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.

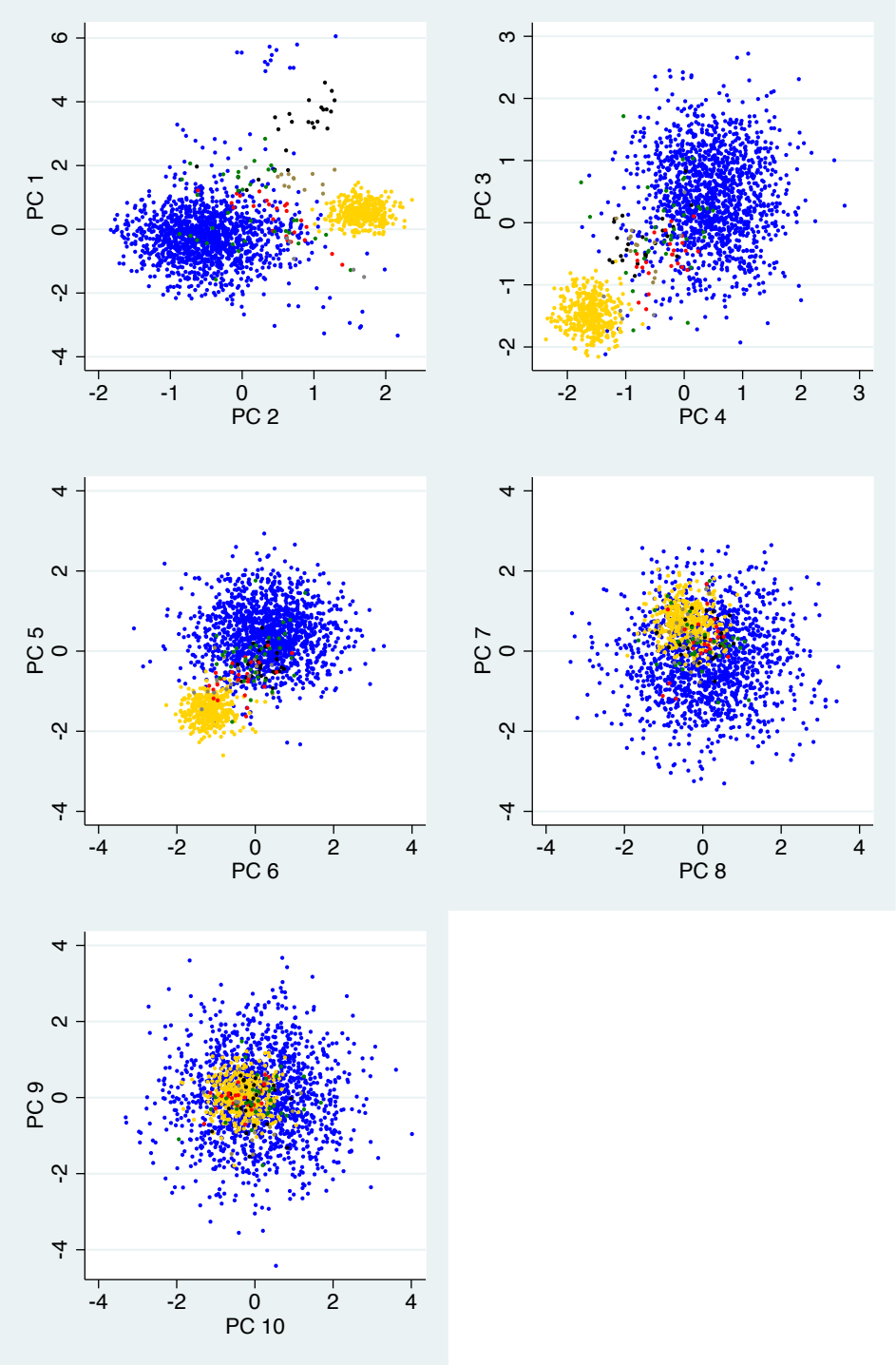
Parents still married at intake: Dichotomous variable equal to one if both the mother and father report still being married at intake and zero if either parent reports that they are single, living as a married couple with someone (parent or other), divorced, separated, widowed, or if the family reports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

