How interactions between ADHD and schools affect educational achievement: a family-based genetically sensitive study

Rosa Cheesman,¹ Espen M. Eilertsen,^{1,2} Ziada Ayorech,¹ Nicolai T. Borgen³, Ole A. Andreassen^{4,5},
Henrik Larsson^{6,7}, Henrik Zachrisson³, Fartein A. Torvik,^{1,2} Eivind Ystrom^{1,8}

¹PROMENTA Research Center, Department of Psychology, University of Oslo, Norway

²Centre for Fertility and Health, Norwegian Institute of Public Health, Norway

³Department of Special Needs Education, University of Oslo, Norway

⁴NORMENT, Division of Mental Health and Addiction, Oslo University Hospital, Oslo, Norway

⁵Institute of Clinical Medicine, University of Oslo, Oslo, Norway

⁶Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden

⁷School of Medical Sciences, Örebro University, Örebro, Sweden

⁸Department of Mental Disorders, Norwegian Institute of Public Health, Oslo, Norway

Abstract

Background: Children with ADHD tend to achieve less than their peers in school. It is unknown whether schools moderate this ADHD deficit. Selection into schools poses a methodological problem.

Methods: We linked data on ADHD symptoms of inattention and hyperactivity and parent-child ADHD polygenic scores (PGS) from the Norwegian Mother, Father, and Child Cohort Study (MoBa) to standardised test results and school identifiers. Using multilevel models, we estimated interactions of school effects with individual differences between students in inattention, hyperactivity, and ADHD-PGS. In our PGS analyses, we ruled out selection by adjusting for parental ADHD-PGS (a within-family PGS design). We then tested whether five measures of the social backgrounds of students at the schools explained any interactions.

Results: Analysis of up to 23,598 students attending 2,579 schools revealed interactions between school and ADHD effects on standardised test results. The variability between schools in the effects of inattention, hyperactivity, and within-family ADHD-PGS on achievement was 0.08, 0.07, and 0.05 SDs, respectively. For example, a one SD increase in inattention changed achievement by -0.23 SDs ($SE=0.009$) on average, but in 2.5% of schools with the weakest effects, the value was -0.07 or less. Schools contributed more to achievement differences for students with higher levels of ADHD, explaining more than four times as much variance in achievement for children with high versus average inattention symptoms. School sociodemographic measures could not explain the ADHD-by-school interactions.

Conclusion: Associations between ADHD and achievement are context dependent. Children with elevated ADHD symptoms and genetic risk perform better in some schools than others. Future research should identify specific school factors that support these students, potentially using the within-family gene-environment interaction approach introduced here.

Introduction

Complex outcomes such as educational achievement result from exchanges between individual characteristics and environmental contexts such as families, schools, and neighbourhoods, over time (Bronfenbrenner & Ceci, 1994). Although schools are an essential context for cognitive and social development, it is not well understood how school environments interact with individual differences between children in their levels of cognitive and behavioural difficulties. Research in this area could eventually inform policymakers and teachers on which school environments best support children at risk of low achievement.

Attention-deficit hyperactivity disorder (ADHD) is of particular interest in this context. Its characteristic symptoms of hyperactivity and inattention loom large at school. ADHD symptoms are common, typically appear in early childhood, and are associated with profound impairments in achievement (DuPaul et al., 2004; Massetti et al., 2008; Polderman, Boomsma, Bartels, Verhulst, & Huizink, 2010; Thapar & Rutter, 2015). Academic impairments are especially severe for inattention symptoms (Pingault et al., 2011; Salla et al., 2016) and may be partly due to general effects on everyday learning, such as spending more time off-task than peers (Kofler, Rapport, & Alderson, 2008). However, the role of schools in the relationship between ADHD and academic difficulties has received relatively little attention. School-based behavioural interventions (e.g., environmental modifications, reinforcement systems, computer-assisted instruction, and peer tutoring) improve behaviour but have modest effects on achievement (DuPaul, Weyandt, & Janusis, 2011; Jangmo et al., 2019). More research is required on school factors that directly address academic skills in ADHD (DuPaul et al., 2011). Identifying which school environments aid the achievements of students with ADHD could help to design policies and interventions.

