

Socioeconomic and Genomic Roots of Verbal Ability

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ABSTRACT

Cognitive ability is one of the most potent and contentious human traits. Many issues surrounding cognitive ability especially those related to heredity is highly charged. Yet, all of the discussion on heredity has been based on non-DNA evidence. It is largely neglected that DNA and environmental data at individual level are indispensable for understanding the development of cognitive ability. In this article, we report findings from a study that uses data from Add Health with genomic measures or polygenic scores (PGS) on cognitive ability. A social-science model including a rich set of SES measures predicts verbal ability well yielding an R^2 of 23.5%. Adding a PGS for education and a PGS for intelligence increases this R^2 by 1.0%. The effects of SES context and the two PGSs are largely independent. Family, school, and neighborhood remain important to verbal ability after an early measure of verbal ability is included as a predictor. Although the influence from the genome is evident, the influences of social environment are critical and cannot be dismissed.

INTRODUCTION

Cognitive ability is one of the most potent human traits. It is potent for at least three reasons. Arguably, cognitive ability is the hallmark of humans that separates them from the other species in the natural world. It is ubiquitous and vital for basic subsistence in deep human history and for contemporary physical and economic well-being. Evidence suggests that more than an inconsequential portion of ability is related to heredity (e.g., Bouchard 1998; Bouchard and McGue 1981). Cognitive ability is as controversial as it is potent. Many issues surrounding cognitive ability especially those related to heredity is highly charged (e.g., Fischer et al. 1996; Herrnstein and Murray 1994; Jensen 1969). Yet, all of the empirical analysis concerning heredity has been based on non-DNA data. It is largely neglected that DNA evidence in combination with socioeconomic (SES) contextual information at individual level is indispensable for understanding how heredity and SES context jointly and interactively mold human cognitive ability. Such DNA evidence just became available recently (Lee et al. 2018; Okbay et al. 2016; Savage et al. 2018; Sniekers et al. 2017).

Understanding the roots of cognitive ability is relevant and important. Cognitive ability has been shown to be one of the best predictors of life outcomes such as educational attainment, occupation achievement, income, wealth, and health (e.g., Farkas et al. 1997; Farkas and Vicknair 1996; Jencks et al. 1979; Taubman and Wales 1974; Taubman and Wales 1972; Wraw et al. 2015). Cognitive ability remains important even if its predictive power for later life outcomes tends to be markedly reduced once education attainment is adjusted. This is so because cognitive ability is a key predictor of academic performance in school as well as educational attainment itself and because it exerts an effect on labor market outcomes independent from education attainment (Jencks et al. 1979; Kerckhoff, Raudenbush and Glennie 2001; Rosenbaum 2001; Spilerman and Lunde 1991). Tests of cognitive ability and related cognitive achievement are routinely and nearly universally used in elementary

and secondary education, college admissions, and admissions of graduate schools and professional schools in the United States (Hauser 2010; Lemann 1999).

The interpretation of the effects of cognitive ability has been intensely contentious. At the heart of the matter is how much weight should be placed on inheritance relative to after-birth learning. Arthur Jensen is considered a leading advocate of the inheritance position. His early work stated, “(t)he best evidence indicates that the means for changing intelligence per se lie in the province of biology rather than psychology or education (Jensen 1969).” He later concluded, “(t)he variance attributed to shared or between-families, environmental factors, which is considerable throughout childhood, gradually shrinks to near-zero between early adolescence and maturity (1997, p.57).” After showing that cognitive ability was a sweeping associative factor of a variety of life outcomes from poverty to educational attainment and to occupational successes, Herrnstein and Murray (1994) professed the view that the differences in intelligence were mostly at the hand of nature and there was little that government policies could change.

Diametrically opposite to Arthur Jensen was Claude Fischer and colleagues (e.g., 1996). They regarded a cognitive test such as the Armed Forces Qualification Test (AFQT) as a test of academic performance from school learning, which is heavily influenced by SES context. Most other commentators on the topic held a view between these two positions, considering cognitive ability a composite outcome that was shaped by both natural endowment and environmental conditions (e.g., Jencks et al. 1979; Scullin et al. 2000; Winship and Korenman 1997).

A fundamental weakness in all previous work on the origin of cognitive ability is the lack of DNA data. As a substitute, biometric studies based on genetic relatedness of twins and sibling were used to estimate the heritability of cognitive ability. Heritability is the proportion of the total variance in a trait due to genetic factors. It estimates the level of

importance of un-identified genetic factors relative to that of non-genetic factors in a trait. It cannot estimate the effects of genotype side by side with the effects of SES context.

Biometric studies yielded a heritability estimate that ranges between 0.2 to 0.8 (e.g., Bouchard 1998; Velden 1997) and a shared non-genetic influence that quickly reduces to zero after childhood (Bouchard 1998; Jensen 1997).

The policy arguments of Jensen (1969) and Herrnstein and Murray (1994) were based on a loose extrapolation of these biometric estimates. Persuasive evidence for policy arguments regarding individuals must be based on necessary genotype and social-context data at individual level. Such analysis cannot be carried out when only biometric data are available. Without such empirical work linking genotype to cognitive ability at individual level, heritability estimates can be easily loosely and even incorrectly interpreted.

With the advent of DNA data and genome-wide association studies (GWAS) on outcomes closely related to cognitive ability, analysis becomes feasible that explicitly incorporates genomic measures. In this article, we report findings from a study that investigates how human genome and environment, especially socioeconomic context, jointly and interactively shape verbal intelligence.

BACKGROUND

Verbal Ability

At Waves I and III, Add Health implemented an abridged version (AHPVT or PVT) of the Peabody Picture Vocabulary Test-Revised (PPVT-R). Our analysis used the percentile rank of PVT ranging 0-100 that was translated from the raw PVT score. PPVT was first published in 1959 and has been revised three times (Dunn and Dunn 2007). Psychological literature considers PPVT an estimate of verbal intelligence (Beres, Kaufman and Perlman 1999; Campbell and Dommestrup 2010). The correlations between PPVT and full-scale intelligence tests were found to be between 0.40 to 0.60 (Beres, Kaufman and Perlman 1999).

Carvajal et al. (1993) empirically estimated the correlation between the Wechsler Intelligence Scale for Children-III and the Peabody Picture Vocabulary Test-Revised among children enrolled in Grades 3, 4, and 5. They obtained statistically significant correlations of .75, .76, and .60, between PPVT and the Wechsler Vocabulary subtest scaled scores, the Wechsler Verbal scores, and the Wechsler Full Scale IQ scores, respectively. Bell et al. (2001) compared PPVT-III and Wechsler Adult Intelligence Scale-Third Edition (WAIS-III) using a sample of 40 adults aged 18-41 with a mean of 22 years. Their results showed that PPVT-III was statistically significantly correlated with the WAIS-III full-scale IQ (FSIQ, 0.40) and verbal IQ (VIQ, 0.46), and that PPVT-III is uncorrelated with performance IQ (PIQ). Compared with full-scale intelligence tests, PPVT is easier-to-use and time-saving. These are decisive advantages in a setting of a large survey like Add Health that includes a large number of survey items besides a verbal ability test.

Cognitive Ability and Life Outcomes

Cognitive ability has a strong positive association with academic performance in school. Examining six longitudinal studies, Jencks et al. (1979, p.102) concluded that the correlational association between the two ranged from 0.40 to 0.63. Using a large, nationally representative and prospective data source of more than 70,000 British students, Deary et al. (2007) showed that cognitive ability measured at 11 years old was a strong predictor of school achievement measured by national examinations on 25 subjects at 16 years old.

The positive association between cognitive ability and educational attainment is also evident. In a study that assembled eight large datasets including several massive state-wide datasets in the United States, Taubman and Wales (1972, p 20) reported a strong positive association between cognitive ability and continuing to college. While about 40-50% of the high school graduates at the 50th percentile of an ability test continued to college, the percentage continuing to college at the 90th percentile reached 80-90% in late 1950s and

1960s when the U.S. higher education expanded. Data from the Wisconsin Longitudinal Study (WLS) showed a positive association between an ability test score and length of schooling (Hauser 2010, p103).

The effect of cognitive ability on later life outcomes becomes complicated when the role of education is taken into account. Drawing data from the National Longitudinal Study of Youth (NLSY), Scullin et al. (2000) investigated the predictive power of AFQT administered when the respondents were 15 to 17 years old, together with educational attainment and race. They reported that although AFQT was predictive of labor market outcomes such as personal incomes, hourly wage, and occupational socioeconomic index (SEI), the predictive powers were substantially reduced when years of schooling was added to the models. The ethnicity analysis showed that cognitive ability had a much smaller effect and educational attainment had a much larger effect for African Americans than for white Americans. Examining about 10,000 Wisconsin high school students followed since their graduation in 1957, Hauser (2010) concluded that the associations of cognitive ability with job performance, occupational prestige, income, and wealth largely disappeared once levels of schooling were adjusted.

The relationship between ability and personal health resembles that between ability and labor market outcomes. When family SES is adjusted and self educational attainment is not adjusted, ability proved to be a strong predictor of general health conditions. A 50-year Scottish longitudinal study on more than 27,000 adults reported that low cognitive ability at 11 years old was strongly associated with higher mortality before age 65 after adjusting for social class and deprivation category (Gruer, Hart and Watt 2017). Using the NLSY-79, Wraw et al. (2015) showed that AFQT was predictive of an array of health indicators measured at age 50 including three overall measures of general health, nine measures of diagnosed illness conditions, and two measures of self-reported conditions after family SES

in childhood was adjusted. Once the respondents' own SES in adulthood including education, income and occupation were adjusted, ability remained a significant predictor for the three overall measures of general health, but for many specific illness conditions, the effects sizes were reduced markedly.

SES Context and Other Environmental Roots of Cognitive Ability

Among social-contextual factors, formal schooling is commonly viewed as the most direct and essential determinant of cognitive ability. Tests of cognitive ability are hardly meaningful out of the context of modern education. In a longitudinal study of the effects of family SES, racial mix, and summer breaks on children's mathematics achievement, Entwisle and Alexander (1992) concluded that "when school is in session, poor children and better-off children perform at almost the same level. Schools seem to be doing a better job than they have been given credit for (pp.82.)" They demonstrated the effect of schooling by showing the loss in mathematics scores after every summer break on the part of children in poverty relative to children not in poverty. Through a natural experiment, Cahan and Cohen (1989) reported a schooling effect distinct from the effect of biological age on an IQ test score that included measurement of fluid intelligence. The study compared fourth graders and fifth graders who were of nearly the same age. Winship and Korenman (1997) reviewed the literature that estimated the effect of education on cognitive ability and carefully reanalyzed the NLSY data used by Herrnstein and Murray (1994). They concluded that each year of education increased 2.7 points of IQ units.

