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Marco Bertoni; Blas A. Marin-Lopez and Anna Sanz-de-Galdeano

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Subjective Gender-Based Patterns in ADHD Diagnosis*

Marco Bertoni† Blas A. Marin-Lopez‡ Anna Sanz-de-Galdeano§

Abstract

The increasing incidence of ADHD diagnosis and its uneven prevalence across demographic groups has sparked debates about misdiagnosis. We use data on individuals' genetic predisposition to ADHD from the Add Health survey of 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 school peers 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 for boys relative to girls.

JEL: I10, I21, J24, J13.

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

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†University of Padova and IZA. Email: marco.bertoni@unipd.it. Bertoni acknowledges funding from a SID grant from the Department of Economics and Management - University of Padova.

‡University of Alicante. Email: blasangel.marin@gmail.com. Marín-López acknowledges financial support from Project PID2021-124237NB-I00 (financed by MCIN/ AEI /10.13039/501100011033/ and by FEDER Una manera de hacer Europa)

§University of Alicante and IZA. Email: anna.sanzdegaldeano@gmail.com. Sanz-de-Galdeano acknowledges financial support from Project PID2021-124237NB-I00 (financed by MCIN/ AEI /10.13039/501100011033/ and by FEDER Una manera de hacer Europa) and from Generalitat Valenciana, Consellería de Innovación, Universidades, Ciencia y Sociedad Digital through project Prometeo CIPROM/2021/068.

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 commonly diagnosed mental health condition, along with anxiety, among 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]

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 by region, gender, and income.[5] In addition, the prevalence of ADHD in the U.S. has been increasing since the late 1990s,[6] along

[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 childhood ADHD lowers the socioeconomic status of parents by reducing their labor supply (and earnings) and 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).

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

A key concern is that diagnosis may be based on relative comparisons of symptoms among peers, rather than an assessment of symptoms against absolute benchmarks.

This issue has received attention because of a growing body of evidence that children who are relatively young for their grade level are more likely to be diagnosed and treated than their older peers. This literature compares students born a few days apart but on two opposite sides of a school entry cut-off date. Despite being of virtually the same age, the rate of ADHD diagnosis is much higher for early starters –who are the youngest in their grade– than for late starters, who end up being the oldest in their grade.[10] Because ADHD is an underlying neurological problem, its preva-

[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 observed 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 reveal 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 biological. The measured response may be functional and physiological, biochemical at the cellular level, or a molecular interaction" (WHO, 1993). Biomarkers include 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 finds no effect of late birth date 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.

lence should not be altered by variations in the relative age for grade of students of the same age caused by discontinuities in school entry cut-off dates. The literature has interpreted these results as evidence of subjective standards in ADHD diagnosis whereby teachers and parents misinterpret the lower maturity of relatively younger students as ADHD symptoms and, as a result, diagnose and treat them for ADHD.

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 susceptibility to ADHD (as measured by the ADHD Polygenic Score –hereafter ADHD PGS–, a summary indicator that proxies for 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.

The rationale behind our strategy is that a higher ADHD PGS rank may increase symptoms' salience and the likelihood of diagnosis, and yet the same student may not be diagnosed in another school grade in which his/her rank is relatively low.

We find robust evidence that relative standards are relevant in ADHD diagnosis, and are 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, attending the same grade, 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 the distribution of genetic predisposition to ADHD, the diagnosis 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.[11] Our study is particularly close to a growing 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 Paffenholz (2023) shows that increasing high school students' ordinal socioeconomic rank within their school and cohort leads to a reduction in depression scores and improved cognitive ability, and that these effects have long lasting consequences for adult depression and college attendance and completion. 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 in 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 in this literature.[12]

Finally, we contribute to an emerging body of work exploiting the increasing

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

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

availability of genetic data in multidisciplinary surveys to study metagenomic effects outside the family, that is, how individuals are affected by the (unobservable) genetic makeup of other individuals in their social network, beyond family members (Domingue et al., 2018; 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, Section 5 reports some internal validity tests, and Section 6 provides a wide range of robustness checks. Finally, Section 7 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 (CPC, 2018). 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.[13] 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, 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 is based 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 health

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

professional (see Section 2.3.2). Saliva samples for DNA extraction were also collected at Wave IV for the in-home sample.[14] 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, mostly 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 subsequent ADHD diagnosis, these indicators are observed by parents and teachers, which in turn may 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 allows us to observe individuals as well as their grade-level peers, thereby allowing us to exploit 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 based on their genetic predisposition to ADHD. This is also important for our identification strategy because genes are fixed at conception and can influence an individual's likelihood of developing ADHD and thus manifesting its symptoms. 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 19,865 students with valid information on gender, school, and grade identifiers. Next, because information on individuals' ADHD diagnosis and genotype is collected in Wave IV, we further restrict the sample to 15,085 individuals who participated in both Waves I and IV. In addition, we are forced to retain only 8,782 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.[15] Our sample further shrinks to 8,412 students once we drop individuals with

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

[15] 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.

invalid sample weights. 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 who do not have at least two boys and two girls, further reducing the number of observations to 8,181 students. The final working sample, after retaining students with no missing values on other characteristics included in the analysis, is 8,179 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 individual (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 number of individual-level characteristics measured at Wave I across the full sample (that is, as big as possible depending on the amount of non missing information for each of the characteristics considered) and our estimation sample. The ADHD diagnosis rate, gender, age, nationality, proportion of students born in the US, proportion of students living with both parents, and parental age are comparable between the two samples. However, the final estimation sample has a higher proportion of White students and lower proportions of Black, Hispanic, and Asian (other backgrounds being the omitted category), and a higher level of the Peabody Picture Vocabulary Test (PPVT), a measure often used as a proxy for academic ability. In Section 6, 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		Male		Female	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Panel A: Outcome and rank variables						
Professional ADHD diagnosis W4	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 measured at Wave 1						
Female	0.49	0.50	0.00	0.00	1.00	0.00
Age in September 1994	15.09	1.76	15.18	1.77	14.99	1.74
Born in the US	0.96	0.19	0.96	0.19	0.96	0.19
White	0.73	0.44	0.72	0.45	0.74	0.44
Black	0.15	0.36	0.15	0.35	0.15	0.36
Hispanic	0.08	0.26	0.08	0.27	0.07	0.26
Asian	0.03	0.16	0.03	0.17	0.02	0.14
Panel C: Family and parental characteristics measured at Wave 1						
Living with both parents	0.72	0.45	0.72	0.45	0.72	0.45
Parental age	41.32	6.28	41.41	6.36	41.23	6.19
Socio-economic status index	0.02	0.96	0.00	0.95	0.04	0.96
Panel D: Characteristics of school-by-grade peers						
Share of females	0.51	0.07	0.52	0.07	0.50	0.07
Average age in September 1994	15.08	1.65	15.09	1.65	15.08	1.65
Share born in the US	0.95	0.09	0.95	0.09	0.95	0.09
Share of whites	0.64	0.31	0.64	0.31	0.65	0.31
Share of blacks	0.20	0.26	0.20	0.26	0.20	0.26
Share of hispanics	0.09	0.16	0.10	0.16	0.09	0.15
Share of asians	0.03	0.08	0.03	0.08	0.04	0.08
Share living with both parents	0.70	0.14	0.70	0.14	0.70	0.14
Average parental age	41.47	2.04	41.46	2.05	41.49	2.03
Average SES index	0.02	0.45	0.01	0.45	0.03	0.45
School-by-grade sample size	24.83	27.97	24.73 27.73	24.93	28.22	
Observations	8,179		3,868		4,311	

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, out of a total of 18 listed, 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.[16]

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

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).[17] Consistent with previous findings (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%) as 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 the gender gap in disruptive behavior is known to affect 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 suspensions and expulsions, are higher for boys than for girls in our data.

In addition, Sciutto et al. (2004) show that teachers refer boys for treatment more often than girls, especially when faced with symptoms of hyperactivity, 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 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,

[17] 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).