Quantifying how much the association between ADHD and achievement varies between schools is a valuable first step towards understanding how schools influence the academic consequences of

ADHD. Addressing this question using a latent approach opens the possibility of capturing effects of all school-level factors driving differences in how much ADHD symptoms affect achievement, including unknown and unmeasured ones. This has the advantage of remaining agnostic to specific environments, which are challenging to identify and measure, and if measured, may represent mere proxies for the ‘true’ interactive school factors (Boardman, Daw, & Freese, 2013; Trejo et al., 2018). After clarifying whether school environments moderate ADHD effects on achievement (i.e., increase or decrease the effect compared to the average across schools), researchers may then identify what these environments are. If available measured school factors cannot account for the latent moderation effects, this would justify hypothesising about other specific school characteristics.

ADHD-by-school interactions are challenging to study. Apparent interaction effects could be driven by non-random selection of families with elevated ADHD symptoms into schools (i.e., parents choosing specific neighbourhoods and schools). This endogeneity problem can be resolved through within-family genetic analysis of children’s ADHD polygenic scores (PGS) adjusted for parents’ ADHD-PGS. ADHD is strongly genetically influenced (70-80% in twin studies) (Faraone & Larsson, 2019), and PGS allow genetic propensity for ADHD to be indexed on the individual level. Importantly, within-family PGS effects (child PGS conditioned on parental PGS) reflect the random segregation of alleles at conception and are uncorrelated with the social environments that parents select. Although the association between parental PGS and child outcomes may capture school selection effects (due to active gene-environment correlation (Plomin, DeFries, & Loehlin, 1977)) the within-family PGS will not. Previous work has demonstrated negative effects of the ADHD-PGS on achievement (Greven, Kovas, Willcutt, Petrill, & Plomin, 2014; Stergiakouli et al., 2017), including within families (Jangmo et al., 2021), but no research has examined between-school differences in this association.

Between-school variation in the effect of ADHD genetic risk on educational achievement would constitute a gene-environment interaction i.e., genes and social environments moderate one other

(Plomin, DeFries, & Loehlin, 1977). In both the educational achievement and ADHD literatures, empirical findings on GxEs have been mixed, with genetic influences being stronger, weaker, or invariable between environments. Studies have focused on interactions with specific measured home environments, such as family socioeconomic status (Figlio, Freese, Karbownik, & Roth, 2017; Gould, Coventry, Olson, & Byrne, 2018; Pennington et al., 2009; Tucker-Drob & Bates, 2016), family chaos (Gould et al., 2018; Wang, Deater-Deckard, Petrill, & Thompson, 2012), parental conflict and involvement (M. A. Nikolas, Klump, & Burt, 2015; M. Nikolas, Klump, & Burt, 2012). Although a few studies have examined GxEs involving schools (Hart, Soden, Johnson, Schatschneider, & Taylor, 2013; Trejo et al., 2018), these have been U.S.-based and have not considered the role of ADHD or its genetic risk.

Here, we explore how educational achievement is shaped by interactions between individual differences in ADHD and school environments. Our primary aim is to estimate total latent between-school variation in the effects of inattention, hyperactivity, and within-family ADHD-PGS on achievement. By using multilevel models, we remain agnostic to the relevant aspects of school environments. Moreover, our within-family PGS analyses rule out selection. Our dataset includes >25,000 Norwegian children's national test results, school identifiers, ADHD symptoms, and parent-offspring PGS for ADHD, from a novel linkage between genetic trio data in the Norwegian Mother, Father, and Child Cohort Study (Magnus et al., 2016) and the Norwegian National Educational Database. Norway is a useful context because the education system is highly standardised, and register data are almost complete. In sum, these data uniquely position us to investigate how genetic propensity interacts with educational contexts in explaining the association between ADHD and achievement.

Methods

The Norwegian context

Relative to other western nations, Norway is a wealthy welfare state with low unemployment and low economic inequality (Eurofound, 2017). The public sector provides various welfare services, including free compulsory education and universal health care. Education is comprehensive with a common curriculum for all students, and there is no tracking/streaming before upper secondary schooling. In elementary school, children primarily attend local schools, defined by their home address. Fewer than 4% of students attend private schools, which are schools with alternative pedagogical traditions, religious schools, or international schools. Child mental health services are almost exclusively public, and children with suspected ADHD or other mental health problems are referred by general practitioners. National guidelines for treatment of ADHD refer to adapted and special needs education as a key intervention, either prioritized before, or supplementary to psychopharmacological treatment. Local school psychology services design individual pedagogical plans based on symptoms and child functioning, not on diagnoses (Helsedirektoratet, 2021).

Sample

The Norwegian Mother, Father, and Child Cohort Study (MoBa; (Magnus et al., 2016)) is a prospective population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health. Pregnant women were recruited from across Norway from 1999 to 2009. The women consented to initial participation in 41% of the pregnancies. The total cohort includes 114,500 children, 95,200 mothers, and 75,200 fathers. To date, 98,110 individuals who are part of a trio (both parents and a child) from MoBa have been genotyped. The current study is based on version 12 of the quality-assured phenotype data files.

