Subjective Gender-Based Patterns in ADHD Diagnosis*

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Abstract

The increasing incidence rate of ADHD diagnosis has sparked debates about over-medicalization and misdiagnosis. We use data on individuals' genetic predisposition to ADHD from the Add Health survey on U.S. schools to uncover relative standards in ADHD diagnosis. We estimate that students' ordinal rank in the genetic predisposition to ADHD among their same-gender grademates has a positive, statistically significant, and substantial causal effect on ADHD diagnosis, holding students' own genetic predisposition to ADHD constant. This effect is mainly driven by boys, contributing to explain the observed higher rate of diagnosis of boys relative to girls for a given genetic ADHD predisposition.

JEL: I10, I21, J24, J13.

Keywords: mental health; ADHD; gender; ADHD polygenic scores; interpersonal comparisons; subjective diagnosis.

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1 Introduction

In 2021, the U.S. Surgeon General issued an advisory identifying a mental health crisis among children and adolescents,[1] and the American Academy of Pediatrics, the American Academy of Child and Adolescent Psychiatry, and the Children's Hospital Association jointly declared a National Emergency in Children's Mental Health.[2] The pandemic has exacerbated mental health challenges, but it is important to recognize that the situation prior to Covid-19 was already very troubling. Even before the pandemic, mental disorders were increasing among children and adolescents, reinforcing their role as the primary catalyst for health-related disability and adverse life outcomes in young people (Kieling et al., 2011; Perou et al., 2013).[3]

Attention-deficit/hyperactivity disorder (ADHD), the focus of this paper, is currently the most frequently diagnosed mental health condition, along with anxiety, in American children and adolescents aged 3-17 years. Nearly 10% of U.S. children have been diagnosed with ADHD by a health care provider, according to recent data for 2016-19 and 2017-18 from the National Survey of Children's Health and the National Health Interview Survey, respectively (Bitsko et al., 2022).

ADHD is a neurodevelopmental disorder characterized by symptoms of inattention, hyperactivity, and impulsivity (APA, 2013; Wolraich et al., 2019). Symptoms of ADHD begin in childhood, when it is usually first diagnosed, and often persist into adulthood. ADHD has been shown to be negatively correlated with human capital accumulation (Currie and Stabile, 2006; Fletcher and Wolfe, 2008), and adult labor market outcomes (Fletcher, 2014), and positively correlated with welfare use (Currie et al., 2010), and criminal activity (Fletcher and Wolfe, 2009), as well as with a wide range of comorbidities and mortality.[4]

[1]The full public statement is available at: http://bitly.ws/IFW6.

[2]The declaration is available at http://bitly.ws/IFZv.

[3]The youth mental health crisis is not limited to the U.S.: according to 2019 statistics collected by UNICEF, suicide was the second leading cause of death among young people in Europe, where only traffic injuries claim more lives of 15-19 year olds (Keeley, 2021).

[4]See for example Faraone et al. (2015); Scott et al. (2017); Sun et al. (2019); Dalsgaard et al. (2015). There is also evidence that children's ADHD reduces parents' socioeconomic status

The prevalence of ADHD varies widely both between and within countries (Charach et al., 2011). ADHD prevalence is generally found to be higher in the U.S. than in Canada and European countries (Charach et al., 2011; Thomas et al., 2015). Within the U.S., there is significant variation among regions as well as by gender and income.[5] In addition, the prevalence of ADHD in the U.S. has been increasing since the late 1990s,[6] along with the prescription of medications to treat the disorder.[7] The effects of ADHD treatment have also been a cause for concern, as research suggests that ADHD medications may not always be beneficial for children in the medium and long term.[8] The variation in the estimated ADHD prevalence within and between countries, together with the upward trends in ADHD diagnosis and treatment, have sparked heated debates about the adequacy of diagnostic and treatment protocols for ADHD. Concerns also stem from the fact that no biological marker is currently diagnostic for ADHD (APA, 2013).[9] As

by lowering their labor supply (and earnings) and reducing relationship stability (Kvist et al., 2013). Erskine et al. (2016) present a review and meta-analysis of the adverse health and psychosocial outcomes associated with ADHD.

[5]ADHD diagnosis rates are higher among boys and children from poorer families (Bitsko et al., 2022; Visser et al., 2014; Akinbami et al., 2011; Xu et al., 2018)

[6]See for example Akinbami et al. (2011); Perou et al. (2013); Visser et al. (2014); Xu et al. (2018).

[7]See for example Girand et al. (2020); Raman et al. (2018); Piper et al. (2018); Bachmann et al. (2017); Visser et al. (2014). A similar trend has been noted worldwide (Dalsgaard et al., 2013).

[8]For example, Currie et al. (2014) show that a large increase in the use of ADHD medications induced by an expansion of prescription drug coverage in Quebec had some negative effects on children both in the medium and long term, some of which are consistent with possible side effects of stimulant medications commonly prescribed for ADHD, particularly depression. In addition, they also uncover a deterioration in important academic outcomes, including grade repetition and math scores. Dalsgaard et al. (2014b) use data from a Danish nationwide cohort study and find that the occurrence of cardiovascular events, while rare, was twice as likely in ADHD stimulant users as in non-users, both in the total national population and in children with ADHD.

[9]A biomarker can be defined as "almost any measurement reflecting an interaction between a biological system and a potential hazard, which may be chemical, physical, or bi-

a result, medical diagnosis of ADHD is usually based on observation of patients and subjective third-party reports from parents and teachers –as is often the case with adolescent mental health diagnoses– which can lead to misdiagnoses.

This issue has garnered more attention due to an increasing body of evidence suggesting that children who are relatively young for their grade level are more likely to be diagnosed and treated than their older peers.[10] Given that ADHD is an underlying neurological problem, its prevalence should not be altered by small variations in the age of children within their grade caused by discontinuities in school entry cut-off dates. Therefore, this evidence is indicative of subjective standards in ADHD diagnosis based on interpersonal comparisons. Understanding the factors that may lead to ADHD misdiagnosis is important because inaccurate diagnoses and the subsequent treatment with ADHD medication may have adverse effecs on health and human capital accumulation,[11] on top of imposing a substantial economic burden on patients, health care systems and societies (Schein et al., 2022).

In this paper, we propose a novel way to detect relative standards in ADHD diagnosis by exploiting the availability of genetic data in Add Health, a longitudinal school-based survey in the United States. We rank students' genetic susceptibil-

ological. The measured response may be functional and physiological, biochemical at the cellular level, or a molecular interaction" (WHO, 1993). Biomarkers encompass a wide range of indicators, from simple measurements such as pulse and blood pressure to basic chemical analyses and complex laboratory tests of blood and various tissues (Strimbu and Tavel, 2010).

[10]This finding has been widely replicated in many countries, such as the U.S. (Elder, 2010; Evans et al., 2010; Layton et al., 2018), Canada (Morrow et al., 2012), Germany (Schwandt and Wuppermann, 2016), the Netherlands (Krabbe et al., 2014), Iceland (Zoëga et al., 2012), Sweden (Halldner et al., 2014; Persson et al., 2021), Taiwan (Chen et al., 2016), and the United Kingdom (Root et al., 2019; Fleming et al., 2022). Denmark is an exception to this pattern, as Pottegärd et al. (2014) and Dalsgaard et al. (2014a) find no association between children's relative age in class and the use of ADHD medication. Dalsgaard et al. (2012) also find no late birthdate effects on ADHD diagnosis in Denmark. See Whitely et al. (2018) for a review of the literature documenting the effect of relative age for grade on ADHD diagnosis and treatment.

[11]See for instance Currie et al. (2014), Dalsgaard et al. (2014b), Ibrahim and Donyai (2015), Carucci et al. (2021) and the references therein.

ity to ADHD (as measured by the ADHD Polygenic Score -hereafter ADHD PGS- a summary indicator of individuals' genetic propensity for the disorder) within their school and grade, and we exploit as-if-random variation within schools in the composition of peers across grades to assess whether a student's ordinal rank in the distribution of genetic susceptibility to ADHD in his or her grade affects the likelihood of diagnosis, holding both his or her age and own genetic susceptibility to ADHD constant.

We find robust evidence that relative standards in ADHD diagnosis are relevant and mainly driven by within-gender comparisons. In particular, we find that a one standard deviation increase in students' ADHD PGS rank within gender and grade increases the probability of ADHD diagnosis by 2.5 percentage points, or 42% of the ADHD diagnosis rate. This effect is large, statistically significant, and driven primarily by boys.

While previous work has emphasized the role of relative age at school entry, our findings suggest that interpersonal comparisons matter for ADHD diagnosis even among children of exactly the same age and with exactly the same genetic susceptibility to ADHD.

In addition, our analysis sheds light on the potential sources of the male-female excess gap in ADHD diagnosis. As there are no significant gender differences in individuals' genetic predisposition to ADHD, this gap is unlikely to be explained by genetic endowments. The medical literature emphasizes that ADHD symptoms tend to manifest differently in boys - who tend to exhibit more externalizing behaviors (e.g., symptoms of hyperactivity) - and girls - who tend to exhibit less disruptive behaviors. Our findings that the relevant peers for ADHD-related comparisons are same-gender grademates rather than all grademates is consistent with the medical evidence, and suggests that third-party assessments (presumably by teachers and/or parents) are based on within-gender comparisons of ADHD manifestations, which in turn later translate into a higher likelihood of ADHD diagnosis for boys whose genetic propensity for ADHD is higher than that of their peers in high school.

Our paper contributes to three strands of literature.

First, we contribute to an extensive literature that examines the escalating mental health challenges experienced by children and adolescents.[12] Our study is especially close to an expanding strand of research that analyzes the influence of children's family and school networks on their mental health. For example, Kiessling and Norris (2023) find that increasing students' ordinal ability rank within their school and grade improves their mental health (as measured by a standard scale used to diagnose depression) and that this effect persists from adolescence into adulthood, while Persson et al. (2021) provide evidence of family spillover effects of marginal ADHD diagnoses by showing that age-for-grade-related marginal diagnoses propagate to younger siblings and cousins. Our study adds to this evidence by showing that interpersonal comparisons based on children's school environment matter beyond relative age-for-grade for later ADHD diagnoses, calling for interdisciplinary and coordinated efforts to improve diagnostic protocols.

