CAMBRIDGE
UNIVERSITY PRESS

Regular Article

Family income and polygenic scores are independently but not interactively associated with cognitive performance among youth genetically similar to European reference populations

S.E. Paul[1], N.M. Elsayed[1] , S.M.C. Colbert[2], R. Bogdan[1], A.S. Hatoum[1] and D.M. Barch[1,2,3]

[1]Department of Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, USA, [2]Department of Psychiatry, Washington University School of Medicine, St. Louis, USA and [3]Department of Radiology, Washington University School of Medicine, St. Louis, USA

Abstract

Cognitive abilities are heritable and influenced by socioeconomic status (SES). It is critical to understand the association between SES and cognition beyond genetic propensity to inform potential benefits of SES-based interventions and to determine if such associations vary across (i) cognitive domains, (ii) facets of SES, and/or (iii) genetic propensity for different aspects of cognition. We examined the contributions of neighborhood socioeconomic advantage, family income, and polygenic scores (PGS) for domains of cognition (i.e., general cognitive ability, executive function, learning and memory, fluid reasoning) in a sample of children (ages 9–10; $n = 5549$) most genetically similar to reference populations from Europe. With some variability across cognitive outcomes, family income and PGS were independently significantly associated with cognitive performance. Within-sibling analyses revealed that cognitive PGS associations were predominantly driven by between-family effects suggestive of non-direct genetic mechanisms. These findings provide evidence that SES and genetic propensity to cognition have unique associations with cognitive performance in middle childhood. These results underscore the importance of environmental factors and genetic influences in the development of cognitive abilities and caution against overinterpreting associations with PGS of cognitive and educational outcomes as predominantly direct genetic effects.

Background

Childhood poverty is robustly associated with general and specific cognitive performance deficits (Clearfield & Niman, 2012; Johnson et al., 2016; Noble et al., 2015; Noble & Giebler, 2020). As early as six months old, youth raised with lower household incomes exhibit poorer performance on measures of total IQ and specific cognitive processes (e.g., executive function, language; Farah, 2017; Johnson et al., 2016; Noble et al., 2015; Noble & Giebler, 2020). An emerging literature also supports independent contributions of neighborhood socioeconomic disadvantage (e.g., a greater percentage of families living in poverty, increased unemployment, lower percentage of educational attainment at the neighborhood level) to cognitive functions and brain maturation in regions associated with cognitive ability above and beyond the contributions of household socioeconomic status (SES; Taylor et al., 2020). Greater educational attainment among children whose families received supplemental income (Akee et al., 2010; Costello et al., 2003, 2010)

and boosts in cognitive performance induced by enriched environments in non-human animal models (Sauce et al., 2018) highlight the plausibility that multiple facets of SES may have a causal impact on child cognition, and heighten the urgency of addressing the epidemic of childhood poverty (DeNavas-Walt & Proctor, 2014; Newhouse et al., 2016).

The moderate heritability of cognitive ability and SES (Tucker-Drob et al., 2013), as well as their shared genetic architecture ($rg = 0.65–0.82$ in adults; Hill, Davies, et al., 2019; Hill, Marioni, et al., 2019), has been used to argue that cognitive deficits related to childhood poverty may be partially attributable to shared genetic liability (Trzaskowski, Harlaar, et al., 2014). Evidence that adolescent cognitive ability is associated with adult neighborhood disadvantage has also been used to argue that associations between cognitive ability and neighborhood disadvantage may be genetically mediated (Ksinan & Vazsonyi, 2021). Shared genetic liability may arise from genetic inheritance that directly influences cognitive ability as well as gene-environment correlations (e.g., between genetic propensity and higher SES environments with generally more access to cognitively stimulating activities that may be more amenable to environmental intervention (Belsky et al., 2018).

Disentangling genetic and socioeconomic status associations with cognition is challenging. Twin studies may not be able to detect genetic influences on SES or the influence of SES on

cognitive development, as family-level SES is typically shared within a twin pair (Trzaskowski, Harlaar, et al., 2014). Twin studies may also modestly overestimate the effect of genetics, given that the equal environments assumption (i.e., that monozygotic twin pairs share the same environmental similarity as dizygotic twin pairs) may not be valid (Felson, 2014). Another approach is to measure genetic influence using polygenic scores (PGS) that effectively represent genome-wide genetic liability to a particular phenotype on an individual (rather than population) level (Wray et al., 2007). Relative to heritability estimates in twin studies that violate the equal environments assumption, genetic similarities detected in GWAS of largely unrelated individuals should be minimally confounded by environmental similarities after accounting for genetic similarity to reference population(s) (and thus any influences of population stratification (Friedman et al., 2021).

Emerging evidence largely based on PGS for educational attainment (EduA) and family-level SES suggests that both PGS and SES have independent effects on cognition (Corley et al., 2023; Judd et al., 2020; Merz et al., 2022; Park et al., 2023; Raffington et al., 2019). Given evidence from twin studies that SES may moderate the heritability of cognitive outcomes, such that heritability is higher in higher SES environments (i.e., the Scarr-Rowe hypothesis; Hanscombe et al., 2012; Rowe et al., 1999), recent work using PGS has also attempted to replicate this finding in the context of individual genetic propensity, with mixed evidence (Corley et al., 2023; Judd et al., 2020; Park et al., 2023; Peñaherrera-Aguirre et al., 2022; Woodley Of Menie et al., 2021).

Focusing on EduA PGS, in particular, may have limitations, given that educational attainment PGS is confounded by factors such as population stratification, assortative mating, and/or gene-environment correlation (e.g., Okbay et al., 2022). Thus, investigations that more comprehensively assess PGS and cognitive abilities are needed. Further, work in the Adolescent Brain Cognitive Development Study (ABCD) sample has shown that both family income (Tomasi & Volkow, 2021) and neighborhood socioeconomic poverty (e.g., median neighborhood income) have unique associations with neurocognitive performance in youth (Hackman et al., 2021; Taylor et al., 2020). Thus, to examine the contributions of both PGS and SES to cognition, it is critical to measure SES comprehensively, including both the familial and neighborhood levels, and to account for genetic propensity for phenotypes, other than EduA, more directly related to cognition. Recent work using the ABCD sample incorporated PGS for cognitive performance and indicators of neighborhood adversity but did not account for family relatedness (Park et al., 2023).

The present study tested the hypothesis that SES (i.e., family income, neighborhood socioeconomic resources) and genetic propensity (polygenic scores [PGS] for educational attainment, intelligence, and executive function) independently contribute to variance in four domains of cognitive ability (i.e., general cognitive ability, executive function, learning and memory, fluid intelligence) in a sample of 5,549 children (ages nine to 10) genetically similar to European reference populations. To reduce the influence of confounding from population stratification, assortative mating, and/or passive gene-environment correlation, we further decomposed PGS variance into between-family and within-family variance (i.e., within-siblings PGS analyses; (Selzam et al., 2019). With this approach, family-mean PGS and individual deviation from that family-mean PGS are both included in the model, disaggregating confounded effects (i.e., family-mean PGS) from effects that more closely approximate direct genetic effects (i.e., individual deviations from family-mean PGS; Selzam et al., 2019).

Finally, given mixed evidence that SES may moderate the heritability of cognitive outcomes (Hanscombe et al., 2012; Rowe et al., 1999), we also tested interactions between each SES metric (i.e., family income and neighborhood advantage) and each PGS.

Method

Statement on ethical regulations

Parents/caregivers provided written informed consent, and children verbal assent, to a research protocol approved by the central institutional review board at the University of California at San Diego for 21 data collection sites across the United States (https://abcdstudy.org/sites/abcd-sites.html) and by the Washington University IRB for the Washington University site.

Participants

Data came from 11,875 children (mean $\pm$ SD age $= 9.91 \pm 0.62$ years; 47.85% girls; 52.1% white; Table 1) who completed the baseline assessment of the ongoing longitudinal Adolescent Brain Cognitive Development (ABCD) Study (release 3.0; https://abcdstudy.org/; Volkow et al., 2018). The study includes multiple sibling and twin pairs and triplets as part of its family-based design (Garavan et al., 2018). Primary analyses were restricted to individuals genetically similar to reference populations from Europe with available genetic data who did not withdraw consent between baseline and later study waves ($n = 5549$), given the lack of other ancestry-specific discovery GWAS of cognitive phenotypes and the low predictive utility of PGS when applied across ancestries (Martin et al., 2019).

Individuals in this sample were 9.9 (± 0.63) years old, on average, and 47.0% of the sample were female (Table 1). Of these 5,549[1] individuals, there were 3,733 singletons as well as 1,816 individuals in 895 families. According to genetic relatedness metrics documented in ABCD release 5.1 ("gen_y_pihat.csv"), there were 863 sibling pairs (197 monozygotic pairs, 296 dizygotic pairs, 357 non-twin sibling pairs, and 13 pairs of unknown relatedness), 21 sibling trios (4 sets of triplets, 4 sets of MZ twins and 1 sibling, 11 sets of DZ twins and 1 sibling, and 2 sets of 3 siblings), one family of 4 individuals (2 sets of MZ twins), one family of five individuals (1 set of triplets, 1 set of DZ twins), and nine families of three individuals of whom only two were included in the subsample of individuals genetically similar to European reference populations (8 DZ twin pairs and 10 sibling pairs among those included).

This subsample of ABCD was slightly older at the time of assessment ($t[11,637] = 2.39$, $p = 0.017$) and exhibited lower neighborhood poverty ($t[9358.5] = -48.1$, $p < 2.2\text{e}{-}16$) and higher household income ($t[9436] = 44.2$, $p < 2.2\text{e}{-}16$) than the remainder of the ABCD sample (Table 1). Relative to the singleton sample, the sibling sample was older ($t[3376.2] = 6.11$, $p = 1.08\text{e}{-}09$) and had lower general ability ($t[3468.8] = -7.90$, $p = 3.60\text{e}{-}15$), higher executive function ($t[3378.6] = 3.22$, $p = 1.28\text{e}{-}03$), higher neighborhood advantage ($t[3535] = 6.34$, $p = 2.61\text{e}{-}10$), and higher household income ($t[3655.1] = 2.54$, $p = 0.011$).

---

[1] Seven individuals who withdrew consent for their data to be shared according to the most recent data release (Release 5.1) were excluded from analyses. In addition, 36 individuals whose neighborhood poverty estimates differed from their siblings were excluded from within-sibling analyses.

