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A twin study of genetic and environmental contributions to attention-deficit/hyperactivity disorder over time

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Background: Attention-deficit/hyperactivity disorder (ADHD) is an increasingly commonly diagnosed neurodevelopmental condition. One possibility is that this reflects a genuine increase in the prevalence of ADHD due to secular environmental changes, yet this hypothesis remains untested. We therefore investigated whether the genetic and environmental variance underlying ADHD, and traits of ADHD, has changed over time. **Methods:** We identified twins born from 1982 to 2008 from the Swedish Twin Registry (STR). We linked the STR with the Swedish National Patient Register and Prescribed Drug Register to identify diagnoses of ADHD and prescriptions of ADHD medication for these twins. We also utilized data collected from participants in the Child and Adolescent Twin Study in Sweden (CATSS), born from 1992 to 2008. Their parents completed a structured ADHD screening tool, which was used to measure traits of ADHD and assign broad screening diagnoses of ADHD. We used the classical twin design to test whether the degree to which variation in these measures was influenced by genetic and environmental variation changed over time. **Results:** We included 22,678 twin pairs from the STR and 15,036 pairs from CATSS. The heritability of ADHD in the STR ranged from 66% to 86% over time, although these fluctuations were not statistically significant. We observed a modest increase in variance in ADHD traits, from 0.98 to 1.09. This was driven by small increases in the underlying genetic and environmental variance, with heritability estimated as 64%–65%. No statistically significant changes in variance in screening diagnoses were observed. **Conclusions:** The relative contribution of genetic and environmental factors to ADHD has remained stable over time, despite its increasing prevalence. Thus, changes in the underlying etiology of ADHD over time are unlikely to explain the increase in ADHD diagnoses. **Keywords:** ADHD; twin study; genetics; environment.

Attention-deficit/hyperactivity disorder (ADHD) is increasingly commonly diagnosed over the last three decades. For example, a global meta-analysis from 2007 reported a pooled prevalence of 5.29%, increasing to 7.2% in a 2015 meta-analysis (Polanczyk, 2007; Thomas, Sanders, Doust, Beller, & Glasziou, 2015). Similarly, ADHD medication is increasingly prescribed in the USA and Europe (Bachmann et al., 2017; Beau-Lejdstrom, Douglas, Evans, & Smeeth, 2016; Castle, Aubert, Verbrugge, Khalid, & Epstein, 2007; Karlstad et al., 2016). There is thus a need to understand why ADHD diagnoses and treatments are increasingly common.

One possibility is that broadened diagnostic criteria may have led to increased recognition of ADHD. Under DSM-5 criteria, published in 2013, ADHD symptoms are required to be present before age 12, compared with age 7 in prior criteria. However, a study of a British cohort reported that applying less stringent age criteria only led to a 0.1% increase in the prevalence of screening diagnoses of ADHD (Polanczyk et al., 2010). Alternatively, the prevalence of ADHD may be genuinely increasing due to secular environmental changes, including increases in existing exposures or the emergence of new exposures. This could include certain environmental factors that are commonly reported in the media, albeit with weak empirical support, such as screen time (Eirich et al., 2022). An additional example with more empirical support is paternal age. Paternal age is associated with ADHD (D'Onofrio et al., 2014), and we recently observed that the mean paternal age has increased over time in Sweden- (Taylor et al., 2020).

Twin studies can test the hypothesis that environmental factors underlying ADHD have changed over time, as they allow the genetic and environmental variance in a trait to be estimated for different groups, such as individuals born in different years. A change in the environment could manifest as an increase in the environmental variance underlying a trait. Assuming that genetic variance in a trait is stable over relatively short periods of time, this would then manifest as a decrease in the heritability of a trait. To illustrate, a twin study examining

Conflict of interest statement: See Acknowledgements for full disclosures.

whether the relative contribution of genetic and environmental factors to autism and autistic traits had changed over time found that the heritability of these phenotypes was relatively stable over time, despite the increased prevalence of autism (Taylor et al., 2020). On the contrary, a Finnish study of alcohol consumption and abstinence found an increase in heritability over time (Virtanen, Kaprio, Viken, Rose, & Latvala, 2019). Twin studies of ADHD and traits of ADHD yield heritability estimates over 70% (Faraone & Larsson, 2019; Greven, Rijsdijk, & Plomin, 2011; Larsson, Chang, D'Onofrio, & Lichtenstein, 2014), indicating that environmental factors may contribute to ADHD. To our knowledge, no prior twin study has investigated whether these etiological influences on ADHD have changed over time.