Second, our work relates to a growing literature that examines the relevance of ordinal rank effects as a specific form of peer effects. Students' ordinal academic rank has been shown to have positive effects on educational attainment (Elsner and Isphording, 2017; Murphy and Weinhardt, 2020; Denning et al., 2021; Elsner et al., 2021; Bertoni and Nisticò, 2023), and wages (Denning et al., 2021), and negative effects on mental health (Elsner and Isphording, 2017; Kiessling and Norris, 2023), and on the likelihood of engaging in risky behaviors and physical fights (Elsner and Isphording, 2018). Moreover, students' ordinal academic rank also affects their choice of subjects in secondary school (Murphy and Weinhardt, 2020), and their choice of specialization at university (Delaney and Devereux, 2021; Elsner et al., 2021; Goulas et al., 2022). However, this is the first paper to examine the consequences of students' ordinal rank in terms of their genetic predisposition to a specific trait, ADHD, which allows us to provide new insights into the drivers of ADHD diagnosis. In addition, we uncover gendered patterns in rank effects, an aspect that, to our knowledge, has received limited attention

[12] See, for example, Kieling et al. (2011); Perou et al. (2013); Gaylor et al. (2023); Keeley (2021) and the references therein.

in this literature.[13]

Finally, we contribute to an emerging body of work exploiting the increasing availability of genetic data in multidisciplinary surveys to study metagenomic effects outside the family, how individuals are affected by the genetic makeup of other individuals (beyond family members) in their social network (Domingue and Belsky, 2017; Sotoudeh et al., 2019; Brunello et al., 2020). By examining the effects of ADHD genetic ordinal rank, we bridge the literatures on ordinal rank and metagenomic effects.

The rest of the paper is organized as follows. Section 2 describes the Add Health database, including the genetic information and the measurement of the relevant variables. Section 3 then explains the identifying variation and the empirical strategy used in the analysis. Section 4 presents the main results of the paper, while Section 5 provides a wide range of robustness checks. Finally, Section 6 concludes the paper.

2 Data

2.1 The Add Health Dataset: Overview and Suitability for our Analysis

We use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative, school-based longitudinal study that began in 1994-1995. The study enrolled 20,745 adolescents in grades 7-12 (age range 12-20) from a stratified sample of 80 high schools and 52 middle schools.[14] Wave I (1994-1995) of Add Health included an in-school questionnaire, administered to all participating students on the day of the interview, that collected information on schools and on students' social and demographic characteristics,

[13] An exception is Delaney and Devereux (2021) who compare the effect of same- and mixed-gendered rank in Math and English on the choice of a STEM major at college in the U.K., but find limited evidence of within-gender comparisons.

[14] The probability of school selection was proportional to school size, and schools were stratified by region, urbanicity, school type, ethnic mix, and size.

including their parental background. In addition, a more detailed in-home interview was conducted with a random sample of approximately 17 males and 17 females within each school and grade, and a parent questionnaire was administered to a parent (usually the resident mother) of each adolescent selected for the in-home sample. The study has followed adolescents from the six Wave I grades in four subsequent waves, including Wave II (1996, age range 12-21, n = 14,738), Wave III (2000-2001, age range 18-27, n = 15,197), Wave IV (2008-2009, age range 24-33, n = 15,701), and most recently Wave V (2016-2018, age range 33-43, n = 12,300).

Our analysis relies primarily on data from Waves I and IV of the Add Health study. Wave I provides us with school and grade identifiers, as well as characteristics of students, their families, and their grademates. Meanwhile, Wave IV provides our outcome variable: whether individuals received a diagnosis of ADHD from a healthcare professional (see Section 2.3.2). Saliva samples for DNA extraction were also collected at Wave IV for the in-home sample.[15] These data are used to measure individuals' genetic predisposition to ADHD, as described in Section 2.4.

In addition, we use several human capital and behavioral indicators mainly from Wave I to provide evidence in Section 2.3.2 that our measure of genetic predisposition to ADHD is correlated with these indicators, as one would expect. In addition to being correlated with later ADHD diagnosis, these indicators are observed by parents and teachers, which may in turn influence their decisions about whether a child needs a medical consultation that may ultimately lead to an ADHD diagnosis.

The Add Health dataset is particularly well suited for our research purposes for several reasons. First, the survey includes a question about ADHD diagnosis, which is our outcome of interest. Second, Add Health is a nationally representative school-based survey that randomly selects students in grades 7-12 from a stratified sample of schools across the United States. This sampling scheme al-

[15] At Wave III of Add Health, DNA was also collected, but only for the full sibling and twin subsamples.

lows us to observe individuals as well as their grade-level peers, thereby allowing us to take advantage of variation across grades within schools, as required by our identification strategy. Third, Add Health provides a polygenic index that serves as a proxy for individuals' genetic predisposition to ADHD, allowing us to rank students' grademates based on their genetic predisposition to ADHD. This is also important for our identification strategy, as genes are fixed at conception and can influence an individual's likelihood of developing ADHD. We will discuss the importance of this factor in Section 3.1.

2.2 Sample Selection and Descriptive Statistics

Our working sample is obtained after applying several selection criteria. Of the 20,745 students surveyed in Wave I, we first retain 18,456 students with valid information on gender, age, race, school and grade identifiers, and sample weights. Next, as information on individuals' ADHD diagnosis and genotype is collected in Wave IV, we further restrict the sample to 14,480 individuals who participated in both Waves I and IV. In addition, we are forced to retain only 8,410 students with valid genetic information. Although Add Health collected saliva samples from 96% of Wave IV participants and 80% consented to the storage of their genetic information, this large reduction in sample size occurs because, after quality control procedures, genotype data were retained for only 9,974 individuals and ADHD PGS information is available for only 9,130 individuals.[16] Finally, because our paper examines gender patterns and our identification relies on variation in individuals' genetic susceptibility to ADHD within schools and across grades, we exclude individuals who belong to school-grade groups with fewer than five students and that do not have at least two boys and two girls, further reducing the number of observations to a final working sample of 8,291 students.

Table 1 displays summary statistics for our outcome variable (Panel A, discussed in the next section) and for the control variables we will use at the individ-

[16]See the Add Health documentation (https://addhealth.cpc.unc.edu/wp-content/uploads/docs/user_guides/AH_GWAS_QC.pdf) for details on genotyping and quality control procedures.

ual (Panel B), family (Panel C), and school-grade level (Panel D). We present data for the full sample and separately for each gender. The parental socioeconomic status index combines information on parental education, parental occupation, household income, household receipt of public assistance, and residential building quality, and is constructed as described in Appendix G of Sanz-de Galdeano and Terskaya (2023).

Table B1 in Appendix B compares a set of individual-level characteristics measured at Wave 1 across the full sample and our estimation sample. The ADHD diagnosis rate, gender, age, nationality, the proportion of students living with both parents, and parental age are comparable between the two samples. However, the final sample has a 7% higher (lower) proportion of White (Hispanic) students, a 0.02SD higher average SES level, and a 0.06SD higher level of the Peabody Picture Vocabulary Test (PPVT), a measure often used as a proxy for academic ability. In Section 5, we thoroughly assess the robustness of our findings to the sample selection criteria we are forced to adopt in order to use the genetic information collected in Wave IV.

2.3 Attention-Deficit/Hyperactivity Disorder (ADHD)

2.3.1 ADHD: Definition and Diagnostic Protocols

Attention-deficit/hyperactivity disorder (ADHD) is one of the most common mental disorders affecting children and adolescents (https://www.cdc.gov/ncbddd/adhd/facts.html). The Diagnostic and Statistical Manual of Mental Disorders (DSM), the manual used by clinicians and researchers to diagnose and classify mental disorders (including ADHD), describes ADHD as a chronic neurodevelopmental disorder characterized by a persistent and pervasive pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development. The American Psychiatric Association published the DSM-V, the 5th edition of the DSM, in 2013 (APA, 2013), but the diagnostic protocols that could be applied to Add Health respondents, given their age, were those of the DSM-IV, the 4th edition of the DSM (APA, 1994). Therefore, we will refer to the DSM-IV

and discuss how it differs from the DSM-V as far as ADHD is concerned (CBHSQ, 2016).

Table 1: Summary Statistics

	All		Males		Females	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Panel A: Outcome and rank variables						
Professional ADHD diagnosis	0.06	0.24	0.08	0.27	0.04	0.20
ADHD PGS rank	0.49	0.31	0.49	0.31	0.49	0.31
ADHD PGS gendered-rank	0.49	0.33	0.49	0.33	0.49	0.33
Panel B: Individual socio-demographic characteristics						
Female	0.49	0.50	0.00	0.00	1.00	0.00
Age	15.91	1.77	16.01	1.77	15.81	1.75
Age^2	256.23	56.39	259.38	57.00	252.95	55.57
Born in the US	0.96	0.19	0.96	0.19	0.96	0.19
White	0.73	0.44	0.73	0.45	0.74	0.44
Black	0.15	0.35	0.15	0.35	0.15	0.35
Hispanic	0.07	0.26	0.08	0.26	0.07	0.26
Panel C: Family and parental characteristics						
Both parents live in hh	0.72	0.45	0.72	0.45	0.72	0.45
Parental age	41.35	6.26	41.45	6.34	41.26	6.18
Socio-economic status	-0.00	1.00	-0.01	1.00	0.01	1.00
Panel D: Grademates characteristics						
Share of female	0.51	0.08	0.50	0.09	0.52	0.07
Share of born in the US	0.95	0.09	0.95	0.09	0.95	0.09
Share of White	0.65	0.31	0.65	0.31	0.65	0.31
Share of Black	0.19	0.26	0.20	0.26	0.19	0.26
Share of Hispanic	0.09	0.16	0.09	0.16	0.09	0.15
Average of age	15.91	1.66	15.92	1.66	15.90	1.66
Share of two parents	0.70	0.14	0.70	0.14	0.70	0.14
Average of parents' age	41.51	2.07	41.52	2.08	41.50	2.06
Average of SES	0.03	0.46	0.03	0.46	0.03	0.45
Group size	24.57	27.79	24.43	27.53	24.70	28.06
N	8,291		3,927		4,364	

Notes: Summary statistics for our estimation sample. Variable means are weighted using Add Health sample weights.

According to the DSM-IV, to be diagnosed with ADHD, an individual must have six or more symptoms of inattention and/or six or more symptoms of hyperactivity-impulsivity that have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level (see the full list of inattention, hyperactivity, and impulsivity symptoms in Appendix A). In addition, some impairment from the symptoms must be present in at least two settings (e.g., school and home), and some hyperactive-impulsive or inattentive symptoms must have been present before age 7 (age 12 in the DSM-V). However, extending the age of onset criterion from age 7 to age 12 in the DSM-V has been

associated with a very small increase in ADHD prevalence, possibly because most adults diagnosed with ADHD recall that their symptoms began before age 12.[17]

2.3.2 ADHD in Add Health: Outcome Variable, Prevalence, and Gendered Patterns

Our outcome variable is a binary indicator derived from the question *"Has a doctor, nurse, or other health care provider ever told you that you have or had: attention problems or ADD or ADHD?"* asked to Add Health respondents in Wave IV.

Panel A of Table 1 shows the percentage of individuals who answered "yes" to this question. The overall prevalence of ADHD diagnoses in our working sample (about 6%) is in the range reported in other papers using diagnosis information from Add Health (Fletcher, 2014), and other US-based surveys, such as the National Survey of Children's Health or the National Health Interview Survey (Bitsko et al., 2022; Bozinovic et al., 2021).[18] Consistent with previous evidence (Bitsko et al., 2022; Skogli et al., 2013; Fletcher, 2014; Bedard and Witman, 2020), we find that ADHD is diagnosed about twice as often in boys (8%) than in girls (4%).