Table 1. Sample characteristics

	M (SD) / n (%)	n complete data
Age, years	9.93 (0.63)	5556
Sex, female	2612 (47.01%)	5556
Combined family income[a]	8.30 (1.64)	5153
Neighborhood Advantage, M (SD)[b]	0.36 (0.52)	5296
Cognition phenotypes		
General Cognitive Ability	0.24 (0.69)	5207
Executive Function	0.06 (0.72)	5207
Learning and Memory	0.15 (0.67)	5207
WISC Matrix Reasoning[c]	10.40 (2.85)	5207

[a]Family income was estimated using binned gross household income and collected from the PhenX questionnaire (see Methods).
[b]Scaled factor score.
[c]Total scaled score. WISC = Wechsler Intelligence Scale for Children.

Measures

Demographic measures

Child demographics

Child age was self-reported and measured in months. Child sex was a caregiver-reported dichotomous variable (Barch et al., 2018).

Familial income

Familial income was estimated using binned gross household income and collected from the PhenX questionnaire (Barch et al., 2018).

Neighborhood advantage (NAdv)

Area deprivation index values were calculated as outlined in previous investigations with the ABCD cohort (Taylor et al., 2020). Briefly, a participant's primary residential address at baseline was geocoded by the Data Analysis, Informatics, and Resource Center of the ABCD Study and were linked to each individual according to their US census tract information (Singh, 2003). Values were multiplied by -1, such that higher values represent more advantage, like family income.

Cognitive measures

Cognitive ability

Three principal components previously derived in the ABCD sample representing general cognitive ability, executive function, and learning and memory were used to index cognitive ability (Thompson et al., 2019). Briefly, a Bayesian Probabilistic Principal Component Analysis (BBPCA) was applied to cognitive tasks from the NIH Toolbox cognition battery, which assesses executive function, attention, processing speed, working memory, episodic memory, and language; the Rey Auditory Verbal Learning Test, which measures auditory learning, memory, and recognition; and the Little Man Task, which assesses visuospatial processing (Luciana et al., 2018). BPPCA component weights for each participant were made available with the ABCD curated data release 2.0.1. The Matrix Reasoning subtest of the Wechsler Intelligence Scale for Children-Fifth Edition (WISC), which indexes fluid and perceptual reasoning important for life function (Burgess et al., 2011; Green et al., 2017) was included as an additional measure of fluid intelligence.

Polygenic Scores (PGS)

Polygenic scores are weighted sums of effect alleles weighted by effect sizes found through GWAS of cognitive phenotypes. Summary statistics from the most well-powered, publicly available genome-wide association studies (GWAS) of three cognitive phenotypes (Educational Attainment (EA PGS), $N = 766,345$ (Lee et al., 2018); common Executive Function (cEF PGS), $N = 427,037$ (Hatoum et al., 2019); and IQ (IQ PGS), $N = 269,867$ (Savage et al., 2018)) were used. PGS were computed using PRS-CS (Ge et al., 2019), a Bayesian approach that incorporates all SNPs (i.e., no p-value thresholding) and utilizes an external linkage disequilibrium (LD) reference panel to account for correlations between SNPs. The "auto" function within the PRS-CS software package was used to compute PGS (see **Supplement** for further details).

Genotyping, quality control, and imputation

The Rutgers University Cell and DNA repository (now incorporated with other companies as Sampled; https://sampled.com/) genotyped saliva samples on the Smokescreen array (Baurley et al., 2016). The genetic data underwent typical quality control procedures following the Ricopili pipeline (Lam et al., 2020). Analyses were restricted to individuals most genetically similar to European reference populations, to match the ancestry makeup of the discovery GWAS. Further details are provided in the Supplement.

Each cognitive outcome (e.g., general cognitive ability, learning and memory, executive function, fluid intelligence), was regressed onto the two SES indicators, the three PGS, age, sex, and the first 10 ancestrally informative principal components (PCs), with a family identifier, and study site included as random effects. Of note, there was no evidence of multicollinearity in these models despite expected correlations between variables (variance inflation factor <2 for every variable in all models, see Supplement Table 1, Supplement Figure 1 for correlations). All continuous variables were standardized before analysis, with SES indicators standardized at the level of the family (i.e., level 2). The false discovery rate was used to correct across all SES p-values ($n = 8$) and, separately, all PGS p-values ($n = 12$).

To test whether any significant PGS effects were inflated by population stratification, assortative mating, and/or passive gene-environment correlation, we conducted within-sibling analyses in which each PGS was decomposed into between- and within-family variance (Selzam et al., 2019). Specifically, both the family average PGS and each individual's deviation from that family average PGS were included in linear mixed-effects models with the SES variables and covariates. Individual-level (within-family) estimates more closely approximate direct, or causal, genetic effects, whereas family-level estimates represent the portion of PGS effects that are confounded (Selzam et al., 2019). The false discovery rate was used to correct across all SES p-values ($n = 12$) and, separately, all PGS p-values ($n = 24$).

To examine whether SES moderates the effects of PGS on cognitive abilities, a series of linear mixed-effects models were run in which each SES variable was interacted with each PGS variable (i.e., six models per cognitive outcome). Moderation models covaried for all covariate-by-SES and covariate-by-PGS interactions (Keller, 2014). FDR was used to correct across all interaction p-values, separately for each SES and PGS (e.g., four income × EduA PGS, four NAdv × cEF PGS, etc.). All analyses were conducted in R version 4.3.2 using the lme4 package, with p-values calculated using lmer Test (D. Bates et al., 2015; Kuznetsova

et al., 2017). Bootstrapped ($n = 200$) confidence intervals were computed for all beta estimates.

Results

Associations between SES and PGS and cognitive outcomes

Family income was significantly and positively associated with cognitive performance across all four examined outcomes (Table 2, Figure 1, (β) ≥ 0.05, $\Delta R^2 \geq 4.00e-04$, $p \leq 3.32e-03$, $p_{FDR} \leq 0.01$), with the largest effect observed for general cognitive ability ($\beta = 0.18$, $\Delta R^2 = 0.031$) and the smallest for executive function ($\beta = 0.05$, $\Delta R^2 = 4.00e-04$). Neighborhood advantage, by contrast, showed no significant associations with cognitive outcomes ($|\beta| \leq 0.03$, $\Delta R2 \leq 1.60e-03$, $p \geq 0.081$) independent of family income.

EduA PGS was significantly and positively associated with general cognitive ability and matrix reasoning ($\beta \geq 0.07$, $\Delta R^2 \geq 3.73e-03$, $p \leq 9.12e-05$, $p_{FDR} \leq 1.82e-04$) but not with executive function or learning and memory ($\beta \leq 0.03$, $\Delta R^2 \leq 7.30e-04$, $p \geq 0.08$). cEF PGS was positively linked to executive function, learning and memory, and matrix reasoning performance ($\beta \geq 0.06$, $\Delta R^2 \geq 3.07e-03$, $p \leq 1.66e-04$, $p_{FDR} \leq 2.50e-04$) but not general cognitive ability ($\beta = 0.01$, $\Delta R^2 = 6.26e-05$, $p = 0.35$). Intelligence PGS was positively associated with general cognitive ability, learning and memory, and matrix reasoning ($\beta \geq 0.08$, $\Delta R^2 \geq 2.94e-03$, $p \leq 1.08e-04$, $p_{FDR} \leq 1.85e-04$) but not executive function ($\beta = -5.17e-04$, $\Delta R^2 = -1.26e-5^2$, $p = 0.98$). Overall, except for cEF PGS, the largest effect sizes between income and PGS and cognition were observed for general cognitive ability, and the smallest for executive function (Figure 1). Significant income and PGS associations were comparable in effect size, with a one standard deviation increase in each linked to 0.05 – 0.19 standard deviation increases in cognitive ability.

Within-sibling results

In a reduced sample of siblings ($n = 1,816$, excluding singletons and 36 participants with within-family differences in neighborhood advantage values), intelligence PGS continued to predict general cognitive ability at both the between-family ($\beta = 0.22$, $\Delta R^2 = 0.020$, $p = 6.08-08$, $p_{FDR} = 1.46e-07$) and within-family ($\beta = 0.06$, $\Delta R^2 = 2.39e-03$, $p = 2.98-04$, $p_{FDR} = 2.05e-03$) levels (Table 3, Figure 2). Between-family ($\beta = 0.13$, $\Delta R^2 = 8.09e-04$, $p = 3.42e-04$, $p_{FDR} = 2.05e-03$), but not within-family ($\beta = 0.019$, $\Delta R^2 = 2.95e-04$, $p = 0.23$), EduA PGS was significantly associated with general cognitive ability. Similarly, between-family ($\beta = 0.16$, $\Delta R^2 = 0.021$, $p = 2.38e-06$, $p_{FDR} = 2.86e-05$), but not within-family ($\beta = 0.026$, $\Delta R^2 = 6.21e-04$, $p = 0.12$), effects of cEF PGS were associated with learning and memory (Table 3, Figure 2). No other PGS associations were significant following false discovery rate correction ($|\beta| \leq 0.10$, $\Delta R2 \leq 5.42e-03$, $p \geq 0.011$, $p_{FDR} \geq 0.052$). Income remained significantly and positively associated with general cognitive ability, learning and memory, and matrix reasoning ($\beta \geq 0.11$, $\Delta R^2 \geq 7.80e-03$, $p \leq 7.49e-03$, $p_{FDR} \leq 2.00-03$; Table 3, Figure 2) but not with executive function ($\beta = 0.037$, $\Delta R^2 = -9.98e-04$, $p = 0.27$; Table 3, Figure 2).

[2]Delta R-squared values were calculated as the R-squared difference between two fixed effects models, which can have a negative value if the predictor is near zero and changes the variance of the random effect parameter when the model is iteratively estimated.

Interactions between SES and PGS

No interactions between SES (i.e., income, neighborhood advantage) and PGS (i.e., EduA, cEF, intelligence) were significant following FDR correction ($|\beta| \leq 0.03$, $\Delta R^2 \leq 7.52e-04$, $p \geq 0.04$, $p_{FDR} \geq 0.39$; Tables 4–7).

