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Shared familial risk factors between autism spectrum disorder and obesity – a register-based familial coaggregation cohort study

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Background: Meta-analyses suggest an association between autism spectrum disorder (ASD) and obesity, but the factors underlying this association remain unclear. This study investigated the association between ASD and obesity stratified on intellectual disability (ID). In addition, in order to gain insight into possible shared etiological factors, the potential role of shared familial liability was examined. **Method:** We studied a cohort of 3,141,696 individuals by linking several Swedish nationwide registers. We identified 35,461 individuals with ASD and 61,784 individuals with obesity. Logistic regression models were used to estimate the association between ASD and obesity separately by ID and sex and by adjusting for parental education, psychiatric comorbidity, and psychotropic medication. Potential shared familial etiologic factors were examined by comparing the risk of obesity in full siblings, maternal and paternal half-siblings, and full- and half-cousins of individuals with ASD to the risk of obesity in relatives of individuals without ASD. **Results:** Individuals with ASD + ID (OR = 3.76 [95% CI, 3.38–4.19]) and ASD−ID (OR = 3.40 [95% CI, 3.23–3.58]) had an increased risk for obesity compared with individuals without ASD. The associations remained statistically significant when adjusting for parental education, psychiatric comorbidity, and medication. Sex-stratified analyses indicated a higher relative risk for males compared with females, with statistically significant interaction effects for ASD−ID, but not for ASD+ID in the fully adjusted model. First-degree relatives of individuals with ASD+ID and ASD−ID had an increased risk of obesity compared with first-degree relatives of individuals without ASD. The obesity risk was similar in second-degree relatives of individuals with ASD+ID but was lower for and ASD−ID. Full cousins of individuals with ASD+ID had a higher risk compared with half-cousins of individuals with ASD+ID). A similar difference in the obesity risk between full cousins and half-cousins was observed for ASD−ID. **Conclusions:** Individuals with ASD and their relatives are at increased risk for obesity. The risk might be somewhat higher for males than females. This warrants further studies examining potential common pleiotropic genetic factors and shared family-wide environmental factors for ASD and obesity. Such research might aid in identifying specific risks and underlying mechanisms in common between ASD and obesity. **Keywords:** Autism; obesity; family factors.

Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by persistent challenges in social communication and interaction alongside restrictive and inflexible behavior patterns and sensory processing alterations leading to impairment in major life areas (American Psychiatric Association, 2013). ASD co-occurs with other neurodevelopmental disorders (e.g. attention-deficit/hyperactivity-disorder (ADHD), intellectual disability (ID), and speech- and language disorders) (Ghirardi et al., 2018; Hoekstra, Happe, Baron-Cohen, & Ronald, 2009; Lundstrom et al., 2015) and internalizing psychiatric disorders (e.g., anxiety and depression) (Hollocks, Lerh, Magiati, Meiser-Stedman, & Brugha, 2018; Hudson, Hall, & Harkness, 2019; van Steensel & Heeman, 2017). ASD has also been associated with negative life outcomes such as limited social integration and poor job prospects (van Heijst & Geurts, 2015; Howlin & Magiati, 2017) as well as high risk for suicidal behaviors (Hirvikoski et al., 2020). Several studies have reported an association between ASD and weight dysregulation. ASD and autistic traits have been associated with both high and low BMI (Kerekes et al., 2015) as well as anorexia nervosa and obesity (Koch et al., 2015; Wentz, Bjork, & Dahlgren, 2017; Westwood et al., 2016) with indications of possible sex differences (Baron-Cohen et al., 2013; Bolte, Ozkara, & Poustka, 2002). A recent meta-analysis estimated the prevalence of obesity in ASD at 21.8% (Li, Xie, Lei, Li, & Lei, 2020), while the WHO have estimated the global prevalence of obesity at 13%. Large population-based

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studies have found an increased risk for obesity in individuals with ASD compared with the general population (de Vinck-Baroody et al., 2015; Dreyer Gillette et al., 2015; Must et al., 2017; Phillips et al., 2014). Meta-analytic evidence on the association between ASD and obesity generally suggests an increased risk for obesity in individuals with ASD. Nevertheless, there is substantial heterogeneity across studies (Kahathuduwa et al., 2019; Zheng et al., 2017) and detailed information about the nature of the association is lacking.

Few studies have explored if ID influences the association between ASD and obesity. This is an important limitation, as ID has demonstrated a robust association with obesity and because there are functional differences between ASD with ID (ASD+ID) and without ID (ASD−ID) (Maiano, Hue, Morin, & Moullec, 2016; Matson & Shoemaker, 2009). Individuals with ASD+ID have been found to have more challenging behaviors than individuals with ASD−ID and they are also less likely to experience gains in social, communication, and repetitive behavior over time, and may not benefit as much from interventions as individuals with ASD−ID (Ben-Itzchak & Zachor, 2007; Fountain, Winter, & Bearman, 2012; Klinger, Cook, & Dudley, 2020).