Previous research suggests that the gender gap in ADHD prevalence may be due to differences in the expression of the disorder in males and females (Quinn, 2008; Skogli et al., 2013; Biederman et al., 2002; Levy et al., 2005). First, girls with ADHD tend to have fewer hyperactive/impulsive symptoms and more inattentive symptoms than boys with ADHD. Moreover, boys with ADHD tend to have more externalizing behaviors, which are more visible and overt, while girls with ADHD tend to have more internalizing comorbidities, which may be less noticeable to teachers, parents, and healthcare providers.

This is not surprising, as it is known that the male-female disruptive behavior

[17] See (CBHSQ, 2016) and references therein.

[18] More recent estimates of ADHD diagnosis based on the NHIS or NSCH are higher than those based on Add Health because Add Health respondents were in grades 7-12 in 1994-95, and ADHD prevalence has been increasing since the late 1990s and early 2000s (Akinbami et al., 2011; Perou et al., 2013; Visser et al., 2014).

gap affects children and adolescents in general, not just those diagnosed with ADHD (Bertrand and Pan, 2013). Consistent with this evidence, Table B2 in Appendix B shows that the prevalence of indicators related to externalizing behaviors, such as being suspended and expelled from school, shoplifting, and engaging in fighting, are significantly higher for boys than for girls in our data, with most gaps being statistically different from zero.

In addition, Sciutto et al. (2004) show that, especially when facing symptoms of hyperactivity, teachers refer boys for treatment more often than girls, even when the symptom profile is the same. These findings do not deny the existence of gender differences in symptom expression, but rather suggest that a gender bias in teachers' perceptions may also influence referral decisions.

The marked differences between boys and girls documented previously suggest that it may be worth considering peer gender as a relevant factor in our analysis. That is, if relative standards do indeed influence ADHD diagnosis, it may be appropriate to compare individuals within their gender group rather than across genders. The extent to which this hypothesis holds is a matter that requires empirical investigation, and we will explore this question further in the following sections.

2.4 Construction of an ADHD Ordinal Polygenic Rank

2.4.1 ADHD Polygenic Scores in Add Health

We construct the school-by-grade ordinal rank for students' genetic predisposition to ADHD using an ADHD polygenic score available for Add Health respondents. Polygenic scores (PGS), sometimes referred to as polygenic indices, polygenic risk scores, or genetic risk scores, are summary measures of an individual's genetic predisposition to an outcome or phenotype of interest (e.g., ADHD, depression, educational attainment, body mass).

The calculation of PGS is based on summary statistics from genome-wide association studies (GWAS). GWAS use a data mining approach to analyze associations between a phenotype and a large number of genetic variants. In approx-

imately 99% of the human genome, there is no variation between individuals. The locations in the genome where there is some variation between individuals are called genetic variants or single nucleotide polymorphisms (SNPs). The estimated associations for each SNP and a phenotype from a GWAS conducted on a large independent sample can be used to construct weights to calculate polygenic scores in independent samples.[19] In Add Health, the PGS are calculated according to the procedure described in Dudbridge (2013). In particular, the raw ADHD PGS for an individual i is calculated as:[20]

$$PGS_i = \sum_{j=1}^{k} \hat{\beta}_j SNP_{ij}, \tag{1}$$

where SNP_{ij} is the allele frequency of SNP j for individual i, and $\hat{\beta}_j$ is the estimated association between SNP j and the probability of being diagnosed with ADHD, obtained in the GWAS conducted by Demontis et al. (2019) using an independent sample of 55,374 individuals (20,183 cases and 35,191 controls) from 12 cohorts of mixed ancestry.[21] Thus, the ADHD PGS is a weighted sum of the regression coefficients $\hat{\beta}_j$ for each SNP from Demontis et al. (2019) and the allele frequencies for the same SNPs in the Add Health genome-wide data. Once calculated, the raw PGS are standardized to have a mean of 0 and a standard deviation of 1 within ancestry groups, to account for between-group population stratification. To control for within-group population stratification, we follow the recommendation to include at least the first five ancestry-specific principal components of the genome-wide data as covariates in all analyses using PGS (Price et al., 2006; Benjamin et al., 2012).

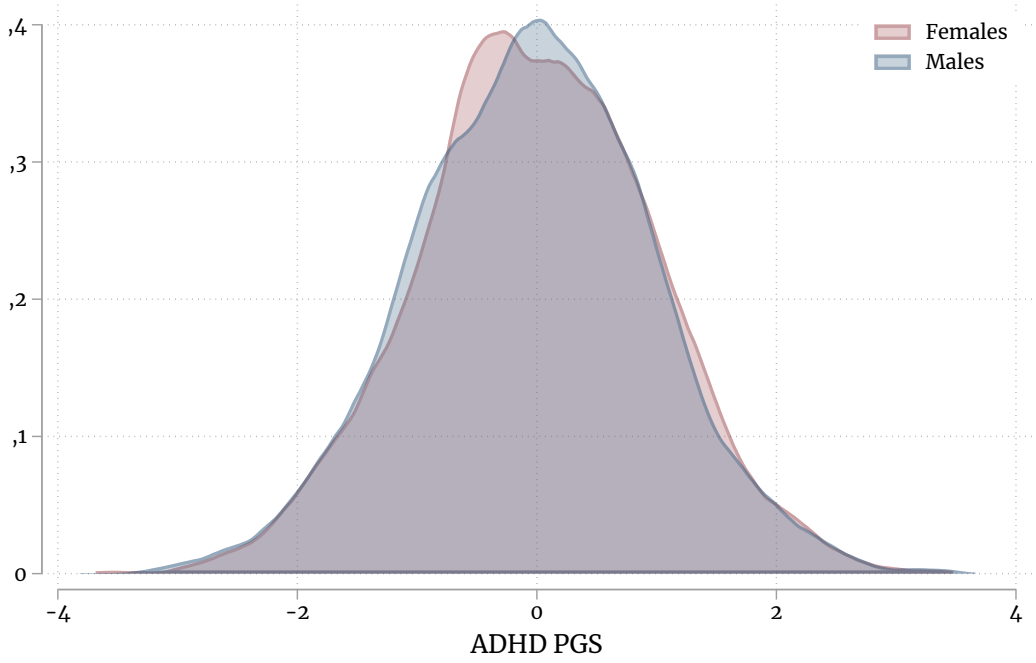
[19] Abdellaoui and Verweij (2021) provides a detailed discussion of polygenic scores and their interpretation.

[20] See the Add Health documentation (https://addhealth.cpc.unc.edu/wp-content/uploads/docs/user_guides/WaveIVPGSRelease2UserGuide.pdf) for details on the construction of polygenic indices in this dataset.

[21] These samples included a population-based cohort from Denmark collected by the Lundbeck Foundation Initiative for Integrative Psychiatric Research, and 11 European, North American and Chinese cohorts aggregated by the Psychiatric Genomics Consortium.

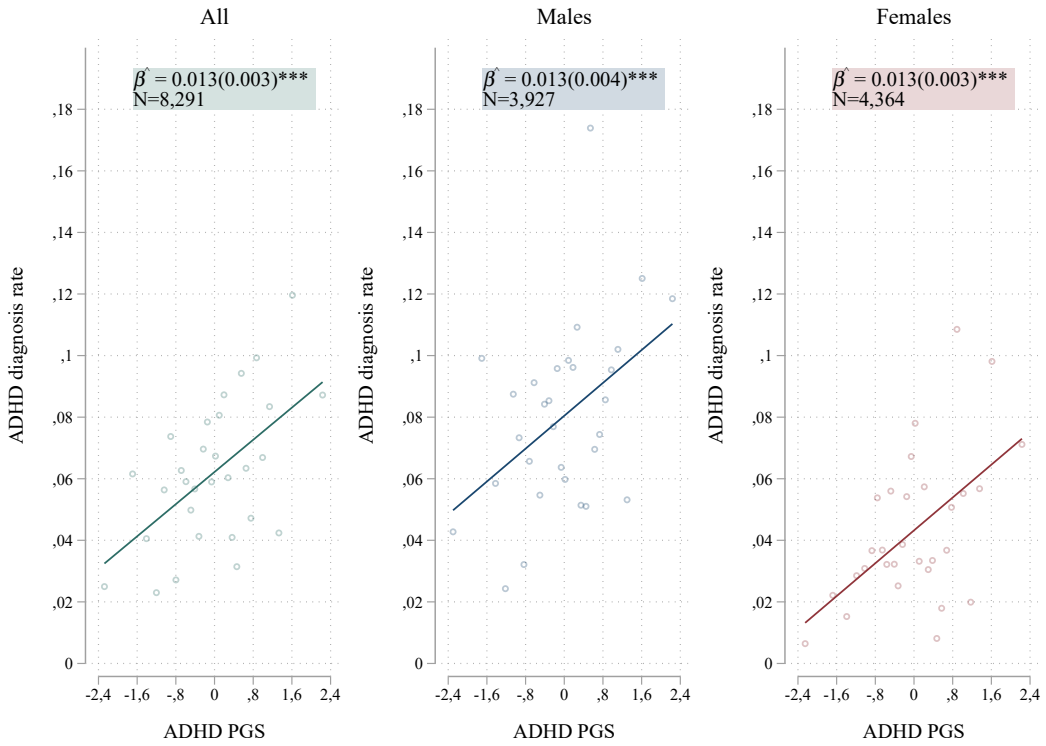
Figure 1 plots the kernel-smoothed densities of Add Health respondents' ADHD PGS, separately by gender. The distributions are approximately normal and do not vary significantly by gender. This evidence illustrates that the previously documented higher prevalence of ADHD in males is unlikely to be due to gender differences in genetic endowments.

Figure 1: ADHD PGS Distribution by Gender



Next, in Figure 2 we show that the ADHD PGS is indeed positively associated with the likelihood of being diagnosed with ADHD in our working sample of Add Health respondents. Specifically, a standard deviation increase in the ADHD PGS increases the odds of being diagnosed with ADHD by 1.3 percentage points for both males and females in our sample. This association is not only large, but also statistically different from zero ($p - value < 0.001$ for both males and females).