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 approximately 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.[18] In Add Health, the PGS are calculated according to the procedure described in Dudbridge (2013). Specifically, the raw ADHD PGS for an individual i is calculated as:[19]

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

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.[20] 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

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

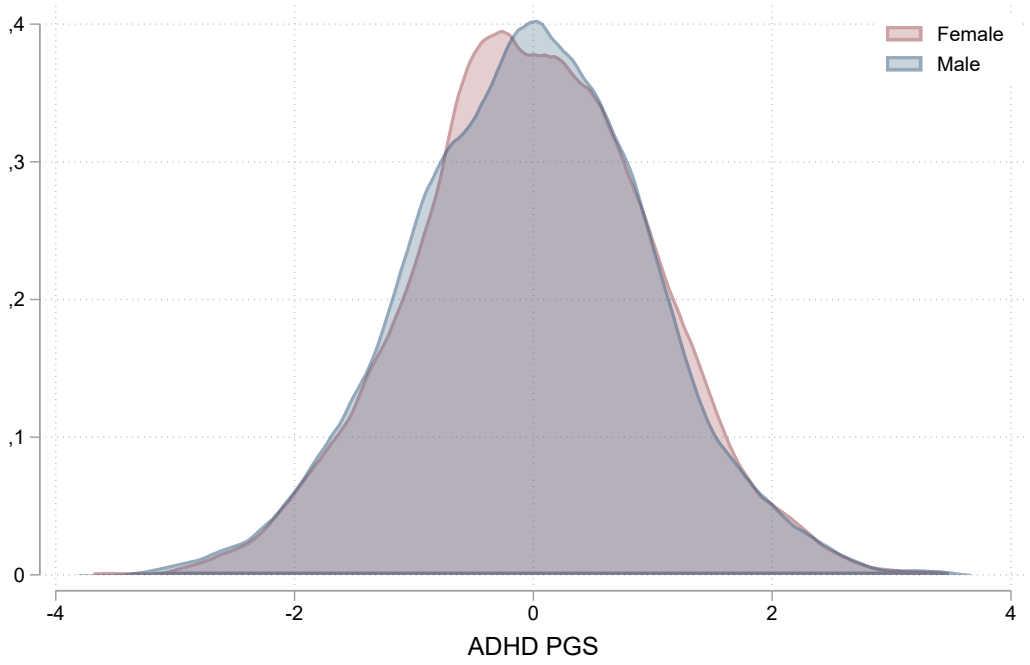
[19] See the Add Health documentation (https://addhealth.cpc.unc.edu/wp-content/uploads/docs/user_guides/WaveIVPGSRelease2UserGuide.pdf) for details on the construction of the polygenic indices in this dataset.

[20] 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.

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).

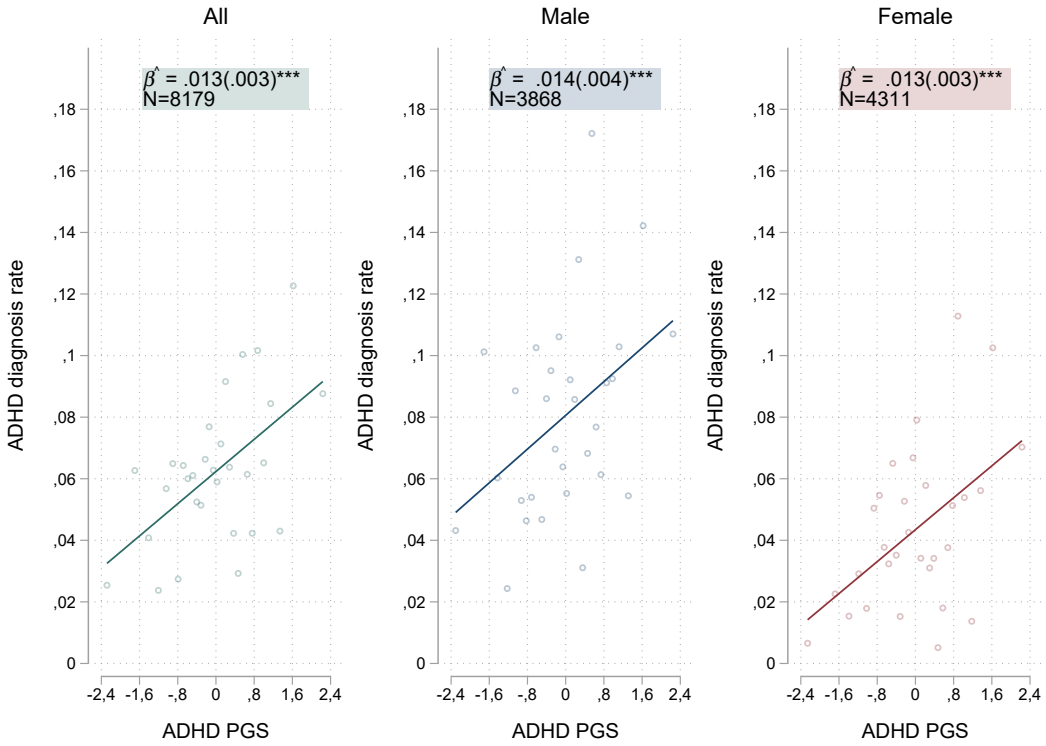
Figure 1 plots the kernel-smoothed densities of Add Health respondents' ADHD PGS, separately by gender. The distributions are approximately normal and do not vary significantly by gender. This indicates that the previously documented higher prevalence of ADHD in males is unlikely to be due to gender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

Figure 1: ADHD PGS Distribution by Gender



Next, in Figure 2 we show that the ADHD PGS is indeed positively associated with the odds 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 1.4 percentage points for males and 1.3 percentage points for 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).

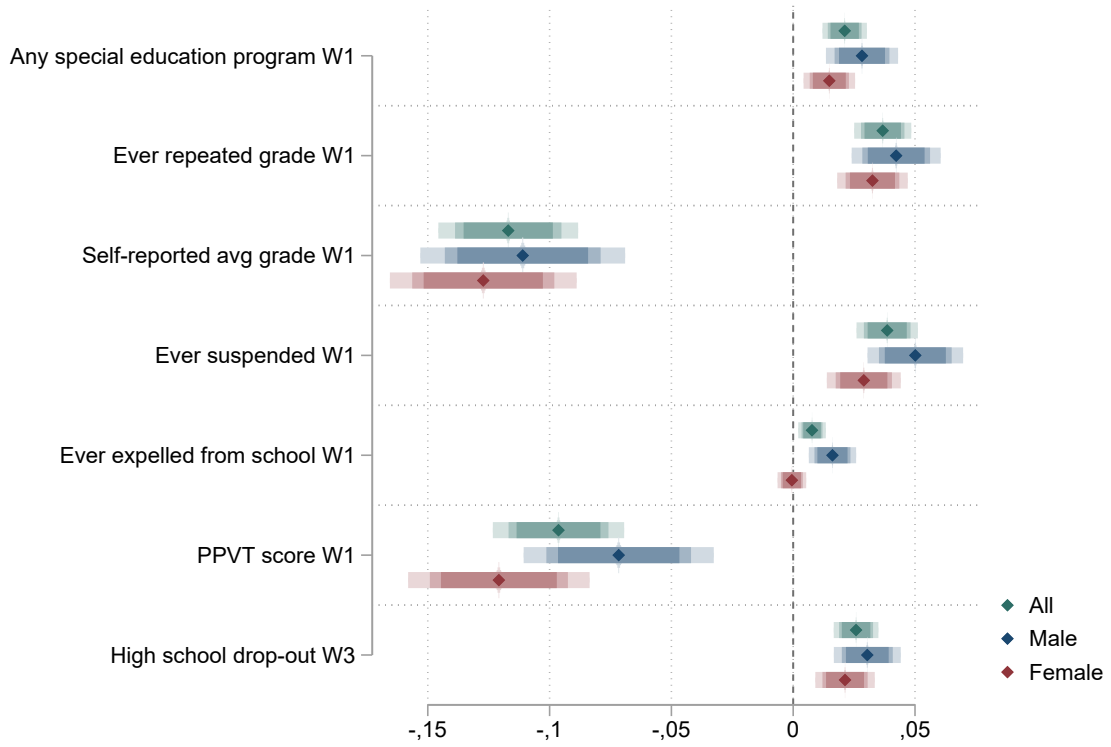
Figure 2: ADHD Polygenic Scores and ADHD Diagnosis



Notes: each graph reports the probability of being diagnosed with ADHD estimated in twenty even-spaced bins of the support of the ADHD PGS. The fitted values of a linear OLS regression of the probability of being diagnosed with ADHD on the ADHD PGS is also graphed, while the ADHD PGS coefficient estimate and its associated standard error are reported at the top of each panel. The ADHD PGS is standardized to have a mean of 0 and a standard deviation of 1.

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. This evidence validates our use of the ADHD PGS as a proxy for manifesting ADHD symptoms that is pre-determined with respect to group assignment.

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. As for the confidence intervals: the darkest, medium-dark, and lightest areas correspond to 90%, 95% and 99% confidence levels, respectively. The ADHD PGS is standardized to have a mean of 0 and a standard deviation of 1.

2.4.2 Main regressor: Ordinal ADHD Polygenic Score Rank

To obtain a measure of the salience of a student's ADHD symptoms within his or her grade prior to school assignment, we calculate students' relative genetic propensity for ADHD based on their absolute rank in the school-by-grade distribution of ADHD PGS.[21] Following Murphy and Weinhardt (2020), we protect against the possibility that measurement error in the ADHD PGS increases multiplicatively farther from the mean and produces 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 a reference for computing the rank. Furthermore, because grades within schools may

[21] 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 the 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.

have different numbers of students, we then transform the absolute rank of students (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. Because 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, parents, or any other environmental factors. 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 the 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 within schools in the Add Health data.

Because 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 parametric 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 would give them more attention. We find this type of sorting implausible. Even if parents could use information about 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 results in 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 fixed effects 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 the school-grade peers' ADHD PGS distribution, as well as other observable peer characteristics such as the proportion of females and all the other school-by-grade covariates summarized in Panel D of Table 1.

We will present several tests in support of the validity of our empirical strategy in Section 5.