The present analyses were performed on a subsample of ~23,000 MoBa children with genome-wide genotype data, parental genotype data, plus administrative records of educational achievement and

school membership linked to MoBa through the Norwegian national ID number system. The administrative data are of high quality, and do not suffer from attrition (Hovde Lyngstad & Skardhamar, 2011; Røed & Raaum, 2003). Analyses were restricted to one child per family, by choosing one sibling at random. Phenotypic-only analyses were additionally limited to ~11,000 MoBa children with data for mother-rated ADHD symptoms at age 8. This lower sample size is mainly due to attrition as the symptom data were collected when children were eight years of age whereas the genotype data were collected at birth. In further analyses, we also restricted the sample to those with complete register data on parental education and income, and school sociodemographics.

Ethics

The establishment of MoBa and initial data collection was based on a license from the Norwegian Data Protection Agency and approval from The Regional Committees for Medical and Health Research Ethics. The MoBa cohort is now based on regulations related to the Norwegian Health Registry Act. The current study was approved by The Regional Committees for Medical and Health Research Ethics (project# 2017/2205).

Measures

School achievement

Standardised national test results for maths and reading at grades 5, 8, and 9, and English at grades 5 and 8 were obtained through linkage to Norway's National Education Database. Introduced in 2007, these tests are mainly used to monitor school development over time. Tests are compulsory, with 96% of all students in Norway taking them; students with special needs and those following introductory language courses may be exempt. Results are conveyed to teachers and parents but have no direct consequence for students. We residualised students' test scores for sex, current age (to capture birth cohort effects), and the exact age when they took the tests. We created 'core

achievement' measures as mean scores at each grade across available subjects (Rimfeld et al., 2018), and centred these to have mean zero, and standard deviation one. Results are presented for overall mean achievement in the main text, and for individual subjects in the Supplementary Information.

Interactions can arise spuriously due to features of outcome distributions (Domingue, Trejo, Armstrong-Carter, & Tucker-Drob, 2020) e.g., a truncated distribution of educational attainment (Trejo et al., 2018). To guard against this, we assessed the mean, variance, and skewness of test outcome distributions (overall and stratified by school performance deciles) and investigated their correlations with item response theory (IRT) scores. The IRT scores, which measure latent ability, are more normally distributed but less contextually meaningful than the main standardised test outcomes returned to teachers.

School codes

We matched children's achievement results to the schools they attended when they took each test. School identifiers were obtained from the National Education Database.

School sociodemographics

To complement the latent analyses, we tested whether specific school-level sociodemographic measures could explain interactions identified through multilevel modelling. We created sociodemographic measures by aggregating administrative data from all parents of students at each school with register data, not only MoBa participants. Measures were intended to capture both the average sociodemographic background among students within each school, and the variability of sociodemographic backgrounds of students within each school. For each school, we included five measures. The first measure was the average years of completed education of parents, converted from Norwegian Standard Classification of Education (NUS2000) categories, and measured when students were 16. The second sociodemographic indicator was the average parental pre-tax annual

income from gainful employment including self-employment but not capital income or social welfare transfers. We averaged the income of both parents across the years that children were aged 11-15, and ranked their income compared to other parents in the same birth cohort. Third and fourth, we measured socioeconomic inequality by calculating Gini coefficients in reported levels of parental education and income, respectively. Gini is a widely used single measure of inequality, and ranges from 0 to 1, with 0 indicating absolute equality and 1 indicating absolute inequality. Fifth, we calculated the proportion of children who are non-Western immigrants and/or who are the children of non-Western immigrants. We created these broad measures in the absence of more detailed school data. Notably, the measures could capture effects intrinsic to specific schools (e.g., peer effects) or broader social stratification (e.g., composition of the school catchment area). If the latter is true, then these variables could be considered additional controls for selection into schools and neighbourhoods.

We used the same measures of parental educational attainment and earned income as individual-level control variables.

ADHD symptoms

The Parent Rating Scale for Disruptive Behaviour Disorders (RS-DBD) (Silva et al., 2005) was used to measure inattention and hyperactivity symptoms when children were aged 8. Items were rated by mothers on a four-point scale (1 = never/rarely, 2 = sometimes, 3 = often, 4 = very often).