In the only available study, the association between autism and obesity failed to reach statistical significance after adjusting for ID (Corvey, Menear, Preskitt, Goldfarb, & Menachemi, 2016). The conclusions that can be drawn from this study are limited however because of the small sample size. Also, parent reports were used to ascertain information about ASD status, severity level, and presence of comorbid conditions. Additional studies with larger samples and clinically ascertained diagnoses are needed to clarify the role of ID in the association between ASD and obesity.

It is currently unclear if the strength of the association between ASD and obesity differs between males and females. One recent population-based study found an association between ASD and obesity in adolescent males, but not in adolescent females (Must et al., 2017). Another study found a stronger association between ASD and obesity in females than in males (Memari, Kordi, Ziaee, Mirfazeli, & Setoodeh, 2012). Some studies report no significant sex differences in the association between ASD and obesity (Criado et al., 2017; de Vinck-Baroody et al., 2015; Healy, Aigner, & Haegele, 2018). The small sample sizes are a limitation in the previous studies that may explain the conflicting results.

Previous studies have not sufficiently clarified the extent to which psychiatric comorbidity and psychotropic medication influences the association between ASD and obesity. Obesity is associated with high rates of comorbid depression, anxiety, and ADHD (Cortese & Tessari, 2017; de Vinck-Baroody et al., 2015; Luppino et al., 2010), and these diagnoses are also common comorbidities to ASD (van Steensel & Heeman, 2017; White, Oswald, Ollendick, & Scahill, 2009). Treatment with antidepressants and antipsychotics is quite common in individuals with ASD, and these medications have been associated with weight gain (Bak, Fransen, Janssen, van Os, & Drukker, 2014; Buck et al., 2014; Fallah et al., 2019; Serretti & Mandelli, 2010). Still, it remains to be investigated if these mental health problems and medications explain the association between ASD and obesity.

No earlier family-based study has explored whether the association between ASD and obesity is influenced by shared familial factors. Both ASD and obesity are highly heritable, with meta-analytic estimates around 64%–91% for ASD and around 47%–90% for obesity (Elks et al., 2012; Silventoinen, Rokholm, Kaprio, & Sorensen, 2010; Tick, Bolton, Happe, Rutter, & Rijsdijk, 2016). Although the high heritability estimates of ASD and obesity do not by default mean that these conditions have causes in common, there is a possibility that the association is partly due to shared familial factors (Martin, Taylor, & Lichtenstein, 2018). A recent Genome-Wide Association Study (GWAS) found a genetic correlation of .09 to .12 between ASD and different levels of obesity (Grove et al., 2019). This study did not investigate if the familial/genetic association was invariant for different types of ASD (with and without ID) or for sexual differences. This remains to be investigated.

The aim of the current study was to estimate the occurrence of obesity in a large population-based cohort of individuals with and without ASD. We explored the risk of obesity, separately for ASD+ID and ASD−ID, as well as for males and females. Further, we analyzed the role of comorbidity with depression, anxiety, substance use disorder (SUD), neurodevelopmental disorders, eating disorders, and medication in the association between ASD and obesity. Finally, the use of family data from the Swedish national multigeneration register-enabled analysis of shared familial risk factors for ASD and obesity.

Methods

Study setting and study population

We used the Medical Birth Register (MBR) to identify all individuals born in Sweden between 1973 and 2006 (n = 3,422,261). By linking information from the MBR with data from the Swedish Total Population Register (STPR), Cause of Death Register and the Multi-Generation Register (MGR), we generated a cohort of 3,141,696 unique eligible individuals after exclusions, see Figure 1. The MGR links those individuals who lived in Sweden from January 1, 1961, and onward to their biological parents and was used to enable identification of full siblings, half-siblings, and cousins. The STPR was used to obtain information on sex and birth year. Individuals with an ASD diagnosis between 1987 and 2006 (n = 35,461) were identified from the Swedish National Patient Register (SNPR). Patients in SNPR can be born in Sweden and in other countries. The individuals with ASD were born between 1973 and 2006. The SNPR includes primary and secondary diagnoses registered as International Classification