3.2 Estimation

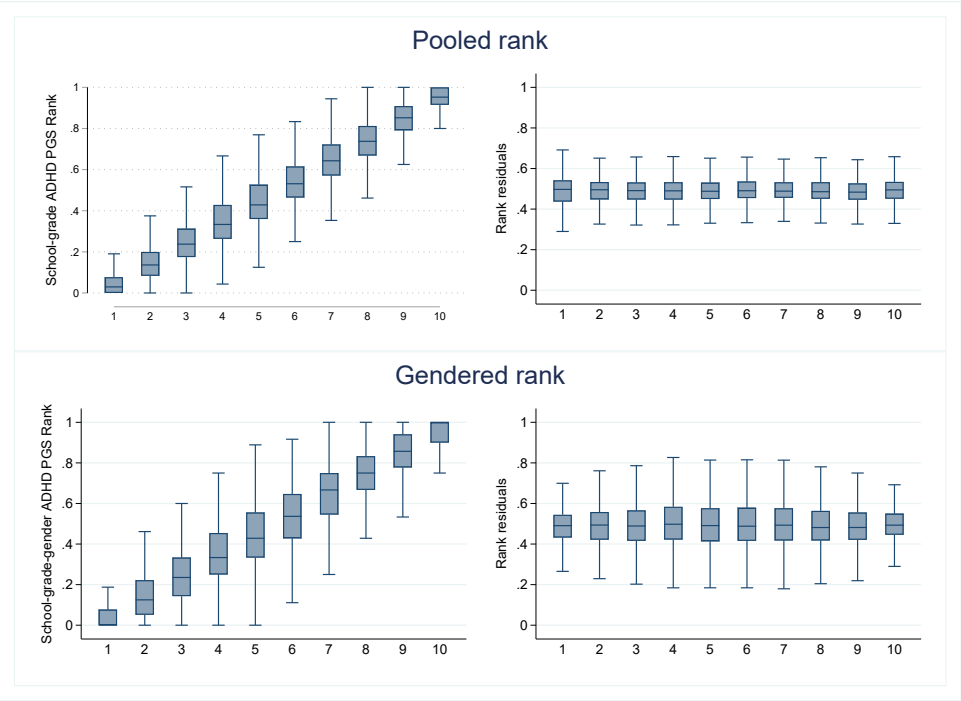
With these considerations in mind, we use Ordinary Least Squares (OLS) to estimate the following empirical model:

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

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 by Wave IV, 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 or her 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 including the following variables: gender, age in September 1994 and its square, indicators for being born in the U.S. and for race and ethnicity –White, Black, Hispanic, Asian, and other, the omitted category– 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). Moreover, ε_{isg} is an idiosyncratic error term, and we account for the dependence of the error term among students enrolled in the same school by clustering the standard errors at the school level (there are 129 schools in our final sample). While Equation (3) imposes several parametric assumptions, in Section 6 we show that our main results hold under many different functional form choices. Furthermore, as discussed in Section 1, we also estimate Equation (3) using the gender-specific ordinal genetic rank within a grade, as well as after splitting the sample by gender. In both cases, our measures of peer composition will be gender-specific. In the latter case, so will the school-by-grade fixed effects and the coefficients associated with the control variables.

Before presenting our main results, it is important to verify that the demanding set of fixed effects and controls included in Equation (3) leaves enough remaining variation in the rank (R_{isg} in Equation (3)) that can be exploited for identification. Following Paffenholz (2023), Figure 4 illustrates that there is considerable variation in the ordinal ADHD PGS rank of students in the same decile of the ADHD PGS distribution observed in the full sample, both in the observed data and when we consider the residuals of a regression of the rank on the fixed effects and controls included in Equation (3). This holds both when we compute the rank within school grade or within gender and school grade. We further report in Table B3 the standard deviation of the observed (gendered) rank and of its residuals, in the full sample as well as in gender-specific subsamples. We find that the residual variation in the rank is between 22.6 and 30.3% of the total, a non-negligible fraction.

Figure 4: Observed and residual variation in the ADHD PGS rank



Notes: the left panels of the figure report the ADHD PGS school-grade rank distribution, for each decile of the ADHD PGS distribution observed in the full sample. The right panels report the distributions for the residuals of a regression of the rank on the fixed effects and controls included in Equation (3). To ease comparison, we re-centered the residuals at the mean value of the rank observed in the full sample. In the top panels we consider the rank observed in the pooled sample, while the gendered rank is used in the bottom panels. The box whiskers represent the median (central line in the box), the 25th and 75th percentiles (lower and upper bounds of the box), and the lowest and highest adjacent values of the local genetic rank, respectively.

Finally, as we will compare the effects that we obtain when we use the pooled and gender-specific ADHD PGS ranks, Figure B1 documents the observed correlation between the two ranks. Although in the full sample the ADHD PGS distribution virtually overlaps across genders (see Figure 1), sampling variation makes it such that there is non-negligible variation in the gender-specific rank for students belonging to the same decile of the pooled rank.[22] The finding is confirmed by the R-squared of a regression of the gendered rank on the pooled rank, that is equal to 0.89 for the full sample and for females, and to 0.86 for males (values well below 1).

[22]The omission from the graph of outliers, with rank values beyond the highest or lowest adjacent values of the distribution, hides the variation in the gendered rank that is present even within the first decile of the pooled rank.

4 Main Results

Table 2 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 of the student's own ADHD PGS, the set of individual controls listed in Table 1, and the mean ADHD PGS of the student's grademates (by gender if the regression uses the gendered version of the rank to fit the specific moment of the distribution). Column (2) enriches the specification by including the variance of the ADHD PGS of the student's school-grade peers (by gender if the regression uses the gendered version of the rank to match the specific moment of the distribution) and the means of other school-grade peer characteristics.[23] Finally, Column (3) reports results from our preferred specification with school-by-grade fixed effects.

Table 2: Average Effects of ADHD PGS Ordinal Ranks on ADHD Diagnosis

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.			
Panel A			
ADHD PGS rank	0.060	0.057	0.065
	(0.045)	(0.045)	(0.044)
R-squared	0.064	0.066	0.115
Panel B			
ADHD PGS gendered-rank	0.076***	0.075***	0.075***
	(0.026)	(0.026)	(0.027)
R-squared	0.065	0.068	0.117
Observations	8,179	8,156	8,179
Own ADHD PGS cubic	Yes	Yes	Yes
Individual controls	Yes	Yes	Yes
ADHD PGS school-grade mean	Yes	Yes	No
ADHD PGS school-grade variance	No	Yes	No
School-grade means of individual controls	No	Yes	No
School and grade FE	Yes	Yes	No
School x grade FE	No	No	Yes

Notes: individual controls include gender, age, and its square, indicators for being born in the U.S. and for race and ethnicity –White, Black, Hispanic, Asian, and other, the omitted category–, 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. School-grade means of individual characteristics exclude the principal components. Estimates are weighted using Add Health sample weights. 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 6.5 per-

[23] The 23 observations lost in Column (2) belong to school-grade samples that are too small to estimate the leave-me-out school-by-grade standard deviation of the ADHD PGS distribution.

centage points, depending on the specification. A clearer picture emerges in Panel B, where we report the effects of the 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 7.5 to 7.6 percentage points, depending on the specification.[24] This result implies that a 1SD increase in the 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 3 we replicate the results in Column (3) of Table 2 –our preferred specification– after splitting the sample by gender. Panel A confirms the presence of a positive but not statistically significant effect of the school-grade ADHD PGS rank computed after pooling males and females, both in the pooled sample and within gender. The evidence in Panel B reveals instead that the significant effect of the within-gender ADHD PGS rank found 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 testing levels for males ($p - value < 0.01$), but not for females ($p - value > 0.1$). The magnitude of the estimated effect of interest is almost twice as large for males (13.2 percentage points) than for females (6.8 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 0.16.[25] When we measure these effects in relative terms, we find that they are quantitatively relevant for both males and females. However, the effect for females is far from reaching standard levels of statistical significance. In particular, a 1SD increase in the within-gender rank for males increases the likelihood of ADHD diagnosis by 4.4 percentage points, or 54.5% of the mean diagnosis rate for males in the sample, while for females the effect is 2.2 percentage points, or 55% of the mean diagnosis rate for females.

We can thus summarize our main findings as follows: the 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 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,

[24] We obtain a slightly larger average effect of 10.4 percentage points (std. err. = 3.3 percentage points) in Column (3) of Table 2 when we allow the controls and school-by-grade fixed effects to have gender-specific coefficients.

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

gender, and their own ADHD PGS levels.

Table 3: Average Effects of ADHD PGS Ordinal Gendered-Rank on ADHD Diagnosis by Gender

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.	All	Male	Female
Panel A			
ADHD PGS rank	0.065	0.070	0.088
	(0.044)	(0.072)	(0.060)
R-squared	0.115	0.205	0.152
Panel B			
ADHD PGS gendered-rank	0.075***	0.132***	0.068
	(0.027)	(0.048)	(0.044)
R-squared	0.117	0.207	0.152
Observations	8,179	3,868	4,311
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
School-grade means of individual controls	No	No	No
School and grade FE	No	No	No
School x grade FE	Yes	Yes	Yes

Notes: individual controls include gender, age and its square, indicators for being born in the U.S. and for race and ethnicity –White, Black, Hispanic, Asian, and other, the omitted category–, 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. School-grade means of individual characteristics exclude the principal components. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5 Internal validity tests

This section provides some tests to assess the empirical plausibility of our identifying assumptions.