We therefore aimed to test for changes in the genetic and environmental variance underlying ADHD over time. We included clinical and subthreshold definitions of ADHD, as there is evidence of genetic links between ADHD and traits of ADHD (Larsson, Anckarsater, Råstam, Chang, & Lichtenstein, 2012; Taylor et al., 2019), while traits of ADHD are also less likely to be influenced by changes in diagnostic practice over time. We assessed whether the genetic and environmental variance in traits and screening diagnoses of ADHD changed over a 16-year period. We then tested whether the heritability of clinical ADHD changed over a 26-year period. If there have been changes in the environmental factors underlying ADHD over time, then we would expect to observe increases in the environmental variance underlying ADHD traits and screening diagnoses and a decrease in the heritability of ADHD.

Methods

Study population

The families of all twins born in Sweden since 1992 are invited to participate in the *Child and Adolescent Twin Study in Sweden* (CATSS) when the twins turn age nine (earlier cohorts included 12-year-olds; Anckarsäter et al., 2011), with a current response rate of 70.2%. CATSS includes twins born since 1992, and recruitment is annual, hence we included twins born between 1992 and 2008. Participants were divided into four birth cohorts: 1992–1995, 1996–1999, 2000–2003, and 2004–2008.

Families who agree to participate in CATSS are also registered in the *Swedish Twin Registry* (*STR*; Zagai, Lichtenstein, Pedersen, & Magnusson, 2019). The STR is a larger register that includes twins who have participated in various twin studies since 1958, covering twins born as long ago as 1886. We linked the STR with the Medical Birth Register (MBR) and identified twins born between 1982 and 2008, meaning that twins were aged 5–31 at the end of follow-up in 2013. Additional opposite-sex twins were identified from the MBR, defined as individuals of the opposite-sex born on the same day to the same parents. STR participants were divided into five birth cohorts: 1982–1991, 1992–1995, 1996–1999, 2000–2003, and 2004–2008. These cohorts have previously been found to be representative of the Swedish population on various characteristics (Taylor et al., 2020).

Variables

ADHD Traits and Screening Diagnoses of ADHD in CATSS were measured using the 19-item ADHD subscale of the Autism-Tics, ADHD, and other comorbidities inventory (A-TAC), a structured parental telephone interview (Hansson et al., 2005). 10 items cover hyperactivity/impulsivity and nine cover inattention. Scores range from 0 to 19. The A-TAC was used as a continuous measure of ADHD traits and to identify individuals with a screening diagnoses of ADHD. Scores over six denote broad ADHD, with sensitivity of 0.64 and specificity of 0.78, and a score of 12.5 denoted strict ADHD, with sensitivity of 0.20 and specificity of 0.97 (Larson et al., 2013). An additional intermediate cutoff of eight was also used. We used these cutoffs to create an ordinal variable.

Clinical ADHD in the STR was defined as either having a diagnosis of ADHD or a prescription of ADHD medication, consistent with prior publications based on Swedish registers (Larsson et al., 2013). Diagnoses of ADHD were identified from the National Patient Register (NPR), which records specialist inpatient and outpatient care in Sweden (Ludvigsson et al., 2011). ADHD was defined as ICD-9 code 314 (hyperkinetic disorder of childhood) or ICD-10 F90 (hyperkinetic disorder). Prescriptions of ADHD medication were extracted from the Prescribed Drug Register, which records dispensations of prescribed medication since July 2005 (Wettermark et al., 2007). Medications included amphetamine, dexamphentamine, methylphenidate, atomoxetine, lisdexamphetamine, and guanphacine.

Statistical analyses

The classical twin design assumes that variation in a trait comprises genetic and environmental components, which are estimated based on comparing within-pair phenotypic resemblance across monozygotic (MZ) and dizygotic (DZ) twins. MZ twins share all of their segregating DNA code as compared to 50% on average in DZ twins, meaning that greater MZ phenotypic resemblance compared to DZ resemblance indicates genetic influences on a trait. Twin models estimate the additive genetic (A), nonadditive genetic (D), shared environmental (C), and nonshared environmental (E) variance underlying a trait; these variance components can be used to calculate the proportion of total phenotypic variance that can be explained by each component. C and D confound one another in the classical twin design, and so only one of these components was estimated. To increase power, we did not estimate both A and D, and fitted ACE models to calculate broad heritability estimates. A comprehensive description of the twin design is given elsewhere (Rijsdijk & Sham, 2002).

ADHD traits. We fitted a univariate ACE model to ADHD traits and estimated A, C, and E variance components, which were summed to give the total variance. We fitted *heterogeneity models* that allowed these components and the mean to differ in each birth cohort. We fitted nested models to test the statistical significance of any differences across cohorts. First, we equated means across birth cohorts, then variances, and finally all parameters. The fit of each nested model was compared to the heterogeneity model using the likelihood-ratio test.

Screening diagnoses of ADHD. We tested whether the variance underlying screening diagnoses of ADHD in CATSS changed over time using an ordinal liability threshold model. This model assumes that a continuous distribution of liability underlies categorical variances and estimates genetic and environmental contributions to the variance in this liability. Thresholds are also estimated, which are *z*-scores corresponding to the proportion of the liability distribution that comprises

affected individuals (presented here as proportions for ease of interpretation). The total variance is typically constrained to 1 in these models to enable model identification; however, it can be freely estimated for ordinal variables if at least one threshold is fixed. We followed this approach. The statistical significance of any changes in the variance underlying screening diagnoses of ADHD over time was assessed as described for traits of ADHD, although thresholds, as opposed to means, were constrained to be equal over time.

