Received: 30 December 2022 | Accepted: 17 August 2023

DOI: 10.1111/dmcn.15757

ORIGINAL ARTICLE

Prevalence of attention-deficit/hyperactivity disorder and autism in 12-year-old children: A population-based cohort

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Funding information
Forskning och Utveckling, Halland, Grant/Award Number: HALLAND-979135; Göteborgs Läkaresällskap, Grant/Award Number: GLS-779231; Svenska Läkaresällskapet, Grant/Award Number: SLS-973255; Västra Götalandsregionen, Grant/Award Number: ALFGBG-507931, ALFGBG-719711 and ALFGBG-965041

Abstract

Aim: To investigate the prevalence of attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) in a population-based birth cohort and correlate the findings with prenatal and perinatal factors. We hypothesized that children born preterm, having experienced preeclampsia or maternal overweight, would have an increased risk of ADHD or ASD.

Method: A Swedish cohort of 2666 children (1350 males, 1316 females) has been followed from birth with parental and perinatal data. The National Board of Health and Welfare's registries were used to collect data regarding perinatal status and assigned diagnoses at the age of 12 years.

Results: The prevalence of ADHD and ASD was 7.6% and 1.1% respectively. Maternal obesity early in pregnancy resulted in a three-fold increased risk of ADHD in the child. Similarly, paternal obesity resulted in a two-fold increased risk. The association was significant also when adjusted for sex, preterm birth, smoking, and lower educational level. The prevalence of ASD was too low for statistically relevant risk factor analyses.

Interpretation: Our results corroborate earlier findings regarding prevalence and sex ratio for both ADHD and ASD. Maternal body mass index and preterm birth were correlated with an ADHD diagnosis at the age of 12 years.

Attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are prevalent neurodevelopmental disorders. The worldwide ADHD prevalence is around 6% among children and adolescents.[1, 2] The prevalence of ASD has been estimated at approximately 1%.[3] ADHD and ASD diagnoses have increased over time; however, during the last decades the prevalence has been stable.[4, 5]

This original article is commented on by Kuhn on pages 411–412 of this issue.

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; ASD, autism spectrum disorder; BMI, body mass index

The current consensus in the field is that the prevalence of ADHD and ASD is relatively constant, but the recognition of the diagnoses, as well as the knowledge and subsequent detection, has increased in recent years.[2, 4–7] Nygren et al. concluded that the increase in the reported prevalence of ASD during the last two decades is the result of a change in definition, broader diagnosis criteria, and an increased awareness of ASD, both among professionals and the general population.[5] A population-based study with 9 594 598 participants in the UK[7] showed an increase in the incidence of ASD between 1998 and 2018, particularly among females and adults. According to the authors, this could be explained by an improved awareness of the symptoms among adults and females during the study period.

The typical symptoms of ADHD are hyperactivity, impulsivity, and/or inattention. The ADHD symptoms should be present continuously for at least 6 months and the ADHD symptomatology onset should occur before the age of 12 years. The ADHD criteria also state that symptoms should be present in different settings and have an impact on general life. ASD is characterized by persistent difficulties with social interaction and social communication, and restricted and repetitive patterns of behaviour, activities, and interests. Symptoms should be present in early childhood and with a severity that limits or impairs everyday functioning.[3]

Both ADHD and ASD are highly heritable, but are also associated with environmental factors.[8, 9] The risk of ADHD was recently shown to covary with preterm birth, small for gestational age, and preeclampsia.[10] Moreover, Andersen et al. showed an increased risk of both ADHD and ASD for the child when the mother was overweight at the onset of pregnancy.[11] Systemic inflammation has been described as the link between increased maternal body mass index (BMI) and neurodevelopment deviances in children.[12] There are few studies that report an association between paternal BMI and ADHD in the offspring.

The aim of this study was to identify the prevalence of ADHD and ASD in a population-based birth cohort of 12-year-old children and the association with perinatal and parental factors. We hypothesized that a high maternal and/or paternal BMI early in pregnancy would be associated with an increased risk of neurodevelopmental disorders in the offspring compared with children in the background population.

