Autism Spectrum Disorder and Anorexia Nervosa in female adolescents: A study of comorbidity and the role of set-shifting abilities

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Abstract

Background: Symptoms of ASD in patients with AN seem to be more prevalent than previously thought. Therefore, investigating the comorbidity between anorexia nervosa (AN) and autism spectrum disorder (ASD) is a growing field of study. The implications of ASD for the clinical outcome of AN as well as the role of SS abilities in the comorbidity between the two disorders are of interest.

Objective: We investigated whether female adolescents and young adults with first onset AN exhibit more symptoms of ASD than healthy controls (HCs). Additionally, we examined whether more ASD symptoms at baseline are associated with a poorer clinical outcome of AN one year later. Finally, we investigated whether more symptoms of ASD at baseline are associated with worse SS abilities in patients with AN.

Method: A total of 130 (AN=65, HC=65) female participants in the age range 12-22 years participated in this study. Two measurement waves were executed (at baseline and at one year follow-up). The ASD symptoms were measured with the Social Responsiveness Scale (12-17 years) and with the Social Responsiveness Scale for Adults (18-22 years). The clinical outcome of AN was measured with the Eating Disorder Examination 12.0 as well as with the standardized body mass index (BMI-SDS). The SS abilities were measured with two tasks. An independent samples t-test was executed in order to examine whether the groups differed with respect to their ASD symptoms at baseline. Additionally, a linear regression analysis was performed in order to investigate the association between ASD symptoms at baseline and the clinical outcome of AN, while controlling for socio-economic status, eating disorder symptoms and BMI-SDS at baseline. A linear regression was carried out in order to examine the association between ASD symptoms at baseline and SS abilities within the AN group.

Results: In line with our hypothesis, participants with AN exhibited significantly more symptoms of ASD than their healthy counterparts. Contrary to our hypothesis, there was no association between symptoms of ASD at baseline and the clinical outcome of AN. There was also no association between symptoms of ASD at baseline and SS abilities in the participants with AN.

Conclusion: This study contributed to the existing body of literature by providing evidence for elevated ASD symptoms in female adolescents and young adults with AN. Additionally, we found no association between ASD symptoms at baseline and the clinical outcome of AN and no association between ASD symptoms at baseline and SS abilities. Future research can focus on investigating the directionality of the relationship between AN and ASD, the possible etiology and the long-term implications for the clinical outcome of AN.

Table of Contents

Introduction

"Highly disabling, possibly chronic, mostly affecting girls and young women, and clear cross-cultural differences in incidence and prevalence rates" – this is a short representation of the statistics around the mental disorder with the highest mortality rate, namely anorexia nervosa (AN) (Simpson, 2002; Zipfel et al., 2000). AN is characterized by a low weight, disturbed body image, and a pervasive fear of weight gain (Yager & Andersen, 2005). AN, meaning loss of appetite, was included as a disorder in the DSM-I in 1952, but documented medical cases of individuals experiencing similar conditions have been around since as early as the 16th century (Keel & Klump, 2003). These findings refute the idea that AN is a modern disease that is merely the product of unattainable body ideals stemming from social media. There is, however, an undisputable link between exposure to thin ideals in the media and the risk of developing eating disorders, especially in children and adolescents (Harrison, 2000). Eating disorders in general and AN in particular are incredibly complex and their etiology and prognosis are formed by a multitude of factors, including biological, social and psychological influences (Pike et al., 2020; Yao et al., 2021). In Europe, the lifetime prevalence of AN is approximately 3.6% and the incidence is around 1.7% (Keski-Rahkonen & Mustelin, 2016). AN is a condition characterized by a heterogeneous disease course and prognosis. In Europe, the statistics point to full recovery in 46% of the cases, a partial improvement in 33.5% of the cases, 20.5% of the cases show a chronic course, and a significant risk of a lethal outcome as a consequence of the disease is present (Steinhausen, 2002). Some of the factors that are known to contribute to a poor prognostic picture include long duration of hospital care (Andrade et al., 2017), low body mass index (BMI) at the time of hospital discharge (Glasofer et al., 2020), personality characteristics like perfectionism, and psychiatric comorbidity (Herzog et al., 1996).

Anorexia Nervosa and Autism Spectrum Disorder

Comorbidity is relatively common in AN, with disorders such as anxiety disorder and depressive disorder being reported in between 50% to 65% of the cases (Herzog et al., 1996). Another disorder that has a high prevalence in AN patients is an autism spectrum disorder (ASD) (Dinkler et al., 2021). ASD is a neurodevelopmental disorder characterized by deficits in social communication, repetitive sensory-motor behaviors, inflexibility and adherence to

routines. It has a heterogenous course and ranges from mild to severe (Lord et al., 2018). Research suggested that as many as 30-37% of patients with AN show symptoms of ASD (Adamson et al., 2020; Tchanturia et al., 2020), compared to around 2.5% in the general population (Xu et al., 2018). Additionally, a study conducted on adolescent girls with ASD showed that they are at a significantly higher risk of exhibiting eating-disordered behaviors than their typically developing peers (Kalyva, 2009). Yet, another study focusing on clinical diagnoses rather than symptoms only, reported that only 4% of the adolescents with AN meet the criteria for a diagnosis of ASD (Rhind et al., 2014), while studies focusing on adults with AN show that they meet the criteria for an ASD diagnosis in more than 20% of the cases (Råstam et al., 2003). These inconsistencies in the literature suggest that more research on the topic is necessary and that age is an important factor to consider when investigating the comorbidity between AN and ASD.

Unlike AN, which typically begins in puberty, ASD is often already visible in young children (Jobs et al., 2019). It is therefore remarkable that a diagnosis of ASD is often secondary to a diagnosis of AN in girls and young women (Vagni et al., 2016). This finding is counterintuitive and points to a fundamental need for better screening for ASD in females. When looking at the bigger picture however, the late diagnosing of ASD in patients with AN can also be considered as a logical consequence of a system that time and again seems to underperform when it comes to diagnosing ASD in girls. Research shows that ASD in girls can have a different phenotype, representing itself differently than it does in boys (Thompson et al., 2003) and is therefore often underdiagnosed (Gould, 2011). For instance, Mandy et al. (2012) reported that girls with ASD exhibit less repetitive stereotyped behaviors as well as less externalizing and more emotional problems than their male peers.

Additionally, the specific implications of ASD symptoms in patients with AN are still unclear. Research suggests that some treatments that are beneficial to girls with AN who do not exhibit autistic traits, do not work so well for their counterparts who score high on autism scales (Tchanturia et al., 2016). Some studies that investigated the recovery outcomes of AN in people diagnosed with ASD, showed that the presence of an ASD diagnosis is associated with poorer recovery outcomes from AN (Nielsen et al., 2015; Wentz et al., 2009). However, it is also valuable to investigate what the influence is of the presence of ASD symptoms on the clinical outcome of AN in people who have not received the diagnosis ASD.