Inattention items (9 in total) included “Fails to give close attention to details or makes careless mistakes in schoolwork” and “Avoids, dislikes or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)”. Hyperactivity items (9 in total) included “Leaves seat in classroom or in other situations in which remaining seated is expected” and “Talks excessively”. We summed the items for the inattention and hyperactivity subscales. For individuals with less than 50% missing data on either scale, missing item values were imputed by the sample

mean. Remaining individuals were excluded from analyses. We then centred sum-scores to have mean zero, and standard deviation one. Inattention and hyperactivity subscales were analysed separately, due to differences in their aetiologies (M. A. Nikolas & Burt, 2010) and associations with achievement (Salla et al., 2016). These symptom measures are intended to capture population variation in ADHD as a dimensional trait, of which clinical ADHD is the extreme (Faraone & Larsson, 2019). Findings based on symptoms rather than diagnoses are more generalisable and robust to variability in diagnostic practices.

Genotype quality control

The current MoBa genomic dataset comprises imputed genetic data for 98,110 individuals (~32,000 parent-offspring trios; before quality control), derived from nine batches of participants, who make up four study cohorts. Within each batch, parent and offspring genetic data were quality controlled separately. Pre-imputation quality control criteria have been described in previous publications and are detailed in the Supplementary Information. We conducted post-imputation quality control, retaining SNPs meeting the following criteria: imputation quality score ≥ 0.8 in all batches, non-duplicated (by position or name), call rate $>98\%$, minor allele frequency $>1\%$, Hardy-Weinberg equilibrium $p < 0.001$, not associated with genotyping batch at the genome-wide level, and not causing a mendelian error. We removed individuals with the following criteria: heterozygosity outliers (F-het ± 0.2), call rate $<98\%$, reported sex mismatching SNP-based sex, duplicates (identified using PLINK's (Chang et al., 2015) `-genome` command as having $\text{pihat} \geq 0.98$, and distinguished from monozygotic twins through linkage to unique IDs in the population register, plus age, sex, and kinship information within MoBa), individuals with excessive numbers of close relatives (cryptic relatedness) and mendelian errors. To minimise environmental confounding, we identified a sub-sample of individuals with European ancestries via principal component analysis using the 1000 Genomes reference; thresholds for exclusion of outliers were based on visual inspection of a plot of principal components 1 and 2. The final numbers of individuals and SNPs passing quality control

were 93,582 and 6,797,215, respectively. Principal components of genetic ancestry were computed for all participants using PLINK's `–within` and `–pca-clusters` commands, based on an LD-pruned version of the final quality-controlled genotype data.

ADHD polygenic scores (PGS)

We generated ADHD-PGS for all individuals in MoBa who passed quality control, based on the latest genome-wide association study of 20,183 individuals of European-associated ancestry diagnosed with ADHD and 35,191 controls (Demontis et al., 2019). We used the PRSice software to calculate scores using all SNPs (i.e., p-value threshold of 1), with clumping parameters $kb=500$, $p=1$, $r^2=.25$ (Choi Shing Wan, n.d.). We excluded 175 MoBa participants who were also included in the BUPGEN and TOP cohorts contributing to the ADHD-GWAS prior to analyses. We computed mid-parental PGS by taking the average maternal and paternal PGS. ADHD-PGS for children from independent families ($N=27,582$) and mid-parental PGS ($N=25,169$; hereafter 'parental PGS') were then centred to have mean zero, and standard deviation one. In all PGS analyses, we included parental PGS as controls, such that effects of offspring PGS are *within-family direct genetic effects*. Notably, within-family designs based on siblings or adoptees rather than parent-offspring trios can also be used to distinguish direct genetic effects from parental genetic effects (Demange et al., 2020). We also included principal components (5 based on maternal data, 5 based on paternal data) to control for population stratification in the parental ADHD-PGS effects.

The main advantage of the within-family ADHD-PGS is that it effectively accounts for differential selection into schools. We checked the clustering of children in schools according to ADHD symptoms and PGS. For inattention and hyperactivity symptoms, and individual child and parental ADHD-PGS, 1-2% of the variance is at the school level, which indicates selection into schools (Supplementary Table 5). However, once parental ADHD-PGS is accounted for, 0% of the variance in child ADHD-PGS is explained by schools. The absence of clustering of child ADHD at the school level

in a within-family ADHD-PGS model implies that we can consider the sorting of students into schools in this model as random and interpret the school slopes causally. Note that the degree of clustering of genetic risk for ADHD in schools is likely to be larger than estimated here using child and parent PGS, which only explain 1.4% and 0.6% of the variance in child ADHD symptoms, respectively (effects were equal for inattention and hyperactivity subscales).