Clinical ADHD. We tested whether the heritability of clinical ADHD in the STR changed over time using a liability threshold model for binary data. Only the proportion of variance in liability explained by genetic and environmental influences can be estimated here, as the total variance has to be constrained to 1 to enable model identification. The statistical significance of any changes in the heritability of ADHD was ascertained as described above.

Additional and sensitivity analyses. As there is evidence that hyperactivity/impulsivity and inattention are partly etiologically distinct (Greven et al., 2011), we repeated our analyses of ADHD traits in CATSS for these domains separately. We also conducted sex-specific analyses due to the sex-biased prevalence of ADHD. We then conducted three sensitivity analyses. First, we assessed whether our results were influenced by the inclusion of ADHD medication by restricting our definition of clinical ADHD to diagnoses of ADHD. Second, we excluded individuals with an inpatient diagnosis of ADHD; ADHD is rarely diagnosed in inpatient settings and hence such individuals could present with a rarer clinical profile. Third, we restricted individuals with ADHD to those diagnosed prior to age 10 to increase comparability with the timing of the CATSS assessments.

Results

Sample sizes and descriptive statistics are shown in Table 1 and separately for each birth year in Table S1. Sex-specific descriptive statistics are shown in Table S2. Univariate twin analyses are shown in Tables S3–S6.

Analyses of ADHD traits and screening diagnoses

ADHD traits. The twin correlations, split by birth cohort, for ADHD traits are shown in Figure 1A and Table S7. MZ correlations ranged from 0.65 to 0.68 and were higher than the DZ correlations in all cohorts, suggesting genetic influences on the variance in ADHD traits. The estimates from the twin model are shown in Figure 2A and Table S8. Fit statistics are in Table 2. There was a statistically significant, small increase in the mean score over time, increasing from a standardized mean of −0.05 (95% CI: −0.08/−0.03) among individuals born from 1992 to 1995 to 0.18 (0.15–0.20) in those born from 2004 to 2008. The total variance increased from 0.98

Table 1 Descriptive statistics by birth cohort

	Overall	1982–1991	1992–1995	1996–1999	2000–2003	2004–2008
CATSS						
N	30,072	–	7,328	7,608	7,410	7,726
Sex & Zygosity						
MZF	4,698 (15.6)	–	1,218 (16.6)	1,060 (13.9)	1,048 (14.1)	1,372 (17.8)
DZF	4,924 (16.4)	–	1,168 (15.9)	1,318 (17.3)	1,232 (16.6)	1,206 (15.6)
MZM	4,332 (14.4)	–	1,114 (15.2)	1,004 (13.2)	952 (12.8)	1,262 (16.3)
DZM	5,686 (18.9)	–	1,454 (19.8)	1,482 (19.5)	1,420 (19.2)	1,330 (17.2)
DZOS	10,432 (34.7)	–	2,374 (32.4)	2,744 (36.1)	2,758 (37.2)	2,556 (33.1)
A-TAC Continuous Scale descriptives						
A-TAC ADHD Mean (*SD*)	2.09 (3.15)	–	1.93 (2.98)	1.89 (2.96)	1.95 (3.04)	2.57 (3.53)
A-TAC hyperactivity/impulsivity mean (*SD*)	1.01 (1.71)	–	0.87 (1.58)	0.95 (1.64)	0.94 (1.65)	1.27 (1.92)
A-TAC inattention mean	1.08 (1.78)	–	1.06 (1.75)	0.94 (1.64)	1.01 (1.73)	1.30 (1.94)
Diagnostic frequencies						
Screening ADHD						
Unaffected	26,686 (89.0)	–	6,585 (90.3)	6,849 (90.3)	6,655 (90.1)	6,597 (85.5)
Broad ADHD	1,326 (4.4)	–	306 (4.2)	340 (4.5)	279 (3.8)	401 (5.2)
Intermediate ADHD	1,321 (4.4)	–	275 (3.8)	262 (3.5)	303 (4.1)	481 (6.2)
Strict ADHD	651 (2.2)	–	129 (1.8)	134 (1.8)	149 (2.0)	239 (3.1)
STR						
N	45,356	11,868	9,038	8,580	8,662	7,208
Sex & Zygosity						
MZF	6,181 (13.6)	2,200 (18.5)	1,309 (14.5)	1,042 (12.1)	1,026 (11.8)	604 (8.4)
DZF	5,660 (12.5)	1,286 (10.8)	1,234 (13.7)	1,286 (15.0)	1,218 (14.1)	636 (8.8)
MZM	5,209 (11.5)	1,484 (12.5)	1,187 (13.1)	976 (11.4)	936 (10.8)	626 (8.7)
DZM	5,920 (13.1)	932 (7.9)	1,480 (16.4)	1,440 (16.8)	1,410 (16.3)	658 (9.1)
DZOS	22,386 (49.4)	5,966 (50.3)	3,828 (42.4)	3,836 (44.7)	4,072 (47.0)	4,684 (65.0)
ADHD clinical diagnosis	1,307 (2.9)	227 (1.9)	338 (3.7)	342 (4.0)	289 (3.3)	111 (1.5)
ADHD medication	1,497 (3.3)	218 (1.8)	348 (3.9)	408 (4.8)	372 (4.3)	151 (2.1)
ADHD diagnosis and/or medication	1,729 (3.8)	286 (2.4)	406 (4.5)	456 (5.3)	407 (4.7)	174 (2.4)

