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Potential mechanisms underlying the association between feeding and eating disorders and autism

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ABSTRACT

There is a reliable association between autism and Feeding and Eating Disorders. Concerningly, where these two conditions co-occur, clinical outcomes of Feeding and Eating Disorders are significantly worse, and treatment less effective, than when the Feeding and Eating Disorders occur in neurotypical individuals. Problematically, the reason for the association between autism and Feeding and Eating Disorders is poorly understood, which constrains advances in clinical care. This paper outlines several possible mechanisms that may underlie the observed association and suggests ways in which they may be empirically tested. Mechanisms are split into those producing an artefactual association, and those reflecting a genuine link between conditions. Artefactual associations may be due to conceptual overlap in both diagnostic criteria and measurement, Feeding and Eating Disorders causing transient autistic traits, or the association being non-specific in nature. A genuine association between autism and Feeding and Eating Disorders may be due to common causal factors, autism directly or indirectly causing Feeding and Eating Disorders, and Feeding and Eating Disorders being a female manifestation of autism.

1. Introduction

Feeding and Eating Disorders (FEDs) are characterised by disturbed eating and eating-related behaviours that significantly impair one's physical and mental health (American Psychiatric Association, 2013). They include anorexia nervosa (AN; severe weight loss, restrictive eating, fear of weight gain and distorted body image), bulimia nervosa (BN; recurrent binge eating followed by inappropriate compensatory behaviours to prevent weight gain), other specified feeding or eating disorders (OSFED; clinically severe difficulties that do not fit into the two aforementioned diagnoses), avoidant restrictive food intake disorder (ARFID; avoidance or lack of interest in food that is not motivated by concerns about weight or body shape), binge eating disorder (BED; regular and uncontrolled overeating), pica (eating things that are not considered food), and rumination disorder (unintentionally regurgitating food). Most research on FEDs and autism thus far has focused on AN and BN.

Autism is a neurodevelopmental condition associated with difficulties in social interaction and communication, and restricted or repetitive patterns of thought and behaviour. In line with the preferences of many in the autistic community, this paper will use identity-first language (i.e., 'autistic person' rather than 'person with autism'; Kenny et al., 2016). Since Gillberg's seminal paper first brought attention to the overlap between FEDs and autism (Gillberg, 1983), there has been a wealth of research substantiating this association (Adams et al., 2022; Carpita et al., 2020; Huke et al., 2013; Zucker et al., 2007; Vissoker et al., 2015; Karjalainen et al., 2019; Spek et al., 2020). For autistic people, clinical outcomes of FEDs tend to be significantly worse than when FEDs occur without autism, and autistic individuals appear to be less responsive to conventional FED treatments (Wentz et al., 2009; Tchanturia, 2021; Nielsen et al., 2015; Westwood and Tchanturia, 2017; Nazar et al., 2018; Zhang et al., 2022). Moreover, those with higher autistic traits form a cluster of AN patients that also show greater anxiety and depression, as well as more severe symptoms and reduced weight gain following treatment (Li et al., 2023; Leppanen et al., 2022; Susanin et al., 2022). Given the clinical implications of this overlap, it is of vital importance that we understand the reasons for it. Understanding the mechanisms responsible may provide useful insight into how we can develop more efficacious treatments for autistic people with FEDs, as well as interventions to prevent the emergence of FEDs.

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This paper will consider whether the evidence provided so far reflects a real association between autism and FEDs, or whether it is an artefact. By artefact, we mean that the association between autism and FEDs does not reflect true covariance between the conditions. An artefactual overlap could arise due to consequences of the FED (such as starvation) causing traits that are misinterpreted as autism; the association not being specific, or due to a third, non-mediating condition; conceptual overlap meaning the conditions are not distinct; or measurement overlap meaning the conditions are not assessed independently. All of these possibilities are reviewed in the first section of the paper. The second section will explore potential explanations for a genuine association. These include FEDs being a female manifestation of autism, the two conditions sharing common or correlated aetiological factors, and autism directly or indirectly causing FEDs (see Fig. 1 for an overview), each of which may act in isolation or in combination. As autism is generally understood to be genetically specified (Tick et al., 2016) and the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-V, American Psychiatric Association, 2013) specifies that symptoms must be present in the early developmental period (in contrast to FEDs which tend to emerge in late adolescence; Solmi et al., 2022), we have not considered the possibility that FEDs might cause autism.

Overall, this paper aims to build upon the work of Brede and colleagues (2020) by providing an overview of the previously-suggested mechanisms that may underlie the association between autism and FEDs, and make some novel suggestions for mechanisms explaining the association. We will also assess the evidential basis for each mechanism and make suggestions for how they could be tested (see Appendix 1). This paper does not seek to provide a comprehensive review of the literature, but rather to provide a conceptual overview to guide empirical work. Searches for relevant literature were conducted on PubMed, initially on 11th July 2022, and subsequently on the 7th April 2023 and 30th July 2023. The reference lists of identified papers were checked to ensure no key papers were missed. (Text Box)

2. Potential mechanisms

2.1. Mechanisms producing an artefactual association

2.1.1. FEDs lead to the same traits as caused by autism

An artefactual association may be produced by the effects of starvation arising from certain FEDs, which may cause individuals to show a phenocopy of autistic traits (Pooni et al., 2012; Pellicano and Hiller, 2013; Westwood et al., 2016; see "consequences of FEDs lead to traits of autism" in Fig. 1). Note that under this account autistic traits are caused by starvation (and therefore do not precede the onset of starvation) and so, to the extent that early onset is a necessary feature of autistic traits (American Psychiatric Association, 2013), autism-consistent traits caused by starvation may be considered as 'pseudo-autistic'.

Consistent with this idea, starvation in FED patients is associated with higher autistic traits, including social withdrawal, repetitive behaviours, and reduced communication skills (Stewart et al., 2017; Mandy and Tchanturia, 2015; Tchanturia et al., 2012a). There is also some evidence to suggest that autistic traits are lower in weight-recovered AN patients compared to those with current AN (Kerr-Gaffney et al., 2021), and decrease in AN patients when assessed a year after weight recovery (Karjalainen et al., 2019). One study found that out of a sample of forty women with AN, 21 scored above the Autism Diagnostic Observation Schedule (Lord et al., 2012) threshold that indicates clinically significant levels of autistic traits, although, of these, only four had demonstrated autistic traits in childhood (Westwood et al., 2018). However, the relevance of this finding is tempered by evidence that, in the general population, a subgroup of females show autistic traits during adolescence and early adulthood which are not overt during childhood, at least by parent report (Pender et al., 2021; Mandy et al., 2018). This may be due to factors such as the increased social demands and stresses of adolescence and early adulthood leading to traits that were always present becoming more noticeable (Mandy et al., 2018).

In contrast to the above evidence, numerous studies have shown that heightened autistic traits are also found in recovered and weight-restored FED samples when compared to healthy individuals, indicating that the presence of autistic traits cannot be entirely explained by starvation (Huke et al., 2013; Bentz et al., 2017; Nazar et al., 2018). Of course, these data are only inconsistent with the idea that autistic traits seen in FED individuals are a product of starvation if one assumes that the effects of starvation do not induce long-term brain changes that persist after the period of starvation. This seems to be a reasonable assumption, with recent studies suggesting that AN-derived brain damage markers (including those due to starvation) do not persist following recovery (Doose et al., 2022; Walton et al., 2022). Arguably

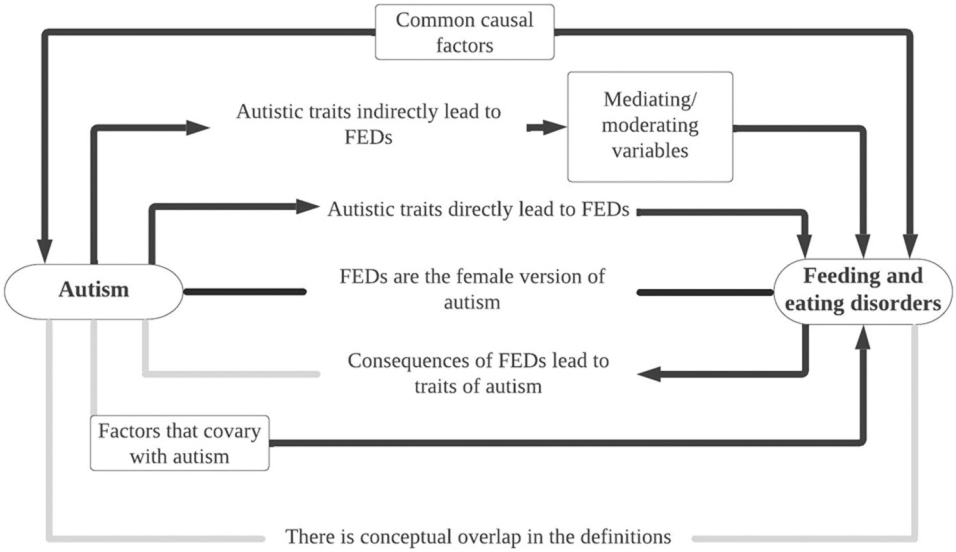

Fig. 1. Note: This model highlights the potential mechanisms responsible for the observed co-occurrence of autism and Feeding and Eating Disorders. The black lines indicate "genuine associations", whereas the grey lines indicate "artefactual" associations. The "conceptual overlap in the definitions" route includes genuine conceptual overlap and overlap in measurement.