Statistical analyses

Table 1 summarises our approach to investigating ADHD-by-school interactions. In short, we estimated the effects of ADHD on standardised test results and tested whether these associations varied between schools. We performed analyses for three indices of ADHD (inattention, hyperactivity, and within-family ADHD-PGS) separately. To ensure that any between-school variability was not simply produced by chance, we formally compared fit statistics for a series of increasingly complex multilevel models (see below).

The base model (Model 1) estimated the association between achievement and ADHD (symptoms or within-family PGS). We pooled data across grades by including individual identification number as a random intercept, and time-point as a fixed effect to account for mean differences in scores across time. Time-point was coded as a continuous variable centred with 0 for grade 9, -1 for grade 8, and -4 for grade 5. Note that the grade 9 composite only includes maths and reading, whereas achievement composites for grades 5 and 8 include all three subjects.

In Model 2, we tested the degree to which achievement varied between schools, by adding a random intercept for schools.

In Model 3, we tested for ADHD-by-school interactions by adding random slopes for the effects of ADHD on achievement across schools. Variation around the average effect of ADHD (or *slope*) across schools is evidence of interaction.

In Model 4, we aimed to correct the school-specific intercepts for potential selection by adding fixed effects for individual-level parental educational attainment and income. Intercepts thus reflect school effects on achievement rather than students' social backgrounds.

In secondary analyses, we tested whether school-level sociodemographic characteristics could explain any observed school effects and ADHD-by-school interactions. Hence, we added fixed effects for school environmental measures (Model 5), and then environment-by-ADHD interaction terms (Model 6). The five school sociodemographic measures were tested jointly.

Table 1: Model-fitting approach. Note: the dependent variable was educational achievement (standardised national test results); ADHD refers to inattention symptoms, hyperactivity symptoms, or the within-family ADHD-PGS – we performed the model comparison procedure separately for these three ADHD indicators.

Model	Fixed effects	Random effects
1) Base	ADHD Grade	Individual child
2) Between-school outcome differences	ADHD Grade	Individual child School
3) ADHD-by-school interaction	ADHD Grade	Individual child School and ADHD
4) ADHD-by-school interaction, adjusted for parental socioeconomic status	ADHD Grade Parental income Parental education	Individual child School and ADHD
5) Accounting for school outcome differences	ADHD Grade Parental income Parental education School average parental income School average parental education	Individual child School and ADHD

	School inequality in parental income	
	School inequality in parental education	
	School proportion of non-western immigrants	
6) Accounting for ADHD-by-school interaction	ADHD	Individual child
	Grade	School and ADHD
	Parental income	
	Parental education	
	School average parental income	
	School average parental education	
	School inequality in parental income	
	School inequality in parental education	
	School proportion of non-western immigrants	
	School average parental income*ADHD	
	School average parental education*ADHD	
	School inequality in parental income*ADHD	
	School inequality in parental education*ADHD	
	School proportion of non-western immigrants*ADHD	

Model fitting and comparisons

Analyses were conducted in R with the lme4 package (Bates, Mächler, Bolker, & Walker, 2015).

Model comparisons are made using the anova() command to assess AIC fit statistics. AIC calculates the trade-off between model fit and model complexity with a penalty for the number of parameters.

If the model with, for instance, the random slopes (ADHD effects) across schools has a lower AIC value than that of a simpler model, this is evidence that ADHD-by-school interactions should be included for an optimal approximation of the underlying data generating processes.

Results

Descriptive statistics

Standardised test outcomes were approximately normally distributed, with no indication of skewness of <-1 or >1 , or of ceiling effects (no students gained the maximum number of points for any test; Supplementary Table 1). This held across deciles of school quality (Supplementary Figures 1-3). Additionally, test outcomes were strongly correlated with item-response theory-derived scores reflecting the latent ability distribution, from the Norwegian Education database (Supplementary Figure 4).

Multilevel models included up to 23,598 children with complete data for achievement, ADHD-PGS, parental ADHD-PGS, and school membership (2,579 unique schools). The average number of students participating in MoBa per school was 17, with a minimum of 1 and a maximum of 54. For the analyses with ADHD symptoms, the sample included 11,737 individuals, from 2,383 schools. Supplementary Table 1 shows descriptive statistics for all variables.

ADHD-by-school interactions influence educational achievement

To explore between-school differences in the effects of ADHD on achievement, we compared models as outlined in Table 1. Model fit improved when ADHD effects on achievement (ADHD measured by inattention symptoms, hyperactivity symptoms, or within-family ADHD-PGS) were allowed to vary between schools, and parental education and income were included as covariates. We now describe the results for the best-fitting model (Model 4). Model fit statistics and results are in Supplementary Tables 2 and 3, respectively.