A-TAC: Autism-Tics, ADHD, and other Comorbidities inventory; CATSS: Child and Adolescent Twin Study in Sweden; DZF: dizygotic female; DZM: dizygotic male; DZOS: dizygotic opposite sex; MZF: monozygotic female; MZM: monozygotic male; STR: Swedish Twin Registry.

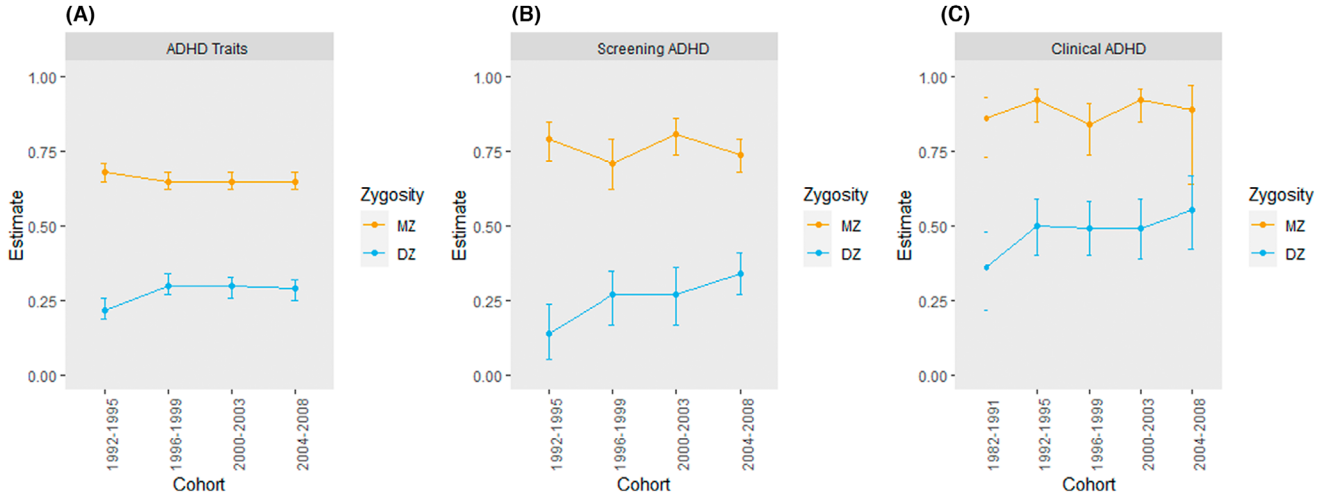

Figure 1 Twin correlations over time. ADHD Traits: total score on the Autism-Tics, ADHD, and other Comorbidities (A-TAC) inventory ADHD subscale. Screening ADHD: ADHD as identified by three screening cutoffs on the A-TAC. Clinical ADHD: diagnosis of ADHD recorded in the NPR and/or prescription of ADHD medication recorded in the Prescribed Drug Register. MZ: monozygotic twins; DZ: dizygotic twins

