Research Article

Admixture Regression, Cognitive Ability, and Educational Attainment: A Social Science Application Using the Add Health Survey

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Socioeconomic inequalities between racial/ethnic groups are a central concern in American social science. Admixture regression methodology (ARM) remains underutilized as an analytic tool for understanding these disparities. ARM exploits natural variation in continental genetic ancestry within socially identified racial/ethnic groups to test whether outcomes track ancestry proportions. Using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), we apply ARM to examine whether continental genetic ancestry predicts a longitudinal measure of general cognitive ability (*g*) and educational attainment among genetically defined African- and Hispanic-ancestry US-born Americans.Measurement invariance held across the African ancestry continuum in both the African- and Hispanic-ancestry samples. However, metric invariance did not hold across the Amerindian ancestry continuum in the Hispanic-ancestry sample, with the *g* factor becoming increasingly defined by vocabulary at higher levels of Amerindian ancestry; individual test results are therefore presented as the primary analysis for this component.We find that African ancestry (in the African-ancestry sample) is strongly negatively associated with both *g* and educational attainment. Amerindian ancestry was also negatively associated with individual cognitive test scores and educational attainment. The model-implied effects, extrapolated to a hypothetical shift from 0% to 100% of the relevant ancestry component (beyond the observed ancestry ranges in these samples), are large: $B = -1.36$ SD units on *g* for African ancestry and $B = -1.75$ SD units on *g* for Amerindian ancestry (the latter to be interpreted with particular caution due to the measurement invariance limitation). After controlling for parental socioeconomic status and interviewer-rated phenotype measures, these associations are partially attenuated but remain statistically significant.Mediation analyses indicate that cognitive ability statistically mediates 68-83% of the African ancestry–educational attainment

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association in the African-ancestry sample and 47-60% of the Amerindian ancestry–educational attainment association in the Hispanic-ancestry sample. Supplemental analyses support the robustness of these patterns across generational subgroups, alternative geographic clustering specifications, and additional phenotypic controls. These results demonstrate the utility of admixture regression as a powerful tool for the social sciences. We situate the findings within a non-specific vertical transmission framework that accommodates genetic, cultural, and other intergenerational mechanisms without assuming their relative contributions.

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1. Introduction

Cognitive ability differences between socially identified racial/ethnic (SIRE) groups in the United States have been documented since the early twentieth century. These differences are of considerable scientific and practical importance. Cognitive ability is one of the strongest individual-level predictors of socioeconomic outcomes, and cognitive ability scores measured during adolescence can account for a substantial portion of SIRE disparities in educational attainment, income, occupational success, and health later in life (e.g., [1]). The societal stakes are high: understanding how these skill gaps arise, how they are perpetuated across generations, and how they might be reduced was ranked fourth among the social sciences' "grand challenge questions that are both foundational and transformative" [2]. Accordingly, cognitive and achievement gaps between American SIRE groups continue to be a major focus of research. One limitation of much prior work, however, is that SIRE categories are often treated as internally homogeneous and monolithic for analytic purposes, thereby leaving substantial within-group variation unexplored.

1.1. Socially defined race, genetic ancestry, and admixture regression methodology

In the United States, individuals are typically classified according to their own or their parents' identification with one or more federally defined broad racial/ethnic classifications (e.g., Asian, Black or African American, White). The Office of Management and Budget [3] notes that these SIRE classifications "should not be interpreted as being primarily biological or genetic in reference" and "may be viewed in terms of social and cultural characteristics as well as ancestry," thereby distinguishing official racial/ethnic classifications from direct measures of genetic ancestry while acknowledging that ancestry

is one component of these classifications. However, OMB's classification system was modeled on the major human geographic races described by naturalists and physical anthropologists during the eighteenth and nineteenth centuries [4]. Because the geographic races described in natural history and physical anthropology broadly correspond to the continental-level genetic or biogeographic ancestry clusters that genomics subsequently identified, socially identified racial classifications and genetic ancestry groups conceptually overlap, even though they are not identical [4]. For this reason, socially identified race and genetic ancestry group membership are empirically correlated (e.g., [5], [6]).