Text box
: To be inputted alongside the introduction if the editor sees fit.

Autism	Autism is a form of neurodevelopmental variance that is currently diagnosed as a discrete entity. It is defined by social and communicative deficits, and restrictive and repetitive patterns of behaviour, interests, and hobbies. Autism is commonly considered to be something one is born with, and although there is no single established cause, there appears to be a genetic and neurological basis.
Feeding and eating disorders	Feeding and eating disorders refer to several conditions which include disturbed eating and eating-related behaviours that significantly impair one's physical and mental health. As with autism, there appear to be multiple potential causes, ranging from biological (e.g.: genetic or hormonal) to environmental factors (e.g.: societal beauty standards or peer influence).

the most persuasive argument comes from the fact that not all FEDs result in starvation (e.g., BED typically results in weight gain), and yet there is preliminary evidence to suggest that these non-restrictive FEDs also frequently co-occur with autism (Nickel et al., 2019; Gesi et al., 2017). Therefore, although starvation effects may increase the level of autistic traits in restrictive BN and AN, it is unlikely that they fully explain the association between FEDs and autism. Whilst one may speculate that overeating could also result in heightened autistic-like traits (e.g., repetitive patterns of binge-eating), to our knowledge there is currently no evidence for this.

Other than the direct effects of starvation, it may also be the case that other symptoms of FEDs cause traits that appear to be autistic in nature. For example, social withdrawal is often observed in autism, due to the social impairments that are characteristic of autism and the adverse social experiences of autistic people (Sasson et al., 2017). Individuals with FEDs may also avoid social situations, due to concerns that others will notice their disordered eating. This withdrawal may lead to social impairment if it impedes the development of age-appropriate social skills.

Longitudinal work suggests that autistic traits precede, and are predictive of, the emergence of FEDs (Solmi et al., 2021; Jacobi et al., 2003; Kalyva, 2009; Karjalainen et al., 2016), suggesting that they are not entirely consequences of the FED due to social withdrawal or starvation. This is substantiated by qualitative work, where women with co-occurring FEDs and autism, or suspected autism, report that their autistic traits were present from childhood (Mandy and Tchanturia, 2015; Brede et al., 2020). Although in some studies autistic traits were notably lower in the recovered groups than in those with an active FED, this may simply be because those with higher autistic traits may be less likely to recover (Nazar et al., 2018; Tchanturia et al., 2017).

Overall, it appears as though starvation and other consequences of eating disorders may account for some of the association between autistic traits and FEDs, but longitudinal studies and research on recovered FED patients, suggest that the association between FEDs and autism is not entirely explained by this. Further longitudinal work would be beneficial to corroborate these findings.

2.1.2. Non-Specific factors that covary with autism cause FEDs

Given that 70% of autistic young people present with at least one co-occurring condition, with 41% showing multiple co-morbidities (Simonoff et al., 2008), it is very possible that the presence of these co-occurring conditions may increase one's risk of developing an FED, rather than autism itself. Thus, the association between FEDs and autism may be present, but not specific to, or caused by, autism, and thus artefactual (see the "factors that covary with autism" route in Fig. 1). Thus, autism could have a common causal basis with a second psychiatric condition, resulting in high co-occurrence between autism and this second condition. The second psychiatric condition may in turn increase the likelihood of developing eating disorders. Critically, autism does not cause either the eating disorder, or the second psychiatric condition. For example, a common genetic component has been identified in both obsessive compulsive disorder (OCD) and autism (Liu et al., 2019), and OCD has been suggested as a risk factor for future FEDs (although prospective longitudinal work has relied on small samples, see Buckner et al., 2010; Micali et al., 2011). This suggestion is supported by the fact that several of the conditions that co-occur with autism also frequently co-occur with FEDs. These include anxiety, depression, attention deficit hyperactivity disorder (ADHD) and the experience of gender dysphoria (Kerr-Gaffney et al., 2018; Kirsch et al., 2020; Postorino et al., 2017; Hollocks et al., 2019; Pezzimenti et al., 2019; Warrier et al., 2020; Milano et al., 2020; Lai et al., 2019; Kaisari et al., 2017; Murphy et al., 2020).

A related explanation is that there is a general underlying 'p factor' explaining symptom co-occurrence, higher levels of which confers a greater risk of any (or multiple) psychiatric condition(s) (Caspi et al., 2014; Murphy et al., 2017). Although it is currently not known exactly how neurodevelopmental conditions can be accommodated within p factor models, it is certainly a possibility that they could be (Ronald, 2019; Selzam et al., 2018). This means that one may be at a high risk of psychopathology, increasing the likelihood of being autistic, as well as developing FEDs, or a condition which predisposes to FEDs.

The non-specific association and p factor model previously described are distinct from a third possibility which may also explain the reported co-occurrence rates, in which autism causes FEDs via a second psychiatric disorder, where the second disorder mediates the link between autism and FEDs, as will be discussed further in Section 2.2.4. As an example of mediation, autism may cause depression (DeFilippis, 2018; Hudson et al., 2019), which may in turn lead to FEDs (Monteleone et al., 2019; Araujo et al., 2010; Stice et al., 2004; Casper, 1998). The crucial fact that makes the mediation model distinct from both the non-specific association and p factor models is that in the mediation model autism is part of the causal chain leading to a FED, and therefore the association is no longer artefactual. Under the mediation model, if autism caused depression, which in turn caused an FED, then an intervention to prevent the autism causing depression would reduce the likelihood of an FED. In contrast, if the p factor increased the likelihood of autism, depression and FED, then interventions designed to prevent autism causing depression would have minimal impact on the likelihood of an FED.

A network-based approach may provide a useful way to test these possibilities (see Robinaugh et al., 2020; Borsboom et al., 2017), by allowing investigation of the relationships between symptoms associated with each of these disorders. This was exemplified by a recent study, which demonstrated that depression and anxiety symptoms, specifically feelings of worthlessness and avoidance of social eating, predicted both ED symptoms and recovery post-treatment in adult AN patients (Elliott et al., 2020). Additionally, Jones and colleagues (2021) used the data from nine published studies of symptoms of several

different disorders, including BN and autism. Network analysis of these symptoms revealed that several symptoms emerged as 'bridge' symptoms (symptoms linking different clinical conditions) across multiple networks, supporting the application of a network-based approach for exploring comorbidity across conditions.

2.1.3. Alexithymia, not autism, drives the association between autism and FEDs

A specific example of a non-specific association described in Section 2.1.2 (the "factors that covary with autism" route in Fig. 1) is demonstrated by the hypothesis that the high co-morbidity between FEDs and autism is driven by alexithymia. This warrants a separate discussion due to alexithymic traits frequently being attributed to autism, thus creating significant potential for alexithymia to be overlooked as a causal factor. Alexithymia is a condition characterised by impairments in identifying and describing one's own emotions (Nemiah et al., 1976) that frequently co-occurs with autism, with rates of alexithymia in autistic populations at around 50%, compared to approximately 5% in the general population (Berthoz and Hill, 2005; Oakley et al., 2020; Kinnaird, Stewart et al., 2019). The increased prevalence of alexithymia in autism is not unique to autism, indeed there are increased rates of alexithymia in numerous clinical populations (Taylor and Bagby, 2004; Leweke et al., 2012; Lumley et al., 1997), including AN (Saure et al., 2022). If alexithymia is a causal factor in the development of FEDs, high co-occurrence will be observed between FEDs and any clinical population characterised by high rates of alexithymia, not just autism (assuming alexithymia is a unitary condition; Hobson et al., 2019). Under this model, autism is *not* part of the causal chain which leads to FEDs. Initial evidence consistent with this 'alexithymia proposal' is that those with FEDs score higher on measures of alexithymia than matched controls (Peres et al., 2020; Kinnaird, Stewart et al., 2020; Westwood et al., 2017; Tchanturia et al., 2012b) and that several conditions which show a high prevalence of alexithymia also show a high degree of co-morbidity with FEDs (Blinder et al., 2006; Corcos et al., 2000). To explore this further, it will be interesting to determine the degree of variability with respect to alexithymia in the FED population, to establish whether there are specific "alexithymic" subgroups, or whether elevated levels of alexithymia are common to all those with FEDs.

