Nature-nurture interplay: Evidence from molecular genetic and pedigree data in Korean American adoptees*§

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Abstract

In a sample of Korean adoptees who have been quasi-randomly assigned to US adoptive families and who have been genotyped, we examine the influences and interplay of genetics ("nature") and shared family environment ("nurture") on a suite of outcomes. We use molecular genetic data to construct polygenic indices (PGIs) that partially predict the outcomes and examine the effects of the PGIs as well as those of a rich set of family variables. We also compare the resemblance of adoptive and biological siblings to decompose outcome variation into shares due to nature and nurture. We find that both nature and nurture causally affect most outcomes and that the influence of the PGIs tends to be of a similar magnitude to that of the observed family variables. Nurture appears particularly important for education, income, and nicotine usage, while nature has a particularly strong influence on GPA, soft skills, cognitive performance, BMI, and height. Nurture effects on education and smoking are partly traceable to rearing parents' genetics. We investigate interactive effects and obtain suggestive evidence that family socioeconomic status and genetic propensity for educational attainment may be substitutes in the human capital production function for cognitive skills.

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1. INTRODUCTION

The nature versus nurture debate has long captured the interest of scholars and lay people alike. Until the middle of the twentieth century, the debate principally involved qualitative observations and philosophical arguments. Over the past few decades, however, behavioral geneticists and economists have collected and analyzed copious amounts of data from twin, pedigree, and adoption studies, and have found that both genetic and environmental factors matter for most traits (Behrman & Taubman 1989; Polderman et al. 2015; Sacerdote 2011; Turkheimer 2000). Meanwhile, since the turn of the century, the advent of molecular genetic data has enabled researchers to study and identify specific genetic variants that influence traits of interest (Beauchamp et al. 2011; Benjamin et al. 2012).

Despite this progress, much remains to be learned about the relative importance of genetic and environmental factors and the dynamics of how they interact. Behavioral genetics research using twins and extended pedigrees relies on a number of identifying assumptions (Goldberger 1979), and inference from adoption studies is limited due to selective placement (whereby children are placed in adoptive families that resemble their biological families) (Bjorklund et al. 2006). Studies using molecular genetic data are often subject to omitted variable bias since an individual's genetics are correlated with their parents' (Kong et al. 2018) and family members' as well as with cultural and environmental factors. Moreover, the study of interactions between genetics and environmental factors has been hampered by gene-environment correlations; for instance, one's family environment is typically correlated with one's genetics and is thus endogenous (Dudbridge & Fletcher 2014). As a result, to date, few studies have successfully documented plausible gene-by-environment ("GxE") interactions (Dick et al. 2015; Hewitt 2012), with the exception of some recent studies that have utilized experimental or quasi-experimental methods to decouple "G" from "E" (e.g., Barcellos et al. 2018; Kuo et al. 2019; Rimfeld et al. 2018; Schmitz & Conley 2016, 2017a).

In a pioneering study, Sacerdote (2007) analyzed data on Korean American adoptees who had been quasi-randomly matched to their adoptive families. This allowed him to obtain unbiased estimates of the effect of family environment on various adoptee outcomes such as education, family income, height, BMI, drinking, and smoking. Following in Sacerdote's footsteps, Fagereng et al. (2021) studied Korean Norwegian adoptees who had also been quasi-randomly assigned to adoptive families and found a causal effect of family background on wealth and investing behavior.

Here, we build on this line of research by analyzing a unique longitudinal dataset of genotyped Korean adoptees from the Minnesota Center for Twin and Family Research (MCTFR)'s Sibling

Interaction and Behavior Study (SIBS) who were quasi-randomly assigned to adoptive families.[1] This allows us to examine what happens when genetic variation is randomly matched to families. The data include 421 Korean-ancestry individuals as well as 141 European ancestry individuals who were adopted by Minnesotan families before age two, 469 European ancestry biological children from comparable families, and rich parental data. (Throughout, we refer to children raised by their biological parents as "biological children" and, following the norm in genetics, to non-Hispanic Whites as "European ancestry individuals".) Over 80% of the adoptees and biological children, as well as most of their rearing parents, have been genotyped. The quasi-random assignment of the Korean adoptees allows us to use several different empirical methods to study nature-nurture interplay. We define this interplay to include both genetic ("nature") and family environment ("nurture") main effects as well as their interaction in the human capital production function (Biroli et al. 2022).

We utilize data across four waves of the MCTFR-SIBS study that contain information on adoptees and biological children from adolescence through early adulthood. The unique longitudinal nature of these data allows us to analyze a suite of 10 socioeconomic, behavioral, and anthropomorphic outcomes, as well as how these outcomes relate to and interact with underlying genetic and family environmental characteristics. The 10 outcomes include educational attainment (EA, in years of education), college completion, grade point average (GPA), soft skills, cognitive performance, income, number of (alcoholic) drinks per week, nicotine usage, BMI, and height. To capture genetic effects, we construct polygenic indices (PGIs) that partially predict these outcomes based on individuals' molecular genetic data. To capture family environmental effects, we use a rich set of characteristics, including a composite score for family socioeconomic status (SES), family income, marital status, number of children, and a parent disinhibition score for antisocial behavior and substance use; maternal and paternal education; and maternal cognitive performance, BMI, height, drinking, and smoking habits.

After describing our data and conducting empirical tests of the quasi-random placement of the Korean adoptees in Section 2, we begin our analysis in Section 3 with a simple variance decomposition exercise. Following Sacerdote (2007), we fit the standard "ACE" model from behavioral genetics to the adoptee and biological sibling data. The model effectively compares the resemblance of adoptive siblings to that of biological siblings to estimate, under a number of assumptions, the share of the variation in an outcome that is due to genetics, the common family environment (shared between siblings reared in the same family), and other factors. Consistent with Sacerdote (2007) and the behavioral genetics literature, our results indicate that both genetics

[1] Genome-wide genotyping involves assaying a subset of an individual's DNA that can be used to predict much of the genome's variation. A related process is sequencing, which consists of identifying the entirety of an individual's DNA. Sequencing data is more expensive and is less commonly collected in population studies.

and common family environment matter for most outcomes. Though imprecise, our estimates suggest that genetic variation plays a larger role for GPA, soft skills, cognitive performance, BMI, and height, accounting for ~30% of the variation in the first three outcomes and over 60% of the variation in the latter two outcomes. Family environment, by contrast, plays a larger role for educational attainment, college completion, income, and nicotine usage, accounting for ~22-28% of the variation in these outcomes.

Next, in Section 4.2, to more directly demonstrate genetic and family environmental influences and to help elucidate the precise variables that account for the family environmental influences, we estimate regressions of each outcome on observed family variables and on the PGIs. In the sample of Korean adoptees, we again observe influences of both sets of variables. The observed (adoptive) family environmental variables jointly account for 8% of the variation in EA and ~6-7% of the variation in log income and BMI. The PGIs jointly account for ~5-7% in the variation in EA, GPA, cognitive performance, and height. Additional regressions of each outcome on each family variable and each PGI separately reveal interesting patterns. For example, adoptee EA is positively associated with parental EA and family SES and income, and negatively with number of (adoptive) siblings. Drinks per week is positively associated with mother nicotine usage and family SES and income. The adoptee-family elasticity of income is 0.286. And all outcomes are significantly associated at the 5% level with one or more PGIs (except income, which is associated at the 10% level with its PGI).

Because of the quasi-random assignment of the Korean adoptees to families, these estimates support a causal interpretation, though some caveats must be kept in mind. First, the estimated effects of the family variables could be due to ommitted and correlated environmental variables. And second, PGIs are only noisy proxies for true underlying genetic effects, and the estimated *magnitude* of their associations with the outcomes is difficult to interpret precisely. Nonetheless, under a simple and plausible assumption, the share of outcome variation accounted for by the PGIs is a lower bound for the share accounted for by genetic factors, and the nonull outcome-PGI associations we report do imply the existence of causal genetic effects. We discuss these interpretative issues in Section 4.1 and Online Appendix E.

In Section 4.3, inspired by Sacerdote (2007), we estimate for each outcome the treatment effects of being assigned to one of three adoptive family types: small and highly educated (Type 1), large and less educated or of low socioeconomic status (Type 3), and all other families (Type 2). In the sample of Korean adoptees, these treatment effects can be interpreted as causal since assignment to family was quasi-random. In that sample, we find treatment effects of being assigned to a Type 1 vs. a Type 3 family on education (+1.3 years of education and +23 percentage points in the probability of completing college) and cognitive performance (+3.5 IQ points). We also

estimate the effects of being in a particular tercile of the outcome-relevant PGI. The highest tercile of the outcome-relevant PGI is significantly associated with 8 of the 10 outcomes.

In Section 4.4, to examine whether family environmental effects may be traced to the effects of one's rearing parents' genetics, we regress each outcome on their outcome-relevant PGI and on those of their two adoptive parents, in the sample of Korean adoptees. Our results point to the existence of indirect genetic effects, or genetic nurture, whereby rearing parents' genetics impacts their children via the family environment (though environmental correlates of parental genetics could also be at play). Holding an adoptee's PGI constant, one-standard deviation increases in the relevant rearing mother and father PGIs increase the adoptee's educational attainment by 0.55 years and their probability of ever having used nicotine by 6.8 percentage points, respectively.

Finally, in Sections 5 and 6, we turn to the interactive dimension of nature-nurture interplay. We examine whether genetic factors and family SES are technical complements or substitutes in the human capital production function for each outcome, using both molecular genetic (Section 5) and pedigree (Section 6) data. In Section 5, we estimate a negative interaction effect on cognitive performance between family SES and the PGIs for both cognitive performance and EA, but find no significant interactions for the other outcomes. The negative interaction for cognitive performance is robust to a number of checks. It suggests that family SES and the PGI of cognitive performance and of EA are technical substitutes in the production function of cognitive performance, such that the effect of the PGI is larger among adoptees in lower-SES families and the effect of family SES is larger among adoptees with lower PGIs. Notably, this result is inconsistent with the Scarr-Rowe hypothesis from psychology, which suggests that genetic factors play a larger role for cognitive performance among higher SES families (Turkheimer, Eric et al. 2011). However, a power analysis suggests that our statistical power to detect a true GxE interaction may be quite limited due to our small sample size, and our estimates with pedigree data in Section 6 are imprecise. We conclude that our negative interaction result should be seen as tentative and that replication in an independent sample is critical.

Taken together, this study and the extensive set of analyses it reports make several key contributions to the rich literature on nature-nurture interplay for economic traits. First, this study replicates and expands Sacerdote's (2007) pioneering analysis of the effects of adoptive family environment in a different dataset of quasi-randomly matched Korean adoptees. It replicates Sacerdote's general result that adoptive family environment causally impacts a suite of outcomes—including educational attainment, BMI, and drinking. It expands Sacerdote's analysis by documenting causal family environmental effects on additional outcomes—including GPA, soft skills, cognitive performance, and personal income—and by reporting additional results and

statistics to help assess the importance of the family environment.[2] Second, this study leverages the quasi-random assignment of the Korean adoptees to demonstrate the existence of causal effects of genetic factors on the studied outcomes, through comparison of biological and adoptive sibling resemblance and through analysis of PGIs constructed with molecular genetic data. While causal genetic effects have long been demonstrated through various research designs, this study provides convergent evidence via a novel research design.[3] Third, this study finds that the influence of the observed PGIs tends to be of a similar magnitude to that of the observed family variables, though this varies across outcomes. While important family variables may be unobserved and PGIs are only noisy proxies for true genetic factors (especially among the Korean adoptees, as we discuss below), this comparison is informative of the variables currently typically available to researchers; it also constitutes a clear demonstration that both nature and nurture matter. Fourth, this study finds evidence consistent with the existence of causal effects of parental genetics, independent of child genetics, on child education and nicotine usage—i.e., genetic nurture. Though environmental or cultural correlates of parental genetics could also be at play, our unique study design rules out one importance source of bias in studies of genetic nurture: assortative mating. Fifth, to our knowledge, this study is the first to examine how genetics and the family environment interact in a dataset where genetics were quasi-randomly matched to family environments—i.e., where family environment is exogenous. Finally, this study serves as an introduction and guide for economists interested in integrating pedigree and molecular genetic data in applied research, introducing fundamental concepts and models and illustrating with a wide array of analyses.

Overall, using a quasi-random design coupled with molecular genetic data and observed characteristics of the family environment, our results show directly that both genetics and the family environment are important. While these findings echo the general consensus in the literature, many scholars and lay thinkers alike still claim that either nature or nurture have negligible influences. For instance, psychologist Jay Joseph (2015) wrote that "[t]he evidence suggests that genes for ... IQ and personality differences, do not exist" (p. 234) and that "traditionally understood social, political, cultural, class, religious, and familial environmental

[2] Among other such results and statistics, we include standard errors on the ACE model parameter estimates; estimate extended ACE models that allow for genetic and common family environmental factors to be correlated or for their relative importance to vary as a function of age or family SES; and estimate the joint influence of observed family variables for each outcome.

[3] All empirical research designs rely on assumptions. Though assumptions are often reasonable and testable, alternative research designs that rely on alternative assumptions can provide valuable additional evidence. For instance, most adoption studies (e.g., Bouchard et al. 1990) may be biased by selective placement; and within-family regressions of outcomes on individual variants or PGIs (e.g., Howe et al. 2022)—which are seen as the gold standard for establishing causal genetic effects—rely on Mendel's First Law, which implies that which sibling inherits which genetic variants is random and independent of other factors that may impact outcomes. However, failures of Mendel's First Law due to meiotic drive have been documented (though not in humans, to the best of our knowledge) (Burt & Trivers 2006).

influences remain the best explanation for differences in human behavior" (p. 251). And behavioral geneticist Robert Plomin (2019) wrote that "growing up in the same family with someone does not make you resemble them beyond your genetic similarity" and that "environmental influences shared by family members do not make a difference" (p. 73). While the latter may conceivably hold for certain psychological and anthropomorphic traits in adulthood, our results imply that family environment does have non-negligible effects on a suite of outcomes, including effects in adulthood on drinking, educational attainment, college completion, and income.[4] Indeed, our findings and those of others (Fagereng et al. 2021; Sacerdote 2007; Silventoinen et al. 2020) are consistent with Jencks' point that psychological traits may be different than economic outcomes (Jencks 1972, as cited in Sacerdote 2007), and suggest that for many of the outcomes of interest to economists and social scientists, the family environment continues to matter after childhood.

More generally, our results help unlock the black box of the human capital production function and contribute to a large body of work in economics that studies nature-nurture interplay in the intergenerational transmission of economic outcomes (e.g., Behrman & Taubman 1976; Björklund & Jäntti 2011; Black et al. 2020; Fagereng et al. 2021). Economists studying the human capital production function have begun integrating genetic data into their analyses (Biroli 2015; Conti & Heckman 2010; Houmark et al. 2021; Rustichini et al.), and the presence (or absence) of genetic effects and their interactions with the environment can provide supporting evidence for theoretical predictions (Biroli et al. 2022). Further, though estimates of heritability and of family environmental effects may not be used to conclusively determine whether policy interventions may ameliorate a trait (Goldberger 1979), policy interventions that mimic environmental improvements we observe across families are unlikely to have large treatment effects on traits with negligible family environmental effects. Our findings of substantial family environmental effects for a suite of traits are consistent with findings that early childhood policy interventions that "emulate the mentoring environments offered by successful families" can have positive long-run effects on non-cognitive skills and socio-economic outcomes (Kautz et al. 2014).

[4] Our outcomes GPA, soft skills, cognitive performance, drinks per week, ever used nicotine, BMI, and height were measured (fully or in part) before adulthood. For some traits, including cognitive performance, drinking, and smoking, family environmental influences decrease and heritability increases as one reaches adulthood (Bouchard 2013; Kendler et al. 2008). For drinks per week, we verified that significant family environmental influences remained when using only data from the second follow-up assessment, when participants were 22 years old on average.

2. DATA

2.1 The Minnesota Center for Twin and Family Research (MCTFR)'s Sibling Interaction and Behavior Study (SIBS)

We analyze a sample of adoptive and non-adoptive families from the Sibling Interaction and Behavior Study (SIBS), a longitudinal study of the Minnesota Center for Twin and Family Research (MCTFR) (McGue et al. 2007). The basic sampling unit in SIBS is a four-member adoptive or non-adoptive nuclear family consisting of a pair of adolescent offspring (at the time of their initial assessment) and their rearing parents. Many of the sampled families have more than two children, but only two children were surveyed in all families. Adoptive and non-adoptive families were recruited through private adoption agencies in Minnesota and through birth records, respectively.

Adoptive families eligible for the study had (1) an adolescent who was adopted between the ages of 11 and 21 and who was placed in their adoptive family's home prior to age two years (mean adoption age of 4.7 months across all adoptees; SD=3.4 months), and (2) another adolescent in their home who was not biologically related to the adoptee. The second child could have been adopted (and placed prior to age two) or biologically related to one or both parents. Non-adoptive families eligible for the study had two full biological adolescent siblings and were selected so that the siblings were comparable to the adoptive sibling pairs in gender and age. In addition, families needed to be living within driving distance of (as much as ~300 miles away from) the labs at the University of Minnesota, siblings could not be more than five years apart in age, and adolescents could not have any physical or mental handicap that would make it difficult for them to complete a daylong intake assessment.

Of the families who were invited to participate, 409 (63%) of the adoptive families and 208 (57%) of the non-adoptive families completed an intake assessment. Differences in socioeconomic status between invited families who completed the assessment and those who did not are minimal (McGue et al. 2007). Of the 409 adoptive families, 124 families were mixed "adoptive/biological families", meaning they had one adolescent adoptee and one adolescent that was a biological child of one or both parents, and 285 were "adoptive/adoptive families" in which both adolescents participating in the study were adopted prior to age two and not biologically related. Our analyses focus primarily on 421 Korean adoptees and secondarily on 471 European ancestry biological children and 141 European ancestry adoptees, as well as on their European ancestry parents. For all practical purposes, the assignment of the Korean adoptees to the adoptive families was random conditional on gender, as we discuss and formally test in Section 2.

SIBS data were collected across four main survey waves, referred to as intake, the first follow-up, the second follow-up, and the third follow-up. Our analyses include data from intake and all three follow-up waves. On average, SIBS participants were in mid-adolescence at intake (mean age=15.0, SD=1.9), late adolescence at their first follow-up (18.3, 2.1), early adulthood at their second follow-up (22.4, 3.5), and in their late-20s through mid-30s at the third and most recent follow-up (32.0, 2.7). At intake, adolescent offspring and their parents completed an in-person five-hour assessment that included interviews and questionnaires, neurocognitive testing, and videotaped family interactions. Follow-ups were conducted via in-person or telephone-based assessments. In 2008 and 2009, blood, saliva, or buccal samples were collected from children and their parents and genotyping was performed (Miller et al. 2012).[5]

One potential limitation of our study is that adoptive families do not constitute a random subset of the population, and are more likely to exhibit greater financial security, marital stability, and mental health than the general population (McGue et al. 2007). For example, in our sample, 65% of the Korean adoptees have an adoptive mother with a college degree and 68% have a father with a college degree, compared to 49% and 48% for the European ancestry biological children.[6] The restricted range in environmental exposures for adopted children may lead to an underestimate of shared environmental effects (Stoolmiller 1998, 1999). Overall, McGue et al. (2007) report that adoptees in the SIBS experienced an 18% reduction of variance in family SES and a 41% reduction of variance in parent disinhibitory psychopathology, though range restriction was found to have no effect on the variance in delinquency, drug use, or IQ among adoptees compared to biological children.

2.2 Variables

2.2.1 Outcomes

Our analysis focuses on a suite of 10 adoptee and biological child outcome variables:

Educational attainment (EA): Years of educational attainment were determined using self-reports from the third follow-up (when participants were 32 years old on average). Participants were asked how many years of education they received, where 12 years is equivalent to a high school degree, 16 years is equivalent to a 4-year college degree, and 20 years is equivalent to a Ph.D. degree.

[5] Typically, a phlebotomist was sent to the participant's home to draw a blood sample, from which DNA was extracted. DNA was successfully obtained from approximately 80% of all eligible offspring and 80% of all eligible parents.

[6] The college graduation rates of the non-adoptive fathers and mothers in our sample are higher than the rates in the general population of adults, but are similar to those of fathers and mothers who live with two or more of their own children in the area where the SIBS families lived (McGue et al. 2007).

College: Receipt of a college degree was also determined using self-reports from the third follow-up. Responses were coded as one if a participant reported receiving a four-year college/university degree, a master's degree, or a doctoral degree, and zero otherwise.

Grade point average (GPA): GPA was assessed using maternal reports of children's overall GPA at the initial intake assessment (when participants were 15 years old on average). Data on actual grades were not collected due to differences in grading formats and standards across school systems; rather, mothers' responses could take on values 0 (failing or much below average), 1 (D's or below average), 2 (C's or average), 3 (B's or better than average), and 4 (A's or much better than average) (Johnson et al. 2007). Johnson et al. (2007) found that these maternal reports were similar or as accurate as teacher reports. Different children were in different school grades at intake and the GPA variable captures GPA at the grade a child was in at intake.

Soft skills: To evaluate soft skills, we used a previously validated composite score of noncognitive skills based on six personality and behavioral measures that were evaluated at intake (McGue et al. 2017). The first three measures, Alienation, Aggression, and Control, capture personality constructs related to self-control and emotional regulation that are relevant for academic success. These were each assessed using 18 items answered on a 4-point scale. The next two measures, Academic Effort and Academic Problems, were based on maternal reports of behavior at school. The Academic Effort scale comprises eight items rated on a 4-point scale that capture effort (e.g., "Turns in homework on time") and motivation (e.g., "Wants to earn good grades). And the Academic Problems scale comprises three items rated on a 4-point scale that assess problematic behaviors in school (e.g., "Easily distracted in class"). The sixth measure in the soft skills composite, Externalizing, was evaluated from interviews of the participating adolescent and their mother and includes the total number of symptoms of attention deficit/hyperactivity disorder (ADHD), conduct disorder (CD), and oppositional defiant disorder (ODD). The final soft skills score was computed by summing all six standardized noncognitive measures and standardizing the result so that it has a mean of zero and unit variance.

Cognitive performance: Cognitive performance was evaluated at intake using an abbreviated version of the WISC-R (for adolescents age 15 years or younger) or the WAIS-R (for adolescents age 16 and older) (McGue et al. 2007). To account for potential differences in the type of cognitive test that was administered, we included a dichotomous variable that is equal to one if an individual was age 16 or older at intake when analyzing cognitive performance. The abbreviated forms of the WISC-R and WAIS-R tests consist of four subsets: two verbal subtests on vocabulary and information and two performance subtests that involve block design and picture arrangement. These subtests were selected because performance on them correlates 0.90 with overall performance computed from all subtests (Kaufman 1990). Scores for the four subtests were adjusted using standard procedures to obtain an IQ score and normed to account for age effects.

Personal income: annual net income (before taxes) from an individual's current job was assessed at the third follow-up assessment. Individuals selected their net income from a series of pre-defined income brackets that increased in increments of $10,000 and ranged from 1 ("I do not have a paying job") to 16 ("$200,000 or more"). We took the midpoint of each category to generate income in dollars, except for the second category ("Less than $10,000")—which we set to $7,500—and the last category—which we set to $250,000. We then used the natural log of income for the analysis, dropping individuals without a paying job.

Drinks per week: Information on alcohol use was assessed at the first three waves (intake and the first two follow-ups). Drinks per week was constructed using participant self-reports from categorical variables that assessed frequency of drinking and quantity of drinks consumed when drinking.[7] Both variables were converted to a weekly scale by taking the midpoint of each numeric range and then normalizing values reported per day or per month to their per-week equivalent. Frequency per week was then multiplied by quantity per week to create the drinks per week variable for each of the first three waves. Participants with more than 50 drinks per week were top coded at 50. A cross-wave summary drinks per week variable was then created by partialling out the effects of age, sex, and age interacted with sex at each wave and then taking the average of the residuals across all three waves.

Ever used nicotine: Participants were asked if they ever smoked or used nicotine at least once without their parents' permission at intake and at the first two follow-up visits. A private, computer administered questionnaire was used. Participants received a one for this variable if they reported smoking or using nicotine at any of the three waves, and zero otherwise. Because the sample was relatively young at intake and in the first two follow-up waves, these assessments are capturing experimental use in adolescence, which research has shown can be a predictor of substance misuse in adulthood (Everett et al. 1999; Grant & Dawson 1997; McGue et al. 2001).

Body mass index (BMI): Height and weight were measured in person for approximately 85% of respondents and were self-reported over the phone for the remaining respondents in the first follow-up wave (when participants were 18 years old on average). Height was recorded in centimeters and weight was recorded in pounds. We converted height in centimeters to meters and weight in pounds to kilograms to calculate BMI.

Height: As just mentioned, self-reported or measured height in centimeters was assessed at the first follow-up wave.

[7] Participants could report frequency of drinking as "non-drinker", "less than once a month", "1-3 times per month", "1-4 times per week", "daily", or "more than once per day". Quantity of drinking was reported as "non-drinker", "1-3 drinks", "4-6 drinks", "7-10 drinks", "11-20 drinks", "21-29 drinks", or "30 or more drinks".

2.2.2 Polygenic indices and principal components

The human genome contains 23 pairs of chromosomes (one set from each parent) comprising a total of roughly 3.1 billion pairs of nucleotides.[8] Most nucleotides are identical across humans, but some vary. There are several types of genetic variants, but by far the most widespread and widely studied are single nucleotide polymorphisms (SNPs). A SNP is a location in the genome where the nucleotides vary across the population. The vast majority of SNPs have only two possible variant nucleotides in the population. To create a variable for each SNP, one of the two nucleotides is selected as the reference and the number of reference nucleotides one has at the SNP is counted.[9]

Over the past decade, large-scale genome-wide association studies (GWAS) of various traits have yielded major advances in human genetics. A GWAS meta-analyzes large samples of genetic data and regresses a trait of interest on millions of SNPs separately to find variants that are associated with the trait after stringent multiple testing corrections are applied. An important application of GWAS findings in social science research has been the use of polygenic indices (PGIs) that utilize individuals' genotypes and each SNP's GWAS coefficient estimate for a trait of interest to partially predict that trait. Specifically, a simple version of the PGI predicting trait Y can be constructed by multiplying the number of reference alleles at each SNP (0, 1, or 2) by that SNP's GWAS coefficient estimate for trait Y, and then summing these values across all SNPs:

$$PGS_i^Y = \sum_{j=1}^{M} \hat{\beta}_j^Y x_{ij}, \quad (1)$$

where j indexes the SNPs, i indexes the individuals, $\hat{\beta}_j^Y$ is the coefficient estimate for SNP j from the GWAS of trait Y, and x_{ij} is the number of reference nucleotides for individual i at SNP j.