Figure 1 shows that the effects of all ADHD indices on achievement vary considerably between schools. On average, a one standard deviation increase in inattention reduces achievement by 23% of a standard deviation ($SE = 0.9\%$), while a one standard deviation increase in hyperactivity reduces

achievement by 9% of a standard deviation ($SE = 0.9\%$). For children's within-family ADHD-PGS, a one standard deviation increase reduces achievement by 7% of a standard deviation ($SE = 1.3\%$). The standard deviations of effects between schools were 0.08, 0.07, and 0.05 for inattention, hyperactivity, and within-family ADHD-PGS, respectively. Therefore, students' inattention symptoms have an effect between $\beta = -0.39$ and $\beta = -0.07$ in 95% of schools, but effects are more extreme than this in 5% of schools (calculated as $-0.23 \pm (1.96 \times 0.08)$, since effects are normally distributed across schools). For the within-family ADHD-PGS, the impact of child ADHD-PGS on achievement is more extreme than double the school-average effect (i.e., $\beta = -0.16$) in 2.5% of the schools with the most negative slopes, while negligible for schools in the opposite tail of the distribution of effects. Between-school differences in within-family ADHD-PGS effects reflect gene-environment interaction, robust to selection.

Note that average effects of ADHD indicators between schools were similar for maths, reading, and English outcomes (Supplementary Table 4; Supplementary Figure 5).

Figure 1: School-level variation in the effect of ADHD measures on achievement.

Note: dashed lines show the average effects of ADHD measures on achievement ($\beta = -0.23, -0.09,$ and -0.07 for inattention, hyperactivity, and within-family ADHD-PGS, respectively). Normal curves show that there is variation in these associations between schools, with SDs of the distributions of effects across schools of 0.08, 0.07, and 0.05 for inattention, hyperactivity, and within-family ADHD-PGS, respectively. Results for the three ADHD variables were obtained from three separate models.

The ADHD-school interaction that we identified implies that not only do ADHD effects vary between schools, but that school effects vary according to student ADHD levels. Figure 2, based on results from Model 4, shows the fraction of variance in achievement that is explained by school differences across ADHD levels. Schools are more important for students with higher compared to average ADHD symptoms and genetic risk. At average levels of ADHD inattention symptoms, differences between schools explained less than 2% of the variance in achievement. However, differences between schools become of greater importance when comparing students with increasingly high inattention symptoms; for those with the highest levels of symptoms, schools explain 10% of the variation in achievement. Nearly the same estimates are found for hyperactivity symptoms. The pattern for ADHD-PGS is similar, only the differences are smaller. Since parental income and

education were controlled for, school effects reflect school ‘value added’, beyond the contribution of family background.

Figure 2: Variation in the effects of schools on achievement by student ADHD measures. Note: on the x-axis, 0= mean level of each ADHD indicator, and values either side represent SDs from the mean, with lines covering only the range of observed variation. Y-axis indicates the % variance explained in achievement by school, controlling for family background.

The correlation between the ADHD effect and the school effect on achievement, known as the slope-intercept correlation, was positive for all ADHD indicators ($r=0.36$, 0.38 , and 0.10 for inattention, hyperactivity, and within-family ADHD-PGS, respectively). This positive correlation means that ADHD has less of an impact on achievement in higher-achieving schools. Indeed, as ADHD effects increase between schools from strongly to weakly negative, overall test results increase. As with the school intercept, the intercept-slope correlation is interpreted for average values of ADHD variables. We found that the correlation becomes more strongly positive with increasing values of student ADHD symptoms and PGS (Supplementary Figure 6).

School sociodemographic measures do not explain the latent interactions

Our five direct measures of school sociodemographics contributed modestly to the between-school differences in achievement. Model 5 showed improved fit relative to Model 4, but the school intraclass correlation – a latent school effect on achievement – reduced merely from 2.8% to 2.4% when including the school covariates. Effect sizes were $\beta=0.01$ for school-average parental education, 0.04 for inequality in parental education, 0.05 for average parental income, -0.05 for inequality in parental income, and 0.06 for the proportion of non-Western immigrants.

None of the five school-level sociodemographic measures could explain ADHD-by-school interactions. This was reflected by reduced fit of Model 6, which included ADHD-by-covariate interactions, relative to Model 5.