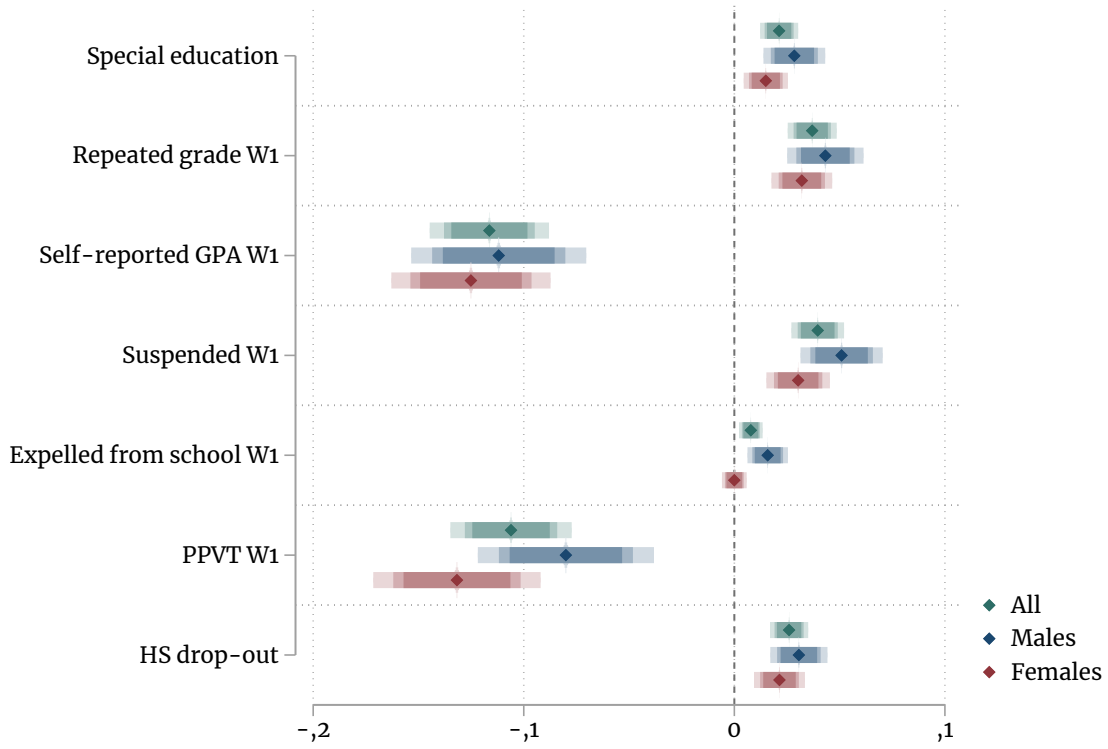
Figure 2: ADHD Polygenic Scores and ADHD Diagnosis



Notes: The probability of being diagnosed with ADHD and the ADHD PGS are plotted on the vertical and horizontal axes, respectively, for all, male, and female individuals in our work sample. The ADHD PGS is standardized to have a mean of 0 and a standard deviation of 1. The table shows, at the top of each panel, the OLS coefficients obtained after regressing the probability of being diagnosed with ADHD on the ADHD PGS and their associated standard errors.

An important concern for our analysis is that, while the value of the ADHD PGS is known to analysts, it is unlikely to be known to parents and teachers. However, parents and teachers do observe some characteristics related to students' ADHD PGS and may therefore respond by consulting or recommending consultation with a health professional. Importantly, Figure 3 indicates that ADHD PGS, while likely unknown to parents and teachers, are strongly associated with potential observable manifestations of ADHD that may lead to a diagnosis.

Figure 3: ADHD Polygenic Scores and ADHD-related Behavioral and Cognitive Indicators



Notes: The table reports the slope coefficients and the associated confidence intervals of linear regressions of the probability of observing each of the outcomes reported in the rows of the panel on the ADHD PGS, for all (green), male (blue), and female (red) individuals in our working sample. The ADHD PGS is standardized to have mean 0 and standard deviation 1.

2.4.2 Main regressor: ADHD Ordinal Polygenic Rank

We compute students' relative genetic predisposition to ADHD based on their absolute rank in the school-by-grade distribution of the ADHD PGS.[22] Following Murphy and Weinhardt (2020), we safeguard against the possibility that measurement error in the ADHD PGS increases multiplicatively further from the

[22]The student with the lowest ADHD PGS in the grade has a rank of 1, the second has a rank of 2, and so on. In case of ties, we assign the lower rank to all students with the same genetic propensity for ADHD, as in Elsner and Isphording (2017, 2018) and Kiessling and Norris (2023), but other ways of correcting for ties produce very similar results.

mean and generates a spurious rank effect by using the uniformly distributed percentiles instead of the raw values of the ADHD PGS level, both as a control and as the reference to compute the rank. Furthermore, since grades within schools may have a different number of students, we then transform students' absolute rank (i.e., $1, 2, 3, \dots$) into a percentile rank using the following expression:

$$R_{isg} = \frac{A_{isg} - 1}{N_{sg} - 1}, \tag{2}$$

where A_{isg} is the absolute genetic ADHD rank of student i in school s in grade g, and N_{sg} is the number of students in grade sg. R_{isg} falls within the unit interval, assigning a value of 0 to the student with the lowest genetic propensity for ADHD and a value of 1 to the student with the highest propensity within a given grade. Since this percentile rank is ordinal and does not contain any cardinal information (i.e., relative information about the genetic tendencies of individuals), we will refer to R_{isg} as the ordinal genetic rank. Ranking individuals on the basis of a given genetic predisposition has the advantage that an individual's genetic makeup is fixed at birth and cannot be influenced by peers, teachers, parental influences, or the environment. This eliminates any concern that the reflection problem might bias our results (Manski, 1993).

3 Empirical Strategy

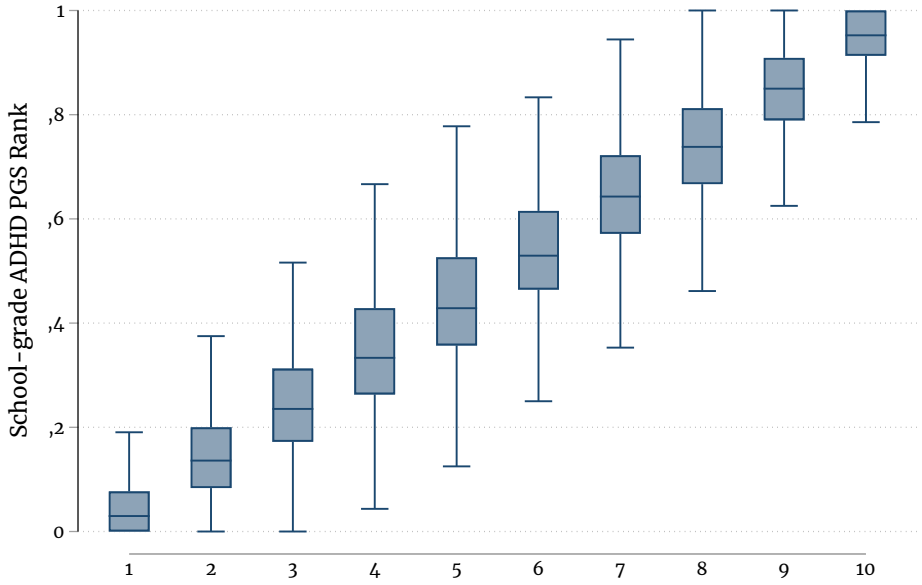
3.1 Identification

Borrowing ideas from Denning et al. (2021), the experiment one would ideally design to determine the effect of within-group ordinal ADHD PGS rank on ADHD diagnosis involves randomly assigning students with the same ADHD PGS level to small groups drawn at random from the population. In this way, all students would be expected to have the same ex-ante ADHD PGS distribution within their group. However, due to small sample variability, the realized group distributions will differ slightly and by chance, thereby generating as-if random variation in ordinal ADHD PGS rank for students with the same ADHD PGS assigned to

different groups.

In the spirit of Hoxby (2000), we mimic this ideal experiment by exploiting the variation in the distribution of ADHD PGS observed across school grades in the Add Health data. Figure 4 illustrates that among students with the same ADHD PGS level, there is considerable variation in the ordinal ADHD PGS rank of students within their grade. For example, students in the fifth decile of the global ADHD PGS distribution rank approximately between the second and eighth deciles of the local ("within school-grade") ADHD PGS distribution.

Figure 4: Global vs. local ADHD Ordinal Polygenic Rank



Notes: This figure shows the relationship between the ordinal genetic rank in the distribution of ADHD PGS (global genetic deciles) and the ordinal genetic rank in the school-grade (local genetic rank). The box whiskers represent the median (blue line), the 25th and 75th percentiles (lower and upper bounds of the box), and the minimum and maximum of the local genetic rank, respectively.

As our analysis pools students with different absolute levels of ADHD PGS, a first requirement for our empirical model is to flexibly control for the mapping between ADHD PGS and ADHD diagnosis. Our baseline specification uses a cubic functional form, but we show that our results are robust to different polynomial choices, as well as to making this mapping school-specific by interacting

the ADHD PGS polynomial with school dummies.

Second, while the predetermined nature of the ADHD PGS with respect to group assignment eliminates concerns about reverse causality or reflection issues, it is still possible that the variation we observe across school grades is due to student sorting. This could occur, for example, if parents attempted to place children with vivid manifestations of ADHD in school grades with few other students with such manifestations, in the hope that teachers could give them more attention. We deem this kind of sorting implausible. Even if parents could use information on past grades to infer the distribution of ADHD PGS that their children might face, small sample variation and grade-specific shocks would still make it unlikely that they could predict the exact distribution realized in each school grade.

We overcome concerns about sorting by including in our model both a comprehensive set of individual pre-determined student characteristics and school and grade or, in our preferred specification, school-by-grade fixed effects.

Murphy and Weinhardt (2020), Elsner et al. (2021), and Delaney and Devereux (2022) highlight that the inclusion of school-by-grade fixed effects leads to a between-group comparison of students with the same ADHD PGS relative to the group mean, but with different ranks due to differences in the distribution of ADHD PGS across groups. By subtracting the group mean from each variable, the within-group estimator does not change the shape of the ADHD PGS distribution, while eliminating differences in mean ADHD PGS across groups. As a result, the inclusion of school-by-grade fixed effects cleans our estimates of the impact of mean-shifting effects common to students in the same school and grade, such as those due to teachers or, importantly, peers.

The specifications with school and grade instead of school-by-grade fixed effects do not share this property, and we account for the joint determination of rank and peer composition highlighted by Bertoni and Nisticò (2023) by including in the model the leave-me-out mean and standard deviation of school-grade peers' ADHD PGS distribution, as well as other observable peer characteristics such as the share of females and all the other school-by-grade covariates summa-

rized in Panel D of Table 1.

In some robustness tests, we also include in our model a set of interaction terms between the first two moments of the ADHD PGS distribution in the school grade and the student's own ADHD PGS. As illustrated by Bertoni and Nisticò (2023) and Denning et al. (2021), this specification effectively controls for non-linear and heterogeneous peer effects, while mimicking the ideal experiment in which students with the same ADHD PGS are assigned to groups with the same ex-ante ADHD PGS distribution.

