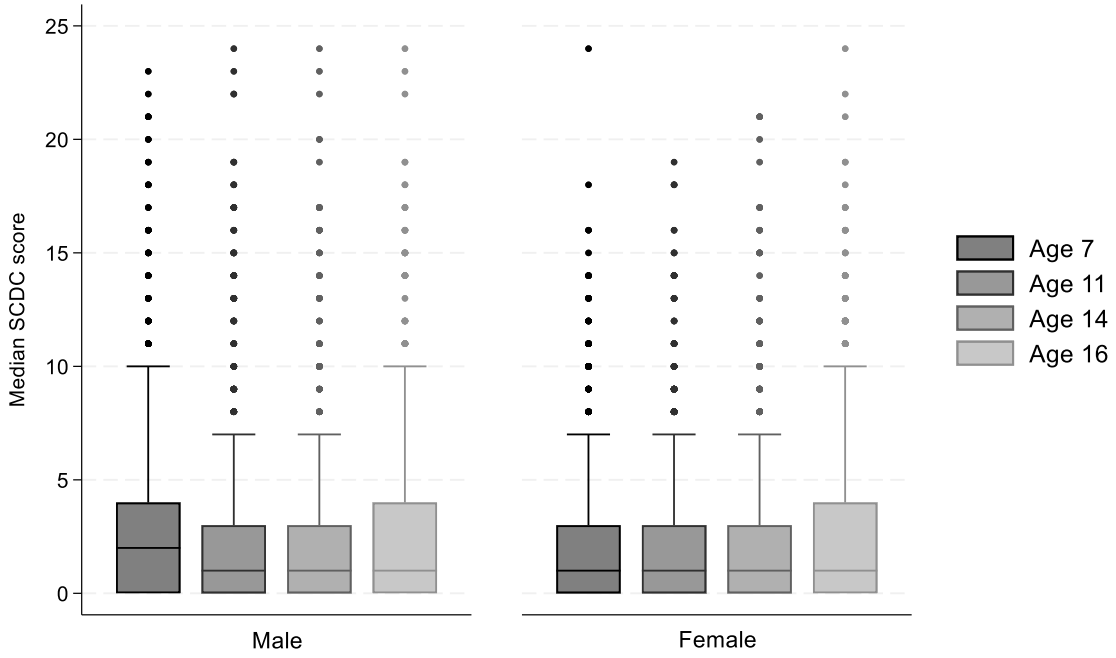


Appendix 4: Flowchart of study participation


N = 15,645
N = 58
Consent withdrawn
N = 754
Not alive at 1 year
N = 911
Not in core
N = 178
One twin removed
N = 13,744 (87.9%)
In core, alive at 1 year, one twin
N = 9,208 (67.0%)
At least one SCDC measurement
N = 9,370 (68.2%)
At least one SDQ measurement
N = 4,536
No SCDC measurements
N = 4,374
No SDQ measurements
N = 5,066
No SCDC and SDQ measurements
N = 8,678 (63.1%)
At least one SCDC and SDQ measurement
N = 5,664
No disordered eating measurements
N = 3,014 (34.7%)
Final sample