Discussion

Using a unique sample of ~23,000 Norwegian children with data on standardised national test results, school identifiers, ADHD symptoms, and parent-offspring ADHD polygenic scores (PGS), we provide new insights into how achievement in this sample is shaped by the interplay between school contexts, and individuals' ADHD symptoms and genetic risk. The availability of parent-offspring ADHD-PGS allowed us to take advantage of the 'natural experiment' of random segregation of parental genetic variation at conception. Using such a within-family ADHD-PGS design is equivalent to randomising children to schools, meaning that selection is unlikely to confound our gene-environment interaction analyses.

We detected an interaction between schools and ADHD, whether ADHD was measured by symptoms of inattention, hyperactivity, or within-family ADHD-PGS. This indicates that the educational consequence of ADHD depends on which school it is expressed in. For example, a one standard deviation increase in inattention scores reduces achievement by 23% of a standard deviation on average, but in the 2.5% of schools where inattention matters the least for achievement, a one standard deviation increase in inattention scores reduces achievement by only 7% of a standard deviation or less. The interaction also indicates that school effects on achievement vary according to students' levels of ADHD symptoms or genetic risk, with larger achievement differences between schools for higher-ADHD students. For example, schools explain 2% of the variance in achievement for those with average inattention levels, versus 8% for students more than two standard deviations above average in inattention. Sociodemographic differences between schools cannot explain the latent ADHD-by-school interaction.

We found that in some schools, student achievement is strongly differentiated by ADHD, in others, not at all. Our formal model comparisons indicated that the between-school variation in ADHD

effects is greater than expected by chance. For individual differences researchers, these school differences imply that associations between ADHD and achievement should be contextualised. For policy makers and teachers, this suggests that the link between ADHD and low achievement is malleable. Although ADHD, on average, has large negative effects on test scores, this does not need to be the case. Further studies with richer data are needed to describe school contexts in which the ADHD penalty is reduced. Nevertheless, the finding of latent between-school variation provides optimism by showing that schools where ADHD effects are small do exist.

The ADHD-by-school interaction also implies that school effects vary according to individual differences in ADHD symptoms and genetic risk. For those interested in education policy, this means that effects of school contexts must be interpreted with reference to individual psychological differences. The small impact of the school on national test scores on average (<3% of the variance) corresponds well with prior evidence from Norway and the UK (Hermansen, Borgen, & Mastekaasa, 2020; von Stumm et al., 2020). However, our results suggest that average estimates of school effects conceal larger impacts for certain children at risk of poor achievement. Students with high ADHD symptoms and genetic risk may achieve differently at different schools, even taking family background into account.

The greater impact of school differences on achievement differences among students with higher ADHD symptoms and genetic risk could reflect variability between schools in support for higher needs students. Special needs education has been shown to affect marginal participants' educational outcomes (Ballis & Heath, 2021), which could mean that variation in such educational practices could contribute to the between-schools differences in ADHD effects. National regulations emphasise inclusive education, with 92% of special needs students remaining in mainstream settings (Nes, 2017). However, schools differ in whether/how funds are allocated to higher needs students, and in how 'inclusivity' is interpreted and implemented. School psychology services are locally

organized (in most cases at a municipal level) and may vary in both level of engagement and type of pedagogical interventions or adaptations they propose for the schools. Moreover, local school organizations within a municipality vary in, for example, how actively the principal facilitates adaptations of classroom practices and collaboration with school psychologists. It has been suggested that many Norwegian schools do not follow the national policy that children should receive support as early as possible for learning difficulties, and instead require them to wait for a referral and diagnosis for ADHD (Haug, 2014). Future work should identify school environments that support high ADHD risk students to perform well. If a policy goal is to reduce school-driven inequalities in achievement, efforts should focus on evening out differences in opportunities for students with high ADHD symptoms and genetic risk between schools.

We also tested whether available measures of school sociodemographics could account for the latent interactions between schools and students' ADHD symptoms and PGS. None of these factors (school-average parental education, income, inequality in education, inequality in income, and proportion of non-western immigrants) appeared to be involved. These measures are likely too broad to capture how schools moderate the effect of ADHD on achievement. Future work should use the framework introduced here to identify the total latent moderation effect by schools, and then see what fraction of this can be captured by more detailed school environmental measures.

Candidate school measures might be the placement of students with elevated ADHD traits (i.e., in the mainstream class or not), time spent outdoors, and the qualifications of teachers and psychologists to cater to children with cognitive and behavioural difficulties (Taylor, Roehrig, Soden Hensler, Connor, & Schatschneider, 2010). If salient school features that aid the achievements of students with ADHD can be identified, then these may provide useful intervention targets. Future research should not only aim to explain the interaction identified within the current methodological framework, but to validate the finding through triangulation with other methods, such as regression discontinuity designs based on policy changes (Barcellos, Carvalho, & Turley, 2018).