Estimating the effect of education on cognitive ability is challenging because the two are intertwined. While academic training in school contributes to cognitive ability, cognitive ability is also related to how fast and efficiently academic subjects are absorbed and to eventual educational attainment. In this project, we estimated the effect of education on PVT measured at Wave 3 after controlling for the exact same version of PVT administered at

Wave 1. A similar strategy was used when estimating the effect of education on cognitive ability in the absence of DNA data (Winship and Korenman 1997, p 220).

Socioeconomic status is a composite concept with multiple dimensions that comprises family financial resources, knowledge, social connections, and the larger social context. SES is typically measured by parental income, education and occupation, one vs two biological parents in the household, sibship size, quality of neighborhood, and quality of school (e.g., Braveman et al. 2005; Link and Phelan 1996). In a meta-analysis of adoption studies of IQ, Locurto (1990) showed that adoption into high SES families raised the adopted children's IQ by 10-12 points. These adoptive children tended to come from low SES families. Entwisle and Alexander (1992) concluded that family SES status had a larger impact than the interruption of schooling from summer breaks on cognitive test scores in the context of elementary education.

Mechanism studies explained why SES context makes a difference in cognitive test scores. Guo and Harris (2000) investigated the mechanisms through which family SES affected children's cognitive ability. Using data from the National Longitudinal Survey of Youth (NLSY), they showed that the effect of family SES was completely mediated by the intervening mechanisms measured by the latent factors of cognitive stimulation in home, home physical environment, parenting style, and child health at birth. Cognitive ability in that study was measured by four Peabody tests including a reading recognition test, a reading comprehension test, and a mathematics assessment test, and PPVT. Hart and Risley (1995) observed children in 42 families for one hour per week for two and a half years and their calculation suggested that large differences existed in the total number of words heard by children from birth to age four across professional families (45 millions), working class families (25 millions), and families in poverty (13 millions). Using the children of the NLSY79, Farkas and Beron (2004) showed that by 36-month of age, large gaps in vocabulary

already emerged across social classes and racial/ethnic groups, and the gaps were not closed afterwards.

Macro historical trend of intelligence tests provides another source of evidence for environmental influences on cognitive ability. The well-known Flynn effect documented the historical rise in intelligence scores (e.g., Flynn 1987). The rise was interpreted as a result from a societal change that placed a great value on rational reasoning (Flynn 2007) as well as general improvements in education, nutrition and health (Flynn 2009). Recent studies reported a leveling off and even a decline in IQ test scores in developed countries starting in the 1990s (Sundet, Barlaug and Torjussen 2004; Teasdale and Owen 2005).

Researchers have long examined the connections between problem behaviors and academic potential (e.g., Hinshaw 1992; Kessler et al. 1995; Malinauskiene et al. 2011; McLeod and Kaiser 2004). Our full models added measures of binge drinking, marijuana use, smoking, and serious delinquency.

Genomic Roots of Cognitive Ability

For decades, the genomic influences on cognitive ability were estimated via biometric studies. The estimated heritability or the proportion of the phenotypic variance in cognitive ability due to genetic factors has a vast range, depending on factors including sample size, the environment associated with the particular population from which the analysis sample is drawn, non-additive genetic variance, the assumptions of assortative mating and equal environment, and whether the data are based on a single genetic-relatedness such as adoptive-apart data or more than one genetic-relatedness such as the classic twin data (e.g., Bouchard and McGue 1981; Plomin and Loehlin 1989; Velden 1997). Heritability estimates, however, cannot be used to investigate the effects of environmental and genomic roots at the individual level.

The efforts linking DNA variation to ability began in earnest in the early 2000. By then, it was evident that intelligence was a complex trait having a polygenic architecture, meaning it is subject to the influences of a large number of genes each with a tiny effect (Glazier, Nadeau and Aitman 2002). The challenge to find specific genes for cognitive ability was enormous. A human genome consists of millions of genetic variants. Testing whether each one of them predicts ability and setting the critical value for significance at the level of 0.05 would by chance generate a huge number of false positive findings. Although the genome is large, it is finite. The solution was to set a stringent critical value of 5×10^{-8} for significance at genome-wide level and to request a replication of a discovered genetic variant in an independent data source. Initial successes of these genome-wide association studies (GWAS) employing several thousands of individuals were performed on human traits such as type 2 diabetes and body mass index (BMI) (Frayling et al. 2007; Zeggini et al. 2007). The number of GWAS-identified genetic loci were small, but tended to be replicated.

It soon became clear that by far the single most important factor in GWAS was sample size. The most readily available large samples were for anthropometric measures such as human height and body mass index (BMI). The 2010 (Allen et al. 2010) and 2014 (Wood et al. 2014) height GWAS, respectively, had a sample size of 183,727 and 253,288 and identified 180 and 423 genetic loci, respectively. A 2017 study based on 711,428 individuals discovered additional 83 rare (minor allele frequency [MAF]<1%) and low-frequency (1%<MAF<5%) coding variants associated with height (Marouli et al. 2017). The 2010 (Speliotes et al.) and 2015 (Locke et al.) GWAS on BMI, respectively, identified 32 and 97 genetic loci. The later round of GWAS generally included all the observations used in a previous round of GWAS. For example, the 2015 BMI GWAS included all the individuals used in the 2010 BMI GWAS. It was of no surprise that a later GWAS tended to replicate the loci identified in an earlier GWAS.

The prospect of discovering specific genes for intelligence appeared more daunting than the genes for anthropometric measures. Human intelligence seems to be much more complicated than height and weight. Even how intelligence is measured can be highly contentious. The candidate-gene approach was called into question when Chabris et al (2012) reported that they could not replicate the previously published associations between 12 specific genetic variants and cognitive ability. An early GWAS based on 3,511 individuals targeting at intelligence failed to find any signal that was genome-wide significant at $P < 10 \times 10^{-8}$ (Davies et al. 2011). In about the same period, genome-wide SNP data analyzed via GREML (Yang et al. 2010; Yang et al. 2011) continued to yield heritability estimates of intelligence about 40-50% (Chabris et al. 2012; Davies et al. 2011).

A 2013 GWAS study of education based on a discovery sample of 101,069 obtained three SNPs with genome-wide significance, one of the three associated with years of education and the other two with a college degree (Rietveld et al. 2013). The effect sizes of these SNPs were about one tenth of those of height and weight. A polygenic score (PGS) from all common SNPs explained about 2% of the variance in both educational attainment and cognitive function. Two subsequent GWAS of educational attainment in 2016 (Okbay et al.) and 2018 (Lee et al.) assembled 293,723 and 1.1 million individuals, and reported 74 and 1,271 independent genome-wide significant SNPs associated with years of education, respectively. The PGS constructed from all common SNPs in the latest GWAS (Lee et al. 2018) obtained an R^2 of about 12% using data from Add Health. Many of the genetic loci were implicated in biological pathways that played a role during prenatal brain development. The estimated genetic overlap or the shared genetic influences between years of education and cognitive performance was about 70% (Supplementary Table 3.1, Okbay et al. 2016), suggesting that the education-based GWAS-identified genetic variants ought be reasonable predictors of cognitive ability.

Two successive GWAS of cognitive ability in 2017 (Sniekers et al.) and 2018 (Savage et al.) employed 78,308 and 269,867 individuals, respectively. The 2018 GWAS identified 205 independent genome-wide significant loci, which included the 15 identified in the 2017 GWAS. The PGS based on the 2018 GWAS obtained an incremental R^2 of about 5% when predicting cognitive ability. The study used a variety of tests measuring cognitive ability because of the analysis sample was assembled from more than a dozen cohorts. These tests either measure fluid intelligence or were used to calculate the Spearman's ρ . The genetic correlation between intelligence and education was estimated again to be about 0.70 with $P=2.5 \times 10^{-287}$ by the whole-genome LD score regression (Bulik-Sullivan et al. 2015), which computed the correlation between the genetic influences behind intelligence and those behind education. The GWAS-identified genes were mostly expressed in brain tissues. In the present analysis, the latest findings of GWAS for intelligence and the findings of GWAS for educational attainment were used to construct the genomic measures to be included in models predicting verbal ability.

In addition to the two genomic measures of educational attainment and intelligence, we tested a number of other genomic measures. Previous work documented the association between academic achievement and educational attainment with obesity (Della Bella and Lucchini 2015; Roskam et al. 2010), general health (Ickovics et al. 2014), health behavior (McLeod and Kaiser 2004), personality (Lynn and Gordon 1961; McKenzie, Taghavi-Khonsary and Tindell 2000; Phillips and Endler 1982), stress (McEwen 2000), and brain size (Pietschnig et al. 2015). To assess the effects of these potential predictors of cognitive ability, we included a number of PGSs that underlie the phenotypic predictors. These PGSs consist of those based on GWAS for general health-related birthweight (Okbay et al. 2016), BMI (Locke et al. 2015), number of cigarettes smoked per day (Furberg et al. 2010), head circumference (Taal et al. 2012), and each of the big five personality traits (agreeableness,

conscientiousness, extraversion, neuroticism, and openness) (de Moor et al. 2012). We included the PGSs that underlied these traits rather than observed traits themselves to reduce the difficulty of interpretation. For example, an observed BMI at 15 years old is likely a result of a host of genetic and environmental influences the individuals experienced up to that point in life. Some of these environmental influences are likely to be correlated with those that affect cognitive development.

Analysis Plan

We conducted two sets of analyses. In the first set, the outcome comprised two measures of verbal ability at Waves 1 and 3 from each individual. A random-effects model was implemented to investigate how verbal intelligence was molded separately and jointly by genomic legacy as well as SES context. Our final models in the first set of analyses added a set of measures on general health, risk health behaviors, a set of PGSs for general health, BMI, smoking, brain size, and personality traits, and a gene by SES-context interaction analysis testing the hypothesis that SES resources increased genomic effects on verbal intelligence. The second set of analyses parallel the first set with one major modification. The outcome in these analyses consisted of only one measure of verbal ability at Wave 3 for each individual and the measure of verbal ability at Wave 1 was included in the model as a predictor. The analyses took advantage of the twice-measured PVT, once at Wave 1 when the respondents were 7th-12th graders and the second time at Wave 3 about 7-8 years later. The inclusion of an earlier verbal ability as a baseline enabled us to test the hypothesis whether SES context continued to exert an effect after earlier verbal ability was conditioned in the model. The inclusion of the baseline verbal intelligence was crucial for testing the effect of education on ability because of the effect of ability on education.