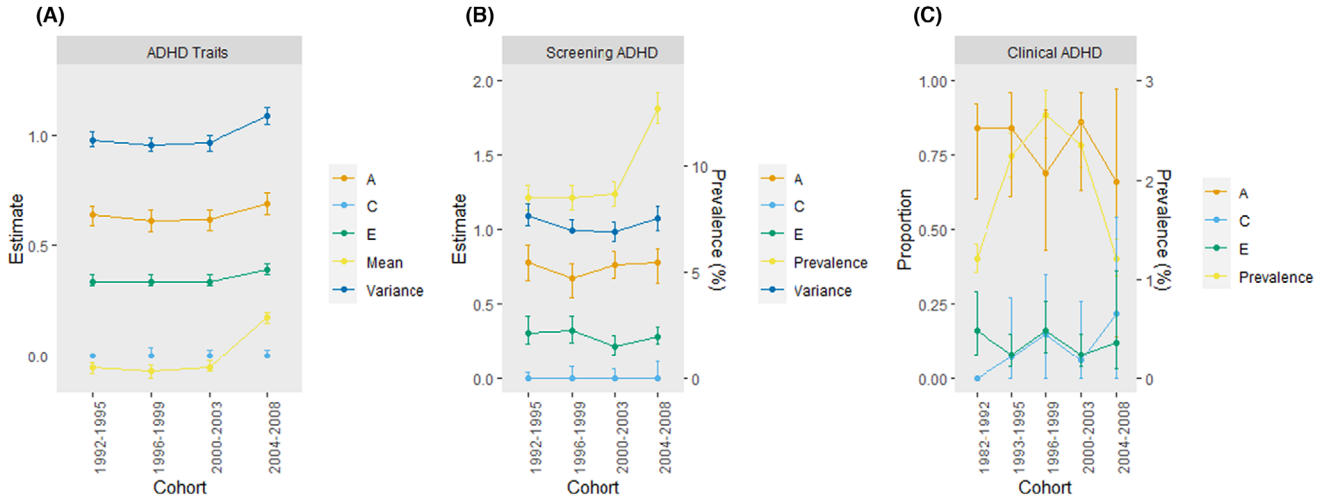

Figure 2 Twin model estimates over time. A: additive genetic variance; C: shared environmental variance; E: nonshared environmental variance. ADHD Traits: total score on the Autism-Tics, ADHD, and other Comorbidities (A-TAC) inventory ADHD subscale. Screening ADHD: ADHD as identified by three screening cutoffs on the A-TAC. Clinical ADHD: diagnosis of ADHD recorded in the NPR and/or prescription of ADHD medication recorded in the Prescribed Drug Register. For ADHD traits and screening ADHD, estimates are given as raw variance components. Means are standardized. For clinical ADHD, estimates are given as the proportion of liability to ADHD explained by each component

(0.95–1.02) to 1.09 (1.05–1.13) over time. These estimates could not be equated without statistically significantly worsening model fit. The variance increase was driven by small increases in both A, from 0.64 (0.59–0.68) to 0.69 (0.64–0.74), and E, from 0.34 (0.32–0.37) to 0.39 (0.37–0.42). C was 0 in all birth cohorts. Since both A and E increase, their relative contributions to the variance in ADHD traits were stable over time, ranging from 64% to 65% for A and 35% to 36% for E.

As the twin correlations did indicate the presence of nonadditive genetic influences (i.e. D), we additionally fitted an ADE model, in which we also modeled sibling contrast effects (i.e. a causal pathway whereby one twin's phenotype directly influences their co-twin's phenotype). In these models, additive genetic influences explained 67%–76% of the variance in traits of ADHD across cohorts, while D was estimated as 0, with the apparent effect of D in the twin correlations likely due to sibling contrast effects. One exception, however, was the cohort born from 2000 to 2003, whereby additive genetic influences explained 48% of the variance and nonadditive genetic influences explained 16% of the variance. However, confidence intervals were wide for both components and included 0, hence caution is needed in interpreting result. The estimates are all provided in Table S9.

Table 2 Twin model fit statistics

Model	−2LL	Parameters	*df*	AIC	Comparison Model	$\Delta\chi^2$	Δ*df*	*p*
ADHD traits								
Fully saturated	81,275.71	40	29,939	21,397.71	–	–	–	–
ACE	81,484.55	16	29,963	21,558.55	Fully Saturated	208.84	24	<.001
Equal means	81,688.55	13	29,966	21,756.55	ACE	204.00	3	<.001
Equal variance	81,526.17	7	29,972	21,582.17	ACE	41.62	9	<.001
Homogeneity	81,743.22	4	29,975	21,793.22	ACE	258.66	12	<.001
Screening ADHD								
Fully saturated	26,545.74	56	29,928	−33,310.26	–	–	–	–
ACE	26,783.83	20	29,964	−33,144.17	Fully Saturated	238.09	36	<.001
Equal thresholds	26,818.18	14	29,970	−33,121.82	ACE	34.35	6	<.001
Equal variance	26,794.49	11	29,973	−33,151.51	ACE	10.66	9	.300
Homogeneity	26,910.34	5	29,979	−33,047.66	ACE	126.51	15	<.001
Clinical ADHD								
Fully saturated	13,549.21	30	45,326	−77,102.79	–	–	–	–
ACE	13,706.50	20	45,346	−76,985.50	Fully Saturated	157.30	20	<.001
Equal thresholds	13,852.02	16	45,350	−76,847.98	ACE	145.52	4	<.001
Equal variance components	13,714.73	8	45,358	−77,001.27	ACE	8.23	12	.767
Homogeneity	13,862.20	4	45,362	−76,861.80	ACE	155.70	16	<.001

−2LL: −2*log-likelihood of the data; *df*: degrees of freedom; AIC: Akaike's information criteria (smaller values indicate better fitting models); $\Delta\chi^2$: −2LL difference between two models, distributed χ^2; Δ*df*: difference in degrees of freedom between models.
Fully Saturated: baseline model of the observed data; ACE: twin model estimating genetic and environmental contributions to the total variance/liability; Homogeneity: model with all parameters constrained to be equal over time.