More specific evidence consistent with the alexithymia proposal, and with potential gender-specific effects of alexithymia, has been provided by a series of cross-sectional studies. Vuillier and colleagues (2020) found that in a non-clinical sample alexithymic traits mediated the relationship between heightened autistic traits and eating psychopathology, accounting for 18% of the variance. They extended these findings in their second study, demonstrating that this mediation effect was partial and specific to females. Among females, autism exhibited both direct effects on FED psychopathology and indirect effects through alexithymia. However, when depression and anxiety scores were added as covariates, the direct effect of autistic traits became non-significant, and only the alexithymic trait "difficulty in identifying feelings" remained a significant indirect predictor. Furthermore, Moseley and colleagues (2023) found that in men, higher autistic traits were not associated with FED psychopathology directly. Instead, autistic traits were associated with increased levels of alexithymia, and these alexithymic traits were associated with greater symptoms of depression and anxiety, which, in turn, were associated with increased FED symptoms. In women, alexithymia mediated the association between autistic traits and FED psychopathology both directly and via the impact of alexithymia on anxious and depressive symptoms. However, these observed gender differences may be an artefact of increased sample sizes and/or an increased range of FED symptom scores in female samples in some studies (with both factors increasing statistical power). This can be seen in Vuillier and colleague's paper.

Although not a direct study of the role of alexithymia in the association between FED and autism, a study by Hobson and colleagues (2020) was consistent with the alexithymia proposal in that it demonstrated that alexithymia increased the chances of meeting the criteria for an autism diagnosis based on the Autism Diagnostic Observation Schedule for both twins at genetic risk of autism and clinical AN samples. Moreover, in Brede and colleagues' qualitative study (2020), one autistic woman with AN described how she sometimes misinterpreted emotions as physical symptoms (consistent with alexithymia), which resulted in her restricting food intake to numb these sensations.

Ideally, the role of alexithymia should be tested using a similar design to that employed by Bird and colleagues (Bird et al., 2010; Cook et al., 2013; Shah et al., 2016). FED symptoms should be measured in autistic and non-autistic participants, with and without co-occurring alexithymia, so that the effects of autism can be established while controlling for alexithymia and vice versa. At the very least, any future study of the link between autism and FEDs should also measure alexithymia. It would also be useful to determine whether levels of alexithymia can be reduced by intervention. Although promising results have been provided by initial studies (Lukas et al., 2019; Levant et al., 2009; Norman et al., 2019), more work is required. If alexithymia can be reduced by an intervention, then experimental studies can be conducted to establish whether this reduction in alexithymia causes a reduction in FED symptoms or risk. An alternative would be to run longitudinal epidemiological studies using random intercept cross-lagged panel models, similar to the design employed by Murray and colleagues (2021), although some researchers have argued that traditional cross-lagged panel models may be preferable (Lüdke & Robitzsch, 2021).

A lot of the caveats mentioned when discussing the association between autism and FEDs also apply to alexithymia. For example, it may be that alexithymic traits are temporarily exacerbated by starvation effects of restrictive FEDs. Additionally, the association may be driven by third variables, such as depression or anxiety. Indeed, some research suggests the association between FEDs and alexithymia may be driven by joint associations with depression (Marchesi et al., 2014; Montebarocci et al., 2011).

2.1.4. Conceptual or measurement overlap

Finally, it could be the case that there is an overlap between autism and FEDs at the conceptual level, rather than an association between distinct conditions, producing an artefactual association ("there is conceptual overlap in the definitions" in Fig. 1). Although there is no overlap in the core criteria for FEDs and autism as set out in The DSM-V (American Psychiatric Association, 2013), there are similarities in the traits that clinicians might seek to identify in order to make a diagnosis, meaning that behaviours linked to one of the conditions may be viewed as a manifestation of the core traits of the other. For example, purging behaviour could either be a classic feature of BN or certain types of AN, or it could be a ritualistic or self-soothing behaviour that one would typically expect in an autistic individual.

Equally, the strict rules surrounding food consumption in AN could also be seen as an autistic trait, in that it reflects adherence to a strict routine motivated by restricted and repetitive behaviour (Marquenie et al., 2011). Kinnaird and Tchanturia explored this further, highlighting that whilst there are common traits between AN and autism across neuropsychological, social and communicative, sensory, and emotion regulation domains, the traits in autism are more general, whereas they are consistently food-focused in AN (Kinnaird and Tchanturia, 2021). Similarly, one study using eye-tracking technology demonstrated that although both participants with AN and autistic participants showed social difficulties, only the autistic participants paid less attention to faces, which is consistent with there being differences in the causes and manifestations of social difficulties across the two groups (Kerr-Gaffney, Jones et al., 2022).

Despite this, it is conceivable that some individuals who would qualify for a diagnosis of autism are instead given FED diagnoses. For example, an FED may be seen as at the core of symptoms such as strict mealtime routines and restricted eating, whilst further autistic traits,

such as non-typical social behaviour, may be assumed to be a side effect of the FED. Whether a diagnosis of autism or FED is deemed to be more appropriate may be influenced by clinicians' biases. For example, females may be more likely to receive a diagnosis of an FED when autism is the root cause of their symptoms, as autism is seen as more likely to affect males and female manifestations of the condition are less known (Lockwood Estrin et al., 2021), and because FEDs are seen as more likely to affect females (Strother et al., 2012). It may be useful to explore these biases further, by providing clinicians with case notes for imaginary patients who show varying degrees of both autistic and FED traits. In some case notes, the gender of patients would be specified, and in others, it would not. Then, whether the clinician recommends that the individual should be further tested for autism, FEDs, or both, and whether this depends on gender, could be ascertained. A similar study has previously been conducted with teachers, focusing purely on autism rather than FEDs, which found that teachers were more likely to suggest autism assessments for boys than for girls with identical symptoms (Whitlock et al., 2020).

Consideration of the above raises questions about the nature and validity of diagnostic categories themselves. First, if an autistic individual has sensory-related issues surrounding food that prevents them from consuming an adequate number of calories a day, if we consider what is best for the patient, would they benefit from a separate diagnosis of ARFID, or is the diagnosis of autism sufficient? The DSM-V currently specifies that with regard to ARFID, a diagnosis can only be given when it occurs in the context of another diagnosis if the severity of the eating disturbance exceeds that typically associated with the other diagnosis and warrants additional clinical attention (American Psychiatric Association, 2013). On a practical level, it could be argued that even if sensory processing difficulties were present in the majority of autistic individuals, in many cases it does not lead to severely restricted eating, and in the cases where it does, it can be treated, meaning that these eating problems may warrant their own separate diagnosis.

Second, is there a benefit to the categorical approach to diagnosis, whereby autism and FEDs are treated as entirely separate concepts, or would an approach similar to that outlined in the Research Domain Criteria (RDoC) be more appropriate? The RDoC is a research framework in which mental health and psychopathology are explored within the major domains of human functioning, rather than within diagnostic categories (Insel et al., 2010). Consequently, the RDoC framework can capture patterns of symptoms that would be seen as co-occurring conditions under the categorical model but does not require any assumption about the primacy of one condition over another, or how symptoms are related to each other. It also accounts for individuals whose symptoms are just below diagnostic thresholds for conditions, which is often observed when assessing autistic traits in women with FEDs (Westwood et al., 2017). Although proposed primarily for research, some argue that the principles of this framework could also be applied clinically (Yager and Feinstein, 2017). Although the RDoC is yet to be widely adopted in autism research, it has been argued that the use of the RDoC framework alongside (as opposed to in place of) more conventional models could be useful. This would allow autistic individuals to identify with the clinically-useful construct of autism and gain access to a community, whilst also addressing the issues associated with diagnostic categories and allowing for co-occurring disordered eating to be treated (Mandy, 2018).

Questions about the use of categorical and dimensional diagnostic systems are part of a broader debate concerning how autism, FEDs and indeed all psychological conditions are conceptualized in psychiatric nosology. Currently, according to 'medical models' (Zachar and Kendler, 2007) they are understood as underlying 'clinical entities', which manifest in the form of the signs and symptoms listed as diagnostic criteria that are set out in DSM-5 and ICD-11 (International Classification of Diseases 11th Revision; World Health Organisation, 2019). As we cannot directly measure these latent clinical entities ('latent factors' within a psychometric approach), we measure their symptoms as a proxy. Crucially, the latent entities are considered discrete and distinct. The medical model approach is what the health care system is currently based upon, and such latent factor models allow for multiple different latent factors to contribute to the presence or severity of particular symptoms. When a diagnosis is made at the symptom, rather than latent factor level, it is therefore not surprising that diagnostic confusion may occur. This is especially pertinent if one takes a dimensional view in which an individual may have different degrees of 'exposure' to the latent factor (or factors affording resilience) such that individual symptoms can vary in strength, and therefore the relative contribution of two latent factors (in this case autism and FEDs) to particular symptoms may also vary across individuals.