Appendix 5: Boxplots of SCDC and SDQ scores


Median SDQ H/I score
10
8
6
4
2
0
Male
Female
Age 7
Age 11
Age 13
Age 16

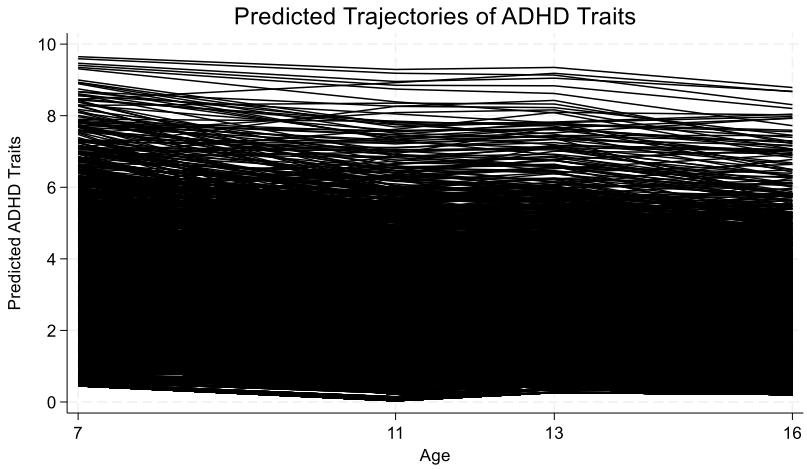

Median SCDC score
25
20
15
10
5
0
Male
Female
Age 7
Age 11
Age 14
Age 16

Appendix 6: Proportion of disordered eating by type and gender

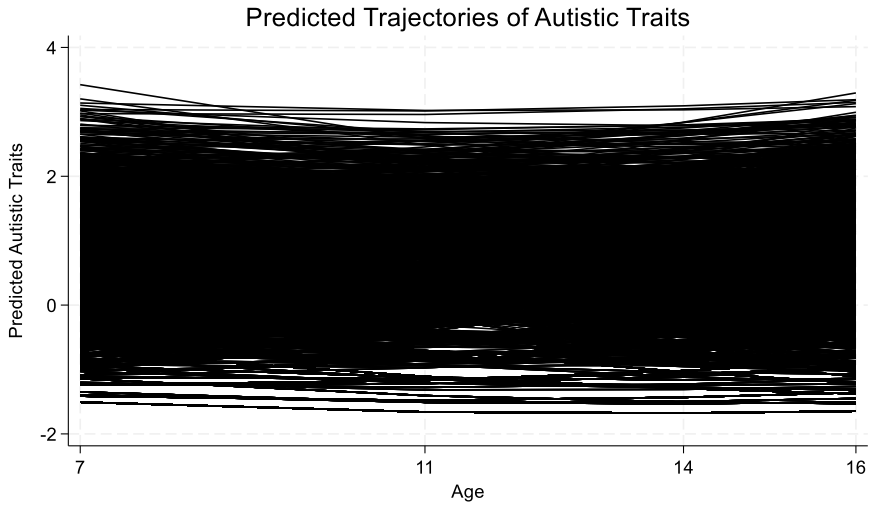
	Binary		Frequency		
	None *n* **(%)**	**Any** *n* **(%)**	**None** *n* **(%)**	**Monthly** *n* **(%)**	**Weekly** *n* **(%)**
Total					
Fasting	2,845 (94.40%)	160 (5.31%)	2,845 (94.40%)	74 (2.46%)	86 (2.85%)
Purging	2,891 (95.92%)	113 (3.75%)	2,891 (95.92%)	34 (1.13%)	79 (2.62%)
Binge eating	2,763 (91.67%)	246 (8.16%)	2,763 (91.67%)	138 (4.58%)	108 (3.58%)
Female					
Fasting	1,765 (58.56%)	148 (4.91%)	1,765 (58.5%)	66 (2.19%)	82 (2.72%)
Purging	1,810 (60.10%)	102 (3.38%)	1,810 (60.10%)	29 (0.96%)	73 (2.42%)
Binge eating	1,711 (56.77%)	201 (6.67%)	1,711 (56.77%)	116 (3.85%)	85 (2.82%)
Male					
Fasting	1,080 (35.83%)	12 (0.40%)	1,080 (35.83%)	8 (0.27%)	4 (0.13%)
Purging	1,081 (35.87%)	11 (0.37%)	1,081 (35.87%)	5 (0.17%)	6 (0.20%)
Binge eating	1,052 (34.90%)	45 (1.49%)	1,054 (34.90%)	22 (0.73%)	23 (0.76%)

Appendix 7: Developmental trajectories for autistic and ADHD traits

ADHD traits growth factor	*M*	*SD*
Intercept	0.295	1.741
Linear Slope	0.009	0.967
Quadratic slope	-.004	0.333



Autistic traits growth factor	*M*	*SD*
Intercept	-.001	1.145
Linear Slope	0.0001	0.346



Appendix 8: Binomial and Multinomial un-adjusted logistic regression results for latent ADHD and Autism growth curve factors and Disordered eating at age 18