Screening diagnoses of ADHD. The twin correlations are shown in Figure 1B and Table S7. MZ correlations ranged from 0.71 to 0.81, and were all higher than the DZ correlations, which varied from 0.14 to 0.34. The prevalence of screening diagnoses of ADHD increased over time, from 9.8% of individuals born from 1992 to 1995 to 14.5% of individuals born from 2004 to 2008 (Figure 2B). As also shown in Figure 2B, the variance in these diagnoses was stable over time and could be equated across cohorts. Genetic variance increased from 0.67 to 0.78 over time, while E increased from 0.22 to 0.32. The heritability of screening diagnoses thus ranged from 68% to 78%, albeit these fluctuations in variance over time were not statistically significant.

Analyses of clinical ADHD

As shown in Figure 1C and Table S10, twin correlations were all higher for MZ than DZ twins, ranging from 0.84 to 0.92 for MZ twins and 0.36–0.55 for DZ twins. Figure 2C and Table S11 show the twin model estimates. An increasing proportion of individuals born from 1982 to 1999 had clinical ADHD, with the proportion decreasing in individuals born after 2000. This is likely due to censoring of the follow-up of these individuals. The heritability of ADHD was consistently high: 0.84 (0.60–0.92) for individuals born 1982–1991, 0.84 (0.61–0.96) for those born 1992–1995, 0.69 (0.43–0.90) for those born 1996–1999, 0.86 (0.63–0.96) for those born 2000–2003, and 0.66 (0.14–0.97) for individuals born 2004–2008. A statistically nonsignificant shared environmental component of 22% was estimated for individuals born 2004–2008. As shown in Table 2, these fluctuations were not statistically significant.

Additional and sensitivity analyses

Dimension- and sex-specific analyses. The results of these additional analyses are shown in Figure 3 and Tables S12–S14. The total variance in both subscales increased, from 0.93 (0.90–0.96) to 1.16 (1.12–1.20) for hyperactivity/impulsivity and from 1.01 (0.98–1.05) to 1.11 (1.07–1.15) for inattention. These increases were driven by small increases in both the genetic and environmental variance. The total variance in ADHD traits also showed an increase in females, from 0.85 (0.80–0.91) to 1.02 (0.96–1.08), and in males, from 1.09 (1.03–1.16) to 1.18 (1.11–1.26). These increases were also driven by small increases in both the genetic and environmental variance. Thus, the increasing variance in ADHD traits over time was not driven by specific ADHD traits or one sex in particular.

Sensitivity analyses. Results of the sensitivity analyses are in Tables S15 and S16. The main results did not change, meaning that our results were not driven by the inclusion of ADHD medication, inpatient diagnoses, or diagnoses before age 9.

Discussion

In this study, we investigated whether the degree to which genetic and environmental factors contribute to traits of ADHD and ADHD diagnoses has changed over time. Although this study did not focus on specific environmental exposures or include genotype data, these results do not support the