First, 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 regress each of the available controls in the vector X_{isg} on the ADHD PGS rank, the cubic polynomial in the ADHD PGS level, the first ten principal components of all genotypes measured in the SNP data matrix and the school-by-grade fixed effects, and check whether or not the estimated rank effects are statistically different from zero. Table 4 reports the results for the within-gender and school-grade rank, while the results for the within-school-grade rank are in Table B4 in Appendix B. Both tables report results for the full sample

and for the gender subsamples. Reassuringly, most of the estimated coefficients are very close to zero in magnitude, and the extensive set of characteristics we test are jointly balanced in the full sample and in the female and male subsamples.

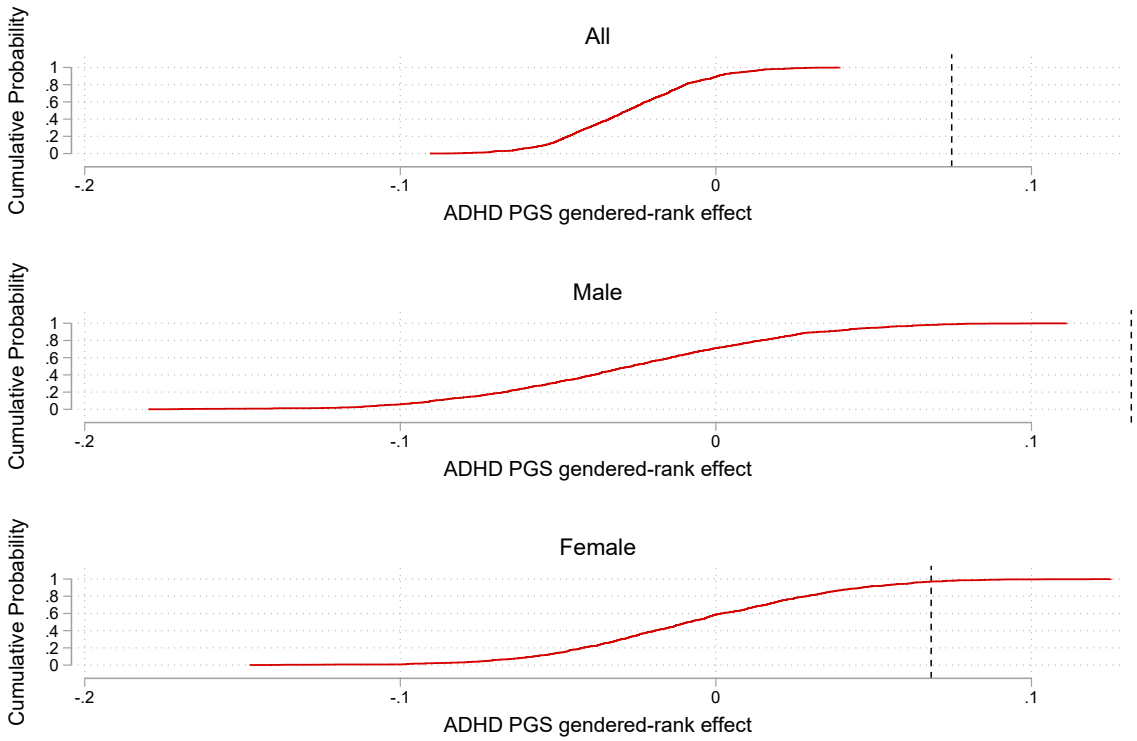
Table 4: Balancing tests: ADHD PGS gendered-rank

	All		Male		Female	
	(1)	(2)	(3)	(4)	(5)	(6)
Dep.Var.:	Coeff.	St. Error	Coeff.	St. Error	Coeff.	St. Error
Female	-0.020	0.101	0.000	.	0.000	.
Age in September 1994	0.033	0.065	0.021	0.102	-0.044	0.102
Born in the US	0.010	0.015	0.028	0.023	0.004	0.031
White	0.037	0.033	-0.075	0.063	0.112	0.068
Black	-0.011	0.022	0.038	0.038	-0.127***	0.043
Hispanic	-0.014	0.027	0.006	0.046	0.012	0.052
Asian	0.001	0.015	-0.015	0.027	0.011	0.022
Living with both parents	0.070	0.050	-0.011	0.092	0.162**	0.078
Respondent parent's age	-0.050	0.642	0.931	1.065	-0.929	1.183
Socio-economic Status	0.036	0.098	0.065	0.155	0.031	0.161
Joint significance ($p-value$)	0.88		0.71		0.19	

Notes: each cell reports the ADHD PGS rank coefficient or standard error from an OLS regression of the covariate listed in the first column on rank, a cubic specification of the ADHD PGS, school-by-grade fixed effects and the first ten principal components of all genotypes measured in the SNP data matrix. Estimates are weighted using Add Health sample weights. Joint significance tests are obtained after jointly estimating the models for all outcomes. Standard errors clustered at the school level * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Second, we also show that two falsification tests based on permutations support the validity of our design. Figure 5 reports the CDF of the empirical distribution of the ADHD PGS gendered-rank effects that we obtain for all students, males, and females, after randomly permuting 1000 times the ADHD PGS level across the students of a given gender in a school-grade, computing the resulting "placebo" ADHD PGS-gendered rank, and estimating Equation (3). The results indicate that randomly drawn ADHD PGS gendered-ranks are very unlikely to generate treatment effects of the size of the ones estimated in the real data, depicted by the vertical line in each panel and reported in Table 3. This evidence suggests that the ADHD PGS rank is not picking up spurious ranks of students within classes that are unrelated to the ADHD PGS rank –and that could manifest even when random ADHD PGS ranks are used. Comparable results are reported in Figure B2 in Appendix B for the case in which we instead randomly permute students' grade within schools and genders, thereby constructing fictitious peer groups.

Figure 5: Falsification test - ADHD PGS permutations within school grades and gender



Notes: the figure reports the CDF of empirical the distribution of the ADHD PGS gendered rank effects estimated using equation Equation (3) after 1000 permutations of the ADHD PGS of same-gender students within school-grades. The vertical line indicates the effect estimated in the observed data (see Table 3).

6 Robustness Tests

In this section, we provide a series of sensitivity tests that confirm the stability of our results. We will focus on the results in Table 3 and assess their robustness along several dimensions, such as the set of individual controls included in the model, functional form choices, alternative rank definitions, timing of ADHD diagnosis, strategic timing of school entry, grade retention, sample selection, and panel attrition. We now describe each set of tests in turn, and report the results in Appendix B.

6.1 Dropping individual level controls

Table B5 reports the estimates that we obtain when we exclude the vector of individual-level controls from the model and only retain the ADHD PGS cubic polynomial, the principal components of all genotypes measured in the SNP data matrix, and the

school-by-grade fixed effects. Results are virtually identical to our baseline ones, reported in Table 3, which is in line with the evidence on covariate balance reported in Table 4.

6.2 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 B6 we assess whether the cubic specification is appropriate by using PGS_{isg} polynomials of different orders, from the first to the seventh, and by nonparametrically controlling for dummies for deciles of the PGS_{isg} distribution. We find results that are remarkably stable in both the full sample and the gender subsamples. Moreover, Table B7 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 proposed by Booij et al. (2017) and include all the interaction terms between a linear (instead of cubic) ADHD PGS trend, the leave-me-out mean, and the leave-me-out standard deviation of its distribution within each school grade. As illustrated by Bertoni and Nisticò (2023) and Denning et al. (2021), this specification effectively controls for nonlinear 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. Table B8 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 quintile of the ADHD PGS distribution has the same effect on ADHD diagnosis as moving from the second to the third quintile (see Gill et al., 2019). We assess the plausibility of this assumption by replacing the linear functional form for the rank with a non-linear one that includes dummies for quintiles of the within-gender-by-school-grade ADHD PGS distribution, taking the third quintile as the omitted reference category. The results –reported in Figure B3– fully support the more parsimonious linear specification used as our benchmark (Table 3).[26]

[26] We also assessed whether the rank effect is heterogeneous depending on students' own ADHD PGS level

6.3 Alternative Rank Definitions

When defining the 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, we show in Table B9 that our results are robust to breaking ties using the average rank, a random order, or the maximum rank.

6.4 Other Ranks

A potential concern with our strategy is that the ADHD PGS rank may actually capture the effect of the ADHD PGS ordinal rank along with the effects of other individual characteristics that are correlated with ADHD PGS and not included in our model. Given the evidence of ability rank effects on individual educational attainment, risky behaviors, 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 address this concern, we augment our baseline specification with a cubic polynomial in students' PPVT test scores –the ability proxy used in the aforementioned studies– and the corresponding ordinal rank. The estimated effects of the ADHD PGS rank obtained from this richer specification are reported in Panel A of Table B10 and are again comparable to our benchmark results.[27] Still, one could argue that since the PPVT is measured after birth, the outcome of the test could be influenced by the genetic predisposition of individuals to ADHD. If this were the case, then the PPVT score would be a "bad" control, and impaired cognitive ability should be considered as a mechanism behind the effect of ADHD PGS rank on ADHD diagnosis. For this robustness test, we followed the existing studies and treated PPVT as a valid measure of ability. In Panel B, however, we report the results obtained when we proxy ability instead with the PGS for educational attainment available in Add Health and fixed at conception.[28] Our results are unchanged.