An alternative, but not necessarily incompatible, framework is exemplified by network-based approaches, where symptoms are conceptualized as an interconnected network, in which certain symptoms can cause the emergence of other symptoms (Robinaugh et al., 2020). Within these networks, clusters of frequently co-occurring symptoms may be interpreted as distinct disorders, or several distinct disorders with high comorbidity or co-occurrence, depending on the existing beliefs and expectations of researchers and clinicians. It is interesting to consider, though somewhat beyond the scope of this article, how the standard medical model and network-based approaches may be combined to understand the link between autism and FEDs. While a standard medical model approach would be to treat the two conditions as due to distinct clinical entities, a network approach is compatible with the idea that symptoms of autism could cause the symptoms of FED. It is uncertain, however, whether if all the symptoms of an FED are present one would be required to assume the presence of a separate underlying clinical entity in addition to autism under the medical model, and if so, whether this clinical entity would be the same as FEDs not resulting from autism. Initial work employing a network approach reported that within adults with AN, poor self-confidence, concerns about eating around others, and concerns about others seeing one's body were the strongest factors linking FED symptoms and autistic traits (Kerr-Gaffney et al., 2020).

There is also the possibility that autism and FED are distinct at the conceptual level, but that the measurement tools we use to assess the degree of autistic traits conflate autistic and FED traits, and vice versa. For example, the Eating Disorder Examination Questionnaire (Fairburn et al., 2008) asks both "Over the past 28 days, on how many days have you eaten in secret (i.e., furtively)?" and "Over the past 28 days, how concerned have you been about other people seeing you eat?". Although this behaviour is most obviously associated with FEDs, it may also reflect avoidance of social situations which require implicit social rules, such as mealtimes, and as such these items may also be endorsed by autistic individuals. The Eating Attitudes Test (EAT26; Garner et al., 1982) asks whether respondents are "aware of the calorie content of foods that I eat, " which may apply to some autistic people given the often-reported preoccupation with numbers in many autistic individuals. Moreover, it asks whether participants "enjoy trying new rich foods," which is unlikely in autistic individuals, given both sensory sensitivities and a preference for routine and repetition.

Conversely, the Autism Quotient (AQ; designed as a measure of autistic traits; Baron-Cohen et al., 2001) asks respondents whether they "find it hard to make new friends." FEDs often make it difficult for individuals to form and maintain friendships, due to factors such as their preoccupation with food, irritability due to hunger, low mood and hospitalisation (Datta et al., 2021; Thelen et al., 1990). The Adult Autism Subthreshold Spectrum (AdAS; Dell'Osso et al., 2017) asks respondents if "at school did you ever avoid eating or playing or doing gymnastics with other children?" Avoiding eating is typical of several FEDs. Additionally, it asks "Have you ever been aggressive because other people wanted to stop you from carrying out your rituals or because they did not let you have your own way?". Again, aggressive behaviour is sometimes observed in those with acute FEDs, especially if they are prevented from engaging in disordered behaviour (Truglia et al., 2006).

Despite the examples listed here, items that may indicate both FEDs and autism are a minority in these questionnaires. Although an FED may push participants who have high levels of subthreshold autistic traits over the threshold, it is unlikely that someone with an FED but low autistic traits would score above the diagnostic threshold, and vice versa. However, to quantify the exact degree of overlap between questionnaire measures of autism and FEDs, factor, latent class, or discriminant, analyses should be conducted. A similar approach has previously been taken to determine the independence of measurement of autistic traits and social anxiety traits (Pickard et al., 2017) and autistic traits and alexithymic traits (Cuve et al., 2022). Another approach is to compare participants diagnosed with both conditions to those diagnosed with only one of the conditions (as ascertained after screening for both conditions) on measures of the second condition. Results of such studies suggest that measurement overlap is not a major factor in explaining the overlap between autism and FEDs (Babb et al., 2022).

In summary, overlap may occur at the conceptual or measurement level, and both may impact diagnosis such that the association between autism and FEDs is artefactually increased. With respect to conceptual overlap, although there is no overlap in the core DSM symptoms, there are some diagnosis-relevant traits that could be applicable to both FEDs and autism. At the level of measurement, there does appear to be a small degree of overlap in questionnaire measures, though not enough to explain the observed association between FEDs and autism. It therefore appears that whilst conceptual and measurement overlap may increase the association between FEDs and autism, it is not sufficient to explain it.

2.2. Mechanisms underlying a genuine association

2.2.1. Eating disorders are a female manifestation of autism

The first explanation for a genuine association that will be discussed is that FEDs are a female manifestation of autism (Gillberg et al., 1983; Carpita et al., 2020; Odent, 2010; Treasure, 2007; see Fig. 1), given that the majority of autistic and non-autistic individuals with FEDs are female (Striegel-Moore et al., 2009) and that the majority of those diagnosed as autistic are male (Loomes et al., 2017). This is consistent with the finding that patterns of narrow interests and restricted and repetitive behaviours are more likely to include dietary habits in autistic females compared to autistic males (Lai et al., 2015). Moreover, both autistic traits and an autism diagnosis predict emotional undereating in girls but not boys (van Hof et al., 2020), and there is a stronger association between autistic traits and total eating problems in females compared to males, as well as more difficulties with eating in social contexts among autistic females compared to autistic males (Lundin Remnélius et al., 2021).

The claim that FEDs are a female manifestation of autism can be interpreted in various ways, and thus far there has not been a precise formulation of what it specifically means. The most extreme interpretation of this claim – that FEDs are only caused by autism in females, and that all autistic females develop an FED – is clearly not supported by the data. The majority of autistic females do not have diagnosed FEDs, score below established thresholds on questionnaires that would indicate the presence of an eating disorder (Karjalainen et al., 2016), and the majority of those with FEDs do not have an autism diagnosis, and score below established thresholds on questionnaires that would indicate clinically significant levels of autistic traits (Carpita et al., 2020; Kerr-Gaffney, Hayward et al., 2021). Moreover, a significant proportion of FED patients are male (Sweeting et al., 2015), and autistic males also show higher FED symptoms and lower body-mass index than neurotypical men (Courty et al., 2013; Sobanski et al., 1999), as well as a greater degree of eating problems (Spek et al., 2020). Although the risk of an FED may be higher in autistic women, it is still elevated in autistic men (Remnelius et al., 2021). Therefore, this extreme interpretation is simply not supported by the data.

However, some argue that because current conceptualizations of autism are modelled around male-typical presentations of autism, the prevalence of autism in women, including women with FEDs, might be underestimated (Dell'Osso et al., 2017; Lai et al., 2011). Westwood and colleagues' (2016) meta-analysis found that although there was a significant difference in the AQ scores of AN patients and healthy controls, women with AN did not tend to meet the cut-off scores that would indicate autism. This is consistent with several possibilities. First, it could reflect a degree of overlap in how we measure autism and AN, as discussed in Section 2.1.4. Second, it could be that women with FEDs show the full range of autistic traits at a low level, or that they only show a subset of autistic traits to a greater degree. Third, it may be that despite a high degree of autistic traits within the women with AN, the AQ scores are not above the threshold as the questionnaire does not capture certain, perhaps more female-typical, autistic traits. Future research would benefit from the use of measures of autistic traits that are designed specifically to capture any female-specific autistic presentation.

The same gender biases that could affect questionnaire measures have also been suggested to influence autism diagnosis, given evidence that girls are less likely to receive a diagnosis than males, despite similar levels of autistic traits (Duvekot et al., 2017). This is substantiated by qualitative work by Brede and colleagues (2020), which demonstrated that of their sample of autistic women with co-occurring AN, all had been in contact with health services for their FED and other mental health conditions before their autism was recognized. Another paper reported an average delay of nine years between an initial mental health diagnosis and an autism diagnosis (Babb et al., 2022) in females with eating disorders, compared to an average delay of 3.5 years from the point at which parents first approached a health professional with their concerns to the confirmation of an autism spectrum disorder diagnosis in a large, male-dominated sample (Crane et al., 2016). Both of these studies are consistent with the notion that autistic traits that go unrecognized in earlier years may manifest as an FED later in life and that a larger delay in females puts them disproportionately at risk, but could also be a result of FED being more readily recognized than autism in females.

It is reasonable to suggest that FEDs are one way that autistic traits may manifest and that females are especially vulnerable to this, given the gendered societal expectations surrounding weight (Murnen and Don, 2012), and the fact that females are less likely to receive an early diagnosis, and therefore support, for their autism (Harrop et al., 2021). Thus, gender may be seen as a moderating factor in the association between autism and FEDs. To corroborate this, future research should explore whether autistic females are at a greater risk of developing various types of FEDs than autistic males. It may be that the gender difference is greater in FEDs that pertain to weight and appearance, such as AN, than those that pertain to sensory experiences of food, like ARFID. Another aspect of the claim that eating disorders might be a female manifestation of autism that is underspecified is whether the claim relates to biological sex or self-identified or experienced gender. The same is often true for the papers addressing this issue. It is important for future research to make this distinction clearer, although effects of the two are often difficult to disentangle (Hines, 2005).