DATA, MEASURES AND METHODS

Data Source

We used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) (<http://www.cpc.unc.edu/projects/addhealth/>), which is an ongoing longitudinal study of a nationally representative sample of more than 20,000 adolescents in grades 7-12 in 1994-95 in the United States who have been followed for more than 20 years through adolescence and the transition to adulthood. Over the years, Add Health has conducted one in-school survey in 1994-1995, and five in-home interviews in 1994-1995 (Wave 1), 1996 (Wave 2), 2001-02 (Wave 3), 2008 (Wave 4), and 2016-8 (Wave 5). Add Health includes more than 3,000 individuals who are identical twins, fraternal twins, full siblings, and half siblings. Add Health has a multiracial and multiethnic sample. The original purpose of Add Health was to understand the causes of health, health behavior, and educational development with special emphasis on the role of social context at the levels of family, neighborhoods, and communities.

We started with a sample of 9,975 individuals for whom GWAS measures were available. Our analysis sample was reduced to 8,116 who were individuals with at least one of the two verbal ability measures. Our final analysis sample consisted of 8,116 individuals. A total of seven individuals have missing values in one of the following covariates: whether the respondent was in school before the interview, binge drinking, and marijuana use and smoking. The small number of missing values were replaced by the sample means. Fifty-one samples with missing values in race/ethnicity were reclassified into the category of Hispanic and others. Missing values in parental SES variables were coded into a separate category.

In January 2015, Add Health completed genome-wide genotyping on the Wave IV participants who consented to archive their DNA for future studies. Of the 15,701 respondents interviewed, 12,200 of the eligible respondents agreed to archive their DNA for future analysis “related to long term health.” The consent was largely uniform across racial/ethnic groups and yielded more than 12,000 samples for genome-wide genotyping.

Add Health utilized two Illumina platforms for GWAS: the Illumina Human Omni1-Quad BeadChip at first and the Illumina Human Omni-2.5 Quad BeadChip at a later time. The two platforms utilized tag SNP technology to identify and included >1.1 million and 2.5 million genetic markers, respectively, derived from phases 1—3 of the International HapMap Project (Altshuler et al. 2005) and the most informative markers from the 1,000 Genomes Project (1KGP) (Altshuler et al. 2010).

Measures of Verbal Ability, SES Context and Other Covariates

Verbal Ability was measured by an abridged version (PVT) of the Peabody Picture Vocabulary Test-Revised (PPVT-R) implemented twice at Waves 1 and 3 at Add Health. PVT included 87 or half of the items of PPVT-R. Our analysis used the PVT percentile rank as the outcome variable. The variable was constructed by computing the unsmoothed percentile rank for the same-age peers at each Add Health Wave. The resulting percentile rank was comparable across age groups at the same Wave.

SES Context. Mother's and father's education were, respectively, coded into a four-category categorical variable with less than high school, high school graduation and some college, at least college degree, and missing. Mother's and father's occupation originally had 16 categories. They were combined into five categories of none and other, which were two of the original categories of Add Health; manual or blue collar; sales, service, or administrative; professional or managerial; and missing. Household income was total family income obtained from the parental questionnaire at Wave 1 in 1994. Household income was coded into a six-category variable with cutoff points at 20th, 40th, 60th, and 80th percentile plus a category of missing. Family structure was measured by a dichotomous variable taking the value of one if the respondent lived with two biological parents and zero if the respondent was from a household of single parent, stepparent, adopted families, and foster homes at Wave 1. Sibship size was the number of children living in the household at Wave 1.

To capture contextual disadvantage at the neighborhood level, we followed the approach previously used by Wodtke, Harding and Elwert (2011). Neighborhood disadvantage was measured by the first principal component from a principal component analysis of six neighborhood measures that included the proportion of households living below the poverty line, the proportion of adults who were unemployed, the proportion of female-headed households, the proportion of adult residents without a high school diploma, the proportion of residents with a college degree, and the proportion of workers holding managerial or professional jobs. In school was coded as one when the respondent was in a school session when interviewed or the respondent was in school in the past school year when interviewed in a summer break; and in school was coded as zero otherwise.

In generating covariates, we took advantage of the longitudinal design of Add Health. Whenever possible, we created time-varying covariates using repeated measures of Add Health. For example, we generated two measures of neighborhood disadvantage derived from two rounds of principal component analysis using data at Waves 1 and 3, respectively. These two measures of neighborhood disadvantage were included longitudinally in our first set of analysis. Similarly, parental education and occupation were also measured at Waves 1 and 3 and included as time-varying covariates in the first set of analyses.

General Health and Health Behaviors. Self-reported health measured general health of the respondents at Waves 1 and 3 and was based on an answer to the question of "(In general, how is your health?)" The answer had five categories of 1=excellent, 2=very good, 3=Good, 4=fair, and 5=Poor. To facilitate interpretation, the variable was reversely coded so that 1=Poor, 2=Fair, 3=Good, 4=Very good, and 5=Excellent, and treated as a continuous variable in analysis. Binge drinking was measured by the number of days the respondents drank 5 or more drinks in a row throughout a year at Waves 1 and 3. The survey response categories of binge-drinking every day or 3 to 5 days a week, 1 or 2 days a week, 2 or 3 days

a month, 3 to 12 times in the past 12 months, 1 or 2 days in the past 12 months, and never were coded into 6-categorical category variable with never as the reference category.

Marijuana use was measured by the number of times the respondents used marijuana during the past 30 days at Waves 1 and 3. Those who skipped the question legitimately were coded as 0 and the variable had a range of 0 to 900. Then the variable was recoded into categories of 0, 1-5, 6-15, 16-30, and 31 or more. Smoking was measured by the number of cigarettes smoked per day over the past 30 days. Those who skipped the question legitimately were coded as 0 and the variable ranged from 0 to 100. The variable was recoded into categories of 0, 1-5, 16-30, and 31-100.

Serious delinquency was constructed from a delinquency scale using 12 questions at Waves 1 and 3. The scale was similar to those widely used in delinquency and criminal behavior research (Thornberry and Krohn 2000). The 12 questions were about physical fighting that resulted in injuries needing medical treatment, use of weapons to get something from someone, involvement in physical fighting between groups, shooting or stabbing someone, deliberately damaging property, pulling a knife or gun on someone, stealing amounts larger or smaller than \$50, breaking and entering, and selling drugs. Respondents were asked to report how many times they had been engaged in the delinquent behaviors in the past 12 months. The answers are none, 1 or 2 times, 3 or 4 times, and 5 or more times. We recoded them into 0, 1, 2, and 3, respectively, for each item. The scores were then summed up and divided it by twelve. The results were rounded up and coded into categories of none, 1 or 2 times, 3 or more times, and missing. *Demographic variables* included gender, age at Waves 1 and 3, and race and ethnicity,

Genomic Measures

The genomic measure on educational attainment and the genomic measure on cognitive ability drew from the estimates of the 2018 GWAS of years of schooling (Lee et al.

2018) and those of 2018 GWAS of intelligence (Savage et al. 2018), respectively. In the case of educational attainment, the GWAS separately regressed each of a large number of single nucleotide polymorphisms (SNPs) on years of schooling where SNPs were a particular type of genetic variables taking values 0, 1, or 2. The value represented the number of risk alleles at the genetic loci for the phenotype. The GWAS obtained one β of a SNP from each of the large number of regressions, with one regression for each SNP. The study identified 1,271 β s significant at the genome-wide level of 5×10^{-8} . To tap all the predictability of a GWAS, a polygenic score (PGS) for individual i is often constructed using all β s from a GWAS as weights for observed number of risk alleles X_{ij} : $PGS_i = \sum_{j=1}^n \beta_{ij} X_{ij}$, where i indexes individual, j indexes SNP, and n stands for the total number of SNPs used in the calculation. The total number of SNPs included in the calculation were a subset of the GWAS SNPs. The subset was obtained from pruning and clumping the original GWAS SNPs. The SNPs in the subset were uncorrelated or weakly correlated with one another. Pruning is used to remove some of the highly correlated SNPs and clumping is used to keep only one SNP in a section in which the SNPs are highly correlated. When n is equal to the entire set of SNPs, it is equivalent to setting $P=1$ when constructing a PGS from GWAS findings, where P is the critical value in a SNP regression.

To facilitate interpretation, a PGS is typically standardized into a Z score: $PGS_i^s = [PGS_i - M]/\sigma$, where M is the mean PGS averaged over all individuals in the sample and σ is the standard deviation of the PGS. When PGS_i^s is included in a regression model predicting verbal ability, its coefficient can be interpreted as the effect of one standard deviation of the PGS on ability. Thus, the standardized PGSs are a way to estimate the overall genomic influence on a phenotype. We have similarly constructed standardized PGSs for intelligence, BMI, head circumference, cigarette smoke per day, birthweight, and the Big Five personality

traits of agreeableness, conscientiousness, extraversion, neuroticism, and openness. In the rest of this article, we used PGS_i to represent PGS_i^s for simplicity.

ANALYTICAL STRATEGIES

Our Add Health data had a hierarchical structure that induces correlation in the outcome variable and that must be addressed statistically in a regression setting (e.g., Diggle, Liang and Zeger 1994). One source of the hierarchy was due to the inclusion of the two measures of verbal ability per individual. The other source of the hierarchy originated from the study design of Add Health, which included a genetic-informative sample consisting of full siblings, DZ twins, MZ twins, and other related individuals. When these siblings were first collected, they were the source of information on inheritance for biometric studies. In the present study, they were complications that needed to be addressed. In our analysis, we addressed the hierarchy of the data using mixed regression models (Raudenbush and Bryk 2002; Searle 1971; Searle, Casella and McCulloch 1992), which are also referred to as random-effects models or multi-level models. The mixed models have long been established in the statistical literature for analysis of data that are not independent. We implemented two types of mixed models written below in the form of multilevel models for the two sets of analyses discussed earlier. The first was a three-level model for the first set of analyses that addressed the repeated measures of verbal ability in addition to the sibling clusters:

$$\begin{aligned}
 Vability_{tij} &= \beta_{0ij(s)} + \mathbf{SES}'_{ij} \mathbf{B}_1 + \mathbf{G}'_{ij} \mathbf{B}_2 + \mathbf{C}'_{ij} \mathbf{B}_3 + e_{tij}, & (\text{level 1 model}) \\
 \beta_{0ij(s)} &= \beta_{j(s)} + v_{ij}, & (\text{level 2 model}) \\
 \beta_{j(s)} &= \beta_0 + \mu_{j(s)}, & (\text{level 3 model}),
 \end{aligned} \tag{1}$$

where *Vability* stands for verbal ability; the subscripts t, i, j , and s index Add Health Wave, individual, sibship and type of sibship, respectively; \mathbf{SES} , \mathbf{G} and \mathbf{C} are, respectively, vectors of SES measures, PGSs, and other variables including demographic indicators and

principle components for addressing population admixture; \mathbf{B}_1 , \mathbf{B}_2 , and \mathbf{B}_3 are vectors representing the effects of these observed variables; and e_{tij} , v_{ij} , and $\mu_{j(s)}$ are random effects at the level of Add Health Wave, individual and sibship, respectively. We estimated models that distinguished different types of sibship and models that did not make that distinction, which was equivalent to ignoring (s). The two sets of estimated coefficients of observed variables were essentially identical. We only presented estimates from the models that did not make the distinction.