When taking a closer look at both disorders, AN and ASD, we can begin to uncover the similarities that present themselves on different levels of functioning – social, emotional, cognitive and neurobiological. First of all, research points to a shared genetic risk for both disorders (Wade et al., 2008a). For instance, studies showed that ASD was overrepresented in first and second degree relatives of patients with AN (Huke et al., 2013; Koch et al., 2015). Zhou et al. (2018) argued that AN and ASD share neurocognitive and temperament endophenotypes. They point to the overlap in neurocognitive deficits in theory of mind and set-shifting (SS), as well as similarity in temperament dimensions, such as perfectionism and negative affectivity (Zhou et al., 2018). Executive functions have been the topic of numerous studies on the relationship between AN and ASD, with SS abilities appearing to be impaired in both disorders (Steinglass et al., 2006; Miller et al., 2015).

Set-shifting abilities, Anorexia Nervosa and Autism Spectrum Disorder

Set-shifting (SS), also referred to as "attentional switching" and "cognitive flexibility", is an executive function related to the ability to shift between different tasks (Miyake et al. 2000). Inefficiencies in SS abilities may present as cognitive inflexibility and rigid thinking, which are typical for individuals with AN (Merwin et al., 2011; Danner et al., 2012).

Previous studies investigating SS abilities in AN showed mixed results. A study by Steinglass et al. (2006) reported that adults diagnosed with AN show deficiencies in their SS abilities, as measured by the Wisconsin Card Sorting Test (WCST). However, this study focused on participants that have been diagnosed with AN for more than 5 years. Additionally, this study only included 15 subjects with AN. It would be valuable to investigate the SS abilities of individuals with AN in a bigger sample. Despite the fact that some studies show impaired SS abilities in AN (Steinglass et al., 2006; Merwin et al., 2011; Tchanturia et al., 2004), inconsistencies in the literature remain. For instance, a study on adolescents with AN using the WCST found no difference in the SS abilities of the group with AN compared to the healthy control group (Fitzpatrick et al., 2012). Additionally, another study suggested a difference in SS abilities of patients with AN when assessed with a subjective (self-report) and an objective (task) measure (Lounes et al., 2011). A common limitation of the aforementioned studies investigating SS abilities in individuals with AN is that they are mostly focused on an adult population. A recent study by Steegers et al. (2021) showed that inefficiencies in SS abilities present as early as the age of 4 were associated with

restrictive eating at the age of 9. The results of this study in combination with the fact that the age of onset of AN is usually in puberty, show that it is important to investigate the SS abilities of adolescents with AN specifically.

When it comes to SS and ASD, studies also have not reached a consensus. The observed behavior of individuals with ASD points to cognitive inflexibility and rigid behavior as expressed by difficulties in changing strategy during everyday activities and deficiencies in adapting their perspective in social situations (Geurts et al., 2009), and some studies show that these difficulties are also reflected in the participants' scores on the WCST (South et al., 2007; Yasuda et al., 2014). Others, however, did not yield the same results and claim that the SS abilities of individuals with ASD are relatively unimpaired (Barnard et al., 2008; Landa & Goldberg, 2005). There are various ways to measure SS abilities, including self-report measures and various tasks such as the WCST, the Trail Making Test (TMT), and the Brixton task (Roberts et al., 2007). Some criticism about the research on SS in ASD concerns the use of the WCST as a measurement instrument (Geurts et al., 2009). Researchers argue that poor performance on the WCST is not necessarily related to cognitive inflexibility but could be the result of deficits in a variety of cognitive processes, including learning from feedback, and working memory (Geurts et al., 2009). According to Geurts et al. (2009), the traditional task scoring system of the WCST does not allow us to distinguish between deficiencies in the different cognitive processes measured by the task. It is therefore not possible to point the specific reason why individuals with ASD perform poorly on this task (Geurts et al., 2009). Considering the fact that studies using the WCST are the only studies consistently showing SS deficits in individuals with ASD, it is interesting to conduct more research using different tasks.

When it comes to investigating SS in people with comorbid AN and ASD, a study by Westwood et al. (2017a) showed that participants with AN who exhibit a greater amount of ASD symptoms score poorer on measures of SS than individuals with AN who do not exhibit ASD symptoms. These findings suggest that underlying impairments in SS abilities could be associated with the relationship between AN and ASD. However, Westwood et al. (2017a) assessed SS abilities with the WCST and it would therefore be valuable to examine the relationship between SS abilities and the two disorders using different tasks. Overall, the literature on SS in patients with both AN and ASD is very limited and more research is necessary to draw meaningful conclusions.

The underlying mechanisms of the relationship between the two disorders are vital in order to understand the comorbidity. This could possibly help mental health professionals in designing tailored prevention programmes. It is also important to understand what the possible implications are for the clinical outcome of AN in individuals with a high amount of ASD symptoms. It is possible that a one-type-fits-all treatment plan is not an adequate option for essentially different patient populations (Westwood & Tchanturia, 2017). Therefore, more research is necessary in order to be able to draw meaningful conclusions and to improve the quality of care for females with comorbid AN and ASD. In order to achieve this, it is important to understand whether individuals with AN and an elevated ASD score show have a poorer clinical outcome after treatment for AN. Despite the growing amount of research on the topic, there are still controversial results when it comes to the comorbidity of ASD and AN. Questions such as how often does it occur, what could explain the relationship, and what could be the potential implications for the clinical outcome of AN remain unanswered. It is possible that some of the inconsistencies in research on comorbidity between AN and ASD could be the result of the use of different diagnostic instruments across studies (Westwood et al., 2017b). Additionally, some studies measure the amount of symptoms of ASD while others only include subjects who score above the cut-off score for receiving a diagnosis of ASD. Moreover, the neurodevelopmental course of ASD and the fact that AN usually has an onset in puberty (Westwood et al., 2016), lead to the conclusion that age is an important factor when considering comorbidity. Some studies even suggest that AN could be the female variant of ASD (Odent, 2010), while others claim that research on young people suggests lower comorbidity rates than previously thought and calls for more longitudinal research as well as female-specific diagnostic tools (Westwood et al., 2017b).

Uncovering the mechanisms through which AN and ASD are related but also how they differ is important and directly related to our ability to design and apply adequate treatment programs. Vital questions that we need to answer concern the extent of the problem, the possible etiology and its meaning for the disease course. Additionally, examining the association between SS and ASD in patients with AN could contribute to the creation of better targeted prevention and treatment programmes. This research will help to bring clarity regarding the comorbidity between AN and ASD in a female adolescent patient population. Additionally, it will map out the possible consequences of a higher amount of symptoms of ASD for the clinical outcome of AN, and it will investigate whether an underlying deficiency in SS abilities is associated with more symptoms of ASD in the participants with AN.

The current study

This study will focus on gathering a better insight into the relationship between AN and ASD. As a part of the ongoing longitudinal BRAVE research (Brain functions and attentional processes in adolescent anorexia nervosa: predictors of its differential course?) in the Erasmus MC Sophia Children's Hospital, this study will investigate a group of patients with AN who have received their diagnosis no more than one year prior to inclusion in the study. The aim of this research is to investigate the amount of autistic symptoms that present themselves in patients diagnosed with first-onset AN, compared to a healthy control (HC) group. We hypothesize that the group of AN patients will experience more ASD symptoms than the HC group (Adamson et al., 2020; Tchanturia et al., 2020).