2.2.2. Common or correlated causal factors

Another explanation for a genuine association between FEDs and autism is that the two conditions have common or correlated causal factors. These causal factors are likely to be observed across multiple different levels; for example, a similar genetic basis for the two conditions may result in overlapping neurological or hormonal atypicalities. Moreover, these causal factors may interact with one another, for example, fewer hormone receptors in the brain may interact with lower circulating levels of those hormones to increase the risk of both autism and FEDs. Although autism emerges significantly earlier than FEDs, this difference in timing may be explained by the onset of puberty, which results in physiological and psychological changes (for example, hormonal changes and altered social expectations) that may cause FEDs to

emerge in those who are already predisposed to them due to shared risk factors between FEDs and autism.

At the most basic level, there may be a genetic component to the overlap between the two conditions. This could mean either that the exact same genes are implicated in both FED and autism, or separate genes that are close together on the chromosome, and therefore are likely to remain together during the process of recombination, cause FEDs and autism independently ('genetic linkage'; Pulst, 1999). In both cases, one would expect to see a correlation between the genes observed in autistic individuals and those with FEDs. Autism is known to have a largely genetic basis, with heritability estimates ranging from 64% to 91% (Tick et al., 2016). FEDs also appear to be highly heritable, although there is a paucity of quantitative genetic research on FEDs other than AN (Bulik et al., 2019), and very little on any shared genetic basis between autism and FEDs. One recent twin study explored the heritability of ARFID, finding a high heritability of 78%, which was maintained when autistic children were excluded from the analysis (Dinkler et al., 2023). Another study included a large autism cohort, within which there was a 21% prevalence of ARFID. Researchers found that among this sample, ARFID had a heritability of 45%, and a genome-wide association study (GWAS) revealed a single hit near ZSWIM6, a gene that prior research has implicated in neurodevelopmental conditions such as autism. Further genetic analysis using polygenic risk scores was consistent with a genetic link to autism (Koomar et al., 2021). As this GWAS analysis was underpowered, it may be that there are further genes implicated in the development of both autism and ARFID that the study was unable to detect. In addition to better-powered GWAS studies, any common genetic influence should also be explored with twin studies (e.g., Hallett et al., 2009; Fagnani et al., 2014).

At the neurological level, it is a possibility that the structural and functional alterations that have been suggested to be associated with autism may also be implicated in AN. For example, atypical development of areas of the brain involved in social interaction, including the amygdala, orbitofrontal cortex and superior temporal sulcus, have been demonstrated in both conditions (Zucker et al., 2007; Gillberg et al., 2007; Björnsdotter et al., 2018). The latter study found lower bilateral superior temporal sulcus grey matter volume in women with AN compared to healthy controls, and also found that left superior temporal sulcus grey matter volume correlated with AQ scores in the AN group. Given that the whole brain search for associations with AQ scores did not reveal any additional areas showing a similar effect, nor an association with the whole brain grey matter volume, the authors concluded that it was unlikely that the observed association between autistic traits and grey matter volume was a generalized effect of grey matter loss due to AN, and was instead specific to areas associated with social cognition (Björnsdotter et al., 2018). Similar variations in grey matter volume in the temporal lobe have been reported in autistic participants (Mueller et al., 2013), although research is highly inconsistent, and has predominantly been conducted on men. Indeed, a recent meta-analysis of the neurobiology of both AN and autism found no overlap between the two, and no consistent neuroanatomical abnormality associated with autism (Sader et al., 2022). Furthermore, a recent study which specifically looked at both AN and ASD traits, found no correlation between ASD traits and brain structure (Halls et al., 2022).

Another factor that has been suggested to be implicated in the aetiology of both FEDs and autism is the oxytocin system (Romano et al., 2016; Quattrocki and Friston, 2014; Giel et al., 2018). Oxytocin is a hormone best known for its role in social behaviours and attachment, but it is also linked to learning, anxiety, feeding and pain perception (Lee et al., 2009). In terms of endogenous levels of oxytocin, one systematic review documented that similarly diminished oxytocin levels have been reported in individuals with AN and autistic individuals, but not BN (Odent, 2010). A later review of oxytocin in FEDs substantiated this, reporting no evidence for alterations of endogenous oxytocin levels in BN, whereas AN seems to be associated with reduced circulating levels of oxytocin (Plessow et al., 2018). However, reduced oxytocin levels in AN may be the result of starvation. Preliminary work has implicated hypermethylation of the OXTR oxytocin receptor gene in AN (Kim et al., 2014; Thaler et al., 2020), and found hypomethylation in males, but not females, with BED (Giel et al., 2022). Atypical methylation of the OXTR gene has also reported in autism, however, the direction of this association appears to vary according to the age of participants and whether autistic traits or diagnoses are considered (Moerkerke et al., 2021). These complex relationships should be explored with further research using larger sample sizes.

Randomised control trials (RCTs), where half the participants are administered oxytocin and half a placebo, have been employed to test the effects of oxytocin on autistic and FED traits. One study found that although intra-nasal oxytocin did not affect weight gain in AN patients, self-reported eating concerns were lower after four to six weeks of treatment, as was cognitive rigidity, and salivary cortisol levels in anticipation of food (Russell et al., 2018). Although Kim and colleagues (2015) noted no difference in consummatory behaviours in patients with AN who took oxytocin, they did note that there was a decrease in calorie consumption over 24 h in patients with BN. Substantiating this, one review reported that exogenous administration of oxytocin reduced food intake in those with BN, but not AN (Plessow et al., 2018). This would suggest that in general, atypical oxytocin function influences eating behaviour, but the exact disorder it leads to depends on the specific mechanism by which it is operating. However, other studies have shown intranasal oxytocin to not affect eating behaviour in either healthy participants, or those with BN or BED (Leslie et al., 2019; Agabio et al., 2016) leading to the very real possibility of a 'file drawer' problem whereby studies finding non-significant effects of oxytocin are not published.

Turning our focus to autism, evidence from intra-nasal oxytocin studies is unconvincing, with one systematic review and meta-analysis highlighting small and non-significant effect on autistic traits (Wang et al., 2019). An alternative explanation could be that oxytocin plays an organisational rather than activational role in the development of autism. Activational effects refer to transient effects of hormones that occur throughout one's lifespan and are captured by studies administering hormones to participants, or measuring hormones in adulthood. Organisational effects occur during critical periods of development and result in long-term consequences. If oxytocin acts on these conditions through organisational effects, then administering it outside a critical period of development would result in no observable effect in an RCT. However, at present there is no evidence for the organisational effects of oxytocin on either autism or FEDs, meaning that currently there is no evidence for atypical oxytocin systems explaining the association between autism and FEDs.

A further factor (potentially related to atypical oxytocin systems; Quattrocki and Friston, 2014) that may be causally related to both autism and FEDs is interoception. Interoception can be defined as one's ability to detect and interpret internal signals of one's own body, such as hunger, satiety and heart rate (Craig, 2002). It is comprised of various dimensions upon which individuals may differ. As well as the accuracy of interoceptive perception and the amount of attention paid to interoceptive signals (Murphy et al., 2019), individuals may also vary in their evaluation of interoceptive signals (Herbert, 2020). All of these dimensions, as well as interactions between dimensions, may be clinically relevant (Garfinkel et al., 2015). Interoceptive accuracy, attention and evaluation may also be separable across interoceptive signals or 'domains', such that, for example, interoception of respiratory and cardiac signals are typical, but interoception of gastric signals atypical (Murphy et al., 2017). Both autism and FEDs have been linked to interoception in theoretical models and by empirical data, and a growing body of research appears supportive of its role in the association between autism and FEDs (Adams et al., 2022). However, it is not yet understood whether the same dimensions and domains of interoception are implicated in autism and FEDs, and many studies rely on problematic

measures of interoception, that either conflate different dimensions of interoception, or are highly susceptible to false positives or false negatives. Given that it appears as though interoception is something that can be trained (e.g., Quadt et al., 2021), it may provide a fruitful option for FED interventions, especially for those who are autistic (for a comprehensive review of the limitation of interoception measures and the potential role of interoception in the association between autism and FED, see Adams et al., 2022).

