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Anorexia Nervosa and Comorbid Autism Spectrum Disorders

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Abstract

Purpose of review

Traits of autism spectrum disorder (ASD) are overrepresented among individuals with anorexia nervosa (AN) and may also moderate the behavioral manifestation of AN. This review aims to provide an overview of AN and comorbid ASD.

Recent findings

Elevated ASD traits do not seem to precede AN among some individuals but are rather related to the illness stage. However, studies have suggested that there are ASD-specific mechanisms for developing AN in a subgroup of individuals with AN. Pronounced traits of ASD and diagnosed ASD are associated with illness prolongation and poorer outcomes in AN, and individuals with AN and elevated ASD traits may benefit less from many of the current treatments. Studies do not support a specific genetic relationship between ASD and AN.

Summary

Recent research encourages the improved recognition of elevated ASD traits in individuals with AN and provides grounds for developing tailored treatments for those with this comorbidity.

Keywords: anorexia nervosa, autism spectrum, autism spectrum disorder, disordered eating, eating disorders, eating and feeding disorders

INTRODUCTION

Autism spectrum disorder (ASD) is a developmental condition characterized by deficits in social interaction and communication, as well as repetitive, stereotyped behavior and interests. ASD is often associated with atypical sensory processing (hyper- or hyposensitivity) [1], poor motor skills, and disturbed eating behaviors, such as food selectivity and routines related to eating [2]. Anorexia nervosa (AN) is an eating disorder whose core characteristics include restriction of energy intake leading to low body weight, intense fear of gaining weight, or persistent behavior that interferes with weight gain and a disturbance in the way in which one's own body weight/shape is experienced [1]. Both elevated ASD traits and diagnosed ASD are overrepresented among individuals with AN [3–8]. Among individuals with AN who fulfill the diagnostic criteria for comorbid ASD, the traits have, by definition, already existed in childhood. In addition, individuals in the acute stage of AN tended to have more ASD traits than before the onset and after recovery from AN. This review provides an overview of AN and comorbid ASD, focusing on ASD traits underlying the development of eating disorder symptoms, discussing possible explanations for the comorbidity, and ASD-specific mechanisms predisposing individuals to AN.

RECENT RESEARCH DEVELOPMENTS

Prevalence of ASD among individuals with AN In Danish and Swedish registry-based studies that identified those who had had ASD diagnoses (before or after AN diagnosis) was 1.3–2.5% in women with AN [9,10**]. The former of these studies, based on nationwide Danish registers, reported the prevalence of ASD in the female controls to be 0.6% [9]; individuals with a diagnosis of AN had a highly elevated risk of receiving a later diagnosis of ASD (hazard ratio =

15.08, 95% confidence interval 12.23–18.58), with the risk being higher for male than for female individuals. Studies that have actively screened for ASD among individuals with AN have consistently reported its prevalence to be between 10% and 30% in female patient samples [4,5,7,8,11,12**]. This is in line with a recent meta-analysis reporting a marked difference in the prevalence of ASD in female individuals between active and passive ascertainment studies [13], suggesting substantial under-detection of ASD in girls and women.

Difficulties in social cognition, interaction, and communication Individuals with acute AN have been shown to have difficulties in emotion recognition, i.e., in the ability to understand and recognize others' emotional expressions, whereas results in recovered individuals are inconsistent [14*,15,16]. In addition, deficits in the theory of mind, which refers to the ability to understand and predict others' mental states, intentions, and behavior, and low cognitive empathy have been reported among individuals with acute AN [17]. Recent studies further reported that difficulties in emotional recognition abilities and low empathy exist specifically in individuals with AN and elevated ASD traits, whereas in individuals with AN and low ASD traits, emotion recognition abilities and empathy are similar to those in healthy controls [14*,18,19]. It has therefore been suggested that deficits in emotion recognition and empathy are not features of AN, but instead reflect the presence of ASD traits in individuals with AN. A distinct subgroup of individuals with acute AN has also been shown to exhibit deficits in social communication and interactions similar to those observed in ASD [6,7,11]. These deficits are characterized by a reduced level of expressive behavior, lack of gesture use, less effective social overtures, and difficulties in social responses. These ASD-like characteristics in social cognition and behavior in individuals with AN are proposed to predispose individuals to social difficulties, such as problems in friendships, loneliness, limited social networks, poor overall

social functioning, and bullying, as well as low self-confidence and social anxiety, all of which are known risk factors for developing AN [20–22].