Additionally, we will investigate whether the amount of ASD symptoms is associated with the clinical outcome of AN. Two measures of clinical outcome will be included – a physiological in the form of standardized body mass index (BMI-SDS) and a psychological – the Eating Disorder Examination score (EDE score). Our hypothesis is that higher scores on ASD scales at baseline will be associated with a poorer clinical outcome of AN, defined as a lower BMI-SDS and higher scores on the EDE after one year (Nielsen et al., 2015; Wentz et al., 2009). When executing the analyses we are going to correct for BMI-SDS and EDE score at baseline by including them as covariates.

Finally, we will investigate whether ASD symptoms are associated with SS abilities in the participants with AN. The hypothesis is that more ASD symptoms in the AN group are associated with a higher error rate (ET) and a slower reaction time (RT) on the SS task (Westwood et al., 2017a).

Method

Study design

The BRAVE research is an extensive longitudinal study based in the Erasmus Sophia Children's hospital, investigating girls and young women with AN as well as healthy controls (HC). The goal of the research is to investigate the brain functions and attention processes in adolescent females with first-onset AN compared to age, gender, socioeconomic status (SES) and education matched healthy controls. There are two measurement waves: one at baseline (T1) and one after one-year follow-up (T2). The first measurements for the participants with

AN took place no more than a year after receiving the diagnosis and the second measurement is conducted one year later. Healthy controls follow a similar procedure. The BRAVE study has been approved by the Medical Ethical Committee of the Erasmus MC-Sophia on 09/12/2016 (1530611) and the study was conducted in agreement with the Declaration of Helsinki (World Medical Association, 2013).

Participants

The total sample consisted of 130 female participants between the ages of 12 and 22 years. Of those participants, 102 were aged 12 - 18 years (AN= 56, HC = 46) and 28 were aged 18 - 22 years (AN = 11, HC = 17). Participants from the AN group had received a DSM-V diagnosis of AN no more than a year prior to inclusion in the study. The participants from the HC group had a normal weight (a body mass index –standard deviation score (BMI-SDS) between +1.3 and -1.3). Both groups had to meet the following inclusion criteria: female gender, age between 12 and 22 years old, sufficient knowledge of the Dutch language, the absence of a psychotic disorder, the absence of a learning disability, Intelligence Quotient (IQ) above 70, and the absence of a severe motor disability. At the end of the baseline measurement, all participants received a small gift of choice (a lip gloss, a notepad or a mini toilet bag). At the end of the second measurement all participants received a 20 euro gift voucher for an online shop.

Materials

Social Responsiveness Scale (SRS-2)

Symptoms of ASD for the participants in the age range 12-17 years are assessed with the Social Responsiveness Scale-2 (SRS-2) (Constantino & Gruber, 2012) filled out by one of the parents – either the mother or the father of the participant. The SRS-2 has good to excellent psychometric properties (Bölte et al., 2008). The internal consistency is α=.91, interrater reliability ranges between α=.76 and α=.95 and the convergent validity with other diagnostic tools is good (Bolte et al. 2008). It is a 65-item questionnaire, comprised of five scales – Social Awareness, Social Cognition, Social Communications, Social Motivations, and Autistic preoccupations. The SRS-2 is scored on a 4-point Likert scale (0-*Not True, 3-Almost always true*). There are a total of 49 regular and 16 reversed items in the questionnaire. An example of a regular item from questionnaire is *"Avoids social interactions with others*", an example of a reversed item is "*Seems confident when interacting with others"*. The raw total

scores range from 0 to 195, with higher scores indicating the presence of more symptoms of ASD. The total T-scores as well as the T-scores of the subscales (corrected for the age of the participants) will be used as continuous variables.

Social Responsiveness Scale for Adults (SRS-A)

The symptoms of ASD for the participants in the age range 18-22 years will be assessed using the Social Responsiveness Scale for Adults (SRS-A) which is a self-report questionnaire with good psychometric properties (Constantino & Todd, 2005). The SRS-A consists of 64 items that are measured on a 4-point Likert scale (*0-Not true, 3-Almost always true*). The SRS-A is comprised by four scales – Social awareness, Social communication, Social motivation, and Rigidity and Restricted interests and repetitive behaviors. The SRS-A consists of 48 regular items and 16 reversed items. An example item from the questionnaire is *"Is emotionally distant, does not show her/his feelings"*. An example of a reversed item is *"Is able to communicate his/her feelings to others"*. The raw total scores range from 0 to 192, with higher scores indicating the presence of more symptoms of ASD. In this research the total T-scores as well as the T-scores of the subscales are used.

Eating Disorder Examination 12.0 (EDE)

Clinical outcome at follow-up (T2) will be assessed using the score on the Eating Disorder Examination 12.0 (EDE). The EDE is a 62-item semi-structured interview with a high inter-rater reliability, aimed at identifying psychopathology related to eating disorders, including AN (Cooper & Fairburn, 1987). The internal reliability of the EDE subscales ranges between α =.63 for the eating concern and α = .88 for the shape concern (Wade et al., 2008b). The EDE has a good construct and criterion validity (Berg et al., 2012).

The interview examines whether the subject has engaged in behaviors indicative of an eating disorder over a 4 week period, it is scored on a 7-point scale, measuring both frequency (*0-"The characteristic is absent", 6-"The characteristic is present every day*") and severity(*0-"The characteristic is absent", 6-"The characteristic is present to an extreme extent"*). An example item from the questionnaire is *"Over the past 28 days, have you gone long periods of time (8 waking hours or more) without eating anything at all in order to influence your shape or weight?"*. The EDE is comprised by four subscales – Restraint, Eating concern, Shape concern, and Weight concern.

Set-Shifting Tasks

The set-shifting abilities of the participants were assessed by two versions of the same set-shifting task. The tasks were developed for this study and based on the category-switch task as described by Wolff and colleagues (Wolff et al., 2016), and the plus-minus task by Miyake and colleagues (Miyake et al., 2000). The two tasks include neutral stimuli (Figure 1) and sports and leisure activities (Figure 2). In both tasks, a cue was shown that indicated what task had to be executed. Afterwards a stimulus was presented and the task, indicated by the cue, had to be executed as quickly as possible using a two button response box (Curdes design). When the cue changed, it demanded from the participant to switch tasks and therefore to shift strategies.

In order to test the hypothesis, error rates (ER) and reaction times (RT) of the set-shifting tasks were combined to form inverse efficiency scores (IES) as RT/(1-ER), as described (Wolff et al., 2016). A higher IES score indicated a poorer performance on the task.