Finally, given the abundant evidence of relative age effects in ADHD diagnosis (see Section 1), we also checked whether the ADHD PGS rank effects persist once we control for the within school-grade age rank. The results in Panel C of Table B10 confirm that this is the case.

and on the standard deviation of the ADHD PGS distribution in school-grades, but we did not find significant evidence of heterogeneous effects along these dimensions.

[27] The different number of observations with respect to Table 3 is due to missing values in the PPVT score.

[28] In particular, the educational attainment PGS we use is calculated using the estimated weights from the GWAS conducted by Lee et al. (2018).

6.5 Timing of ADHD diagnosis

As noted in Section 2.3.1, our dependent variable measures whether Add Health respondents had ever been diagnosed with ADHD by Wave IV. Figure B4 reports the distribution of age at diagnosis of ADHD by gender, and shows that –especially among males– a significant proportion of cases are diagnosed before students meet their middle/secondary school peers. We first check that our results are not picking up a spurious effect of cases diagnosed before school entry age –which we set at 5– or even before middle/secondary school age –which we set at 9– by replicating our benchmark estimations (Table 3) after removing from the sample those students who had already been diagnosed with ADHD before these ages. The results in Panels A and B of Table B11 are comparable to our benchmark.[29] Finally, we also assess that our uncovered effects are not entirely driven by cases of ADHD diagnosed after high school, when subjective peer comparisons take place. We do this by re-estimating our baseline models after redefining our dependent variable as a dummy equal to 1 if students received a diagnosis by age 18 and equal to 0 if they received it later or never, based on information available through Wave IV. The results in Panel C are again comparable to our baseline. If anything, the gender gap shrinks somewhat in this specification.

6.6 Strategic Timing of School Entry and grade retention

Another assumption behind our identification strategy is that there is no sorting of students according to the expected composition of the school-grade. Parents could potentially manipulate their children's placement by delaying school entry conditional on their choice of a particular school. While we consider this type of sorting to be unlikely, to assess whether our results are robust to potential concerns about strategic timing of school entry, 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 a full grade, thereby eliminating students who may be late entrants as well as grade repeaters. The results in Table B12 are again comparable to our benchmark.

Grade retention may also be a concern for our strategy, as students in our data were first interviewed in Add Health Wave I when they were in different grades. A consequence of this sampling scheme is that, for those first interviewed in later grades, we observe grade composition only after a potentially endogenous process of grade retention may have taken place. Figure B5 reports the set of ADHD PGS

[29] A potential concern with the robustness check in which we drop individuals diagnosed before age 9 is that, if there is little mixing of students between primary and secondary schools, there may be some overlap of students in primary and middle/secondary schools. As a result, we do not overemphasize the relevance of this exercise.

gendered-rank effect estimates and associated 99, 95, and 90 percent confidence intervals that we obtain when we retain in the estimation sample only students who were enrolled in progressively lower grades at Wave I. The dashed line shows the estimate that includes all students up to grade 12. While the precision of the estimates decreases as we drop students, our main results are qualitatively unchanged.

As an alternative, following Bertoni et al. (2020) and Rodriguez-Planas et al. (2022) we also change our definition of school peers and use school-by-birth cohort groups instead of school-by-grade ones. Note, however, that while birth cohorts do not reflect school starting age choices or grade retention, the relevance of this peer group may be lower for interpersonal comparisons. We then re-compute the rank using this peer group definition, and replace the school-by-grade dummies with school-by-cohort ones. Results obtained using this alternative strategy are reported in Panel A of Table B13 and are qualitatively similar to those from our preferred specification, although somewhat smaller in magnitude. In this regard, note that the sampling scheme adopted by Add Health selected students attending schools at a given point in time. As a consequence, we only observe early-starters (who are already in high school) in the youngest cohort observed in the data, and either late starters or grade-repeaters (who have not yet left high school) in the oldest ones. To sidestep this issue, we replicate our analyses after keeping only students born between 1977 and 1981, for whom we observe samples that are as complete as possible. Our results –reported in Panel B of Table B13– are similar to our benchmark even in this case. Overall, the results in this subsection suggest that school starting age choices and grade retention do not severely threaten the validity of our research design.

6.7 Sample Selection Criteria

Another potential 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 dropped 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 explained in Section 2, the combination of these two factors leads us to work with a sample of 8,412 students out of the 20,745 who were present in Wave I. We then lose some more observations due to the unavailability of valid sample weights and the peer-group size requirement our research question requires, which leads to a final sample size of 8.179 students.

This reduction in sample size 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 students' genetic predisposition to 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.[30]

We assess the impact of these potential problems on our estimates by relying on two reweighting strategies. First, in Panel A of Table B14 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), and interviewer fixed effects, which serve as exclusion restrictions to identify the selection equation. There are 568 interviewer in the final sample, but we lose 182 observations in the estimation due to lack of variation in the attrition probability within 80 interviewers. Interviewer fixed effects are jointly significant in the selection equation for all, male, and female student, with $p - values < 0.01$. 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 in line with the baseline estimates reported in Table 3.

Second, in Column (2) of Table B14, we address the issue of measurement error in the rank due to missing data on the ADHD PGS by calculating 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-grade groups where 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 rank measurement error caused by sample selection by comparing the rank constructed in the full and selected samples for variables that are observed for all students in Wave I. 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 I sample and in our final estimation sample is very high (0.97).

[30] Add Health's random sampling of students within school cohorts also introduces measurement error. According to the simulations carried out by Elsner and Isphording (2017), this source of measurement error attenuates the estimates of rank effects.

7 Conclusion

ADHD diagnoses and prescriptions for ADHD medication have grown dramatically in recent decades. These large increases have raised concerns about the subjective nature of ADHD diagnosis, a debate further fueled by the fact that ADHD 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 of 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 susceptibility to ADHD in his or her school-grade affects the likelihood of being diagnosed.[31]

We find that a one standard deviation increase in students' ordinal rank in the genetic predisposition to ADHD among their same-gender peers is associated with a 2.4 percentage point increase in the odds of being diagnosed with ADHD, holding both students' age and their own genetic predisposition to ADHD constant. This effect is both statistically significant and large, accounting for 40% 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 in Denmark between children who are relatively older and younger than their classmates, which is an exception to the general pattern, may be related to the comparatively 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 re-

[31] We are aware that ranking individuals on the basis of their position in a PGS distribution is subject to uncertainty, an issue that has recently been thoroughly analyzed by Muslimova et al. (2023), who show that measurement error in the PGS is an important cause of the lack of rank concordance across different polygenic scores, while also providing insightful applications to cardiovascular disease and educational attainment. It is important to clarify, however, that our research design is aimed at providing evidence (or lack of evidence) of relative norms in ADHD diagnosis, but is by no means suitable for implementing individualized interventions and/or individualized medication recommendations in clinical settings, which is beyond the scope of this paper.

sponsible for the diagnosis and subsequent initiation of pharmacological treatment. Therefore, the consideration of age-for-grade (Persson et al., 2021),[32] as well as promoting specialists' 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.[33]

Finally, 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.

[32] Persson et al. (2021) document that the younger siblings and cousins of age-for-grade induced marginally diagnosed children are also more likely to be diagnosed with and treated for ADHD in Sweden. In light of this evidence, they recommend that physicians adjust the screening protocol so that it attaches less weight to information about a diagnosis of an older family member when he or she is relatively young-for-grade.

[33] See https://ur1.app/h2qU7 for an independent evaluation of the Focus ADHD program and the use of the QbTest conducted by the Institute of Mental Health.

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Appendices - For Online Publication

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).
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.

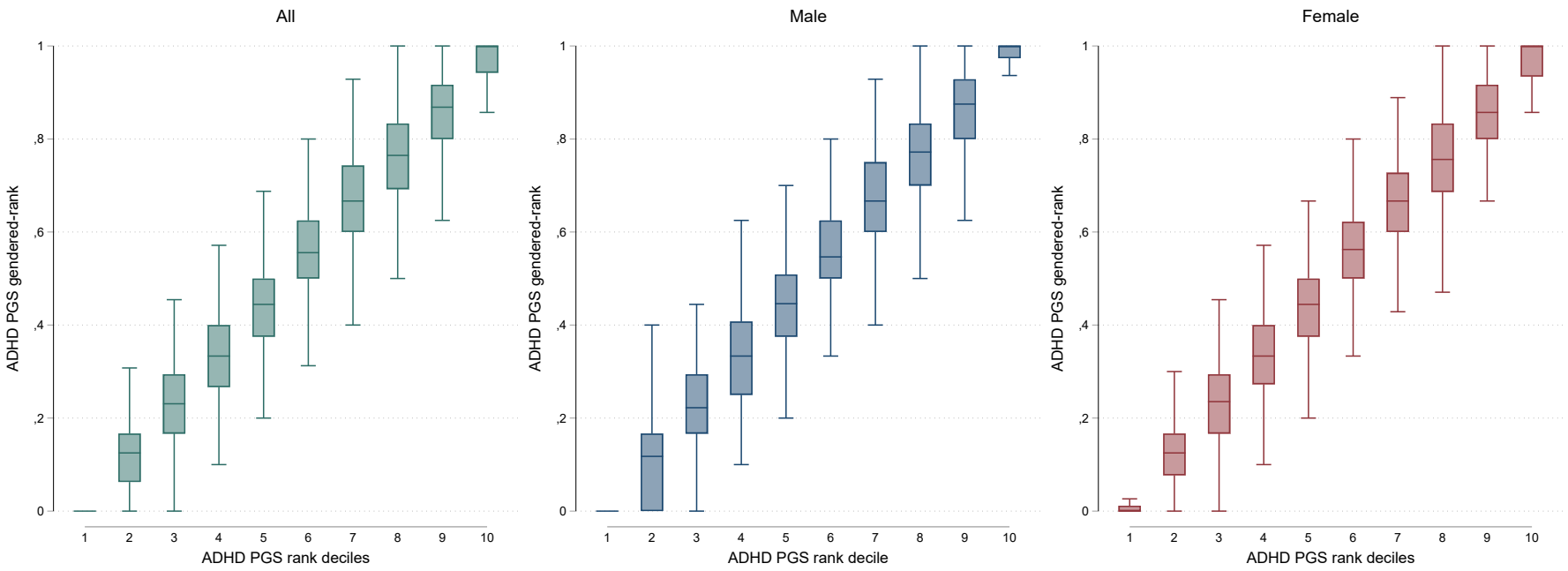
Impulsivity

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/.