Discussion

The present study had three aims: (a) test whether indicators of family and neighborhood socioeconomic status (SES) are independently associated with different aspects of cognitive ability in a large sample of youth, beyond contributions from polygenic scores (PGS) of educational attainment (EduA), intelligence (IQ), and common executive function (cEF); (b) test whether any significant PGS effects are attributable to between-family and/or within-family effects through within-siblings analyses; and (c) test whether SES moderates associations between PGS and cognitive outcomes. Broadly, family income, but not neighborhood advantage, and PGS were significantly and independently associated with cognitive abilities with similar effect sizes, with some variability across PGS indicators and cognitive outcome. Except for Intelligence PGS and general cognitive ability, no within-family PGS effects were detected. Finally, no significant interactions between SES and PGS were observed after correction for multiple comparisons. These findings provide further evidence for the importance of modifiable environmental factors and genetic influences in the development of cognitive abilities.

Concerning SES, family income was robustly associated with all cognitive outcomes, accounting for indicators of genetic propensity, whereas neighborhood advantage was not. The magnitude of the associations between income and each cognitive outcome was 1.18 to 2.17 times larger than most significant PGS associations with the same outcomes, with the exceptions of intelligence PGS and general cognitive ability and cEF PGS and executive function (1.06–1.40 times larger than income associations). Altogether, these results show that family income and estimates of genetic propensity for cognitive phenotypes derived from current GWAS in youth genetically similar to European reference populations show similar magnitudes of association with cognitive outcomes.

The lack of significant neighborhood advantage associations is not consistent with previous evidence of independent contributions of family SES and neighborhood resources (Taylor et al., 2020) and area deprivation (Park et al., 2023) to cognition in the ABCD dataset or with other investigations of putative effects of neighborhood disadvantage on performance on various cognitive tasks (Kalb et al., 2023; Wodtke, Ard, et al., 2022; Wodtke, Ramaj, et al., 2022). There are several potential explanations for this divergence. First, the variability of NAdv among Black participants in ABCD is greater than that among White participants (Taylor et al., 2020). Although race and genetic ancestry should not be conflated, it may be that the present study's reliance on individuals most genetically similar to European reference populations may have truncated the variability in NAdv and thus the magnitude of association with cognitive outcomes. Further, Taylor and colleagues (2020) utilized the full ABCD sample in analyses, so the present study in the smaller subsample may have simply been underpowered to detect such effects. Second, Taylor and colleagues (2020) did not account for PGS in their analyses, possibly inflating income and neighborhood poverty estimates. Third, Park and colleagues (2023) used the area deprivation index rather than a latent neighborhood poverty factor like that in our analyses, and they did not appear to account for family relatedness. Other
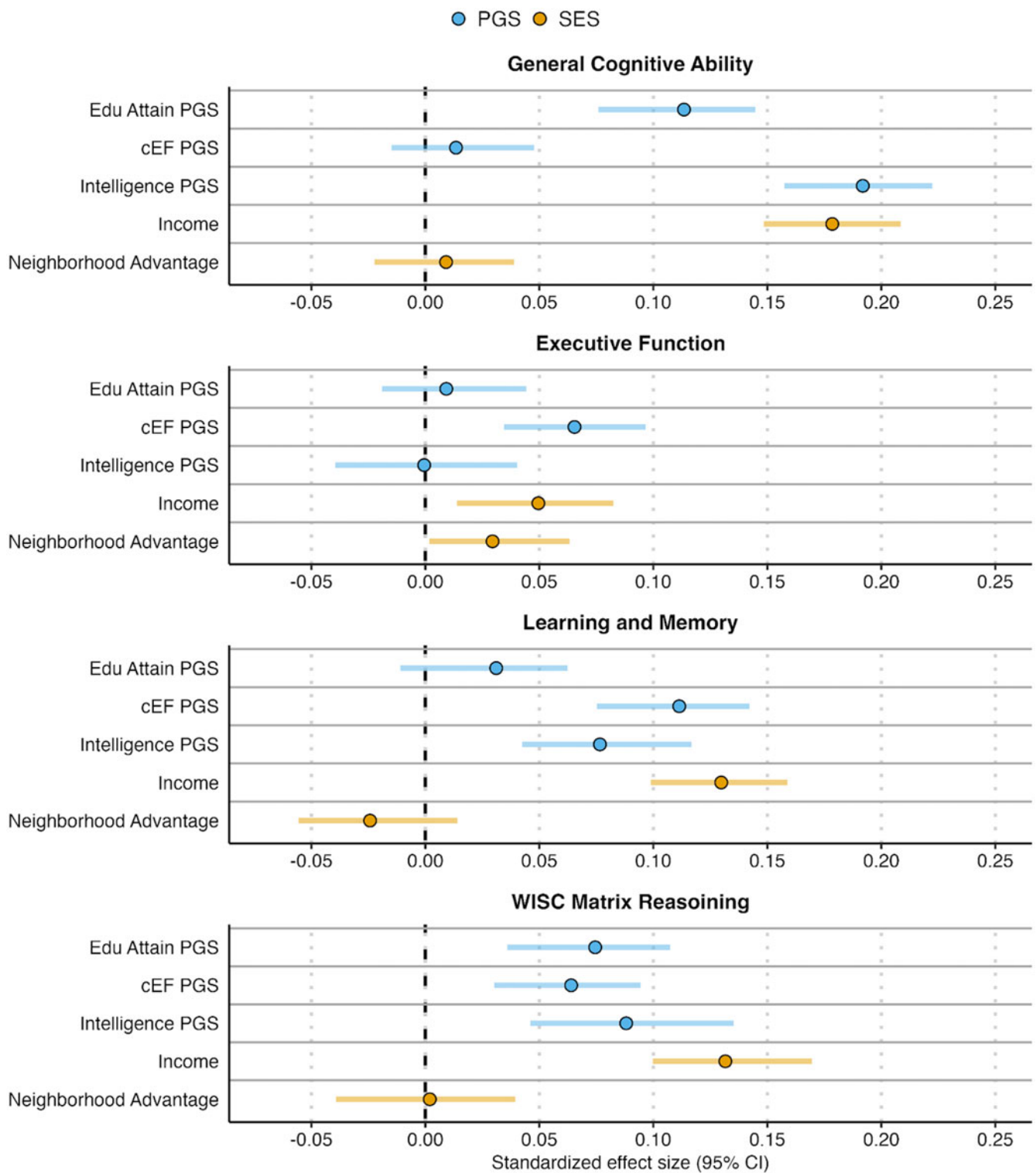
Figure 1. Associations between PGS, family income, and neighborhood advantage and cognitive performance. Polygenic score (PGS) associations are shown in blue, and SES associations are shown in orange. Estimates are standardized betas, with 95% confidence intervals. Edu Attain = educational attainment; cEF = common executive function; PGS = polygenic score.

investigations did not account for family income as was done in the present study, possibly explaining our null findings relative to other investigations.

Similar to prior work (Judd et al., 2020; Merz et al., 2022; Park et al., 2023; Peñaherrera-Aguirre et al., 2022; Raffington et al.,

2019), the present study found that EduA PGS was associated with cognitive ability. The largest effect found was for general cognitive ability and no significant association was detected for executive function, similar to Merz and colleagues (2022), or for learning and memory. Building upon this literature, the current study found

Table 2. Associations between PGS, family income, and neighborhood advantage and cognitive performance

	Standardized Beta (β)	STE	95% CI	*p*	*pFDR*
General Cognitive Ability					
EduA PGS	0.11	0.02	0.08, 0.14	1.14E-12	6.86E-12
cEF PGS	0.01	0.01	−0.01, 0.05	0.35	0.42
INT PGS	0.19	0.02	0.15, 0.23	1.61E-26	1.93E-25
Income	0.18	0.02	0.15, 0.21	9.48E-31	7.58E-30
Neighborhood Advantage	0.01	0.02	−0.02, 0.04	0.56	0.63
Executive Function					
EduA PGS	0.01	0.02	−0.02, 0.05	0.60	0.66
cEF PGS	0.07	0.02	0.03, 0.10	3.91E-05	1.06E-04
INT PGS	−5.17E-04	0.02	−0.04, 0.04	0.98	0.98
Income	0.05	0.02	0.02, 0.08	3.32E-03	0.01
Neighborhood Advantage	0.03	0.02	−0.01, 0.06	0.08	0.13
Learning and Memory					
EduA PGS	0.03	0.02	6.69E-04, 0.07	0.08	0.10
cEF PGS	0.11	0.02	0.08, 0.14	1.93E-12	7.74E-12
INT PGS	0.08	0.02	0.04, 0.12	1.08E-04	1.85E-04
Income	0.13	0.02	0.09, 0.17	3.23E-14	1.29E-13
Neighborhood Advantage	−0.02	0.02	−0.06, 0.01	0.16	0.21
WISC Matrix Reasoning					
EduA PGS	0.07	0.02	0.04, 0.11	9.12E-05	1.82E-04
cEF PGS	0.06	0.02	0.04, 0.10	1.66E-04	2.50E-04
INT PGS	0.09	0.02	0.04, 0.14	4.41E-05	1.06E-04
Income	0.13	0.02	0.10, 0.17	6.69E-14	1.78E-13
Neighborhood Advantage	1.95E-03	0.02	−0.03, 0.04	0.91	0.91

PGS = Polygenic Score; EduA = educational attainment; cEF = common executive function; INT = intelligence. pFDR represents FDR-corrected *p*-values, separately for Income/Neighborhood Advantage (*n* = 8 tests) and the PGS (*n* = 12 tests).

that intelligence PGS mirrored these associations, showing larger effects than EduA for general cognitive ability and matrix reasoning and also showing a significant association with learning and memory. PGS for common executive function (cEF) was associated with larger effects than the other PGS for executive function and learning and memory and was also associated with matrix reasoning. Altogether, these findings suggest that matrix reasoning is most broadly associated with estimates of genetic propensity toward cognitive ability and educational attainment and that executive function is the least strongly predicted by PGS overall and specifically linked to cEF PGS. These findings support the importance of examining multiple indicators of cognitive ability and genetic propensity to parse differences across cognitive domains.