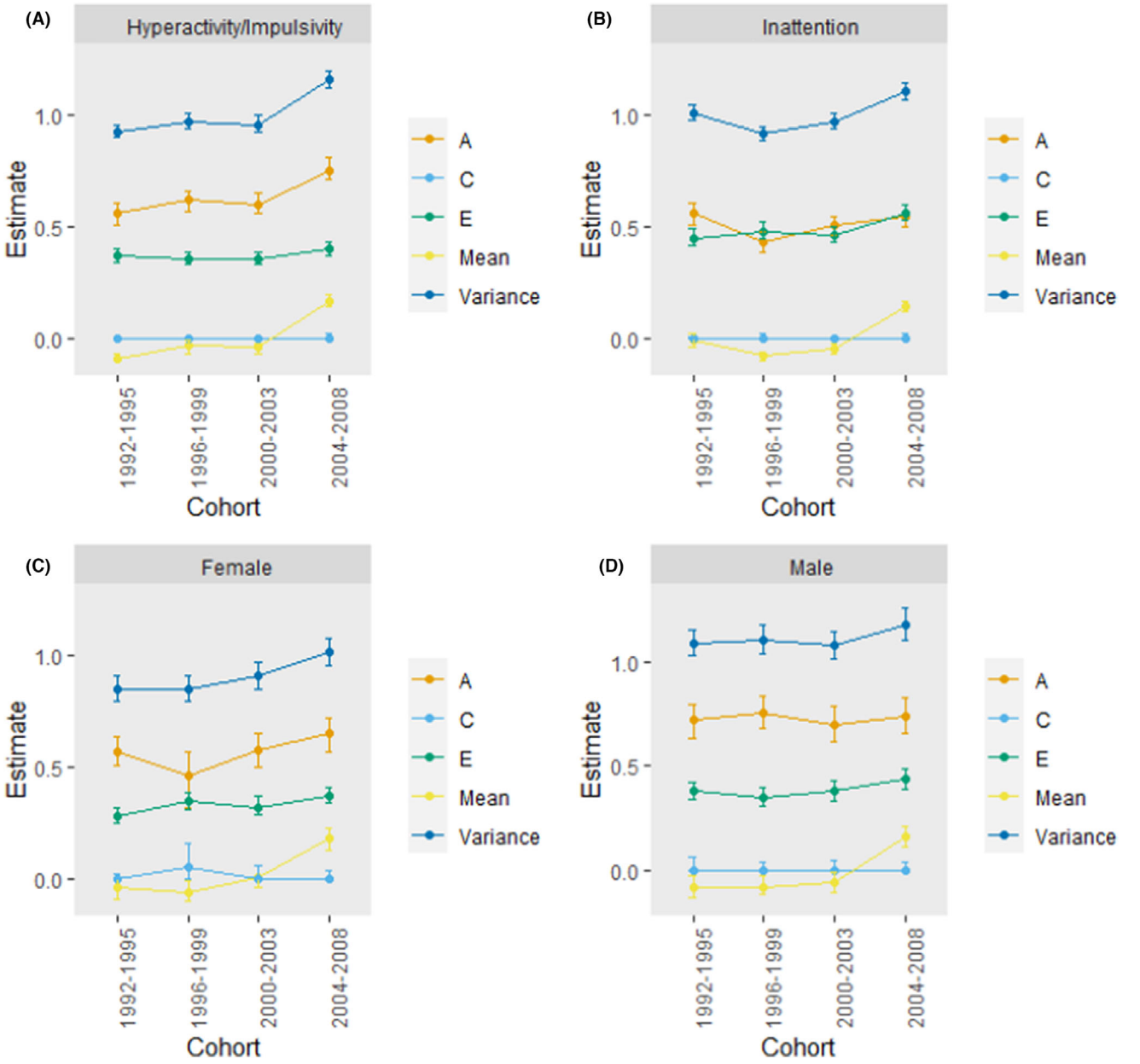

Figure 3 Subscale- and sex-specific results. A: additive genetic variance; C: shared environmental variance; E: nonshared environmental variance. ADHD Traits: total score on the Autism-Tics, ADHD, and other Comorbidities (A-TAC) inventory ADHD subscale. Screening ADHD: ADHD as identified by three screening cutoffs on the A-TAC. Clinical ADHD: diagnosis of ADHD recorded in the NPR and/or prescription of ADHD medication recorded in the Prescribed Drug Register. Sex-specific results are shown for the total A-TAC ADHD scale, rather than for specific subscales. All estimates are the raw variance components

hypothesis that the increased prevalence of ADHD is due to changes in the environment, as such changes would be expected to increase the degree to which environmental factors contribute to ADHD. Rather, these results indicate that the increasing prevalence of ADHD is explained by other factors, such as increased diagnosis of a strongly heritable condition.

Several additional analyses added robustness to our results in showing that a larger change in the variance in ADHD was not masked by grouping different sexes or traits together. We repeated our analyses for core ADHD subdimensions separately due to evidence that these domains are, to a degree, etiologically distinct (Greven et al., 2011). There is evidence that the skewed sex ratio observed in ADHD prevalence has decreased over time (Huang, Chu, Cheng, & Weng, 2014), and so we performed sex-specific analyses. However, all of these analyses yielded similarly small increases in the variance underlying ADHD to our main analyses, thus adding further support to the result that the environmental variance in ADHD has remained stable over time.

Our results are instead in line with prior twin studies of ADHD, and further affirm that ADHD, and its related traits, are highly heritable (Greven et al., 2011; Larsson et al., 2012, 2014). Nonetheless, our heritability estimates were not 100%, meaning that environmental contributions to

ADHD cannot be ruled out. Many prior studies of environmental factors linked with ADHD have focused on early life exposures (Carlsson, Molander, Taylor, Jonsson, & Bölte, 2021). Unmeasured familial confounding (i.e. an association arising from shared familial causes between an exposure and an outcome) appears to account for the association between some of these factors and ADHD, such as maternal psychosocial adversity (Rosenqvist, Sjölander, Ystrom, Larsson, & Reichborn-Kjennerud, 2019). Such shared genetic causes may be captured in the heritability estimates for ADHD in a twin model, meaning that an increase in such factors could serve to increase the genetic variance in ADHD over time. Furthermore, the association between some environmental exposures and ADHD has been shown to persist even after adjusting for familial confounding, including low birth weight (Pettersson et al., 2015) and advanced paternal age (D'Onofrio et al., 2014). Our results thus allow for these factors to contribute to ADHD, but the weak variance increases over time suggest that they are unlikely to explain why ADHD diagnoses have increased.