We computed or obtained PGIs predicting seven outcomes: EA, cognitive performance, income, drinks per week, whether one ever was a smoker, BMI, and height, for the European and Korean ancestry MCTFR-SIBS participants and their parents. We used these PGIs in our analyses involving these seven outcomes.[10] We also used the EA PGI in our analyses involving college, GPA, and soft skills, because trait-specific PGIs were either unavailable or were less predictive of these outcomes than the EA PGI, which was constructed using data from a large GWAS involving

[8] Technically, there are 3.1 billion *pairs* of *nucleotide pairs*. The pairs refer to the separate contribution from mother and father, while the nucleotide pairs are due to the paired helical structure of DNA. Due to a property called complementarity, the nucleotides in the nucleotide pairs are always matched in a predictable fashion, so it suffices to observe one nucleotide to know the other. For that reason, following common practice, we will only refer to 3.1 billion *pairs of nucleotides*.

[9] Since one may have inherited the reference nucleotide from neither, either, or both their mother and father, the SNP variable is equal to 0, 1, or 2. For more details, see Beauchamp et al. (2011b) and Benjamin et al. (2012).

[10] We used the PGI predicting whether one ever was a smoker in our analyses of our "ever used nicotine" outcome; though the two traits differ, they are similar.

more than one million individuals.[11] We standardized all PGIs separately in the MCTFR-SIBS samples of European and of Korean ancestry individuals, so that they have mean zero and unit variance in each sample. Online Appendix A and Online Appendix Table A.1 provide technical details on how we constructed or obtained the PGIs and list the GWAS estimates we used to construct the PGIs.

To control for confounding from population stratification—which arises when SNPs are correlated to genetic, environmental, or cultural factors that impact traits of interest[12]—we used the software PLINK (Chang et al. 2015) to apply principal components analysis (PCA) to the SNP data to compute the main axes of genetic variation that arise from systematic ancestry differences. As is standard practice (Beauchamp et al. 2011; Price et al. 2006), we then used the top 10 resulting principal components (PCs) as controls in all our empirical analyses that involve a PGI. For the European ancestry individuals, we used PCs computed in the entire European ancestry (i.e., non-Hispanic White) MCTFR-SIBS sample, and for the Koreans, we used PCs computed in the sample of Koreans only. Further, in our analyses that involve PGIs, when we analyze the European ancestry biological children (and, in some analyses, the European ancestry adoptees), we always analyze them separately from the Korean adoptees.

For each of our 10 outcomes, Figure 1 shows the incremental R^2 of the outcome-relevant PGI in the samples of Korean adoptees, European ancestry parents, and European ancestry biological children. Incremental R^2 is defined as the increase in R^2 from adding the PGI to a regression of the predicted outcome on sex, birth year, and the top ten PCs. Among (European ancestry) parents, the PGIs predict approximately 28%, 10%, 8%, and 4% of the variation in height, EA, cognitive performance, and income, respectively.

As indicated above, we used coefficient estimates from GWAS of European ancestry individuals to construct all the PGIs, including those for the Korean adoptees. To avoid bias due to population stratification, GWAS are typically performed within ancestry groups, and GWASs of non-European individuals have so far not reached very large sample sizes for the outcomes we analyze. PGIs constructed with estimates from GWASs of a given ancestry typically predict poorly in individuals of different ancestries (Carlson et al. 2013; Martin et al. 2017), so it was to be

[11] Specifically, the largest GWAS of college to date (to the best of our knowledge) involved only 280,007 individuals (Okbay et al. 2016); unsurprisingly, the PGI of EA is a better predictor of college. For soft skills, a PGI constructed with data from a GWAS of non-cognitive skills (Demange et al. 2021) performs substantially worse than our PGI of EA. And (to the best of our knowledge) no large-scale GWAS of GPA exists.

[12] Hamer (2000) provides an interesting illustrative example of how such confounding can happen.

expected that our PGIs would have lower predictive power in the sample of Korean adoptees.[13,14] Despite this limitation, we achieve a fairly high level of PGI prediction in that sample: our PGIs account for approximately 7%, 6%, 5%, and 2% of the variation in height, EA, cognitive performance, and income, respectively.

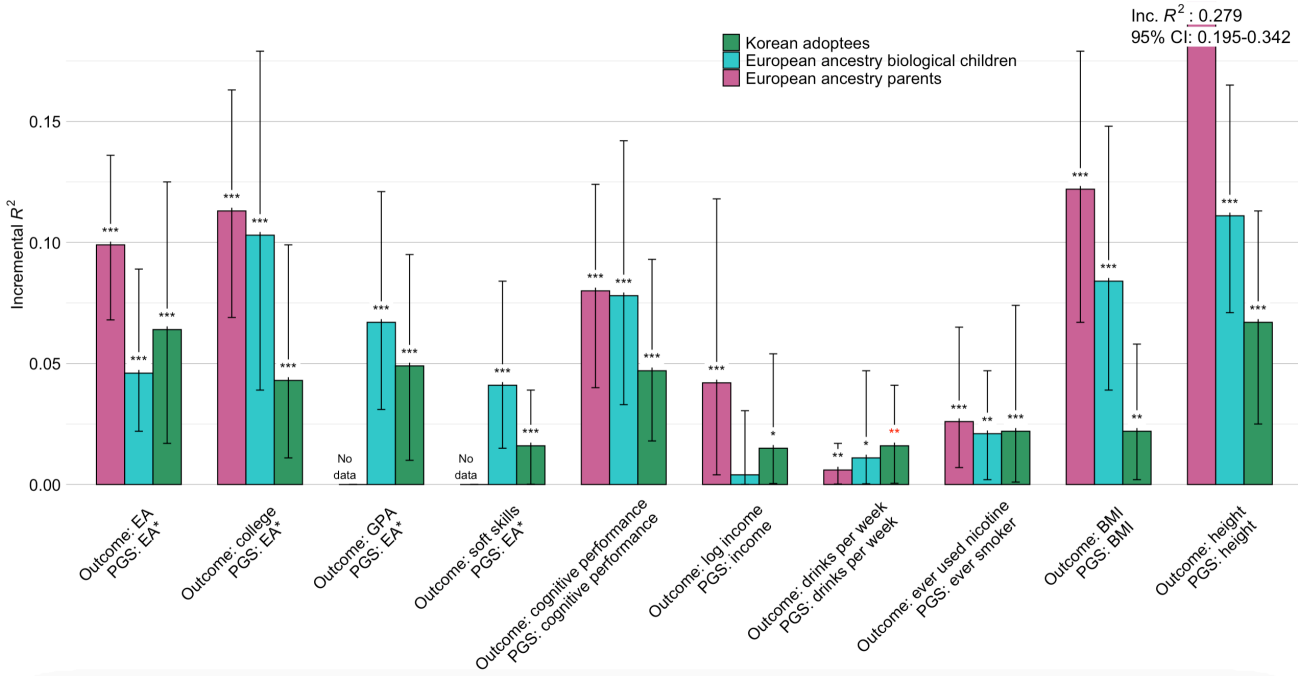

Figure 1. Incremental R^2 of the outcome-relevant PGI for each of the 10 outcomes. Incremental R^2 is defined as the increase in R^2 from adding the PGI to a regression of the predicted outcome on sex, birth year, and the top ten PCs. For the binary outcomes (college and ever used nicotine), a logistic regression was estimated and the Nagelkerke's R^2 was used. The stars above the bars indicate the significance of the coefficient of the PGI in the regression of the outcome on the PGI (and the controls); red stars indicate the coefficient has the opposite sign from what one would expect (likely due to lack of statistical power). Error bars indicate 95% confidence intervals for the incremental R^2's and were estimated with the bootstrap method.

However, the PGI of drinks per week is noisy and has low predictive power: its incremental R^2's in the samples of European ancestry parents and biological children do not exceed 1.1% and it has the wrong sign in the sample of Korean adoptees. For that reason, we will not use that PGI

[13] This may occur because a given SNP's GWAS coefficient estimate captures both that SNP's effect as well as the effects of correlated (and often unobserved) SNPs and other genetic variants, and because the SNPs' correlation patterns vary across ancestries. This may also occur because SNP effects may vary across ancestries, possibly because their effects vary across environments and environments vary across ancestries.

[14] In addition, our PGIs were constructed using SNP coefficient estimates (the $\hat{\beta}_j^Y$'s) from GWASs that regressed an outcome Y on non-adopted individuals' SNPs and omitted their parents' SNPs. Because the parents' SNPs are correlated with the individuals' SNPs and may affect Y via the common family environment, there may be omitted-variable bias in the $\hat{\beta}_j^Y$'s, and thus bias in our PGIs. In the terminology of Section 4.4 below, that biased part of a PGI captures some of the indirect effects (including genetic nurture), while the unbiased part captures direct effects. Among the Korean adoptees, due to quasi-random placement, the part of their PGIs that captures indirect effects is not correlated with their rearing parents' genetics and amounts to just another source of noise in their PGIs, thus further increasing attenuation bias.

in this study; we will only use the remaining six PGIs, of EA, cognitive performance, income, ever smoker, BMI, and height.

2.2.3. Other variables

Family socioeconomic status (SES): to reduce multiple-hypothesis testing and capture the multidimensional nature of socioeconomic background, in some analyses we used a family SES composite score that consists of parent self-reports of their occupation and education at intake and of their gross household income at the first follow-up assessment (McGue et al. 2017). Occupations were coded using the Hollingshead scale of occupational status, which ranges from 1 to 7 (Hollingshead 1957); the scale was reverse coded so that higher values represent higher status. The SES measure was computed by standardizing and then averaging the three individual measures, and then standardizing the resulting average so that it has mean zero and unit variance.

Baseline family variables: To capture family environmental variation, the following set of "baseline family variables" was used in many of our analyses: mother's years of education, mother's cognitive performance, mother's drinks per week, mother's ever used nicotine, mother's BMI, mother's height, mother's age when child was born, father's years of education, father's age when child was born, family SES, log family income, parent disinhibition score, number of siblings in the rearing family, whether the family was a mixed biological or adoptive family, whether the family lived in a city or suburb, and whether the parents were still married at intake. Online Appendix B provides detailed descriptions of these variables.

Baseline control variables: all analyses included our baseline control variables. These include sex, birth year, age at which the outcome was measured, placement age (for the adoptees only), and, for the regressions with a PGI, the 10 top PCs of the ancestry-specific SNP data. For analyses of cognitive performance, the baseline controls also include a dichotomous variable indicating whether or not a participant was age 16 or more at intake (and therefore took the WAIS-R instead of the WISC-R IQ test). For analyses of drinks per week and of ever used nicotine, the baseline controls also include age at intake and at each of the first two follow-up assessments, since these outcomes were constructed based on measures taken at each of these three assessments.

2.3 Analysis sample

We only analyzed individuals of Korean and European (non-Hispanic White) ancestry, who comprise more than 90% of the MCTFR-SIBS sample. Since all the European ancestry adoptees and biological children and the vast majority of the Korean adoptees in the sample have European ancestry parents, we excluded from the sample the seven Korean adoptees who have a non-European ancestry parent. In our analyses that do not involve molecular genetic data, we focused on the remaining 421 Korean adoptees, 471 European ancestry biological children, and 141

European ancestry adoptees, and on their European ancestry parents. Of these, genotypic data were available for 361 Korean adoptees, 411 European ancestry biological children, and 122 European ancestry adoptees, and for the majority of their parents (see Table 1).

As is customary when analyzing molecular genetic data, we examined the data to detect "genetic outliers". Genetic outliers are individuals whose recorded ancestry differs from the ancestry that can be inferred from their genetic data. We identified them by plotting and visually inspecting the top 10 PCs of the genetic relatedness matrix of the full sample of MCTFR-SIBS individuals who have been genotyped. Online Appendix C shows the plots of the top 10 PCs and provides further details. We identified no genetic outliers among the Korean adoptees or among the European ancestry adoptees, but 4 among the European ancestry biological children, 10 among the (European ancestry) adoptive and biological fathers and another 10 among the (European ancestry) adoptive and biological mothers. We dropped these outliers from any analyses that involve PGIs but kept them in the analyses that do not involve PGIs.

Table 1 shows descriptive statistics for the Korean adoptees, European ancestry adoptees, and European ancestry biological children who have been genotyped and are not genetic outliers, as well as for their parents. The average placement age of the Korean adoptees is 5.2 months (SD = 2.7; all adoptees were adopted before age 24 months). 39% of Korean adoptees are male compared to 47% of European ancestry biological children. Several characteristics at intake are distributed similarly for Korean adoptees (KA) and European ancestry biological children (EABC), including age at intake (KA: mean=15.0, SD=1.9; EABC: mean=14.9, SD=1.9), birth year (KA: 1985.9, 2.8; EABC: 1986.7, 2.8), cognitive performance (KA: 108.4, 13.8; EABC: 108.3, 12.9), and GPA (KA: 3.4, 0.8; EABC: 3.5, 0.7). At the first follow-up visit Korean adoptees were shorter (165.2 cm, 7.9 cm) than European ancestry biological children (172.8 cm, 8.6 cm) and had a slightly lower BMI (23.1, 3.9) than biological children (23.6, 4.5). At the third follow-up wave, Korean adoptees and European ancestry biological children had similar EA (KA: 16.1, 2.1; EABC: 16.2, 1.9) and log income (KA: 10.9, 0.7; EABC: 10.9, 0.7).

< Table 1 goes about here >

2.4 Quasi-random placement of the Korean adoptees

2.4.1 Adoption process

The adoption process for Koreans adoptees in the MCTFR-SIBS study resembles the adoption process described in detail in Sacerdote (2007).[15] While Sacerdote analyzed Korean adoptees adopted by families from across the US through the Holt adoption agencies, MCTFR-SIBS adoptees were adopted by Minnesotan families through one of three private adoption agencies. Briefly, the process typically took ~12-18 months from initial application to the child's placement in the adoptive home. Steps to adoption included filing an application, participating in a home study assessment, attending adoption education classes, passing a criminal background check, being matched with an adoptee, reviewing a referral statement and accepting the match, flying the adoptee to the US, and legally adopting the child in family court. US and South Korean law required that adoptive parents had a family income above 125 percent of the poverty level, were between the ages of 25 and 45, had been married for at least three years, and had at most four children before adoption.

As with the data analyzed by Sacerdote, adoptive parents in our study were matched to children on a first-come, first-served basis, with factors that are plausibly uncorrelated with adoptee characteristics—such as the timing of an application—determining which adoptee got matched to which family. Nonetheless, parents in our data were able to request an adoptee of a given sex. Since we control for sex in all our analyses, this should not bias our results. In addition, parents in our data could specify that they were not comfortable with a number of severe medical conditions prior to being matched, and had the opportunity to decline an adoptee after they had been matched. In principle, this could lead to correlations between biological and adoptive parent characteristics and indicate that adoptee placement was not quasi-random. In practice, however, this is unlikely to be the case. First, few children with disabilities or special needs were included in our sample because the SIBS excluded children with a mental or physical disability that would preclude them from full participation in the initial intake assessment. Second, because the number of parents wishing to adopt far exceeded the number of children available for adoption, parents rarely refused a match. And third, the referral statements were based on what the private adoption agencies knew about the adoptees and their parents, which usually was very little.[16]

[15] One of the authors of this study, Matt McGue, has been closely in touch with the adoption agencies since the inception of MCTFR-SIBS and has intricate knowledge of the adoption process. This summary is based on his knowledge and a review of relevant documents.

[16] Tellingly, MCTFR researchers once attempted to extract data from these referral statements to analyze them in their research, but ultimately abandoned that endeavor because so little useful data could be extracted. Basic information on the health of the baby (such as their birth weight or whether there were problems with delivery) was sometimes but not consistently available.

2.4.2 Testing for quasi-random placement

To test whether the Korean adoptees were quasi-randomly allocated to the adoptive families, we followed Sacerdote (2007) and Fagereng et al. (2021) and regressed pre-determined adoptee variables on adoptive family variables. Following Sacerdote and Fagereng et al., we include among our set of dependent variables the adoptee's sex and placement age (in months), and we include among our regressors the father's and mother's years of education and the log of family income. In addition, we include among our dependent variables the adoptees' 6 PGIs, and among our regressors the remaining 13 baseline family variables. All regressions control for adoptee birth year and its square.[17]

Table 2 reports, for each regression, the estimated coefficients as well as the *F* statistic and corresponding *P* value from the *F* test for the joint significance of the family variables.[18] Only when "male" is the dependent variable is the null hypothesis of no association with the family variables rejected at the 5% level; this is not surprising, since parents had the opportunity to request an adoptee of a given sex. Since we control for sex in our analyses in the rest of the paper, this should not be a source of bias. Among the 112 estimated coefficients on the 16 baseline family variables from the 7 other regressions, only 6 (5.4%) are significant at the 5%, which is in line with what one would expect if the data captured only noise. These results are consistent with quasi-random assignment of the Korean adoptees to their adoptive families conditional on sex.

< Table 2 goes about here >

Since the PGIs are noisy variables, their lack of association with any of the regressors may not be surprising. As an additional test, we switched the right-hand-side variables and (most of) the left-hand-side variables and regressed the previous regressions' regressors on the adoptees' PGIs (while still controlling for birth year and its square), and then conducted *F* tests to evaluate the joint significance of the PGIs. The *F* stat was significant at the 5% level in only 1 (6.3%) of the 16 regressions and was not significant at the 10% level in any other regression, as one would expect if the data captured only noise.

We repeated these analyses in the small sample of European ancestry adoptees who have been genotyped. That sample is about one third the size of the sample of Korean adoptees, which limits statistical power, but the PGIs' incremental R^2's are higher in that sample, which increases power.

[17] To maximize regression sample size, missing observations were recoded as 0 and dummies indicating missing observations were included for five family variables with high numbers of missing observations (mother's cognitive performance, mother's BMI, mother's height, father's age when child was born, and log family income).

[18] For the regression with "male" as the dependent variable, a logistic regression was estimated and joint significance was tested with a Wald test.

As can be seen in Online Appendix Table G.1, among the regressions of sex, placement age, and the PGIs on the baseline family variables, the full *F* test is significant at the 5% level in 6 of the 8 regressions and at the 10% level in all but 1 of the 8 regressions. And among the regressions of each of the 16 baseline family variables on the adoptees' PGIs, the *F* test for the joint significance of the PGIs is significant at the 5% level for 2 of the regressions and at the 10% level for another 2 of the regressions.

In sum, we find no evidence of selective placement for the sample of Korean adoptees, except with respect to sex. At the same time, and as expected, we find evidence of selective placement for the sample of European ancestry adoptees.

3. NATURE AND NURTURE: EVIDENCE FROM PEDIGREE DATA

To begin our analysis of nature-nurture interplay, we follow Sacerdote (2007) and use the standard "ACE" model from behavioral genetics (Plomin et al. 2001) to decompose the variance of each of the 10 outcomes into shares explained by additive genetic factors (A)[19], the common family environment (C), and unexplained factors (E). The genetic factors and common family environment can be thought of as capturing nature and the part of nurture that is shared among siblings reared in the same family, respectively; the unexplained factors may include individual environments that are not shared with other siblings (some of which can also be thought as nurture) as well as measurement error. The ACE model assumes that A, C, and E contribute linearly and additively to the outcome of interest Y, normalized to have mean zero and unit variance:

$$Y^{std} = A + C + E,$$

where $Y^{std} = (Y - E[Y])/\sigma_Y$. Furthermore, the model assumes that A, C, and E are independent, that there is no assortative mating, and that A includes both the direct and indirect effects of genetics.[20] The latter include what behavioral geneticists call active gene-environment correlation (rGE)—whereby genetics impact the outcome indirectly by influencing the individual's choice of a causal environment—as well as evocative rGE—whereby one's genetics evokes an environmental response that causally impacts the outcome.

Taking the variance of both sides of the equation yields:

$$\sigma^2_{Y^{std}} = 1 = \sigma^2_A + \sigma^2_C + \sigma^2_E.$$

[19] Additive genetic factors refer to genetic effects that are linear in the number of variants one has at a location in the genome and that do not involve interactions between genetic variants at different locations in the genome. For most traits, much of the genetic variation is accounted for by additive genetic factors (Hill et al. 2008).

[20] More sophisticated models have been developed to account for sibling-specific effects, gene-environment correlations, assortative mating, genetic dominance, and epistasis (e.g., Keller et al. 2009). However, these models are not identified in our sample of adoptive and biological siblings.

In other words, the variation in a standardized outcome is equal to the sum of the variation from genetic factors (σ_A^2), the common family environment (σ_C^2), and the nonshared environment (σ_E^2). The outcome's heritability is defined as the share of the outcome's variance that is due to genetic factors: $h^2 = \sigma_A^2 / \sigma_{Y^{std}}^2 = \sigma_A^2$.

We assume that adoptive siblings share no genetics but share the same family environment, and that biological siblings share half of their genetics[21] and also share the same family environment. It follows that the correlations between two biological siblings and between two adoptive siblings are equal to:

$$Corr_{\text{bio}}(Y_1, Y_2) = \frac{1}{2}\sigma_A^2 + \sigma_C^2;$$
$$Corr_{\text{adopt}}(Y_1, Y_2) = \sigma_C^2,$$

where we use subscripts 1 and 2 to index the (arbitrarily chosen) first and second individual in each pair. From here, $h^2 = \sigma_A^2 = 2(Corr_{\text{bio}}(Y_1, Y_2) - Corr_{\text{adopt}}(Y_1, Y_2))$, $\sigma_C^2 = Corr_{\text{adopt}}(Y_1, Y_2)$, and $\sigma_E^2 = 1 - \sigma_A^2 - \sigma_C^2$.

Importantly, because adoptive siblings only share a common family environment after adoption has taken place, C does not capture pre-adoption environmental influences, such as the in-utero environment nor the family or orphanage environment before adoption. The part of these influences that is not selected or evoked by one's genetics is captured by E (recall that active and evocative rGE are captured by A). By contrast, in (non-adopted) twin studies that estimate the ACE model by comparing the resemblance of monozygotic twins to that of dizygotic twins, C does capture these influences.

To obtain precise estimates of σ_A^2, σ_C^2, and σ_E^2, we employ the generalized method of moments (GMM). We obtain such estimates for each residualized outcome $\tilde{Y}$, where $\tilde{Y}$ is the outcome Y purged of the effects of a vector X that includes the baseline control variables, a dummy indicating adoptee vs. biological child status, and an intercept.[22] We treat as an observation each sibling pair in the sample used above to compute sibling correlations, and let $1\{BS\}$ and $1\{AS\}$ denote dummy variables indicating if the pair contains two biological siblings or two adoptive siblings. Further, we let β denote the coefficient on the purged covariates X (so, $\tilde{Y} = Y - X\beta$), and estimate β along with all the other parameters via GMM. We obtain the following moment conditions:

$$E\left[1\{BS\}\left(\tilde{Y}_1\tilde{Y}_2/\sigma_{\tilde{Y}}^2 - 1/2\,\sigma_A^2 - \sigma_C^2\right)\right] = 0;$$
$$E\left[1\{AS\}\left(\tilde{Y}_1\tilde{Y}_2/\sigma_{\tilde{Y}}^2 - \sigma_C^2\right)\right] = 0;$$

[21] In the presence of assortative mating, the correlation between the additive genetic factors among two biological siblings may not be 0.5. If assortative mating is positive, that correlation will be larger than 0.5 and our ACE heritability estimates in our sample of adoptive and biological siblings will be biased upwards.

[22] Adoption age was included among the baseline control variables and was coded to 0 for the biological children.

$$E[1 - \sigma_A^2 - \sigma_C^2 - \sigma_E^2] = 0;$$
$$E\left[\left(\tilde{Y}_1\right)^2 + \left(\tilde{Y}_2\right)^2 - 2\sigma_{\tilde{Y}}^2\right] = 0;$$
$$E[\tilde{Y}_1 X_1 + \tilde{Y}_2 X_2] = 0.$$

3.1 Results

Panel A of Table 3 shows adoptive and biological sibling correlations for each residualized outcome $\tilde{Y}$. Adoptive sibling correlations were computed among families with at least one Korean adoptee (families with only non-Korean adoptees were not used because non-Korean adoptees may have been non-randomly assigned to their families). Biological sibling correlations were computed for the European ancestry biological children only.

Reflecting a higher degree of genetic similarity, the biological sibling correlations are higher than the adoptee correlations and are statistically significant (at the 5% level) across the board, except for log income (for which the sample is small). For example, the correlation between biological siblings for cognitive performance at intake is 0.317, which is higher than the correlation of 0.080 for adopted siblings. Biological siblings also have substantially higher correlations for GPA, soft skills, BMI, and height. On the other hand, for educational attainment, college, drinks per week, and ever used nicotine, correlations are more similar for biological and adopted siblings. For log income, the adoptive sibling correlation is larger, though not significantly so.

< Table 3 goes about here >

Panel B of Table 3 shows the resulting GMM estimates. For educational attainment, college completion, log income, and ever used nicotine, between 22% and 28% of the variance can be explained by the common family environment; for drinks per week, the corresponding figure is 12.3%. For all these outcomes, the share explained by genetics is not significant (though the standard errors are large). By contrast, for cognitive performance, BMI, and height, 33.0%, 84.0%, and 62.2% of the variance is explained by genetics, respectively, while the share explained by common family environment is much smaller. Genetics explains ~30% and family environment ~15% of the variance in both GPA and soft skills.

Though our estimates are imprecise (as expected due to the small size of our sample), overall they are broadly consistent with those in the literature (Polderman et al. 2015). For phenotypes that are identical or similar to those that were examined by Sacerdote (college, drinking, smoking, BMI, and height), most of Sacerdote's point estimates are within our (wide) confidence intervals. In addition to sampling variation, a possible reason for differences between our estimates and Sacerdote's is differences in respondents' age. Sacerdote's adoptive and biological sibling samples

were aged 19-40 at the time of data collection, while the majority of respondents in our data were still teenagers at intake and at the first follow-up wave, when GPA, soft skills, cognitive performance, drinks per week, ever used nicotine, BMI, and height were measured.

More generally, when interpreting our results, it is important to keep in mind that these outcomes may not have been fully realized at measurement for many respondents. In particular, the heritabilities of cognitive performance, drinking, and smoking have been shown to be smaller in early childhood and to increase with age (Bouchard 2013; Kendler et al. 2008). Conversely, common environmental influences on these traits, as well as on savings behavior (Cronqvist & Siegel 2015), have been found to become much smaller in early or middle adulthood. This may explain why our heritability estimate for cognitive performance is smaller than other estimates in the literature that suggest more than half of the variation in cognitive performance can be explained by genetic factors (e.g., Bouchard & McGue 1981; Plomin et al. 2001; Polderman et al. 2015). This may also explain why we find significant (but still small) common family environmental influences on height. We test for such age interactions just below, but find no support for them in our data (which are not ideal for this test).