Cognitive and behavioral inflexibility Cognitive inflexibility has been shown to manifest specifically among individuals with AN and elevated ASD traits [23]. Numerous studies have also reported that individuals with AN have difficulties in set-shifting, a component of cognitive flexibility [24–27]. Similar to deficits in social interaction, difficulties in set-shifting have been found to be pronounced in the acute stage of AN, whereas findings in recovered individuals are inconsistent [28–30]. However, recent meta-analyses showed that the majority of studies that investigated set-shifting in recovered individuals reported that set-shifting difficulties persist after recovery [3,25]. Difficulties in set-shifting may contribute to inflexible behavior commonly seen in AN, such as compulsive rituals and routines around eating, weight, exercising, body checking, obsessive behavior, and restrictive thinking style [31]. Not surprisingly, cognitive inflexibility has been reported to be associated with more severe rituals around eating among individuals with AN [32]. Cognitive inflexibility is also suggested to be related to excessive self-control and perfectionism, which are common traits in individuals with AN [31,33]. Inflexible behavior and thinking style may also hamper recovery, and it has been proposed that cognitive rigidity is an important factor contributing to treatment resistance and maintenance of eating disorders [12**,31].

Atypical sensory processing Individuals with acute AN have been shown to have high sensory sensitivity, and high sensation avoiding and atypical sensory processing appear prominent specifically among individuals with AN and high ASD traits [34*,35,36]. Sensory symptoms have been reported to be associated with the severity of eating disorder symptoms in individuals

with acute AN [36]. In two recent qualitative studies, including participants with both AN and ASD, participants described that they had food-specific sensory sensitivities related to food texture, taste, smell, or mixing foods and that restrictive eating was often related to the sensory properties of food [12**,37]. In addition to eating disturbances, sensory processing is also involved in the subjective experience of body schema. Therefore, it is plausible that atypical sensory processing may contribute to disturbed body image, for instance, through proprioception and kinesthesia (sensing body positions and boundaries of the body) [12**,35]. A meta-analysis showed that individuals with current and past AN had multisensory impairments in body perception, including deficits in tactile and proprioceptive sensory processing [38]. To conclude, sensory processing atypicality may underlie some of the core symptoms of AN [34*].

Trajectory of ASD traits Mixed results have been reported for the course of elevated ASD traits in AN; elevated traits have been reported to both precede the onset and remain after the recovery of AN [3,4,15,32,39*,40], and that individuals who later develop an eating disorder did not have elevated ASD traits in childhood nor post-disorder [10**15]. A recent population-based twin study did not find evidence of elevated ASD traits at age 9 years among those who later developed AN, but elevated ASD traits at age 18 years in individuals with acute AN were observed [10**]. The authors suggested several explanations for their findings, including that ASD traits in cognitively able individuals did not manifest at age 9 years because of camouflaging of symptoms and lower social demands in childhood, that the parental interview used in the study did not capture ASD traits in girls because of the putative gender-biased criteria of ASD, and that elevated ASD traits are present only in a subgroup of individuals with AN. Another recent population-based cohort study reported that greater

childhood ASD traits at age 7 years were associated with eating disorder symptoms at age 14 years [39**]. The study also reported a dose-response effect, where elevated ASD traits were associated with more frequent eating disorder symptoms, implying that ASD traits may present a risk factor contributing to the development of eating disorder symptoms.

It has also been proposed that elevated ASD traits arise as a consequence of acute eating disorder-related processes, such as starvation. However, studies consistently show a lack of association between the level of (elevated) ASD traits and (lower) body mass index (BMI), and that there are no BMI differences in AN when comparing those with low ASD traits to those with high ASD traits or diagnosed ASD [8,19–21]. To our knowledge, there is no evidence of an association between starvation and ASD traits.

It is also important to note that studies investigating recovered individuals do not include non-recovered individuals; in patient samples, a marked number of individuals with AN do not achieve full recovery, and ASD traits are associated with poorer outcomes [8,20,21,41]. It is thus possible that studies investigating recovered individuals have fewer participants with AN and elevated ASD traits if the recovery rate is lower in this population.