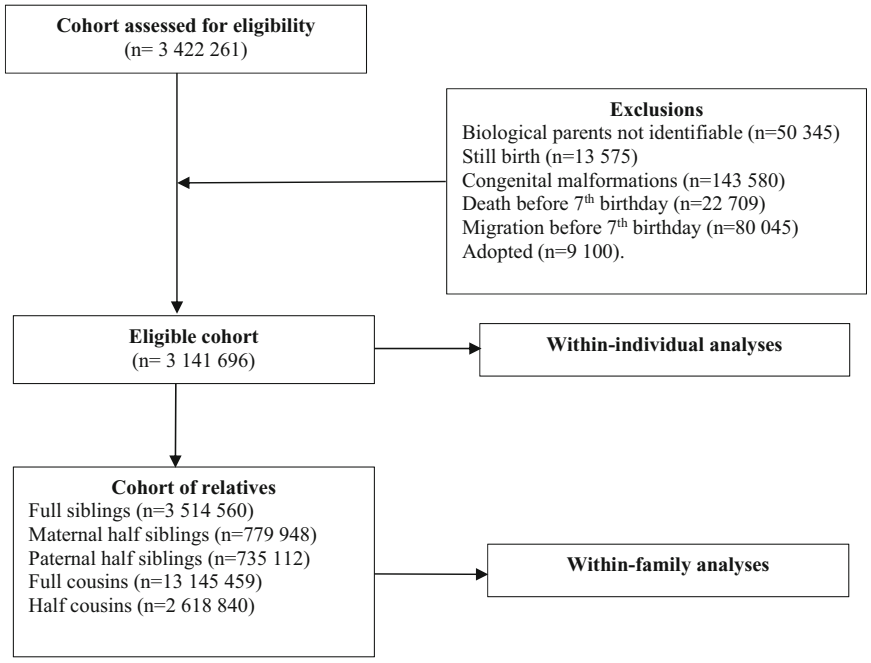

Figure 1 Flow chart of the study population

of Diseases (ICD) diagnosis codes. The SNPR was also used to identify comorbid psychiatric disorders. Additional information about psychiatric comorbidity was obtained from the Swedish Prescribed Drug Register, which contains national information on all dispensed prescribed medication since July 2005.

Family education level was identified by highest education level among any of the biological parents and categorized by three levels: elementary (school year 1–9), secondary (school year 10–14), and higher (university and higher). Three different registers were used for the identification of education levels. For years 1970 to 1984, we used Census (population and housing census); for years 1985 to 1989, register-based labor market statistics (RAMS) which is a database combing statistics containing information on employment, commuters, employees, and industrial structures; and for years 1990 to 2006 the longitudinal integration database for educational and social sectors, health and insurance, as well as labor market (LISA).

Ascertainment of ASD diagnosis and dichotomization of ASD with and without ID

Individuals with an ASD diagnosis at age >1 between 1974 and 2006 were identified from the SNPR. ICD-9 ASD diagnoses from 1987 to 1996 were converted to corresponding ICD-10 ASD diagnoses from 1997 and onward using a conversion instrument provided by the Swedish National Board of Health and Welfare. The included diagnoses in the final cohort were autism (F84.0), Asperger syndrome (F84.5), atypical autism (F84.1), and pervasive developmental disorder – not otherwise specified (F84.9), other childhood disintegrative disorder (F84.3), and other pervasive developmental disorders (F84.8). The dichotomization into ASD+ID or ASD−ID was based on registered ICD codes for intellectual disability: Mild mental retardation (F70), Moderate mental retardation (F71), Severe mental retardation (F72), Profound mental retardation (F73), Other mental retardation (F78), and Unspecified mental retardation (F79). The validity of psychiatric diagnoses in the SNPR has generally considered to be high, and the validity of the ASD diagnoses specifically has also been found to be high (Ludvigsson et al., 2011; Ludvigsson, Reichenberg, Hultman, & Murray, 2013).

Ascertainment of obesity

The SNPR was used to identify individuals diagnosed with obesity at age >5 between 1978 and 2006 (ICD-8 1969–1986 277,99; ICD-9 1987–1996 278A, 278B; ICD-10 1997–2013 E65, E66.0, E66.1, E66.2, E66.8, E66.9).

Ascertainment of psychiatric comorbidity

Based on previous research, neurodevelopmental disorders, eating disorders, substance use disorder, depression, and anxiety were included as possible covariates. Lifetime psychiatric comorbidity with ADHD was extracted from the SNPR using ICD-9 codes: 314J, 314W, 314X and ICD-10 code F90, and from the Swedish Prescribed Drug Register using the Anatomical Therapeutic Chemical (ATC) codes N06BA01 (amphetamine), N06BA02 (dexamphetamine), N06BA04 (methylphenidate), N06BA09 (atomoxetine), and N06BA12 (lisdexamfetamine). Lifetime comorbidity with depression at age >5 was identified by ICD-8 codes 296.2, 298.0, and 300.4; ICD-9 codes 296B, 298A, and 311; and ICD-10 codes F32 and F33. Lifetime comorbidity with anxiety at age >5 was identified by ICD-8 codes 300; ICD-9 codes 300, ICD-10 codes F40-F42, F44-45, and F48. Lifetime comorbidity with substance use disorder at age >10 was identified with ICD-8 codes 303 and 304; ICD-9 codes 303-305; ICD-10 codes F10-F19. Lifetime comorbidity with neurodevelopmental disorders at age >2 was identified with ICD-10 codes F80.0-F80.2, F80.4, F80.8, F80.9, F81.0, F81.2, F81.8, F81.9, and F82. Lifetime comorbidity with eating disorders at age >10 was identified as ICD-9 codes 307B or 307F and ICD-10 codes F50.0-F50.3. Antidepressant and antipsychotic medication use was extracted from the Swedish Prescribed Drug Register using Anatomical Therapeutic Chemical (ATC) codes (full details are provided in Table S1).