3.2 Extensions of the ACE models

Following Fagereng et al. (2021), we use the fact that we have three types of sibling pairs—adoptive-adoptive, adoptive-biological, and biological-biological—to extend the ACE model by relaxing the assumption that genetics and shared environment are always uncorrelated. Taking the variance of y for the biological children now yields

$$\sigma^2_{Y^{std}_{BC}} = \sigma^2_A + \sigma^2_C + 2\gamma + \sigma^2_E,$$

where $\gamma = Cov(A, C)$ among the biological children. Due to quasi-random assignment, $Cov(A, C) = 0$ for the Korean adoptees. For the adoptive-adoptive pairs, $Cov(A_1, C_2) = Cov(A_2, C_1) = 0$; for the adoptive-biological pairs, $Cov(A_1, C_2) = 0$ and $Cov(A_2, C_1) = \gamma$; and for the biological-biological pairs, $Cov(A_1, C_2) = Cov(A_2, C_1) = \gamma$. From this, four moments conditions can be derived and the four parameters of the extended ACE model can be estimated. Online Appendix D provides additional details and list the GMM moment conditions.

Online Appendix Table G.2 shows the GMM estimates. Estimates of γ, the covariance between genetics and the common environment, are mostly small in magnitude and none is significantly different from zero. Fagereng et al. (2021) also find little evidence of correlation between A and C. This may be because two countervailing forces cancel each other out: A correlates positively with parental genetics and thus with a good home environment, but negatively with parental investments—if the latter are motivated by an attempt to compensate for lower A. Across the outcomes, estimated variance shares due to genetics and the common family

environment resemble those from the baseline ACE model, though the estimated variance share due genetics is noticeably larger for EA ($h^2 = \sigma_A^2 = 0.381$) and cognitive performance (h^2=0.527), and lower for BMI ($h^2 = 0.411$).

To test for age interactions in the ACE model, we estimated another extended version of the ACE model—one that allows the age at which an outcome was measured to moderate the effects of the additive genetic, common environmental, and unexplained factors:

$$\tilde{Y} = (a_0 + a_1 \cdot age)A + (c_0 + c_1 \cdot age)C + (e_0 + e_1 \cdot age)E.$$

Online Appendix Figure G.1 plots, for each outcome, the estimated share of the outcome variation that is attributable to each factor as a function of age at measurement. Overall, we find little evidence that age at measurement moderates the relative importance of the factors, though our estimates are imprecise. One possible reason for this lack of evidence is that age may truly have no moderating effects on the ACE estimates; other reasons include the small size of our sample as well as the limited age range we observe. Interestingly—and consistent with the above discussion—for height, we find suggestive (but statistically insignificant) evidence that heritability increases and common family environmental influences decrease with age. Online Appendix D provides further detail on this extended model, how we estimated it via GMM, and how we plot and report the results.

4. NATURE AND NURTURE: EVIDENCE FROM MOLECULAR GENETIC DATA

The findings from the previous section, along with the extensive behavioral genetics literature (e.g., Fagereng et al. 2021; Sacerdote 2007; Silventoinen et al. 2020), suggest that both genetics and the common family environment matter for many social scientific outcomes. However, the precise genetic and family environmental variables that matter remain elusive. In this section, we leverage the quasi-random assortment of the Korean adoptees into families to analyze the contributions of specific family environmental variables and of the PGIs to each of the 10 outcomes.

4.1 Interpreting estimates from regressions on family variables and PGIs in our sample of Korean adoptees

To help organize thoughts, let us once again consider the ACE model from the previous section, but let us relax the assumption that A, C, and E are uncorrelated. As is well known, in a sample of biological children (or non-randomly assigned adoptees), regressing a child outcome Y on family environmental variables (e.g., parental EA) may yield biased estimates, since parents

share genetics with their children and the family environmental variables may thus be correlated with the ommitted A. Similarly, regressing Y on children's genetic variables (e.g., their PGIs) may yield biased estimates, since the omitted C may be correlated with the parents' genetics, which in turn are correlated with the children's genetics. Our sample of quasi-randomly placed Korean adoptees allows us to mostly circumvent these issues, since there is plausibly no correlation between A and C in our sample.

4.1.1 Interpreting estimates from regressions on family variables

Because of the quasi-random assortment of Korean adoptees into families, estimates from regressions of Y on family variables can support a causal interpretation, but with one caveat. The caveat is that the family variables' estimated effects could be due to correlated variables that were omitted from the regressions rather than to the family variables themselves.

4.1.2 Interpreting estimates from regressions on PGIs

Three caveats must be kept in mind when interpreting estimates from regressions of Y on PGIs. The first caveat is that, due to their correlation with biological parents' genetics, the Korean adoptees' PGIs may still be correlated with pre-adoption environmental influences (including the in-utero environment) that were not selected nor evoked by the adoptees' genetics. As a result, the adoptee PGIs' estimated effects may capture part of E. However, since only pre-adoption factors can generate a correlation between the PGI and E and all adoptees were adopted at a very young age, this is unlikely to introduce more than a negligible amount of bias. We cannot directly observe the correlation between the PGI and E in our data, so we formalize this as an assumption:

Assumption 1: the covariance between the PGI and E, $\sigma_{PGI,E}$, is null or small in magnitude.[23]

The second caveat is that, even under Assumption 1, one cannot interpret an estimated outcome-PGI association as an estimate of *the effect of A*. The reason is that, as already mentioned, PGIs are only imprecise measures A, and this is especially the case for the Korean adoptees. The third caveat is that, even under Assumption 1, the estimated outcome-PGI association is not an unbiased estimate of *the causal effect of the PGI*. We define the causal effect of the PGI on Y as $\beta \equiv \Delta_Y/\Delta_{PGI}$, where Δ_Y denotes the expected change in Y that would ensue if a fictitious experimenter were to permute chromosomes across individuals at conception in a way that increased one's PGI by Δ_{PGI}.[24] An estimated outcome-PGI association is not an unbiased estimate

[23] The proof of Proposition 1 in Online Appendix E clarifies what is meant by "small in magnitude".

[24] For this thought experiment to work, one cannot permute single SNPs, as these tend to be correlated with nearby variants and these correlations are baked into the PGIs; one must instead permute large independent blocks of correlated DNA, such as the chromosomes. Also, note that Δ_Y denotes the *expected* change in Y, as opposed to the

of β because population stratification and assortative mating generate correlations between the PGI and other genetic factors that are omitted from our regressions. Online Appendix E provides more details.

Because of these last two caveats, it is difficult to precisely interpret the *magnitude* of the estimated association between an outcome Y and a PGI. Nonetheless, estimates from our regressions of Y on the PGIs in our sample of Korean adoptees are interesting because they can be used to obtain a lower bound for the share of the variation in Y that is attributable to A (σ_A^2). Proposition 1 formalizes this statement.

Proposition 1. Under Assumption 1, in a regression of Y on some PGIs, the regression's true (population) R^2 is a lower bound for σ_A^2: $R^{o\,2}_{PGI} \leq \sigma_A^2$.

Online Appendix E provides the proof. A corollary of Proposition 1 is that a nonnull outcome-PGI association implies the existence of causal genetic effects.

4.2 Multiple regression results

Table 4 presents results from regressions that showcase the contributions of family environmental variables and the PGIs to the 10 outcomes in the sample of Korean adoptees. Specifically, we regressed each outcome on the set of baseline family variables, the adoptees' six PGIs, and the baseline control variables.[25] For each outcome, we then computed the incremental adjusted R^2's of the block of family variables and of the block of PGIs.[26] We define the incremental adjusted R^2 of each of these two blocks of variables as the difference between the adjusted R^2 of the regression on the baseline controls and the block and the adjusted R^2 of the same regression in the same sample but without the block. We also conducted tests of the joint significance of the variables in each block.[27,28]

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actual change. The expectation is taken over all possible chromosome permutations across the individuals in the population. Δ_Y captures the expected rather than actual change in Y because the PGI is only a noisy proxy for the true additive genetic factor, and its signal-to-noise ratio may vary across chromosomes; as a result, some permutations would increase Y by more than $\beta\Delta_{PGI}$, while other permutations would increase Y by less than $\beta\Delta_{PGI}$.

[25] We jointly include the six PGIs in the regression of each outcome because each outcome-relevant PGI is noisy and the other PGIs often have incremental explanatory power. Of course, if an outcome-relevant PGI were a perfect measure of the outcome's additive genetic factor A, other PGIs would have no incremental explanatory power.

[26] We use the adjusted R^2 because it yields a less biased estimate of the true (population) R^2, and it is an unbiased estimator of the true R^2 when the latter is 0. Because of the small size of our sample, this makes a noticeable difference.

[27] For the continuous variables, OLS regressions were estimated and a F test was used to test joint significance. For the binary outcomes (college and ever used nicotine), logistic regressions were estimated, McFadden's adjusted pseudo R^2 was used, and a Wald test was used to test joint significance.

[28] As in the adoptee random placement analysis above, to maximize regression sample size, missing observations were recoded to 0 and dummies indicating missing observations were included for five family variables with high numbers of missing observations (see the table note for details). This does not bias the estimated coefficients but does reduce the effective variation in the five family variables, and so biases our incremental R^2 estimates downwards.

< Table 4 goes about here>

A few interesting patterns emerge. First, both genetic and family environmental variables matter. The baseline family variables are jointly siginificant (at the 5% level) for EA, cognitive performance, log income, and BMI. They account for 8% of the variation in EA; for ~6-7% of the variation in log income and BMI; and for 1.5% and ~3% of the variation in cognitive performance and soft skills, respectively. Perhaps reassuringly, they also account for less 1% of the variation in height. As for the PGIs, they are jointly siginificant and account for 5-7% of the variation in EA, GPA, cognitive performance, and height.[29]. The PGIs are also jointly singificant in the regressions of college completion and soft skills. While the PGIs are not jointly significant in the regressions of log income, drink per week, ever used nicotine, and BMI, these outcomes are significantly associated with single PGIs, as we discuss below. By Proposition 1, the above PGI variance shares are estimates of lower bounds for σ_A^2 for the corresponding outcomes, and the significant outcome-PGI associations do imply a rejection of the null of no genetic effects on the outcomes.

To examine the role of specific variables, we regressed each outcome on each baseline family variable and each PGI separately while controlling for the baseline controls, in the sample of Korean adoptees. This analysis is similar to Sacerdote's (2007) analysis of which aspects of the family environment are the most important for adoptees' outcomes, though Sacerdote did not have molecular genetic data and so could not include PGIs in his regressions.

The top panel of Online Appendix Table G.4 shows the results for the baseline family variables. As mentioned, because of the quasi-random assignement of the adoptees to families, these estimates can support a causal interpretation of the effects of each rearing family variable, though the estimated effects could be due to ommitted correlated variables. Of note, our estimates reveal that higher parental education, family SES, and family income are associated with higher adoptee education, income, and drinking. For instance, one extra year of either maternal or paternal education is associated with a 0.23-year increase in adoptee education. A one-standard-deviation increase in family SES is associated with 0.66 extra year of education, a 10-percentage-point increase in the probability of attending college, a 10% increase in income, and 1.1 additonal drinks per week. And the adoptee-family elasiticity of income is 0.286, implying that a one-percent increase in adoptive family income is associated with a 0.286% increase in adoptee income.

Adoptive parents' substance use habits (or their correlates) also impact adoptees, with adoptees whose mother ever used nicotine consuming an extra 1.7 alcoholic drinks per week on

[29] Online Appendix Table G.3 report analogous regressions in the sample of biological children. The family environment and the PGIs are both highly correlated with multiple outcomes. However, as mentioned above, since biological children share both genetics and environment with their parents, these associations are likely confounded.

average, and with a one-standard-deviation increase in parent disinhibition associated with a 2.6-point decrease in cognitive performance. We also see a negative effect of family size on adoptee educational, cognitive, and labor market performance: having an additional (adoptive) sibling is associated with a 5.6-percentage-point decrease in the probability of attending college, with 1.3-point decrease in cognitive performance, and with a 5.6% decrease in income. This could be due to lower per-child parental investment in larger families (Downey 1995) and suggests the existence of a tradeoff between the quantity and quality of children (Becker 1960).

As shown in the bottom panel of Online Appendix Table G.4, the PGIs are strongly associated with related outcomes. Clearly demonstrating nonzero effects of genetics on the outcomes, 9 of the 10 outcomes are significantly associated at the 5% level with at least one of the PGIs; the remaining outcome, income, is associated at the 10% level (with the PGI of income). We find that a one-standard-deviation increase in the PGI of EA is associated with 0.5 additional years of EA, a 7-percentage-point higher probability of having completed college, 3 additional IQ points, 0.9 fewer drinks per week, as well as with a higher GPA and increased soft skills. One-standard-deviation increases in the PGIs predicting whether one ever was a smoker, BMI, and height are associated with a 5-percentage-point higher probability of ever having smoked, a 0.6-point increase in BMI, and a 2-centimeter increase in height, respectively. Again, a causal interpretation can be warranted, though the above-mentioned caveats must be kept in mind.

We caution against too close a comparison of the overall influence of the family variables and PGIs. Not only are the PGIs noisy proxies for the true genetic effects (especially among the Korean adoptees), but the family variables could also have been measured with error and it is possible that family variables other than the ones we observe would have stronger effects. Nonetheless, our results indicate that among variables typically available to current-day researchers, the explanatory power of genetic and family environmental variables is of a similar order of magnitude, with both sets of variables explaining positive (i.e., nonzero) shares of the variation in most outcomes we study. Our results also constitute a clear demonstration that both nature and nurture matter.

4.3 Nature and nurture treatment effects

Next, inspired by Sacerdote (2007), we estimated the treatment effect of being assigned to a particular family type. We considered three different family types based on parental education, the number of children in the family, and family SES.[30] Type 1 families are defined as families with three or fewer children and where both parents have a four-year college degree (44% of Korean adoptees and 22% of European ancestry nonoadoptees). Type 3 families are defined as families (i)

[30] Sacerdote (2007) did not use family SES to define the three family types; given our small sample size, we also used family SES to define Type 3 families, to ensure the number of families is not too unbalanced across the three family types.

with four or more children and where neither parent has a four-year college degree or (ii) in the bottom quintile of the SES distribution (14% of Korean adoptees and 33% of biological children); Type 2 families are the families that are neither Type 1 nor Type 3 (43% of Korean adoptees and 45% of biological children).

To complement this analysis of the effects of nurture based on the three family types, we analyzed the effects of nature using dummy variables indicating one's tercile in the distribution of each outcome-relevant PGI (with the third tercile corresponding to the highest PGIs).

For the continuous outcomes, treatment effects were estimated with OLS regressions using the following specifications:

$$Y = \beta_0 + \beta_1 FT1 + \beta_2 FT2 + \beta_3 Controls + \varepsilon$$
$$Y_i = \gamma_0 + \gamma_2 GT2 + \gamma_3 GT3 + \gamma_3 Controls + \varepsilon,$$

where Y is the outcome of interest, $FT1$ and $FT2$ are dummies indicating Type 1 and Type 2 families, and G$T2$ and $GT3$ are dummies indicating the second and the third PGI terciles. The ommitted categories are the Type 3 families and the first PGI tercile. Thus, β_1 is the treatment effect of being assigned to a Type 1 family relative to being assigned to a Type 3 family, and γ_3 is the effect of having a PGI in the highest tercile relative to having a PGI in the lowest PGI tercile. For the binary outcomes, analogous logistic regressions were estimated. In the sample of Korean adoptees, the family-type treatment effects can be interpreted as causal since assignment to family was quasi-random. And the significant PGI-tercile effects imply causal genetic effects, with the caveats discussed above.

< Table 5 goes about here >

Table 5 shows the results for the sample of Korean adoptees.[31] Overall, we observe effects of both nurture and nature. Panel A shows the estimated effects of being assigned in the different family types. We see benefits of being in a Type 1 family on EA, having a college degree, and cognitive performance. Adoptees who were quasi-randomly placed in Type 1 families have on average 1.3 additional years of education, have a 23-percentage-point higher probability of having a college degree, and scored 3.5 IQ points higher on the cognitive performance assessment, compared to adoptees who were placed in Type 1 families.

Panel B of Table 5 shows the estimated effects of the PGI tercile dummies. As expected, and consistent with the results reported in Figure 1 and Online Appendix Table G.4, being in the third tercile of the outcome-relevant PGI is significantly associated with all outcomes, except ever

[31] Online Appendix Table G.5 shows the corresponding results for the sample of European ancestry biological children. These estimates cannot be interpreted as causal due to the correlation between genetic and family environmental factors.

smoker (and drinks per week, whose outcome-relevant PGI we dropped from the analysis). For instance, being in the third tercile of the EA PGI is associated with 1.1 extra year of education and a 16-percentage-point higher probability of having a college degree, relative to being in the first tercile; and being in the third tercile of the cognitive performance, income, and height PGIs is associated with 6.3 extra IQ points, a 28% higher income, and 3.8 extra centimeters of height, respectively.

4.4 Indirect genetic effects (genetic nurture)

Above, we peeked into the black box of the latent common family environment factor represented by C, and found that variables such as rearing family education, SES, income, substance use habits, and size (or their correlates) account for part of C's effects. These family variables, in turn, have been shown or are likely to be under genetic influences (Plomin & Bergeman 1991). This leads to the question of how much of C can be traced to the effects of one's rearing parents' genetics.

To begin addressing this question, let us again consider the ACE model, allowing A and C to be correlated, but with C decomposed into components that capture the common family environment correlated with the rearing parents' genetics and the residual common family environment:

$$Y = A + C + E = A + \left(P_{A_m,A_f}C + M_{A_m,A_f}C\right) + E,$$

where P_{A_m,A_f} is the projection matrix of A_m and A_f and $M_{A_m,A_f} = I - P_{A_m,A_f}$ (so P_{A_m,A_f} and M_{A_m,A_f} projects onto and off the space spanned the rearing mother's and father's additive genetic factors). Direct genetic effects stem from an individual's own genome and are represented by A. Indirect genetic effects, or *geneitc nurture*, are the effects of the rearing parents' genomes that act via the family environment (Kong et al. 2018). They are "indirect" because they do not stem from an individual's own genome, and are captured by $P_{A_m,A_f}C$. $P_{A_m,A_f}C$ may also capture environmental and cultural factors that are part of C and that correlate with parents' genetics while not being endogenous to it (i.e., population stratification). Finally, the *population* effect is the estimated association between the outcome and an individual's genome, represented by $A + \rho$, where ρ accounts for the correlation between A and $P_{A_m,A_f}C$.

Following Okbay et al. (2022), we estimate the direct and indirect genetic effects *of a PGI* by regressing an individual's outcome on the individual's PGI and their rearing parents' PGIs:

$$Y = \mu + \delta PGI + \alpha_m PGI_m + \alpha_f PGI_f + u_i,$$

where δ captures the PGI's direct effect while α_m and α_f capture the parental PGIs' indirect effects (genetic nurture) as well as effects due to population stratification. Importantly, because of the

quasi-random assignment of the adoptees, one important potential confound in other genetic nurture studies' estimates of α_m and α_f—assortative mating—is not present in our study. As discussed in Young et al. (2019) and Okbay et al. (2022), since a PGI is a noisy proxy for A, estimates of α_m and α_f from typical samples may also capture bias due to assortative mating, since assortative mating generates correlations between parental PGIs and the component of a child's A that is not captured by their noisy PGI. That bias is not present in our study, since parental PGIs are uncorrelated with the child's A due to quasi-random assignment.

Online Appendix Table G.6 reports the results for each outcome with the outcome-relevant PGI (except for drinks per week, whose PGI we dropped) in the sample of Korean adoptees. Since our primary interest is to assess whether part of C can be traced to rearing parents' genetics, the table also reports the incremental adjusted R^2 of the parents' PGIs, defined as the difference between the adjusted R^2 of the regression that includes the parents' PGIs and the adjusted R^2 of the same regression in the same sample but without these PGIs. The table also reports the results of F tests of the joint significance of the parental PGIs.[32] We find that for EA, ever used nicotine, and possibly BMI, part of C can indeed be traced to rearing parents' genetics. Rearing parents' PGIs of EA and BMI account for 6.8% and 1.8% of the variation in the adoptees' EA and BMI, respectively. A one-standard-deviation increase in the rearing mother's PGI of EA is associated with a 0.55-year increase in adoptee EA, and one-standard-deviation increases in the rearing father's and mother's PGIs of ever smoking are associated with 6.8- and 4.7-percentage-point increases in adoptee nicotine usage, respectively.

Again, because of the quasi-random assignment of the adoptees to their adoptive parents, these estimates of α_m and α_f can support a causal interpretation and are consistent with the existence of genetic nurture, with the caveat that non-endogenous correlates of the parents' PGIs—but not assorative mating (as in other studies of genetic nurture)—could in principle account for the parental PGIs' estimated effects (Nivard et al. 2022). These results are consisent with a growing body of work that has documented associations between parental PGIs and child outcomes after controlling for child PGI (e.g., Cheesman et al. 2020a; Demange et al. 2020; Kong et al. 2018).

[32] As before, for the two binary outcomes, logistic regressions were estimated, Nagelkerke's pseudo R^2 was used, and a Wald test statistic was conducted (instead of a F test).

5. NATURE AND NURTURE INTERACTIONS: EVIDENCE FROM MOLECULAR GENETIC DATA

We now turn to the interactive dimension of nature-nurture interplay and examine whether genetics and the common family environment interact in the human capital production function for each of the 10 outcomes. Suppose the production function for outcome Y is given by

$$Y = A + C + (A \times C) + E,$$

where we again allow A and C to be correlated and where $(A \times C)$ represents an interaction between genetics and the family environment. We will begin our empirical investigation using the outcome-relevant PGIs and family SES (as a rough measure of the common family environment) using the following model:

$$Y = \beta_0 + \beta_1 SES + \beta_2 PGI + \beta_3 (SES \times PGI) + \epsilon. \quad (5.1)$$

We say there is a gene-environment interaction ("GxE") if $\beta_3 \neq 0$.[33] The PGI and family SES are technical complements in the human capital production function if $\beta_3 > 0$ and technical substitutes if $\beta_3 < 0$.[34] This simple specification is commonly used in the GxE literature. Though more complex models have incorporated individual or parental investment functions that respond to one's genetics and environment (see, e.g., Biroli et al. 2022 and Houmark et al. 2021), we adopt this specification because our primary objective is to test whether GxE interactions are present at all.

While there has been a plethora of GxE studies over the past decade, only a few robust, replicable, and plausible gene-environment interactions have been documented to date, as most GxE studies have suffered from a number of methodological limitations (Dick et al. 2015; Domingue et al. 2020; Hewitt 2012). For instance, most GxE studies to date have explored interactions between genetics and environmental variables that are not exogenous (e.g., Caspi et al. 2002, 2003). This casts doubt on whether any identified interactions truly are gene-environment interactions and not simply proxying for environment-environment or gene-gene interactions

[33] To develop some intuition for this, rewrite equation (5.1) by grouping the terms that involve F, and observe that when $\beta_3 \neq 0$ the effect of SES on Y is modulated by the PGI: $Y = \beta_0 + (\beta_1 + \beta_3 PGI)SES + \beta_2 PGI + \epsilon$. If we instead group the terms that involve the PGI, we see that SES modulates the effect of the PGI: $Y = \beta_0 + \beta_1 SES + (\beta_2 + \beta_3 SES)PGI + \epsilon$. Statistically, these two interpretations are indistinguishable.

[34] Specifically, the PGI and family environment are technical complements if the marginal effect of the PGI on Y increases as family environment improves (and vice versa): $\frac{\partial^2 Y}{\partial PGI\, \partial SES} = \frac{\partial^2 Y}{\partial SES\, \partial PGI} = \beta_3 > 0$; they are technical substitutes if the marginal effect of the PGI decreases as family environment improves (and vice versa): $\frac{\partial^2 Y}{\partial PGI\, \partial SES} = \frac{\partial^2 Y}{\partial SES\, \partial PGI} = \beta_3 < 0$.

(Schmitz & Conley 2017).[35] A second common limitation is that many studies fail to adequately control for confounders that may bias interaction effect estimates; as Keller (2014) demonstrates, it is important to interact control variables with both the interacted environmental variable and the interacted genetic variable. A third limitation is that for variables that have no natural scale, any estimated GxE interaction could be an artifact of the way a variable was scaled.[36] Finally, many studies have insufficient statistical power due to the small size of the samples they analyzed and the likely small effects of the interactions of interest.

Here, we address the first three of these limitations by leveraging the Korean adoptees' quasi-random placement; by properly interacting control variables as recommended by Keller (2014); and by verifying the robustness of our significant baseline results to changes in the scale of the dependent and family environmental variables whose scales are arbitrary. We discuss the issue of statistical power in Section 5.2.

We estimate β_3 using equation (5.1) but with the control variables. In our first model (Model I), we include the control variables but do not include their interactions. In our second model (Model II), we include the control variables as well as their interactions with the PGI and family SES, as recommended by Keller (2014):

$$Y = \beta_0 + \beta_1 SES + \beta_2 PGI + \beta_3 (SES \times PGI) + \beta_4 Controls + \beta_5 Controls \times SES + \beta_6 Controls \times PGI + \epsilon,$$

where $Controls$ denotes a vector of control variables. In our baseline specification, we include the baseline control variables. All regressions, including those of the binary outcomes[37], were estimated by OLS, with standard errors clustered at the family level.

5.1 Results

Table 6 reports our baseline results for the 10 outcomes in the sample of Korean adoptees. For Model I, we report the estimates of the coefficients on the PGI, family SES, and their interaction. For Model II, we only report the estimates for the PGI-family SES interaction, because the estimates for the PGI and family SES are difficult to interpret without taking into account the estimates on the interacted controls and the values of the control variables. In all specifications,

[35] For example, Caspi et al. (2003) reported a significant interaction effect between the 5-HTT gene and experiencing stressful life events on depression, but since experiencing stressful life events is heritable, it is possible that the result instead reflects a gene-gene interaction.

[36] For example, height can be measured in centimeters and income in dollars, but a measure of personality based on the sum of ordinal response variables typically does not have a natural scale. This complicates the interpretation of any estimated GxE interaction. To illustrate, there may be no GxE interaction in the model with the square root of the personality measure ($\sqrt{Y} = \gamma_1 SES + \gamma_2 PGI + u$), but this could still imply an interaction in the model with the actual measure ($Y = (\gamma_1 SES + \gamma_2 PGI + u)^2 = {\gamma_1}^2 SES^2 + {\gamma_2}^2 PGI^2 + \gamma_1 \gamma_2 (SES \times PGI) + residuals$).