Figure 1

SS neutral task

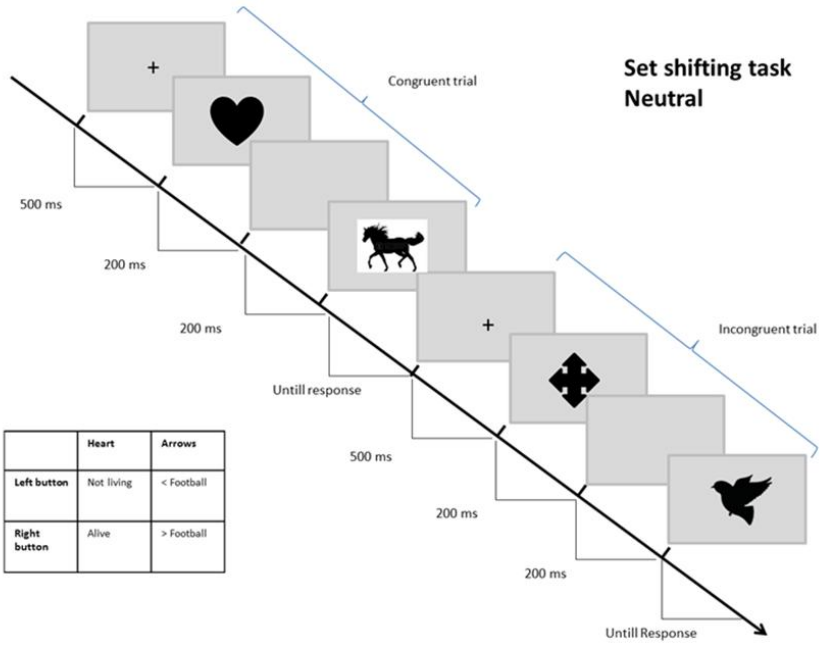
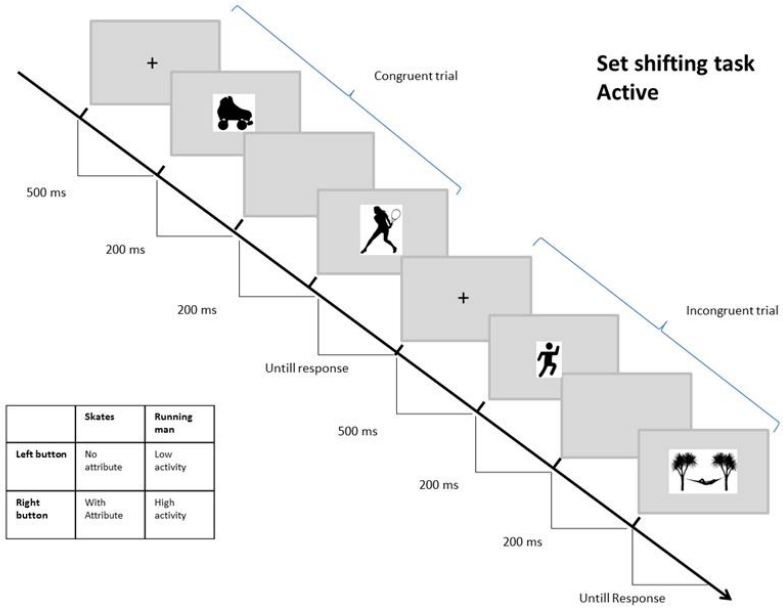


Figure 2

SS active task



Procedure

In- and outpatients with AN who met the eligibility criteria were recruited from the child- and adolescent psychiatry department of the Erasmus MC-Sophia. In addition, we collaborate with 12 other mental health institutions in the area of Rotterdam that could introduce the study to potential participants. All participants can also find information about the study online on the website of the Erasmus MC and on the social media accounts of the BRAVE research. Finally, participants can also be informed about the study via-via and contact the researchers via the contact form on the website or via the reply-form in our folder. The first step when selecting participants is the screening procedure. On the basis of eligibility criteria it is decided whether the participant can take part in the research.

Before the screening procedure the participants are asked to sign a consent form for contact. Upon reception of this consent form, the researchers contact the potential participants for the screening procedure. If the participant meets the inclusion criteria, they receive packages containing materials with information about the study. In case a participant is under

16 years old the parents also need to provide consent. After 14 days, the participants are contacted again via the telephone. During this conversation they decide whether they want to participate in the study and are provided with the opportunity to ask any questions regarding participation. If a participant decides that they want to take part in the study, they are asked to send their original signed consent forms via the post. Afterwards a consent meeting is scheduled during which the short screening procedure is repeated again and the EDE is administered among other interviews and questionnaires assessing psychopathology. During this meeting two appointments are made in random order. During one of the appointments neuropsychological assessments are made. The neuropsychological assessment includes the completion of the SS neutral and active tasks. Additionally, the participants undergo a variety of neuropsychological tests, such as an IQ test and other tasks assessing cognitive skills like memory and attention. During the other meeting an MRI scan, eye-tracking and physical health assessments take place. Additionally, some physical measures are taken, including height and weight in order to calculate the BMI-SDS.

The parents of the participants are invited to fill out several online questionnaires including the SRS-2. Participants who are 18 years old or older, receive the SRS-A via email and fill it in online.

The above described procedure is repeated one year after the first measurement. During that year, the participants with AN will be treated with care as usual for their AN, yet the type and duration of treatment they are receiving differs slightly per institute and by participant characteristics. Healthy controls will not receive any form of intervention between T1 and T2. Information about the type of treatment that participants receive is collected by means of a questionnaire which is administered by phone at T2. For this thesis, the measurements of interest are the score on the EDE, BMI-SDS, the score on the SRS/SRS-A, as well as the performance on the SS tasks.

Data Analyses

Firstly, the descriptive statistics of age, BMI-SDS, eating disorder symptoms (EDE), ethnicity and SES (educational level of the mother) were reviewed in order to provide and overall picture of the sample characteristics. In order to examine whether the two groups differed on age, eating disorder symptoms and BMI-SDS, multiple independent samples t-tests were executed. A Chi-squared test was executed in order to check whether the groups differed on the categorical variables ethnicity and SES (educational level of the mother).

The first research question concerns the difference in ASD symptoms between patients with AN and HCs. The participants were divided in two groups depending on their age range. For the participants in the age range 12-17 years the total T-score of the SRS-2 was used as a continuous dependent variable. For the participants in the age range 18-22 years the total T-score of the SRS-A was used as a continous dependent variable. In order to answer this question two independent samples t-tests were executed and the following variables were included in the analysis: independent variable Group (AN/ HC) and dependent variable SRS-2 total T-score at T1 (continuous) or SRS-A total T-score at T1 (continuous). By using the t-scores of the SRS-2/SRS-A, we corrected for the participants' age and gender. The differences on subscale level between the groups were investigated with a MANOVA for the age range 12-17 years and with a Man Whitney U test for the age range 18-22 years. It was decided to execute two different tests because the assumptions for MANOVA were not met in the sample of participants aged 18-22 years.

The second research question concerns the implications of ASD symptoms for the clinical outcome of AN. This questions included only the AN group and was tested using a linear regression analysis to measure the association between ASD symptoms at T1 and eating disorder symptoms/BMI-SDS at T2. Studies show that SES can be related to the severity and clinical outcomes of AN in children and adolescents (Li et al., 2021). Therefore, when conducting the analyses investigating the clinical outcomes of AN, SES, as measured by the educational level of the mother, was used as a covariate. Additionally, we adjusted the model for the baseline scores of the EDE and the BMI-SDS by adding them as covariates. Since the homoscedasticity and normality assumptions were not met for the participants in the age range 18-22 years, it was decided to perform a weighted least squares linear regression which is robust to these violations.

Lastly, linear regression analyses were executed in order to check whether there was an association between the ASD symptoms at baseline and the SS abilities of the participants with AN. This analysis only included participants from the AN group at T1. No covariates were included in this analysis.