Familial aggregation and genetic studies of ASD and AN In a nationwide Danish registry study, a family history of ASD was associated with an increased risk of AN and *vice versa*, implying a shared genetic background between the disorders [9]. However, the risk of developing AN also increased in individuals with family members with major depression or other mental disorders. The study also failed to find a dose-response effect for familial ASD according to the degree of shared genetic material for vulnerability to AN. The authors concluded that the

results did not support the hypothesis that ASD acts as a specific genetic or familial risk factor for developing AN. Based on national registers, the study included only diagnosed disorders, thus covering a more severe spectrum of disorders. In other studies, elevated ASD traits, such as deficits in cognitive flexibility, poor theory of mind, and weak central coherence, have been reported among non-affected sisters and mothers of probands with AN, suggesting familial aggregation of ASD traits in individuals with AN [27,32,42,43].

Recent genome-wide studies, including several thousands of diagnosed cases with ASD and AN, have not reported significant genetic overlap between the disorders beyond what psychiatric disorders in general share [44,45]. Instead, AN, obsessive-compulsive disorder, and schizophrenia have demonstrated significant sharing of genetic risk factors, and the same is true for ASD and schizophrenia [44,45]. To conclude, the existing data do not support a specific genetic relationship between ASD and AN.

ASD-specific mechanisms for developing AN Among those who fulfill ASD criteria, ASD traits have, by definition, presented early in development, and AN is likely to be secondary to ASD. Two recent qualitative studies including participants with AN and ASD have investigated autism-specific mechanisms for developing AN [12**,37]. Almost all participants in these studies reported that body image issues were less relevant in their eating disorder, but rather that their ASD-related features had predisposed them to AN. Participants described that social and emotional difficulties resulted in self-image problems and made participants feel confused, faulty, or different. Furthermore, individuals with AN with ASD described that their eating disorder symptoms arose primarily from an inflexible thinking style and increased sensory sensitivity. The authors concluded that ASD-related difficulties contribute to the

development of eating disorder symptoms through both direct and indirect pathways. A direct pathway involves aspects related to the core symptoms of ASD, such as sensory processing issues, inflexibility, and social difficulties (see Table 1). Additionally, via an indirect pathway, ASD-related difficulties were suggested to result in negative emotional consequences, low self-esteem, and feeling different, which can lead to the use of restrictive eating as a maladaptive coping strategy. To conclude, ASD may predispose individuals to develop an eating disorder and AN in individuals with high ASD traits is likely to differ from AN in individuals with low ASD traits.

Treatment outcome and treatment modifications Elevated ASD traits are associated with a poorer prognosis of AN. Among individuals with AN and elevated ASD traits, the treatment time for AN and illness duration have been shown to be higher [8,20,21,32,46–48]. These individuals are also more often admitted to inpatient treatment, and antipsychotic medication is used more frequently than in individuals with AN and low ASD traits [20,21]. Additionally, individuals with AN and high ASD traits have been shown to terminate the treatment of AN prematurely more often than those with low ASD traits [48]. Long-term follow-up studies have shown that in those who benefit from treatment, ASD traits are not associated with physical recovery reflected by BMI [20,21]. However, poorer psychological recovery has been reported to be associated with elevated ASD traits [21] and increased residual symptoms after treatment, such as higher weight and shape concerns, dietary restrictions, poorer psychosexual and mental state, problems in peer relationships, and general difficulties [20,21]. These residual symptoms may increase the likelihood of future relapse [21]. It has been proposed that individuals with AN and elevated ASD traits benefit less

from current treatment methods, and therefore, treatment modifications that take into account elevated ASD traits are needed [49**].

A recent study aimed to develop a treatment pathway for individuals with AN and ASD [49**]. Treatment modifications were planned specifically around special sensory needs and individual modifications of psychological treatment (Table 1). In a follow-up study, the authors reported that this treatment pathway for patients with AN and ASD was associated with reduced treatment length and was cost-effective [47]. The authors concluded that it is crucial to recognize elevated ASD traits in AN, educate healthcare professionals on the comorbidity of the two disorders, and develop further treatment modifications. Other recent studies found that individual cognitive remediation therapy (CRT) was beneficial for those whose AN is accompanied by high ASD traits [50*,51]. In contrast, one study reported that among individuals with AN and elevated ASD traits, the CRT group did not improve with respect to flexibility or motivation [52]. Thus, individuals with AN and elevated ASD traits could benefit from individual treatment adaptations that take into account their neuropsychological characteristics and resemble neuropsychological rehabilitation targeted at ASD.