3.2 Estimation

Following these considerations, we estimate with Ordinary Least Squares (OLS) the following empirical model:

$$y_{isg} = \alpha_{sg} + \beta R_{isg} + g(PGS_{isg}) + X'_{isg}\delta + \varepsilon_{isg}, \tag{3}$$

where y_{isg} is our outcome, a dummy variable equal to 1 if student i attending school s and grade g has ever been diagnosed with ADHD, and 0 otherwise; α_{sg} are school-by-grade fixed effects; R_{isg} is our main regressor of interest, i.e., student i's ordinal genetic rank within his/her school grade; $g(PGS_{isg})$ is a cubic polynomial function in the student's own ADHD PGS; and $\mathbf{X_{isg}}$ is a vector of individual-specific controls, which includes the following variables: gender, age and its square, indicators for being born in the US and for race (white, black, hispanic, other), an indicator for whether both parents live in the household, parental age, an index of socioeconomic status, and the first ten principal components of all genotypes measured in the SNP data matrix - to control for population stratification. Finally, ε_{isg} is an idiosyncratic error term, and we account for the dependence of the error term on students enrolled in the same school by clustering the standard errors at the school level (there are 130 schools in our data).

As expected, we will also estimate Equation (3) taking into account gender-specific ordinal genetic rank within a school grade, as well as after splitting the

sample by gender. In these cases, our measures of peer composition will be gender-specific, and so will be the school-by-grade fixed effects.

3.3 Assessing the Plausibility of the Identifying Assumption

A first important check is to verify that the demanding set of fixed effects and controls included in Equation (3) leaves enough remaining variation in rank (R_{isg} in Equation (3)) that can be exploited for identification. To this end, we report in Table B3 in Appendix B the standard deviation of the error term of a regression of rank on the fixed effects and controls included in Equation (3). We repeat this exercise when we compute rank within school grade or within gender and school grade, and in the full sample as well as in gender-specific subsamples. We find that the residual variation in rank is between 22.6 and 39.4% of the total, a non-negligible fraction.

Next, we perform a series of balancing tests to determine whether, despite the non-random assignment of students to grades, our identification strategy can attenuate the potential correlation between student characteristics and their ADHD PHG rank, thereby replicating a situation in which rank is as good as randomly assigned for students with the same ADHD PGS level. To do this, we estimate Equation (3) using each of the available controls in the vector X_{isg} as the dependent variable, and check whether or not the estimated rank effects are statistically different from zero. Table 2 reports the results for within-school-grade rank, while the results for within-gender and school-grade rank are in Table 3. Both tables report results for the full sample and for the gender subsamples. Reassuringly, the estimated coefficients are very close to zero in magnitude, and only 6 out of 114 are statistically different from zero, a result consistent with a Type I error rate of 5 percent.

Table 2: Balancing tests: ADHD PGS rank

	All		Males		Females	
	(1)	(2)	(3)	(4)	(5)	(6)
	Coeff.	St. Error	Coeff.	St. Error	Coeff.	St. Error
Female	-0.120	0.086	0.000	.	0.000	.
Age (in months)	-0.008	0.115	-0.040	0.182	0.023	0.152
Born in the US	0.004	0.035	0.036	0.050	-0.027	0.050
White	0.065	0.071	0.030	0.103	0.134	0.101
Black	-0.066	0.041	0.016	0.059	-0.116*	0.066
Hispanic	-0.017	0.040	-0.050	0.058	-0.003	0.066
Raised by two parents	0.001	0.084	-0.123	0.136	0.106	0.129
Respondent parent's age	0.481	1.099	1.905	1.799	-0.762	1.708
Socio-economic Status	0.205	0.165	0.248	0.251	0.192	0.236
PC1	0.007	0.005	0.014	0.012	0.001	0.004
PC2	0.005	0.004	0.009**	0.004	-0.005	0.006
PC3	-0.000	0.003	-0.006	0.005	0.007	0.005
PC4	0.002	0.003	0.009	0.007	-0.003	0.004
PC5	0.001	0.004	-0.000	0.007	0.004	0.005
PC6	-0.000	0.004	0.001	0.005	-0.001	0.007
PC7	-0.006	0.004	-0.003	0.004	-0.008	0.007
PC8	0.004	0.004	0.007	0.006	-0.000	0.006
PC9	-0.001	0.003	-0.000	0.006	-0.003	0.003
PC10	0.001	0.004	-0.006	0.005	0.011*	0.007

[1] Estimates are weighted using Add Health sample weights. [2] Standard errors clustered at the school level * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 3: Balancing tests: ADHD PGS gendered-rank

	All		Males		Females	
	(1)	(2)	(3)	(4)	(5)	(6)
	Coeff.	St. Error	Coeff.	St. Error	Coeff.	St. Error
Female	0.005	0.099	0.000	.	0.000	.
Age (in months)	0.017	0.069	0.017	0.111	-0.060	0.102
Born in the US	0.008	0.015	0.029	0.026	-0.001	0.028
White	0.034	0.033	-0.072	0.060	0.092	0.069
Black	-0.009	0.021	0.038	0.035	-0.124***	0.042
Hispanic	-0.010	0.028	0.010	0.044	0.022	0.053
Raised by two parents	0.055	0.050	-0.018	0.091	0.118	0.075
Respondent parent's age	-0.172	0.624	0.985	1.049	-1.181	1.166
Socio-economic Status	0.025	0.099	0.053	0.160	-0.001	0.161
PC1	0.001	0.002	0.003	0.002	0.001	0.003
PC2	0.000	0.002	0.002	0.003	-0.002	0.004
PC3	0.001	0.002	-0.004	0.003	0.005	0.003
PC4	-0.002	0.002	0.001	0.002	-0.004	0.003
PC5	0.000	0.002	-0.003	0.003	0.007**	0.003
PC6	0.002	0.002	-0.001	0.003	0.005	0.005
PC7	-0.004	0.003	-0.002	0.003	-0.008	0.006
PC8	0.002	0.002	0.002	0.003	0.001	0.004
PC9	0.004**	0.002	0.004	0.003	0.002	0.002
PC10	0.001	0.002	-0.001	0.002	0.007	0.005

[1] Estimates are weighted using Add Health sample weights. [2] Standard errors clustered at the school level * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

4 Main Results

Table 4 reports estimates of the effect of ADHD PGS rank on the probability of receiving a professional ADHD diagnosis. Column (1) includes school and grade fixed effects, a cubic polynomial in the student's own ADHD PGS, the set of individual controls listed in Table 1, and the mean ADHD PGS of the student's school-grade peers. Column (2) enriches the specification by including

the variance of the ADHD PGS for students' school-grade peers and the means of other school-grade peer characteristics. Finally, Column (3) reports results from our preferred specification with school-by-grade fixed effects.

Table 4: Average Effects of ADHD PGS Ordinal Ranks on ADHD Diagnosis

	(1)	(2)	(3)
Professional ADHD diag.			
Panel A			
ADHD PGS rank	0.057	0.055	0.069
	(0.040)	(0.040)	(0.043)
R-squared	0.062	0.063	0.117
Panel B			
ADHD PGS gendered-rank	0.073***	0.073***	0.075***
	(0.025)	(0.025)	(0.027)
R-squared	0.063	0.065	0.118
Observations	8,291	8,291	8,291
Own ADHD PGS cubic	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes
ADHD PGS school-cohort mean	Yes	Yes	No
ADHD PGS school-cohort variance	No	Yes	No
Further school-cohort means	No	Yes	No
School and Cohort FE	Yes	Yes	No
School x Cohort FE	No	No	Yes

[1] Estimates are weighted using Add Health sample weights.

[2] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Panel A shows the results when we compute the ADHD PGS rank after pooling males and females in the school grade. It shows a positive but statistically insignificant effect of the ADHD PGS rank on ADHD diagnosis, ranging from 5.7 to 6.9 percentage points, depending on the specification. A clearer picture emerges from Panel B, where we report the effect of within-gender ADHD PGS

rank. We find that moving from the bottom to the top of the school grade in terms of ADHD PGS increases the probability of being diagnosed with ADHD by 7.3 to 7.5 percentage points, depending on the specification. This result implies that a 1SD increase in rank –equal to 0.33– increases the probability of diagnosis by 2.5 percentage points, or 42% of the ADHD diagnosis rate in the sample, a large effect that is statistically significant and remarkably stable across specifications.

Next, in Table 5 we replicate results in Column (3) of Table 4 - our preferred specification - after splitting the sample by gender. Panel A confirms the absence of a significant effect of the school-grade ADHD PGS rank computed after pooling males and females, both in the pooled sample and within gender.

On the contrary, the evidence in Panel B indicates that the significant impact of the within-gender ADHD PGS rank uncovered in the full sample is mostly driven by males. We find that the impact of the ADHD PGS gendered rank on the likelihood of ADHD diagnosis is statistically significant at standard levels of testing for males ($p - value < 0.01$) but not for females ($p - value > 0.1$). The magnitude of the estimated effect of interest is more than twice as large for males (13.3 percentage points) than for females (6 percentage points), and the p-value of a one-tailed test for the hypothesis that the effect is larger for males than for females is 0.11, at the margin of significance according to standard practice.[23] When we measure these effects in relative terms, we find that they are quantitatively relevant for both males and females, although the effect for females is far from reaching standard levels of statistical significance. In particular, a 1SD increase in within-gender rank for males increases the likelihood of ADHD diagnosis by 4.4 percentage points, or 55% of the mean diagnosis rate for males in the sample, while for females the effect is 2 percentage points, or 50% of the mean diagnosis rate for females.

We can thus summarize our main findings as follows: comparing ADHD PGS rank within gender and school grade has a substantial and significant effect on the likelihood of receiving a professional diagnosis of ADHD, and this effect is

[23]See McShane et al. (2019) for a discussion of the use of thresholds to decide on statistical significance.

primarily driven by males. In contrast, the ADHD PGS rank within school grade, after pooling gender, is clearly not as relevant. This set of findings is consistent with the hypothesis that teachers and families apply heuristic, subjective standards and rely on interpersonal comparisons when assessing the likelihood that a student has ADHD. In particular, they appear to assess ADHD issues by gauging the relative manifestations of students' symptoms within gender and school grade, even conditional on students' age, gender, and their own ADHD PGS levels.

Table 5: Average Effects of ADHD PGS Ordinal Gendered-Rank on ADHD Diagnosis by Gender

	(1)	(2)	(3)
Professional ADHD diag.	All	Males	Females
Panel A			
ADHD PGS rank	0.069	0.073	0.086
	(0.043)	(0.070)	(0.057)
R-squared	0.117	0.206	0.150
Panel B			
ADHD PGS gendered-rank	0.075***	0.133***	0.060
	(0.027)	(0.048)	(0.042)
R-squared	0.118	0.208	0.150
Observations	8,291	3,927	4,364
Own ADHD PGS cubic	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes
ADHD PGS school-grade mean	No	No	No
ADHD PGS school-grade variance	No	No	No
Further school-grade means	No	No	No
School and Grade FE	No	No	No
School x Grade FE	Yes	Yes	Yes

[1] Estimates are weighted using Add Health sample weights.

[2] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5 Robustness Tests

In this section, we provide a series of tests that corroborate the validity of our results. We will focus on the results in Table 5, and assess their robustness along several dimensions, including functional form choices; alternative rank definition; the timing of ADHD diagnosis; the strategic timing of school entry; sample selection, and panel attrition. We now describe each set of tests in turn, and report the results in Appendix B.