Our mixed model for the second set of analyses was a two-level model that used verbal ability at Wave 3 as the dependent variable and estimated the effects of the same set of predictors while controlling for verbal ability at Wave 1:

$$VabilityW3_{ij} = \beta_{0j(s)} + \alpha VabilityW1_{ij} + \mathbf{SES}'_{ij} \mathbf{B}_1 + \mathbf{G}'_{ij} \mathbf{B}_2 + \mathbf{C}'_{ij} \mathbf{B}_3 + e_{ij}, \quad (\text{level 1 model}) \quad \beta_{0j(s)} = \beta_0 + \mu_{j(s)}, \quad (\text{level 2 model}). \quad (2)$$

Both (1) and (2) were random-intercept models. Conditional on the random intercepts at the individual level and the level of sibling clusters, the siblings and repeated measures were assumed to be independent.

Population admixture or population stratification is a major concern in genetic association studies. Population groups separated over the past 50,000-100,000 years are likely to have developed private genetic variants that differ across population groups and that are unrelated to cognitive ability. If these population groups differ in ability test scores for social-contextual and other environmental reasons and if these variants are not controlled properly, the association between the private variants and test scores could be erroneously interpreted as causal. The error can be avoided by the common practice of including the ten or so largest principle components (PCs) in the regression that links genetic variants to a phenotype (Price et al. 2006). The PCs represent ancestral genetic differences among population groups and

are highly correlated with self-reported race/ethnicity. Gene-environment interaction terms can be added to models (1) and (2) readily.

FINDINGS

Table 1 showed the means and the standard deviations of the continuous variables, and the percentage distribution of categorical variables used in the analysis. The descriptive statistics were provided for Wave 1 data, Wave 2 or Wave 3 data and the combined data from Waves 1, 2 and/or 3. The predictors measured at two Waves were used as time-varying covariates when verbal ability at Waves 1 and 3 were used in the first set of analyses.

Table 1 about here

Table 2 presented the coefficients and their standard errors of a 3-level random-effects model of verbal ability. The three levels were, respectively, at ability measure for either Add Health Wave 1 or Wave 3, individual, and family sibship. The random effects at the individual level accounted for the correlation of the two measures of verbal ability at Waves 1 and 3 from one person. The random effects at the family level addressed the unobserved effects at the level of family sibship. Little surprise that the two random effects at the individual level and family level were highly statistically significant. Alternative models were estimated that replaced a single random effect at the family level by a number of random effects characterizing different types of sibship. The resulting effects of observed covariates and their standard errors remained largely unchanged. Seven of the ten principal components that were meant to control for population admixture were statistically significant and most were highly significant. In each of the four models, 8,116 individuals contributed 15,766 measures of verbal ability, indicating that only about 5% of the individuals contributed a single measure.

Model 1 was a traditional social-science model that did not consider genomic influences. Controlling for demographic factors, all social-contextual characteristics

significantly and simultaneously predicted verbal ability in directions consistent with expectation. Individuals whose mothers with at least a college degree scored 10.7 points higher on the verbal ability test than those whose mothers with an education less than high school. The effect of father's education was weaker. Those whose fathers with at least a college degree scored 3.5 points higher than those whose fathers with an education less than high school. Individuals whose mothers holding a professional and managerial job scored about 1.6 points higher than those whose mothers have a manual or blue-collar job. The effect of father's occupation was larger. The comparable effect of fathers holding a professional and managerial job was about six points relative to a manual and blue-collar job. The effect of household income was substantial. Individuals living in a household in the top 20% income group scored about 10 points higher than those in the lowest 20% income group. Individuals living in a household with 3 to 5 siblings scored six points lower than those who were the only child in a family. An increase in one standard deviation in the index of the neighborhood disadvantage was associated with a decrease of 0.7 point of verbal ability score. Those who were in a school session when taking the verbal ability test scored 5.5 points higher than those who were not in a school session.

Models 2, 3 and 4 showed that when only random-effects and principal components are included in the model, one standard deviation of the education PGS and the ability PGS were associated with 6.2 and 2.0 points of verbal ability, respectively, when these PGSs were singly included in the model. The comparable effects were 6.0 and 1.4 points when the PGSs were simultaneously included in the same model. These results established the importance of the two PGSs.

Table 2 about here

Models 1 through 6 in Table 3 added a component of SES context at a time allowing the effect of each of the SES components to be evaluated when the education PGS and the

ability PGS were already in the model. All of the SES components were significantly predictive of verbal ability. The effects of these SES components were larger than in Model 1 in Table 2 because the SES components in Models 1 through 6 were not adjusted one another as they were in Model 1 in Table 2. Since the two PGSs were included in the models, these results were the first indicator of the extent to which the SES components were associated with the included genomic measures.

Model 7 in Table 3 included all SES components simultaneously as well as the two PGSs. This model could be compared with Model 1 in Table 2 to assess the impact of the two PGSs on the effects of SES factors. All SES components that were significant in the model without the PGSs remained significant in the model with the PGSs. As expected, the inclusion of the PGSs attenuated the SES effects, but the attenuation was moderate. In most cases, the inclusion of the PGSs reduced the SES effects in Model 1 in Table 2 by about 10% or less. For example, the two categories of mother's education "high-school graduation/some college" and "at least college degree", respectively, were associated with 5.24 points and 10.7 additional points in the verbal ability test score relative to "less than high school" in the model without the PGSs. In comparison, the two comparable estimates in the model with the two PGSs were 4.95 and 10.1, which represented reductions of 5.5% and 5.6%, respectively. The models in Table 3 demonstrated the importance of both SES context and the two PGSs for verbal ability. The models also showed that the correlations between the SES predictors and the two PGSs were modest and that the SES factors and the education PGS predicted verbal ability by and large independently.

Table 3 about here

The social-science model (Model 1 in Table 2) had an R^2 of 0.235. The three R^2 s in Models 2, 3, and 4 were 0.152, 0.137, and 0.154, respectively. Models 2, 3, and 4 added the education PGS alone, the ability PGS alone, and both PGSs, respectively, without including

the SES predictors. The estimated two R^2 s were consistent with the estimated effect sizes and levels of significance for the two PGSs. The ability PGS did explain a significant part of the variation in verbal ability net of the education PGS, but the latter seems dominated the genomic effects. With the full social-science model plus the two PGSs, Model 7 in Table 3 had an R^2 of 0.255. Subtracting this R^2 of .235 from Model 1 in Table 2 yields an incremental R^2 of .020 (.255-.235=.020). Part of this incremental R^2 of 2.0% was due to the included PCs measuring population admixture. These PCs were highly correlated with race/ethnicity and the inclusion of the PCs was responsible for the dramatic reduction in the effects of race/ethnicity. Excluding the R^2 due to PCs, the two PGSs accounted for an incremental R^2 of 1%.

Models 1 in Table 4 added a set of predictors measuring general health and health behaviors and a set of PGSs measuring genomic influences on general health, body mass index, number of cigarettes smoked per day, head circumference, and each of the Big Five personality traits to Model 7 in Table 3. The estimated effects tended to be modest or non-existent. Self-reported health was positively associated with verbal ability, an increase of each category in a 5-category scale associated with an increase of one half-point of the ability score. Those who used marijuana 16-30 times over the past 30 days had a verbal ability score about 2.7 points lower than those who did not use the drug in the same period. Those who smoked 16-30 cigarettes per day scored about two points lower than those who did not smoke. Out of the nine PGSs, only PGS for conscientiousness was significantly associated with verbal ability.

The gene-SES interaction analysis included all the interactions between the education PGS and SES components. The chi square test using the loglikelihood values between the model without the interaction terms (Model 1 in Table 4) and the model with the interaction

terms (Model 2 in Table 4) had a chi square value of 30 for 26 degrees of freedom, which is not significant at the level of 5 percent.

Table 4 about here

Table 5 presented the findings of our second set of analyses. The table displayed coefficients and their standard errors of random-effects models predicting verbal ability measured at Wave 3 conditional on verbal ability measured at Wave 1. Otherwise, the models paralleled the models presented in Tables 2, 3, and 4. In other words, these models estimated the effects of SES factors, the education and ability PGSs, and other predictors on verbal ability measured at Wave 3 while controlling for the same version of verbal ability measured at Wave 1 about seven years ago. The sample size or the number of individuals included in the analysis was reduced to 7,647 from 8,116 because an individual had to contribute two measures of verbal ability to be included in the analysis.

Model 1 in Table 5 showed the baseline effects of the education and ability PGSs. As expected, the PVT score at Wave 1 was highly predictive of the PVT score at Wave 3. An increase of one point in the Wave-1 test score was associated with an increase of 0.65 point in the Wave-3 test score. The effect of one standard deviation of the education PGS was reduced from 6.0 points in Model 4 in Table 2 to 2.2 points in Model 1 in Table 5 and the PGS for intelligence lost its significance. Model 2 in Table 5 added the full set of SES predictors. Consistent with expectation, the SES findings tended to diminish in effect size and level of significance. Out of all SES predictors that continued to exert their importance after Wave-1 verbal ability was controlled, years of schooling by Wave 3 and neighborhood disadvantage at Wave 1 were the most remarkable. Each additional year of schooling was associated with almost two points on the PVT test. Neighborhood disadvantages at Wave 1 and 3 were both included in the same model. It was neighborhood disadvantage in earlier life that turned out

to be negatively associated with verbal ability. Living with two biological parents was negative and marginally significant.

Table 5 about here

DISCUSSION AND CONCLUSION

Our social-science model predicted verbal ability well. After age, gender and race/ethnicity were adjusted, mother's education, father's education, mother's occupation, father's occupation, household income, sibship size, neighborhood disadvantage, immigration status, and in school over the past year were all significantly and largely independently associated with verbal ability. This social-science model had an R^2 of 0.235.