[37] We estimated linear probability models via OLS for the binary outcomes because, as Ai & Norton (2003) show, it is difficult to interpret coefficients on interaction terms in probit and logit models.

the family environment variable is family SES. For each outcome, we use the outcome-relevant PGI. For the cognitive performance outcome, we also conduct the analysis with the PGI of EA, because EA is highly genetically correlated with cognitive performance (Okbay et al. 2016)[38] and because the effective sample size of the GWAS of EA whose summary statistics we used to construct the PGI of EA is more than twice as large as that of the corresponding GWAS of cognitive performance.

< Table 6 goes about here >

As expected, the estimates for Model I imply that the PGIs are all significantly and positively associated with their corresponding outcomes. Family SES is also positively associated with most outcomes (though not significantly so, except for EA, College, and cognitive performance). In both Models I and II, we estimate negative interaction effects on cognitive performance between family SES and the PGIs of both cognitive performance and EA, but find no significant interactions for the other outcomes. The estimates of the interaction effect on cognitive performance with the PGI of cognitive performance are marginally significant ($P = 0.06$ in both models) and those with the PGI of EA are more highly significant ($P = 0.0014$ in both models). These negative interactions imply that family SES and the PGIs for cognitive performance and EA are technical substitutes in the production function of cognitive performance, such that the effects of the PGIs are larger among lower-SES families and the effect of family SES is larger among adoptees with lower PGIs.

Figure 2 helps visualize these results. We defined four quadrants based on whether an adoptee's EA PGI and family SES are above or below the respective medians in the sample of genotyped Korean adoptees. For each quadrant, the figure shows mean cognitive performance, after first residualizing cognitive performance on the baseline controls and then adding the predicted value of cognitive performance based on the controls evaluated at their means. As can be seen, family SES and the EA PGI affect cognitive performance, but only when the EA PGI or family SES are low; among adoptees in high SES families, the EA PGI has little effect, and among high-EA-PGI adoptees, family SES has little effect.

[38] The genetic correlation between two traits is the correlation between the additive genetic components of the two traits. Under some assumptions, it can be shown that the genetic correlation is also equal to the correlation across SNPs between the SNPs' true effect sizes on the two traits (Bulik-Sullivan et al. 2015).

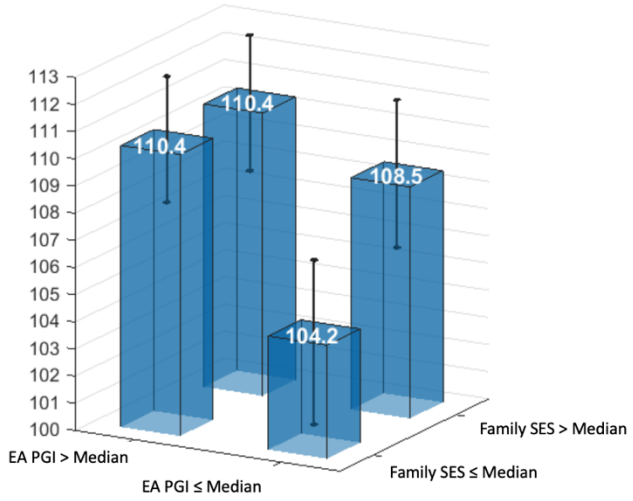

Figure 2. Mean cognitive performance among the Korean adoptees by EA PGI × family SES quadrants, conditional on the control variables.

We conducted a suite of robustness checks to assess the robustness of the negative interaction between family SES and the PGIs of cognitive performance and EA on cognitive performance, using Model II. We examined if the negative interaction is robust to limiting the sample to the male or female adoptees only; to splitting the sample at the median measurement age (15 years old, at intake); to scale transformations of the cognitive performance and family SES variables; to conditioning on an extensive set of control variables; and to dichotomizing the family SES variable by replacing it by a dummy indicating whether one's family SES is above the median.[39] The set of extensive control variables include the baseline controls plus the rearing mother's and father's ages when the child was born, the number of adoptive and biological siblings, and dummies indicating whether the family is a mixed biolgical and adoptive (vs. a purely adoptive) family, whether the adoptees' adoptive parents reside in a city or suburb, and whether they were still married at intake. Online Appendix Table G.7 shows the results; they are remarkably robust, with all estimates negative and most estimates with the PGI of EA significant at the 5% level.

We repeated the baseline analyses in the sample of European ancestry biological children. As can be seen in Online Appendix Table G.8, there is little evidence of interactions between the PGIs and family SES in that sample (except for the outcome college attendance, for which we estimate

[39] If we group the terms that involve PGI in our baseline specification, we obtain $Y = \beta_0 + \beta_1 F + (\beta_2 + \beta_3 F)PGI + \epsilon$. Such a specification, with continuous PGI and F variables, is commonly used in the GxE literature, but if $\beta_3 < 0$ it implies that PGI has a negative impact on adoptees from families with a sufficiently high F (for whom $\beta_2 + \beta_3 F < 0$). Dichotomizing F (i.e., family SES) allows us to circumvent that issue. We also verified that the negative interaction result is robust to dichotomizing the cognitive performance PGI instead of family SES.

a negative interaction significant at the 5% level). The absence of an interaction effect for cognitive performance in the sample of biological children stands in contrast to the presence of a negative interaction among the Korean adoptees. One explanation for this discrepancy is that the latter negative interaction is a false positive result; another possibility is that the estimate of the interation effect in the sample of biological children is biased because family SES is not exogenous and is correlated with genetic propensity for SES in that sample.

These results relate to a sizeable literature that uses twin- or pedigree-based research designs to test the Scarr-Rowe hypothesis, according to which the heritability of cognitive performance is reduced in lower SES families. A recent meta-analysis by Tucker-Drob & Bates (2016) found support for the hypothesis among U.S.-based studies, but not in studies from Western Europe or Australia, where the range of family environments may be more restricted. In the largest study to date, however, Figlio et al. (2017) found no evidence that heritability varied as a function of family SES. Recently, Rask-Andersen et al. (2021), using molecular genetic data, found that the SNP heritability of fluid intelligence and educational attainment, as well as the predictive power of PGIs for these traits, are higher among lower SES families. Of note, these studies analyzed data from non-adoptive families, so the environmental variables analyzed were not exognous to the genetic factors. Here, in a sample of quasi-randomly assigned Korean adoptees, we find suggestive evidence that genetic influences are stronger in lower SES families, which is consistent with the Rask-Andersen et al. (2021) results, but contrary to what the Scarr-Rowe hypothesis predicts. We note, however, that our sample does not contain many families characterized by poverty and privation, so our results are not informative about the relative importance of genetic influences in such families.

5.2 Power considerations

With a sample of only 361 genotyped Korean adoptees, statistical power to detect a GxE interaction may be limited. To further evaluate this, we derived an expression to calculate statistical power analytically under simple assumptions, and verified the results through simulations. Both our calculations and simulations suggest that statistical power to estimate a significant GxE effect in our sample may be limited. For example, if we assume that the R^2 of the GxE interaction term is $R^2_{GxE} = 0.01$ (which is ~20% as large as the R^2 of the PGI and ~50% as large as that of family SES in simple regressions), then power is only ~45%; if we instead assume that R^2_{GxE} =0.005, then power is ~25%. To obtain at least 80% power, we need to assume that $R^2_{GxE} \geq \sim 0.025$, which is ~50% as large as the R^2 of the PGI and may thus be unrealistic given the literature's limited success in identifying robust GxE effects so far. Given this and the well-known fact that significant results tend to be particularly large in magnitude when the null hypothesis is true and power is limited (Gelman & Carlin 2014), our finding of a significant GxE interaction between the PGI of EA and

family SES on cognitive performance should be taken as no more than tentative until replication is possible in a larger, independent sample. Online Appendix F includes the derivations of the analytical expression to compute power and provides more detail on the simulations as well as the Stata code for the simulations.

6. NATURE AND NURTURE INTERACTIONS: EVIDENCE FROM PEDIGREE DATA

To further test for complementarities and substitution effects between genetic and environmental factors in the human capital production function, we leveraged our pedigree data. We estimated an extended ACE model that allows for moderating effects of (adoptive) family SES on the relative influences of additive genetic, common environmental, and unexplained factors. This extended ACE model[40] is identical to the one from Section 3 that allowed for moderating influences of age at measurement, but with family SES replacing age as the moderating variable:

$$\tilde{Y} = (a_0 + a_1 SES)A + (c_0 + c_1 SES)C + (e_0 + e_1 SES)E.$$

A positive estimate of a_1 would imply an increasing share of the outcome variance attributable to additive genetic factors as a function of family SES, and would suggest compelementarities between genetics and family SES. Online Appendix D provides further details on this extended ACE model and on how we estimated it via GMM.

Online Appendix Figure G.2 plots, for each outcome and as a function of (adoptive) family SES, the shares of the outcome variance that are attributable to additive genetic, common environmental, and unexplained factors, as well as the outcome variance. Overall, we find little evidence that family SES and genetics are complements or subsitutes, or that family SES moderates the relative importance of the three factors. For cognitive performance, heritability is flat at ~0.3 over the range of observed family SES. This again contradicts the Scarr-Rowe hypothesis, according to which cognitive performance has a higher heritability among higher-SES families (this also does not support our above finding of a gene-environment interaction on cognitive performance between family SES and the PGIs of cognitive performance and of educational attainment). As with the model with age as the moderating factor, possible reasons for this lack of evidence include a true lack of moderating effects (which would imply our results are true negatives) as well as the small size of our sample and the limited range of observed family SES in our data (which would imply our results are false negatives).

[40] The behavioral genetics literature discussed in Section 5 regarding the Scarr-Rowe hypothesis has mainly relied on such extended ACE models estimated with pedigree data.

7. CONCLUSION

In this study, we leveraged a unique dataset of Korean-American adoptees who were quasi-randomly assigned to adoptive families *and* who have been genotyped, to study nature-nurture interplay for 10 outcomes. Our results suggest that both nature and nurture play a fundamental role in shaping socioeconomic outcomes in adolescence and adulthood. In general, family environment appears to have particularly strong influences on educational outcomes, income, and nicotine usage, whereas genetics appear to have stronger influences on GPA, soft skills, cognitive performance, BMI, and height. However, most outcomes are jointly influenced by both genetics and the common family environment. Our analyses and data allow us to peek into the black box of the common family environment and imply that parental EA, income, SES, and substance use habits, as well as family size (or their correlates) play important roles for some of the outcomes. We also find evidence consistent with the existence of genetic nurture (though we cannot rule out confounding by cultural or environmental factors). Finally, we document a robust negative GxE interaction on cognitive performance between family SES and the PGI for EA (and that of cognitive performance), suggesting they may be substitutes in the human capital production function.

Several limitations of our analyses should be mentioned. First, the small size of our sample of Korean adoptees limits statistical power and reduces the precision of our estimates. While results from the ACE variance decomposition affirm the importance of both genetic and family environmental factors, they are not precise enough to draw firm conclusions regarding their relative importance for most of the studied outcomes. Further, the negative interaction between family SES and genetics on cognitive performance must be seen as suggestive until replicated in an independent and larger dataset. Second, the PGIs are imprecise proxies for the true genetic factors and the family variables that we observe may fail to capture important dimensions of the family environment. Thus, results from our regressions of outcomes on the PGIs and family variables establish lower bounds for the roles of genetics and family environment and only inform the relative importance of variables that are *currently* typically available to researchers. Third, although the quasi-random assignment of the Korean adoptees improves the internal validity of this study, thereby addressing several limitations in the current GxE literature, our study is still subject to concerns surrounding its external validity. These include concerns that adoptive families do not constitute a representative subset of the population, which could result in the underestimation of environmental effects due to a restricted range of exposures (Stoolmiller 1998, 1999). In addition, Korean adoptees (and adoptees in general) may be treated differently by parents or educators or exhibit traits that differ from a representative population. While our sample is too small to weigh in on this, recent research conducted in a large sample of Korean adoptees in

Norway suggests this type of bias is minimal (Fagereng et al. 2021). Fourth, most of our outcomes—including cognitive performance, drinking, and nicotine usage—were measured (partly or fully) before adulthood. There is evidence that for these three outcomes and for other traits like savings behavior, the common family environment becomes much less important in adulthood (Bouchard 2013; Cronqvist & Siegel 2015; Kendler et al. 2008). Nonetheless, for drinking, educational attainment, college completion, and income, we find evidence of common family environmental effects in adulthood.

Finally, we are limited in the extent to which we can assess potential mechanistic pathways between family characteristics, genetic factors, and adoptee outcomes. There is a substantial literature on parental investments and their reaction to children's endowments (e.g., Aizer & Cunha 2012; Becker & Tomes 1976; Heckman & Mosso 2014), including recent evidence based on molecular genetic data that parents respond to their children's genetics (Breinholt & Conley 2023; Fletcher et al. 2020; Houmark et al. 2021; Sanz-de-galdeano & Terskaya 2019). While our results show that adoptees' genetics influence their outcomes, we did not examine whether that influence is moderated in part by adoptive parents' reactions to the adoptees' genetics. And while we document *common* family environmental effects, we did not explore whether family effects are heterogenous across siblings, including as a function of their genetics or sex.

Overall, our research design demonstrates the usefulness of studying quasi-randomly assigned adoptees when incorporating genetic data into economic analyses. As the costs of genotyping and whole genome sequencing continue to fall, future work in larger samples—like Sacerdote's (2007) sample of Korean adoptees from Holt or the large sample of Norwegian Korean adoptees analyzed by Fagereng et al. (2021)—could be incredibly useful in obtaining more precise estimates of nature and nurture and in studying how they interact through possible dynamic complementarities over the lifecycle. This would lead to an enhanced understanding of the human capital production function and of the factors that contribute to inequality and intergenerational mobility, with important policy implications.

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Table 1: Summary statistics

	Korean adoptees			European ancestry adoptees			European ancestry bio. children		
	Mean	Std. dev.	*N*	Mean	Std. dev.	*N*	Mean	Std. dev.	*N*
Children									
Genotyped & not a genetic outlier	0.86	0.35	421	0.87	0.34	141	0.87	0.33	471
Genotyped, non-outlier children									
Baseline controls									
Male	0.39	0.49	361	0.54	0.50	122	0.47	0.50	411
Age at intake	15.00	1.86	361	15.05	2.19	122	14.87	1.89	411
Age at first follow-up	18.37	2.10	354	18.40	2.38	119	18.16	1.99	401
Age at second follow-up	22.36	1.78	346	22.68	2.09	115	22.25	1.83	393
Age at third follow-up	32.38	2.57	249	32.02	2.63	94	31.63	2.54	313
Age 16 or older at intake	0.25	0.43	361	0.31	0.47	122	0.27	0.45	411
Birth year	1985.98	2.77	361	1986.43	2.97	122	1986.71	2.78	411
Placement age (in months)	5.20	2.66	361	2.50	3.18	122	.	.	0
Number of siblings in the rearing family	1.59	1.08	361	1.37	0.76	122	2.29	1.33	411
Outcomes									
EA	16.22	2.12	233	15.51	2.15	85	16.12	1.86	288
College	0.73	0.45	233	0.52	0.50	85	0.74	0.44	288
GPA	3.43	0.77	355	3.00	1.02	117	3.46	0.74	395
Soft skills	0.10	0.97	361	-0.37	0.95	122	0.10	0.99	410
Cognitive performance	108.45	13.79	361	104.88	14.90	122	108.49	12.94	409
Log income	10.85	0.66	210	10.85	0.54	74	10.87	0.66	264
Drinks per week	0.15	6.57	361	0.51	6.71	122	0.31	6.62	411
Ever used nicotine	0.75	0.44	361	0.80	0.41	122	0.67	0.47	411
BMI	23.06	3.90	309	23.35	5.15	93	23.56	4.46	355
Height	165.20	7.86	309	171.37	8.03	93	172.80	8.57	355

(Continues)

Table 1 (Continued): Summary statistics

	Korean adoptees			European ancestry adoptees			European ancestry bio. children		
Variable	Mean	Std. dev.	*N*	Mean	Std. dev.	*N*	Mean	Std. dev.	*N*
Baseline family variables									
Mother's EA	16.59	1.81	360	15.97	1.73	122	15.96	1.71	411
Mother's cognitive performance	115.86	13.80	291	107.16	12.47	77	109.80	13.61	296
Mother's drinks per week	2.87	4.60	359	1.98	3.61	119	2.99	4.92	406
Mother ever used nicotine	0.23	0.42	361	0.19	0.40	119	0.20	0.40	409
Mother's BMI	27.82	7.01	285	28.18	5.38	80	27.66	5.98	320
Mother's height	165.13	6.43	290	166.48	7.42	88	166.07	6.48	328
Mother's age when child was born	33.06	3.60	361	32.40	3.55	117	29.70	4.22	409
Father's EA	16.96	1.79	359	16.24	1.87	121	15.96	2.00	409
Father's age when child was born	34.75	3.96	336	33.79	3.15	111	31.56	5.07	354
Family SES	0.25	0.90	361	-0.09	0.97	118	-0.20	1.03	402
Log family income	11.34	0.46	306	11.31	0.58	97	11.26	0.47	289
Parent disinhibition score	-0.31	0.80	359	-0.36	0.76	122	0.00	1.03	411
Number of siblings in the rearing family	1.59	1.08	361	1.37	0.76	122	2.29	1.33	411
Mixed biological & adoptive family	0.17	0.37	361	0.20	0.40	122	0.22	0.41	411
Family lives in a city or suburb	0.72	0.45	360	0.59	0.49	122	0.75	0.43	409
Parents still married at intake	0.92	0.28	361	0.92	0.28	122	0.87	0.34	411
Other variables									
Mother is genotyped	0.86	0.35	361	0.84	0.36	122	0.91	0.29	411
Father is genotyped	0.71	0.45	361	0.75	0.44	122	0.69	0.46	411
Family type dummies									
Type 1 family	0.44	0.50	361	0.31	0.47	122	0.22	0.42	411
Type 2 family	0.43	0.50	361	0.47	0.50	122	0.45	0.50	411
Type 3 family	0.14	0.35	361	0.25	0.43	122	0.33	0.47	411

Note: Summary statistics for all variables were computed at the level of the children, including summary statistics for the mother, father, and family variables (therefore, if a mother, father, of family has two genotyped, non-outlier children, then it will be double-counted). The Type 1, Type 2, and Type 3 family dummy variables are defined in Section 4.3. Summary statistics for PGIs and PCs are not reported as these do not have a natural scale and were therefore all standardized, separately in the MCTFR-SIBS samples of European and Korean ancestry individuals, so that they have mean zero and unit variance in each sample.

Table 2: Tests of random placement of the Korean adoptees

	(1) Male	(2) Placement age	(3) PGI of EA	(4) PGI of cog. perf.	(5) PGI of income	(6) PGI of ever smoker	(7) PGI of BMI	(8) PGI of height
Baseline family variables								
Mother's EA	0.017	-0.042	0.043	0.045	0.044	-0.038	-0.044	0.044
	(0.020)	(0.133)	(0.046)	(0.048)	(0.050)	(0.042)	(0.043)	(0.041)
Mother's CP	0.002	0.001	-0.002	-0.003	-0.001	0.000	-0.000	-0.001
	(0.002)	(0.012)	(0.005)	(0.005)	(0.005)	(0.004)	(0.005)	(0.005)
Mother's DPW	0.006	-0.005	-0.002	0.017	-0.006	0.015	0.006	-0.002
	(0.005)	(0.031)	(0.012)	(0.013)	(0.012)	(0.010)	(0.011)	(0.012)
Mother ever used nicotine	0.030	0.814**	0.031	-0.099	0.007	0.023	-0.024	-0.151
	(0.055)	(0.331)	(0.145)	(0.145)	(0.146)	(0.123)	(0.128)	(0.129)
Mother's BMI	0.000	-0.075**	0.007	0.003	0.007	-0.006	-0.002	0.004
	(0.004)	(0.031)	(0.010)	(0.010)	(0.009)	(0.009)	(0.008)	(0.009)
Mother's height	0.010**	-0.032	-0.020*	-0.017*	-0.014	0.021**	0.005	-0.002
	(0.004)	(0.036)	(0.011)	(0.010)	(0.010)	(0.009)	(0.009)	(0.009)
Mother's age when child was born	-0.017*	0.049	0.005	-0.006	-0.007	0.012	0.008	-0.016
	(0.009)	(0.064)	(0.023)	(0.022)	(0.020)	(0.020)	(0.021)	(0.021)
Father's EA	-0.009	-0.220	0.062	0.041	0.076	0.005	-0.011	-0.034
	(0.021)	(0.196)	(0.055)	(0.055)	(0.061)	(0.044)	(0.044)	(0.051)
Father's age when child was born	-0.004	0.045	-0.006	0.006	-0.003	-0.000	-0.001	-0.012
	(0.007)	(0.072)	(0.018)	(0.019)	(0.018)	(0.019)	(0.019)	(0.018)
Family SES	0.008	0.573	-0.277	-0.250	-0.302	-0.014	0.121	-0.017
	(0.071)	(0.514)	(0.175)	(0.177)	(0.193)	(0.135)	(0.142)	(0.155)
Log family income	-0.088	-0.874*	0.258	0.303	0.325	-0.116	-0.186	0.015
	(0.095)	(0.509)	(0.216)	(0.209)	(0.236)	(0.211)	(0.212)	(0.208)
Parent disinhibition score	0.046	-0.437**	-0.059	-0.102	-0.102	-0.041	0.095*	0.136*
	(0.032)	(0.212)	(0.071)	(0.069)	(0.072)	(0.080)	(0.054)	(0.075)
Number of siblings in the rearing family	-0.054**	0.067	0.034	-0.008	0.012	0.054	0.014	0.014
	(0.022)	(0.180)	(0.056)	(0.057)	(0.056)	(0.060)	(0.054)	(0.048)
Mixed biological & adoptive family	0.129**	-0.139	0.245*	0.168	0.252*	-0.006	0.093	0.211
	(0.060)	(0.400)	(0.147)	(0.137)	(0.150)	(0.136)	(0.147)	(0.162)
Family lives in a city or suburbs	-0.021	-0.517	0.001	0.057	-0.036	-0.049	-0.239**	0.225*
	(0.053)	(0.545)	(0.140)	(0.133)	(0.140)	(0.125)	(0.120)	(0.125)
Parents still married	0.067	0.609	0.143	0.204	0.138	-0.570**	-0.235	0.169
	(0.084)	(0.432)	(0.208)	(0.242)	(0.219)	(0.230)	(0.197)	(0.252)
Observations	414	414	354	354	354	354	354	354
R^2	0.187	0.093	0.043	0.045	0.049	0.069	0.038	0.066
Test statistic, joint signif. of family var.	43.860	1.302	0.818	0.658	0.858	1.549	0.943	1.289
P value	0.002	0.173	0.697	0.871	0.646	0.0632	0.536	0.183

Note: All regressions control for adoptee birth year and its square. To maximize regression sample size, missing observations were coded as 0 and dummies indicating missing observations were included for five baseline family variables with high numbers of missing observations (mother's cognitive performance, mother's BMI, mother's height, father's age when child was born, log family income). The family variables for the tests of joint significance include the baseline family variables as well as these five dummies. For the continuous outcomes, OLS regressions were estimated and the test statistic for joint significance is the *F* statistic. For the binary outcome (male), a logistic regression was estimated, the reported coefficients are average marginal effects, Nagelkerke's pseudo R^2 was used, and the test statistic for joint significance is the Wald statistic. Robust standard errors clustered at the family level are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 3: Correlations in outcomes among pairs of adopted and biological siblings and resulting variance decomposition estimates from the ACE model

	Panel A: Adoptive and biological siblings correlations				Panel B: Estimated proportion of outcome explained by genetics (σ_A^2), common family env. (σ_C^2), and unexplained factors (σ_E^2)		
	Adoptive sibling correlation	*N (pairs)*	Biological sibling correlation	*N (pairs)*	σ_A^2	σ_C^2	σ_E^2
EA	0.225*	103	0.323**	89	-0.074	0.282***	0.792***
					(0.302)	(0.112)	(0.224)
College	0.268**	104	0.295**	89	0.070	0.254***	0.676***
					(0.323)	(0.105)	(0.262)
GPA	0.118	238	0.294***	176	0.310*	0.130**	0.561***
					(0.204)	(0.065)	(0.167)
Soft skills	0.141*	247	0.282***	181	0.281*	0.141**	0.577***
					(0.210)	(0.064)	(0.176)
Cognitive perf.	0.080	246	0.317***	181	0.330**	0.091*	0.579***
					(0.196)	(0.066)	(0.157)
Log income	0.241*	85	0.113	78	-0.245	0.228***	1.017***
					(0.270)	(0.087)	(0.221)
Drinks per week	0.122	212	0.197**	155	0.145	0.123**	0.732***
					(0.220)	(0.069)	(0.183)
Ever used nicotine	0.233**	169	0.295**	133	0.176	0.218***	0.606***
					(0.250)	(0.078)	(0.208)
BMI	0.165*	188	0.486***	150	0.840***	0.144**	0.015
					(0.257)	(0.074)	(0.211)
Height	0.136	188	0.417***	150	0.622***	0.112**	0.266*
					(0.214)	(0.068)	(0.176)

Note: Adoptive sibling correlations were computed among families with at least one Korean adoptee; biological sibling correlations were computed for the European ancestry biological children only. In Panel A, correlations were estimated after partialling out the effects of a vector *X* that includes the baseline control variables, a dummy indicating adoptee vs. biological child status, and an intercept. In Panel B, GMM was used to estimate the ACE variance shares (σ_A^2, σ_C^2, and σ_E^2), as described in the text. We do not constrain estimates of variance shares to be nonnegative (e.g., for σ^2_A for EA and log income). GMM standard errors are in parentheses. Since we are working with variances, *P* values for the variance shares were computed against a one-sided alternative. *** p<0.01, ** p<0.05, * p<0.1

Table 4: Regressions of Korean adoptee outcomes on family environmental variables and adoptee PGSs

	(1) EA	(2) College	(3) GPA	(4) Soft skills	(5) Cognitive performance	(6) Log income	(7) Drinks per week	(8) Ever used nicotine	(9) BMI	(10) Height
$\Delta\bar{R}^2$, family variables	0.080***	-0.037	-0.017	0.029*	0.015**	0.061***	-0.025	-0.063	0.066***	0.006*
Joint significance (*P*)	<0.001	0.194	0.788	0.069	0.048	0.002	0.257	0.591	0.006	0.099
$\Delta\bar{R}^2$, adoptee PGSs	0.056***	0.002**	0.072***	0.015**	0.052***	-0.011	-0.007	0.000	0.022	0.061***
Joint significance (*P*)	<0.001	0.040	<0.001	0.027	<0.001	0.661	0.578	0.140	0.108	<0.001
Observations	226	226	348	354	354	205	354	354	305	305
$\bar{R}^2$, all variables	0.176	-0.019	0.103	0.127	0.123	0.122	0.016	-0.040	0.115	0.598

Note: All regressions include the baseline control variables. To maximize regression sample size, missing observations were coded as 0 and dummies indicating missing observations were included for five family variables with high numbers of missing observations (mother's cognitive performance, mother's BMI, mother's height, father's age when child was born, log family income). The family variables include the baseline family variables as well as these five dummies. The adoptee PGIs include the PGIs of EA, cognitive performance, income, ever smoker, BMI, and height. For the continuous outcomes, OLS regressions were estimated, the adjusted R^2 was used, and the test for joint significance is the *F* test. For the binary outcomes (college and ever used nicotine), logistic regressions were estimated, McFadden's adjusted pseudo R^2 was used, and the test for joint significance is the Wald test. The incremental adjusted R^2 ($\Delta\bar{R}^2$) of each block of variables is the difference between the adjusted R^2 of the regression of the outcome on the controls and the variables in the block, and that of the same regression (in the same sample) but on the controls only. The stars on the $\Delta\bar{R}^2$'s indicate the significance level of the associated test for joint significance.
*** p<0.01, ** p<0.05, * p<0.1.