What this paper adds

- The prevalence of attention-deficit/hyperactivity disorder (ADHD) was 7.6%.
- The prevalence of autism spectrum disorder was 1.1%.
- Increased maternal and paternal body mass index were risk factors for ADHD in children.
- Parental smoking correlated with ADHD in children.
- Parental educational level was related to the occurrence of ADHD in children.

METHOD

Participants

This study is part of a population-based longitudinal birth cohort comprising 2666 children. The children were born in the Halland region in south-western Sweden, between October 2007 and December 2008. In total, there were 3860 births in the region during the recruitment period. The parents of 3856 newborn infants in the region were asked to participate in the cohort during their first visit to the child health care centre. Of these, 376 parents chose not to participate and 814 did not respond to the request. The study cohort was originally described by Almqvist-Tangen et al. in 2012.[13] Except for the consent form being in Swedish, there were no limitations to the recruitment. The study population was considered to be representative of children born in Sweden during the same time period regarding sex, birthweight, and gestational age.[13]

Study design

Background data were collected through parent data forms and the National Medical Birth Register. Data on ADHD and ASD diagnoses and medical treatment with stimulants were collected from the National Board of Health and Welfare registries. The collection of data until 31st December 2019 was approved by the National Board of Health and Welfare on 12th January 2021. The data were obtained from the National Patient Register, the National Prescribed Drug Register, and the Cause of Death Register.

The study was approved by the regional ethical review board in Lund (299/2007 and 141/2018). Written informed consent was obtained from the parents when they chose to participate in the study.

Variables

The main outcome variable of the study was the prevalence of ADHD and ASD in the study population, based on diagnostic codes as registered in the National Patient Register. The diagnostic codes from the National Patient Register have been cross-correlated with data on prescribed drugs from the National Prescribed Drug Register. Six individuals had been prescribed central stimulants without a corresponding ADHD diagnosis. Five of those had received several prescriptions of methylphenidate, a pharmaceutical only prescribed to treat ADHD. These individuals have therefore been treated as if they had an ADHD diagnosis.

One child had methylphenidate prescribed once and was not considered treated for ADHD.

Parental and perinatal data were collected through a questionnaire given to the parents during their first visit to the child health care centre. Where there were data from both the National Board of Health and Welfare's registries and self-reported responses from the questionnaire, priority has been given to the registry data. The self-reported and registry data were generally in agreement. However, the self-reported data (e.g. on weight) tended to be rounded off to the nearest multiple of five. Further variable descriptions and missing data can be found in Appendix S1.

Statistical methods

The statistical analysis was conducted using IBM SPSS Statistics version 28 (IBM Corp., Armonk, NY, USA), NumPy,[14] and Pandas.[15] Dependent variables were diagnoses for ADHD or ASD. Independent variables were sex, gestational age, parental smoking, BMI groupings, and education levels, all either categorical or ordinal and chosen based on previous research and socioeconomic factors.[2, 10, 11] Where there were missing data points, the affected individuals have been excluded from that subanalysis. Statistically significant differences between independent variables were analysed using Pearson's χ^2 tests. Comparisons between groups with BMI as a quantitative variable were made with one-sample *t*-test. A *p*-value below 0.05 was considered significant. Crude and multivariable binary logistic regression analyses were conducted to investigate associations between all the independent variables when predicting the likelihood of ADHD.

RESULTS

From the original population-based birth cohort of 2666 children, this study successfully followed up 2658 children. Two children from the original cohort were deceased and six children were listed as emigrated and therefore not included (see Figure S1).

Table 1 shows the characteristics of the study population, including 1313 females and 1345 males. Among the children born preterm, 16 were born before week 32 and the lowest recorded gestational age in the cohort was week 27. At the time of follow-up, the children had a median age of 11 years 7 months 28 days with a range from 11 years 0 months 0 days to 12 years 2 months 30 days. The prevalence of ADHD and ASD was 7.6% ($n=203$) and 1.1% ($n=28$) respectively; 22 of the children with ASD also had ADHD. When divided into subgroups, the ASD cases were too few for further statistically relevant analyses; two cases were born before week 37 and four cases had a mother who smoked at the time of pregnancy.