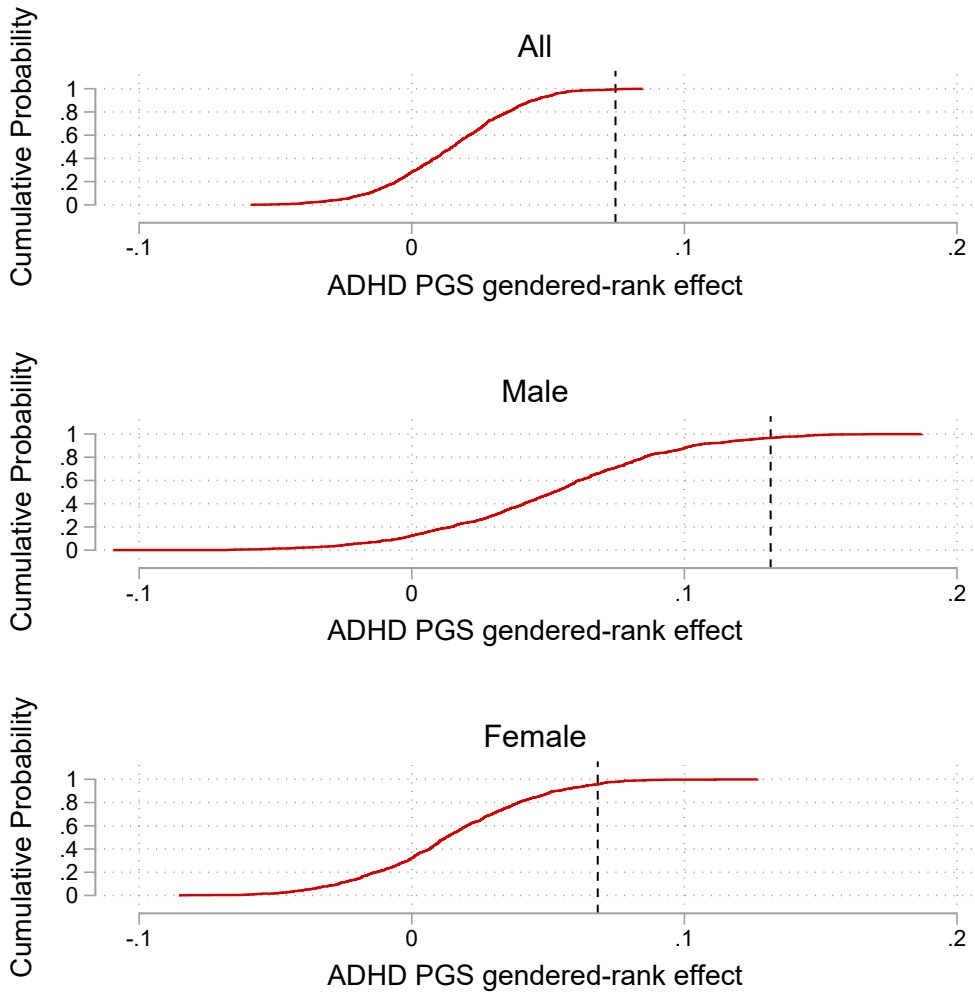
Appendix B Additional Figures and Tables

Figure B1: Correlation between the gendered and pooled ADHD PGS rank



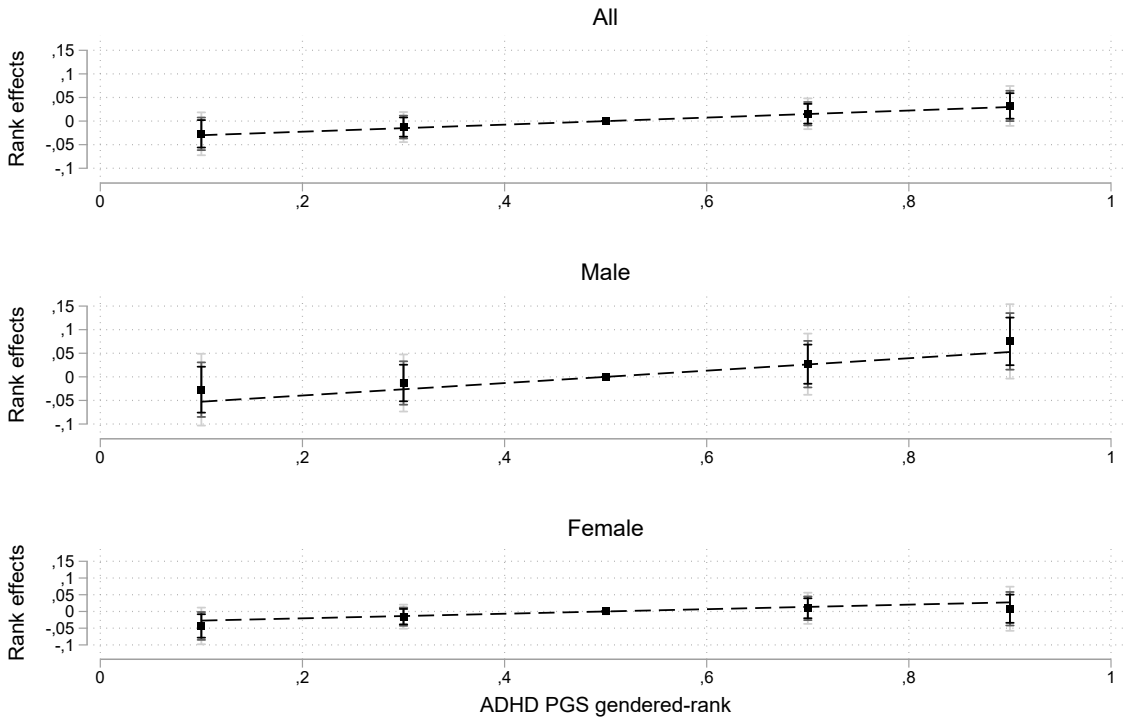
Notes: the panels of the figure report the ADHD PGS school-grade-gender rank distribution, for each decile of the ADHD PGS school-grade distribution observed in the full and in gender-specifc samples. The box whiskers represent the median (central line in the box), the 25th and 75th percentiles (lower and upper bounds of the box), and the lowest and highest adjacent values of the local genetic rank, respectively.

Figure B2: Falsification Test - Grade Permutations within Schools and Gender



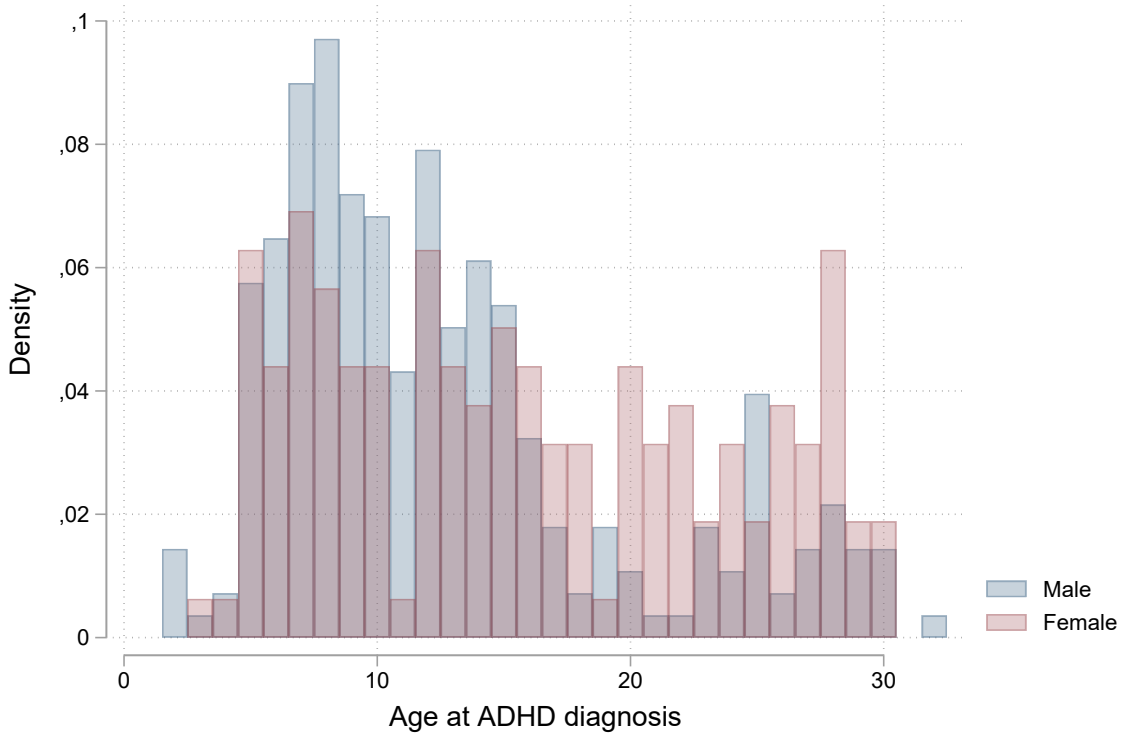
Notes: the figure reports the CDF of the empirical distribution of the ADHD PGS gendered rank effects estimated using equation Equation (3) after 1000 permutations of the grades of same-gender students within schools. The vertical line indicates the effect estimated in the observed data (see Table 3).