CONCLUSIONS

This review aims to present recent research developments regarding the comorbidity of AN and ASD. Individuals with AN often have widespread ASD-like characteristics, such as deficits in social perception, interaction and behavior, cognitive inflexibility, and atypical sensory processing. These ASD-like characteristics appear to be prominent in acute AN, and to some extent, these characteristics may be related to the illness stage. However, in a subgroup of

individuals, autism-related traits appear to predispose individuals to developing AN. It is important to recognize these individuals, as it may lead to the development of tailored treatments and improved outcomes in AN.

KEY POINTS

- Many individuals with AN have elevated ASD traits, such as deficits in social perception, interaction, social behavior, cognitive inflexibility, and atypical sensory processing.
- In some individuals, elevated ASD traits may be a consequence of acute eating disorder-related processes.
- A subgroup of individuals with AN fulfills the diagnostic criteria for ASD. In this subgroup, ASD traits were already present in childhood.
- Elevated ASD traits in individuals with AN are associated with longer treatment periods, worse treatment outcomes, prolonged AN, and a higher likelihood of chronic eating disorders.
- Individuals with AN and comorbid ASD may benefit less from many of the current treatments; therefore, tailored treatments should be developed.

Table legends

Table 1. Characteristics of autism spectrum disorders in anorexia nervosa, and implications for treatment.

Abbreviations: ASD = autism spectrum disorder, AN = anorexia nervosa, CRT = cognitive remediation therapy [12**,19,34*,35–37,49**,50*,53*].

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Conflicts of Interest

Conflicts of Interest: None.

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* Qualitative study analyzing the experiences of caregivers of individuals with AN and ASD. The themes discussed included the ways that ASD symptoms were interlinked with AN, quality of problems that individuals with ASD and AN face in treatment, and how AN treatment could be improved by taking ASD traits into account.

Table 1.

ASD characteristics in individuals with AN	Ways that symptoms contribute to the development of AN	Possible difficulties faced in treatment	Possible treatment modifications
Difficulties in social cognition, e.g., difficulties in emotion recognition, low cognitive empathy, poor theory of mind	• Predisposed to negative experiences that increase the risk of low self-esteem and feelings of being "faulty" and loneliness • Increased likelihood of developing social anxiety • Camouflaging (masking of social difficulties) may cause exhaustion and loneliness	• Difficulties in communicating emotions • Communication problems may hamper the therapeutic relationship • Literal interpretation may cause misunderstandings	• Individual CRT or neuropsychological rehabilitation that takes individual needs into account • Working with emotion identification • Clear and logical communication style • More flexibility in therapeutic sessions • Using alternative methods of communication, e.g., writing
Cognitive inflexibility and rigidity	• Increased likelihood of developing rigid eating habits • Increased likelihood of being interested in food- and weight-related counts	• Difficulties to distinguish which rigid routines occur due to ASD or AN • May hamper recovery by undermining the ability to make changes	• Individual CRT or neuropsychological rehabilitation that takes individual needs into account • In some cases, it may be better to accept routines than try to change them • A predictable schedule in treatment
Atypical sensory processing	• Food-related sensory sensitivities, i.e., texture, smell, taste, and temperature, may lead to avoiding some foods • Eating-related sensations, e.g., stomach fullness, may feel very unpleasant and cause avoidance of eating • Low sensation sensitivity may make it challenging to recognize hunger and satiety • May contribute to body image disturbance	• Challenges with food plans that do not take into account the sensory needs • May be hard for healthcare professionals to distinguish whether food avoidance is motivated by sensory sensitivity • Treatment environment may cause sensory overload	• Modifications of food plan during the treatment, e.g., soft texture of food, no mixing of foods • Modification of treatment environment, e.g., minimizing background noises and using neutral colors • "A sensory box" that includes tools helping with sensory overload, e.g., earplugs