Table 2 shows the statistics for the frequencies of ADHD based on perinatal and parental data. Of the children with ADHD, 142 were male and 61 were female (i.e. a male:female ratio of 2.3:1). For ASD, the corresponding numbers were 20 male and eight female, corresponding to a male:female ratio of 2.5:1. Among children born preterm, 13.7% had an ADHD diagnosis at the age of 12 years, compared to 7.3% among children born at term, a nearly two-fold increase. Among the three children in the study cohort with no reported gestational age, one child had both ADHD and ASD. The birthweight indicates no preterm birth in accordance with Swedish references for infant size at birth[16] and assuming this individual was born at term it would only have marginal effect on the risk of ADHD (yielding $\chi^2=6.31$, $p=0.012$ compared to $\chi^2=6.41$, $p=0.011$, reported in Table 2).

A strong association was found between the mother being overweight or obese at the beginning of pregnancy and the risk of an ADHD diagnosis in the child. Table 2 shows the difference in the prevalence of ADHD, when the mother had normal weight, as opposed to overweight or obesity. When the mother had a BMI exceeding 25 kg/m^2 the child's risk of ADHD doubled (11.4%) compared to when the mother had a normal weight (5.7%). The association between maternal BMI in early pregnancy and prevalence of ADHD in the offspring was dose-dependent, that is, there was a stronger association between maternal obesity and offspring with ADHD ($\chi^2=42.58$, $p<0.001$) than maternal overweight and offspring with ADHD ($\chi^2=8.44$, $p=0.004$).

Figure 1 illustrates the risk of ADHD in the child as a function of parental BMI. Among the 201 children with ADHD, 50.7% ($n=102$) had a mother with normal weight, 28.4% ($n=57$) children had a mother with overweight, and 20.9% ($n=42$) had a mother with obesity. The corresponding numbers for children with no ADHD diagnosis are 68.5% ($n=1673$), 23.3% ($n=569$), and 8.3% ($n=202$) respectively. BMI could not be calculated because of missing weight and/or height for 0.5% ($n=13$) of mothers.

In the paternal BMI analysis, there were 333 (12.5%) missing cases. Paternal BMI did not show any significant association with the risk of ADHD in the child (using a cut-off of BMI ≥25 kg/m^2). However, among fathers with obesity (BMI ≥30 kg/m^2), 12.8% of their children had ADHD, compared to 6.4% of the children with normal-weight fathers (< 25 kg/m^2). Furthermore, the maternal and paternal BMI were statistically significantly correlated (r = 0.21) Out of the 2317 children, 8.0% ($n=186$) had a father with obesity. Of these, 23.1% ($n=43$) also had a mother with obesity.

Binary logistic regression

Binary logistic regression analyses were conducted as crude and multivariable analyses (see Table 3). On average, children born before week 37 had 2.13 (95% confidence interval [CI] 1.21–3.74) times the odds of having ADHD. Moreover, maternal overweight or obesity at the onset of pregnancy corresponded to an increased risk of ADHD in the child with a factor of 1.89 (95% CI 1.40–2.54). Preterm birth, maternal smoking, or BMI above 2 kg/m^2 were shown to be significant as independent variables when calculating the probability of ADHD.

TABLE 1 Population characteristics including perinatal and parental factors from birth to study period with the outcome of attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD).

	Birth cohort ($n = 2666$)	Study population ($n = 2658$)	ADHD ($n = 203$)	ASD ($n = 28$)
Sex				
Males	1350	1345	142	20
Females	1316	1313	61	8
Birthweight (g)				
< 2500	90	90	9	2
2500–4499	2468	2461	182	24
≥ 4500	107	106	12	2
Gestational age (weeks)				
< 37	117	117	16	2
≥ 37	2545	2538	186	25
Preeclampsia				
Yes	77	77	9	2
No	2589	2581	194	26
Small for gestational age				
Yes	44	44	5	1
No	2484	2484	186	43
Maternal age (years)				
< 25	330	329	40	5
25–34	1803	1796	129	17
≥ 35	533	533	34	6
Maternal education (years)				
≤ 12	1303	1298	126	13
> 12	1328	1325	75	14
Maternal smoking				
Yes	215	214	30	4
No	3443	3452	173	24
Maternal BMI (kg/m^2)				
< 25	1783	1775	102	18
≥ 25	873	870	99	10
Paternal education (years)				
≤ 12	1598	1592	149	20
> 12	884	880	39	6
Paternal smoking				
Yes	258	258	33	4
No	3400	3408	170	24
Paternal BMI (kg/m^2)				
< 25	1181	1180	75	9
≥ 25	1152	1145	96	13

Abbreviation: BMI, body mass index.