However, Model 1 in Table 2 is not a "pure" social-science model. Parental genomes likely have a role in shaping parental SES such as parental education, occupation, and income as well as the neighborhood the family lives and the schools children attend. These SES characteristics are expected to be correlated with children's PGSs predicting verbal ability. The cause of the correlation can be traced to parental genomes which at the same time influence SES environment and transmit 50% of the maternal genetic materials and 50% of paternal genetic materials to children.

Adding children's PGSs to the social-science model tests the relative importance of individuals' own genome vs. the importance of social context. When both in a model (Model 7 in Table 3), both continued to be significantly predictive of verbal ability. All SES predictors that were predictive of verbal ability without the PGSs remained significantly predictive of verbal ability with the PGS. The model that included both SES factors and the ability-related PGSs yielded an R^2 of .255, which represented an increase of an R^2 of 1% over the social-science model, excluding the impact the PCs. The correlation between SES and the PGSs was expected to reduce both estimates when both are in the same model. This was what we observed in the empirical estimates. Both the effect of the education PGS and the effect of

the intelligence PGS were reduced about approximately 23% from 6.00 to 4.59 and from 1.43 to 1.10, respectively. The reductions in effects of SES context were mostly about 10% or less.

The side by side inclusion of the two PGS predicting verbal ability and SES context in the same model yields a number of insights. First, after the two PGSs are taken into consideration, SES still dominates the influences on verbal ability, with the PGSs accounting for an additional R^2 of 1.0%. Second, given the moderate correlation between SES context and the PGSs, the two seem to influence verbal ability largely independently. Third, even if some of SES context is genomic, the non-genomic SES context seems pivotally important. The empirically estimated moderate correlation between the two PGSs and SES context suggests that most of the hugely important SES context is social rather than genomic. How much of SES context is social and how much is genomic will be estimated more accurately and more directly when data become available on both maternal genome and paternal genome. Fourth, when both SES context and the PGSs were included in a model, the effects of both were estimated with greater accuracy. The estimated effects of SES context were less contaminated by parental genomic sources and more truly the effects of non-genomic SES context. The effects of the PGSs predicting verbal ability were, to an extent, purified of parental genomic sources and became more truly a reflection of children's own genome rather than his or her parents' genomes.

Adding measures of general health and health behaviors showed that general health was positively associated with verbal ability and, smoking and marijuana use were negatively associated with verbal ability. These effects were small and adding these factors to the model did not affect the estimates of SES and PGS predictors already in the model. Compared with parental SES factors, general health and health behaviors seemed to be of less consequences to verbal ability. The influences of these classes of predictors appeared to be mostly unrelated.

Adding PGSs for BMI, birthweight, head circumference, number of cigarettes smoked per day, and personality traits hardly yielded any additional significant explanatory power to the model. Two explanations explained the lack of significant findings. First, some of the GWAS were based on a data source that was not sufficiently large to generate a reliable PGS. For example, the GWAS from which the PGSs for the personality traits were derived was based on about 20,000 individuals (de Moor et al. 2012). In contrast, the successful GWAS of educational attainment and cognitive ability were all based on a data source at least several times larger. Second, our findings showed that SES predictors tended to be more impactful than other predictors such as general health and health behaviors. These PGSs were PGSs of non-SES factors. Even if the PGS for BMI was based on a sufficiently large data source (Locke et al. 2015), the link between BMI and verbal ability was likely to be weak or even non-existent. Previously identified link between observed BMI and cognitive ability is worth revisiting because the phenotype of BMI is likely have been subject to substantial SES influences.

Including interaction terms between the education PGS and SES did not add additional explanatory power to the model. Gene-environment interaction analysis holds a particularly prominent position in social genomics that aims to understand the importance of social-contextual environment while taking into consideration of genomic influences. Our analyses indicated that sufficient power of GxE interaction analysis was difficult to come by unless one or a very small number of GxE interaction terms were targeted theoretically to reduce the number of tests.

Fresh insights were obtained by conditioning the analyses on Wave-1 verbal ability. A coefficient of about 0.6 of Wave-1 PVT suggested that the Wave-1 verbal ability predicted the Wave-III verbal ability with 60% accuracy. With the Wave-1 verbal ability included in the model as a predictor, the estimated effects of the education PGS, SES predictors, and

other predictors carried a distinct meaning. These effects represented the effects above and beyond Wave-1 verbal ability, or above and beyond the effects of the measured and unmeasured PGSs and SES and other predictors that had already acted upon Wave-1 verbal ability.

The PGS for education was still significantly predictive of Wave-3 verbal ability, but the effect size was reduced by about two thirds. Out of the SES factors, number of years of schooling by Wave 3 and neighborhood disadvantage were significantly predictive of Wave-3 verbal ability at the level of 0.001; and living with two biological parents were predictive of verbal ability at .10. These three social-structural influences were at the levels of family, school, and neighborhood, respectively. The large effect of year of schooling by Wave 3 was particularly noteworthy. The coefficient of 1.94 implied that each additional year of schooling was associated with about two points of verbal ability. Assessing causal effects of schooling on verbal ability was difficult. While more schooling helps grow verbal ability, individuals with higher verbal ability are likely to seek and attain more education. We took three measures to address the difficulty. First, the model controlled for an earlier version of verbal ability. Second, the model controlled for age at which Wave-3 test was taken. Third, we measured schooling by the number of years of schooling before verbal ability was taken at Wave 3.

Our analysis demonstrated the importance of genomic and social-contextual roots of verbal ability. Unlike the indirect measurement used in biometric studies, our analysis measured genomic roots by the education PGS and the ability PGS based on molecular SNP data at individual level. Though the two PGSs accounted for only about one percent of the variance in verbal ability, the estimated effects were substantial in size, highly significant, and largely invariant to whether the model included variables of SES context, general health and health behaviors, and other PGSs.

In all probability, the measurement of genomic root of verbal ability will improve. Substantially larger samples than currently used could be assembled to hunt more genes in fresh GWAS, which focus common alleles with MAF greater than 5%. A recent study (Marouli et al. 2017) showed that SNPs with MAF<5% can be examined when an extremely large sample is available and that these rare variants tend to have much larger effects than those common variants. These foreseeable advances plus unforeseeable advances will in all likelihood raise the percentage of variance that can be explained by genomic measures.

The cluster of SES factors proved to be of foremost importance to verbal ability with the two GPSs in the model. Social and educational context continued to be important to verbal ability measured between ages 18 and 25 after Wave-1 verbal ability measured at ages 12 and 19 were conditioned in the model. Before the era of molecular genomics, the identification of genomic root of cognitive ability relied on biometric studies. Unequipped with molecular genetic measures at individual level that are naturally correlated to individual-level SES-contextual measures, biometric studies undervalue the importance of environment including SES context. The results from biometric studies are often cited as evidence to support the dismissal of the importance of SES context in policy discussion (Herrnstein and Murray 1994; Jensen 1997).

Social scientists have long considered SES context fundamentally important in shaping life outcomes including cognitive ability (e.g., Duncan, Brooksgunn and Klebanov 1994; Fischer et al. 1996; Hauser 2010; McLeod and Shanahan 1993). With genomic influences measured at individual level and included in the model, we confirmed the fundamental importance of SES context. After all, the worth of cognitive ability including verbal ability depends heavily on the context of modern education and society. At the same time, the modern educational system and society depend on the social fabrics such as family, schools, and neighborhoods to bring about the intellectual potential of individuals. The

eminence of these social fabrics will persist unless their functions are served by institutions other than family, schools, and neighborhoods.

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Table 1. Descriptive statistics: Add Health by wave

Variable	Wave 1 (N=7,674)		Wave 2 or 3 (N=8,092)		Wave 1 & Wave 2 or 3 (N=8,116)	
	Mean or %	S.D.	Mean or %	S.D.	Mean or %	S.D.
PVT Percentile Score (Verbal Ability)	50.2	28.7	50.6	29.3	50.4	29.0
Polygenic Scores (PGS) Predicting Cognitive Ability						
PGS for education	0.0	1.0	0.0	1.0	0.0	1.0
PGS for cognitive ability	0.0	1.0	0.0	1.0	0.0	1.0
SES Context						
Mother's education						
Less than high school	15.1		15.0		15.0	
High school graduation/some college	54.7		54.4		54.6	
At least college graduation	25.8		26.5		26.1	
Missing	4.4		4.1		4.3	
Father's education						
Less than high school	15.0		14.8		14.9	
High school graduation/some college	52.9		52.9		52.9	
At least college graduation	24.8		25.5		25.1	
Missing	7.3		6.8		7.1	
Mother's occupation						
None & other	29.0		21.1		25.0	
Manual or blue collar	16.5		19.3		17.9	
Sales, service, or administrative	22.0		25.5		23.8	
Professional or managerial	27.4		29.8		28.6	
Missing	5.1		4.3		4.7	
Father's occupation						
None & other	14.1		9.5		11.7	
Manual or blue collar	32.4		37.2		34.8	
Sales, service, or administrative	5.1		5.9		5.5	
Professional or managerial	20.4		22.8		21.6	
Missing	28.0		24.8		26.4	
Household income						
0-20 percentile	13.9		13.8		13.9	
20-40 percentile	13.6		13.5		13.5	
40-60 percentile	17.1		16.9		17.0	
60-80 percentile	17.7		17.6		17.6	
80-100 percentile	16.6		16.6		16.6	
Missing	21.1		21.6		21.4	
Family Structure						
Live with Two Biological Parents	0.48		0.48		0.48	
Sibling size						
No sibling	4.1		4.0		4.1	
1 to 2 sibling(s)	49.0		49.0		49.0	
3 to 5 siblings	28.6		28.7		28.7	
6 to 20 siblings	18.2		18.2		18.2	
Other and Missing	0.1		0.1		0.1	
Neighborhood Disadvantages	0.0	1.0	0.0	1.0	0.0	1.0
In School	0.9	0.3	0.1	0.4	0.5	0.5
Demographics						
Age	16.0	1.7	22.3	1.8	19.2	3.6
Gender						
Female	0.5		0.5		0.5	
Male	0.5		0.5		0.5	
Race & Ethnicity						
White	59.9		60.0		59.9	
Black	20.1		20.1		20.2	
Asian	5.4		5.4		5.4	
Hispanic	14.2		14.1		14.1	
Native American & Others	0.5		0.5		0.5	