Owing to advances in genetics, an individual's genetic ancestry can now be estimated with high precision as admixture proportions traceable to historically semi-isolated biogeographic ancestry groups—such as Sub-Saharan African, European, East Asian, and Amerindian—which reflect overall lineage relative to ancestral source populations [7]. Critically, because socially-identified racial classifications imperfectly overlap with genetic ancestry groups, members of the same SIRE classification can differ considerably in genetic ancestry. For example, individuals who identify as Black or African American may have African ancestry ranging from roughly 2% to 100% [8], while those who identify as Hispanic may vary widely in their proportions of European, Amerindian, and African ancestry.

This within-group variation in ancestry creates a natural experiment, in the sense that ancestry proportions vary substantially among individuals who share the same broad SIRE classification. If outcomes track genetic ancestry within, or controlling for, socially identified group identity and other pertinent variables, factors associated with genetic lineage—rather than factors that affect group members irrespective of ancestry—are implicated in the causal structure underlying group differences. For this reason, regressing health or behavioral outcomes on ancestry proportions in admixed populations — known as admixture regression methodology (ARM) — has become a standard tool in genetic epidemiology for quantifying ancestry-associated variation. In these regression models, SIRE is often included and treated as a "surrogate to an array of social, cultural, behavioral, and environmental variables" alongside other pertinent demographic variables [6]. Applications of ARM span behavioral traits (such as alcohol dependence, cigarette smoking, and sleep depth), disease states (such as asthma, cardiovascular disease, and cancer), and anthropometric features (such as height and BMI) [9].

Despite this wide acceptance in genetic epidemiology, extending ARM to the social sciences has met with reluctance and at times outright hostility (e.g., [10]), even though the only fundamental methodological change is the choice of dependent variables [11]. In large part, this is because associations between genetic

ancestry and heritable traits, after controlling for appropriate confounders, can be interpreted as evidence for ancestral genetic differences— an inference frequently made in genetic epidemiology. Yet suggesting an ancestral genetic basis for socioeconomically relevant behavioral differences between racial/ethnic groups remains taboo in much contemporary Western social science [12].

This concern about genetic attributions has led many social scientists to adopt a strong social-constructivist view of SIRE, according to which racial classifications bear little to no meaningful relationship to biological variation and attempts to relate them to genetics risk reifying racial hierarchy [13][14][15]. In stronger formulations, this position leads researchers such as Herd et al. [16] to argue that "[g]iven what is known about ancestry, it makes little sense to examine racial group genetic variation, especially in the American context"—effectively foreclosing the kind of within-group ancestry analyses that are routine in genetic epidemiology.

1.2. Vertical and horizontal transmission frameworks

While strong constructivist positions are difficult to sustain empirically, the broader concern about prematurely attributing behavioral differences to genetics is not unreasonable. For this reason, we have previously clarified that associations between genetic ancestry and behavioral traits can be interpreted within the framework of non-specific vertical transmission models, as employed in the "deep roots" economic and social science literature (e.g., [17][18][19][20]). Vertical transmission, in this context, refers to the intergenerational transmission of traits or trait-relevant influences through genetic, vertical cultural, or other lineage-linked channels, without assuming a primary pathway. These models posit initial differences among the source populations of the admixed groups that are carried forward across generations, producing correlations between ancestry proportions and outcomes. Because biogeographic ancestry proportions approximate the fraction of genetic—and, less exactly over many generations, genealogical—ancestors deriving from each ancestral population, persistent intergenerationally transmitted differences are expected to scale, at least approximately, with the proportion of ancestry derived from different source populations [21][22]. This framework, while not excluding genetic transmission, accommodates the possibility that ancestry-associated variation reflects intergenerationally transmitted environmental influences, including cultural and institutional legacies [23]. It also connects ARM with the broader research program on persistent intergenerational transmission, which faces similar uncertainty about mechanisms (e.g., [24][25]).