This study has several limitations. First, the generalisability of the findings is limited. Analyses included a subsample of MoBa participants with European ancestries, as defined by principal component analysis of the genetic data. The findings cannot be uncritically generalized to children with non-European ancestries, and replication of our findings in more diverse samples is essential. Generalisability issues also may stem from selection into the MoBa genetics sample, and attrition linked to ADHD. However, the school identifiers and standardised national test results cover the Norwegian population and are not affected by these issues. Second, our analyses underestimate the main and interactive effects of ADHD genetic risk. The latest ADHD-PGS explains only 5.5% of the variance in case-control ADHD (Demontis et al., 2017), despite the high twin heritability of 70-80% (M. A. Nikolas & Burt, 2010). Moreover, the PGS is based on mean effects of SNPs on ADHD case-status across international education systems. These might not be the SNPs with effects that are sensitive to school context in Norwegian young people. However, it is an advantage of this study that we had within-family ADHD-PGS to provide a strict test for gene-environment interaction. Compared to the measured ADHD symptoms, the ADHD-PGS is less prone to selection issues, more normally distributed, and not subject to attrition.

Conclusion

In sum, we found evidence of an interaction between students' ADHD symptoms and PGS, and their schools. This means that the performance gap associated with ADHD differs between schools, and the impact of school on achievement varies according to individual differences in ADHD. Children with elevated ADHD symptoms and PGS perform better in some schools than others. Knowing the characteristics of these environments requires future research, but this study indicates that one can expect to find them. Given the high individual and societal costs of ADHD and low educational

achievement (Du Rietz et al., 2020), the potential profitability of identifying these school qualities is great.

Key points and relevance

- What's known: ADHD impairs educational achievement, but it is unknown whether schools can influence this (and how).
- What's new: We found that schools moderate the effects of ADHD on achievement. We demonstrated this not only using ADHD symptoms but using within-family ADHD-PGS, where selection is controlled for.
- What's relevant for policy: Schools alter the impact of ADHD and create particularly strong differences in achievement among high-ADHD risk students. We need further research to discover the mechanisms behind this.
- What's relevant for science: We have developed a robust framework to investigate gene-environment interactions based on multilevel modelling of within-family data that could be used in future studies.

Acknowledgements and funding

RC and EY are supported by the Research Council of Norway (288083). HZ, EY and NTB are supported by the ERC consolidator grant EQOP 'Socioeconomic gaps in language development and school achievement: Mechanisms of inequality and opportunity' (818425). ZA is supported by a Marie Skłodowska Curie Action Individual Fellowship from the European Union (894675). FAT is supported by the Research Council of Norway (300668 and 273659). This work was partly supported by the Research Council of Norway through its Centres of Excellence funding scheme, project number 262700. The Norwegian Mother, Father and Child Cohort Study is supported by the Norwegian Ministry of Health and Care Services and the Ministry of Education and Research. We are grateful to all the participating families in Norway who take part in this on-going cohort study. We thank the Norwegian Institute of Public Health (NIPH) for generating high-quality genomic data. This research is part of the HARVEST collaboration, supported by the Research Council of Norway (#229624). We also thank the NORMENT Centre for providing genotype data, funded by the Research Council of Norway (#223273), South East Norway Health Authority and KG Jebsen Stiftelsen. We further thank the Center for Diabetes Research, the University of Bergen for providing genotype data and performing quality control and imputation of the data funded by the ERC AdG project SELECTIONPREDISPOSED, Stiftelsen Kristian Gerhard Jebsen, Trond Mohn Foundation, the Research Council of Norway, the Novo Nordisk Foundation, the University of Bergen, and the Western Norway health Authorities (Helse Vest). Henrik Larsson reports receiving grants from Shire Pharmaceuticals; personal fees from and serving as a speaker for Medice, Shire/Takeda Pharmaceuticals and Evolan Pharma AB; and sponsorship for a conference on attention-deficit/hyperactivity disorder from Shire/Takeda Pharmaceuticals and Evolan Pharma AB, all outside the submitted work.

Correspondence to

Rosa Cheesman r.c.g.cheesman@psykologi.uio.no; Eivind Ystrøm eivind.ystrom@psykologi.uio.no

Analysis code can be found at https://github.com/rosacheesman/ADHD_schools/.

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