C. ANALYSIS OF GENETIC OUTLIERS

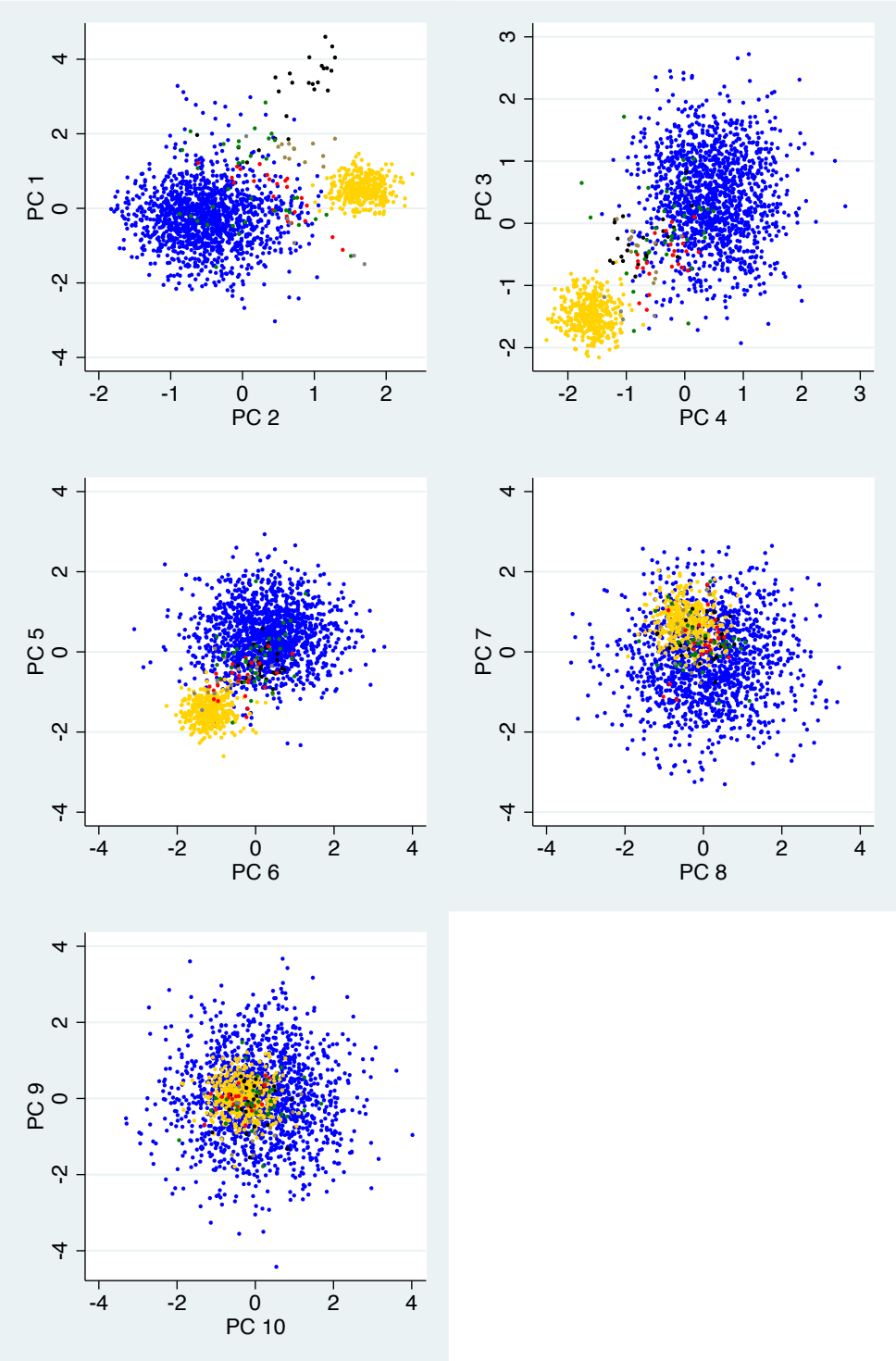
To identify the genetic outliers among the European and Korean ancestry individuals, we plotted and visually inspected the top 10 PCs of the genetic relatedness matrix of the full sample of MCTFR-SIBS individuals who have been genotyped. Appendix Figure C.1 shows these plots.