This non-specific vertical transmission framework is pragmatic: whether genetic, vertical cultural, or other transmission mechanisms are more plausible will depend on the trait, its heritability, and the ancillary evidence. For example, if a trait has no relevant genetic variance within the populations under study, then ancestry-associated genetic differences in that trait would not be expected given the logical and mathematical relationship between within- and between-group heritabilities under the common causation assumption; consequently, nonzero within-population heritability is a necessary, though not sufficient, condition for interpreting ancestry–trait associations genetically [26]. Nonetheless, a vertical cultural transmission model may often be of interest, as would generally be the case in the social sciences. While simple ARM designs cannot distinguish between genetic and vertical cultural transmission, more elaborate approaches — for example, incorporating local ancestry as an additional term in the regression model — can do so. In some contexts, understanding the specific mechanisms underlying intergenerational transmission matters; in others, it may not.

This vertical transmission model can be contrasted with horizontal transmission models, according to which SIRE-related differences are driven by common factors that affect members of SIRE groups as such, rather than in proportion to individual-level ancestry. Such factors may include cultural norms, dialect, language, involuntary minority status, and SIRE-based segregation. Most commonly cited examples of structural racism— for example, voter suppression, Jim Crow laws, segregated housing, and redlining [27][28][29]— operate at the level of socially identified group membership rather than scaling with individual-level genetic ancestry. To the extent that outcomes track genetic ancestry within SIRE groups, the explanatory scope of purely horizontal transmission models is reduced relative to that of vertical transmission models.

1.3. Prior research on admixture, cognitive ability, and socioeconomic outcomes

The earliest cognition-related admixture studies, which relied on phenotype-based estimates of admixture and recorded genealogy rather than genomic data, were conducted in the early-to-mid twentieth century. These studies examined whether European admixture was associated with scholastic achievement and cognitive test scores among admixed African American, Native American, and Hispanic populations. Most reported a modest positive correlation between measures of European ancestry and cognitive or achievement test scores, with a median correlation of approximately $r = .16$ [18].

These modest effect sizes were frequently interpreted as too small to be practically meaningful —a fundamentally flawed conclusion since it ignores the substantial attenuating effect of ancestry range

restriction within socially identified groups [18]. The research program was nonetheless largely abandoned, partly on account of this misinterpretation and partly for other reasons.

More recent work using genomic measures of ancestry has confirmed and extended these early findings. Continental genetic ancestry proportions have been shown to predict both socioeconomic status (SES) and cognitive ability at the regional level across American nations and their first-order administrative subdivisions [30]. A large meta-analysis of 88 epidemiological studies conducted across the Americas, with a plurality of studies from the United States, found that measures of SES covary with continental ancestry at the individual level, including within local regions and within SIRE groups [9].

At the individual level, ARM analyses have similarly reported robust associations between ancestry and general cognitive ability (g) after controlling for parental SES and genetically predicted markers of skin, hair, and eye color, used as proxies for potential phenotype-based discrimination. These ARM studies further indicate that the ancestry–g association is substantially mediated by brain features [31]. A preprint by Guo et al. (2019, [32]) reported parallel findings in the Add Health sample: African and Amerindian ancestry principal components predicted Picture Vocabulary Test scores among African and Hispanic Americans. Unfortunately, these results were never published in full and the relationships were not explored in detail.

While this body of evidence is growing, it is still based on a relatively small number of samples and datasets. Independent replication across additional datasets and methods is therefore needed to strengthen confidence in the findings.

1.4. The current study

To our knowledge, no study has tested whether cognitive ability mediates the association between genetic ancestry and socioeconomic outcomes in adulthood. Prior admixture research has largely been limited to children and adolescents. Moreover, only one small substudy known to the current authors has included interviewer-rated color as a phenotype control, despite color-based discrimination frequently being invoked as an explanation for group differences.