Maternal age was excluded from the regression analysis since low maternal age was strongly associated with low maternal educational level; only 9% of mothers below the age of 25 years had more than 12 years of education. When maternal age was included in the multivariate analysis, only a negligible effect on other factors was seen, including no effect on the significance level.

DISCUSSION

This study showed that the prevalence of ADHD and ASD in 12-year-old children in a population-based longitudinal birth cohort in south-western Sweden was 7.6% and 1.1% respectively. Preterm birth, parental smoking, and lower parental education was confirmed to have an association with

TABLE 2 Attention-deficit/hyperactivity disorder (ADHD) frequencies and the differences associated with perinatal and parental data.

	ADHD (*n*)	%	χ^2	*p*
Sex	142	10.6		
Males	61	4.6	32.92	<0.001[a]
Females				
Gestational age (weeks)				
<37	16	13.7		
≥37	186	7.3	6.41	0.011[a]
Maternal age (years)				
<25	40	12.1		
≥25	163	7.1	10.88	<0.001[a]
Maternal education				
≤12	126	9.7		
>12	75	5.7	15.18	<0.001[a]
Maternal smoking				
Yes	30	14.0		
No	173	7.1	13.44	<0.001[a]
Maternal BMI (kg/m^2)				
<25	102	5.7		
≥25	99	11.4	26.38	<0.001[a]
Paternal education				
≤12	149	9.4		
>12	39	4.4	19.58	<0.001[a]
Paternal smoking				
Yes	33	12.8		
No	170	7.1	10.76	<0.001[a]
Paternal BMI (kg/m^2)				
<25	75	6,4		
≥25	96	8.4	3.51	0.061

Statistical comparison using Pearson's χ^2 test, all done one by one.

[a]p <0.05 was considered significant. Abbreviation: BMI, body mass index.

ADHD in the child. In accordance with the hypothesis, high maternal and paternal BMI was shown to associate dose-dependently with the risk of ADHD in the child.

The prevalence of ADHD in this study population was higher than what has previously been described: 7.6% as compared to 5.9% (95% CI 4.6–7.5).[1] Willcutt also looked at the prevalence of ADHD in studies using different types of diagnostic algorithms, concluding that studies using symptom gauging by parents or teachers yield significantly higher prevalence figures.[1] The ADHD diagnostic criteria were most recently updated in the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) in 2013.[3] The differences between DSM-5 and previous editions include the criterion for the age at onset being changed to symptoms being present before the age of 12 years from the age of 7 years.[3, 17] The prevalence of ASD in the study population (1.1%) was like that in previous publications (1.0%).[18] The majority of children (79%) diagnosed with ASD were also diagnosed with ADHD, in accordance with previous findings.[19]

The male preponderance in ADHD[2] and ASD[7] is well known. In this study population, males outnumbered females with a sex ratio of 2.3:1 for ADHD and 2.5:1 for ASD. These numbers are in accordance with what has previously been found in larger population-based studies covering ADHD.[2] Willcutt found a relationship of 2.3:1 for ADHD between the age of 6 years and 12 years.[1] In 2022, Russell et al. showed a ratio of males to females with ASD of 4:1 in a population-based study. They also found a decreasing male to female ratio during recent years.[7]

We found an increased risk of ADHD in children born preterm, although few children were born before 32 weeks' gestation in this cohort. It has previously been shown that being born before 32 weeks' gestation yields an increased risk of both ADHD[20] and ASD.[21] Moreover, a recently published study showed a dose-dependent increase in ADHD depending on how preterm the infant was born.[10] Because of the small sample size in this cohort, we have not been able to confirm an association between either preeclampsia or small for gestational age and the prevalence of ADHD and ASD, as published by Beer et al.[10]