Immigration Status						
US Born	0.69		0.68		0.68	
Speaking English at Home	0.92		0.92		0.92	
General Health and Health Behaviors						
Self-reported General Health	3.9	0.9	4.0	0.9	3.9	0.9
Binge Drinking						
0 times a year	73.5		50.8		73.6	
1 to 12 times a year	15.6		27.3		15.5	
2 or 3 days a month	4.7		9.0		4.6	
1 or 2 days a week	3.7		8.8		3.8	
3 to 5 days a week/(Almost) Every day	2.5		4.0		2.5	
Marijuana Use						
0 time in past 30 days	84.7		77.2		80.9	
1 to 5 times	10.4		11.3		10.9	
6 to 15 times	2.0		3.7		2.9	
16 to 30 times	2.1		6.3		4.3	
31 times and above	0.8		1.4		1.1	
Smoking						
0 cigarettes per day	74.2		65.3		69.6	
1 to 5 cigarettes	16.8		12.9		14.8	
6 to 15 cigarettes	6.4		12.8		9.7	
16 to 30 cigarettes	2.4		8.5		5.5	
31 to 100 cigarettes	0.3		0.6		0.5	
Serious Delinquency						
None	50.5		71.5		61.3	
1 or 2 times	46.0		25.2		35.3	
3 or more times	2.2		0.4		1.3	
Missing	1.4		2.8		2.1	
Other Polygenic Scores						
PGS for Birthweight	0.0	1.0	0.0	1.0	0.0	1.0
PGS for BMI	0.0	1.0	0.0	1.0	0.0	1.0
PGS for Head Circumference	0.0	1.0	0.0	1.0	0.0	1.0
PGS for Number of Cigarette Per Day	0.0	1.0	0.0	1.0	0.0	1.0
PGS for Agreeableness	0.0	1.0	0.0	1.0	0.0	1.0
PGS for Conscientiousness	0.0	1.0	0.0	1.0	0.0	1.0
PGS for Extraversion	0.0	1.0	0.0	1.0	0.0	1.0
PGS for Neuroticism	0.0	1.0	0.0	1.0	0.0	1.0
PGS for Openness	0.0	1.0	0.0	1.0	0.0	1.0

Table 2. Coefficients (standard errors) of random-effects models predicting verbal ability by SES context and ability-related PGSs, with two measures of verbal ability for each individual at Waves 1 and 3

Predictors	Model 1	Model 2	Model 3	Model 4
	SES context coefficient (S.E.)	Education PGS coefficient (S.E.)	Ability PGS coefficient (S.E.)	Both PGSs coefficient (S.E.)
PGSs Predicting Cognitive Ability				
PGS for Education		6.200(.45)***		6.007(.45)***
PGS for Cognitive Ability			2.029(.39)***	1.434(.38)***
SES Context				
Mother's Education				
Less than high school	-			
High school graduation/some college	5.242(.99)***			
At least college graduation	10.746(1.23)***			
Missing	-0.415(1.81)			
Father's Education				
Less than high school	-			
High school graduation/some college	3.391(.96)***			
At least college graduation	3.473(1.18)**			
Missing	1.275(1.70)			
Mother's Occupation				
Manual or blue collar	-			
None & Others	0.231(.70)			
Sales, service, or administrative	1.320(.72)+			
Professional or managerial	1.611(.74)*			
Missing	-0.313(1.47)			
Father's Occupation				
Manual or blue collar	-			
None & Others	0.468(.75)			
Sales, service, or administrative	2.848(1.03)**			
Professional or managerial	6.082(.67)***			
Missing	1.040(.73)			
Household Income Wave 1				
0-20 percentile	-			
20-40 percentile	3.989(1.02)***			
40-60 percentile	6.034(1.00)***			
60-80 percentile	7.757(1.04)***			
80-100 percentile	9.800(1.10)***			
Missing	1.646(.93)+			
With 2 Biological Parents Wave 1	-0.295(.65)			
Sibship size				
No sibling	-			
1 to 2 siblings	-3.918(1.32)**			
3 to 5 siblings	-6.439(1.35)***			
6 to 20 siblings	-7.967(1.41)***			
Other and Missing	13.343(7.31)+			
Neighborhood Disadvantages	-0.713(.18)***			
In School	5.479(.56)***			
Demographics				
Age	0.732(.08)***			
Female	-2.238(.51)***			
Race and Ethnicity				
White	-			
Black	-18.055(.72)***			
Asian	-7.801(1.23)***			
Hispanic	-8.686(.92)***			
Native Americans and Others	-7.695(3.71)*			
Immigration Status				
US Born	4.422(.55)***			
Speaking English at Home	3.954(1.20)***			
Population Admixture				
PC1		-374.485***	-694.361***	-295.294***
PC2		357.307***	241.943***	339.937***
PC3		-385.081***	-432.084***	-391.083***
PC4		44.477+	56.369*	48.011+
PC5		171.596***	184.998***	176.640***
PC6		-101.202***	-98.751***	-92.553***

PC7		35.205	13.758	38.405
PC8		-79.652**	-80.670**	-79.474**
PC9		-18.876	-41.840	-18.592
PC10		24.839	15.807	24.627
Constant	22.773(2.68)***	50.374(.28)***	50.404(.28)***	50.375(.28)***
Random Effects				
σ_u^2 , family-level	2.692(0.04)***	2.833(.03)***	2.858(.03)***	2.831(.03)***
σ_v^2 , person-level	2.544(0.05)***	2.542(.05)***	2.537(.05)***	2.541(.05)***
σ_e^2 , wave-level	2.796(0.01)***	2.790(.01)***	2.790(.01)***	2.790(.01)***
Model-level Parameters				
(-2)LogLikelihood	143,432	144,287	144,451	144,273
Person-observations	15,766	15,766	15,766	15,766
Number of Families	7,324	7,324	7,324	7,324
Number of Persons	8,116	8,116	8,116	8,116
OLS R-squared	0.235	0.152	0.137	0.154

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Only indicators of level of significance are provided for the estimated PCs.

Table 3. Coefficients (standard errors) of random-effects models predicting verbal ability by SES context and ability-related PGSs, with two measures of verbal ability for each individual at Waves 1 and 3

Predictors	Model 1 Education coefficient(S.E.)	Model 2 Occupation coefficient(S.E.)	Model 3 Income coefficient(S.E.)	Model 4 Family structure coefficient(S.E.)	Model 5 Neighborhood coefficient(S.E.)	Model 6 In school coefficient(S.E.)	Model 7 All SES coefficient(S.E.)
PGSs Predicting Cognitive Ability							
PGS for Education	5.133(.43)***	5.444(.43)***	5.362(.44)***	5.757(.44)***	5.971(.44)***	5.800(.44)***	4.588(.42)***
PGS for IQ	1.171(.37)**	1.305(.37)***	1.253(.37)***	1.333(.38)***	1.361(.38)***	1.331(.38)***	1.095(.36)**
SES Context							
Mother's Education							
Less than high school	-						-
High school graduation/some college	6.724(1.00)***						4.947(.98)***
At least college graduation	13.922(1.21)***						10.092(1.22)***
Missing	0.777(1.72)						-0.786(1.79)
Father's Education							
Less than high school	-						-
High school graduation/some college	4.191(.96)***						3.121(.95)***
At least college graduation	5.759(1.18)***						3.042(1.17)**
Missing	0.547(1.45)						1.046(1.68)
Mother's Occupation							
Manual or blue collar		-					-
None & Others		0.625(.70)					0.163(.69)
Sales, service, or administrative		3.545(.72)***					0.974(.72)
Professional or managerial		6.055(.71)***					1.582(.73)*
Missing		-1.457(1.24)					-0.634(1.46)
Father's Occupation							
Manual or blue collar		-					-
None & Others		0.003(.76)					0.309(.75)
Sales, service, or administrative		4.729(1.03)***					2.659(1.02)**
Professional or managerial		8.261(.65)***					5.591(.66)***
Missing		-0.552(.64)					1.003(.72)
Household Income at Wave 1							
0-20 percentile			-				-
20-40 percentile			5.357(1.03)***				3.744(1.00)***
40-60 percentile			8.696(.99)***				5.353(.99)***
60-80 percentile			12.012(1.00)***				6.814(1.02)***
80-100 percentile			17.032(1.01)***				8.709(1.08)***
Missing			4.199(.93)***				1.398(.92)
With 2 Biological Parents at Wave 1				3.249(.57)***			-0.183(.64)
Sibship size							
No sibling				-			-
1 to 2 siblings				-3.266(1.37)*			-3.609(1.30)**

3 to 5 siblings					-6.868(1.41)***		-5.671(1.33)***
6 to 20 siblings					-9.026(1.47)***		-6.833(1.39)***
Other and Missing					12.850(7.60)+		12.084(7.19)+
Neighborhood Disadvantages						-1.076(.18)***	-0.693(.18)***
In School							6.473(.56)***
Demographics							
Age	0.091(.04)*	0.029(.04)	0.099(.04)*	0.101(.04)*	0.092(.04)*	0.870(.08)***	0.716(.08)***
Female	-2.141(.51)***	-2.288(.51)***	-2.281(.52)***	-2.321(.52)***	-2.486(.53)***	-2.634(.52)***	-2.077(.50)***
Race and Ethnicity							
White	-	-	-	-	-	-	-
Black	1.569(2.66)	2.175(2.67)	1.425(2.69)	4.135(2.73)	4.033(2.74)	3.726(2.72)	-0.209(2.59)
Asian	-3.388(3.35)	-3.085(3.37)	-0.758(3.39)	-1.570(3.43)	-0.860(3.45)	-1.344(3.42)	-3.660(3.26)
Hispanic	-1.408(1.40)	-1.718(1.40)	-1.656(1.41)	-2.203(1.43)	-2.119(1.44)	-2.260(1.43)	-1.126(1.36)
Native Americans and Others	1.484(3.91)	1.895(3.93)	3.102(3.95)	2.280(4.01)	2.486(4.03)	2.416(3.99)	1.489(3.80)
Immigration Status							
US Born	4.509(.56)***	4.608(.56)***	4.503(.57)***	4.515(.57)***	4.887(.58)***	4.704(.57)***	4.121(.55)***
Speaking English at Home	4.892(1.22)***	6.670(1.22)***	5.898(1.23)***	8.769(1.24)***	8.222(1.24)***	8.600(1.23)***	3.707(1.20)**
Population Admixture							
PC1	-429.595***	-369.770***	-294.229**	-404.555***	-434.359***	-455.959***	-282.941**
PC2	116.949+	144.085*	178.676**	139.380*	166.484*	162.662*	127.632*
PC3	-198.696***	-248.015***	-237.030***	-243.276***	-264.309***	-262.267***	-176.929***
PC4	23.618	19.702	20.905	18.190	18.735	19.815	30.903
PC5	139.695***	135.268***	131.594***	124.307***	135.734***	131.474***	132.129***
PC6	-131.597***	-119.966***	-126.233***	-134.386***	-148.398***	-147.541***	-86.651***
PC7	28.300	26.047	31.696	26.388	19.419	19.096	41.333+
PC8	-91.102***	-68.398**	-73.471**	-74.720**	-74.770**	-73.752**	-75.959**
PC9	-10.704	-13.253	-7.041	-7.605	-12.434	-13.762	-0.271
PC10	4.290	15.362	23.604	18.059	21.737	22.015	6.164
Constant	31.190(1.69)***	37.146(1.72)***	33.125(1.74)***	41.657(2.11)***	38.518(1.66)***	20.102(2.28)***	19.230(2.72)***
Random Effects							
σ_u^2 , family-level	2.707(.04)***	2.730(.03)***	2.743(.03)***	2.771(.03)***	2.789(.03)***	2.781(.03)***	2.657(.04)***
σ_v^2 , person-level	2.569(.04)***	2.550(.05)***	2.560(.05)***	2.566(.05)***	2.558(.05)***	2.538(.05)***	2.537(.05)***
σ_e^2 , wave-level	2.792(.01)***	2.797(.01)***	2.790(.01)***	2.790(.01)***	2.791(.01)***	2.794(.01)***	2.795(.01)***
Model-level Parameters							
(-2)LogLikelihood	143,590	143,735	143,739	143,951	144,060	143,964	143,163
Person-observations	15,766	15,766	15,766	15,766	15,766	15,766	15,766
Number of Families	7,324	7,324	7,324	7,324	7,324	7,324	7,324
Number of Persons	8,116	8,116	8,116	8,116	8,116	8,116	8,116
OLS R-squared	0.216	0.210	0.201	0.183	0.176	0.191	0.255