Any discussion of interoception must include consideration of alexithymia, as atypical interoception is thought to cause alexithymia (Brewer et al., 2015, 2016; Murphy et al., 2018). Indeed, empirical evidence suggests that when accounting for alexithymia, the association between autism and interoception becomes non-significant, whilst alexithymia remains significantly associated with interoception when controlling for autism (Brewer et al., 2015; Murphy et al., 2018). To our knowledge, only two papers have considered empirical data on alexithymia, autism, interoception, and FEDs. One found that in a sample of 37 participants with AN, there was no relationship between objective interoceptive accuracy, as measured by the heartbeat tracking task, and alexithymia or autistic traits, after controlling for anxiety and depression (Kinnaird, Stewart, 2020). However, most patients in this study were taking psychotropic medications, which may have affected results, and the heartbeat tracking task has frequently been suggested to be an invalid measure of interoceptive accuracy (Desmedt et al., 2018). The other study was a meta-analysis, which reported that although there was no relationship between interoception and alexithymia in healthy populations, lowered interoception was related to heightened alexithymia in both FED and autistic samples (Trevisan et al., 2019). For this analysis, interoception was defined broadly, encompassing objective interoceptive accuracy and subjective measures of interoceptive accuracy and attention, as well as interoceptive magnitude (self-reported intensity of sensations in response to experimentally induced stimuli), interoceptive detection (perception of stimuli being either present or absent) and interoceptive insight (the correspondence between subjective and objective interoceptive accuracy). Again, there are significant limitations to the majority of the methods used to assess interoception (Adams et al., 2022), as well as issues with treating interoception as a unitary construct (Murphy et al., 2019). In summary, interoception may underlie a degree of the association between autism and FEDs, however, methodological improvements are required before this possibility can be investigated.

2.2.3. *Autism directly causes FEDs*

Another proposal involving a genuine association is that the traits of autism directly result in the development of FEDs. Indeed, there is evidence that autistic traits demonstrated by the age of seven can predict FEDs arising during adolescence (Solmi et al., 2021; Carter Leno et al., 2022), and it is possible that different autistic traits are associated with different risk profiles for different FEDs (Dell'Osso et al., 2018). Under Brede and colleagues' (2020) model of how autism may lead to FEDs, autistic individuals may avoid mealtimes because of the social challenges associated with them. Spending mealtimes with others may be especially stressful to autistic individuals due to the number of implicit rules and expectations surrounding the occasion. This could lead to an avoidance of mealtimes, and the formation of negative associations with food leading to restrictive FEDs (see also Bourne, 2022), as well as a limited ability to model norms of healthy eating habits by observing peers (Higgs and Thomas, 2016; although it should be noted that modelling from peers is not necessarily advantageous if the peers being observed have disordered eating themselves).

Another way in which autism may directly cause FEDs relates to the sensory hypersensitivities observed in many autistic people. Hypersensitivity specifically to particular food textures, tastes, smells or appearances has been reported to occur in approximately 90% of autistic children and 95% of autistic adults (Marshall et al., 2015; Crane et al., 2009). Given that hypersensitivity to certain foods is predictive of food avoidance or refusal (Lane et al., 2010; Nimbley et al., 2022), it may underlie restrictive FEDs. Consistent with this idea is the finding that AN patients report a sensory profile of hypersensitivity combined with avoidance (Zucker et al., 2013). Interviews with autistic women with FEDs also support this notion (Kinnaird, Norton, 2019), with many reporting that they would avoid foods because of sensory issues. Conversely, autistic individuals who are highly sensation seeking might eat too much of certain foods that have particular textures or tastes, thus resulting in bingeing behaviours. Differences at various levels of sensory processing may result in FEDs, and the specific FED that arises may depend on the way in which the individual is affected. Whilst heightened perception, aversive processing and an avoidance reaction could conceivably result in ARFID, reduced perception, positive processing and an approach reaction may lead to BED. Interestingly, this may suggest that for a number of autistic patients diagnosed with AN, a diagnosis of ARFID may be better suited, given the role of sensory factors in their symptoms and the absence of weight and shape concerns.

Following their interviews, Kinnaird and colleagues conducted further studies specifically focused on sensory perception in autism and FEDs. They found that among a group of participants with AN, those with higher AQ traits had significantly higher scores for smell, vision, texture and total sensitivity, although not taste, sound and touch. However, in a regression analysis, autistic traits were related to sensitivity to smell only after accounting for other factors such as depression. This latter finding prompted the suggestion that the increased sensitivity seen in those with high autistic traits may in fact not be due to autism, but instead to a co-occurring third factor (Kinnaird, Dandil, 2020).

In addition to issues with sensory sensitivity, gastrointestinal symptoms are reported by up to 85% of autistic young people (Hsiao, 2014). There is also a high reported overlap between gastrointestinal conditions and FEDs. Although the link between gastrointestinal conditions and FEDs is most typically explored within the context of FEDs causing gastrointestinal conditions (Santonicola et al., 2019), there is emerging evidence of a bidirectional relationship for certain conditions, such as between coeliac disease and AN (Avila et al., 2019; Satherley et al., 2015). For example, Sainsbury and colleagues (2013) found that greater gastrointestinal symptoms at diagnosis of coeliac disease predicted the development of dysfunctional restrictive eating patterns due to anxiety about gastrointestinal symptoms.

The rigid and repetitive behaviours associated with autism could also lead to restrictive FEDs. Food consumption may be highly selective, and any departure from fixed mealtime routines may lead to no food being consumed at all. Furthermore, autistic individuals may attempt to manage their intolerance of uncertainty by controlling aspects of their life such as diet, which provides predictability. This may also cause food neophobia, as they avoid eating any foods they do not deem as "safe" (Wallace et al., 2018). In autistic children, strong associations were reported between feeding and eating behaviours and restricted and repetitive thoughts and behaviours (Johnson et al., 2014; Zickgraf et al., 2022). A recent scoping review on the association between ARFID and autism highlighted that certain traits appeared to co-occur in autistic children and those with disturbed eating patterns, such as a preference for routine, cognitive rigidity and intolerance of uncertainty, which could manifest as an insistence on the same food or drink, or mealtime routines (Bourne et al., 2022).

Cognitive rigidity in particular could result in the adoption of inflexible and restrictive internalised rules around eating and weight, which result in more disordered eating (Brown et al., 2012). Indeed, cognitive rigidity has been demonstrated to relate to eating disorder symptoms, and has been found specifically in AN patients (Arlt et al., 2016; Tenconi et al., 2023). Similarly, eating behaviours could become a special interest for an autistic person, as they may become fixated on habits such as counting calories or macronutrients, or develop an obsession with eating certain foods.

2.2.4. Autism indirectly causes FEDs

It may be that there is a genuine association between autism and FEDs because certain features of autism *indirectly* increase one's risk of developing an FED; i.e., whether or not autistic traits result in FEDs depends on the presence of mediating factors. This is perhaps best conceptualized within a wider developmental psychopathology framework, in which development is both active and dynamic, with both environmental- and individual-level factors interacting to determine mental health outcomes throughout one's lifespan (Rutter and Sroufe, 2000). Mental health states may evoke certain responses from the environment which, in turn, continue to shape one's mental health. Being autistic may evoke certain experiences from the environment, which may interact with individual-level factors to confer risk to FEDs. The developmental stage at which one is exposed to these risk factors may also have an effect. For example, body image is particularly salient during puberty, so may be a critical developmental period when considering eating disorders. Within the context of autism, adolescence may mark a period of increased bullying for not "fitting in," which in turn may increase negative affect, thus increasing risk for eating disorders. For a more comprehensive outline of this idea, see Rutter and Rutter, (1993) and Mandy and Lai, (2016).

Autism may indirectly play a causal role in the development of FEDs via emotion dysregulation. Overwhelming sensory sensations, a departure from routine, intolerance of uncertainty, bullying or associated mental illnesses like depression and anxiety (Hollocks et al., 2019) may all result in extreme negative feelings, and this may lead to higher negative emotionality in autistic than non-autistic people (Mazefsky, 2015). The difficulties with social communication that many autistic individuals experience may restrict them from accessing protective factors that confer resilience such as a social support network, or group physical activities. Autistic individuals may also be more likely than neurotypicals to attempt to manage these negative emotions through harmful strategies (Mazefsky, 2015). For example, starvation or over-eating may be used to numb overwhelming negative feelings (Brede et al., 2020). It has been shown that caloric deprivation is associated with the downregulation of physiological arousal (Miller et al., 2003), and over-eating is a frequently used method of emotion regulation (de Campora et al., 2016). If emotional dysregulation is part of the causal chain between autism and the development of FEDs, then there is potential for intervention to address this. In autistic individuals with FEDs, a particular focus could be given to identifying healthier ways to regulate emotions in therapy, such as reappraisal of negative thoughts and mindfulness, which evidence suggests autistic individuals are less likely to engage in spontaneously (Samson et al., 2015). More broadly, societal interventions to reduce autism-linked negative emotional states (through antibullying campaigns, the design of environments to be more inclusive for those with sensory sensitivities, etc.) may also act to reduce the link between autism and FEDs via emotional dysregulation.