The assumptions for all statistical tests were checked prior to executing the analyses. Data analyses were performed using SPSS Statistics (version 26).

Results

The groups differed significantly on the variables BMI-SDS *(t* (113) = 8.07, *p* = .000) and EDE *(t* (78.95) = -17.74, *p* = .000). The results showed that, on average, the participants in the AN group had a lower BMI at baseline (*M*= -1.19 ,*SD*=1.23) than the participants in the HC group (*M*= 0.46*, SD*= 0.95). Additionally, the participants in the AN group had higher EDE scores at baseline (*M*= 3.45*, SD*=1.26) than the participants in the HC group (*M*= 0.32*,SD*= 0.53).

The groups did not differ on the baseline characteristics age *(t* (128) = 1.70, *p* = .092), ethnicity (X^2 (2) = 4.53, p= .104) and educational level of the mother (X^2 (2) = 2.51 , p= .286). The descriptive statistics at baseline from the AN group and the HC group are presented in Table 1.

Table 1

Descriptive statistics of baseline characteristics of participants with AN and HCs

Variable	AN		HC		*p*
	N	*M (SD)*	*N*	*M (SD)*	
Age (years)	65	15.66 (2.16)	65	16.29 (2.07)	.092
EDE total T-score	60	3.45 (1.26)	60	0.32 (.53)	.000*
BMI-SDS	55	-1.19 (1.23)	60	0.46 (0.95)	.000*
SS active task IES score					
- Age range 12-17 years	44	1446.36 (609.51)	-	-	-
- Age range 18-22 years	10	1234.42 (374.73)	-	-	-
SS neutral task IES score					
- Age range 12-17 years	44	1217.81 (623.73)	-	-	-
- Age range 18-22 years	10	982.75 (376.71)	-	-	-
Ethnicity	65		65		.104
- Dutch	61	93.9%	63	96.9%	
- Western	3	4.6%	0	0%	
- Non-Western	1	1.5%	2	3.1%	
Educational level of the mother	61		60		.286
- Low	8	12.3%	3	4.6%	
- Medium high	20	30.8%	21	32.3%	
-High	33	55.4%	36	55.4%	
Illness duration (months)	65	4.91 (3.77)	-	-	

Note. AN = Anorexia Nervosa. HC = Healthy Control. N=sample size. *M* = Mean. *SD* = Standard Deviation. EDE = Eating Disorder Examination. BMI-SDS = standardized body mass index. IES= inverse efficiency score. SS active task = set-shifting active task. SS neutral task = set-shifting neutral task. * = $p<0.05$

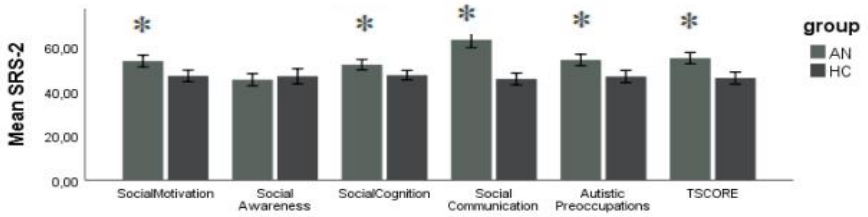
Comparing symptoms of ASD at baseline between AN and HC

The first research question concerned the difference in ASD symptoms between the AN group and the HC group at baseline. For the participants in the age range 12-17 years, this question was answered by conducting an independent samples t-test with Group (AN/HC) as a categorical independent variable and the score on the SRS-2 (N(AN) = 54, N(HC)=48) as a continuous dependent variable. The assumptions were checked prior to executing the analysis. The normality assumption was met for the AN group but not for the HC group. However, according to the central limit theorem (CLT) if the sample size is larger than 20 per group, the data is robust to this violation. The Levene's test showed that the assumption for equality of variances was not violated (p=.391), therefore we assumed that the variances were equal when interpreting the results of the t-test. We found a significant difference at baseline between the group with AN and the control group on the level of symptoms of ASD *(t* (100) = -4.87, p = .000). On average, the participants in the AN group had higher ASD scores on the SRS-2 (M=55.30, SD=9.27) than the participants in the HC group (M=46.27, SD=9.41). The descriptive statistics and corresponding p-values are illustrated in Table 2.

Additionally, a MANOVA was executed to check for the differences between the groups on subscale level. The normality assumption was violated for the control group for the subscales social cognition, social communication, and autistic preoccupations and for the AN group for the subscales social awareness, social communication, autistic preoccupations. However, according to the CLT the data is robust to this violation. The Levene's test for equality of variances showed that the assumption for homogeneity of variances is not met for the social communication subscale (p<.001). However, because the sample sizes are approximately equal, the MANOVA is robust to this violation. Wilk's Lambda showed a significant difference at baseline between the AN and the HC group (λ=.549, F(5.96) = 15.80, p=.000, η2= .45). The test of between-subject effect showed that the AN group had more ASD symptoms at baseline compared to the HC group for the variables social motivation (F (1,101) = 12.95 p=.001, η2=.12), social cognition (F(1,101) = 8.92, p= .004, η2=.08), social communication (F (1,101) = 61.76, p=.000, η2=.38), and autistic preoccupations (F (1,101) = 15.89, p=.000, η2=.14), but not for the variable social awareness (F (1,101) = 0.51, p=.510) (see Figure 3).

Figure 3

Mean SRS-2 scores on subscale level of participants with AN and for HCs aged 12-17 years



*Note: AN: anorexia nervosa; HC: healthy controls. * = p<0.05*

Table 2

Autistic trait scores of participants with AN and for HCs aged 12-17 years

Variable	AN N=54		HC N=48		*p*
	M	*SD*	*M*	*SD*	
SRS-2 Total T-score	55.30	9.27	46.37	9.41	.000*
Social Awareness T-score	45.54	10.10	47.08	11.77	.510
Social Cognition T-score	52.33	8.57	47.58	7.35	.004*
Social Communication T-score	63.61	13.00	45.83	9.28	.000*
Social Motivation T-score	53.98	9.82	47.25	8.97	.001*
Autistic Preoccupations T-score	54.48	9.40	47.00	9.53	.000*

Note. AN = Anorexia Nervosa. HC = Healthy Control. N=sample size. *M* = Mean. *SD* = Standard Deviation. *=p<0.05

For the participants in the age range 18-22 an independent samples t-test was performed with Group (AN/HC) as a categorical independent variable and the score on the SRS-A (N(AN) = 11, N(HC)=17) as a continuous dependent variable. The assumptions were checked prior to executing the analysis. The normality assumption was met for both groups. The Levene's test showed that the assumption for equality of variances was violated (p=.014).

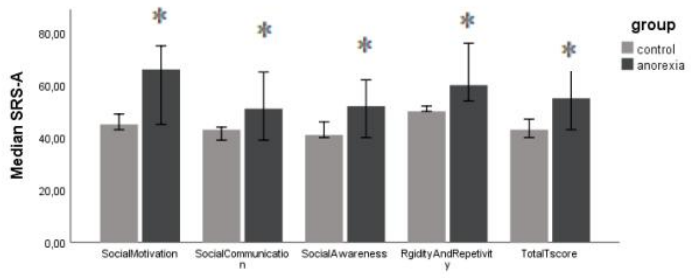
Therefore, the row with equal variances not assumed was used when interpreting the t-test. The results showed that there was a significant difference between the AN and the HC group at baseline on symptoms of ASD (t (12,51) = -3.67, p = .003). On average, the participants in the AN group had higher ASD scores (M=56.45, SD=10.93) than the participants in the HC group (M=43.65, SD=4.78).