Statistical analyses

First, the association between ASD and obesity was evaluated by comparing the risk of obesity between individuals with and without ASD. Logistic regression models were used to estimate the strength of the association between ASD and obesity. The

measures of association are presented as odds ratios (ORs) with 95% confidence intervals (CIs). The crude associations were adjusted for sex and birth year. The associations were then adjusted for maternal and paternal education, psychiatric comorbidity, and medications. All analyses were stratified by sex and were conducted separately for the ASD+ID and ASD−ID groups. In order to test if there were significant differences in the association of ASD and obesity, an interaction term of ASD and sex was included.

In the second series of the analyses, shared familial risk factors were assessed by comparing risk estimates separately for first-degree relatives (full siblings), second-degree relatives (maternal- and paternal half-siblings), and third-degree relatives (full- and half-cousins). Logistic regression was used to estimate the risk of obesity among relatives of ASD and relatives of individuals without ASD across the different levels of relatedness. Measures of association were presented as ORs with 95% CIs. We used cluster-robust estimation to adjust for nonindependence of the data within family clusters. In accordance with earlier research from your group, the familial analyses were adjusted for sex and birth year in the patients and their relatives (Chen et al., 2017, 2019; Yao et al., 2019).

Increased risk of obesity among relatives of individuals with ASD compared with relatives of individuals without ASD would suggest familial effects on the association between ASD and obesity.

All statistical analyses were performed in SAS 9.4 and STATA 16.0.

Sensitivity analyses

For sensitivity analyses, the pattern of associations between ASD and obesity was first explored by restricting the ascertainment of diagnoses to ICD-10 only, rather than by ICD-8, ICD-9, and ICD-10. We also further restricted the definition to ICD-10 codes from specialist outpatient care only rather than from inpatient and outpatient specialist care. The rationale for restrictions is that diagnostic assessment of ASD was rare in Sweden before 1990 and most patients are diagnosed in the specialized outpatient care. ICD-10 was published 1997 in Sweden and outpatient data were included in the SNPR from 2001 and onward. Before 2001, the SNPR only contained diagnoses recorded in inpatient patient care.

Results

Descriptive statistics

Among the 3,140,696 individuals eligible for the study in this Swedish nationwide cohort, 35,461 individuals (r females) had an ASD diagnosis (1.18% prevalence). Of these, 6,025 individuals had ASD+ID (0.19% prevalence) and 29, 436 had ASD−ID (0.94%). The prevalence of obesity was 5.94% in the ASD+ID group and 5.30% in the ASD−ID group, while the corresponding prevalence estimates in individuals without ASD were 1.97% (see Table 1). Mean age at first obesity diagnosis were lower for individuals with ASD+ID (14.32 years) and ASD−ID (16.67 years) than for individuals without ASD (21.22 years). This difference was statistically significant ($p < .000$). The familial aggregation analyses included 3,824,736 full siblings, 14,041,940 full cousins, and 2,929,492 half-cousins.

The association between ASD and obesity

Individuals with ASD+ID (OR = 3.76 [95% CI, 3.38–4.19]) and ASD−ID (OR = 3.40 [95% CI, 3.23–3.58]) had an increased risk for obesity compared with individuals without ASD. The associations were

Table 1 Descriptive statistics of the total study population (n = 3,141,696)