We also observed that the mean level of ADHD increased over time, which may appear to counter the above arguments. Similar patterns were observed in prior CATSS papers on ADHD traits (Rydell, Lundström, Gillberg, Lichtenstein, & Larsson, 2018), as well as autistic traits (Taylor et al., 2020). These increases were all small, however; the observed mean ADHD trait score increased by less than half a unit (i.e. less than 0.5, with a possible range of responses from 0 to 19) between individuals born from 1992 to 1995 and those born from 2004 to 2008. An individual born more recently would thus not be expected to show a drastically increased number of ADHD traits compared with an individual born in the early 19,902, with such an increase likely to be statistically significant due to our large sample size.

Taken together with existing evidence that the level of ADHD traits has remained consistent over time in the general population (Rydell et al., 2018), our results indicate that the prevalence of ADHD has not increased. While one study reported that broadening diagnostic criteria had a minimal impact on the prevalence of ADHD (Polanczyk et al., 2010), a meta-analysis reported that accounting for assessment methods attenuated differences in ADHD prevalence across countries (Polanczyk, Willcutt, Salum, Kieling, & Rohde, 2014). This suggests that increasingly sensitive tools may be being used to diagnose ADHD. It is also possible that ADHD is increasingly recognized by parents, teachers, and clinicians, all of which may have contributed to its apparent increase in prevalence. For example, children's access to clinical support is contingent on parents and/or teachers recognizing their difficulties. If parents and teachers have become more aware of ADHD, then this may increase the likelihood of a child coming to clinical attention. ADHD is also increasingly recognized in adults, meaning that increased diagnosis of ADHD in adults may contribute to its observed prevalence (Bachmann et al., 2017). Adults and parents of children with ADHD may thus have become more willing to seek a diagnosis of ADHD. Finally, individuals with milder ADHD traits may be more likely to receive a diagnosis in contemporary clinical practice (Kazda, Bell, Thomas, McGeechan, & Barratt, 2019). While our study did not test any of the possibilities discussed here, the lack of evidence for etiological changes in ADHD over time indicates that these alternative explanations for the apparent increase in ADHD should be studied further.

The main strengths of this study are the large twin cohorts, with registry linkage, which allowed us to study ADHD over a period of almost 30 years. We also focused on multiple definitions of ADHD. However, there is a need to assess whether our findings generalize to countries outside Sweden, since environmental factors may have changed more profoundly outside Sweden. It would also be beneficial for future studies to focus on specific exposures associated with ADHD to assess whether their association with ADHD has changed over time. Such an approach would also make it possible to examine whether new exposures associated with ADHD have arisen over time. Finally, we only studied ADHD traits and screening diagnoses in children. Future studies should assess whether similar results are observed in adults.

Conclusions

Overall, we did not find evidence that the etiology of ADHD has changed over time. The environmental variance underlying ADHD showed only a very weak increase, which is unlikely to account for an increase in ADHD over time, thus speaking against the notion of secular changes in the environment leading to a genuine increase in the prevalence of ADHD. We suggest that future research would be best targeted toward better understanding how changes in assessments and recognition of ADHD across different developmental periods have impacted on the reported prevalence of ADHD.

Supporting information

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

Table S1. Descriptive statistics for each birth year in CATSS.
Table S2. Descriptive statistics by sex for each birth cohort.
Table S3. Twin correlations.
Table S4. Assumptions testing.

Table S5. Univariate twin model fit statistics.
Table S6. Univariate estimates.
Table S7. Twin correlations split by birth cohort.
Table S8. Twin model estimates by birth cohort.
Table S9. Estimates from ADE-s model of ADHD traits.
Table S10. Twin correlations split by birth cohort.
Table S11. Twin model estimates split by birth cohort.
Table S12. Subscale- and sex-specific twin correlations over time.
Table S13. Twin model fit statistics for subscale- and sex-specific analyses.
Table S14. Estimates for subscale- and sex-specific analyses.
Table S15. Twin model fit statistics for sensitivity analyses.
Table S16. Twin model estimates for sensitivity analyses.

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Data availability statement

We are unable to share data publicly due to Swedish laws around privacy. Researchers interested in accessing data from the Swedish Twin Registry may submit an application to do so, subject to approval from the STR steering committee and relevant ethical approvals. Full details can be found on the STR website: https://ki.se/en/research/the-swedish-twin-registry.

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Key points

- Attention-deficit/hyperactivity disorder (ADHD) is an increasingly commonly diagnosed neurodevelopmental condition, with a strong increase in the recorded prevalence over the past two decades.
- Here we tested whether the degree to which genetic and environmental factors contribute to ADHD, and subthreshold traits of ADHD, has changed over time.
- Overall, the results do not suggest that the relative importance of genetic and environmental factors for ADHD has changed over time. It is therefore unlikely that drastic changes in the environment can explain why the prevalence of ADHD has increased so sharply.

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