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.

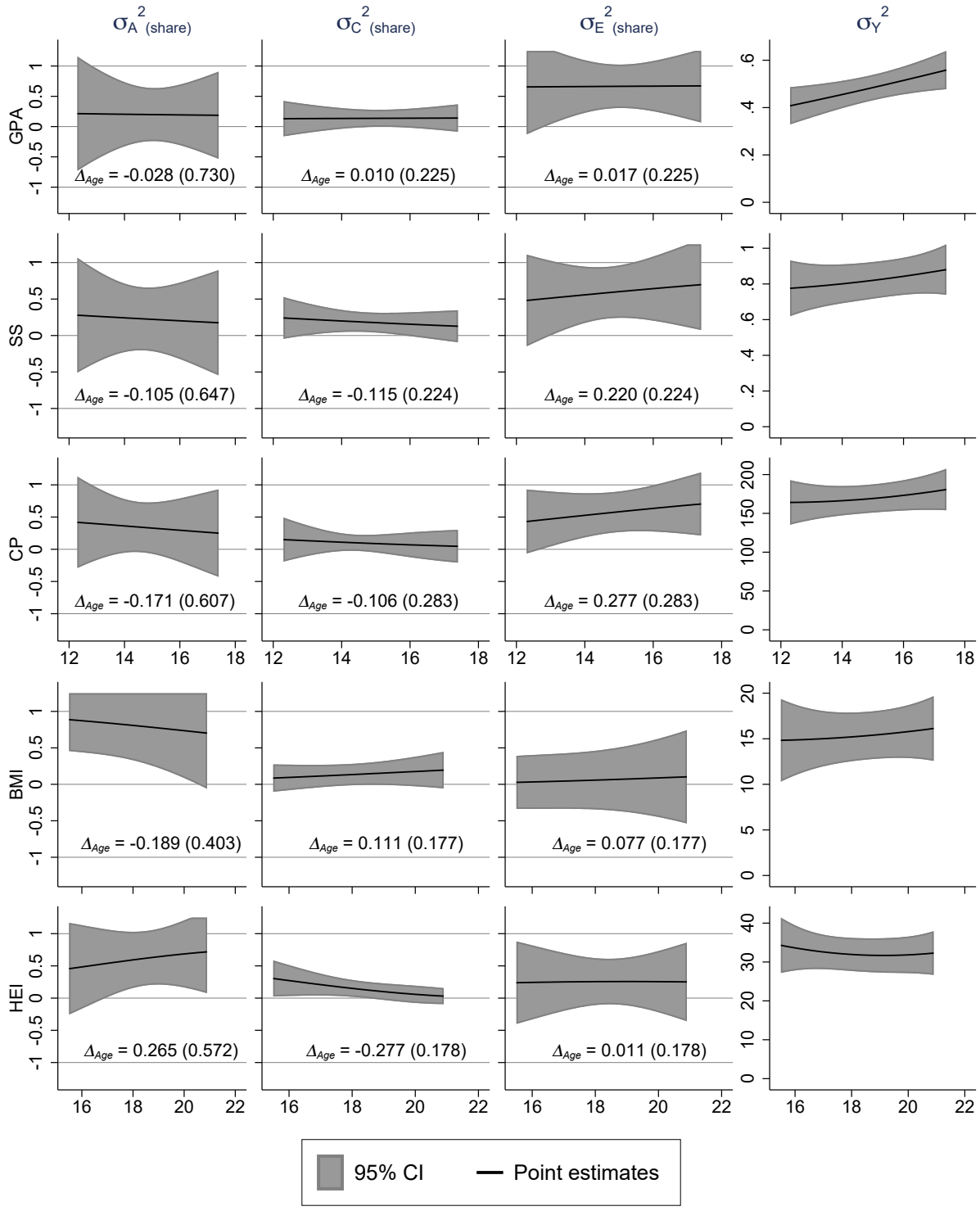
F.5 Stata code for the simulations

```
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'
```