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Only level of significance are provided for the estimated PCs.

Table 4. Coefficients (standard errors) of random-effects models predicting verbal ability by SES context, ability-related PGSs, general health and health behaviors, and other PGSs, with two measures of verbal ability for each individual at Waves 1 and 3

Variables	Model 1 Full Model coefficient(S.E.)	Model 2 Interaction coefficient(S.E.)
PGSs Related to Cognitive Ability		
PGS for Education	4.413(.42)***	0.605(1.75)
PGS for IQ	0.981(.36)**	1.005(.36)**
SES Context		
Mother's Education		
Less than high school	-	-
High school graduation/some college	4.877(.98)***	4.738(.99)***
At least college graduation	9.912(1.21)***	9.817(1.22)***
Missing	-0.722(1.78)	-0.819(1.80)
Father's Education		
Less than high school	-	-
High school graduation/some college	3.125(.95)***	3.313(.96)***
At least college graduation	3.097(1.17)**	3.099(1.18)**
Missing	1.034(1.68)	1.042(1.69)
Mother's Occupation		
Manual or blue collar	-	-
None & Others	0.180(.69)	0.095(.69)
Sales, service, or administrative	0.965(.71)	0.893(.72)
Professional or managerial	1.606(.73)*	1.552(.73)*
Missing	-0.318(1.46)	-0.332(1.46)
Father's Occupation		
Manual or blue collar	-	-
None & Others	0.306(.75)	0.331(.75)
Sales, service, or administrative	2.672(1.02)**	2.779(1.06)**
Professional or managerial	5.485(.66)***	5.504(.68)***
Missing	0.936(.72)	1.010(.72)
Household Income at Wave 1		
0-20 percentile	-	-
20-40 percentile	3.640(1.00)***	3.953(1.03)***
40-60 percentile	5.230(.99)***	5.599(1.01)***
60-80 percentile	6.615(1.02)***	7.016(1.05)***
80-100 percentile	8.477(1.08)***	8.833(1.13)***
Missing	1.262(.92)	1.719(.95)+
With 2 Biological Parents at Wave 1		
	-0.348(.64)	-0.259(.65)
Sibship size		
No sibling	-	-
1 to 2 siblings	-3.634(1.29)**	-3.897(1.30)**
3 to 5 siblings	-5.687(1.33)***	-5.892(1.33)***
6 to 20 siblings	-6.724(1.39)***	-6.801(1.40)***
Other and Missing	12.255(7.17)+	11.848(7.22)
Neighborhood Disadvantages		
	-0.680(.18)***	-0.786(.19)***
In School		
	5.053(.56)***	5.055(.56)***
General Health and Health Behaviors		
Self-reported Health		
	0.488(.21)*	0.478(.21)*
Binge Drinking		
0 times a year	-	-
1 to 12 times a year	0.972(.45)*	0.956(.45)*
2 or 3 days a month	-0.277(.70)	-0.312(.70)
1 or 2 days a week	-0.204(.75)	-0.241(.75)
3 to 5 days a week/(Almost) Every day	-1.171(.99)	-1.215(.99)
Marijuana Use		
0 time in past 30 days	-	-
1 to 5 times	-1.496(.58)**	-1.501(.58)**
6 to 15 times	0.111(1.03)	0.091(1.03)
16 to 30 times	-2.659(.89)**	-2.656(.89)**
31 times and above	0.518(1.60)	0.501(1.60)
Smoking		
0 cigarettes per day	-	-
1 to 5 cigarettes	-0.555(.52)	-0.525(.52)
6 to 15 cigarettes	-1.683(.65)**	-1.648(.65)*
16 to 30 cigarettes	-2.336(.83)**	-2.356(.83)**

31 to 100 cigarettes	-3.053(2.48)	-3.162(2.48)
Serious Delinquency		
None	-	-
1 or 2 times	-0.211(.40)	-0.198(.40)
3 or more times	-0.954(1.53)	-0.894(1.53)
Missing	-2.879(1.16)*	-2.809(1.16)*
Other Polygenic Scores		
PGS for Birthweight	-0.370(.46)	-0.334(.46)
PGS for BMI	-0.628(.56)	-0.617(.56)
PGS for Head Circumference	0.725(.91)	0.692(.91)
PGS for Cigarette per day	-0.493(.36)	-0.467(.36)
PGS for Agreeableness	-0.204(.39)	-0.220(.39)
PGS for Conscientiousness	-0.846(.37)*	-0.852(.37)*
PGS for Extraversion	0.355(.35)	0.375(.35)
PGS for Neuroticism	-0.066(.37)	-0.075(.37)
PGS for Openness	0.836(.44)+	0.830(.44)+
Interactions between SES & Education PGS		
PGS*Mother's Education		
PGS*Less than High School		-
PGS*High School		-0.188(.96)
PGS*College		1.281(1.19)
PGS*Missing		-1.168(1.82)
PGS*Father's Education		
PGS*Less than High School		-
PGS*High School		1.122(.94)
PGS*College		0.997(1.16)
PGS*Missing		2.241(1.75)
PGS*Mother's Occupation		
PGS*Manual or blue collar		-
PGS*Missing		-0.295(.67)
PGS*None & Other		-0.400(.74)
PGS*Sales, service, or administrative		-1.789(.71)*
PGS*Professional or managerial		-0.576(1.52)
PGS*Father's Occupation		
PGS*Manual or blue collar		-
PGS*Missing		0.126(.85)
PGS*None & Other		-0.690(1.23)
PGS*Sales, service, or administrative		-0.314(.75)
PGS*Professional or managerial		0.065(.70)
PGS*Household Income at Wave 1		
PGS*0-20 percentile		-
PGS*20-40 percentile		0.680(.92)
PGS*40-60 percentile		0.477(.98)
PGS*60-80 percentile		0.478(1.06)
PGS*80-100 percentile		0.645(1.12)
PGS*Missing		1.496(.84)+
PGS*Family Structure		
PGS*With 2 Biological Parents at Wave 1		-0.137(.70)
PGS*Sibling size		
PGS*No sibling		-
PGS*1 to 2 siblings		3.337(1.37)*
PGS*3 to 5 siblings		2.757(1.38)*
PGS*6 to 20 siblings		3.177(1.41)*
PGS*Other and Missing		3.016(7.36)
PGS*Neighborhood Disadvantages		-0.292(.15)+
PGS*At School in the Past Year		-0.401(.30)
Demographics		
Age	0.717(.08)***	0.728(.08)***
Female	-2.326(.51)***	-2.379(.51)***
Race and Ethnicity		
White	-	-
Black	-0.288(2.59)	-0.211(2.61)
Asian	-3.401(3.25)	-3.467(3.25)
Hispanic	-1.148(1.36)	-0.992(1.36)
Native Americans and Others	1.431(3.79)	1.612(3.79)
Immigration Status		
US Born	3.893(.55)***	3.921(.55)***

Speaking English at Home	4.273(1.20)***	4.217(1.20)***
Population Admixture		
PC1	-350.729*	-351.162*
PC2	200.507**	203.686**
PC3	-171.204***	-170.923***
PC4	43.381	42.859
PC5	123.981***	126.325***
PC6	-84.702**	-82.235**
PC7	47.315+	47.428+
PC8	-77.312**	-77.982**
PC9	9.024	9.146
PC10	4.833	4.484
Constant	18.175(2.86)***	17.831(2.87)***
Random Effects		
σ_u^2 , family-level	2.650(.04)***	2.646(.04)***
σ_v^2 , person-level	2.536(.05)***	2.538(.05)***
σ_e^2 , wave-level	2.793(.01)***	2.793(.01)***
Model-level Parameters		
(-2)LogLikelihood	143,082	143,052
Person-observations	15,766	15,766
Number of Families	7,324	7,324
Number of Persons	8,116	8,116
OLS R-squared	0.261	0.263

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Only indicators of level of significance are provided for the estimated PCs.