Latent Trajectory factor	Any disordered eating at 18 years	Frequency of any disordered eating at 18 years	
	Any OR	**Monthly RRR**	**Weekly RRR**
ADHD intercept	0.91 (0.83, 0.99), $p = .031$	0.83 (0.73, 0.95), $p = .005$	0.97 (0.87, 1.09), $p = .616$
ADHD slope	0.71 (0.53, 0.97), $p = .029$	0.65 (0.41, 1.01), $p = .056$	0.76 (0.52, 1.12), $p = .167$
ADHD quadratic	0.47 (0.21, 1.08), $p = .074$	0.36 (0.11, 1.23), $p = .104$	0.56 (0.20, 1.62), $p = .285$
Autism Intercept	0.47 (0.21, 1.08), $p < .001$	1.24 (1.05, 1.46), $p = .010$	1.28 (1.10, 1.49), $p = .001$
Autism Slope	2.00 (1.44, 2.79), $p < .001$	1.71 (1.07, 2.74), $p = .025$	2.28 (1.49, 3.49), $p <.001$

OR = Odds ratio, RRR = relative risk ratio

Latent Trajectory factor	Any Fasting at 18 years	Frequency of Fasting at 18 years	
	Any OR	**Monthly RRR**	**Weekly RRR**
ADHD intercept	0.93 (0.82, 1.06), $p = .278$	0.89 (0.73, 1.08), $p = .222$	0.97 (0.82, 1.15), $p = .698$
ADHD slope	0.74 (0.47, 1.16), $p = .189$	0.67 (0.34, 1.31), $p = .240$	0.80 (0.44, 1.45), $p = .459$
ADHD quadratic	0.52 (0.15, 1.79), $p = .299$	0.45 (0.07, 2.82), $p = .396$	0.58 (0.11, 2.99), $p = .510$
Autism Intercept	1.28 (1.07, 1.52), $p = .006$	1.24 (0.97, 1.60), $p = .090$	1.31 (1.03, 1.66), $p = .026$
Autism Slope	1.92 (1.18, 3.14), $p = .009$	2.06 (1.02, 4.18), $p = .045$	1.81 (0.94, 3.51), $p = .076$
	Any purging at 18 years	**Frequency of purging at 18 years**	
ADHD intercept	0.88 (0.75, 1.03), $p = .097$	0.86 (0.64, 1.14), $p = .281$	0.88 (0.73, 1.07), $p = .192$
ADHD slope	0.71 (0.42, 1.21), $p = .210$	0.82 (0.32, 2.15), $p = .692$	0.67 (0.35, 1.27), $p = .212$

ADHD quadratic	0.58 (0.13, 2.51), p = .464	1.23 (0.9, 16.87), p = .877	0.42 (0.07, 2.41), p = .330
Autism Intercept	1.26 (1.02, 1.55), p = .030	1.36 (0.94, 1.97), p = .105	1.21 (0.95, 1.56), p = .124
Autism Slope	2.62 (1.48, 4.63), p = .001	1.71 (0.61, 4.82), p = .309	3.15 (1.61, 6.16), p = .001
	Any binge eating at 18 years	**Frequency of binge eating at 18 years**	
ADHD intercept	0.97 (0.88, 1.07), p = .540	0.90 (0.78, 1.04), p = 0.134	1.05 (0.91, 1.22), p = .506
ADHD slope	0.86 (0.59, 1.24), p = .419	0.79 (0.48, 1.31), p = .358	0.93 (0.55, 1.57), p = .788
ADHD quadratic	0.75 (0.27, 2.07), p = .580	0.61 (0.16, 2.41), p = .482	0.93 (0.22, 3.90), p = .916
Autism Intercept	1.21 (1.05, 1.40), p = .008	1.15 (0.96, 1.39), p = .132	1.30 (1.05, 1.60), p = .017
Autism Slope	1.49 (0.99, 2.23), p = .055	1.40 (0.82, 2.39), p = .220	1.63 (0.90, 2.95), p = .107

OR = Odds ratio, RRR = relative risk ratio