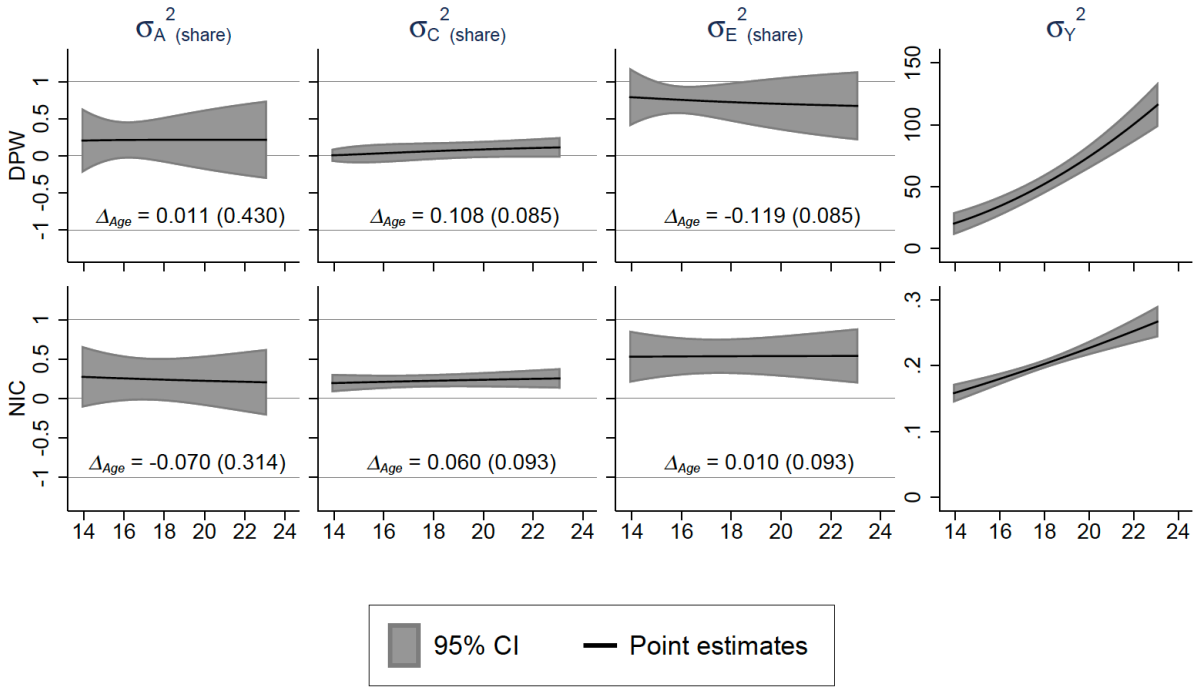
```
    *****
    * Generate the FAMID's
    qui gen FAMID=round(_n/2)     if _n<=2*`N_FAMID_pair'
    qui replace FAMID=_n-`N_FAMID_pair' if _n>2*`N_FAMID_pair'