	ASD+ID	ASD−ID	No ASD
N total	6,435 (0.20%)	30,700 (0.97%)	3,104,561
Females	2,002 (0.13%)	9,341 (0.61%)	1,534,314
Males	4,023 (0.25%)	20,095 (1.25%)	1,607,386
Mean age at first registered ASD diagnosis (*SD*)	13.15 (7.84)	16.54 (8.21)	–
Mean age at first obesity diagnosis (*SD*)	14.32 (6.65)	16.67 (8.03)	21.22 (10.29)
Obesity			
All	358 (5.94%)	1,560 (5.30%)	59,866 (1.97%)
Females	133 (6.66%)	614 (6.57%)	40,430 (2.73%)
Males	225 (5.56%)	946 (4.71%)	19,436 (1.24%)
Comorbid disorder			
ADHD	2,055 (34.11%)	14,587 (49.55%)	89,565 (2.97%)
Other neurodevelopmental disorder	875 (13.60%)	2,308 (7.52%)	
Depression	634 (9.85%)	8,042 (26.19%)	119,354 (3.84%)
Anxiety	641 (10.64%)	7,319 (24.86%)	121,330 (3.91%)
SUD	263 (4.37%)	2,968 (10.08%)	104,274 (3.36%).
Eating disorder	86 (1.33%)	962 (3.13%)	
Psychotropic medication	1019 (15.84)	3,897 (12.69%)	
Mother education level			
Elementary	1,076 (17.98%)	3,655 (12.43%)	393,802 (12.69%)
Secondary	3,308 (50.77%)	14,485 (49.27%)	1,498,062 (48.25%)
Higher	1,870 (31.25%)	11,259 (38.30%)	1,205,499 (38.83%)
Father education level			
Elementary	1,303 (21.76%)	5,193 (17.72%)	605,716 (19.51%)
Secondary	3,098 (51.75%)	15,292 (52.17%)	1,529,377 (49.27%)
Higher	1,586 (26.49%)	8,826 (30.11%)	955,397 (30.78%)

marginally attenuated when adjusting for parental education (ASD+ID OR = 3.44 [95% CI, 3.09–3.83]; ASD−ID OR = 3.35 [95% CI, 3.18–3.53]), and further attenuated but remained statistically significant when also adjusting for psychiatric comorbidity (ASD+ID OR = 2.17 [95% CI, 1.94–2.43]; ASD−ID OR = 1.67 [95% CI, 1.58–1.77]) and medication (ASD+ID, OR = 1.97 [95% CI, 1.76–2.21]; ASD−ID, OR = 1.56 [95% CI, 1.48–1.66]). Sex-stratified analyses indicated a higher relative risk for males compared with females, with statistically significant interaction effects for ASD−ID ($p < .0001$.), but not for ASD+ID ($p = .888$) in the fully adjusted model (ASD−ID Male OR = 1.67 [95% CI, 1.55–1.80]; Female OR = 1.34 [95% CI, 1.22–1.46]; ASD+ID Male OR = 2.04 [95% CI, 1.77–2.36]; Female OR = 1.71 [95% CI, 1.43–2.05]) (see Table 2).

Familial coaggregation between ASD and obesity

First-degree relatives of individuals with ASD+ID (full siblings OR = 1.78 [95% CI, 1.54–2.06] and ASD−ID (full siblings OR = 1.79 [95% CI, 1.67–1.92]) had an increased risk of obesity compared with first-degree relatives of individuals without ASD. The obesity risk was similar in second-degree relatives of individuals with ASD+ID (maternal half-siblings OR = 1.63 [95% CI, 1.25–2.14]; paternal half-siblings OR = 1.71 [95% CI, 1.29–2.25]), but was lower for ASD−ID (maternal half-siblings OR = 1.41 [95% CI, 1.23–1.61]; paternal half-siblings OR = 1.21 [95% CI, 1.03–1.41]). Full cousins of individuals with ASD+ID had a higher risk compared with half-cousins of individuals with ASD+ID (full cousins OR = 1.37 [95% CI, 1.25–1.50]; half-cousins OR = 1.17 [95% CI, 1.01–1.36]). As similar difference in the obesity risk between full cousins and half-cousins was observed for ASD−ID (full cousins OR = 1.20 [95% CI, 1.15–1.25]; half-cousins OR = 1.12 [95% CI, 1.05–1.21]) (see Table 3 and Figure 2).

Sensitivity analyses

The pattern of associations between ASD and obesity were the same when restricting the diagnoses to ICD-10, but not when restricting to ICD-10 diagnoses in outpatient mental health services only. In the main analyses, the CIs were nonoverlapping in the ASD−ID group between males and females in both the crude and adjusted models. The CI`s were overlapping between males and females in the ASD−ID group in all models when restricting to outpatients. See Tables S2 and S3.

Discussion

In this large population-based study, we found an increased risk for obesity in individuals with ASD while controlling for sex, parental education level, psychiatric comorbidity, and medication, which is consistent with previous research (de Vinck-Baroody et al., 2015; Dreyer Gillette et al., 2015; Must et al., 2017; Zheng et al., 2017) and highlights the need for intervention studies and treatment guidelines for co-occurring obesity in individuals with ASD. The mean age for obesity diagnosis was lower for individuals with ASD+ID (14.32 years) and ASD−ID (16.67 years) than for individuals without ASD (21.22 years). All children and adolescents in Sweden are included in school healthcare-based health screenings following BMI and weight development throughout the 9-year compulsory school. Therefore, the age differences in the first obesity diagnoses most probably are not due selection effects of having a diagnosis of ASD. Rather, these differences probably reflect the heightened risk for obesity in individuals with ASD. These results indicate that preventive programs and interventions regarding overweight should begin early in life for individuals with ASD.