5.1 Functional Form Choices

Our baseline specification in Equation (3) adopts a cubic functional form to model the relationship between PGS_{isg} and the outcome. However, spurious rank effects could arise as a result of an incorrect choice of functional form. In Table B4 we assess whether the cubic specification is appropriate by using PGS_{isg} polynomials of different orders, from the first to the seventh, and find results that are remarkably stable in both the full sample and the gender subsamples. Moreover, Table B5 shows that our results hold even when we allow this mapping to be school-specific by interacting the (cubic) PGS_{isg} polynomial with school dummies.

Furthermore, our main specification does not account for the possibility of heterogeneous effects of the ADHD PGS that depend on the school-grade distribution of the PGS. While the inclusion of school-by-grade fixed effects implicitly controls for homogeneous school-by-grade peer effects, it does not safeguard against the possibility that rank effects may actually capture heterogeneous peer effects (Denning et al., 2021; Bertoni and Nisticò, 2023). To address this concern, we adopt the specification suggested by Booij et al. (2017) and include interaction terms between the ADHD PGS and the leave-me-out mean and standard deviation of its distribution within each school grade. Table B6 shows that the results are again fairly stable.

Finally, the specification used in Equation (3) assumes that rank effects are linear. For instance, under linearity moving from the first to the second quar-

tile of the ADHD PGS distribution has the same effect on ADHD diagnosis as moving from the second to the third quartile (see Gill et al., 2019). We assess the plausibility of this assumption by replacing the linear functional form for rank with a non-linear one that includes dummies for quartiles of the within-gender-by-school-grade ADHD PGS distribution, taking the second quartile as the omitted reference category. Results in Figure B1 are supportive of the linear specification.[24]

5.2 Alternative Rank Definitions

In defining rank, we have broken ties by following the default definition first adopted by Murphy and Weinhardt (2020) and assigning the lower rank to all students. While this is an arbitrary choice, in Table B7 we show that our results are robust to breaking ties using the average rank, a random order, or the maximum rank.

5.3 Ability Rank

A potential concern related to our strategy is that the ADHD PGS rank may actually be capturing the impact of ordinal rank along with other individual traits that are correlated with the ADHD PGS and not included in our model. Given the evidence of ability rank effects in determining individual educational attainment, risky behaviours, and mental health in the Add Health cohorts (Elsner and Isphording, 2017, 2018; Kiessling and Norris, 2023), a prime candidate among such traits is individual ability. To dispel this concern, we augment our baseline specification with a cubic polynomial in students' PPVT test scores - the ability proxy

[24]While there is some evidence of non-linear effects at the bottom of the rank distribution for males, the differences with the linear specification are not statistically significant. We also assessed whether the rank effect is heterogeneous depending on students' quartile in the ADHD PGS and SES distribution in the full sample, but we failed to find significant evidence of heterogenous effects along these two dimensions.

used in the above-mentioned studies - and the corresponding ordinal rank.[25] The estimated effects of the ADHD PGS rank obtained from this richer specification are reported in Table B8, and are again comparable to our benchmark findings.

5.4 Timing of ADHD diagnosis

As stated in Section 2.3.1, our dependent variable measures whether Add Health respondents had ever been diagnosed with ADHD by Wave IV. Figure B2 reports the age at ADHD diagnosis, and shows that a substantial share of cases is diagnosed before pupils meet their middle/secondary school peers. We verify that our results are not picking up a spurious effect on the diagnosis that took place before school starting age - that we set at 5 - or even before middle/secondary school age - that we set at 9 - by replicating our main estimations in Table 5 after dropping from the sample those pupils who had already been diagnosed with ADHD before those ages. Results in Table B9 are comparable to our benchmark.[26]

5.5 Strategic Timing of School Entry

Another assumption behind our identification strategy is that there is no sorting of students according to the expected composition of the school-grade. Parents may potentially manipulate their children's placement by delaying school entry conditional on their choice of a particular school. While we deem this kind of sorting as unlikely, to assess whether our results are robust to potential concerns

[25] One could argue that, since it is measured after birth, the outcome of the PPVT test could be influenced by individuals' genetic predisposition to ADHD. If this was the case, then the PPVT score would be a "bad" control, and impaired cognitive ability shall be considered as a mechanism behind the impact of ADHD PGS rank on ADHD diagnosis. For this robustness test, we nonetheless conform with the existing studies and treat PPVT as a valid measure of ability.

[26] A potential concern about the age-9 placebo is that schoolmates in primary and middle/secondary schools could in part overlap if there was little mixing of students between primary and secondary school. As a result, we do not over-emphasize the validity of this exercise.

about strategic school entry timing, we follow Elsner and Isphording (2017) and replicate our main estimates after restricting the sample to age bands of 0.4 years around the mean age of an entire grade, thereby eliminating students who may be late entrants as well as grade repeaters. The results in Table B10 are again comparable to our benchmark.

5.6 Sample Selection Criteria

Another potential source of concern relates to the sample selection criteria we are forced to adopt in order to use the genetic information collected in Wave IV. On the one hand, approximately 25% of students drop out of the survey between Waves I and IV. On the other hand, of the 96% of students who were asked to participate in DNA collection in Wave IV, only 80% consented to long-term archiving and were then eligible for genotyping. In addition, quality control protocols also affected the actual availability of genetic data. As mentioned in Section 2, the combination of these two factors leads us to work with a final sample of 8,410 students out of the 20,745 who were present in the Wave 1 sample. This problem has two important consequences. First, we are working with a final sample that is selected and may not be representative of the population of interest, and a relevant concern in our case is that selection may depend on pupils' genetic predisposition for ADHD. Second, we can only construct the school-grade ADHD PGS rank for the subset of students who are included in our final sample and for whom information on the ADHD PGS is available, thereby introducing a source of error in the measurement of rank.

We assess the consequences of these potential issues for our estimates by relying on two re-weighting strategies.

First, in Column (1) of Table B11 we follow Mazzonna and Peracchi (2017) and address non-random sample selection due to attrition and/or the unavailability of valid genetic data by estimating in the full Wave I sample a probit model for not being present in the final sample conditional on the same set of individual controls used in our benchmark Equation (3), interviewer fixed effects, and a set of variables taken from the interviewer questionnaire administered in Wave I

that serve as exclusion restrictions to identify the selection equation.[27] We then multiply the inverse of the predicted probabilities of not being in the final sample obtained for each student by their Add Health sampling weights, and we use the resulting weights when re-estimating our main model in the final sample. The results are qualitatively similar to the baseline estimates reported in Table 5.

Second, in Column (2) of Table B11, we address the issue of measurement error in rank due to missing data on the ADHD PGS by computing the retention rate in each school-grade-gender group and then using the retention rates as weights (combined with sampling weights) in our main regression model estimated in the final sample. By giving more weight to school grades in which more students are retained, this strategy effectively mitigates the problem of measurement error. Again, reassuringly, the results are very comparable to our baseline.

Finally, we can also assess the severity of the measurement error in rank induced by sample selection by comparing the rank constructed in the full and selected samples for variables that are observed in Wave I for all pupils. As discussed above, one such variable is the PPVT test. We find that, in our final estimation sample, the correlation between the PPVT school-by-grade rank computed in the full Wave 1 sample and in our selected sample is above 0.964 - a very high value.

6 Conclusion

ADHD diagnoses and prescriptions for ADHD medication have increased dramatically in recent decades. This large increase has raised concerns about the subjective nature of ADHD diagnosis, a debate further fueled by the fact that ADHD

[27] The Wave I Interviewer Survey Questions used are: 1) *Where was the interview conducted?*; 2) *How would you describe the immediate area or street (one block, both sides) where the respondent lives?*; 3) *What type of residence is most common on the street (one block, both sides) where the respondent lives?*; 4) *How well kept are most of the buildings on the street?*; 5) *When you went to the respondent's home, did you feel concerned for your safety?*; 6) *Number of interruptions during the interview*; 7) *Did the respondent ever appear embarrassed about answering questions during the interview?*.

is diagnosed much more frequently in boys than in girls, although there are no significant gender differences in individuals' genetic predisposition to ADHD.

We use data on individuals' genetic predisposition to ADHD from the Add Health survey on U.S. schools to investigate the role of interpersonal comparisons in the diagnosis of ADHD among U.S. adolescents. We examine whether a student's ordinal rank in the distribution of genetic propensity for ADHD in their school-grade affects the likelihood of being diagnosed.

We find that a one standard deviation increase in students' ordinal rank in the genetic predisposition to ADHD among their same-gender grademates is associated with a 2.5 percentage point increase in the odds of being diagnosed with ADHD, holding students' age as well as their own genetic predisposition to ADHD constant. This effect is both statistically significant and large, accounting for 42% of the average diagnosis rate in our sample. Moreover, we find that this effect is mostly driven by boys, as the estimated rank effect for the subsample of girls fails to reach standard levels of statistical significance.

Our findings shed new light on the factors that may explain the gap in diagnosis rates between boys and girls. In addition, our findings highlight the critical role of children's environment in ADHD diagnosis and the importance of promoting interdisciplinary and coordinated efforts to improve ADHD diagnosis and minimize its subjective component.

For example, Pottegärd et al. (2014) argue that the fact that no significant differences in ADHD medication use are found between young and old children in Denmark, which is an exception to the general pattern, may be related to the relatively low use of ADHD medication and/or the common practice of delaying school entry for relatively young children in the country. Dalsgaard et al. (2014a) and Dalsgaard et al. (2012) suggest that another explanation for the Danish exception may be differences in diagnostic assessment for ADHD. In Denmark, only specialists (child psychiatrists and pediatricians) are responsible for the diagnosis and subsequent initiation of pharmacological treatment. Therefore, the consideration of age-for-grade as well as the promotion of specialist involvement in the diagnosis of ADHD may be avenues worth exploring by educators, healthcare

providers, and health policymakers.

In addition, a number of initiatives are underway to improve the assessment process for ADHD. In England, since April 2020, the Academic Health Science Networks have been supporting National Health Service mental health trusts and community pediatric services through the Focus ADHD program (https://acesse.dev/COORN). This program supports the implementation of an objective computer-based assessment tool (QbTest) to complement (but not replace) current clinical assessment processes. The technology, which helps inform clinical decisions by measuring the three core components of ADHD (attention, impulsivity and activity), has shown promising results to date.[28]

In addition, recent advances in neuroimaging-based tools that provide a comprehensive assessment of brain morphology, microstructure, and connectivity changes associated with ADHD may complement clinical assessment for the diagnosis of ADHD in children (Lin et al., 2023).

The widely documented psychosocial and economic burden of ADHD and the importance of timely and accurate diagnosis indicate that further research and initiatives in this area are urgently needed.

[28]See https://ur1.app/h2qU7 for a recent independent evaluation undertaken by the Institute of Mental Health into the roll-out of the Focus ADHD program and the use of the QbTest.