Figure B3: Nonlinear ADHD PGS Gendered-rank by Gender.



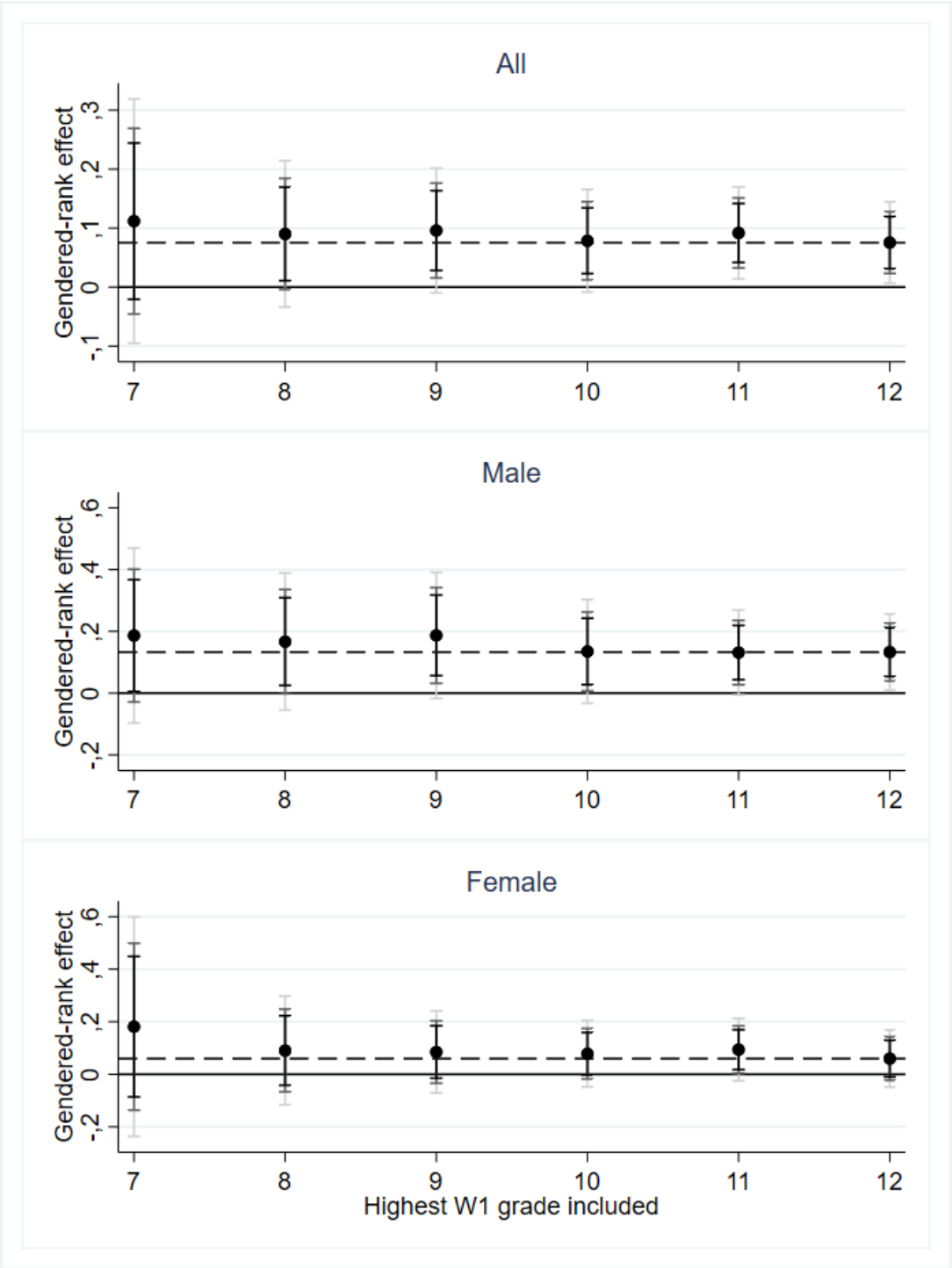
Notes: the figure reports the estimated effects of being in quintile 1, 2, 4, or 5 vs. the omitted quintile 3 of the ADHD PGS gendered rank distribution, and their corresponding 99, 95 and 90 confidence intervals. The estimate of the specification with a linear rank effect –as in Equation (3)– is also reported. The p-values for joint equality of the rank quintile effects are equal to 0.07, 0.06, and 0.29 for all, male and female students, respectively.

Figure B4: Age at ADHD Diagnosis - by gender



Notes: the sample includes students in our estimation sample who report that they have received a professional ADHD diagnosis by Wave 4.

Figure B5: ADHD PGS Gendered-rank Effects - Retaining in the estimation sample only students enrolled in progressively lower grades at Wave I.



Notes: the horizontal line indicates the effects estimated including all grades. The number of observations are 1,157, 2,296, 3,799, 5,401, 6,885 and 8,791 when we retain all students up to grades 7, 8, 9, 10, 11, and 12, respectively. Analogous figures for males only are equal to 514, 1,064, 1,777, 2,550, 3,282, and 3,868. For females, they are equal to 643, 1,232, 2,022, 2,851, 3,603, and 4,311.

Table B1: Comparison of Full and Estimation Sample Characteristics.

	Full sample			Estimation sample		
	(1)	(2)	(3)	(4)	(5)	(6)
	Mean	Std.Dev.	Obs.	Mean	Std.Dev.	Obs.
Professional ADHD diagnosis W4	0.06	0.23	14478	0.06	0.24	8179
Female	0.49	0.50	18456	0.49	0.50	8179
Age in September 1994	15.12	1.77	18456	15.09	1.76	8179
Born in the US	0.94	0.25	18456	0.96	0.19	8179
White	0.65	0.48	18456	0.73	0.44	8179
Black	0.16	0.37	18456	0.15	0.36	8179
Hispanic	0.12	0.32	18456	0.08	0.26	8179
Asian	0.04	0.19	18456	0.03	0.16	8179
Living with both parents	0.71	0.46	18456	0.72	0.45	8179
Parental age	41.42	6.40	18456	41.32	6.28	8179
Socio-economic status index	0.00	1.00	18456	0.02	0.96	8179
PPVT score W1	0.00	1.00	17581	0.09	0.93	7816

Notes: summary statistics for the full and estimation samples. Outcome means are weighted using Add Health sample weights.

Table B2: Cognitive Outcomes and Externalizing Behaviors by Gender

	All			Male			Female		
	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs
Any special education program W1	0.10	0.30	7184	0.13	0.34	3414	0.07	0.25	3770
Ever repeated grade W1	0.22	0.41	8173	0.27	0.44	3863	0.16	0.37	4310
Self-reported avg grade W1	-0.01	1.01	8106	-0.15	1.02	3825	0.14	0.98	4281
Ever suspended W1	0.27	0.44	8176	0.35	0.48	3865	0.18	0.39	4311
Ever expelled from school W1	0.04	0.20	8176	0.06	0.24	3865	0.02	0.15	4311
PPVT score W1	0.09	0.93	7816	0.12	0.93	3686	0.05	0.94	4130
High school drop-out W3	0.12	0.32	8179	0.13	0.33	3868	0.11	0.31	4311

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

Table B3: Identifying Variation

	(1)	(2)	(3)
	All	Male	Female
Panel A: Standard deviation in the raw data			
ADHD PGS rank	0.31	0.31	0.31
ADHD PGS gendered-rank	0.33	0.33	0.33
Panel B: standard deviation of residuals after including school x grade FE, ADHD PGS cubic, individual controls			
ADHD PGS rank	0.07	0.07	0.07
ADHD PGS gendered-rank	0.13	0.11	0.10

Notes: the table reports the standard deviation of the ADHD PGS rank or gendered rank observed in the raw data (Panel A) and the standard deviation of the residuals of the regression of the same variables on the set of controls and fixed effects listed in the Panel heading (Panel B). Estimates are weighted using Add Health sample weights.