    *****
    * 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


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

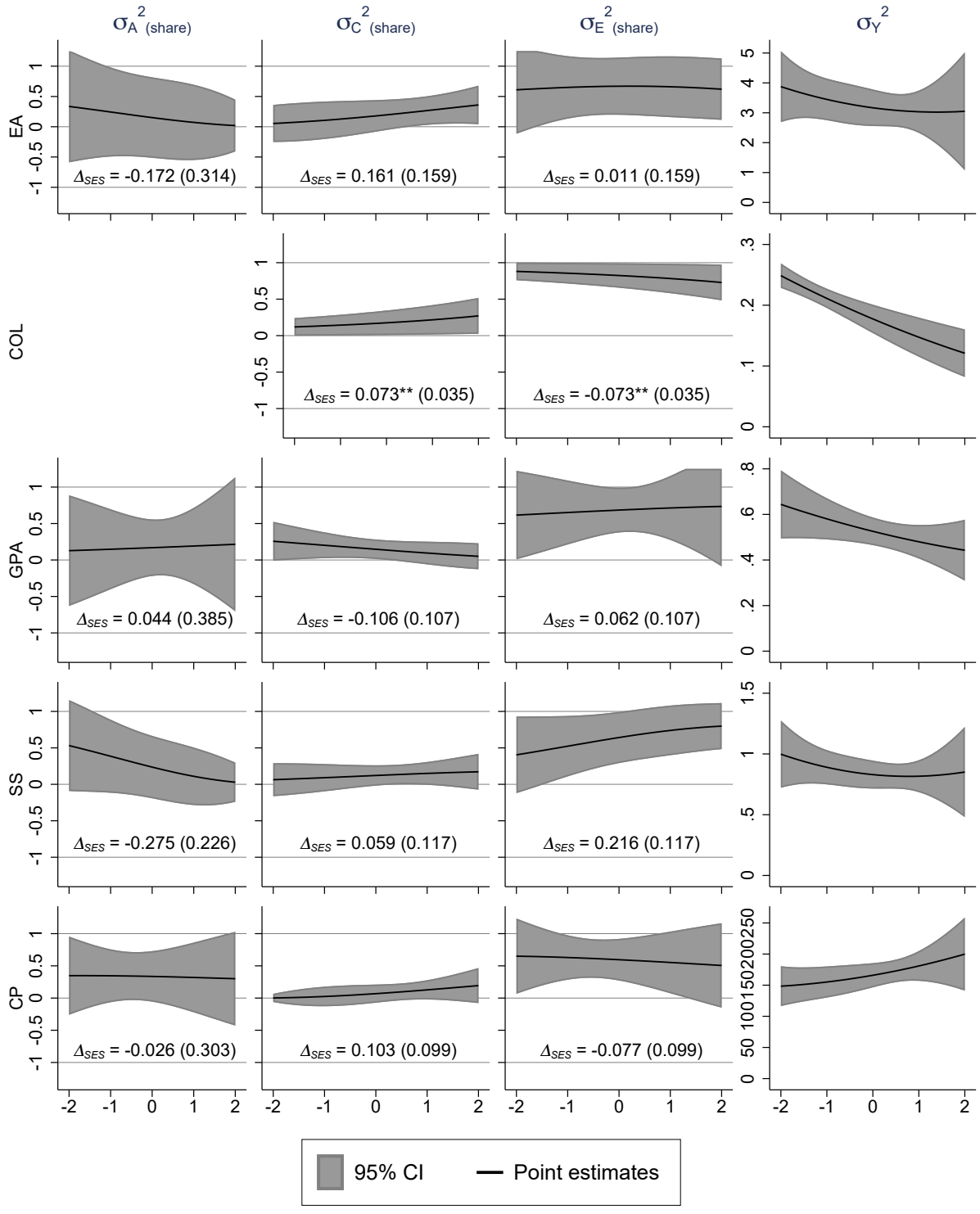
G. ADDITIONAL FIGURES AND TABLES