A non-parametric Mann-Whitney U test was executed in order to investigate the group differences on subscale level. It was chosen to do a Mann-Whitney U test instead of a MANOVA because the assumption for equality of variances for the subscales social communication, social motivation and rigidity and repetivity was violated. Since the sample sizes of the groups are not approximately equal (HC=17, AN=11), the MANOVA is not robust to this violation. The Shapiro Wilk's test showed that the normality assumption for the HC group for the scales social awareness and rigidity and repetivity was also violated. However, the Mann-Whitney U test is robust to this violation.

Across all scales the AN participants exhibited a higher amount of ASD symptoms than the participants in the HC group (see Figure 4). The AN group (N=11) had more ASD symptoms at baseline compared to the HC group (N=17) for the variables social communication, social motivation, rigidity and repetivity and social awareness. The Mann Whitney U test results are illustrated in Table 3.

Figure 4

Median SRS-A on subscale level of participants with AN and for HCs aged 18-22 years



*Note: AN: anorexia nervosa; HC: healthy controls. * = p<.05*

Table 3

Results of Mann Whintey U test on the significance of the differences of SRS-A subscale t-scores in participants aged 18-22 years

Variable	U	*z*	*r*	*p*
Social Motivation T-score	26.00	-3.19	.60	.001*
Social Communication T-score	44.50	-2.32	.44	.021*
Social Awareness T-score	45.00	-2.29	.43	.022*
Rigidity and Repetivity T-score	12.50	-3.95	.73	.000*

Note. AN = Anorexia Nervosa. HC = Healthy Control. N=sample size. *M* = Mean. *SD* = Standard Deviation.
*= $p < .05$

Symptoms of ASD at baseline and the clinical outcome of AN

The second research question investigated the association between ASD symptoms at baseline and the clinical outcome of AN one year later. This question concerned only the AN group and was tested using a linear regression analysis with the SRS-2 scores at T1 as a continuous independent variable and either the EDE total score or the BMI-SDS at T2 as continuous dependent variables. Additionally, we corrected for EDE and BMI-SDS scores at T1, as well as for SES by including them as covariates in the linear regression. All assumptions were checked prior to executing the analysis. The sensitivity assumption check showed no outliers as examined by Cook's, Mahalanobis and the Leverage values. Additionally, examination of the scatterplots showed that the linearity, homoscedasticity and normality assumptions were also met.

The linear regression analysis (Table 4) showed that there was no significant association between ASD symptoms at baseline and symptoms of AN at T2 ($F(1,28) = 0.94$, p=.341). After adjusting the model for the covariates EDE score at T1 and SES, there was a significant effect of the model (F(3.28)=3.38, p= .034) however, this was due to the fact that that the covariate EDE T1 had a significant effect *(t* (28) = 2.76, p = .011). The association between ASD symptoms and symptoms of AN at T2 remained non-significant *(t* (28) = -0.35, p = .730).

The analysis showed no significant association between ASD symptoms and the BMI-SDS at T2 ($F(1,20) = 1.848$, $p=.190$). After adjusting the model for the covariates BMI-SDS at T1 and SES, there was still no significant effect ($F(3,20)=2.15$, $p=.131$). None of the covariates was significantly associated with the BMI-SDS at T2. The linear regression results are illustrated in Table 4 and Table 5.

Table 4

Multiple linear regression results for autistic traits at baseline and EDE T2 12-17 years

	B for EDE at T2	S.E.	β	*t*	*p*	R^2	Adjusted R^2
Unadjusted model						0.03	0.00
Constant	3.62	1.45		2.49	.019		
SRS-2 T1	-0.03	0.03	-0.18	-0.97	.341		
Adjusted model						0.29	0.20
Constant	-0.06	1.88		-0.03	.977		
SRS-2 T1	-0.01	0.02	-0.06	-.35	.730		
SES	0.34	0.31	0.19	1.09	.288		
EDE T1	0.51	0.19	0.48	2.76	.011		

Note. EDE T2= eating disorder examination at T2; B=unstandardized regression coefficient; S.E.=standard error of the coefficient; β=standardized regression coefficient; CI=confidence interval; LL=lower limit; UL=upper limit; $R^{2=}$standardized effect size. SRS T1= mean t-score of the SRS at baseline; Adjusted model= adjusted for the covariates EDE T1, SES=socioeconomic status.

Table 5

Multiple linear regression results for autistic traits at baseline and BMI-SDS T2 12-17 years

BMI-SDS T2	B	S.E.	β	*t*	*p*	R^2	Adjusted R^2
Unadjusted model						0.09	0.04
Constant	0.76	1.15		0.66	.518		
SRS-2 T1	-.003	0.02	-0.30	-1.36	.190		
Adjusted model						0.26	0.15
Constant	0.28	1.60		0.18	.863		
SRS-2 T1	-0.01	0.02	-0.08	-0.35	.734		
SES	-.004	0.33	-0.02	-0.11	.916		
BMI-SDS T1	0.41	0.21	0.48	1.98	.064		

Note. BMI-SDS=standardized body-mass index; B=unstandardized regression coefficient; S.E.=standard error of the coefficient; β=standardized regression coefficient; CI=confidence interval;

LL=lower limit; UL=upper limit;$R^{2=}$standardized effect size; SRS-2 T1= mean t-score of the SRS-2 at baseline; Adjusted model= adjusted for the covariates BMI-SDS T1, SES=socioeconomic status.

Table 6

Means and standard deviations for anorexia nervosa participants for BMI-SDS, eating disorder symptoms (EDE total score and subscales Restraint, Food concerns, Weight concerns and Body shape concerns) at T2 12-17

Variable	AN T2		
	N	*M*	*SD*
BMI-SDS	25	-.67	1.19
EDE total score	30	2.30	1.31
Restraint	30	1.85	1.63
Food concerns	30	1.51	1.00
Weight concerns	30	2.85	1.65
Body shape concerns	30	3.01	1.70

Note. AN = Anorexia Nervosa. BMI-SDS:Body mass index standardized. EDE:Eating Disorder Examination. N=sample size. *M* = Mean. *SD* = Standard Deviation.

For the participants in the age range 18-22 a weighted least squares regression was performed. The results showed a non-significant relationship between symptoms of ASD and eating disorder symptoms (Table 7) $F(1,7) = 0.00$, $p = .998$). After adjusting the model for the covariates EDE at T1 and SES, the model was significant $F(3,7) = 9.46$, $p = .027$). However, the covariate SES had a significant association with symptoms of AN at T2 *(t* (7) = -4.58, $p = .010$). There was still no significant association between symptoms of ASD and symptoms of AN at T2 t (7) = 0.65, $p = .553$).