A dose-dependent association was found between the mother being overweight or obese in early pregnancy and the risk of ADHD in the offspring. This association has previously been shown in several publications during the last decade.[22–25] However, paternal BMI was also associated with an increased risk of offspring with ADHD. Moreover, the increased risk of offspring with ADHD could not be explained by an association between maternal and paternal overweight or obesity. Previous studies have described the causality between maternal obesity and an increased risk of ADHD in the child due to systemic inflammation.[12] This does not, however, explain the association between paternal obesity and an increased risk of offspring with ADHD. It could be that what is actually seen is the heredity association between an increased probability of ADHD and overweight or obesity in the mother and father. In future studies, this heredity component must be further evaluated to determine the extent to which overweight and obesity in the parents is an expression of the hereditary nature of ADHD.

According to the estimation by the Swedish Public Health Agency in 2021, 52% of the Swedish population (58% of all males, 46% of all females) between the age of 16 and 84 years are overweight or obese.[26] The study population has comparable prevalence for both sexes; however, the prevalence tends to increase with age. Overweight or obesity was found in 33% of the mothers early in pregnancy and in 49% of the fathers. Weight and height data for the fathers were only based on self-reported values in the parental survey. The pregnancy could not be expected to have a significant impact on the fathers' weight. However, the drop-out rate regarding the fathers' weight and height was notable. The association between elevated parental BMI and neurodevelopmental diagnoses in the children requires greater preventive efforts.

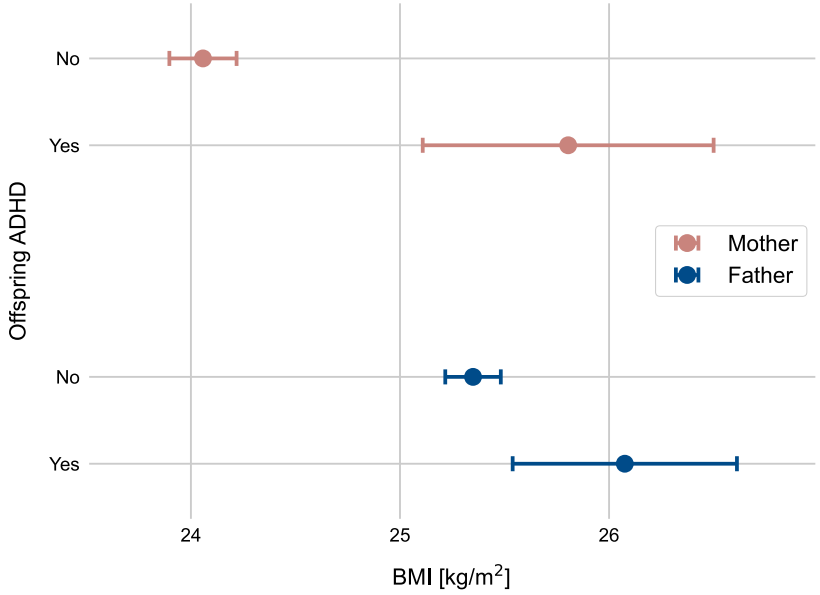

FIGURE 1 The risk of attention-deficit/hyperactivity disorder (ADHD) in the child as a function of maternal (pink) and paternal (blue) body mass index (BMI) at the beginning of pregnancy. Values plotted as mean with 95% confidence interval.

TABLE 3 Results from the binary logistic regression analyses.

	Crude analysis OR (95% CI)		Multivariable analysis OR (95% CI)	
	ADHD (n = 202–200) vs no ADHD (n = 2421–2453)	p	ADHD (n = 198) vs no ADHD (n = 2413)	p
Sex				
Females	1.00		1.00	
Males	2.41 (1.76–3.29)	<0.001[b]	2.39 (1.74–3.27)	<0.001[b]
Gestational age (weeks)				
≥37	1.00		1.00	
<37	2.01 (1.16–3.50)	0.013[b]	2.13 (1.21–3.74)	0.008[b]
Maternal education[a]				
>12	1.00		1.00	
≤12	1.84 (1.37–2.49)	<0.001[b]	1.61 (1.18–2.21)	0.003[b]
Maternal smoking[a]				
No	1.00		1.00	
Yes	2.21 (1.45–3.37)	<0.001[b]	1.64 (1.05–2.58)	0.031[b]
Maternal BMI[a] (kg/m^2)				
<25	1.00		1.00	
≥25	2.05 (1.53–2.75)	<0.001[b]	1.89 (1.40–2.54)	<0.001[b]