It is important to note that it can be very difficult to identify indirect effects in general, but particularly of neurodevelopmental conditions such as autism. Many of the autistic traits that could directly cause FEDs may also have an indirect effect on FEDs. For example, rigid thinking could directly cause restrictive eating behaviours, or it may lead to emotional distress, which is then managed by restrictive eating. Also difficult to disambiguate is the case in which autism causes FEDs (directly or indirectly), versus the case in which autism and FEDs have common causal factors. For example, if certain genes are responsible for both autism and FEDs, then it would logically entail that autistic traits correlate with FED symptoms, whether autism causes the FED or not.

The crucial question then becomes whether the genes cause FEDs *via* the autistic traits, or independently of them. In principle this question can be answered via an intervention study in which action is taken to reduce the degree of autistic traits that are specifically implicated in the development of FEDs (rather than general autistic traits), to see if doing so reduces the chances of developing an FED. In practice, there is limited evidence of an acceptable quality to suggest interventions reduce autistic traits (Green and Garg, 2018), but more success may be achieved if one has a specific model of how the autistic traits cause the FED (e.g., via emotion dysregulation) and specifically intervene to impact the hypothesized causal link (e.g., with emotion regulation training). If the intervention reduces the odds of developing a FED, then we could conclude at least some genetic influence acts via the autistic traits. If there is no effect of the intervention on the odds of developing an FED, however, then the converse cannot be concluded. No effect of an intervention to reduce autistic traits may simply mean that the influence of autistic traits has already occurred prior to the intervention. The fact that autism is a neurodevelopmental condition means one cannot intervene before the autism may have influenced later FED outcomes (note: intervention may be possible before autism symptoms become apparent, but this is not the same as before autistic traits may have impacted the odds of developing an FED). Obviously, it should be acknowledged that whether interventions to reduce autistic traits are either ethical or necessary is a controversial issue. Numerous organisations and individuals within the autistic community, including those aligned with the neurodiversity movement, have argued that autism should not be treated as a disorder, and thus should not be seen as something to "cure" (see Kapp, 2020). Therefore, this approach of attempting to minimise traits associated with autism may be seen as pathologizing a cognitive difference that should instead be accepted.

2.2.5. Moderating factors

The strength of association between autism and FEDs is likely to be subject to moderation by several factors, which provides opportunity for intervention. One such moderator could be exposure to certain ideas about food and weight. Given the societal standards of thinness and the focus on diet culture in popular media (Marks et al., 2020), adhering to these values with restrictive eating may seem to autistic individuals as a way to belong socially.

Another example relates to the rigid, absolute cognitions associated with autism (Stark et al., 2021), which may result in harmful thoughts about food or diet if the autistic individual has been exposed to certain ideas about food. For example, restrictive FEDs in autistic individuals may be driven by beliefs that all fat is bad (Brede et al., 2020), or being skinnier is more attractive. Equally, extreme interpretations of ideas such as food being needed for you to grow "big and strong" may lead to binge eating. In each of these examples, the ideas and knowledge about food that autistic people have been exposed to act as a moderating factor between their autistic traits and FED symptoms, which opens the potential for intervention. For example, autistic children could be taught more healthy and balanced ideas about food as part of the early support they receive for their autism. Moreover, parents can be warned to be mindful of the way they speak with their children about food.

Other moderating factors could include the age of receiving an autism diagnosis (Mandy et al., 2022), gender, how well the environment is suited to the autistic individual, and stressful life events, among many potential others. With respect to the age of autism diagnosis, if one receives a diagnosis earlier then the direct effects of autism on FED may be reduced, for example by enabling parents to identify foods that are less aversive for the child and adapting mealtimes. It may also reduce the indirect effects of autism, for example, early intervention for their autism may equip the children with non-food-related emotion regulation strategies. Later diagnosis means these opportunities for positive intervention are missed.

Some factors are likely to operate as both moderators and mediators. This can be illustrated by the example of depression. Depression may be a mediator, in that one's experience of being autistic may cause the development of depression, which in turn plays a causal role in the development of an FED. It may also be a moderator, in that it may emerge independently of autism, but increase the likelihood of developing an eating disorder compared to autistic individuals without depression. Determining which role depression plays in any individual with both autism and FEDs is likely to require clinical interviews to

determine whether autism is causally involved in the depression.

3. Further questions and considerations

3.1. Does the relationship between autism and FEDs differ for specific eating disorders?

It is reasonable to assume that the strength of the association between autism and FEDs, and the mechanisms underlying the association, may vary depending on the specific FED. Some studies have found no difference in the prevalence of autism across FED diagnoses. Vagni and colleagues (2016) found no difference across female patients with AN, BN, or BED. Other empirical work suggests that although the direction of the relationship remains the same across FEDs, the strength of this association varies. Dell'Osso and colleagues (2018) found that within non-autistic adults with FEDs, autistic traits were higher in those with AN compared to patients with binge-purging behaviours. Similarly, Numata and colleagues (2021) found that autistic traits were higher in FED patients who did not show self-induced vomiting, and highest in patients with BED. Thus far, most of these studies are limited by small sample sizes, with only thirty to forty participants in each FED group.

Overall, there is a paucity of research into FEDs other than AN and BN and their association with autism. Pica and rumination may be especially linked to autism, however, they have so far been largely neglected in the literature. The reason for this lack of research may be because studies of autism predominantly recruit participants without intellectual disability, and intellectual disability is known to be associated with both pica and rumination (Gravestock et al., 2000). Given that a high proportion of autistic people do have an intellectual disability, with one review suggesting rates are as high as 50% (Russell et al., 2019), further research should aim to explore the association between autism and all FEDs in more representative samples.

The recency of ARFID's addition to the DSM means that there is currently a lack of research on its association with autism. This is especially concerning, as the importance of sensory issues and emotional dysregulation in both conditions makes a causal connection a possibility, and available research indicates a high rate of co-occurrence (Sanchez-Cerezo et al., 2023). In addition, it may be the case that for individuals diagnosed with both autism and FEDs other than ARFID, a diagnosis of ARFID would better reflect their symptoms. Indeed, one qualitative study supports this conjecture, with several participants with co-occurring AN and autism reporting that they lacked the motivations necessary for an AN diagnosis, such as a desire for thinness and weight loss. Instead, motivations such as a need for control and sensory difficulties were cited as more important to them (Kinnaird, Norton, 2019).

A recent study by Inoue and colleagues (2021) demonstrated that distinct causal mechanisms may underlie the associations between autism and different FEDs. They recruited a sample of children with either AN or ARFID. Among those who had AN, the group had higher parent-reported autistic traits than age-matched healthy controls, however there were no significant correlations between autistic traits and FED symptoms. They speculated that this may be because autistic traits were associated with the onset, but not the maintenance of AN. The reverse was found in the ARFID group, where there were no significant differences in parent-reported autistic traits between the ARFID group and the age-matched healthy controls, although autistic traits did correlate with eating disorder symptoms within the ARFID group. However, it is important to note that the EAT26 was used to measure eating disorder symptoms (Maloney et al., 1989), which includes subscales about preoccupations with thinness, dieting, social pressure to eat, and purging, which may not be as applicable to ARFID. These findings can be understood within a developmental psychopathology framework, whereby common risk factors are shared across FEDs, and their interactions with other environmental or individual variables, or the stage of development at which they are present, confer particular risk for a specific FED. This is illustrated in Fig. 2.

In terms of the potential mechanisms highlighted in this paper, different mechanisms may be more applicable to certain FEDs than others. For example, emotional dysregulation and interoceptive difficulties may be more likely to result in AN or BED, whilst sensory sensitivities may result in ARFID. It could also be that there are similar risk factors for different FEDs, but that the expression of these risk factors depends on environmental triggers, which vary across FEDs. For example, absolute thinking could result in either BED or AN, depending on the type of information one has been exposed to.

Even within specific FEDs, it may be possible to identify different causal pathways for the association with autism. For example, ARFID can be split into three separate facets: a lack of appetite, sensory issues, and anxiety about adverse consequences of eating (e.g., fears of choking on food; Thomas et al., 2017). Whilst the first could be a product of atypical interoception shared by both autism and ARFID, the second may relate more to the sensory sensitivities associated with autism, and the third may relate more to the role of co-occurring conditions such as anxiety.

3.2. Longitudinal research

There is a pressing need for longitudinal research to assess the mechanistic links discussed here more thoroughly. Indeed, it is a limitation of this paper that much of the research discussed relies on cross-sectional studies with fewer than a hundred participants, which is not sufficient to explore the complexity of all the potential mechanisms.

One benefit of longitudinal work is that one can explore how the nature of the relationships between autism and FEDs change over time.