Table 5: Treatment effects of family type and PGI tercile for the Korean adoptees

	Panel A: Effect of family type				Panel B: Effect of PGI tercile				
	Type 1	Type 2	*N*	R^2	PGI	Tercile 3	Tercile 2	*N*	R^2
EA	1.320***	1.269***	261	0.099	EA	1.113***	0.716**	231	0.136
	(0.390)	(0.388)				(0.340)	(0.359)		
College	0.226***	0.176**	262	0.111	EA	0.163***	0.150**	231	0.237
	(0.0823)	(0.0789)				(0.0575)	(0.0606)		
GPA	-0.0657	0.0399	414	0.066	EA	0.354***	0.316***	355	0.132
	(0.130)	(0.132)				(0.0992)	(0.101)		
Soft skills	-0.0834	0.0398	421	0.105	EA	0.371***	0.201	361	0.151
	(0.135)	-0.137				(0.116)	(0.123)		
Cog. performance	3.547**	1.492	421	0.065	Cog. perf.	6.294***	1.894	361	0.127
	(1.788)	(1.830)				(1.773)	(1.902)		
Log income	0.146	0.212*	236	0.060	Income	0.277**	-0.010	208	0.159
	(0.125)	(0.120)				(0.108)	(0.120)		
Drinks per week	1.346	-0.233	421	0.017	Drinks per week	--	--	--	--
	(1.189)	(1.217)							
Ever used nicotine	-0.0221	-0.0588	361	0.120	Ever Smoker	0.0742	-0.00150	361	0.16
	(0.0713)	(0.0735)				(0.0556)	(0.0524)		
BMI	-0.476	0.0417	350	0.050	BMI	1.513***	0.365	309	0.097
	(0.669)	(0.702)				(0.569)	(0.545)		
Height	0.0906	-0.921	350	0.532	Height	3.791***	1.771***	309	0.586
	(1.057)	(1.064)				(0.749)	(0.676)		

Note: Each row in each panel represents a separate regression of an outcome on family type dummies (Panel A) or PGI tercile dummies (Panel B), with the Type 3 dummy omitted from the Panel A regressions and the Tercile 1 dummy omitted from the Panel B regressions. Panel B regressions are estimated in the sample of genotyped individuals only. All regressions include the baseline control variables (including the 10 top PCs of the ancestry-specific SNP data for the Panel B regressions). Type 1 families are defined as those with three or fewer children whose two parents each have a four-year college degree; Type 3 families are defined as those (i) with four or more children and where neither parent has a four-year college degree or (ii) in the bottom quintile of the SES distribution; Type 2 families are the families that are neither Type 1 nor Type 3. For the continuous outcomes, OLS regressions were estimated. For the binary outcomes (college and ever used nicotine), logistic regressions were estimated, the reported coefficients are average marginal effects, and Nagelkerke's pseudo R^2 was used. Robust standard errors are in parentheses and are clustered at the family level.
*** p<0.01, ** p<0.05, * p<0.1.

Table 6: Baseline GxE specification in the sample of Korean adoptees

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Dependent variable	EA	College	GPA	Soft skills	Cognitive performance	Cognitive performance	Log income	Ever used nicotine	BMI	Height
PGI	EA	EA	EA	EA	Cognitive performance	EA	Income	Ever smoker	BMI	Height
Panel A: Model I (without the interacted controls)										
PGI	0.543***	0.083***	0.182***	0.124***	3.400***	3.762***	0.078*	0.047*	0.464*	2.017***
	(0.125)	(0.028)	(0.042)	(0.047)	(0.668)	(0.705)	(0.040)	(0.026)	(0.249)	(0.335)
Family SES	0.713***	0.111***	0.017	-0.001	1.438**	1.795**	0.096*	0.007	0.022	0.414
	(0.144)	(0.034)	(0.048)	(0.054)	(0.725)	(0.749)	(0.056)	(0.026)	(0.241)	(0.338)
PGI x family SES	-0.032	-0.001	-0.037	0.031	-1.247*	-2.483***	0.042	0.017	0.377	0.059
	(0.130)	(0.031)	(0.050)	(0.049)	(0.648)	(0.766)	(0.038)	(0.025)	(0.243)	(0.361)
R^2	0.237	–	0.142	0.146	0.156	0.174	0.156	–	0.099	0.614
Panel B: Model II (with the interacted controls, following Keller 2013)										
PGI x family SES	-0.025	0.004	-0.049	-0.016	-1.464*	-2.847***	0.058	0.020	0.351	-0.143
	(0.167)	(0.037)	(0.056)	(0.059)	(0.763)	(0.880)	(0.048)	(0.031)	(0.244)	(0.425)
R^2	0.313	–	0.218	0.237	0.225	0.263	0.262	–	0.212	0.650
Observations	231	231	355	361	361	361	208	339	309	309

Note: Model I in Panel A includes the baseline control variables. Model II in Panel B also includes the interactions of these baseline controls with family SES and with the PGI, and is otherwise identical to Model I. Only the coefficient on the PGI x family SES interaction is reported for Model II, as the interacted controls make the coefficients on the PGI and Family SES difficult to interpret. For all outcomes (including the binary outcomes), OLS regressions were estimated. The number of observations is the same in Panels A and B for each outcome. Standard errors clustered at the family level are in parentheses.

*** p<0.01, ** p<0.05, * p<0.1

Nature-nurture interplay: Evidence from molecular genetic and pedigree data in Korean American adoptees*§

ONLINE APPENDIX

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A. DETAILS ON MOLECULAR GENETIC DATA PROCESSING AND POLYGENIC INDEX (PGI) CONSTRUCTION

Genome-wide genotyping was conducted on MCTFR studies using the Illumina Human660W-Quad array (Illumina, Inc., San Diego, CA). A total of 527,829 SNPs were genotyped (Miller et al. 2012).

To construct the PGI of a given outcome, we used available coefficient estimates (i.e., the $\hat{\beta}_j^Y$'s) from the largest possible GWAS of the outcome conducted among individuals of European descent, as listed in Appendix Table A.1. For the PGIs of educational attainment (EA), cognitive performance, income, and drinks per week (DPW), estimates from large GWASs of related outcomes were also available, so we used the multi-trait analysis of GWAS (MTAG) software (Turley et al. 2018). For the PGIs of ever smoker, height, and BMI, MTAG was not used as no large-scale GWAS of related traits were available (to our knowledge). MTAG combines summary statistics from GWAS estimates of related traits to generate more precise coefficient estimates for each of the jointly analyzed traits, thereby boosting statistical power to detect PGI associations for these traits. For instance, as shown in Appendix Table A.1, to generate the SNP coefficient estimates used to construct the PGI of drinks per week, we jointly analyzed estimates from a GWAS of drinks per week with those from a GWAS of the Alcohol Use Disorders Identification Test (AUDIT).

The MCTFR European ancestry individuals were used in the original GWAS meta-analysis for EA, cognitive performance, income, and DPW. To avoid overfitting, these individuals needed to be removed from the original GWAS to obtain new summary statistics before constructing the PGIs. While we were able to obtain new summary statistics for income and DPW with MCTFR European ancestry individuals removed, we were not able to obtain summary statistics without these individuals from the GWAS of cognitive performance by Savage et al. (2018), whose estimates are among those we used to construct the PGIs of EA and of cognitive performance. For these two outcomes, for the European ancestry individuals, we used "multi-trait" (i.e., MTAGed) PGIs from the SSGAC PGI Repository, which provides PGI based on summary statistics from GWAS that excluded MCTFR individuals (Becker et al. 2021). MCTFR-SIBS individuals of Korean ancestry were not included in the Savage et al. (2018) GWAS, so we used the Savage et al. (2018) estimates to construct their PGIs.

To maximize the predictive power of the PGIs, we utilized the software tool PRS-CS (Ge et al. 2019) to construct the PGIs, rather than simply taking the weighted sum of each individual's SNPs, as in Equation (1) of the main text. The PGIs of EA and cognitive performance we obtained from the SSGAC PGI repository for the European ancestry individuals were constructed using the software tool LDpred (Vilhjálmsson et al. 2015), assuming the infinitesimal model (see Becker et al., 2021 for more details). Both PRS-CS and LDpred use Bayesian methods to adjust the estimated GWAS coefficients to account for the fact they were estimated in regressions that did not control for correlated nearby

SNPs. To do so, both software use an external sample to model local linkage disequilibrium (LD) patterns (i.e., correlations between SNPs) in order to convert the GWAS regression coefficients from the GWAS summary statistics to partial regression coefficients (equivalent to the regression coefficients one would obtain from controlling for neighboring SNPs in the GWAS). PRS-CS also applies Bayesian shrinkage to the partial regression coefficients. The resulting partial regression coefficients are then used as weights in the PGIs. These features substantially improve the predictive performance of PRS-CS PGIs over most existing methods (Ge et al., 2019). To construct our PRS-CS PGIs, we used the 1000 Genomes European populations to estimate local LD patterns and calculated the shrunken partial regression coefficients for the SNPs. The PGIs were constructed using the ~450,000 to ~475,000 SNPs that were originally genotyped in MCTFR, successfully merged to GWAS or MTAG summary statistics, and survived all default software filters. Only genotyped SNPs were used to construct the PRS-CS PGIs (i.e., imputed SNPs were not used for these PGIs; imputed SNPs were used for the PGIs we obtained from the PGI repository).

Appendix Table A.1 below provides more details on PGI construction. For each of the seven PGIs we constructed, it shows the source of the GWAS summary statistics that were used to construct the PGI; the adoptee and biological child outcomes for which we used the PGI in our analyses; the other GWASs that were used with MTAG (if applicable for the PGI); the number of individuals in the original GWAS or the effective GWAS sample size (for the PGIs obtained with MTAG); the number of SNPs that were used to construct the PGI[1]; and whether MCTFR was included in the original GWAS meta-analysis and thus needed to be excluded to produce the summary statistics we used to construct the PGIs, to avoid overfitting.

[1] Not all the ~475,000 SNPs that were originally genotyped in MCTFR and survived all default PRS-CS filters could be used to construct the PGIs, since SNPs also had to be present in the GWAS summary statistics to be used.

Appendix Table A.1. Polygenic index (PGI) construction details

PGI	Outcomes analyzed with the PGI	Base GWAS	MTAGed GWASs	Effective GWAS sample size	Number of SNPs used to construct PGI	Exclusions from original GWASs
Educational attainment (EA[*])	EA College GPA Soft skills	Lee et al. (2018) [EA]	Lee et al. (2018) [cog. perf.] Savage et al. (2018) [cog. perf.[#]]	852,303	451,830	Data from MCTFR and 23andMe were excluded from both Lee et al. GWASs
Cognitive performance[*]	Cognitive performance	Savage et al. (2018) [cog. perf.[#]]	Lee et al. (2018) [cog. perf.] Lee et al. (2018) [EA]	414,022	451,830	
Income	Income	Kweon et al. (unpublished) [income]	Lee et al. (2018) [EA]	688,845	473,426	Data from MCTFR were excluded from Lee et al. GWAS
Drinks per week	Drinks per week	Liu et al. (2019) [drinks per week]	Sanchez-Roige et al. (2019) [AUDIT[&] total score]	599,173	474,873	Data from MCTFR and 23andMe were excluded from both Liu et al. GWASs
Ever smoker	Ever used nicotine	Liu et al. (2019) [ever smoker#]	--	1,232,091	474,881	
BMI	BMI	Loh et al. (2018) [BMI]	--	457,824	476,022	---
Height	Height	Loh et al. (2018) [height]	--	457,303	476,022	---

Note: PGIs were constructed using the software tool PRS-CS. For the PGI of EA, cognitive performance, income, and drinks per week, MTAG was used prior to PRS-CS to combine the estimates from the "base GWAS" with those from the "MTAGed GWAS"; for these PGI, the effective GWAS sample size is calculated by MTAG as the sample size that would have been needed for the mean chi-square statistics across the SNPs in the base GWAS to be equal to that attained by MTAG. For the PGI of ever smoker, BMI, and height, only the estimates from a "base GWAS" were used; for these PGI, the effective sample size is simply the sample size of the base GWAS. "Exclusions from original GWASs" indicates cohorts whose data were included in the original GWAS meta-analyses listed in the "Base GWAS" and "MTAGed GWAS" columns but excluded from the GWAS meta-analyses whose estimates we used to construct the PGIs (data from which MCTFR was excluded were needed to avoid overfitting; data from 23andMe could not be used due to access restrictions).

[*] The PGIs of EA and of cognitive performance constructed as described in this table were only used for the Korean adoptees; for European ancestry individuals, we used the "multi-trait" (i.e., MTAGed) PGIs of EA and of cognitive performance from the SSGAC PGI repository (Becker et al. 2021). We did so to avoid overfitting, as data from MCTFR European ancestry individuals were included in the Savage et al. GWAS and estimates from an equivalent GWAS excluding these data could not be obtained.

[#] For consistency with our terminology in the rest of the paper, we use the labels "cognitive performance" to refer to what Savage et al. call "intelligence" and "ever smoker" to refer to what Liu et al. call "smoking initiation".

[&] "AUDIT" stands for Alcohol Use Disorders Identification Test.

B. ADDITIONAL INFORMATION REGARDING VARIABLE DEFINITION

The main text describes in detail how the adoptee and biological child outcomes variables were constructed. Here we provide additional details regarding the construction of the family background variables.

Mother's or father's years of education: At intake, mothers and fathers were asked for information on the highest education degree they obtained. Responses were categorized on a five-point scale as "less than HS", "high school", "some college", "college", or "professional". We used the International Standard Classification of Education (ISCED) framework to convert highest degree obtained into years of education. Specifically, individuals reporting less than high school were assigned 10 years of education, a high school degree was converted to 13 years, some college was converted to 15 years, a college degree was converted to 17 years, and a professional degree was converted to 19 years.

Mother's cognitive performance: Mother's cognitive performance was assessed at the first follow-up using an abbreviated form of the Weschler Adult Intelligence Scale-Revised (WAIS-R) that consisted of two performance (Block Design and Picture Arrangement) and two verbal (Vocabulary and Information) subtests (Wechsler 1981). Prorated IQs, derived from the four subtests following standard procedures, have been shown to correlate 0.90 with IQs based on all Weschler subtests (Kaufman 1990).

Mother's drinks per week (DPW): Information on mother's alcohol use was assessed at intake. As with the adolescents, DPW was constructed using participant self-reports from categorical variables that assessed frequency of drinking and quantity of drinks consumed when drinking.[2] Both variables were converted to a weekly scale by taking the midpoint of each numeric range and then normalizing values reported per day or per month to their per-week equivalent. Frequency per week was then multiplied by quantity per week to create the DPW variable. Participants with more than 50 DPW were top coded at 50.

Mother ever used nicotine: Mothers were asked if they ever smoked or used nicotine at least once during their intake visit. Participants who reported smoking or using nicotine received a one for this variable and zero otherwise.

Mother's BMI: The height and weight of mothers was recorded in the first follow-up wave. Height was recorded in centimeters and weight was recorded in pounds. Weight was measured in person for

[2] Participants could report frequency of drinking as "non-drinker", "less than once a month", "1-3 times per month", "1-4 times per week", "daily", or "more than once per day". Quantity of drinking was reported as "non-drinker", "1-3 drinks", "4-6 drinks", "7-10 drinks", "11-20 drinks", "21-29 drinks", or "30 or more drinks".

the ~85% of respondents who came in to be interviewed and was measured via self-report over the phone for the remaining 15% of the respondents. We converted height in centimeters to meters and weight in pounds to kilograms to calculate BMI.

Mother's height: Mother's height in centimeters was measured at the first follow-up wave. Height was measured in person for the 85% of respondents who came in to be interviewed and via self-report over the phone for the remaining 15% of the sample.

Mother's or father's age when child was born: To construct age when child was born, we subtracted the mother's or father's birth year from the child's birth year for all adopted and non-adopted adolescents.

Log family income: Self-reported family income was assessed at the first follow-up wave. Parents selected their gross household income from a series of pre-defined income brackets that ranged from 1 ("less than $10,000/year") to 15 ("over $100,000/year"). We took the midpoint of each category to generate income in dollars except for the first income category, which was set to $7,500, and the final income category, which was set to $125,000, and then used the natural log of income for analysis.

Parent disinhibition score: Detailed information on psychological assessment of parental disinhibition and the construction of the parent disinhibition score is available in McGue et al. (2007). Briefly, the parent disinhibition score utilizes information from the Structured Clinical Interview for DSM-III-R (SCID-R) with updated interviews from DSM-IV criteria to assess antisocial personality disorder (DSM-III-R Personality Disorders, SCID-II). Parents were also administered the expanded substance abuse module (SAM) that was updated to cover DSM-IV criteria. All clinical assessments were performed during intake. The final score sums together the standardized (log transformed) symptom scales for adult antisocial behavior (AAB), alcohol abuse, and substance abuse for both the mother and the father. The composite score was still created if data was missing for either parent (i.e., up to three missing indicators were allowed). The final composite score was standardized in the sample of non-adoptive families.

Number of siblings in the rearing family: Reported by parents at intake; includes both adopted and non-adopted siblings.

Mixed biological and adoptive family: Dichotomous variable that equals one if the family has both biological and adopted children and zero otherwise.

Family lives in a city or suburb: Dichotomous variable that equals one if the family reports living in a large city or in the suburbs at intake, and zero if the family lives in a medium or small city or in a rural area.

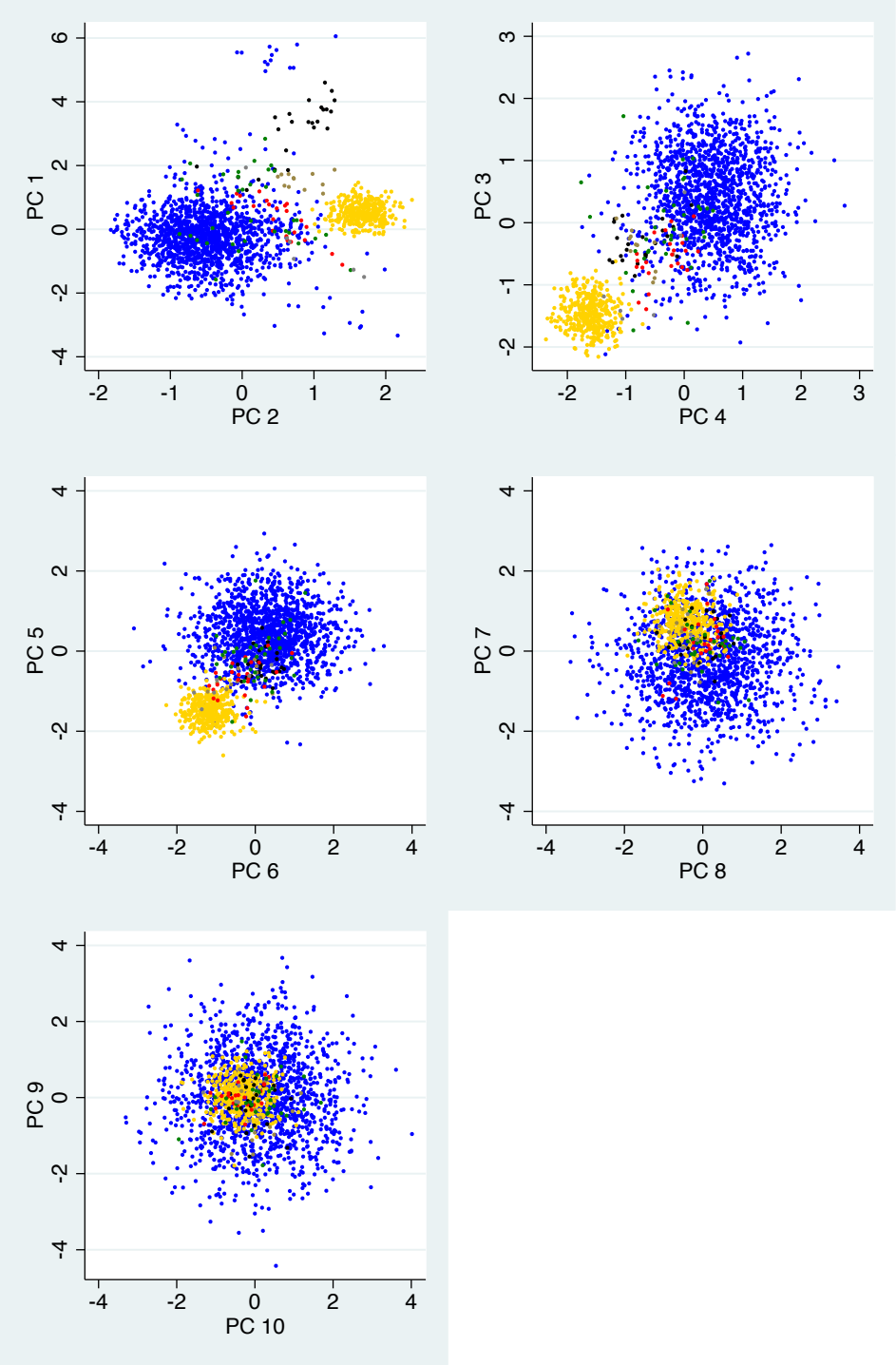
Parents still married at intake: Dichotomous variable equal to one if both the mother and father report still being married at intake and zero if either parent reports that they are single, living as a married couple with someone (parent or other), divorced, separated, widowed, or if the family reports never being married.

C. ANALYSIS OF GENETIC OUTLIERS

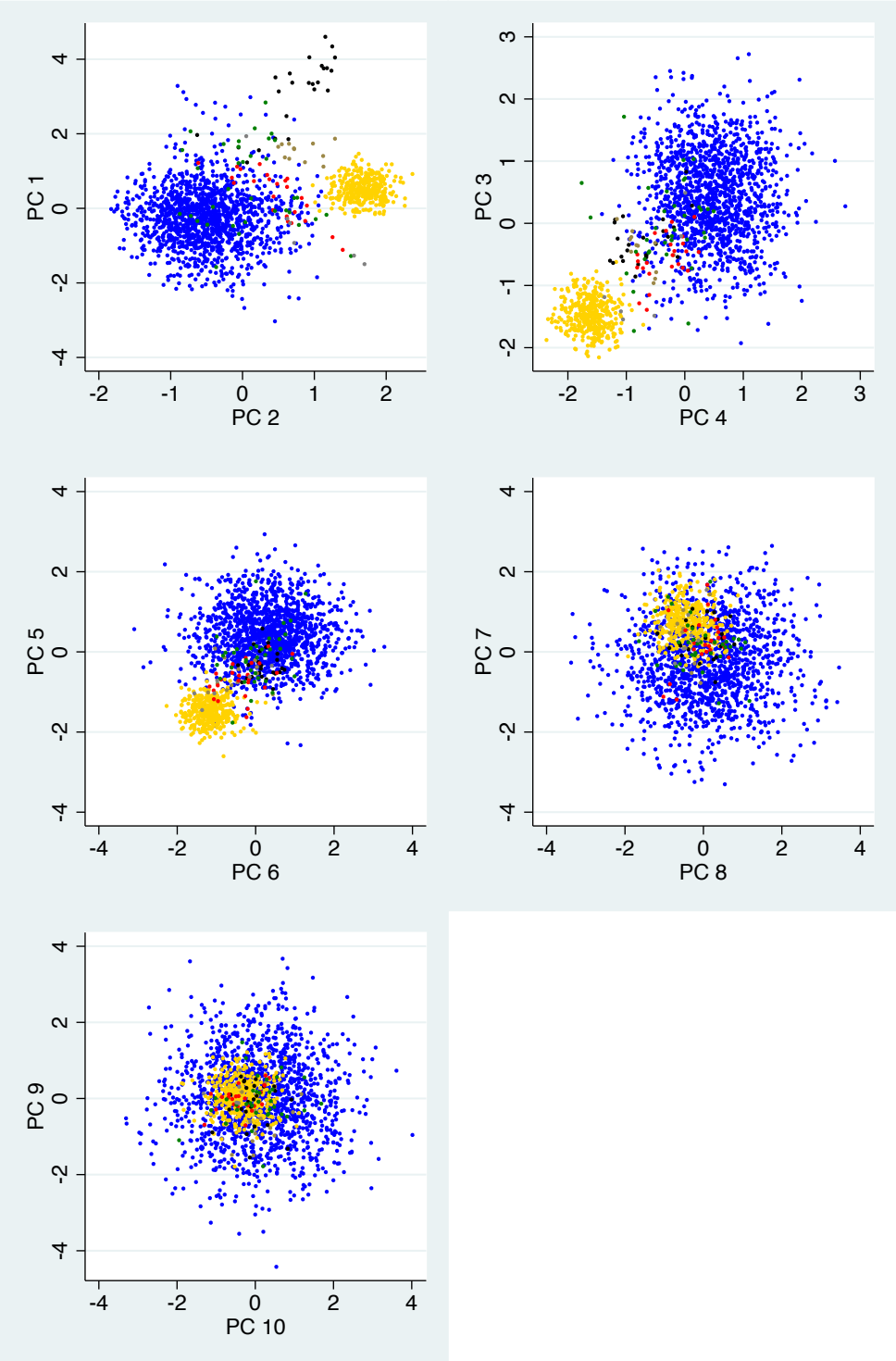
To identify the genetic outliers among the European and Korean ancestry individuals, we plotted and visually inspected the top 10 PCs of the genetic relatedness matrix of the full sample of MCTFR-SIBS individuals who have been genotyped. Appendix Figure C.1 shows these plots.