Table B4: Balancing tests: ADHD PGS rank

	All		Male		Female	
	(1)	(2)	(3)	(4)	(5)	(6)
Dep.Var.:	Coeff.	St. Error	Coeff.	St. Error	Coeff.	St. Error
Female	-0.143	0.091	0.000	.	0.000	.
Age in September 1994	0.038	0.110	-0.025	0.167	0.104	0.154
Born in the US	0.011	0.037	0.040	0.051	-0.033	0.055
White	0.079	0.071	0.033	0.107	0.164*	0.095
Black	-0.078*	0.043	0.005	0.062	-0.134**	0.066
Hispanic	-0.016	0.040	-0.031	0.060	-0.020	0.065
Asian	0.007	0.025	-0.020	0.030	0.013	0.034
Living with both parents	0.024	0.088	-0.097	0.139	0.121	0.134
Respondent parent's age	0.483	1.190	1.848	1.849	-0.771	1.798
Socio-economic Status	0.209	0.165	0.220	0.244	0.230	0.227
Joint significance ($p-value$)	0.60		0.90		0.30	

Notes: each cell reports the ADHD PGS rank coefficient or standard error from an OLS regression of the covariate listed in the first column on rank, a cubic specification of the ADHD PGS, school-by-grade fixed effects and the first ten principal components of all genotypes measured in the SNP data matrix. Estimates are weighted using Add Health sample weights. Joint significance tests are obtained after jointly estimating the models for all outcomes. Standard errors clustered at the school level * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B5: Robustness Test: Excluding Individual Controls

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.	All	Male	Female
ADHD PGS gendered-rank	0.077***	0.131***	0.068
	(0.028)	(0.050)	(0.044)
Observations	8,179	3,868	4,311
R-squared	0.101	0.197	0.144

Notes: we replicate Table 3 but excluding individual-level controls with the exception of the principal components of all genotypes measured in the SNP data matrix. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B6: Robustness Test: Different Functional Forms for the ADHD PGS

Dep.Var.: Professional ADHD diag.	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
ADHD PGS functional form	Linear	2nd order	3rd order	4th order	5th order	6th order	7th order	decile dummies
Panel A: All								
ADHD PGS gendered-rank	0.075***	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)	(0.025)
Observations	8,179	8,179	8,179	8,179	8,179	8,179	8,179	8,179
R-squared	0.116	0.116	0.117	0.117	0.117	0.117	0.117	0.117
Panel B: Male								
ADHD PGS gendered-rank	0.131***	0.131***	0.132***	0.133***	0.133***	0.133***	0.133***	0.127***
	(0.048)	(0.048)	(0.048)	(0.048)	(0.048)	(0.048)	(0.048)	(0.046)
Observations	3,868	3,868	3,868	3,868	3,868	3,868	3,868	3,868
R-squared	0.207	0.207	0.207	0.207	0.207	0.208	0.208	0.209
Panel C: Female								
ADHD PGS gendered-rank	0.070	0.070	0.068	0.068	0.068	0.069	0.069	0.075*
	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)	(0.040)
Observations	4,311	4,311	4,311	4,311	4,311	4,311	4,311	4,311
R-squared	0.150	0.150	0.152	0.152	0.152	0.152	0.153	0.153

Notes: we replicate Table 3 but using different functional forms for the ADHD PGS. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B7: Robustness Test: Inclusion of School-specific Cubic ADHD PGS Polynomials

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.	All	Male	Female
ADHD PGS gendered-rank	0.066**	0.143**	0.088
	(0.029)	(0.059)	(0.053)
Observations	8,179	3,868	4,311
R-squared	0.183	0.349	0.264

Notes: we replicate Table 3 but including school-specific cubic ADHD PGS polynomials for all, males and females. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B8: Robustness Test: Allowing for a Non-Linear and Heterogeneous Structure for ADHD PGS Peer Effects

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.	All	Male	Female
ADHD PGS gendered-rank	0.068**	0.110**	0.073
	(0.028)	(0.052)	(0.048)
Observations	8,156	3,854	4,302
R-squared	0.117	0.209	0.152

Notes: we replicate Table 3 but include all the interaction terms between a linear (instead of cubic) ADHD PGS trend, the leave-me-out mean, and the leave-me-out standard deviation of its distribution within each school-grade. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B9: Robustness Test: Other Rank Definitions

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.	All	Male	Female
Panel A: Ties are assigned the average rank			
ADHD PGS gendered-rank	0.072***	0.128***	0.064
	(0.027)	(0.049)	(0.045)
Observations	8,179	3,868	4,311
R-squared	0.116	0.207	0.152
Panel B: Randomly break ties			
ADHD PGS gendered-rank	0.068**	0.124**	0.056
	(0.027)	(0.048)	(0.043)
Observations	8,179	3,868	4,311
R-squared	0.116	0.207	0.152
Panel C: Ties are assigned the maximum value			
ADHD PGS gendered-rank	0.067**	0.121**	0.057
	(0.027)	(0.048)	(0.044)
Observations	8,179	3,868	4,311
R-squared	0.116	0.207	0.152

Notes: we replicate Table 3 but changing the way we break ties. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B10: Robustness test: Controlling for Ability and Age Rank

	(1)	(2)	(3)
Dep. Var: Professional ADHD diag.	All	Male	Female
Panel A: Including PPVT rank			
ADHD PGS gendered-rank	0.086***	0.127**	0.078*
	(0.029)	(0.052)	(0.043)
Observations	7,816	3,686	4,130
R-squared	0.122	0.223	0.159
Panel B: Including Education PGS rank			
ADHD PGS gendered-rank	0.076***	0.134***	0.069
	(0.027)	(0.048)	(0.044)
Observations	8,179	3,868	4,311
R-squared	0.117	0.209	0.154
Panel C: Including age rank			
ADHD PGS gendered-rank	0.074***	0.133***	0.069
	(0.027)	(0.049)	(0.044)
Observations	8,179	3,868	4,311
R-squared	0.118	0.208	0.152

Notes: we replicate Table 3 but include the additional within school-grade rank variables indicated in the title of each Panel. Each regression also contains the own cubic polynomial of the variables used to construct the ranks indicated in each Panel. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B11: Robustness Test: Age at ADHD Diagnosis

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.	All	Male	Female
Panel A: Drop if diagnosed before age 5			
ADHD PGS gendered-rank	0.070**	0.129***	0.057
	(0.027)	(0.048)	(0.044)
Observations	8,170	3,861	4,309
R-squared	0.118	0.210	0.153
Panel B: Drop if diagnosed before age 9			
ADHD PGS gendered-rank	0.045*	0.104**	-0.005
	(0.024)	(0.046)	(0.035)
Observations	8,047	3,775	4,272
R-squared	0.109	0.197	0.155
Panel C: Dep.var.: diagnosed *by age 18*			
ADHD PGS gendered-rank	0.075***	0.105**	0.088**
	(0.024)	(0.048)	(0.036)
Observations	8,179	3,868	4,311
R-squared	0.132	0.231	0.163

Notes: we replicate Table 3 after changing the estimation sample or the definition of the dependent variable. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B12: Robustness Test: Sample Restricted to Students 0.4 Years Around the Mean Age of Their School-grade

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.	All	Male	Female
ADHD PGS gendered-rank	0.109**	0.208**	0.170
	(0.044)	(0.090)	(0.105)
Observations	3,499	1,710	1,789
R-squared	0.240	0.405	0.341

Notes: we replicate Table 3 after restricting the estimation sample as described in the Table title. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B13: Robustness Test: Defining School Peers using School-by-Birth Cohorts instead of School-by-Grade Groups

	(1)	(2)	(3)
Dep.Var.: Professional ADHD diag.	All	Male	Female
Panel A: All birth cohorts			
ADHD PGS gendered-rank	0.034	0.099*	0.007
	(0.029)	(0.051)	(0.042)
Observations	7,994	3,783	4,211
R-squared	0.120	0.229	0.173
Panel B: 1977-1981 cohorts only			
ADHD PGS gendered-rank	0.032	0.104*	-0.008
	(0.034)	(0.059)	(0.044)
Observations	7,007	3,330	3,677
R-squared	0.118	0.212	0.167

Notes: we replicate Table 3 after changing the definition of school peers from schools-by-grade groups to school-by-birth cohort ones, constructing rank at this level of aggregation and using school-by-birth cohort instead of school-by-grade dummies. Panel B only retains birth cohorts born between 1977 and 1981. Estimates are weighted using Add Health sample weights. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B14: Robustness Test: Sample Selection

	(1)	(2)	(3)
Dep. Var: Professional ADHD diag.	All	Male	Female
Panel A: Ex-ante attrition probability weights			
ADHD PGS gendered-rank	0.075**	0.145***	0.079*
	(0.030)	(0.054)	(0.047)
Observations	7,997	3,789	4,208
R-squared	0.125	0.229	0.154
Panel B: Retention rate weights			
ADHD PGS gendered-rank	0.069**	0.126***	0.068
	(0.026)	(0.046)	(0.043)
Observations	8,179	3,868	4,311
R-squared	0.111	0.195	0.144

Notes: we replicate Table 3. Estimates are weighted using a combination of the Add Health sampling weights and ex-ante attrition probabilities or retention rates. Standard errors clustered at the school level in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.