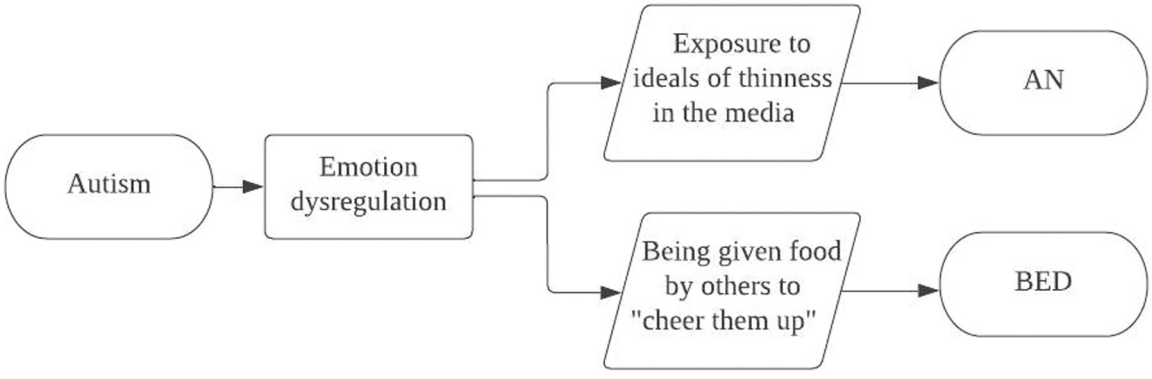

Fig. 2. Note: A simplified model to illustrate the principles of the developmental psychopathology framework. Here autism leads to emotion dysregulation, but the particular Eating Disorder engendered by the emotional dysregulation depends on specific environmental influences (here the attitudes to food to which the individual is primarily exposed to).

The association between autism and FEDs may vary according to age, with one study highlighting a greater association in older participants (Vuillier et al., 2020). However, the cross-sectional design of this study does not exclude the possibility of cohort effects. It is also possible that the time of onset of FEDs may differ between autistic and non-autistic people, especially if there are slightly different causal pathways underlying the development of the FEDs. Although eating problems are more common in autistic compared to typically developing children (Baraskewich et al., 2021), it is currently unknown whether these eating problems develop into FEDs with age. If they do, this could lead to an earlier emergence of FEDs in autistic compared to non-autistic people.

Longitudinal research would allow for a better understanding of whether autistic traits cause the onset or maintenance of FEDs. To establish whether autistic traits could cause the onset of FED, researchers can test whether autistic traits at one time point (for example, mid-childhood) predict the emergence of an FED at a second time point (for example, mid-adolescence) within a sub-sample of those who have never had an FED before. Ideally, this would be replicated across time points, and assessed by methods such as a cross-lagged panel analysis. To establish whether autistic traits maintain FEDs, researchers could separate a sub-sample of participants who already have an FED and assess whether autistic traits predict either the length of symptoms/treatment or changes in FED symptoms.

3.3. Do the profile of FEDs differ in autistic versus non-autistic people?

Another important question is whether the way FEDs manifest is markedly different in autistic versus neurotypical people. As well as some potential causes being specific to autism, as this paper discusses, there is also the possibility that the symptoms of the FEDs themselves are different in autistic people. If the causes and symptoms were different in autistic individuals, this may explain why traditional methods of FED treatment are not as effective in this patient group (Tchanturia, 2021). As with most issues discussed here, it is clear more research is required.

3.4. Clinical implications

There are certain clinical implications that can be drawn from this paper. First, it highlights a need to screen FED patients for co-occurring autism at the point at which they receive their FED diagnosis. The response to treatment for FEDs may vary in autistic and non-autistic individuals, and thus recognition of autism in patients with FEDs could enable more targeted interventions to be employed.

Given that autistic FED patients benefit less from conventional interventions than non-autistic individuals (Babb et al., 2022; Tchanturia, 2021), further work is needed to develop services to make them more accessible and effective (Babb et al., 2022; Li, Hutchings-Hay et al., 2022). One such intervention is the PEACE pathway, which has shown initial indications of efficacy (Li, Halls et al., 2022). Qualitative (e.g., Babb et al., 2021; Brede et al., 2022) and quantitative (Babb et al., 2022) evidence suggests that relatively simple, low-cost modifications can be made to the physical and social clinical environment to make FED services more accessible to autistic people, such as modifying the sensory environment in clinic; adapting how staff communicate with autistic clients; and adjusting the way in which therapies are delivered. It is also possible that autism-specific interventions will need to be developed and tested, to accommodate the fact that autism-specific mechanisms may cause and maintain some FEDs (Brede et al., 2021).

In line with the discussion raised in both Section 2.1.4 (Conceptual or Measurement Overlap) and Section 2.2.1 (Eating Disorders are a Female Manifestation of Autism), clinicians should be made more aware of the overlap between FEDs and autism, and any potential diagnostic bias with regards to gender. This will help to reduce the likelihood of, for example, a male missing out on appropriate support for a FED because it is assumed to be a manifestation of autism.

In general, a thorough empirical testing of the mechanisms proposed in this paper will allow a greater understanding of how eating disorders emerge, both generally, and specifically within autistic individuals. This understanding will facilitate better treatment. For example, identification of a mediating variable between autism and FED could mean that the mediating variable could be treated to reduce the occurrence of FEDs in the autistic population.

4. Conclusions

High co-occurrence has been observed between autism and FEDs, and where this co-occurrence exists, clinical outcomes of FEDs have been demonstrably worse. There are many possible explanations for this observed association between autism and FEDs, which this paper has systematically outlined. It is unlikely that FEDs are purely a female form of autism, however, autistic females have an especially high risk of developing FEDs. At present, although research on common causal factors is limited, the existing research suggests these may exist. There is slightly more research to suggest that autistic traits, such as sensory hypersensitivity, insistence on routine, absolute thinking and social impairments may either directly or indirectly cause FEDs. However, it is also very possible that the observed association may be due to third variables, such as alexithymia, depression, or anxiety. In addition to the potential for these third variables to wholly account for the relationship, it may also be that they influence the association between autism and FEDs, either through moderating or mediating the autism-FED association. Whilst starvation may enhance the degree of overlap between FEDs and autistic traits, it appears insufficient to fully explain the association between the conditions. Similarly, although there is likely some degree of conceptual overlap, this also does not appear sufficient to fully explain the association. Despite the quantity of existing research on the association between autism and FEDs, it has largely lacked a unifying theoretical framework. By outlining the potential mechanisms by which autism may be associated with FEDs, and providing direction for future research (see Appendix 1), we hope to facilitate the empirical testing of these pathways, to garner a better understanding of the association between autism and FEDs and improve clinical outcomes.

Appendix A

Mechanism	Proposed method(s) of testing
Artefactual	
Consequences of FEDs lead to traits of autism	Longitudinal studies looking at traits of both autism and abnormal eating behaviour, as well as autism and FED diagnoses, from early childhood to adulthood.
Factors that covary with autism (including alexithymia)	Research on whether autism fits into a p factor model (do symptoms of autism correlate with other psychiatric conditions?).Mediation analyses of covarying factors such as depression. Network analysis.2×2 design splitting the influence of other factors and autism. Including alexithymia specifically as a covariate in any study of autism.Intervention studies.Longitudinal epidemiological studies using random intercept cross-lagged panel models, or traditional cross-lagged panel models.
There is conceptual overlap in the definitions	Explore clinician's biases with imaginary case notes with demographic features either included or omitted.Research employing the RDoC framework.Network analysis.Combining research using the above two methods with research based around a medical model.

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(*continued*)

Mechanism	Proposed method(s) of testing
– Diagnosis	Factor analyses of questionnaire measures of autism and FEDs.Latent class analyses of questionnaire measures of autism and FEDs.
– Measurement	Discriminant analyses of questionnaire measures of autism and FEDs.
Genuine	
FEDs are the female version of autism	Use of measures of autism that are designed specifically to capture female specific presentations.Determining whether autistic females are at a greater risk of developing various types of FEDs than males.
Common causal factors	Further quantitative genetic research on FEDs other than AN, and on the shared genetic basis between autism and FEDs.Further neurological research on brain alterations in autistic *females,* and FEDs other than AN.Research into organisational influences of oxytocin on autism and FEDs.Intervention studies that seek to train interoception.
Autistic traits directly lead to FEDs	Mediation and moderation models.
Autistic traits indirectly lead to FEDs	Mediation and moderation models.Longitudinal research based around a developmental psychopathology framework.Intervention studies* - e.g.: does teaching better methods of emotional regulation reduce ED symptoms in those with autism?

Note: *We would like to note that the use of interventions to reduce autistic traits generally is extremely controversial, and therefore we have not included this in our table of suggested future research. Instead, we refer specifically here to intervening at the level of potential mediating factors (here the example is given of emotional regulation). Please see Section 2.2.4 for a more comprehensive discussion of this issue.

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