The results also showed a non-significant association between symptoms of ASD and BMI-SDS (Table 8) ($F(1,4) = 1.39$, $p = .324$). After adjusting the model for the covariates BMI-SDS at T1 and SES the model was still not significant ($F(3,4) = 3.04$, $p = .393$). None of the covariates had a significant effect.

Table 7

Multiple linear regression results for autistic traits at baseline and EDE T2 18-22 years

EDE T2	B	S.E.	β	*t*	*p*	R^2	Adjusted R^2
Unadjusted model						.000	-.167
Constant	3.653	2.743		1.332	.231		
SRS-A T1	.000	.053	-.001	-.003	.998		
Adjusted model						.876	.784
Constant	3.774	1.873		2.015	.114		
SRS-A T1	.015	.024	.118	.647	.553		
SES	-.958	.209	-.976	-4.578	.010		
EDE T1	.182	.246	.146	.742	.499		

Note. BMI-SDS=standardized body-mass index; B=unstandardized regression coefficient; S.E.=standard error of the coefficient; β=standardized regression coefficient; CI=confidence interval; LL=lower limit; UL=upper limit; $R^{2=}$standardized effect size; SRS-A T1= mean t-score of the SRS-A at baseline; Adjusted model= adjusted for the covariates BMI-SDS T1, SES=socioeconomic status.

Table 8

Multiple linear regression results for autistic traits at baseline and BMI-SDS T2 18-22 years

BMI-SDS T2	B	S.E.	β	*t*	*p*	R^2	Adjusted R^2
Unadjusted model						0.32	.09
Constant	-14.10	13.28		-1.06	.366		
SRS-A T1	0.24	0.20	0.56	1.18	.324		
Adjusted model						0.90	.61
Constant	2.16	11.38		0.19	.881		
SRS-A T1	0.06	0.34	0.15	0.19	.882		
SES	-1.61	6.03	-0.42	-0.27	.834		
BMI-SDS T1	2.09	1.85	1.24	1.13	.46		

Note. BMI-SDS=standardized body-mass index; B=unstandardized regression coefficient; S.E.=standard error of the coefficient; β=standardized regression coefficient; CI=confidence interval; LL=lower limit; UL=upper limit; $R^{2=}$standardized effect size; SRS-A T1= mean t-score of the SRS-A at baseline; Adjusted model= adjusted for the covariates BMI-SDS T1, SES=socioeconomic status.

Table 9

Means and standard deviations for participants with anorexia nervosa for BMI-SDS, eating disorder symptoms (EDE total score and subscales) at T2 18-22 years

Variable	AN		
	T2		
	N	*M*	*SD*
BMI-SDS	5	-0.74	2.37
EDE total score	8	3.33	0.85
Restraint	8	2.73	1.68
Food concerns	8	1.80	1.35
Weight concerns	8	3.56	1.24
Body shape concerns	8	3.81	1.10

Note. AN = Anorexia Nervosa. BMI-SDS:Body mass index standardized. EDE:Eating Disorder Examination. N=sample size. *M* = Mean. *SD* = Standard Deviation.

Symptoms of ASD at baseline and SS abilities in AN

A linear regression analysis was executed to examine whether there is a significant association between SS abilities, as measured by an active and a neutral SS task, and symptoms of ASD, as measured by the SRS-2 at baseline. The outliers assumption check (as measured by Mahalanobis and the Levarage values) showed that there was one outlier that significantly influenced the results and it was therefore decided to remove it from the analysis. The normality assumption was checked by examining a Q-Q plot which showed that the residuals were normally distributed. The linearity and homoscedasticity assumptions were met.

The analysis (Table 10) showed that, among participants with AN, there was no significant association between symptoms of ASD and SS abilities, as measured by the neutral task, and($F(1,9) = 0.45$, $p=.523$). There was also no significant association between ASD symptoms at baseline and the active SS tasks ($F(1,9) = 0.31$, $p=.593$).

Table 10

Linear regression results for set-shifting neutral, set-shifting active with autistic traits as independent variable

Variable	B for SRS-2	S.E.	β	*t*	*p*	95% CI for B		R^2
						LL	UL	
SS neutral	4.63	10.57	.07	.438	.664	-16.68	25.92	.004
SS active	8.92	10.65	.13	.837	.407	-12.57	30.40	.016

Note. SS neutral = neutral set-shifting task. SS active = active set-shifting task.B=unstandardized regression coefficient; S.E.=standard error of the coefficient; β=standardized regression coefficient; CI=confidence interval; LL=lower limit; UL=upper limit;$R^{2=}$standardized effect size;

For the participants in the age range 18-22, a linear regression analysis was performed with the SRS-A score as a continuous independent variable and the SS tasks (neutral or active) as continuous dependent variables. The outliers assumption check (as measured by Mahalanobis, Cook's and the Levarage values) showed that there were no outliers. The normality assumption was checked by examining a Q-Q plot which showed that the residuals were normally distributed. The linearity and homoscedasticity assumptions were met.

The linear regression analysis (Table 13) showed that there was no significant association between the symptoms of ASD and the netral SS task ($F(1,9) = .45$, $p=.523$). The analysis showed no significant association between symptoms of ASD and the active SS task ($F(1,9) = .31$, $p=.592$).

Table 11

Linear regression results for SS neutral, SS active with SRS-2 as independent variable

Variable	B	S.E.	β	*t*	*p*	95% CI for B		R^2
						LL	UL	
SS neutral	-9.61	14.39	-.23	-0.67	.523	-42.79	23.57	0.05
SS active	-8.05	14.43	-.19	-0.56	.592	-41.32	25.23	0.04

Note. SS neutral = neutral set-shifting task. SS active = active set-shifting task.B=unstandardized regression coefficient; S.E.=standard error of the coefficient; β=standardized regression coefficient; CI=confidence interval; LL=lower limit; UL=upper limit; $R^{2=}$standardized effect size;

Discussion

The aim of this study was to examine whether participants with AN experience more symptoms of ASD than the HC group. Additionally, we investigated whether symptoms of ASD at baseline are associated with the clinical outcome of AN one year later. Finally, this study examined whether symptoms of ASD are associated with SS abilities in the participants with AN. The results showed that, line with our hypothesis, ASD symptoms were elevated in participants with AN compared to healthy controls. Contrary to our hypothesis, there was no relationship between symptoms of ASD and the clinical outcome of AN. Additionally, there was no relationship between the symptoms of ASD and the SS abilities of patients with AN.

ASD symptoms at baseline

Patients with AN experienced more symptoms of ASD at baseline than their HC counterparts, which is in line with our hypothesis. Patients with AN showed more problems in the areas of social cognition, social motivation, social communication, autistic preoccupations, social awareness and rigidity and repetivity.

Our findings are in line with previous research that found that both adolescents and young adults with AN exhibit more symptoms of ASD than healthy controls (Adamson et al., 2020; Tchanturia et al., 2020; Dinkler et al., 2021). The results of our study point to the fact that females with AN have more difficulties in initiating and maintaining social interactions than their healthy counterparts. Additionally, it is important to note that the effect sizes across scales ranged from medium ($\eta 2$=.08) to large ($\eta 2$=.38) (Lakens, 2013). Research shows that the effect size is an essential indicator of clinical relevance (Hojat & Xu, 2004). Therefore, the effect sizes indicate that the results of this study go beyond mere statistical significance and can potentially be interpreted as clinically relevant.