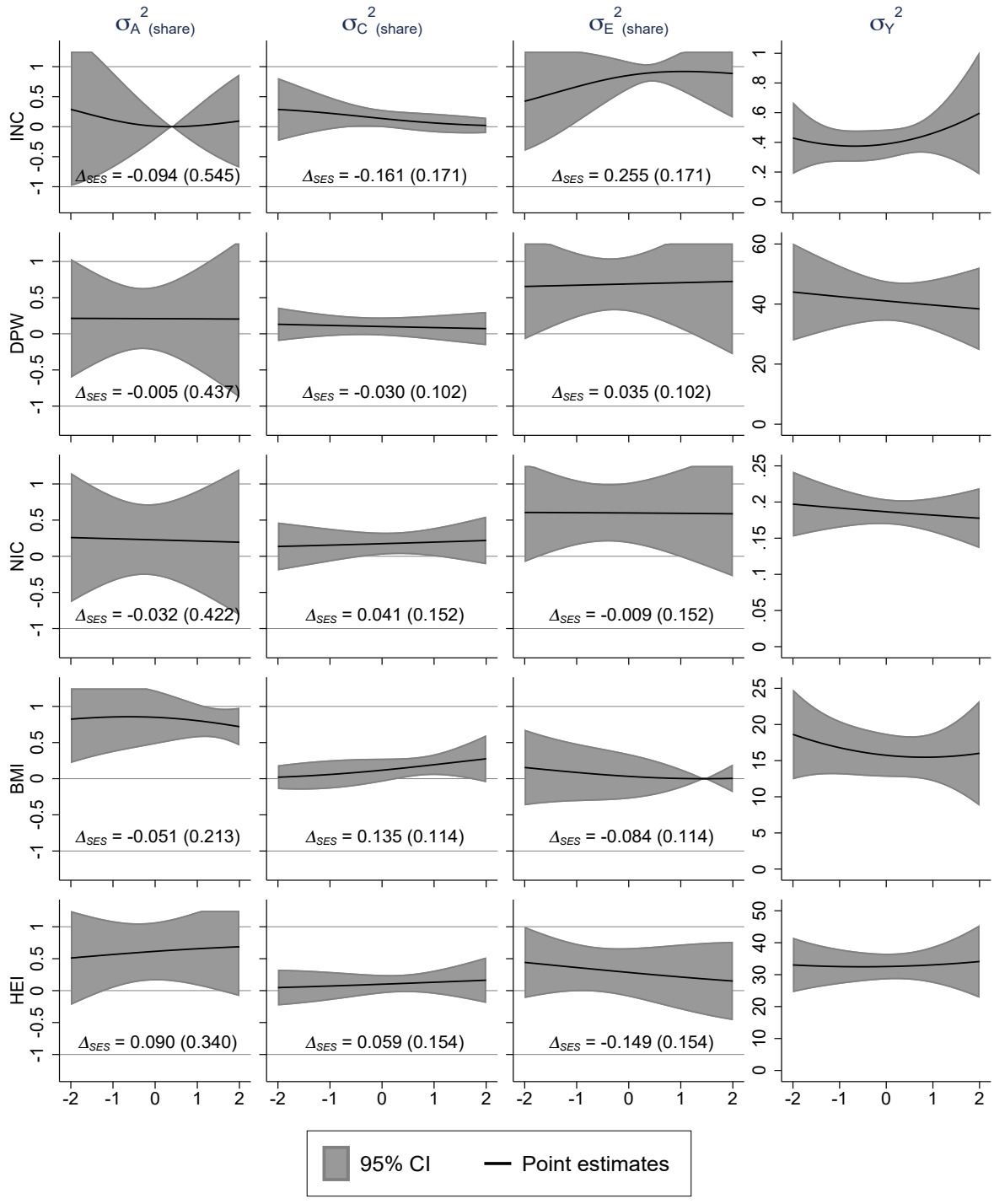
Appendix Figure G.1. *(Continues)*



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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