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Appendices

Appendix A DSM-IV criteria for ADHD diagnosis

ADHD diagnosis requirements:

1. 18 ADHD symptoms are divided into two symptom domains (inattention and hyperactivity/impulsivity), of which at least six symptoms in one domain are required for diagnosis. Symptoms must have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level.
2. Some hyperactive-impulsive or inattentive symptoms must have been present before age 7 years.
3. Some impairment from the symptoms is present in at least two settings (e.g., at school [or work] and at home).
4. There must be clear evidence of clinically significant impairment in social, academic or occupational functioning.
5. The symptoms do not occur exclusively during the course of a pervasive developmental disorder, schizophrenia, or other psychotic disorders and is not better accounted for by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, or a personality disorder).

DSM-IV symptoms by domain:

Inattention

1. Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.
2. Often has difficulty sustaining attention in tasks or play activity.
3. Often does not seem to listen when spoken to directly.

4. Often does not follow through on instructions and fails to finish schoolwork, chores or duties in the workplace (not due to oppositional behavior or failure to understand instructions).
5. Often has difficulty organizing tasks and activities.
6. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework).https://www.overleaf.com/project/6138
7. Often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books or tools).
8. Is often easily distracted by extraneous stimuli.
9. Is often forgetful in daily activities.

Hyperactivity symptoms

10. Often fidgets with hands or feet or squirms in seat.
11. Often leaves seat in classroom or in other situations in which remaining seated is expected.
12. Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness).
13. Often has difficulty playing or engaging in leisure activities quietly.
14. Is often "on the go" or often acts as if "driven by a motor".
15. Often talks excessively.

Impulsivity

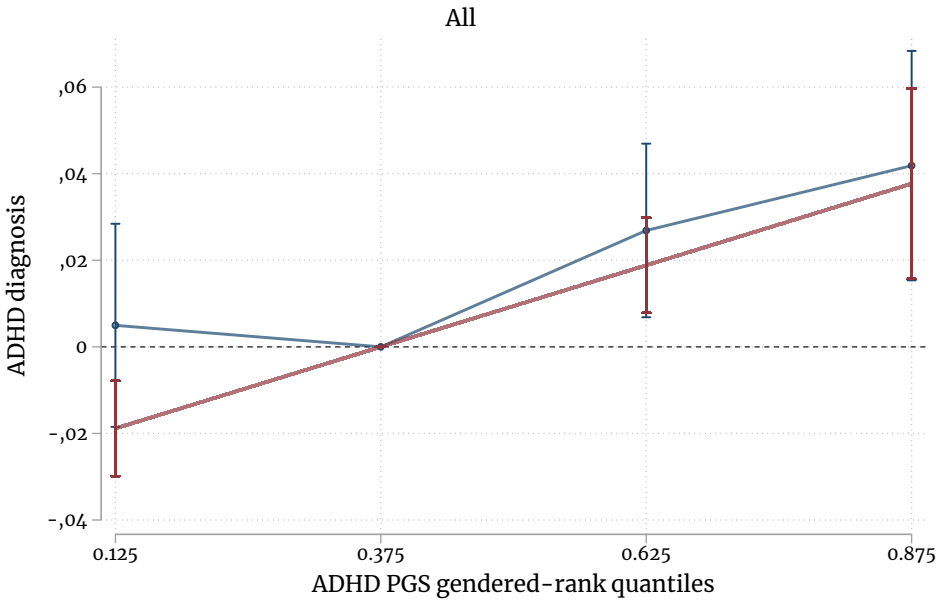
16. Often blurts out answers before questions have been completed.
17. Often has difficulty awaiting turn.
18. Often interrupts or intrudes on others (e.g., butts into conversations or games).

Source: https://www.ncbi.nlm.nih.gov/books/NBK519712/table/ch3.t3/.

Appendix B Additional Figures and Tables

Figure B1: Non-linear ADHD PGS gendered-rank

(a) Non-linear ADHD PGS gendered-rank



(b) Non-linear ADHD PGS gendered-rank by gender

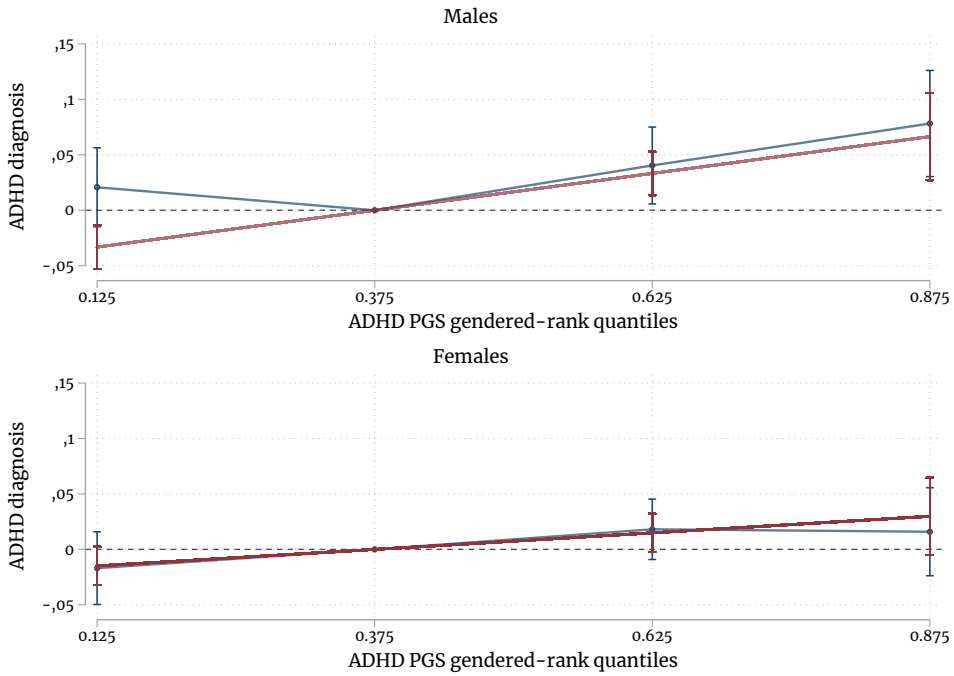


Figure B2: Age at ADHD diagnosis

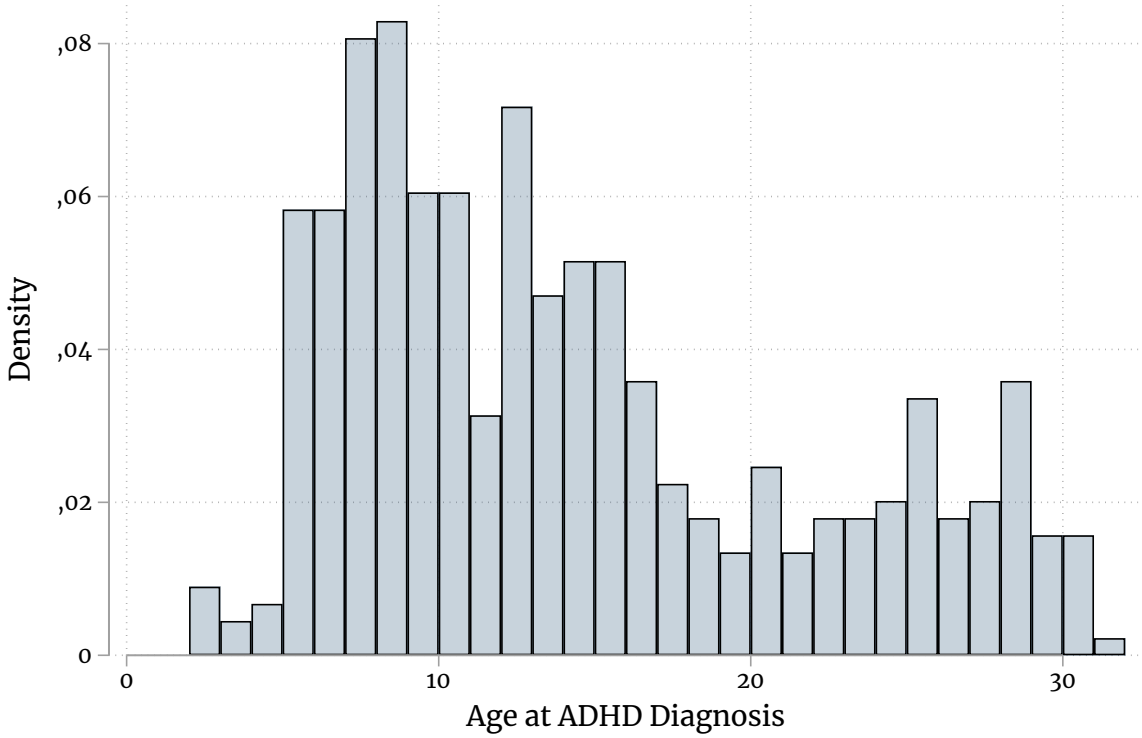


Table B1: Comparing representative versus estimation sample characteristics

	Representative sample			Estimation sample		
	(1)	(2)	(3)	(4)	(5)	(6)
	Mean	Std.Dev.	Obs.	Mean	Std.Dev.	Obs.
Professional ADHD diagnosis	0.06	0.23	14478	0.06	0.24	8291
Female	0.48	0.50	18456	0.49	0.50	8291
Age	15.94	1.78	18456	15.91	1.77	8291
Age^2	257.29	56.89	18456	256.23	56.39	8291
Born in the US	0.94	0.25	18456	0.96	0.19	8291
White	0.64	0.48	18456	0.73	0.44	8291
Black	0.16	0.37	18456	0.15	0.35	8291
Hispanic	0.12	0.33	18456	0.07	0.26	8291
Both parents live in hh	0.70	0.46	18456	0.72	0.45	8291
Parental age	41.48	6.44	18456	41.35	6.26	8291
Socio-economic status (std.)	-0.02	1.02	18456	-0.00	1.00	8291
PPVT W1 (std.)	-0.06	1.04	17581	0.00	1.00	7920

Notes: Summary statistics for the entire sample. Outcome means are weighted using Add Health sample weights.