For the first time, we demonstrated that relatives of individuals with ASD have a higher risk for obesity compared with relatives to individuals without ASD. We found that the strength of the familial coaggregation of ASD and obesity attenuated with decreasing genetic and environmental relatedness (i.e. from full siblings to half-cousins), in particular for ASD−ID. This pattern indicates that at least part of the shared familial factors (genetic and/or shared environmental factors) between ASD and obesity is

Table 2 Associations between ASD and obesity

	ASD+ID				ASD−ID			
	Crude	Adjusted for SES	Adjusted for comorbidity	Adjusted for medication	Crude	Adjusted for SES	Adjusted for comorbidity	Adjusted for medication
Obesity								
All	3.76 (3.38–4.19)	3.44 (3.09–3.83)	2.17 (1.94–2.43)	1.97 (1.76–2.21)	3.40 (3.23–3.58)	3.35 (3.18–3.53)	1.67 (1.58–1.77)	1.56 (1.48–1.66)
Females	2.98 (2.51–3.53)	2.73 (2.30–3.25)	1.89 (1.58–2.26)	1.71 (1.43–2.05)	2.79 (2.57–3.02)	2.75 (2.53–2.99)	1.41 (1.29–1.55)	1.34 (1.22–1.46)
Males	4.11 (3.60–4.70)	3.73 (3.25–4.27)	2.24 (1.94–2.58)	2.04 (1.77–2.36)	3.65 (3.42–3.90)	3.57 (3.34–3.82)	1.80 (1.66–1.94)	1.67 (1.55–1.80)

The relative risk for the ASD groups compared with the general population expressed as odds ratios (95% confidence interval). Adjusted for SES, adjusted for sex, birth year, maternal, and paternal education; ASD, autism spectrum disorder; Crude, adjusted for sex and birth year; ID, intellectual disability; OR, odds ratios. Adjusted for Comorbidity, adjusted for sex, birth year, maternal, and paternal education, ADHD, and other neurodevelopmental disorders, substance abuse, depression, anxiety, and eating disorders. All ORs are significant at $p < .0001$.