As can be seen from the figure, PC 1 mainly discriminates European ancestry individuals (the blue dots) from African Americans (the Black dots), though there are a number of European ancestry outliers with a high PC 1 and whom we identify as outliers. PC 2 mainly discriminates European from Korean ancestry individuals, though again there are a few European ancestry outliers with a high PC 2 and whom we identify as outliers. Finally, together PCs 3 and 4 also discriminate European from Korean ancestry individuals, though again there are a few European ancestry individuals in the Korean cloud, whom we also identify as outliers. Specifically, we identified as outliers the European ancestry individuals whose PC 1 is larger than 4, whose PC 2 is larger than 1.1, or whose PCs 3 and 4 are smaller than -1 and -0.9, respectively. The remaining PCs do not as clearly discriminate between European, Korean, and other ancestries, and so we did not use them to identify outliers.

Appendix Figure C.2 shows plots of the top 10 PCs without the outliers. As can be seen, there is now no more than minimal overlap between the European ancestry, Korean ancestry, and African American individuals.



Appendix Figure C.1. Top 10 principal components (PCs) of the genetic relatedness matrix of the full sample of MCTFR-SIBS genotyped individuals, plotted for all SIBS genotyped individuals. Blue dots represent European ancestry individuals, yellow dots Asians (Koreans), black dots African Americans, brown dots South Asians, red dots Hispanics, green dots Pacific Islanders, and grey dots all other individuals.



Appendix Figure C.2. Top 10 principal components (PCs) of the genetic relatedness matrix of the full sample of MCTFR-SIBS genotyped individuals, excluding the outliers. Blue dots represent European ancestry individuals, yellow dots Asians (Koreans), black dots African Americans, brown dots South Asians, red dots Hispanics, green dots Pacific Islanders, and grey dots all other individuals.

D. EXTENSIONS OF THE ACE MODEL

Here, we detail the three extensions of the ACE model mentioned in the main text.

D.1 Correlation between genetics and the shared family environment

In Section 3 of the main text, we relax the standard ACE-model assumption that A and C are uncorrelated. We use GMM to estimate the resulting extended ACE model. Additional moment conditions are required due to the introduction of a new parameter ($\gamma \coloneqq Cov(A, C)$) and to the fact that the outcome variance for biological children now differs from that for adoptive children. The resulting GMM moment conditions are:

$$\left[1\{AA\}\left(\widetilde{Y}_1\widetilde{Y}_2/\sigma^2_{\widetilde{Y}_A} - \sigma_c^2\right)\right] = 0;$$
$$E\left[1\{AB\}\left(\widetilde{Y}_1\widetilde{Y}_2/(\sigma^2_{\widetilde{Y}_A}r) - (\sigma_c^2 + \gamma)/r\right)\right] = 0;$$
$$E\left[1\{BB\}\left(\widetilde{Y}_1\widetilde{Y}_2/(\sigma^2_{\widetilde{Y}_A}r^2) - (0.5\sigma_a^2 + \sigma_c^2 + 2\gamma)/r^2\right)\right] = 0;$$
$$E[1 - \sigma_A^2 - \sigma_C^2 - \sigma_E^2];$$
$$E\left[1\{AA\}\left(\widetilde{Y}_1\widetilde{Y}_1 + \widetilde{Y}_2\widetilde{Y}_2 - 2\sigma^2_{\widetilde{Y}_A}\right)\right] = 0;$$
$$E\left[1\{AB\}\left(\widetilde{Y}_1\widetilde{Y}_1 + \widetilde{Y}_2\widetilde{Y}_2 - \sigma^2_{\widetilde{Y}_A} - \sigma^2_{\widetilde{Y}_A}r^2\right)\right] = 0;$$
$$E\left[1\{BB\}\left(\widetilde{Y}_1\widetilde{Y}_1 + \widetilde{Y}_2\widetilde{Y}_2 - 2\sigma^2_{\widetilde{Y}_A}r^2\right)\right] = 0;$$
$$E[\tilde{Y}_1X_1 + \tilde{Y}_2X_2] = 0;$$

where $1\{BB\}$, $1\{AA\}$, $1\{AB\}$ are dummies indicating biological-biological, adoptee-adoptee, and biological-adoptee sibling pairs; $\sigma^2_{\widetilde{Y}_A}$ denotes the variance of the residualized outcome $\tilde{Y}_A$ among adoptees; and r is not estimated as a parameter but is a shorthand for $\sqrt{\sigma_A^2 + \sigma_C^2 + \sigma_E^2 + 2\gamma}$, which is the square root of the ratio of the variance of the residualized outcome among biological children to that among adoptees (i.e., $r = \sqrt{\sigma^2_{\widetilde{Y}_B}/\sigma^2_{\widetilde{Y}_A}} = \sqrt{\sigma_A^2 + \sigma_C^2 + \sigma_E^2 + 2\gamma}$). As for the (non-extended) ACE model, we let β denote the coefficient on the purged covariates X (so, $\tilde{Y} = Y - X\beta$), and estimate β along with all the other parameters via GMM (so $\tilde{Y}$ is a shorthand for $Y - X\beta$).

Unlike Fagereng et al. (2021), we did not find that most outcomes have a larger variance among biological children than among adoptees, and so we did not introduce an additional parameter to allow the outcome variance to further vary (beyond what is already predicted by our extended ACE model) between biological children and adoptees.

D.2 Moderation of genetic and environmental effects by age or family SES

In Section 3 of the main text, we allow the age at which the outcome was measured to moderate the effects of additive genetic, common family environment, and unexplained factors (while maintaining the standard ACE-model assumption that A and C are uncorrelated). And in Section 6, we allow these effects to be moderated by family SES.

Formally, we let

$$\tilde{Y}_i = (a_0 + a_1 M_i)A_i + (c_0 + c_1 M_i)C_i + (e_0 + e_1 M_i)E_i,$$

where A, C, and E are now assumed to have unit variance; M denotes the moderating factor (age at outcome measurement or family SES); $i\epsilon\{1,2\}$ indexes sib i in a given pair; and, as before, $\tilde{Y} = Y - X\beta$ denotes the outcome Y purged of the covariates X. As before, X contains the baseline control variables, a dummy indicating adoptee vs. biological child status, and an intercept; in addition, when M is not among the baseline controls (i.e., when M is family SES), X also contains M.

To derive the moment conditions, consider first the variance and covariances implied by the model:

$$\begin{aligned}
E[\tilde{Y}_i^2] &= (a_0 + a_1 M_i)^2 + (c_0 + c_1 M_i)^2 + (e_0 + e_1 M_i)^2 \\
&= [{a_0}^2 + {c_0}^2 + {e_0}^2] + [2(a_0 a_1 + c_0 c_1 + e_0 e_1)]M_i + [{a_1}^2 + {c_1}^2 + {e_1}^2]{M_i}^2 \ (i\epsilon\{1,2\}); \\
E_{BS}[\tilde{Y}_1 \tilde{Y}_2] &= \frac{1}{2}(a_0 + a_1 M_1)(a_0 + a_1 M_2) + (c_0 + c_1 M_1)(c_0 + c_1 M_2) \\
&= \left[\frac{1}{2}a_0^2 + c_0^2\right] + \left[\frac{1}{2}a_0 a_1 + c_0 c_1\right](M_1 + M_2) + \left[\frac{1}{2}a_1^2 + c_1^2\right]M_1 M_2; \\
E_{AS}[\tilde{Y}_1 \tilde{Y}_2] &= (c_0 + c_1 M_1)(c_0 + c_1 M_2) \\
&= [c_0^2] + [c_0 c_1](M_1 + M_2) + [c_1^2]M_1 M_2.
\end{aligned}$$

This yields constrained regressions of $\tilde{Y}_i^2$ on $\boldsymbol{M_i} = [1, M_i, M_i^2]'$ among all (adopted and biological) children; of $\tilde{Y}_1 \tilde{Y}_2$ on $\boldsymbol{M_{1,2}} = [1, M_1 + M_2, M_1 M_2]'$, separately among biological siblings and among adoptive siblings (note that $M_1 = M_2$ when family SES is the moderator). Let $\widetilde{\tilde{Y}^2}$, $\tilde{Y}_1\widetilde{\tilde{Y}_{2BS}}$, and $\tilde{Y}_1\widetilde{\tilde{Y}_{2AS}}$ denote the residuals from these regressions:

$$\begin{aligned}
\widetilde{\tilde{Y}^2} &\coloneqq \tilde{Y}^2 - [{a_0}^2 + {c_0}^2 + {e_0}^2] - [2(a_0 a_1 + c_0 c_1 + e_0 e_1)]M - [{a_1}^2 + {c_1}^2 + {e_1}^2]M^2; \\
\tilde{Y}_1\widetilde{\tilde{Y}_{2BS}} &\coloneqq \tilde{Y}_1 \tilde{Y}_2 - \left[\frac{1}{2}a_0^2 + c_0^2\right] - [a_0 a_1 + 2c_0 c_1]M - \left[\frac{1}{2}a_1^2 + c_1^2\right]M^2; \\
\tilde{Y}_1\widetilde{\tilde{Y}_{2AS}} &\coloneqq \tilde{Y}_1 \tilde{Y}_2 - [c_0^2] - [2c_0 c_1]M - [c_1^2]M^2.
\end{aligned}$$

We obtain the following 10 moment conditions (where the first 3 lines each contain 3 moment conditions):

$$E\left[1\{BS\}\,\tilde{Y}_1\widetilde{\tilde{Y}_{2BS}}\,\boldsymbol{M_{1,2}}\right] = 0;$$

$$E\left[1\{AS\}\,\tilde{Y}_1\widetilde{\tilde{Y}_{2AS}}\,\boldsymbol{M_{1,2}}\right] = 0;$$
$$E\left[\widetilde{\tilde{Y}_1}^2\boldsymbol{M}_1 + \widetilde{\tilde{Y}_2}^2\boldsymbol{M}_2\right] = 0;$$
$$E[\tilde{Y}_1 X_1 \;+\; \tilde{Y}_2 X_2] = 0.$$

For the outcomes DPW and NIC, which were measured three times (at intake and at the first two follow-ups), for the case where age is the moderator, we treated each pair of measurement (for a sib pair) as a separate observation (instead of computing a summary variable that combines the information from the three waves, as we do for all other analyses); these observations were treated as a panel and we clustered standard errors at the sib-pair level. For all other outcomes and for the case where family SES is the moderator, we treated the outcome data in the same way as for all other analyses.

To help interpret the above model's estimates, let $\sigma^2_{\tilde{Y}|M}$ denote the moderator-dependent outcome variance, and let $\sigma^2_{A\,|M}$, $\sigma^2_{C\,|M}$, and $\sigma^2_{E\,|M}$ denote the outcome variance that is attributable to additive genetic, common environmental, and unexplained factors, respectively:

$$\sigma^2_{A\,|M} = (a_0 + a_1 M)^2;$$
$$\sigma^2_{C|M} = (c_0 + c_1 M)^2;$$
$$\sigma^2_{E|M} = (e_0 + e_1 M)^2;$$
$$\sigma^2_{\tilde{Y}|M} = \sigma^2_{A|M} + \sigma^2_{C|M} + \sigma^2_{E|M}.$$

We then define the moderator-dependent *shares* (or fractions) of the outcome variance that are attributable to additive genetic, common environmental, and unexplained factors:

$$\sigma^2_{A(share)} = \sigma^2_{A\,|M}/\sigma^2_{\tilde{Y}|M};$$
$$\sigma^2_{C(share)} = \sigma^2_{C\,|M}/\sigma^2_{\tilde{Y}|M};$$
$$\sigma^2_{E(share)} = \sigma^2_{E\,|M}/\sigma^2_{\tilde{Y}|M},$$

where we omit M as a subscript for notational simplicity.

Finally, to further help interpret the estimates, we define a metric, Δ_M, that indicates the predicted change in each variance share ($\sigma^2_{A(share)}$, $\sigma^2_{C(share)}$, and $\sigma^2_{E(share)}$) for a given outcome as one moves from a low level of the moderator M to another, higher level. For the results with family SES as the moderator, for a given outcome, the metric Δ_{SES} indicates the difference in each share associated with a change from a family SES of -1 to a family SES of 1. For the results with age at outcome measurement as the moderator, the metrics Δ_{age} indicates the difference in each share associated with a change from a 10th to the 90th percentile of the age distribution for the outcome.

Appendix Figures G.1 and G.2 show, for each outcome, these shares as a function of age and family SES.

E. INTERPRETING ESTIMATES FROM REGRESSIONS ON PGIS

This Appendix complements Section 4.1.2 in the main text. It explains why our estimated outcome-PGI associations in the sample of Korean adoptees are not unbiased estimates of the causal effect of the PGIs and proves Proposition 1.

E.1 Why estimated outcome-PGI associations are not unbiased estimates of the causal effects of the PGIs

Consider a fictitious world with no assortative mating or population stratification and where an experimenter can permute chromosomes across individuals at conception. Let us assume that A is uncorrelated with C (as in our sample of Korean adoptees) and with E. And let us distinguish, in the ACE model, between the part of A that is predicted by the PGI ($\beta \cdot PGI$) and the remaining part (A'):

$$Y = A + C + E = (\beta \cdot PGI + A') + C + E.$$

In that setting, regressing Y on the PGI yields an unbiased estimate of β, which is the causal effect of the PGI on Y, as defined in the main text. Next, consider the real world, in which assortative mating and population stratification cannot be assumed away. These generate correlations between the PGI and A'. Thus, regressing Y on the PGI does not yield an unbiased estimate of β, due to the now correlated omitted variable A'.

E.2 Proof of Proposition 1

Here we show formally that under Assumption 1, the true (population) R^2 of the PGI in a regression of the outcome Y on the PGI is no larger than that additive genetic variance in Y (Proposition 1). The proof is for the case with a single PGI, but can easily be generalized for the case with multiple PGIs.

For simplicity (and without loss of generality), let Y be standardized, with zero mean and unit variance. If we regress Y on the PGI only, the estimated coefficient on the PGI will be

$$\hat{\beta} = \mathrm{Cov}(PGI, Y)/\mathrm{Var}(PGI) = \mathrm{Cov}(PGI, \beta \cdot PGI + A' + C + E)$$
$$= \beta + \sigma_{PGI,A'} + \sigma_{PGI,C} + \sigma_{PGI,E} = \beta + \sigma_{PGI,A'} + \sigma_{PGI,E},$$

where $\sigma_{PGI,A'}$, $\sigma_{PGI,C}$, and $\sigma_{PGI,E}$ are the covariances between the PGI and A', C, and E; $\sigma_{PGI,C} = 0$ due to the quasi-random assignment of the adoptees; and where the other terms are defined in the text. Using the fact that both the PGI and Y have unit variance, it follows that the true (population) R^2 of the PGI is:

$$R^{o\,2}_{PGI} = \hat{\beta}^2 = \beta^2 + {\sigma_{PGI,A'}}^2 + {\sigma_{PGI,E}}^2 + 2\,\beta\sigma_{PGI,A'} + 2\sigma_{PGI,E}\sigma_{PGI,A}$$
$$= \left[\beta^2 + 2\,\beta\sigma_{PGI,A'}\right] + \left[{r_{PGI,A'}}^2\sigma^2_{A'} + \vartheta\left(\sigma_{PGI,E}\right)\right],$$

where r denotes a correlation; where we've used the equality $\sigma_{PGI,A} = \beta + \sigma_{PGI,A'}$; and where $\vartheta(\sigma_{PGI,E}) \equiv \sigma_{PGI,E}(\sigma_{PGI,E} + 2\sigma_{PGI,A})$ is a quadratic function of $\sigma_{PGI,E}$. Let $\Im$ denote the interval $[-2\sigma_{PGI,A}, 0]$ if $\sigma_{PGI,A}$ is positive, and the interval $[0, -2\sigma_{PGI,A}]$ otherwise. ϑ is negative in $\Im$'s interior (and reaches its minimum at $\sigma_{PGI,E} = -\sigma_{PGI,A}$), is equal to 0 at the interval border points, and is positive but remains small outside but close to $\Im$ (since ϑ is differentiable). Comparing ${R^o}^2_{PGI}$ with $\sigma_A^2 = [\beta^2 + 2\,\beta\sigma_{PGI,A'}] + [\sigma_{A'}^2]$, we see that ${R^o}^2_{PGI} \leq \sigma_A^2$ if $\sigma_{PGI,E}$ is within or sufficiently close to $\Im$, such that $\vartheta(\sigma_{PGI,E}) \leq (1 - {r_{PGI,A'}}^2)\sigma_{A'}^2$. In other words, if $\sigma_{PGI,E}$ is null or "small in magnitude" (Assumption 1), such that it falls in or near $\Im$, then ${R^o}^2_{PGI} \leq \sigma_A^2$. ■

Finally, we note that Proposition 1 likely holds when $\sigma_{PGI,E}$ is not so small. Recall that A' captures the part of A that is orthogonal to the PGI in the absence of assortative mating or population stratification. A' can be decomposed into two components: one component that captures the effects of variants that are not captured by the PGI (recall that the PGI is constructed using less than 500,000 SNPs), and a second component that is due to the fact that the variants used to construct the PGI are measured with noise. Assortative mating or population stratification may generate a correlation between the PGI and the first component of A', but not between the PGI and the second component. Because our PGIs certainly are very noisy—especially when predicting among the Korean adoptees—that second component of A' is likely large, and the correlation between the PGI and A' ($r_{PGI,A'}$) is unlikely to be large. Thus, even if $\sigma_{PGI,E}$ lies far outside of $\Im$ and $\vartheta(\sigma_{PGI,E})$ is thus positive, it is unlikely that it will be larger than $(1 - {r_{PGI,A'}}^2)\sigma_{A'}^2$. Thus, Proposition 1 holds even if $\sigma_{PGI,E}$ is not so small. And as discussed in the main text, $\sigma_{PGI,E}$ is likely small.

F. STATISTICAL POWER TO ESTIMATE A SIGNIFICANT GxE INTERACTION

F.1 Framework and derivations

With a sample of only 361 genotyped Korean adoptees, statistical power to detect a GxE interaction may be limited. To further evaluate this, we derived an expression to calculate statistical power analytically under simple assumptions, and we verified the results through simulations.

Consider the GxE model:

$$Y = \beta_0 + \beta_1 F + \beta_2 PGI + \beta_3 (F \times PGI) + ControlsTerms + \epsilon.$$

Assume that Y, F, and PGI are standard normal variables with mean 0 and variance 1, and that ϵ is also normally distributed. $ControlsTerms$ captures all the control terms in the regression, which may include the terms for the baseline controls as well as for their interactions with F and PGI. Let k denote the total number of covariates in the regression. Consistent with the quasi-random assignment of the adoptees to the families, we assume that F and PGI are independent. It follows that $\mathrm{Var}(F \times PGI) = E[F^2 PGI^2] - E[F]^2 E[PGI]^2 = E[F^2]E[PGI^2] = 1$; that $\mathrm{Cov}(F \times PGI, F) = E[F^2 PGI] - E[F \cdot PGI]E[F]$=0; and similarly that $\mathrm{Cov}(F \times PGI, PGI) = 0$. We further assume that the controls are independent from the other variables. It follows from all this that

$$\mathrm{Var}(Y) = {\beta_1}^2 + {\beta_2}^2 + {\beta_3}^2 + \sigma^2_{ControlTerms} + \sigma^2_\epsilon = 1.$$

Since $F \times PGI$ is orthogonal to both F and PGI, we can rewrite our model as

$$Y = \beta_0 + \beta_3 (F \times PGI) + \{\beta_1 F + \beta_2 PGI + ControlTerms + \epsilon\} = \beta_0 + \beta_3 (F \times PGI) + \xi,$$

where $\xi = \beta_1 F + \beta_2 PGI + ControlTerms + \epsilon$. For simplicity, we assume that ξ is normally distributed; since the regression includes a constant, we can also assume, without loss of generality, that ξ has mean 0. From our other assumptions, ξ has variance $\sigma^2_\xi = 1 - {\beta_3}^2$ and is orthogonal to $F \times PGI$. Further, the share of the variation in the regression accounted for by $F \times PGI$ is $R^2_3 = {\beta_3}^2$, so $\sigma^2_\xi = 1 - R^2_3$. Therefore, the problem boils down to computing the power of a simple linear regression with a normally distributed covariate and error term, albeit with a degrees-of-freedom adjustment to account for the covariates captured by ξ.

Note that the variance of the OLS estimator $\hat{\beta}_3$ is $\sigma^2_3 \equiv \mathrm{Var}(\widehat{\beta_3}) = \frac{\sigma^2_\xi}{[SST_3(1-R^2_{3,other})]}$, where SST_3 is the total sum of squares in $F \times PGI$ and $R^2_{3,other}$ is the R^2 of a regression of $F \times PGI$ on all the other covariates and a constant. Next, observe that $R^2_{3,other} = 1 - \frac{SSR_{3,other}}{SST_3}$, where $SSR_{3,other}$ is the residual sum of squares from the regression of $F \times PGI$ on all the other covariates and a constant. In turn, $SSR_{3,other} = (N-k) \cdot \hat{\sigma}^2_{u_{3,other}}$, where $N - k = N - (k-1) - 1$ is the numbers of degrees of

freedom in that regression, and $\hat{\sigma}^2_{u_{3,other}}$ is the unbiased estimator of the error variance in that regression. Since $F \times PGI$ is independent of all the other covariates, the true error $u_{3,other}$ in that regression is equal to $F \times PGI$, and so the true error variance is $\sigma^2_{u_{3,other}} \approx \text{Var}(F \times PGI) = 1$. It follows that $\sigma^2_{\widehat{\beta_3}} \approx \frac{\sigma^2_\xi}{[SST_3(1-R^2_{3,other})]} = \frac{\sigma^2_\xi}{\left[SST_3\left(1-\left(1-\frac{SSR_{3,other}}{SST_3}\right)\right)\right]} = \frac{\sigma^2_\xi}{SSR_{3,other}} = \frac{\sigma^2_\xi}{N-k} = \frac{1-{\beta_3}^2}{N-k} = \frac{1-R^2_3}{N-k}$.[3]

Power is given by

$$\text{Prob}\left(\left|\frac{\widehat{\beta_3}}{\hat{\sigma}_3}\right| > z_{\alpha/2}\right) \approx \text{Prob}\left(\left|\frac{\beta_3 + z\sigma_3}{\hat{\sigma}_3}\right| > z_{\alpha/2}\right) \approx \text{Prob}\left(\left|\frac{\beta_3}{\sigma_3} + z\right| > z_{\alpha/2}\right)$$

$$= \text{Prob}\left(\left|\sqrt{N-k} \cdot \beta_3/\sigma_\xi + z\right| > z_{\alpha/2}\right)$$

$$= \text{Prob}\left(\sqrt{N-k} \cdot \beta_3/\sigma_\xi + z > z_{\alpha/2}\right) + \text{Prob}\left(\sqrt{N-k} \cdot \beta_3/\sigma_\xi + z < -z_{\alpha/2}\right)$$

$$= \left\{\Phi\left(\sqrt{N-k} \cdot \beta_3/\sigma_\xi - z_{\alpha/2}\right)\right\} + \left\{1 - \Phi\left(\sqrt{N-k} \cdot \beta_3/\sigma_\xi + z_{\alpha/2}\right)\right\}$$

$$= \Phi\left(\sqrt{(N-k) \cdot R^2_3/(1-R^2_3)} - z_{\alpha/2}\right) + 1 - \Phi\left(\sqrt{(N-k) \cdot R^2_3/(1-R^2_3)} + z_{\alpha/2}\right),$$

where $\Phi(\cdot)$ is the cumulative distribution function of a standard normal variable; $z_{\alpha/2}$ is the critical value at the α level of significance; the sampling variation in $\widehat{\beta_3}$ is approximately equal to $z\sigma_3$, where $z \sim N(0,1)$, in sufficiently large samples by the Central Limit Theorem; and where we use the approximation $\sigma_3 \approx \hat{\sigma}_3$.

The above derivations ignore the family structure of the data, assuming no intrafamily correlation among the variables and no clustering of the errors. Moulton derived a formula, known as the Moulton factor, that indicates by how much the conventional OLS variance formula understates the true variance of an OLS estimator when there is intraclass correlation (Angrist & Pischke 2009; Moulton 1986). In the case of a bivariate regression of Y on $F \times PGI$ (i.e., if we ignore the other covariates), the formula is

[3] Observe that the variances of the OLS estimators $\widehat{\beta_1}$ and $\widehat{\beta_2}$ are similarly given by $\sigma_1 \approx (1-{\beta_1}^2)/(N-k)$ and $\sigma_2 \approx (1-{\beta_2}^2)/(N-k)$, so the variance of $\widehat{\beta_3}$ will be similar to that of $\widehat{\beta_1}$ and $\widehat{\beta_2}$ if ${\beta_3}^2$ is similar to ${\beta_1}^2$ and ${\beta_2}^2$. If that is the case, the power to estimate the main effects β_1 and β_2 will be similar to the power to estimate the coefficient on the interaction. By contrast, in a blog post, statistician Andrew Gelman has shown that under some basic assumptions, a much larger sample is needed to have sufficient statistical power to estimate an interaction rather than to estimate a main effect (Gelman 2018). The discrepancy arises because Gelman assumed that the interaction was only half the size of the main effects and because in his framework the standard errors of the interaction were roughly twice as large as those of the main effects.

$$\frac{\sigma^2_{3,correct}}{\sigma^2_{3,conventional}} = 1 + \left[\frac{\mathrm{Var}(n_g)}{\bar{n}} + \bar{n} - 1\right]\rho_x\rho,$$

where $\sigma^2_{3,correct}$ and $\sigma^2_{3,conventional}$ are the correct and conventional variances of $\widehat{\beta_3}$. In the current setting, $\mathrm{Var}(n_g)$ is the variance in family size and $\bar{n}$ is the average number of adoptees per family (here, families have either one or two adoptees); ρ_x is the intra-class (i.e., intra-family) correlation of $F \times PGI$; and ρ is the intra-class correlation of the error term ϵ. (The Moulton factor it the square root of the ratio $\sigma^2_{3,correct}/\sigma^2_{3,conventional}$.)

Here, because of the quasi-random assignment of the Korean adoptees to their adoptive families, adoptive siblings in the same family are unrelated. As a result, their PGIs are uncorrelated and so are their PGIs interacted with their family variable F. Thus, the intra-class correlation of $F \times PGI$ is zero, and the Moulton factor is unity, implying that the conventional OLS variance formula is accurate.