Odds ratio (OR) with 95% confidence intervals (CI) for having attention-deficit/hyperactivity disorder (ADHD), given sex, gestational age, and possible risk factors and/or confounders.

[a]In the crude analysis, sex is controlled for gestational age and vice versa. Statistical analysis of a possible association between ADHD and maternal education, smoking and body mass index (BMI) were run one by one controlling for sex and gestational age. In the multivariable analysis, all variables were introduced in one model with ADHD as the dependent variable.

[b]p <0.05 was considered statistically significant.

As expected, we found significant associations between ADHD and lower maternal age, lower maternal and paternal education, as well as smoking. Hypothetically, the above factors could be attributed to ADHD in the parents, that is, the increased probability of ADHD in the offspring is the expression of a hereditary causality. We have no ADHD symptom data for the parents, neither as children nor adults. Moreover, both the ADHD criteria[27] and the view of ADHD as a disease have been subject to change over time.[6] Obesity and ADHD are known to be associated

in both children and adolescents.[28] Likewise, there is an association between overweight/obesity and low education, daily smoking, and lower self-reported well-being.[29] Despite this, we found a substantial association between the probability of ADHD and the maternal BMI subgroup early in pregnancy in regression analyses when adjusting for education and smoking.

The main strength of this study was that a population-based cohort was followed from birth and that these data were combined with nationwide registry data, that is, data based on biological, prenatal, and perinatal variables from a cohort study were combined with diagnostic coding for ADHD and ASD and the prescription of specific drugs. The study design makes it possible to discuss both prevalence and associations. There are, however, some intrinsic limitations regarding this study methodology. Due to low sample size, the associations between perinatal data and ASD were not significant and therefore not presented. A follow-up study spans a long time and, because of legislation, data on exposures are limited to the variables that were initially included. In this case, specifically, there is the potential issue that while maternal weight was both measured and self-reported, paternal weight was solely self-reported, leading to an absence of data. If more accurate, objective, and complete paternal anthropometric measures were collected, an association with the prevalence of ADHD, indicating heredity rather than perinatal adverse environmental effects, may be found.

Conclusion

The prevalence and sex ratios for both ADHD and ASD largely confirm previous findings. Increased parental weight, smoking, and lower educational level seem to be associated with an increased risk of ADHD in the offspring. Whether this is because of an unfavourable perinatal environment, an increased risk of overweight in mothers with ADHD, or rapid weight gain in the offspring, which, in turn, is known to coexist with neurodevelopmental disorders, needs to be addressed in further studies.

ACKNOWLEDGEMENTS

We are very grateful to the children and parents participating in this study. The authors would also like to thank Gerd Almqvist-Tangen and Bernt Alm, and Maria Andersson and Anders Holmén for creating and managing the database.

The study has been financially supported by the Research and Development Department of the Region of Halland (HALLAND-979135), the Swedish Society of Medicine (SLS-973255), the Gothenburg Society of Medicine (GLS-779231), and the Swedish state under the agreement between the Swedish government and the Region Västra Götaland (ALFGBG-507931, -719711 and -965041).

The authors report no conflicts of interest in this article and the funders did not participate in the work.

DATA AVAILABILITY STATEMENT

Data available on request from the authors.

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SUPPORTING INFORMATION

The following additional material may be found online:
Appendix S1: Supporting methods
Figure S1: Flowchart describing the study population

How to cite this article: Fast K, Wentz E, Roswall J, Strandberg M, Bergman S, Dahlgren J. Prevalence of attention-deficit/hyperactivity disorder and autism in 12-year-old children: A population-based cohort. Dev Med Child Neurol. 2024;66:493–500. https://doi.org/10.1111/dmcn.15757