In this study, results highlight that in middle childhood both income and genetic propensity are independently associated with both relatively stable cognitive abilities (e.g., fluid intelligence and IQ) as well as those that undergo substantial development through adolescence (e.g., EF). More explicitly, each cognitive domain examined (i.e., general ability, fluid intelligence, executive function, learning/memory) has varying trajectories of development from middle childhood through adulthood (Diamond, 2006, 2013; Ferrer et al., 2009; Greiff et al., 2015; McKean et al., 2015;

Petscher et al., 2018). Despite stability differences between domains there is ample evidence that adolescent cognitive ability strongly predicts ability across the lifespan (Icenogle et al., 2019), and that adolescent cognitive ability may mediate associations between early-life SES and later-life cognition (Zheng et al., 2019), underscoring the importance of examining relationships between income, genetic propensity, and cognition in adolescence. Moreover, there is evidence that the same genes are associated with domains of cognition throughout the lifespan and that cognitive ability at different ages is genetically correlated (Haworth et al., 2010; Mollon et al., 2021; Trzaskowski, Yang, et al., 2014). Altogether, although some change may occur due to increasing heritability of cognition over the lifespan (Mollon et al., 2018, Trzaskowski et al., 2014) and increasing correlation between genetic influences and specific measured environments, the effects presented here can be generally expected to persist and likely increase in magnitude into adolescence and young adulthood.

Notably, results of the within-siblings analyses suggested that most of these significant PGS associations were attributable to between-family, but not within-family, variability in PGS. That is, accounting for the family PGS means, individual-level PGS for EduA was not associated with general cognitive ability or matrix reasoning, nor was individual-level intelligence PGS with learning
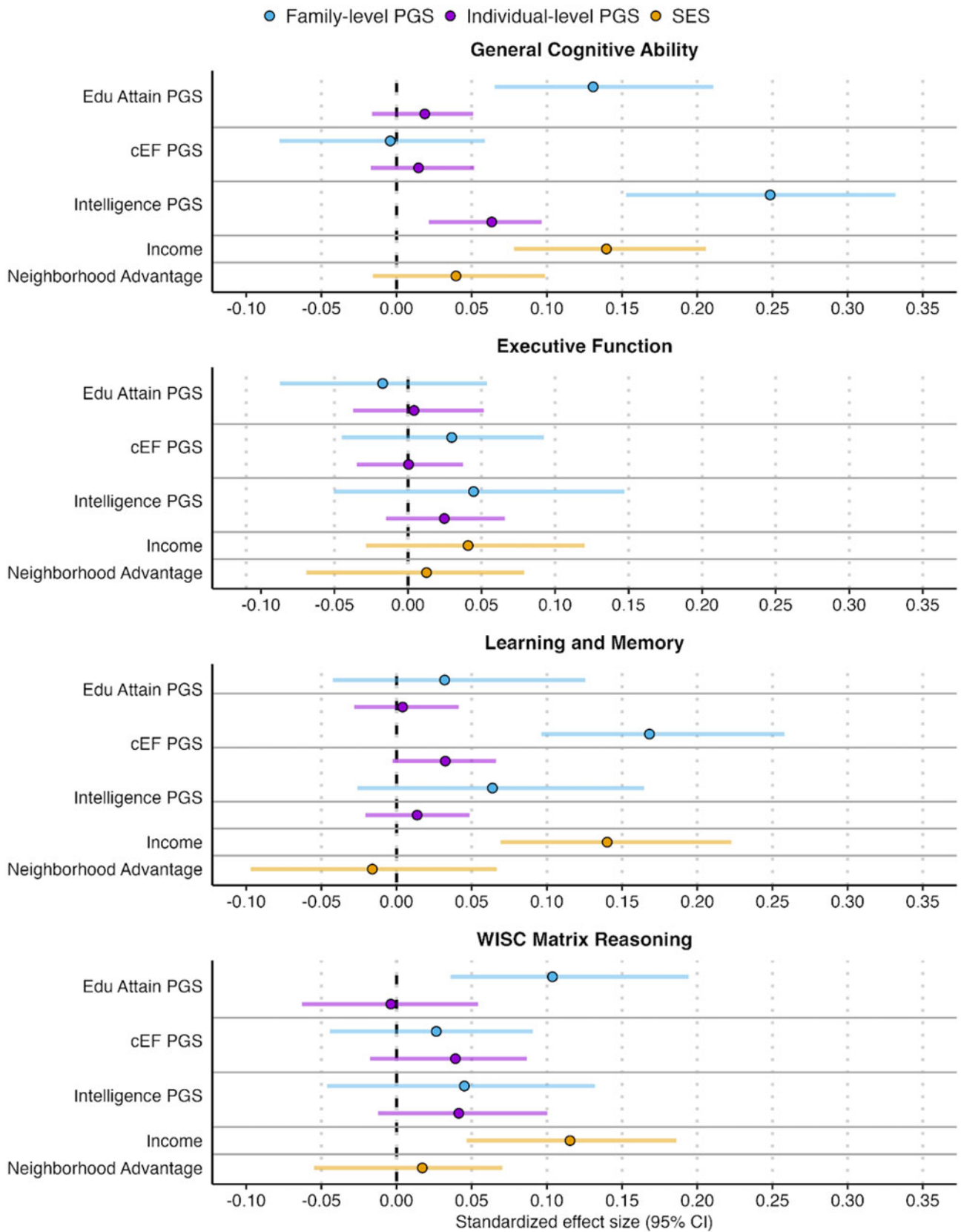
Figure 2. Between- and within-family PGS associations with cognition. Polygenic score (PGS) associations with cognition are decomposed into between- (blue) and within- (purple) family estimates; SES associations are shown in orange. Estimates are standardized betas, with 95% confidence intervals.

Table 3. Results for within-sibling analyses

	Standardized Beta (β)	STE	95% CI	p	pFDR
General Cognitive Ability					
Income	0.14	0.03	0.07, 0.21	4.38E-05	3.50E-04
Neighborhood Advantage	0.04	0.03	−0.04, 0.10	0.23	0.41
EduA PGS - between	0.13	0.04	0.06, 0.22	8.70E-04	0.01
EduA PGS - within	0.02	0.02	−0.01, 0.06	0.26	0.57
cEF PGS - between	−4.19E-03	0.04	−0.07, 0.07	0.91	0.94
cEF PGS - within	0.01	0.02	−0.01, 0.05	0.38	0.63
INT PGS - between	0.25	0.04	0.18, 0.33	4.00E-08	9.59E-07
INT PGS - within	0.06	0.02	0.02, 0.10	4.64E-04	3.71E-03
Executive Function					
Income	0.04	0.04	−0.03, 0.11	0.25	0.41
Neighborhood Advantage	0.01	0.03	−0.04, 0.07	0.72	0.72
EduA PGS - between	−0.02	0.04	−0.10, 0.06	0.67	0.85
EduA PGS - within	4.11E-03	0.02	−0.04, 0.04	0.83	0.94
cEF PGS - between	0.03	0.04	−0.04, 0.09	0.42	0.63
cEF PGS - within	4.21E-04	0.02	−0.04, 0.04	0.98	0.98
INT PGS - between	0.04	0.05	−0.06, 0.15	0.34	0.63
INT PGS - within	0.02	0.02	−0.02, 0.07	0.24	0.57
Learning and Memory					
Income	0.14	0.04	0.08, 0.22	1.74E-04	6.98E-04
Neighborhood Advantage	−0.02	0.04	−0.08, 0.06	0.66	0.72
EduA PGS - between	0.03	0.04	−0.02, 0.11	0.46	0.63
EduA PGS - within	4.03E-03	0.02	−0.03, 0.05	0.82	0.94
cEF PGS - between	0.17	0.04	0.09, 0.25	1.59E-05	1.91E-04
cEF PGS - within	0.03	0.02	−4.90E-03, 0.07	0.06	0.26
INT PGS - between	0.06	0.05	−0.05, 0.16	0.19	0.52
INT PGS - within	0.01	0.02	−0.02, 0.05	0.47	0.63
WISC Matrix Reasoning					
Income	0.12	0.03	0.04, 0.18	1.00E-03	2.67E-03
Neighborhood Advantage	0.02	0.03	−0.05, 0.10	0.62	0.72
EduA PGS - between	0.10	0.04	0.02, 0.19	0.01	0.06
EduA PGS - within	−3.76E-03	0.03	−0.08, 0.06	0.89	0.94
cEF PGS - between	0.03	0.04	−0.05, 0.10	0.46	0.63
cEF PGS - within	0.04	0.03	−0.02, 0.09	0.16	0.52
INT PGS - between	0.05	0.05	−0.06, 0.13	0.33	0.63
INT PGS - within	0.04	0.03	−0.02, 0.10	0.17	0.52

PGS = Polygenic Score; EduA = educational attainment; cEF = common executive function; INT = intelligence. pFDR = FDR-corrected p-values, separately for Income/Neighborhood Advantage (*n* = 8 tests) and the PGS (*n* = 24 tests).

and memory or matrix reasoning or cEF PGS with executive function or learning and memory. In these cases, the observed PGS effects may be attributable to population stratification, assortative mating, or passive gene-environment correlation, rather than to direct genetic effects (Selzam et al., 2019). Both between- and within-family PGS effects were detected for intelligence PGS in association with general cognitive ability, with the between-family

PGS association being approximately four times larger than the within-family PGS association. These findings converge with prior work showing that, particularly for cognitive phenotypes, between-family PGS effects eclipse within-family PGS effects (e.g., Okbay et al., 2022; Selzam et al., 2019). Shared environment has been shown to capture a particularly large amount of variability in phenotypes such as IQ and educational attainment, relative to

Table 4. Interactions between PGS & SES in models predicting general cognitive ability

Model	Standardized Beta (β)	STE	95% CI	p	pFDR
EduA PGS	0.21	0.02	0.17, 0.24	3.54E-29	
Income	0.17	0.02	0.13, 0.20	1.51E-19	
Income * EduA PGS	−0.03	0.01	−0.06, 2.14E-04	0.05	0.39
EduA PGS	0.23	0.02	0.20, 0.27	1.74E-38	
Neighborhood Advantage	0.06	0.02	0.02, 0.10	9.264E-04	
Neighborhood Advantage * EduA PGS	−0.02	0.01	−0.05, 0.01	0.20	0.67
cEF PGS	0.14	0.02	0.10, 0.18	1.52E-14	
Income	0.21	0.02	0.18, 0.25	4.13E-29	
Income * cEF PGS	−0.01	0.01	−0.04, 0.01	0.29	0.67
cEF PGS	0.14	0.02	0.10, 0.18	3.64E-16	
Neighborhood Advantage	0.08	0.02	0.05, 0.12	1.29E-05	
Neighborhood Advantage * cEF PGS	0.01	0.01	−0.01, 0.04	0.31	0.67
INT PGS	0.26	0.02	0.22, 0.30	1.13E-47	
Income	0.18	0.02	0.14, 0.21	1.65E-21	
Income*INT PGS	−0.03	0.01	−0.06, -2.01E-03	0.02	0.39
INT PGS	0.29	0.02	0.25, 0.33	1.11E-58	
Neighborhood Advantage	0.07	0.02	0.03, 0.11	9.12E-05	
Neighborhood Advantage*INT PGS	2.06E-03	0.01	−0.03, 0.03	0.88	0.91

PGS = Polygenic Score; EduA = educational attainment; cEF = common executive function; INT = intelligence. pFDR represents FDR correction across all interaction p-values, separately for each SES and PGS (e.g., 4 Income × EduA PGS, 4 Neighborhood Advantage × cEF PGS, etc).