F.2 Calculations

In our data, when the dependent variable is cognitive performance, there are $N = 361$ genotyped Korean adoptees with nonmissing data. In the Model II specification, there are 3 covariates of interest (F, PGI, and $F \times PGI$), and the 15 baseline controls together with their interactions with family SES (F) and the PGI use 45 control terms, so $k = 3 + 3 \cdot 15 = 48$ and $N - k = 313$. We regressed cognitive performance on family SES to obtain the estimate $\widehat{R_1^2}$ =0.022 (equal to the R^2 of the regression) and, separately, on the PGI of cognitive performance or EA, which yielded the estimates $R^2_{2\,(PGS\ of\ CP)}$ =0.056 and $R^2_{2\,(PGS\ of\ EA)}$ =0.053, respectively.[4]

Plugging in $N - k = 313$, R_1^2 =0.02, R_2^2 =0.05, and R_3^2 =0.01 (which assumes that the R^2 of the GxE interaction is 20% as large as the R^2 of the PGI and 50% as large as that of family SES) in the above power formula and using the $\alpha = 0.05$ level of significance, we obtain an estimate of 43% for the power to obtain a significant estimate of β_3. If we instead assume that R_3^2 =0.005, then we estimate that power is 24%, and if we assume that R_3^2 =0.025, then power is 83%.

F.3 Simulations

To verify these calculations, we conducted simulations. The simulations included 361 observations and 15 control variables that are independent from one another and from other variables,

[4] The corresponding estimates for the R^2 of $F \times PGI$ are $R^2_{2\,(F\times PGS\ of\ CP)} = 0.0011$ and $R^2_{2\,(F\times PGS\ of\ EA)} = 0.0077$, but these are in-sample estimates that relate directly to β_3, the parameter for which we wish to evaluated statistical power.

as well as the interactions of the control variables with F and PGI. We modeled the family structure of the data, with 123 families of two adoptees and 115 singletons, and assumed intra-class correlation coefficients of 0, 1, and 0.3 for PGI, F, and ϵ, respectively.[5] As in our actual analyses, we clustered the errors at the family level. The Stata code for the simulations is included below in this Appendix.

Assuming R_1^2 =0.02, R_2^2 =0.05, and R_3^2 =0.01 and using the $\alpha = 0.05$ level of significance, we obtain a power estimate of 46%; if we instead assume that R_3^2 =0.005 and then R_3^2 =0.025, then power is 27% and 83%, respectively. These power estimates from our simulations are strikingly similar to those obtained above with our analytical formula.

F.4 Conclusion

In sum, statistical power to estimate a significant GxE effect depends on the true R^2 of the GxE interaction. Under optimistic assumptions about that true R^2 (e.g., $R^2_{F\times PGI} \geq 0.025$), power is adequate ($> 80\%$); however, under more conservative assumptions (e.g., $R^2_{F\times PGI} \leq 0.01$), power is limited. Thus, our finding of a significant GxE interaction between the PGI of EA and family SES on cognitive performance should be taken as no more than tentative until it is replicated (or not) in a larger, independent sample.

[5] Based on our above discussion of the Moulton factor formula, the assumed intra-class correlations for F and ϵ should not affect power, given the assumed intra-class correlation of 0 for PGS; simulations confirmed that this is indeed the case.

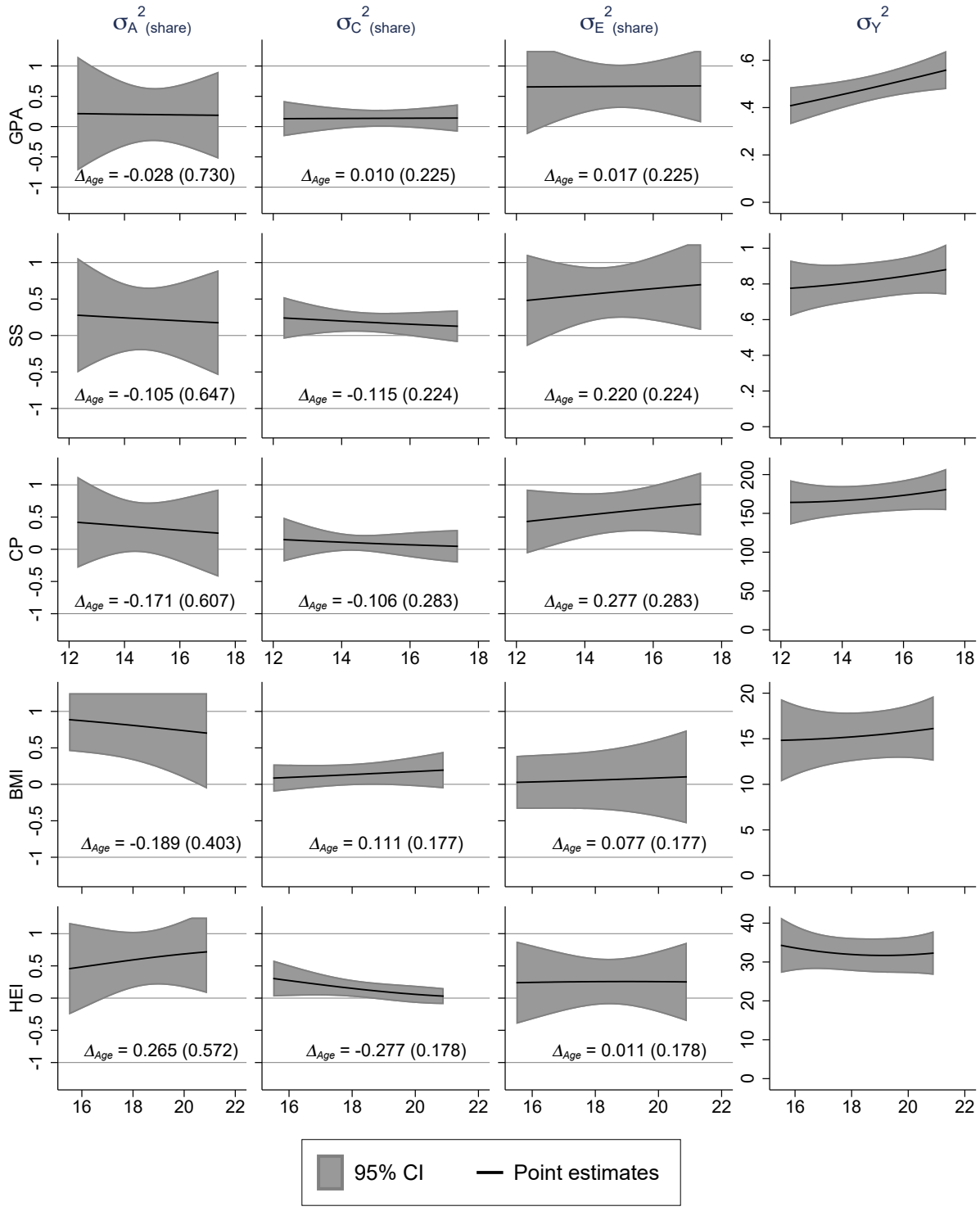
F.5 Stata code for the simulations

```
clear all
set maxvar 12000
set matsize 11000
cap log close
set more off


*****************************************************************
*****************************************************************

* 1. SIMULATING THE GxE data
* The model is y = B1*G + B2*F + B3*(G*F)+eps
* All variables are normally distributed with mean 0 and variance 1
* (except eps, which is scaled so that var(y)=1)
* In the regression, we control for controls that only capture noise, to account
* for the degrees of freedom these take

***************
* => USER INPUT NEEDED HERE:
local R2_G=0.05
local R2_F=0.02
local R2_GxF=0.005
***************

* The assumed intraclas (intrafamily) correlations for G, F, and eps;
*    (note: since G_intrafam_corr=0 for the Korean adoptees, the Moulton factor
*     is 1, so Eps_intrafam_corr does not matter)
local G_intrafam_corr=0
local F_intrafam_corr=1
local Eps_intrafam_corr=0.3

local Nobs=361
local Ncontrols=15

* For the clusters (in the analysis sample, there are 123 pairs of adoptees
* that share a FAMID (=246 adoptees) and 115 adoptees each with their own FAMID)
local N_FAMID_pair=123

local Nsim=10000

forval sim =1/`Nsim' {

    clear

    qui set obs `Nobs'

    local B1=sqrt(`R2_G')
    local B2=sqrt(`R2_F')
    local B3=sqrt(`R2_GxF')

    * Note: for simplicity, for this simulation, we assume that the controls
    *  only apture noise, so the following holds:
    local var_eps=1-`R2_G'-`R2_F'-`R2_GxF'
```

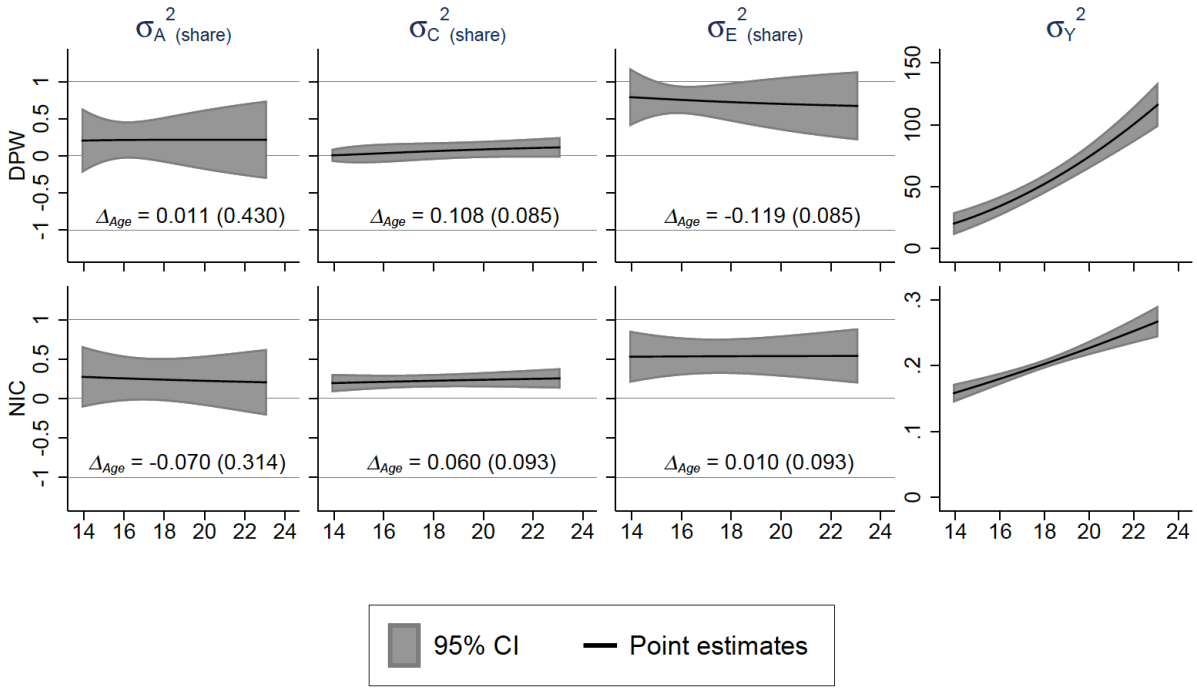
```
    *****
    * Generate the FAMID's
    qui gen FAMID=round(_n/2)     if _n<=2*`N_FAMID_pair'
    qui replace FAMID=_n-`N_FAMID_pair' if _n>2*`N_FAMID_pair'

    *****
    * Generate the G variable with the WF structure:
    qui gen G_fam = rnormal(0,sqrt(`G_intrafam_corr')) ///
      if FAMID[_n]!=FAMID[_n-1]
    qui replace G_fam = G_fam[_n-1]     if FAMID[_n]==FAMID[_n-1]
    gen G = G_fam + rnormal(0,sqrt(1-`G_intrafam_corr'))

    * Generate the F variable with the WF structure:
    qui gen F_fam = rnormal(0,sqrt(`F_intrafam_corr')) ///
      if FAMID[_n]!=FAMID[_n-1]
    qui replace F_fam = F_fam[_n-1]     if FAMID[_n]==FAMID[_n-1]
    gen F = F_fam + rnormal(0,sqrt(1-`F_intrafam_corr'))

    * Generate the GxF variable:
    gen GxF = G*F

    * Generate the error, with the WF structure:
    qui gen eps_fam = rnormal(0,sqrt(`Eps_intrafam_corr'*`var_eps')) ///
      if FAMID[_n]!=FAMID[_n-1]
    qui replace eps_fam = eps_fam[_n-1] if FAMID[_n]==FAMID[_n-1]
    gen eps = eps_fam + ////
      rnormal(0,sqrt((1-`Eps_intrafam_corr')*`var_eps'))

    *****
    * Generate the control variables:
    forval k=1/`Ncontrols' {
        gen control`k' =rnormal(0,1)
        gen control`k'_X_G =rnormal(0,1)
        gen control`k'_X_F =rnormal(0,1)
    }

    *****
    gen y = `B1'*G + `B2'*F + `B3'*GxF + eps
    qui reg y G F GxF control*, cluster(FAMID)

    local t = _b[GxF]/_se[GxF]
    local pvalue = 2*ttail(e(df_r),abs(`t'))

    mat sim_res[`sim',1]=`pvalue'
    mat sim_res[`sim',2]=(`pvalue'<0.05)

    if `pvalue'<`alpha' {
        local count_signif_simlns=`count_signif_simlns'+1
        }

    * At every 50 iterations, display where we're at in the the for loop
    if mod(`sim',50)==0 {
        display "`sim'"
        }
}
```

```
local power_in_percent=`count_signif_simlns'/`Nsim'*100
display "Power = `power_in_percent'%"



************************************************************
************************************************************

* 2. ANALYTICAL POWER FORMULA

***************
* => USER INPUT NEEDED HERE:
local R2_G=0.05
local R2_F=0.02
local R2_GxF=0.025
local NminusK=313
local alpha=0.05
***************

scalar power_analytical = ///
  normal(sqrt(`NminusK'*`R2_GxF'/(1-`R2_GxF')) ///
  -invnormal(1-`alpha'/2)) + 1  ///
  - normal(sqrt(`NminusK'*`R2_GxF'/(1-`R2_GxF')) ///
  + invnormal(1-`alpha'/2))

display power_analytical


******************************************************
******************************************************
```

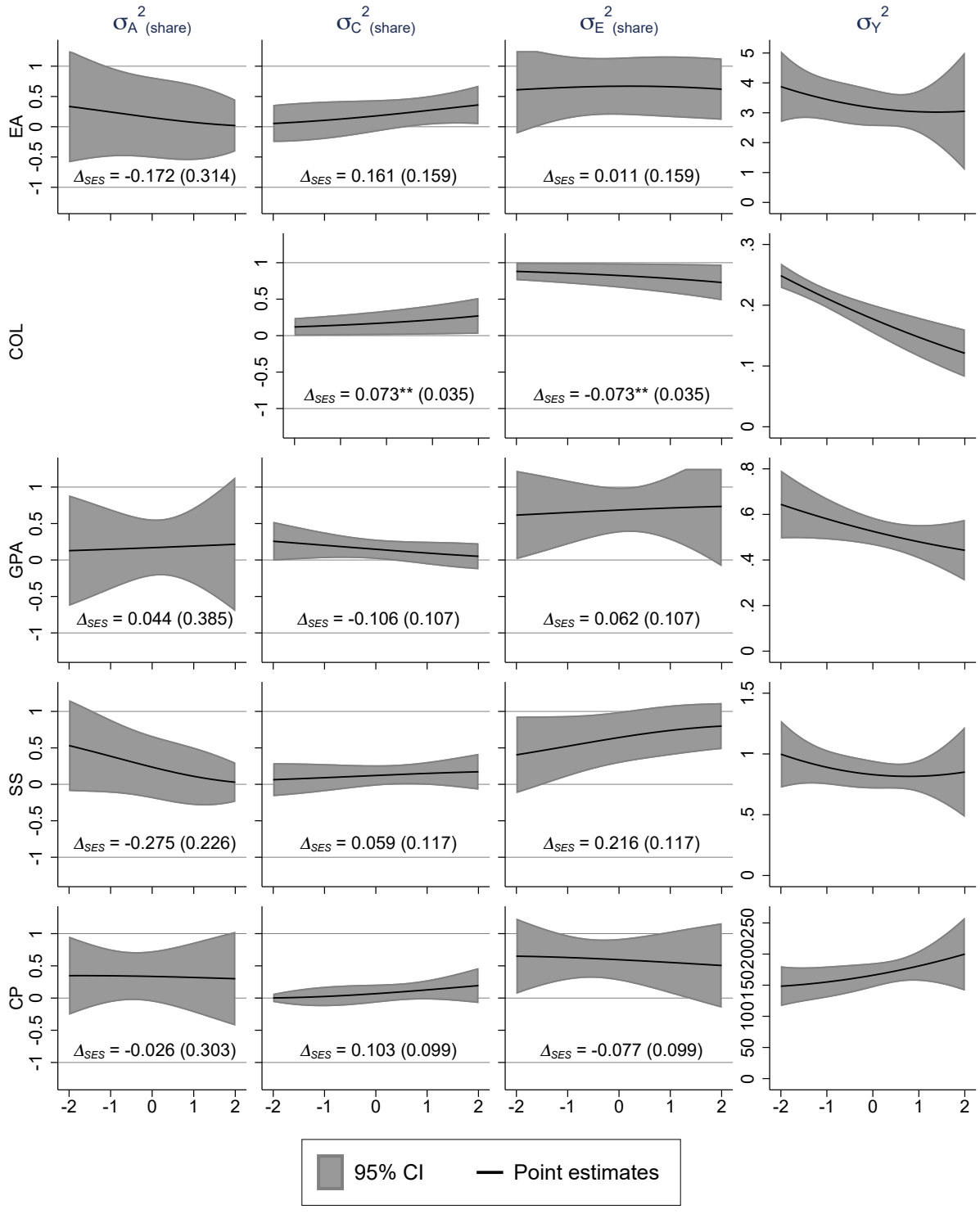
G. ADDITIONAL FIGURES AND TABLES



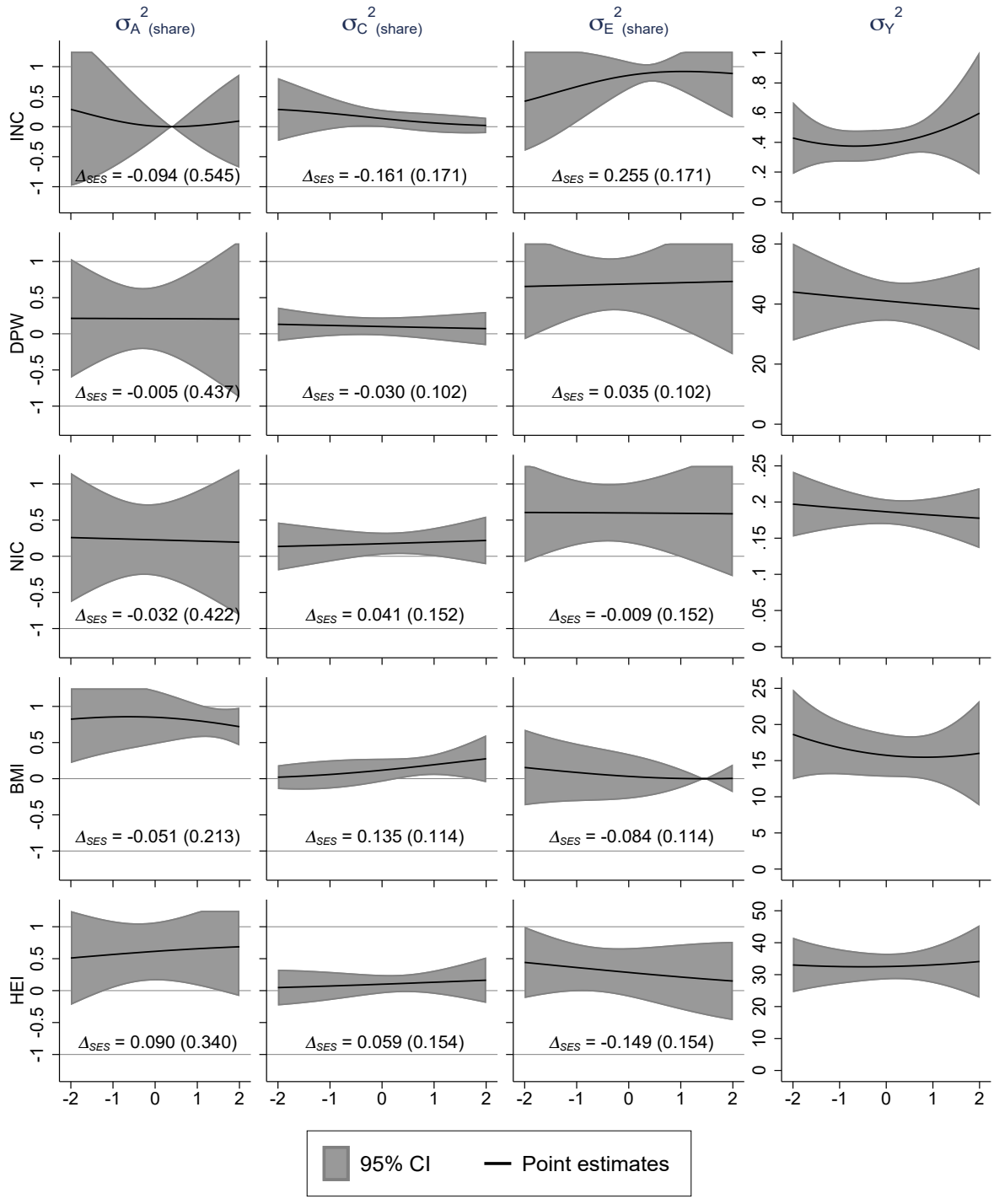
Appendix Figure G.1. *(Continues)*



Appendix Figure G.1. Variance decomposition estimates from the extended ACE model that allows for moderating influences of age at trait measurement. Each subfigure shows, for each outcome (in each row), the share of the residualized-outcome variance that is attributable to additive genetic factors ($\sigma^2_{A\,(share)}$, in Column 1), common environmental factors ($\sigma^2_{C\,(share)}$, in Column 2), and individual environmental factors ($\sigma^2_{C\,(share)}$, in Column 3), as well as the residualized-outcome variance (σ^2_Y, in Column 4), as functions of the age at which the trait was measured (on the *x*-axis). For the outcomes DPW and NIC, which were measured at three different waves, each pair of measurements (for each sib pair) from each wave was treated as a separate observation; these observations were treated as a panel and we clustered standard errors at the sib-pair level (all other outcomes were measured only once). In Columns 1-3, the subfigures' vertical axes are truncated at +/- 1.25. No results are shown for the outcomes EA, COL, and INC, because these were measured at the third follow-up wave and their age a measurement is highly collinear with birth year; further, EA and COL are not measurement-age-dependent. Δ_{age} is a metric that indicates the predicted change in each variance share as one moves from 10th to the 90th percentile of the distribution of age at measurement for the outcome. Metric standard errors are in parentheses. See Appendix D for additional details.
* p<0.1 ** p<0.05 *** p<0.01



Appendix Figure G.2. *(Continues)*



Appendix Figure G.2. Variance decomposition estimates from the extended ACE model that allows for moderating influences of (adoptive) family SES. Each subfigure shows, for each outcome (in each row), the share of the residualized-outcome variance that is attributable to additive genetic factors ($\sigma^2_{A\,(share)}$, in Column 1), common environmental factors ($\sigma^2_{C\,(share)}$, in Column 2), and individual environmental factors ($\sigma^2_{C\,(share)}$, in Column 3), as well as the residualized-outcome variance (σ^2_Y, in Column 4), as functions of family SES (on the *x*-axis). For the outcome COL, convergence could not be achieved for the extended ACE model; results are shown instead for the extended CE model (i.e., without the additive genetic factor). For the outcome NIC, which was measured at three different waves, convergence could not be

achieved when controlling for all three ages at measurement (which are highly multicollinear); results are shown instead controlling for age at the first and third (but not second) follow-ups. In Columns 1-3, the subfigures' vertical axes are truncated at +/- 1.25. Δ_{SES} is a metric that indicates the predicted change in each variance share as one moves from a family SES of -1 to a family SES of 1. Metric standard errors are in parentheses. See Appendix D for additional details.
* p<0.1 ** p<0.05 *** p<0.01

Appendix Table G.1: Tests of random placement of the European ancestry adoptees

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Male	Placement age	PGI of EA	PGI of cognitive performance	PGI of income	PGI of ever smoker	PGI of BMI	PGI of height
Baseline family variables								
Mother's EA	0.032	0.428	-0.168	-0.166	-0.065	-0.102	0.190*	0.012
	(0.036)	(0.332)	(0.112)	(0.106)	(0.108)	(0.095)	(0.108)	(0.098)
Mother's CP	0.003	-0.030	-0.013	-0.023*	-0.015	0.015	-0.018**	-0.008
	(0.004)	(0.032)	(0.015)	(0.013)	(0.013)	(0.012)	(0.009)	(0.014)
Mother's DPW	-0.029***	-0.084	-0.042	0.003	-0.029	-0.023	0.009	0.014
	(0.009)	(0.092)	(0.037)	(0.050)	(0.032)	(0.032)	(0.034)	(0.030)
Mother ever used nicotine	-0.164*	-0.912	0.124	0.082	0.456	0.066	-0.043	0.490
	(0.098)	(1.058)	(0.371)	(0.354)	(0.352)	(0.277)	(0.266)	(0.295)
Mother's BMI	-0.038***	0.078	-0.010	0.057**	-0.000	-0.024	0.020	0.040
	(0.011)	(0.092)	(0.031)	(0.028)	(0.028)	(0.031)	(0.025)	(0.029)
Mother's height	0.024***	0.031	-0.025**	-0.025*	-0.017	0.011	-0.003	0.027**
	(0.006)	(0.035)	(0.011)	(0.014)	(0.013)	(0.012)	(0.017)	(0.013)
Mother's age when child was born	-0.010	-0.099	0.021	0.007	0.040	-0.050	-0.057	0.039
	(0.014)	(0.120)	(0.040)	(0.038)	(0.043)	(0.045)	(0.037)	(0.036)
Father's EA	0.068*	-0.154	0.115	0.156	0.205*	-0.152*	-0.043	0.048
	(0.041)	(0.303)	(0.103)	(0.115)	(0.112)	(0.088)	(0.100)	(0.100)
Father's age when child was born	-0.019	-0.047	-0.023	0.030	-0.070	0.029	0.018	-0.061
	(0.014)	(0.098)	(0.042)	(0.046)	(0.044)	(0.051)	(0.049)	(0.047)
Family SES	-0.201*	0.181	0.015	0.046	-0.382	0.281	-0.079	-0.085
	(0.115)	(0.759)	(0.266)	(0.295)	(0.308)	(0.274)	(0.291)	(0.261)
Log family income	0.246*	-0.892	-0.486	-0.630*	0.116	0.140	0.510	-0.310
	(0.147)	(1.185)	(0.392)	(0.355)	(0.365)	(0.330)	(0.338)	(0.339)
Parent disinhibition score	0.073*	0.278	-0.009	0.068	0.044	0.103	0.232*	0.058
	(0.040)	(0.285)	(0.137)	(0.130)	(0.134)	(0.121)	(0.130)	(0.146)
Number of siblings in rearing family	-0.000	0.446	-0.036	-0.082	0.021	-0.085	-0.057	-0.170
	(0.046)	(0.398)	(0.139)	(0.128)	(0.140)	(0.111)	(0.131)	(0.119)
Mixed biological & adoptive family	0.048	-1.534**	0.106	0.192	0.018	-0.315	0.409	0.188
	(0.101)	(0.615)	(0.280)	(0.285)	(0.308)	(0.302)	(0.283)	(0.277)
Family lives in a city or suburbs	-0.274***	-0.384	0.198	0.430*	0.202	0.076	-0.432*	0.490**
	(0.087)	(0.589)	(0.251)	(0.236)	(0.235)	(0.238)	(0.224)	(0.205)
Parents still married	-0.281*	2.456*	-0.131	-0.399	0.068	-1.334***	-0.373	-0.759
	(0.170)	(1.324)	(0.923)	(0.530)	(0.784)	(0.456)	(0.725)	(0.520)
Observations	127	127	102	102	112	112	112	112
R^2	0.397	0.181	0.242	0.294	0.207	0.155	0.211	0.273
Test statistic, joint signif. of family var.	43.84	0.669	2.906	3.662	2.210	1.992	1.604	3.706
P value	0.002	0.851	<0.001	<0.001	0.006	0.016	0.070	<0.001