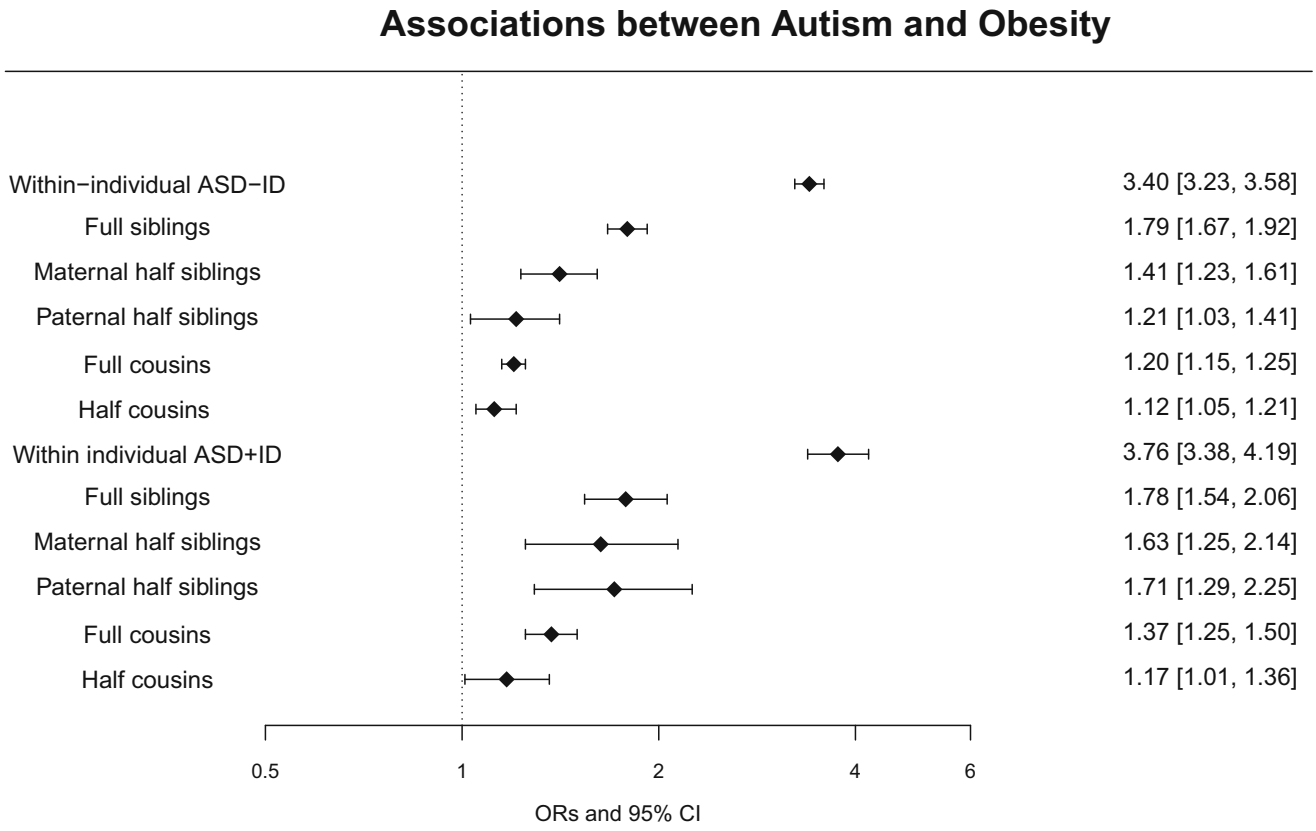

Figure 2 Within individual and within family associations between ASD and obesity

due to genetic factors, which is consistent with a recent GWAS (Grove et al., 2019) reporting on a statistically significant genetic correlation (rG = 0.092–0.127).

The genetic overlap between ASD and obesity might be explained by common mechanisms such as reward circuitries associated with both eating behavior and repetitive and restricted behaviors (Kohls, Antezana, Mosner, Schultz, & Yerys, 2018; Loviglio et al., 2017; Maillard et al., 2015). This warrants further studies examining potential common pleiotropic genetic factors and shared family-wide environmental factors for ASD and obesity. Such research might aid in identifying specific risks and underlying mechanisms in common between ASD and obesity, which in turn may facilitate improved intervention or prevention. We also note that the strength of the association in full siblings between ASD and obesity was weaker compared to previously observed full-sibling associations between ASD and other psychiatric conditions (e.g. ADHD) (Ghirardi et al., 2018; Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010; Wang et al., 2020). Furthermore, compared to previous familial coaggregation studies of ASD and other psychiatric conditions (Sundelin et al., 2016; Wang et al., 2020), we found less strong support for decrease in the obesity risk as a function of genetic relatedness. Together, this suggests a stronger genetic overlap between ASD and other psychiatric conditions than

Table 3 Familial coaggregation of ASD and obesity

	ASD total crude		ASD+ID adjusted*		ASD−ID adjusted[a]	
	OR (95% CI)	*p*-Value	OR (95% CI)	*p*-Value	OR (95% CI)	*p*-Value
Full siblings	1.71 (1.60–1.82)	<.0000	1.78 (1.54–2.06)	<.0000	1.79 (1.67–1.92)	<.0000
Maternal half-siblings	1.37 (1.22–1.55)	<.0000	1.63 (1.25–2.14)	<.0000	1.41 (1.23–1.61)	<.0000
Paternal half-siblings	1.25 (1.09–1.43)	>.002	1.71 (1.29–2.25)	<.0000	1.21 (1.03–1.41)	<.018
Full cousins	1.19 (1.14–1.24)	<.0000	1.37 (1.25–1.50)	<0.000	1.20 (1.15–1.25)	<.0000
Half-cousins	1.11 (1.04–1.19)	<.001	1.17 (1.01–1.36)	0.032	1.12 (1.05–1.21)	>.001

Risk of obesity in relatives of individuals with ASD, compared with relatives of individuals without ASD. ASD total, ASD with intellectual disability + ASD without intellectual disability; ASD, autism spectrum disorder; CI, confidence interval; ID, intellectual disability; OR, odds ratio.
[a]Adjusted for sex, sex relative, birth year, and birth year relative.

for ASD and obesity and also that shared family-wide environmental factors may play a more important role for ASD and obesity than for ASD and other psychiatric conditions.

Our finding of an association between autism and obesity in both ASD−ID and ASD+ID, suggests that the association between autism and obesity is not entirely driven by ID, a known risk factor for obesity (Maïano, Hue, Morin, & Moullec, 2016). This finding is inconsistent with a previous study indicating that the association between ASD and obesity no longer reached significance when controlling for ID (Corvey et al., 2016). One possible explanation for the discrepant findings may be differences in the age distribution of the study participants across the two studies. The previous study investigated the association between ASD and obesity in children (Corvey et al., 2016), while the present study explored the same association across the lifespan. The association between ASD and obesity while controlling for ID could be explored further in future studies by using an ID only comparison group, preferably using a population-based sample. Our finding that it is something specific to ASD that increases the risk for obesity has important clinical implications. Individuals with ASD should be screened for the risk for obesity, regardless of ID status. Clinicians should also screen for ASD in individuals with obesity.