Table 5. Interactions between PGS & SES in models predicting executive function

Model	Standardized Beta (β)	STE	95% CI	p	pFDR
EduA PGS	−0.02	0.02	−0.05, 0.03	0.44	
Income	0.05	0.02	3.63E-03, 0.09	0.03	
Income*EduA PGS	3.32E-03	0.02	−0.02, 0.04	0.83	0.91
EduA PGS	−0.01	0.02	0.05, 0.02	0.52	
Neighborhood Advantage	0.07	0.02	−0.03, 0.10	1.04E-03	
Neighborhood Advantage*EduA PGS	0.02	0.01	−0.01, 0.04	0.25	0.67
cEF PGS	0.07	0.02	0.04, 0.11	1.20E-04	
Income	0.04	0.02	4.73E-03, 0.07	0.06	
Income* cEF PGS	−0.01	0.02	−0.04, 0.02	0.37	0.70
cEF PGS	0.09	0.02	0.05, 0.13	4.38E-06	
Neighborhood Advantage	0.06	0.02	0.03, 0.10	1.20E-03	
Neighborhood Advantage* cEF PGS	−0.01	0.01	−0.04, 0.02	0.63	0.90

(*Continued*)

Table 5. (*Continued*)

Model	Standardized Beta (β)	STE	95% CI	p	pFDR
INT PGS	0.03	0.02	7.98E-04, 0.07	0.08	
Income	0.04	0.02	−4.05E-03, 0.08	0.07	
Income*INT PGS	3.23E-03	0.01	−0.03, 0.03	0.83	0.91
INT PGS	0.04	0.02	5.97E-04, 0.08	0.03	
Neighborhood Advantage	0.06	0.02	0.01, 0.10	1.70E-03	
Neighborhood Advantage*INT PGS	0.01	0.02	−0.02, 0.04	0.49	0.84

PGS = Polygenic Score; EduA = educational attainment; cEF = common executive function; INT = intelligence. pFDR represents FDR correction across all interaction p-values, separately for each SES and PGS (e.g., 4 Income × EduA PGS, 4 Neighborhood Advantage × cEF PGS, etc).

Table 6. Interactions between PGS & SES in models predicting learning and memory

Model	Standardized Beta (β)	STE	95% CI	p	pFDR
EduA PGS	0.09	0.02	0.05, 0.13	1.76E-05	
Income	0.10	0.02	0.05, 0.14	6.81E-06	
Income*EduA PGS	0.01	0.02	−0.03, 0.04	0.67	0.90
EduA PGS	0.10	0.02	0.06, 0.14	1.22E-07	
Neighborhood Advantage	3.75E-03	0.02	−0.04, 0.04	0.85	
Neighborhood Advantage*EduA PGS	−0.01	0.01	−0.04, 0.02	0.66	0.90
cEF PGS	0.15	0.02	0.12, 0.19	2.76E-15	
Income	0.11	0.02	0.07, 0.15	1.09E-07	
Income*cEF PGS	0.02	0.02	−0.01, 0.05	0.18	0.67
cEF PGS	0.16	0.02	0.12, 0.20	1.30E-17	
Neighborhood Advantage	0.01	0.02	−0.04, 0.05	0.54	
Neighborhood Advantage*cEF PGS	0.03	0.01	3.62E-03, 0.06	0.04	0.39
INT PGS	0.14	0.02	0.10, 0.18	2.52E-12	
Income	0.10	0.02	0.05, 0.14	2.22E-06	
Income*INT PGS	0.02	0.02	−0.02, 0.05	0.29	0.67
INT PGS	0.15	0.02	0.11, 0.19	1.08E-14	
Neighborhood Advantage	0.01	0.02	−0.03, 0.05	0.71	
Neighborhood Advantage*INT PGS	0.02	0.02	−0.01, 0.05	0.14	0.67

PGS = Polygenic Score; EduA = educational attainment; cEF = common executive function; INT = intelligence. pFDR represents FDR correction across all interaction p-values, separately for each SES and PGS (e.g., 4 Income × EduA PGS, 4 Neighborhood Advantage × cEF PGS, etc).

some physical and mental health phenotypes, suggesting that PGS of IQ and EduA may be especially confounded by gene-environment correlation (Polderman et al., 2015; Selzam et al., 2019).

The hypothesis that SES may moderate the effect of genetic propensity on cognitive outcomes was not supported by the present data. Although inconsistent, particularly across countries, much of the evidence for what has been named the Scarr-Rowe hypothesis has come from twin studies showing that SES moderates the heritability of cognitive ability (e.g., T. C. Bates et al., 2013; Harden et al., 2007; Rowe et al., 1999; Tucker-Drob et al., 2011; Turkheimer et al., 2003). Using polygenic scores allows this hypothesis to be tested among singletons as well as twins and overcomes the erroneous (in this case) twin assumption that there is no covariance between additive genetic and shared environmental influences on a trait. At the same time, the variance explained by PGS is substantially less than heritability estimates and does not fully capture an individual's

Table 7. Interactions between PGS & SES in models predicting wisc matrix reasoning

Model	Standardized Beta (β)	STE	95% CI	p	pFDR
EduA PGS	0.15	0.02	0.10, 0.19	1.67E-11	
Income	0.13	0.02	0.08, 0.18	4.91E-09	
Income*EduA PGS	1.89E-03	0.02	−0.04, 0.04	0.91	0.91
EduA PGS	0.17	0.02	0.13, 0.22	8.23E-16	
Neighborhood Advantage	0.05	0.02	0.01, 0.09	0.03	
Neighborhood Advantage*EduA PGS	−2.76E-03	0.02	−0.04, 0.03	0.86	0.91
cEF PGS	0.15	0.02	0.11, 0.19	8.79E-13	
Income	0.16	0.02	0.12, 0.20	2.16E-13	
Income* cEF PGS	−4.85E-03	0.02	−0.04, 0.03	0.77	0.91
cEF PGS	0.16	0.02	0.12, 0.20	1.40E-14	
Neighborhood Advantage	0.06	0.02	0.02, 0.10	2.86E-03	
Neighborhood Advantage* cEF PGS	0.02	0.01	−0.01, 0.05	0.16	0.67
INT PGS	0.16	0.02	0.11, 0.21	8.40E-14	
Income	0.14	0.02	0.11, 0.18	1.16E-10	
Income*INT PGS	0.01	0.02	−0.03, 0.04	0.60	0.90
INT PGS	0.19	0.02	0.15, 0.24	1.30E-18	
Neighborhood Advantage	0.05	0.02	0.01, 0.10	0.01	
Neighborhood Advantage*INT PGS	0.01	0.02	−0.02, 0.05	0.38	0.70

PGS = Polygenic Score; EduA = educational attainment; cEF = common executive function; INT = intelligence. Betas are standardized. pFDR represents FDR correction across all interaction p-values, separately for each SES and PGS (e.g., 4 Income × EduA PGS, 4 Neighborhood Advantage × cEF PGS, etc).

full genetic propensity. Further, interactions in general, and in particular those involving potentially noisy measures, such as polygenic scores, are often quite small and difficult to detect (e.g., Vize et al., 2023). The present findings align with some prior research estimating interactions between PGS and SES in association with cognitive ability (e.g., Merz et al., 2022; von Stumm et al., 2023), including in the ABCD sample (e.g., Judd et al., 2020), but contrasts other work detecting PGS-by-SES interactions, also using the ABCD sample (e.g., Peñaherrera-Aguirre et al., 2022). The latter study was limited by analyses that used different genotype quality control standards and did not correct for multiple testing in assessing statistical significance of the interactions. Overall, these results contribute to a complicated literature by showing that, in a large sample of children from across the U.S. who are genetically similar to European reference populations, genetic influences on cognition derived from three GWAS of aspects of cognition are not moderated by childhood family SES or neighborhood poverty across a range of cognitive measures.

Strengths of this study include the large sample size; the decomposition of polygenic score variance into between- and within-family variance to better account for potentially confounding influences of population stratification, assortative mating, and or passive gene-environment correlation; and the use of both family- and neighborhood-level SES, multiple polygenic scores, and multiple cognitive outcomes. Further, by leveraging genome-wide data and measured SES, we were able to estimate the independent effect of SES on cognitive abilities, accounting for genetic influences on cognition that may share variance with SES.

Limitations to the present study should be considered in interpreting the results. First, our sample is restricted to individuals most genetically similar to reference populations from Europe. Given the intersectionality between SES and race (which is correlated with but distinct from ancestral background) alongside typically higher levels of both familial and neighborhood poverty among BIPOC populations, our findings may not generalize to all ancestral backgrounds or racial groups. There is a dearth of well-powered GWAS of cognitive phenotypes in individuals with less genetic similarity to European reference populations, precluding the estimate of PGS in these groups in ABCD, but the exclusion of these individuals from research on this topic is a major limitation. Second, polygenic scores include information only from common genetic variants, and thus, in conjunction with the still-limited power of PGS to explain the common variant heritability, are underestimates of the true genetic effect on cognition. Further, rare genetic variants and de novo mutations not incorporated into the PGS may interact with SES (Ganna et al., 2016; Rask-Andersen et al., 2021), and our approach was not able to account for this possibility.