Since this study did not investigate directionality, it is not possible to identify whether symptoms of ASD developed before or after the symptoms of AN. However, some research suggests that symptoms of ASD can impact the development of AN in two different ways. On the one hand, increased rigidity can impact the development of inadequate eating patterns and on the other hand, problems in social communication can contribute to the development of emotional problems and low self-esteem (Saure et al., 2021).

ASD symptoms and clinical outcome of AN

Contrary to our hypothesis, a higher amount of ASD symptoms at baseline was not associated with a poorer clinical outcome of AN after one year. This finding is not in line with previous research that has found that the presence of ASD symptoms has a negative effect on the clinical outcome of AN (Nielsen et al., 2015; Wentz et al., 2009). However, both studies had a significantly longer follow-up (10 and 18 years respectively) than our study, which could have influenced the differences in the results.

The results of our study could be explained in several ways. First, one possible reason for the lack of effect could be the small sample size. In the age range 12-17, there were 30 participants, and in the age range 18-22, there were only a total of 8 participants in the sample. It is possible that the lack of power increased the probability of a Type II error and therefore prevented us from finding an effect (Akobeng, 2016). Another explanation for our findings could be the fact that the treatment of AN is a long-term endeavour and can often last more than two years (Verheij, 2004; Wojciechowski, 2004). However, the measurement assessing clinical outcome was conducted only one year after the baseline measurement. Therefore, it is possible that for some participants this one year was not sufficient to achieve a favourable clinical outcome. Additionally, we cannot exclude the possibility that the participants who exhibited a higher amount of ASD symptoms actually received interventions that were tailored to their specific needs. Research shows that when psychological interventions are tailored to the needs of the client, the treatment can be successful in people with elevated ASD symptoms (Karim, 2021).

ASD symptoms and SS abilities

Contrary to our expectations, ASD symptoms were not associated with SS abilities. Our findings are in line with previous studies, which suggests that whether or not ASD symptoms are associated with poorer SS abilities is often dependent on the task used to examine SS abilities (Barnard et al., 2008; Landa et al., 2005). Even though a certain level of inflexibility is common in individuals with ASD, it is possible that the tasks used to measure SS abilities do not always match the patterns of inflexibility exhibited in ASD.

The WCST is often used to measure SS abilities and interestingly it is often studies that use this task that show the deficits in SS in people with ASD. However, the WCST has been criticized as a measure of SS abilities (Geurts et al., 2006). Therefore, the use of different instruments to measure SS abilities could explain the inconsistency between our research and

studies that do show an association between SS abilities and ASD (South et al., 2007; Yasuda et al., 2014).

Our results are not in line with the study of Westwood et al. (2017a) who did find an association between SS abilities and symptoms of ASD in individuals with AN. However, their study also examined SS abilities using the WCST, which could be one of the reasons for the difference in results. Additionally, Westwood et al. (2017a) looked at individuals who scored at or above the clinical cut-off score of an ASD measure, meaning that they focused on individuals with clinical or sub-clinical symptoms of ASD, while most of our participants scored below the cut-off on our ASD measure. Finally, Westwood et al. (2017a) only included participants from a single in-patient treatment institution. The fact that their patient sample required in-patient care, could suggest that patient's symptoms of AN were more severe and therefore less comparable to our sample.

Strengths and limitations

This study used multiple versions of an SS task different from the WCST, which helped to diversify the pool of tasks used to measure SS abilities in research. Another strength of our study is the diverse age range of our participants. The fact that the sample size of the participants with AN in the age range 18-22 was limited can be explained by the fact that AN often has an onset in puberty and our population of interest constituted individuals with first-onset AN that were diagnosed no more than one year prior inclusion in the study. Another strength of our study was the comprehensive instrument pool, which made it possible to look at different components of AN. Using a reliable and extensive measure like the EDE helped us examine psychological components such as body shape concerns and adding an objective clinical outcome measure in the form of BMI-SDS made our results even more comprehensive.

A limitation of our study concerns the limited power, specifically with regards to the second and third research questions. It is possible that the small sample size influenced the power of the study and increased the probability of a Type II error. Additionally, considering the fact that the results of studies investigating SS abilities in patients with AN differ depending on the task used to measure SS abilities, it would have been informative to use multiple measures of SS abilities, including a self-report questionnaire, and to compare whether there is a difference in the scores on the different measures. Another limitation of

this study that has to be considered is that data collection took place both before and during the COVID-19 pandemic. Research has found that the lockdown measures have had an impact on the mental health of adolescents, and that patients who suffer from mental disorders such as AN have been particularly vulnerable (Schlegl, 2020). Therefore, it is possible that the results of this study have been indirectly influenced by the lockdown measures. For example, it is possible that not all patients with AN had access to regular treatment sessions or that the social isolation could have contributed to high stress and the worsening of symptoms related to AN, which could have in turn had implications for the clinical outcome of AN.

Implications and future research suggestions

This is one of the first studies to examine both ASD symptoms and SS abilities in individuals with first-onset AN. The finding that individuals with AN exhibit more symptoms of ASD than healthy controls once again highlights the importance of further examination of the relationship between the two disorders. Some studies suggest that the development of ASD symptoms in individuals with AN can be the result of a prolonged disease course (Hiller et al., 2013). However, the fact that our study only included participants that were relatively recently diagnosed and still found an effect, suggests that there is a different explanation for this relationship. Even though we did not find an association between SS abilities and symptoms of ASD, mental health professionals highlight that rigidity and inflexibility in behavior are a shared component of both disorders (Robertson et al., 2021; D'Cruz et al. 2013). Therefore, it is essential to further examine the specific similarities on cognitive and behavioral level between the two disorders, identify possible mediators of their relationship, and examine the possible long-term implications of ASD symptoms for the clinical outcome of AN. Moreover, it would be valuable to research the patterns of deficiency in SS abilities in AN and ASD with a more diverse instrument pool, going beyond the WCST, and comparing self-report and objective measures. Additionally, it is important to investigate whether the symptoms of ASD actually precede the development of AN and whether individuals with elevated ASD symptoms are at a higher risk for the development of AN. Such findings would be essential for the development of AN prevention and treatment programs. Finally, including a larger sample size and controlling for the effects of the COVID-19 pandemic when investigating the relationship between ASD symptoms and the clinical outcome of AN is recommended.

Conclusion

In conclusion, our study adds to the body of literature investigating the comorbidity between AN and ASD, the implications for the clinical outcome and the role of SS abilities. We provided evidence for elevated symptoms of ASD in both female adolescents as well as young adults with AN. We did however not find an association between symptoms of ASD and the clinical outcome of AN. Additionally, we did not find a relationship between symptoms of ASD and SS abilities in participants with AN. These findings need to be interpreted with caution, since there are different explanations that can be responsible for the lack of effect. Our study investigated a topic that has not been extensively researched in a unique sample - relatively recently diagnosed individuals with first-onset AN within a broad age range. As such, this study can provide important insights to mental health professionals and suggest valuable future directions in the research of AN, ASD and SS abilities.

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