Appendix 1. Descriptive statistics for the sample for the 2nd set of analyses requiring two measure of verbal ability from each individual (N=7,647)

Variable	Wave I		Wave III	
	Mean or %	S.D.	Mean or %	S.D.
PVT Percentile Score (Verbal Ability)	50.2	28.7	50.7	29.3
Polygenic Scores (PGS) predicting cognitive ability				
PGS for Education	0.0	1.0	-	-
PGS for IQ	0.0	1.0	-	-
Years of Education	-	-	13.2	1.9
SES Context				
Mother's Education				
Less than high school	15.1		-	
High school graduation/some college	54.7		-	
At least college graduation	25.8		-	
Missing	4.4		-	
Father's Education				
Less than high school	15.0		-	
High school graduation/some college	52.9		-	
At least college graduation	24.8		-	
Missing	7.3		-	
Mother's Occupation				
None & Other	29.0		-	
Manual or blue collar	16.5		-	
Sales, service, or administrative	22.0		-	
Professional or managerial	27.4		-	
Missing	5.1		-	
Father's Occupation				
None & Other	14.1		-	
Manual or blue collar	32.4		-	
Sales, service, or administrative	5.1		-	
Professional or managerial	20.4		-	
Missing	28.0		-	
Household Income at Wave 1				
0-20 percentile	13.9		-	
20-40 percentile	13.5		-	
40-60 percentile	17.1		-	
60-80 percentile	17.7		-	
80-100 percentile	16.7		-	
Missing	21.1		-	
Family Structure				
Lived with Two Biological Parents	0.5		-	
Sibling size				
No sibling	4.1		-	
1 to 2 sibling(s)	49.0		-	
3 to 5 siblings	28.6		-	
6 to 20 siblings	18.2		-	
Other and Missing	0.1		-	
Neighborhood Disadvantages	0.0	1.0	0.0	1.0
In School				
Demographics	0.9	0.3	0.1	0.4
Age	-	-	22.3	1.8
Gender				
Female	0.5		-	
Male	0.5		-	
Race & Ethnicity				
White	59.9		-	
Black	20.1		-	
Asian	5.4		-	
Hispanic	14.2		-	
Native American or Others	0.5		-	

Immigration Status				
US Born	0.69		-	
Speaking English at Home	0.92		-	
General Health and Health Behaviors				
Self-reported General Health	3.9	0.9	4.0	0.9
Binge Drinking				
0 times a year	73.5		50.8	
1 to 12 times a year	15.6		27.2	
2 or 3 days a month	4.7		9.1	
1 or 2 days a week	3.8		8.9	
3 to 5 days a week/(Almost) Every day	2.5		4.0	
Marijuana Use				
0 time in past 30 days	84.7		77.2	
1 to 5 times	10.4		11.4	
6 to 15 times	2.0		3.8	
16 to 30 times	2.1		6.2	
31 times and above	0.8		1.5	
Smoking				
0 cigarettes per day	74.2		65.5	
1 to 5 cigarettes	16.8		12.7	
6 to 15 cigarettes	6.4		12.8	
16 to 30 cigarettes	2.4		8.4	
31 to 100 cigarettes	0.3		0.6	
Serious Delinquency				
None	50.5		71.6	
1 or 2 times	46.0		25.0	
3 or more times	2.2		0.4	
Missing	1.4		2.9	
Other Polygenic Scores				
PGS for Birthweight	0.0	1.0	-	-
PGS for BMI	0.0	1.0	-	-
PGS for Head Circumference	0.0	1.0	-	-
PGS for Cigarette per day	0.0	1.0	-	-
PGS for Agreeableness	0.0	1.0	-	-
PGS for Conscientiousness	0.0	1.0	-	-
PGS for Extraversion	0.0	1.0	-	-
PGS for Neuroticism	0.0	1.0	-	-
PGS for Openness	0.0	1.0	-	-

Table 5. Coefficients (standard errors) of random-effects models predicting verbal ability at Wave 3 conditional on verbal ability at Wave 1

Predictors	Model 1	Model 2	Model 3	Model 4
	PGS coefficient (S.E.)	SES+PGS coefficient (S.E.)	ALL coefficient (S.E.)	Interactions coefficient (S.E.)
PVT Percentile Score at Wave 1	0.647(.01)***	0.580(.01)***	0.578(.01)***	0.578(.01)***
Polygenic Scores (PGSs) Predicting to Cognitive Ability				
PGS for Education	2.234(.41)***	1.407(.40)***	1.411(.40)***	2.540(1.71)
PGS for IQ	0.176(.35)	0.083(.34)	0.064(.34)	0.064(.34)
SES Context				
Years of Education by Wave 3		1.943(.15)***	1.925(.15)***	1.932(.15)***
Mother's Education				
Less than High School		-	-	-
High school graduation/some college		1.801(1.04)+	1.679(1.04)	1.671(1.04)
At least college graduation		2.903(1.32)*	2.751(1.32)*	2.739(1.32)*
Missing		1.837(1.89)	1.821(1.89)	1.806(1.89)
Father's Education				
Less than High School		-	-	-
High school graduation/some college		-0.699(1.04)	-0.740(1.04)	-0.739(1.04)
At least college graduation		-0.274(1.30)	-0.207(1.30)	-0.202(1.30)
Missing		0.031(1.88)	0.085(1.88)	0.101(1.87)
Mother's Occupation (Wave 1)				
Manual or blue collar		-	-	-
None & Others		0.393(.74)	0.268(.74)	0.264(.73)
Sales, service, or administrative		0.941(.80)	0.907(.80)	0.890(.80)
Professional or managerial		0.664(.82)	0.650(.82)	0.636(.82)
Missing		0.521(1.61)	0.451(1.61)	0.427(1.61)
Father's Occupation (Wave 1)				
Manual or blue collar		-	-	-
None & Others		0.120(.76)	0.204(.76)	0.203(.76)
Sales, service, or administrative		-1.432(1.14)	-1.381(1.14)	-1.362(1.14)
Professional or managerial		1.890(.73)**	1.888(.73)**	1.907(.73)**
Missing		-0.626(.75)	-0.590(.75)	-0.576(.75)
Household Income at Wave 1				
0-20 percentile		-	-	-
20-40 percentile		1.483(.93)	1.380(.93)	1.356(.92)
40-60 percentile		0.901(.92)	0.752(.92)	0.720(.92)
60-80 percentile		0.953(.96)	0.908(.96)	0.882(.96)
80-100 percentile		1.493(1.03)	1.463(1.03)	1.458(1.03)
Missing		-0.497(.87)	-0.618(.87)	-0.627(.87)
With 2 Biological Parents at Wave 1		-1.162(.63)+	-1.194(.63)+	-1.188(.63)+
Sibling size				
No sibling		-	-	-
1 to 2 siblings		0.250(1.21)	0.276(1.21)	0.282(1.21)
3 to 5 siblings		-0.663(1.24)	-0.654(1.24)	-0.648(1.24)
6 to 20 siblings		-1.166(1.30)	-1.072(1.30)	-1.061(1.30)
Other and Missing		4.812(6.59)	4.579(6.58)	4.633(6.58)
Neighborhood Disadvantages (Wave 1)		-0.976(.29)***	-0.988(.29)***	-0.986(.29)***
Neighborhood Disadvantages (Wave 3)		-0.285(.25)	-0.285(.25)	-0.286(.25)
General Health and Health Behaviors				
Self-reported Health (Wave 1)			-0.320(.27)	-0.320(.27)
Binge Drinking (Wave 1)				
0 times a year			-	-
1 to 12 times a year			0.169(.71)	0.162(.71)
2 or 3 days a month			2.215(1.20)+	2.210(1.20)+
1 or 2 days a week			-3.322(1.33)*	-3.307(1.33)*
3 to 5 days a week/(Almost) Every day			-2.999(1.61)+	-2.998(1.61)+
Marijuana Use (Wave 1)				
0 time in past 30 days			-	-
to 5 times			-0.066(.86)	-0.053(.86)
6 to 15 times			-0.970(1.76)	-0.955(1.76)
16 to 30 times			4.712(1.76)**	4.709(1.76)**
31 times and above			-0.997(2.69)	-0.981(2.69)
Smoking (Wave 1)				
0 cigarettes per day			-	-
1 to 5 cigarettes			-0.659(.69)	-0.665(.69)

6 to 15 cigarettes			-1.686(1.08)	-1.721(1.08)
16 to 30 cigarettes			0.567(1.66)	0.514(1.66)
31 to 100 cigarettes			0.815(4.43)	0.761(4.43)
Serious Delinquency (Wave 1)				
None			-	-
1 or 2 times			-0.578(.51)	-0.568(.51)
3 or more times			0.307(1.75)	0.337(1.75)
Missing			-4.927(2.07)*	-4.888(2.07)*
Other PGSs				
PGS for Birthweight			-0.270(.43)	-0.271(.43)
PGS for BMI			-0.294(.53)	-0.297(.53)
PGS for Head Circumference			1.280(.86)	1.274(.86)
PGS for Cigarette per day			0.393(.34)	0.390(.34)
PGS for Agreeableness			-0.328(.36)	-0.327(.36)
PGS for Conscientiousness			-0.055(.35)	-0.058(.35)
PGS for Extraversion			-0.578(.33)+	-0.576(.33)+
PGS for Neuroticism			-0.197(.34)	-0.197(.34)
PGS for Openness			0.371(.41)	0.366(.41)
Interaction				
PGS for Edu*Years of Edu by Wave 3				-0.086(.13)
Demographics				
Age (Wave 3)	0.850(.14)***		0.879(.14)***	0.883(.15)***
Female	-0.753(.48)		-0.931(.49)+	-0.933(.49)+
Race and Ethnicity				
White	-		-	-
Black	1.132(2.41)		1.122(2.41)	1.088(2.41)
Asian	2.641(3.07)		2.601(3.07)	2.580(3.07)
Hispanic	-0.339(1.26)		-0.648(1.26)	-0.671(1.26)
Native Americans or Others	2.293(3.53)		1.935(3.53)	1.934(3.53)
Immigration Status				
US Born	1.066(.52)*		1.038(.52)*	1.027(.52)*
Speaking English at Home	-1.199(1.13)		-1.047(1.13)	-1.037(1.13)
Population Admixture				
PC1	-151.995***	-226.129*	-105.504	-103.845
PC2	18.771	93.332	109.501	108.998
PC3	-54.920*	13.423	31.085	30.362
PC4	-3.058	8.696	-7.791	-7.797
PC5	28.394	36.174	19.341	19.701
PC6	-66.250**	-21.115	-41.813	-42.520+
PC7	-18.412	-8.406	-7.963	-8.119
PC8	-16.950	-13.021	-14.630	-14.711
PC9	-27.943	-19.892	-17.510	-17.684
PC10	3.608	0.544	-3.734	-3.707
Constant	18.165(.52)***	-24.438(3.76)***	-22.726(3.95)***	-22.857(3.96)***
Random Effects				
σ_u^2 , family-level	2.052(.13)***	2.018(.13)***	2.026(.13)***	2.026(.13)***
σ_v^2 , person-level	2.970(.02)***	2.942(.02)***	2.937(.02)***	2.937(.02)***
Model-level Parameters				
-2 Log-Likelihood	68247	67800	67754	67754
Number of Families	7,647	7,647	7,647	7,647
Number of Persons	6,921	6,921	6,921	6,921
OLS R-squared	0.485	0.514	0.517	0.517

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1

Only indicators of level of significance are provided for the estimated PCs.