Third, the data used in this study are cross-sectional due to the availability of certain cognitive tasks at baseline but not follow-up waves of ABCD (Luciana et al., 2018; Thompson et al., 2019). Prior work examining specific cognitive measures in ABCD suggested that despite age-related change, there is considerable stability in individual differences of cognitive performance from ages 9–10 to 11–12 (Anokhin et al., 2022). Here, we were interested in broad cognitive constructs that mapped most closely onto the GWAS used for our PGS, and because these broader cognitive components can only be computed at one time point, we were restricted to baseline cognitive and SES data. The nascent literature indicates that early life and sustained exposure to poverty has differential associations with cognition relative to those with more transient or later development poverty (Brooks-Gunn & Duncan, 1997; Korenman et al., 1995; Najman et al., 2009; National Institute of Child Health and Human Development Early Child Care Research Network, 2005), and thus it is unclear whether the effects associated with SES in this study reflect persistent or temporally limited relationships. Given possible changes in SES and in the strength of associations between polygenic propensity and SES and cognitive ability over the lifespan (Brooks-Gunn & Duncan, 1997; Korenman et al., 1995; Najman et al., 2009; National Institute of Child Health and Human Development Early Child Care Research Network, 2005), it will be important for future work to whether changes in SES are associated with changes in cognitive ability accounting for, or in interaction with, polygenic propensity.

wFourth, the observed associations were relatively small in effect ($\beta \leq 0.193$, $R^2 \leq 0.031$ in primary analyses), suggesting that large proportions of variance in the cognitive abilities examined are still unexplained. Other influences, including but not limited to maternal exposure to adversity and stress effects on the fetus, caregiver and child stress, mental health, parenting behavior, cognitive stimulation in the home, nutritional deprivation, greater exposure to environmental toxins, and parent genetic variants for cognition that were not passed to their children but shaped their cognitive environment, are also important factors to consider (See reviews: Johnson et al., 2016; Merz et al., 2019; Pace et al., 2017; Perkins et al., 2013). Each of these aforementioned factors (e.g., household stress, increased risk of mental health diagnoses, parental behaviors) are correlated with childhood poverty and are potential environmental mechanisms linking childhood poverty with adverse outcomes, including lower performance on cognitive measures. Future work may address some of these limitations through the incorporation of parental cognitive performance measures, in addition to measures of other influences that may shape child cognition and cognitive environment. Fifth, the inclusion of PGS and heritable environmental factors (e.g., SES) in the same regression can create spurious associations (Akimova et al., 2021). In that case, the effect of the environment should increase, while the effect of genetics should decrease. We did not find this to be the case in our models, suggesting that the associations we observed are not spurious or driven by conditioning on a collider. Relatedly, prior work shows that gene × environment interactions may be inflated in the case of gene-environment correlations (Akimova et al., 2021; Kazma et al., 2011; Lindström et al., 2009). Because we did not detect any significant interactions, we do not believe this is a concern in our analyses.

Broadly, this study extends prior work by examining multiple SES, PGS, and cognitive outcomes and testing whether PGS associations may be attributable to direct genetic effects or, rather, are confounded. We show that the PGS effects are largely confounded (e.g., by population stratification, assortative mating, and/or gene-environment correlation) and are smaller than the effects observed for family income. These findings highlight the unique importance of a modifiable environmental factor, SES, to childhood cognitive ability and lend further empirical support to those investigating the effects of interventions designed to alleviate childhood poverty, such as direct payments designed to increase family financial stability and enhance child cognitive and psychosocial functioning (Rojas et al., 2020). This work emphasizes the continued need for public policy solutions to the problem of childhood poverty. Future work is needed to examine mechanisms through which genes and SES may influence cognitive ability (e.g., brain structure, stress, physical and mental health), which may also hold implications for intervention efforts and local and federal policy aimed at reducing child poverty and its sequelae.

References

Akee, R. K. Q., Copeland, W. E., Keeler, G., Angold, A., & Costello, E. J. (2010). Parents' incomes and children's outcomes: A quasi-experiment. *American Economic Journal. Applied Economics*, 2(1), 86–115. https://doi.org/10.1257/app.2.1.86

Akimova, E. T., Breen, R., Brazel, D. M., & Mills, M. C. (2021). Gene-environment dependencies lead to collider bias in models with polygenic scores. *Scientific Reports*, 11(1), 9457. https://doi.org/10.1038/s41598-021-89020-x

Anokhin, A. P., Luciana, M., Banich, M., Barch, D., Bjork, J. M., Gonzalez, M. R., Gonzalez, R., Haist, F., Jacobus, J., Lisdahl, K., McGlade, E., McCandliss, B., Nagel, B., Nixon, S. J., Tapert, S., Kennedy, J. T., & Thompson, W. (2022). Age-related changes and longitudinal stability of individual differences in ABCD Neurocognition measures. *Developmental Cognitive Neuroscience*, 54, 101078. https://doi.org/10.1016/j.dcn.2022.101078

Barch, D. M., Albaugh, M. D., Avenevoli, S., Chang, L., Clark, D. B., Glantz, M. D., Hudziak, J. J., Jernigan, T. L., Tapert, S. F., & Yurgelun-Todd, D. (2018). Demographic, physical and mental health assessments in the adolescent brain and cognitive development study: Rationale and description. *Developmental Cognitive Neuroscience*, 32, 55–66.

Bates, D., Maechler, M., Bolker, B., Walker, S., Christensen, R. H. B., Singmann, H., Dai, B., Grothendieck, G., Green, P., & Bolker, M. B. (2015). Package lme4. *Convergence*, 12(1), 2.

Bates, T. C., Lewis, G. J., & Weiss, A. (2013). Childhood socioeconomic status amplifies genetic effects on adult intelligence. *Psychological Science*, 24(10), 2111–2116.

Baurley, J. W., Edlund, C. K., Pardamean, C. I., Conti, D. V., & Bergen, A. W. (2016). Smokescreen: A targeted genotyping array for addiction research. *BMC Genomics*, 17(1), 145. https://doi.org/10.1186/s12864-016-2495-7

Belsky, D. W., Domingue, B. W., Wedow, R., Arseneault, L., Boardman, J. D., Caspi, A., Conley, D., Fletcher, J. M., Freese, J., Herd, P., Moffitt, T. E., Poulton, R., Sicinski, K., Wertz, J., & Harris, K. M. (2018). Genetic analysis of social-class mobility in five longitudinal studies. *Proceedings of the National Academy of Sciences of the United States of America*, 115(31), E7275–E7284. https://doi.org/10.1073/pnas.1801238115

Brooks-Gunn, J., & Duncan, G. J. (1997). The effects of poverty on children. *The Future of Children*, 7(2), 55–71. https://doi.org/10.2307/1602387

Burgess, G. C., Gray, J. R., Conway, A. R., & Braver, T. S. (2011). Neural mechanisms of interference control underlie the relationship between fluid intelligence and working memory span. *Journal of Experimental Psychology-General*, 140(4), 674–692.

Clearfield, M. W., & Niman, L. C. (2012). SES affects infant cognitive flexibility. *Infant Behavior and Development*, 35(1), 29–35. https://doi.org/10.1016/j.infbeh.2011.09.007

Corley, J., Conte, F., Harris, S. E., Taylor, A. M., Redmond, P., Russ, T. C., Deary, I. J., & Cox, S. R. (2023). Predictors of longitudinal cognitive ageing from age 70 to 82 including APOE e4 status, early-life and lifestyle factors: The Lothian Birth Cohort 1936. *Molecular Psychiatry*, 28(3), 1256–1271.

Costello, E. J., Compton, S. N., Keeler, G., & Angold, A. (2003). Relationships between poverty and psychopathology: A natural experiment. *JAMA*, 290(15), 2023–9. https://doi.org/10.1001/jama.290.15.2023

Costello, E. J., Erkanli, A., Copeland, W., & Angold, A. (2010). Association of family income supplements in adolescence with development of psychiatric and substance use disorders in adulthood among an American Indian population. *JAMA : The Journal of the American Medical Association*, 303(19), 1954–1960. https://doi.org/10.1001/jama.2010.621

DeNavas-Walt, C., & Proctor, B. D. (2014). Income and poverty in the United States: 2013. 72.

Diamond, A. (2006). The early development of executive functions. In *Lifespan cognition: Mechanisms of change* (pp. 70–95). Oxford University Press. https://doi.org/10.1093/acprof:oso/9780195169539.003.0006

Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, 64, 135–168. https://doi.org/10.1146/annurev-psych-113011-143750

Farah, M. J. (2017). The neuroscience of socioeconomic status: Correlates, causes, and consequences. *Neuron*, 96(1), 56–71. https://doi.org/10.1016/j.neuron.2017.08.034

Felson, J. (2014). What can we learn from twin studies? A comprehensive evaluation of the equal environments assumption. *Social Science Research*, 43, 184–199.

Ferrer, E., O'Hare, E. D., & Bunge, S. A. (2009). Fluid reasoning and the developing brain. *Frontiers in Neuroscience*, 3(1), 46–51. https://doi.org/10.3389/neuro.01.003.2009

Friedman, N. P., Banich, M. T., & Keller, M. C. (2021). Twin studies to GWAS: There and back again. *Trends in Cognitive Sciences*, 25(10), 855–869. https://doi.org/10.1016/j.tics.2021.06.007

Ganna, A., Genovese, G., Howrigan, D. P., Byrnes, A., Kurki, M. I., Zekavat, S. M., Whelan, C. W., Kals, M., Nivard, M. G., Bloemendal, A., Bloom, J. M., Goldstein, J. I., Poterba, T., Seed, C., Handsaker, R. E., Natarajan, P., Mägi, R., Gage, D., Robinson, E. B. . . . Neale, B.M. (2016). Ultra-rare disruptive and damaging mutations influence educational attainment in the general population. *Nature Neuroscience*, 19(12), 1563–1565. https://doi.org/10.1038/nn.4404

Garavan, H., Bartsch, H., Conway, K., Decastro, A., Goldstein, R. Z., Heeringa, S., Jernigan, T., Potter, A., Thompson, W., & Zahs, D. (2018). Recruiting the ABCD sample: Design considerations and procedures. *Developmental Cognitive Neuroscience*, 32, 16–22. https://doi.org/10.1016/j.dcn.2018.04.004

Ge, T., Chen, C-Y., Ni, Y., Feng, Y.-C. A., & Smoller, J. W. (2019). Polygenic prediction via bayesian regression and continuous shrinkage priors. *Nature Communications*, 10(1), 1776.