Note: This table mirrors Table 2 but reports analyses in the sample of European ancestry adoptees (instead of the sample of Korean adoptees). All regressions control for adoptee birth year and its square. To maximize regression sample size, missing observations were coded as 0 and dummies indicating missing observations were included for five baseline family variables with high numbers of missing observations (mother's cognitive performance, mother's BMI, mother's height, father's age when child was born, log family income). The family variables for the tests of joint significance include the baseline family variables as well as these five dummies. For the continuous outcomes, OLS regressions were estimated and the test statistic for joint significance is the *F* statistic. For the binary outcome (male), a logistic regression was estimated, the reported coefficients are average marginal effects, Nagelkerke's pseudo R^2 was used, and the test statistic for joint significance is the Wald statistic. Robust standard errors clustered at the family level are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Appendix Table G.2: Sibling correlations in outcomes among adoptive-adoptive, adoptive-biological, and biological-biological pairs and resulting variance decomposition estimates from the extended ACE model

	Panel A: Sibling correlations among adoptive-adoptive, adoptive-biological, and biological-biological pairs						Panel B: Estimated proportion of outcome variance explained by genetics (σ_A^2), common family env. (σ_C^2), gene-environment correlation (σ_{AC}), and unexplained factors (σ_E^2)			
	Adoptive-adoptive sib correlation	*N (pairs)*	Adoptive-biological sib correlation	*N (pairs)*	Biological-biological sib correlation	*N (pairs)*	σ_A^2	σ_C^2	σ_E^2	γ
EA	0.226**	76	0.346*	27	0.358***	89	0.381*	0.292***	0.326*	-0.131
							(0.248)	(0.093)	(0.209)	(0.058)
College	0.242**	77	0.378*	27	0.290**	89	-0.128	0.261***	0.867***	0.052
							(0.347)	(0.109)	(0.308)	(0.073)
GPA	0.127*	172	0.086	66	0.303***	176	0.389**	0.135**	0.476***	-0.026
							(0.223)	(0.063)	(0.201)	(0.054)
Soft skills	0.175**	175	0.055	72	0.280***	181	0.350**	0.148***	0.502***	-0.034
							(0.208)	(0.063)	(0.190)	(0.054)
Cognitive perf.	0.006	175	0.222*	71	0.303***	181	0.527***	0.032	0.441**	0.014
							(0.212)	(0.068)	(0.196)	(0.069)
Log income	0.264**	63	0.175	22	0.115	78	-0.071	0.233***	0.839***	-0.050
							(0.371)	(0.082)	(0.359)	(0.089)
Drinks per week	0.140*	150	0.098	62	0.188**	155	0.157	0.113*	0.731***	0.001
							(0.283)	(0.071)	(0.289)	(0.078)
Ever used nicotine	0.137	119	0.425***	50	0.310**	133	-	-	-	-
BMI	0.169*	134	0.143	54	0.490***	150	0.411*	0.133*	0.456**	0.136
							(0.254)	(0.092)	(0.246)	(0.115)
Height	0.209**	134	-0.070	54	0.411***	150	0.636***	0.093*	0.271*	-0.007
							(0.206)	(0.069)	(0.184)	(0.058)

Note: Adoptive-adoptive sibling correlations were computed among sibling pairs comprising at least one Korean adoptee (as well as another Korean or a European ancestry adoptee); adoptive-biological sibling correlations were computed among sibling pairs comprising one Korean adoptee and one European ancestry biological children; and biological-biological sibling correlations were computed among sibling pairs comprising two European ancestry biological children. In Panel A, correlations were estimated after partialling out the effects of a vector *X* that includes the baseline control variables, dummies indicating European vs. Korean ancestry and adoptee vs. biological child status, and an intercept. In Panel B, GMM was used to estimate the extended ACE model parameters (σ_A^2, σ_C^2, σ_E^2, and γ), as described in Appendix D. For the outcomes EA, College, and Income, which were measured at the third follow-up, convergence could not be achieved when controlling for age at the third follow-up (which is highly collinear with birth year); results are shown instead without controlling for age at the third follow-up. For the outcome Drinks per week, which was measured at three different waves, convergence could not be achieved when controlling for all three ages at measurement (which are highly multicollinear); results are shown instead controlling for age at the first and third (but not second) follow-ups. Estimates for ever used nicotine are omitted due to convergence issues. We do not constrain estimates of variance shares to be nonnegative (e.g., for σ_A^2 for EA and log income). GMM standard errors are in parentheses. Since we are working with variances, *P* values for the variance shares were computed against a one-sided alternative.
*** p<0.01, ** p<0.05, * p<0.1

Appendix Table G.3: Regressions of White non-adoptee outcomes on family environmental variables and non-adoptee PGSs

	(1) EA	(2) College	(3) GPA	(4) Soft skills	(5) Cognitive performance	(6) Log income	(7) Drinks per week	(8) Ever used nicotine	(9) BMI	(10) Height
$\Delta\bar{R}^2$, family variables	0.148***	0.155***	0.111***	0.116***	0.096***	0.035	0.036	0.009	0.134***	0.088***
Joint significance (p)	<0.001	<0.001	<0.001	<0.001	<0.001	0.416	0.110	0.183	<0.001	<0.001
$\Delta\bar{R}^2$, adoptee PGSs	0.073***	0.070***	0.090***	0.075***	0.093***	0.003**	0.022***	-0.002**	0.091***	0.123***
Joint significance (p)	<0.001	<0.001	<0.001	<0.001	<0.001	0.018	0.002	0.039	<0.001	<0.001
Observations	273	273	380	393	391	253	393	393	339	339
$\bar{R}^2$, all variables	0.181	0.134	0.177	0.273	0.202	0.100	0.151	0.076	0.299	0.659

Note: Note: This table mirrors Table 4 but reports analyses in the sample of European ancestry biological children (instead of the sample of Korean adoptees). All regressions include the baseline control variables. To maximize regression sample size, missing observations were coded as 0 and dummies indicating missing observations were included for five family variables with high numbers of missing observations (mother's cognitive performance, mother's BMI, mother's height, father's age when child was born, log family income). The family variables include the baseline family variables as well as these five dummies. The adoptee PGIs include the PGIs of EA, cognitive performance, income, ever smoker, BMI, and height. For the continuous outcomes, OLS regressions were estimated, the adjusted R^2 was used, and the test for joint significance is the F test. For the binary outcomes (college and ever used nicotine), logistic regressions were estimated, McFadden's adjusted pseudo R^2 was used, and the test for joint significance is the Wald test. The incremental adjusted R^2 ($\Delta\bar{R}^2$) of each block of variables is the difference between the adjusted R^2 of the regression of the outcome on the controls and the variables in the block, and that of the same regression (in the same sample) but on the controls only. The stars on the $\Delta\bar{R}^2$'s indicate the significance level of the associated test for joint significance.

*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table G.4: Single variable regressions of Korean adoptee outcomes on baseline family variables and adoptee PGIs

	(1) EA	(2) College	(3) GPA	(4) Soft skills	(5) Cognitive performance	(6) Log income	(7) Drinks per week	(8) Ever used nicotine	(9) BMI	(10) Height
Baseline family variables										
Mother's EA	0.225***	0.021	-0.032	-0.039	-0.210	0.007	0.175	-0.004	0.025	0.075
	(0.076)	(0.015)	(0.023)	(0.031)	(0.394)	(0.028)	(0.182)	(0.013)	(0.165)	(0.195)
Mother's cognitive performance	0.017	0.003	-0.001	-0.010**	0.018	-0.008**	0.000	-0.002	-0.022	-0.008
	(0.011)	(0.002)	(0.003)	(0.004)	(0.052)	(0.003)	(0.028)	(0.002)	(0.015)	(0.028)
Mother's drinks per week	0.019	0.004	-0.001	-0.003	0.044	-0.004	0.072	-0.004	-0.106**	0.083
	(0.033)	(0.007)	(0.010)	(0.012)	(0.164)	(0.011)	(0.084)	(0.005)	(0.049)	(0.057)
Mother ever used nicotine	0.259	0.055	-0.027	-0.112	-0.097	0.039	1.686**	0.033	0.250	-0.104
	(0.353)	(0.069)	(0.095)	(0.133)	(1.652)	(0.128)	(0.797)	(0.053)	(0.613)	(0.766)
Mother's BMI	-0.028	-0.003	-0.003	-0.015*	0.049	-0.002	-0.036	0.003	-0.001	-0.060
	(0.021)	(0.005)	(0.006)	(0.008)	(0.099)	(0.007)	(0.059)	(0.004)	(0.030)	(0.050)
Mother's height	0.042*	0.005	-0.005	-0.021**	-0.001	0.017*	0.041	0.003	0.047	0.024
	(0.024)	(0.005)	(0.008)	(0.009)	(0.123)	(0.010)	(0.053)	(0.004)	(0.037)	(0.053)
Mother's age when child was born	0.054	0.004	-0.004	-0.016	0.200	-0.011	0.072	-0.004	-0.104	-0.046
	(0.042)	(0.009)	(0.012)	(0.016)	(0.204)	(0.014)	(0.097)	(0.007)	(0.071)	(0.100)
Father's EA	0.225***	0.026	0.006	-0.006	0.869**	0.020	0.427**	0.009	-0.070	0.156
	(0.084)	(0.016)	(0.024)	(0.029)	(0.421)	(0.030)	(0.195)	(0.014)	(0.125)	(0.194)
Father's age when child was born	0.032	0.003	-0.008	-0.016	0.045	0.014	0.028	-0.002	0.033	0.052
	(0.038)	(0.008)	(0.012)	(0.016)	(0.198)	(0.015)	(0.102)	(0.006)	(0.063)	(0.093)
Family SES	0.660***	0.095***	-0.006	-0.008	1.147	0.099*	1.149***	0.000	0.102	0.328
	(0.145)	(0.028)	(0.048)	(0.054)	(0.756)	(0.053)	(0.370)	(0.023)	(0.239)	(0.343)
Log family income	1.105***	0.202***	0.015	0.125	2.485	0.286***	2.231***	0.028	0.316	0.836
	(0.306)	(0.063)	(0.091)	(0.112)	(1.665)	(0.089)	(0.690)	(0.045)	(0.446)	(0.615)
Parent disinhibition score	-0.124	-0.014	-0.093*	0.030	-2.613***	0.040	0.290	0.022	0.131	0.820
	(0.205)	(0.036)	(0.051)	(0.061)	(0.896)	(0.051)	(0.524)	(0.039)	(0.278)	(0.535)
Number of siblings in the rearing family	-0.185	-0.056***	-0.013	-0.001	-1.343**	-0.056*	-0.234	0.016	0.068	-0.143
	(0.140)	(0.021)	(0.035)	(0.048)	(0.605)	(0.029)	(0.300)	(0.022)	(0.199)	(0.303)
Mixed biological & adoptive family	0.572	0.057	0.032	-0.016	0.791	-0.002	-0.729	-0.008	-0.871	2.141**
	(0.429)	(0.078)	(0.119)	(0.129)	(2.056)	(0.133)	(0.952)	(0.065)	(0.532)	(0.830)

continues

Appendix Table G.4 (continued): Single variable regressions of Korean adoptee outcomes on family environmental variables and adoptee PGIs

	(1) EA	(2) College	(3) GPA	(4) Soft skills	(5) Cognitive performance	(6) Log income	(7) Drinks per week	(8) Ever used nicotine	(9) BMI	(10) Height
Family lives in a city or suburb	0.577	0.130**	0.017	0.002	0.655	0.142	0.663	-0.062	-0.044	0.653
	(0.389)	(0.065)	(0.097)	(0.120)	(1.660)	(0.115)	(0.796)	(0.054)	(0.521)	(0.797)
Parents still married at intake	0.001	0.082	0.234	0.391*	4.501*	0.016	-0.731	-0.074	-1.157	-1.310
	(0.487)	(0.094)	(0.178)	(0.215)	(2.353)	(0.136)	(1.185)	(0.102)	(1.170)	(1.187)
Genetic Variables										
PGI of EA	0.490***	0.071***	0.174***	0.131***	3.116***	0.070	-0.689**	-0.017	-0.368	0.391
	(0.128)	(0.026)	(0.042)	(0.045)	(0.661)	(0.043)	(0.321)	(0.022)	(0.264)	(0.323)
PGI of cognitive performance	0.541***	0.084***	0.169***	0.124**	3.120***	0.060	-0.645*	-0.024	-0.295	0.545*
	(0.136)	(0.027)	(0.041)	(0.049)	(0.654)	(0.048)	(0.350)	(0.021)	(0.244)	(0.310)
PGI of income	0.504***	0.068***	0.176***	0.129***	2.922***	0.080*	-0.616*	-0.005	-0.540**	0.321
	(0.125)	(0.025)	(0.039)	(0.044)	(0.671)	(0.043)	(0.324)	(0.022)	(0.250)	(0.341)
PGI of ever smoker	-0.086	-0.042	-0.059	-0.081	0.086	-0.005	0.352	0.054**	0.316	-0.005
	(0.137)	(0.025)	(0.041)	(0.054)	(0.714)	(0.048)	(0.305)	(0.024)	(0.243)	(0.329)
PGI of BMI	-0.104	-0.026	-0.117***	-0.089*	-1.111	-0.005	0.026	0.021	0.593**	0.250
	(0.144)	(0.026)	(0.036)	(0.047)	(0.717)	(0.047)	(0.336)	(0.021)	(0.237)	(0.289)
PGI of height	0.199	0.035	0.085**	0.061	-0.273	-0.010	0.120	0.018	0.120	2.022***
	(0.145)	(0.027)	(0.041)	(0.049)	(0.699)	(0.047)	(0.361)	(0.023)	(0.235)	(0.305)

Note: For each outcome, the table reports estimates from separate regressions of the outcome on each family environmental variable and each PGI. All regressions include the baseline control variables (including the 10 top PCs of the ancestry-specific SNP data). For the continuous outcomes, OLS regressions were estimated. For the binary outcomes (college and ever used nicotine), logistic regressions were estimated, and the reported coefficients are average marginal effects. Due to varying numbers of missing observations, sample sizes vary between 164 and 361 across all the regressions. Robust standard errors clustered at the family level are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table G.5: Treatment effects of family type and PGI tercile for the European ancestry biological children

	Panel A: Effect of family type				Panel B: Effect of PGI tercile				
	Type 1	Type 2	*N*	R^2	PGI	Tercile 3	Tercile 2	*N*	R^2
EA	1.241***	0.903***	310	0.098	EA	1.063***	0.426	282	0.117
	(0.294)	(0.261)				(0.285)	(0.282)		
College	0.338***	0.158***	310	0.142	EA	0.312***	0.089*	282	0.190
	(0.0750)	(0.0512)				(0.066)	(0.054)		
GPA	0.351***	0.160*	455	0.061	EA	0.357***	0.231**	394	0.098
	(0.0898)	(0.0927)				(0.0938)	(0.097)		
Soft skills	0.541***	0.211*	470	0.127	EA	0.467***	0.358***	408	0.180
	(0.116)	(0.118)				(0.109)	(0.112)		
Cognitive performance	6.660***	3.114**	469	0.097	Cog. perf.	7.551***	3.525**	407	0.159
	(1.727)	(1.447)				(1.625)	(1.439)		
Log income	0.0514	0.112	286	0.078	Income	0.138	0.202**	261	0.137
	(0.101)	(0.0948)				(0.0954)	(0.096)		
Drinks per week	-0.456	-0.611	471	0.012	--	--	--	--	--
	(0.814)	(0.771)							
Ever used nicotine	-0.0344	0.0257	415	0.178	Ever Smoker	0.0879	0.169***	411	0.249
	(0.0658)	(0.0557)				(0.0539)	(0.0506)		
BMI	-0.0294	-0.358	395	0.058	BMI	2.896***	1.155**	355	0.188
	(0.658)	(0.617)				(0.580)	(0.745)		
Height	1.466	0.372	395	0.484	Height	6.541***	2.579***	355	0.603
	(0.949)	(0.802)				(0.807)	(0.745)		

Note: This table mirrors Table 5 but reports analyses in the sample of European ancestry biological children (instead of the sample of Korean adoptees). Each row in each panel represents a separate regression of an outcome on family type dummies (Panel A) or PGI tercile dummies (Panel B), with the Type 3 dummy omitted from the Panel A regressions and the Tercile 1 dummy omitted from the Panel B regressions. Panel B regressions are estimated in the sample of genotyped individuals only. All regressions include the baseline control variables (including the 10 top PCs of the ancestry-specific SNP data for the Panel B regressions). Type 1 families are defined as those with three or fewer children whose two parents each have a four-year college degree; Type 3 families are defined as those (i) with four or more children and where neither parent has a four-year college degree or (ii) in the bottom quintile of the SES distribution; Type 2 families are the families that are neither Type 1 nor Type 3. For the continuous outcomes, OLS regressions were estimated. For the binary outcomes (college and ever used nicotine), logistic regressions were estimated, the reported coefficients are average marginal effects, and Nagelkerke's pseudo R^2 was used. Robust standard errors are in parentheses and are clustered at the family level.
*** p<0.01, ** p<0.05, * p<0.1."

Appendix Table G.6: Genetic nurture estimates for the Korean adoptees

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Dependent variable	EA	College	GPA	Soft skills	Cognitive performance	Log income	Ever used nicotine	BMI	Height
PGI	EA	EA	EA	EA	Cognitive performance	Income	Ever smoker	BMI	Height
Panel A: With mother's and father's PGSs									
Child's PGI	0.429***	0.045	0.185***	0.192***	2.619***	0.053	0.115***	0.627**	1.609***
	(0.158)	(0.032)	(0.054)	(0.054)	(0.870)	(0.063)	(0.027)	(0.261)	(0.387)
Mom's PGI	0.546***	0.050	-0.052	-0.075	-0.606	0.054	0.047*	0.542*	0.134
	(0.181)	(0.041)	(0.058)	(0.074)	(1.113)	(0.061)	(0.025)	(0.274)	(0.360)
Dad's PGI	0.200	0.045	0.048	0.020	1.000	-0.078	0.068**	0.248	0.164
	(0.154)	(0.029)	(0.043)	(0.054)	(0.767)	(0.056)	(0.029)	(0.203)	(0.393)
$\Delta\bar{R}^2$ (parents' PGIs)	0.068***	0.010	-0.002	-0.003	<0.001	0.004	0.013**	0.018*	-0.003
Joint sig. of parents' PGIs (p)	0.003	0.112	0.431	0.599	0.370	0.284	0.028	0.078	0.860
N	151	151	236	238	238	138	238	213	213

Note: All regressions include the baseline control variables. For the continuous outcomes, OLS regressions were estimated, the adjusted $\bar{R}^2$ was used, and the test for joint significance is the F test. For the binary outcomes (college and ever used nicotine), logistic regressions were estimated, the reported coefficients are average marginal effects, McFadden's adjusted pseudo $\bar{R}^2$ was used, and the test for joint significance is the Wald test. The incremental adjusted ($\Delta\bar{R}^2$) of the parents' PGIs is the difference between the adjusted $\bar{R}^2$ of the regression of the outcome on the child's PGI and the controls, and that of the same regression (in the same sample) but without the parents' PGIs. The stars on the $\Delta\bar{R}^2$'s indicate the significance level of the associated test for joint significance, as indicated by the P values in the following row. Robust standard errors clustered at the family level are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table G.7: Robustness checks for GxE models of cognitive performance in the sample of Korean adoptees

Robustness check	(1) Baseline	(2) Males	(3) Females	(4) <15 yrs. old	(5) ≥15 yrs. old	(6) $(\text{cog. perf.})^2$ instead of cog. perf.	(7) $\sqrt{\text{cog. perf.}}$ instead of cog. perf.	(8) $(\text{SES} + 5)^2$ instead of SES	(9) $\sqrt{\text{SES} + 5}$ instead of SES	(10) Extensive set of controls	(11) Interaction with high family SES dummy
Panel A: Model II (with the interacted controls, following Keller 2013) with the PGI of cognitive performance											
PGI of cog. perf.	-1.464*	-3.512*	-0.662	-2.178	-2.542**	-284.536*	-0.075**	-0.154*	-6.213*	-1.069	-2.047
x family SES	(0.763)	(1.858)	(1.040)	(1.361)	(1.130)	(161.020)	(0.038)	(0.081)	(3.220)	(1.034)	(1.536)
R^2	0.225	0.383	0.238	0.292	0.371	0.225	0.225	0.228	0.224	0.245	0.237
Panel B: Model II (with the interacted controls, following Keller 2013) with the PGI of EA											
PGI of EA	-2.847***	-4.956***	-1.314	-3.427**	-3.156**	-570.044***	-0.143***	-0.276***	-12.666***	-2.506**	-2.467
x family SES	(0.880)	(1.859)	(1.178)	(1.662)	(1.215)	(182.860)	(0.044)	(0.090)	(3.814)	(1.039)	(1.496)
R^2	0.263	0.422	0.257	0.318	0.350	0.261	0.262	0.264	0.261	0.297	0.252
Observations	361	141	220	171	190	361	361	361	361	335	361

Note: Panels A and B report estimates from models with the PGIs of cognitive performance and of EA, respectively. The table reports robustness checks for GxE models of cognitive performance in the sample of males (col. 2) and females (col. 3) only; in the sample of adoptees who were less than (col. 4) and at least (col. 5) 15 years old when cognitive performance was measured; with the dependent variable cognitive performance replaced by its square (col. 6) and its square root (col. 7); with the family SES variable replaced by $(\text{SES} + 5)^2$ (col. 8) and $\sqrt{\text{SES} + 5}$ (col. 9); with the extensive controls as well as their interactions with family SES and with the PGI (col. 10); and with a specification in which we dichotomize the family SES variable by replacing it by a dummy indicating whether one's family SES is above the median (among the Korean adoptees; col. 11). Column (1) reports the baseline estimates (which also appear in Table 6). In addition to the PGI x family SES (or high family SES dummy, in col. 11) term, all models include the PGI, family SES, the baseline control variables, as well as the baseline controls interacted with family SES (or the high family SES dummy) and with the PGI. Only the estimate of the coefficient on the PGI x family SES interaction is reported, as the interacted controls make the coefficients on the PGI and family SES difficult to interpret. The extensive controls include the baseline controls as well as the rearing mother's and father's ages when the child was born, the number of siblings in the rearing family, and dummies indicating whether the family is a mixed biological and adoptive family (vs. a purely adoptive family), whether the adoptees' adoptive parents reside in a city or suburb, and whether they were still married at intake. For all outcomes (including the binary outcomes), OLS regressions were estimated. The number of observations is the same in Panels A and B for each column. Standard errors clustered at the family level are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Appendix Table G.8: Baseline GxE specification in the sample of European ancestry biological children

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Dependent variable	EA	College	GPA	Soft skills	Cognitive performance	Cognitive performance	Log income	Ever used nicotine	BMI	Height
PGI	EA	EA	EA	EA	Cognitive performance	EA	Income	Ever smoker	BMI	Height
Panel A: Model I (without the interacted controls)										
PGI	0.316***	0.082***	0.176***	0.188***	3.440***	3.715***	0.018	0.059**	1.370***	3.076***
	(0.120)	(0.026)	(0.039)	(0.053)	(0.699)	(0.697)	(0.040)	(0.025)	(0.276)	(0.331)
Family SES	0.483***	0.138***	0.116***	0.148***	2.024***	2.001***	0.089**	-0.006	0.043	0.322
	(0.111)	(0.024)	(0.036)	(0.047)	(0.606)	(0.610)	(0.039)	(0.023)	(0.252)	(0.340)
PGI x family SES	-0.027	-0.057**	0.002	0.066	-0.008	0.296	-0.037	0.001	-0.167	0.181
	(0.120)	(0.025)	(0.038)	(0.043)	(0.639)	(0.594)	(0.039)	(0.024)	(0.264)	(0.358)
R^2	0.177	–	0.148	0.217	0.201	0.212	0.148	–	0.207	0.629
Panel B: Model II (with the interacted controls, following Keller 2013)										
PGI x family SES	-0.182	-0.076**	0.013	0.076*	-0.029	0.306	0.033	0.007	0.025	-0.026
	(0.149)	(0.036)	(0.036)	(0.046)	(0.634)	(0.673)	(0.044)	(0.025)	(0.266)	(0.365)
R^2	0.275	–	0.217	0.280	0.244	0.272	0.220	–	0.258	0.666
Observations	277	277	387	400	398	398	259	374	348	348

Note: This table mirrors Table 6 but reports analyses in the sample of European ancestry biological children (instead of the sample of Korean adoptees). Model I in Panel A includes the baseline control variables. Model II in Panel B also includes the interactions of this baseline set of controls with family SES and with the PGI, and is otherwise identical to Model I. Only the coefficient on the PGI x Family SES interaction is reported for Model II, as the interacted controls make the coefficients on the PGI and Family SES difficult to interpret. For all outcomes (including the binary outcomes), OLS regressions were estimated. The number of observations is the same in Panels A and B for each outcome. Standard errors clustered at the family level are in parentheses.

*** p<0.01, ** p<0.05, * p<0.1

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