Table B2: Cognitive Outcomes and Externalizing Behaviors by Gender

	All			Male			Female			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(4)-(7)
	Mean	Std. Dev.	N	Mean	Std. Dev.	N	Mean	Std. Dev.	N	Diff
Panel A: Cognitive outcomes										
Special education	0.10	0.30	7288	0.13	0.34	3468	0.07	0.25	3820	0.063***
Repeated grade W1	0.22	0.41	8285	0.27	0.44	3922	0.16	0.37	4363	0.104***
Self-reported GPA	-0.00	1.00	8216	-0.14	1.01	3882	0.15	0.97	4334	-0.286***
Suspended W1	0.27	0.44	8288	0.35	0.48	3924	0.18	0.39	4364	0.168***
Expelled from school W1	0.04	0.20	8288	0.06	0.23	3924	0.02	0.15	4364	0.035***
GPA	-0.00	1.00	5803	-0.20	1.01	2673	0.19	0.95	3130	-0.388***
HS drop-out	0.12	0.32	8291	0.13	0.33	3927	0.11	0.31	4364	0.018***
Panel B: Risky behaviors										
Regular smoker W1	0.18	0.38	8242	0.17	0.37	3901	0.19	0.39	4341	-0.021**
Got drunk during the past year W1	0.29	0.45	8275	0.29	0.46	3917	0.29	0.45	4358	0.006
Ever tried marijuana W1	0.28	0.45	8216	0.30	0.46	3878	0.26	0.44	4338	0.033***
Ever tried other illegal drugs W1	0.13	0.33	8224	0.12	0.33	3881	0.13	0.34	4343	-0.007
Panel C: Behavioral outcomes										
Paint graffiti or signs	0.12	0.45	8257	0.14	0.50	3908	0.09	0.38	4349	0.054***
Deliberately damage property	0.24	0.58	8254	0.34	0.69	3908	0.14	0.41	4346	0.200***
Lie parents or guardians	0.87	1.03	8241	0.82	1.01	3902	0.93	1.04	4339	-0.108***
Shoplift	0.37	0.78	8249	0.42	0.83	3899	0.31	0.72	4350	0.107***
Physical fight	0.45	0.76	8246	0.59	0.85	3899	0.30	0.63	4347	0.288***
Hurt someone badly	0.24	0.58	8248	0.35	0.69	3899	0.12	0.40	4349	0.228***
Run away from home	0.11	0.39	8261	0.09	0.37	3909	0.12	0.41	4352	-0.026***
Drive a car without permission	0.13	0.46	8261	0.15	0.51	3909	0.11	0.42	4352	0.039***
Steal more than 50 dollars	0.07	0.35	8257	0.09	0.40	3906	0.05	0.28	4351	0.040***
Go into a house to steal	0.07	0.34	8256	0.08	0.38	3903	0.05	0.30	4353	0.033***
Threaten to use a weapon to get something	0.05	0.29	8256	0.08	0.34	3905	0.03	0.21	4351	0.046***
Sell marijuana or other drugs	0.14	0.55	8249	0.20	0.64	3900	0.08	0.42	4349	0.112***
Steal less than 50 dollars	0.32	0.74	8247	0.38	0.79	3898	0.26	0.68	4349	0.118***
Take part in a group fight	0.25	0.59	8254	0.30	0.64	3905	0.20	0.52	4349	0.096***
Act loud, rowdy, or unruly in a public place	0.72	0.90	8252	0.73	0.92	3905	0.71	0.88	4347	0.022
Delinquency scale	4.13	4.98	8233	4.74	5.51	3891	3.49	4.28	4342	1.252***

[1] Notes: Summary statistics for our estimation sample.

[2] Variable means are weighted using Add Health sample weights.

Table B3: Identifying Variation

	(1)	(2)	(3)
	All	Males	Females
Panel A: School x Grade residual variation			
ADHD PGS rank	0.31	0.31	0.31
ADHD PGS gendered-rank	0.33	0.33	0.33
Panel B: School x Grade + ADHD PGS cubic polynomial residual variation			
ADHD PGS rank	0.07	0.07	0.07
ADHD PGS gendered-rank	0.13	0.14	0.13

[1] The table reports the standard deviation of the residuals of regressions of rank on the controls and fixed effects listed in the heading of each panel. [2] Estimates are weighted using Add Health sample weights.

Table B4: Robustness Test: Different Functional forms for the ADHD PGS Polynomial by Gender

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Professional ADHD diag.	Linear	2nd order	3rd order	4th order	5th order	6th order	7th order
Panel A: All							
ADHD PGS gendered-rank	0.075***	0.075***	0.075***	0.075***	0.075***	0.075***	0.075***
	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)	(0.027)
Observations	8,291	8,291	8,291	8,291	8,291	8,291	8,291
R-squared	0.117	0.117	0.118	0.118	0.118	0.118	0.118
Panel B: Males							
ADHD PGS gendered-rank	0.132***	0.133***	0.133***	0.134***	0.134***	0.134***	0.134***
	(0.048)	(0.048)	(0.048)	(0.048)	(0.048)	(0.048)	(0.048)
Observations	3,927	3,927	3,927	3,927	3,927	3,927	3,927
R-squared	0.208	0.208	0.208	0.209	0.209	0.209	0.209
Panel C: Females							
ADHD PGS gendered-rank	0.061	0.061	0.060	0.059	0.059	0.060	0.060
	(0.042)	(0.042)	(0.042)	(0.042)	(0.042)	(0.042)	(0.042)
Observations	4,364	4,364	4,364	4,364	4,364	4,364	4,364
R-squared	0.149	0.149	0.150	0.150	0.151	0.151	0.151

[1] We replicate the specification in Column (1) of Table 5.

[2] Estimates are weighted using Add Health sample weights. [3] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B5: Robustness Test: Inclusion of School-specific Cubic ADHD PGS Polynomials

	(1)	(2)	(3)
Professional ADHD diag.	All	Males	Females
ADHD PGS gendered-rank	0.068**	0.146**	0.081
	(0.028)	(0.058)	(0.049)
Observations	8,291	3,927	4,364
R-squared	0.183	0.347	0.262
Own ADHD PGS cubic	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes
ADHD PGS school-grade mean	No	No	No
ADHD PGS school-grade variance	No	No	No
Further school-grade means	No	No	No
School and Grade FE	No	No	No
School x Grade FE	Yes	Yes	Yes

[1] We replicate Table 5 but we now include school-specific cubic ADHD PGS polynomials for all, males and females. [2] Estimates are weighted using Add Health sample weights. [3] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B6: Robustness Test: Allowing for a Non-Linear and Heterogeneous Structure for ADHD PGS Peer Effects

	(1)	(2)	(3)
Professional ADHD diag.	All	Males	Females
ADHD PGS gendered-rank	0.076***	0.133***	0.059
	(0.026)	(0.048)	(0.039)
Observations	8,291	3,927	4,364
R-squared	0.118	0.209	0.151
Own ADHD PGS cubic	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes
ADHD PGS school-grade mean	No	No	No
ADHD PGS school-grade variance	No	No	No
Further school-grade means	No	No	No
School and Grade FE	No	No	No
School x Grade FE	Yes	Yes	Yes

[1] We replicate Table 5 but we now include interaction terms between the leave-me-out school grade mean and standard deviation of the ADHD PGS distribution and the cubic ADHD PGS polynomial. [2] Estimates are weighted using Add Health sample weights. [3] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B7: Robustness Test: Other Rank Definitions

	(1)	(2)	(3)
Professional ADHD diag.	All	Males	Females
Panel A: Ties are assigned the average rank			
ADHD PGS gendered-rank	0.073***	0.131***	0.062
	(0.028)	(0.049)	(0.043)
Observations	8,291	3,927	4,364
R-squared	0.118	0.208	0.150
Panel B: Randomly break ties			
ADHD PGS gendered-rank	0.072***	0.126***	0.059
	(0.027)	(0.047)	(0.042)
Observations	8,291	3,927	4,364
R-squared	0.118	0.208	0.151
Panel C: Ties are assigned the maximum value			
ADHD PGS gendered-rank	0.067**	0.124**	0.062
	(0.028)	(0.048)	(0.043)
Observations	8,291	3,927	4,364
R-squared	0.118	0.208	0.150
Own ADHD PGS cubic	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes
ADHD PGS school-grade mean	No	No	No
ADHD PGS school-grade variance	No	No	No
Further school-grade means	No	No	No
School and grade FE	No	No	No
School x Grade FE	Yes	Yes	Yes

[1] Estimates are weighted using Add Health sample weights.

[2] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B8: Robustness test: Controlling for Ability Rank

	(1)	(2)
Professional ADHD diag.		
Panel A: All		
ADHD PGS rank	0.087***	0.078***
	(0.028)	(0.027)
R-squared	0.124	0.119
Observations	7,920	8,291
Panel B: Male		
ADHD PGS gendered-rank	0.131**	0.138***
	(0.052)	(0.048)
R-squared	0.223	0.211
Observations	3,741	3,927
Panel C: Female		
ADHD PGS gendered-rank	0.069*	0.060
	(0.042)	(0.042)
R-squared	0.157	0.152
Observations	4,179	4,364
PPVT rank	Yes	No
Own PPVT cubic	Yes	No
EA PGS rank	No	Yes
Own EA PGS cubic	No	Yes

[1] Estimates are weighted using Add Health sampling weights.

[2] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B9: Robustness Test: Age at ADHD Diagnosis

	(1)	(2)	(3)
Professional ADHD diag.	All	Males	Females
Panel A: Drop if diagnosed before age 5			
ADHD PGS gendered-rank	0.071***	0.131***	0.049
	(0.027)	(0.048)	(0.042)
Observations	8,282	3,920	4,362
R-squared	0.119	0.211	0.151
Panel B: Drop if diagnosed before age 9			
ADHD PGS gendered-rank	0.044*	0.102**	-0.010
	(0.023)	(0.046)	(0.033)
Observations	8,157	3,833	4,324
R-squared	0.112	0.199	0.154
Own ADHD PGS cubic	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes
ADHD PGS school-grade mean	No	No	No
ADHD PGS school-grade variance	No	No	No
Further school-grade means	No	No	No
School and grade FE	No	No	No
School x Grade FE	Yes	Yes	Yes

[1] Estimates are weighted using Add Health sample weights.

[2] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B10: Robustness Test: Sample Restricted to Students 0.4 Years Around the Mean Age of Their School-grade

	(1)	(2)	(3)
Professional ADHD diag.	All	Male	Female
ADHD PGS gendered-rank	0.097**	0.158*	0.125
	(0.045)	(0.088)	(0.107)
Observations	3,593	1,768	1,825
R-squared	0.245	0.402	0.336
Own ADHD PGS cubic	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes
ADHD PGS school-grade mean	No	No	No
ADHD PGS school-grade variance	No	No	No
Further school-grade means	No	No	No
School and Grade FE	No	No	No
School x Grade FE	Yes	Yes	Yes

[1] Estimates are weighted using Add Health sample weights.

[2] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B11: Robustness Test: Sample selection

	(1)	(2)
Weights:	Ex-ante attrition probabilities	Retention rates
Panel A: All		
ADHD PGS gendered-rank	0.071**	0.071***
	(0.028)	(0.027)
Observations	8,291	8,291
R-squared	0.137	0.112
Panel B: Males		
ADHD PGS gendered-rank	0.092*	0.129***
	(0.051)	(0.046)
Observations	3,927	3,927
R-squared	0.250	0.197
Panel C: Females		
ADHD PGS gendered-rank	0.085	0.065
	(0.052)	(0.043)
Observations	4,364	4,364
R-squared	0.164	0.143
Own ADHD PGS cubic	Yes	Yes
Individual controls	Yes	Yes
ADHD PGS school-grade mean	No	No
ADHD PGS school-grade variance	No	No
Further school-grade means	No	No
School and Grade FE	No	No
School x Grade FE	Yes	Yes

[1] Estimates are weighted using a combination of the Add Health sampling weights and ex-ante attrition probabilities or retention rates.

[2] Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.