Green, C. T., Bunge, S. A., Briones Chiongbian, V., Barrow, M., & Ferrer, E. (2017). Fluid reasoning predicts future mathematical performance among children and adolescents. *Journal of Experimental Child Psychology*, 157, 125–143. https://doi.org/10.1016/j.jecp.2016.12.005

Greiff, S., Wüstenberg, S., Goetz, T., Vainikainen, M.-P., Hautamäki, J., & Bornstein, M. H. (2015). A longitudinal study of higher-order thinking skills: Working memory and fluid reasoning in childhood enhance complex problem solving in adolescence. *Frontiers in Psychology*, 6, 1060. https://doi.org/10.3389/fpsyg.2015.01060

Hackman, D. A., Cserbik, D., Chen, J.-C., Berhane, K., Minaravesh, B., McConnell, R., & Herting, M. M. (2021). Association of local variation in neighborhood disadvantage in metropolitan areas with youth neuro-cognition and brain structure. *JAMA Pediatrics*, 175(8), e210426. https://doi.org/10.1001/jamapediatrics.2021.0426

Hanscombe, K. B., Trzaskowski, M., Haworth, C. M. A., Davis, O. S. P., Dale, P. S., & Plomin, R. (2012). Socioeconomic status (SES) and children's intelligence (IQ): in a UK-representative sample SES moderates the environmental, not genetic, effect on IQ.. *PLOS ONE*, 7(2), e30320. https://doi.org/10.1371/journal.pone.0030320

Harden, K. P., Turkheimer, E., & Loehlin, J. C. (2007). Genotype by environment interaction in adolescents' cognitive aptitude. *Behavior Genetics*, 37(2), 273–283. https://doi.org/10.1007/s10519-006-9113-4

Hatoum, A. S., Mitchell, E. C., Morrison, C. L., Evans, L. M., Keller, M. C., & Friedman, N. P. (2019)GWAS of over 427,000 individuals establishes GABAergic and synaptic molecular pathways as key for cognitive executive functions'. *bioRxiv*, 674515.

Haworth, C. M., Wright, M. J., Luciano, M., Martin, N. G., de Geus, E. J., van Beijsterveldt, C. E., Bartels, M., Posthuma, D., Boomsma, D. I., Davis, O. S., Kovas, Y., Corley, R. P., Defries, J. C., Hewitt, J. K., Olson, R. K., Rhea, S. A., Wadsworth, S. J., Iacono, W. G., McGue, M., Thompson, L. A., . . . Plomin, R. (2010). The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Molecular Psychiatry*, 15(11), 1112–1120.

Hill, W. D., Davies, N. M., Ritchie, S. J., Skene, N. G., Bryois, J., Bell, S., Di Angelantonio, E., Roberts, D. J., Xueyi, S., Davies, G., Liewald, D. C. M., Porteous, D. J., Hayward, C., Butterworth, A. S., McIntosh, A. M., Gale, C. R., & Deary, I. J. (2019). Genome-wide analysis identifies molecular systems and 149 genetic loci associated with income. *Nature Communications*, 10(1), 5741. https://doi.org/10.1038/s41467-019-13585-5

Hill, W. D., Marioni, R. E., Maghzian, O., Ritchie, S. J., Hagenaars, S. P., McIntosh, A. M., Gale, C. R., Davies, G., & Deary, I. J. (2019). A combined analysis of genetically correlated traits identifies 187 loci and a role for neurogenesis and myelination in intelligence. *Molecular Psychiatry*, 24(2), 169–181. https://doi.org/10.1038/s41380-017-0001-5

Icenogle, G., Steinberg, L., Duell, N., Chein, J., Chang, L., Chaudhary, N., Di Giunta, L., Dodge, K. A., Fanti, K. A., Lansford, J. E., Oburu, P., Pastorelli, C., Skinner, A. T., Sorbring, E., Tapanya, S., Uribe Tirado, L. M., Alampay, L. P., Al-Hassan, S. M., Takash, H. M. S., & Bacchini, D. (2019). Adolescents' cognitive capacity reaches adult levels prior to their psychosocial maturity: Evidence for a, maturity gap, in a multinational, cross-sectional sample. *Law and Human Behavior*, 43(1), 69–85. https://doi.org/10.1037/lhb0000315

Johnson, S. B., Riis, J. L., & Noble, K. G. (2016). State of the art review: Poverty and the developing brain. *Pediatrics*, 137(4), e20153075.

Judd, N., Sauce, B., Wiedenhoeft, J., Tromp, J., Chaarani, B., Schliep, A., van Noort, B., Penttilä, J., Grimmer, Y., Insensee, C., Becker, A., Banaschewski, T., Bokde, A. L. W., Quinlan, E. B., Desrivières, S., Flor, H., Grigis, A., Gowland, P., Heinz, A., Ittermann, B., . . . Klingberg, T. (2020). Cognitive and brain development is independently influenced by socioeconomic status and polygenic scores for educational attainment. *Proceedings of the National Academy of Sciences of the United States of America*, 117(22), 12411–12418.

Kalb, L., Lieb, R., Ludwig, N., Peterson, R., Pritchard, A., Ng, R., Wexler, D., & Jacobson, L. (2023). Association between neighborhood deprivation and child cognition in clinically referred youth. *Journal of Developmental & Behavioral Pediatrics*, 44(8), e543–e550.

Kazma, R., Babron, M.-C., & Génin, E. (2011). Genetic association and gene-environment interaction: A new method for overcoming the lack of exposure information in controls. *American Journal of Epidemiology*, 173(2), 225–235. https://doi.org/10.1093/aje/kwq352

Keller, M. C. (2014). Gene × environment interaction studies have not properly controlled for potential confounders: The problem and the (simple) solution. *Biological Psychiatry*, 75(1), 18–24. https://doi.org/10.1016/j.biopsych.2013.09.006

Korenman, S., Miller, J., & Sjaastad, J. (1995). Long-term poverty and child development in the United States: Results from the NLSY. *Children and Youth Services Review*, *17*, 127–155. https://doi.org/10.1016/0190-7409(95)00006-X

Ksinan, A. J., & Vazsonyi, A. T. (2021). Understanding neighborhood disadvantage: A behavior genetic analysis. *Journal of Criminal Justice*, *73*, 101782. https://doi.org/10.1016/j.jcrimjus.2021.101782

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. (2017). LmerTest package: Tests in linear mixed effects models. *Journal of Statistical Software*, *82*, 1–26.

Lam, M., Awasthi, S., Watson, H. J., Goldstein, J., Panagiotaropoulou, G., Trubetskoy, V., Karlsson, R., Frei, O., Fan, C. C., De Witte, W., Mota, N. R., Mullins, N., Brügger, K., Lee, S. H., Wray, N. R., Skarabis, N., Huang, H., Neale, B., Daly, M. J., Mattheisen, M., . . . Ripke, S. (2020). RICOPILI: Rapid imputation for COnsortias PIpeLIne. *Bioinformatics*, *36*(3), 930–933.

Lee, J. J., Wedow, R., Okbay, A., Kong, E., Maghzian, O., Zacher, M., Nguyen-Viet, T. A., Bowers, P., Sidorenko, J., Karlsson Linnér, R., Fontana, M. A., Kundu, T., Lee, C., Li, H., Li, R., Royer, R., Timshel, P. N., Walters, R. K., Willoughby, E. A. . . . Cesarini, D. (2018). Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals. *Nature Genetics*, *50*(8), 1112–1121. https://doi.org/10.1038/s41588-018-0147-3

Lindström, S., Yen, Y.-C., Spiegelman, D., & Kraft, P. (2009). The impact of gene-environment dependence and misclassification in genetic association studies incorporating gene-environment interactions. *Human Heredity*, *68*(3), 171–181. https://doi.org/10.1159/000224637

Luciana, M., Bjork, J. M., Nagel, B. J., Barch, D. M., Gonzalez, R., Nixon, S. J., & Banich, M. T. (2018). Adolescent neurocognitive development and impacts of substance use: Overview of the adolescent brain cognitive development (ABCD) baseline neurocognition battery. *Developmental Cognitive Neuroscience*, *32*, 67–79.

Martin, A. R., Kanai, M., Kamatani, Y., Okada, Y., Neale, B. M., & Daly, M. J. (2019). Clinical use of current polygenic risk scores may exacerbate health disparities. *Nature Genetics*, *51*(4), 584–591. https://doi.org/10.1038/s41588-019-0379-x

McKean, C., Mensah, F. K., Eadie, P., Bavin, E. L., Bretherton, L., Cini, E., & Reilly, S. (2015). Levers for language growth: Characteristics and predictors of language trajectories between 4 and 7 Years. *PLOS ONE*, *10*(8), e0134251. https://doi.org/10.1371/journal.pone.0134251

Merz, E. C., Strack, J., Hurtado, H., Vainik, U., Thomas, M., Evans, A., & Khundrakpam, B. (2022). Educational attainment polygenic scores, socioeconomic factors, and cortical structure in children and adolescents. *Human Brain Mapping*, *43*(16), 4886–4900.

Merz, E. C., Wiltshire, C. A., & Noble, K. G. (2019). Socioeconomic inequality and the developing brain: Spotlight on language and executive function. *Child Development Perspectives*, *13*(1), 15–20.

Mollon, J., Knowles, E. E. M., Mathias, S. R., Gur, R., Peralta, J. M., Weiner, D. J., Robinson, E. B., Gur, R. E., Blangero, J., Almasy, L., & Glahn, D. C. (2021). Genetic influence on cognitive development between childhood and adulthood. *Molecular Psychiatry*, *26*(2), 656–665. https://doi.org/10.1038/s41380-018-0277-0

Najman, J. M., Hayatbakhsh, M. R., Heron, M. A., Bor, W., O'Callaghan, M. J., & Williams, G. M. (2009). The impact of episodic and chronic poverty on child cognitive development. *The Journal of Pediatrics*, *154*(2), 284–289.e1. https://doi.org/10.1016/j.jpeds.2008.08.052

National Institute of Child Health and Human Development Early Child Care Research Network (2015). Duration and Developmental Timing of Poverty and Children's Cognitive and Social Development From Birth Through Third Grade. *Child Development*, *76*(4), 795–810. https://doi.org/10.1111/j.1467-8624.2005.00878.x

Newhouse, D., Suarez-Becerra, P., & Evans, M. C. (2016), *New Estimates of Extreme Poverty for Children*, 25.