In contrast to previous research (Must et al., 2017), reporting an association between ASD and obesity only in males, we were able to demonstrate that the association between ASD and obesity was present in both males and females. The nonsignificant association in females in the study by Must et al. (2017) might be due to limited statistical power. Our study findings possibly suggest a stronger association between ASD and obesity in males compared with females in the ASD−ID group. A research implication of our robust findings of associations between ASD and obesity regardless of sex and possible sex differences is that future studies on this association should include both sexes and also have sufficient power for sex-specific analyses. Clinically, both men and women with ASD should be screened for obesity risk.

In accordance with earlier studies (Dreyer Gillette et al., 2015; de Vinck-Baroody et al., 2015; Must et al., 2017; Zheng et al., 2017), the association between ASD and obesity was somewhat attenuated but remained significant when controlling for psychiatric comorbidity, including ADHD, which in itself is associated with obesity (Chen et al., 2017; Cortese et al., 2016). This is in line with an earlier population-based study, which also found an effect of ASD on high body mass index even in the absence of ADHD (Kerekes et al., 2015). Finding that the association between ASD and obesity remains significant when controlling for both internalizing and externalizing problems, and neurodevelopmental disorders is interesting in the light of the recent findings of a general psychopathology factor that posits that most of the shared variance between psychiatric disorders can be accounted for by a common factor (Caspi et al., 2014; Pettersson, Larsson, & Lichtenstein, 2016; Selzam, Coleman, Caspi, Moffitt, & Plomin, 2018). Our results suggest that it is something above and beyond other psychiatric comorbidities (i.e. anxiety, depression, SUD, and ADHD) or psychotropic medication that links ASD with obesity. One potential factor might be food selectivity due to sensory processing alterations, which is heritable and common in ASD (Chen et al., 2017; Chistol et al., 2018; Cortese et al., 2016; Smith et al., 2016; Taylor et al., 2018). Another explanation could be inflexibility which is common in ASD, leading to difficulties in switching between different kinds of food (Zickgraf, Richard, Zucker, & Wallace, 2020). It is also possible that lifestyle factors such as sedentary behaviors can explain part of the association between ASD and obesity (Jones et al., 2017). This warrants further exploration, preferably in genetically sensitive studies.

Strengths and limitations of the study should be taken into account when interpreting the validity of our findings. An important strength of the study is the large population-based sample collected from several nation-wide Swedish registers enabling use of the entire Swedish population as study base. It decreases the risk of misclassification, selection bias, and recall bias or unwillingness to report sensitive data. Our sample of 3.1 million individuals was created from the Swedish Total Population Register (STPR). Individuals moving to Sweden and planning to stay there for ≥1 year will in most cases be recorded in the TPR. Close to 100% of births and deaths, and 95% of immigrations are reported to the TPR within 30 days and with a higher proportion over time. About 25% of the Swedish population has foreign background, so we can expect that our cohort is quite diverse in terms of ethnicity (Ludvigsson et al., 2016).

Another strength is that we had power enough to detect the robust associations between ASD and obesity in both males and females. A possible limitation of the study is the assessment of depression in the SNPR. Mild depression is often treated within primary care services rather than in specialized psychiatric care (Sundquist, Ohlsson, Sundquist, & Kendler, 2017). We therefore most probably only included the most severe cases of depression in the adjusted analyses. In the same vein, the coverage of ID in the SNPR is likely limited too. This is probably also the case with obesity. The individuals with obesity who are present in the SNPR are probably the most severe cases who often develop complications as a result of their obesity and therefore need specialized health care.

Conclusions

We found an increased risk for obesity in both ASD+ID and ASD−ID when controlling for

socioeconomic factors, psychiatric comorbidity, and medications, and observed statistically significant associations in both males and females. Overall, the effect of sex was limited, but with possible stronger associations between ASD and obesity in males than in females in the ASD−ID group. The familial coaggregation analyses suggested that the association between ASD and obesity is partly due to shared familial factors. Further research should be encouraged to investigate the neurobiological and behavioral mechanisms underlying the association between ASD and obesity.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Table S1. Associations between ASD and obesity- ICD-diagnoses only.

Table S2. Associations between ASD and obesity-outpatients only.

Table S3. Anatomical therapeutical chemical codes and corresponding medical name.

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Key points

- ASD and obesity tend to co-occur, but little is known about the nature of the association and the influence of ID, sex, psychiatric comorbidity, medications, and SES.
- In a nationally representative cohort of 3,141,696 individuals, we showed that ASD is associated with obesity when controlling for all covariates.
- This association is partly due to shared familial factors (genetic and/or shared environmental factors).
- Individuals with ASD should be assessed for obesity risk. Furthermore, screening for ASD should be included for individuals with obesity, especially in patients with unsuccessful treatment attempts.

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