Noble, K. G., & Giebler, M. A. (2020). The neuroscience of socioeconomic inequality. *Current Opinion in Behavioral Sciences*, *36*, 23–28. https://doi.org/10.1016/j.cobeha.2020.05.007

Noble, K. G., Houston, S. M., Brito, N. H., Bartsch, H., Kan, E., Kuperman, J. M., Akshoomoff, N., Amaral, D. G., Bloss, C. S., Libiger, O., Schork, N. J.,

Murray, S. S., Casey, B. J., Chang, L., Ernst, T. M., Frazier, J. A., Gruen, J. R., Kennedy, D. N., Van Zijl, P., Mostofsky, S., . . . Sowell, E. R. (2015). Family income, parental education and brain structure in children and adolescents. *Nature Neuroscience*, *18*(5), 773–778.

Okbay, A., Wu, Y., Wang, N., Jayashankar, H., Bennett, M., Nehzati, S. M., Sidorenko, J., Kweon, H., Goldman, G., Gjorgjieva, T., Jiang, Y., Hicks, B., Tian, C., Hinds, D. A., Ahlskog, R., Magnusson, P. K. E., Oskarsson, S., Hayward, C., Campbell, A., . . . Young, A. I. (2022). Polygenic prediction of educational attainment within and between families from genome-wide association analyses in 3 million individuals. *Nature Genetics*, *54*(4), 437–449. https://doi.org/10.1038/s41588-022-01016-z

Pace, A., Luo, R., Hirsh-Pasek, K., & Golinkoff, R. M. (2017). Identifying pathways between socioeconomic status and language development. *Annual Review of Linguistics*, *3*(1), 285–308. https://doi.org/10.1146/annurev-linguistics-011516-034226

Park, J., Lee, E., Cho, G., Hwang, H., Kim, B., Kim, G., Joo, Y. Y., & Cha, J. (2023). Gene-environment pathways to cognitive development and psychotic-like experiences in children. *eLife*, *12*. https://doi.org/10.7554/eLife.88117.1,

Peñaherrera-Aguirre, M., Woodley, M. A., Sarraf, M. A., & Beaver, K. M. (2022). Social adversity reduces polygenic score expressivity for general cognitive ability, but not height. *Twin Research and Human Genetics*, *25*(1), 10–23.

Perkins, S. C., Finegood, E. D., & Swain, J. E. (2013). Poverty and language development: Roles of parenting and stress. *Innovations in Clinical Neuroscience*, *10*(4), 10–19.

Petscher, Y., Justice, L. M., & Hogan, T. (2018). Modeling the early language trajectory of language development when the measures change and its relation to poor reading comprehension. *Child Development*, *89*(6), 2136–2156. https://doi.org/10.1111/cdev.12880

Polderman, T. J., Benyamin, B., De Leeuw, C. A., Sullivan, P. F., Van Bochoven, A., Visscher, P. M., & Posthuma, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics*, *47*(7), 702–709.

Raffington, L., Czamara, D., Mohn, J. J., Falck, J., Schmoll, V., Heim, C., Binder, E. B., & Shing, Y. L. (2019). Stable longitudinal associations of family income with children's hippocampal volume and memory persist after controlling for polygenic scores of educational attainment. *Developmental Cognitive Neuroscience*, *40*, 100720. https://doi.org/10.1016/j.dcn.2019.100720

Rask-Andersen, M., Karlsson, T., Ek, W. E., & Johansson, Å. (2021). Modification of heritability for educational attainment and fluid intelligence by socioeconomic deprivation in the UK biobank. *American Journal of Psychiatry*, *2020*, https://doi.org/10.1176/appi.ajp.2020.20040462

Rojas, N. M., Yoshikawa, H., Gennetian, L., Rangel, M. L., Melvin, S., Noble, K., Duncan, G., & Magunson, K. (2020). Exploring the experiences and dynamics of an unconditional cash transfer for low-income mothers: A mixed-methods study. *Journal of Children and Poverty*, *26*(1), 64–84. https://doi.org/10.1080/10796126.2019.1704161

Rowe, D. C., Jacobson, K. C., & Van den Oord, E. J. (1999). Genetic and environmental influences on vocabulary IQ: Parental education level as moderator. *Child Development*, *70*(5), 1151–1162. https://doi.org/10.1111/1467-8624.00084

Sauce, B., Bendrath, S., Herzfeld, M., Siegel, D., Style, C., Rab, S., Korabelnikov, J., & Matzel, L. D. (2018). The impact of environmental interventions among mouse siblings on the heritability and malleability of general cognitive ability. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1756), 20170289. https://doi.org/10.1098/rstb.2017.0289

Savage, J. E., Jansen, P. R., Stringer, S., Watanabe, K., Bryois, J., De Leeuw, C. A., Nagel, M., Awasthi, S., Barr, P. B., Coleman, J. R., Grasby, K. L., Hammerschlag, A. R., Kaminski, J. A., Karlsson, R., Krapohl, E., Lam, M., Nygaard, M., Reynolds, C. A., Trampush, J. W., . . . D., Posthuma (2018). Genome-wide association meta-analysis in 269,867 individuals identifies new genetic and functional links to intelligence. *Nature Genetics*, *50*(7), 912–919.

Selzam, S., Ritchie, S. J., Pingault, J.-B., Reynolds, C. A., O'Reilly, P. F., & Plomin, R. (2019). Comparing within-and between-family polygenic score prediction. *The American Journal of Human Genetics*, *105*(2), 351–363.

Singh, G. K. (2003). Area deprivation and widening inequalities in US mortality, 1969-1998. *American Journal of Public Health*, *93*(7), 1137–1143.

Taylor, R. L., Cooper, S. R., Jackson, J. J., & Barch, D. M. (2020). Assessment of neighborhood poverty, cognitive function, and prefrontal and hippocampal volumes in children. *JAMA Network Open*, *3*(11), e2023774–e2023774.

Thompson, W. K., Barch, D. M., Bjork, J. M., Gonzalez, R., Nagel, B. J., Nixon, S. J., & Luciana, M. (2019). The structure of cognition in 9 and 10 year-old children and associations with problem behaviors: Findings from the ABCD study's baseline neurocognitive battery. *Developmental Cognitive Neuroscience*, *36*, 100606.

Tomasi, D., & Volkow, N. D. (2021). Associations of family income with cognition and brain structure in USA children: Prevention implications | molecular psychiatry. *Molecular Psychiatry*, https://www.nature.com/articles/s41380-021-01130-0

Trzaskowski, M., Harlaar, N., Arden, R., Krapohl, E., Rimfeld, K., McMillan, A., Dale, P. S., & Plomin, R. (2014). Genetic influence on family socioeconomic status and children's intelligence. *Intelligence*, *42*(100), 83–88. https://doi.org/10.1016/j.intell.2013.11.002

Trzaskowski, M., Yang, J., Visscher, P. M., & Plomin, R. (2014). DNA evidence for strong genetic stability and increasing heritability of intelligence from age 7 to 12. *Molecular Psychiatry*, *19*(3), 380–384.

Tucker-Drob, E. M., Briley, D. A., & Harden, K. P. (2013). Genetic and environmental influences on cognition across development and context. *Current Directions in Psychological Science*, *22*(5), 349–355. https://doi.org/10.1177/0963721413485087

Tucker-Drob, E. M., Rhemtulla, M., Harden, K. P., Turkheimer, E., & Fask, D. (2011). Emergence of a gene$\\times$ socioeconomic status interaction on infant mental ability between 10 months and 2 years. *Psychological Science*, *22*(1), 125–133.

Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I. I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, *14*(6), 623–628. https://doi.org/10.1046/j.0956-7976.2003.psci_1475.x

Vize, C. E., Baranger, D. A., Finsaas, M. C., Goldstein, B. L., Olino, T. M., & Lynam, D. R. (2023). Moderation effects in personality disorder research. *Personality Disorders: Theory, Research, and Treatment*, *14*(1), 118.

Volkow, N. D., Koob, G. F., Croyle, R. T., Bianchi, D. W., Gordon, J. A., Koroshetz, W. J., Pérez-Stable, E. J., Riley, W. T., Bloch, M. H., Conway, K., Deeds, B. G., Dowling, G. J., Grant, S., Howlett, K. D., Matochik, J. A., Morgan, G. D., Murray, M. M., Noronha, A., Spong, C. Y. . . . Weiss, S. R. B. (2018). The conception of the ABCD study: From substance use to a broad NIH collaboration. *Developmental Cognitive Neuroscience*, *32*, 4–7. https://doi.org/10.1016/j.dcn.2017.10.002

von Stumm, S., Malanchini, M., & Fisher, H. L. (2023). The developmental interplay between the p-factor of psychopathology and the g-factor of intelligence from age 7 through 16 years. *Development and Psychopathology*, 1–10.

Wodtke, G. T., Ard, K., Bullock, C., White, K., & Priem, B. (2022). Concentrated poverty, ambient air pollution, and child cognitive development. *Science Advances*, *8*(48), eadd0285.

Wodtke, G. T., Ramaj, S., & Schachner, J. (2022). Toxic neighborhoods: The effects of concentrated poverty and environmental lead contamination on early childhood development. *Demography*, *59*(4), 1275–1298.

Woodley Of Menie, M. A., Peñaherrera-Aguirre, M., Dunkel, C. S., & Sarraf, M. A (2021). Evidence for the scarr-rowe effect on genetic expressivity in the health and retirement study. *Twin Research and Human Genetics: The Official Journal of the International Society for Twin Studies*, *24*(2), 110–115. https://doi.org/10.1017/thg.2021.13

Wray, N. R., Goddard, M. E., & Visscher, P. M. (2007). Prediction of individual genetic risk to disease from genome-wide association studies. *Genome Research*, *17*(10), 1520–1528. https://doi.org/10.1101/gr.6665407

Zheng, D., Chen, J., Wang, X., & Zhou, Y. (2019). Genetic contribution to the phenotypic correlation between trait impulsivity and resting-state functional connectivity of the amygdala and its subregions. *NeuroImage*, *201*, 115997. https://doi.org/10.1016/j.neuroimage.2019.07.010