Following the careful sequential analytic approach outlined by Hu et al. (2019, [20]), we apply ARM to data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Focusing on the African-ancestry and Hispanic-ancestry samples, we examine three main questions:

1. Is continental genetic ancestry associated with a longitudinal measure of general cognitive ability (*g*) and with adult educational attainment?
2. Do these associations remain after controlling for parental socioeconomic status (SES) and interviewer-rated race-associated phenotypes, especially skin color?
3. Does cognitive ability mediate the relationship between genetic ancestry and adult educational attainment?

2. Methods

2.1. Dataset

We analyzed data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a large-scale, nationally representative longitudinal study that began with U.S. adolescents in grades 7-12 and has followed them into adulthood (see https://addhealth.cpc.unc.edu/ for details). The study began with over 20,000 participants in grades 7-12 during the 1994-1995 school year and collected data through an in-school survey and subsequent in-home interviews: Wave I in 1994-1995, Wave II in 1996, Wave III in 2001-2002, Wave IV in 2008-2009, and Wave V in 2016-2018.

Analyses began with 9,130 participants with available precomputed polygenic scores and genetic principal components (PCs) in Wave IV. Add Health assigned these individuals to genetically identified European, African, Hispanic, and East Asian ancestry groups using the PSANCEST variable, following the procedure detailed in [33]. In brief, Add Health computed the means and standard deviations of the first two ancestry PCs among socially identified White, Black, and Hispanic respondents and then assigned individuals in the full genotyped sample to an ancestry group if their values on both PCs fell within the specified one- to two-standard-deviation window around that group's mean.

We excluded (a) individuals missing both verbal vocabulary tests, (b) members of the East Asian ancestry subsample, (c) socially identified Asian respondents in any ancestry group, and (d) individuals born outside the United States. East Asian individuals were excluded because the available ancestry PCs do not adequately distinguish East Asian from Amerindian ancestry, which would confound the Amerindian admixture regression. Foreign-born individuals were excluded because the cognitive battery is culturally and linguistically loaded — two of the four measures are picture vocabulary tests administered in English — making scores for non-native-raised individuals non-comparable.

These exclusions left 5,657 European-, 1,921 African-, and 813 Hispanic-ancestry individuals. Mean differences were computed for the European-, African-, and Hispanic-ancestry groups, but ARM analyses were limited to the African- and Hispanic-ancestry samples because the European-ancestry sample contains little non-European ancestry variation.

2.2. Variables

Age. Age at Wave IV was calculated as a continuous measure in years by subtracting the respondent's birth year and month (H4OD1Y, H4OD1M) from the Wave IV interview year and month (IYEAR4, IMONTH4), with months expressed as fractions of a year.

Sex. The Wave IV variable BIO_SEX4 was recoded as 1 = female and 0 = male.

Region of residence. The REGION variable coded the U.S. Census region (Northeast, Midwest, South, or West) of the participants at the time of the Wave IV interview and was retained as a factor. Missing region values were imputed as the modal region within each PSANCEST group.

School attended. For robustness analyses, a factor variable for school attended was created from the PSUSCID variable and used as an alternative clustering variable instead of REGION. Because school attendance in the United States is strongly assortative by family SES and residential context, school ID was used as a robustness clustering variable rather than as the primary geographic clustering variable.

Socially-identified race/ethnicity (SIRE) and Hispanic nationality. The genetically defined African-ancestry sample had an estimated African ancestry range of 55-100% and consisted of 1,891 socially identified non-Hispanic Black respondents, 24 Hispanic respondents, 4 White respondents, and 1 Native American respondent, with SIRE data missing for one individual. The genetically defined Hispanic-ancestry sample had estimated Amerindian and African ancestry ranges of 8-50% and 0-26%, respectively, and consisted of 1 socially identified non-Hispanic Black respondent, 704 Hispanic respondents, 102 non-Hispanic White respondents, and 6 Native American respondents. Binary SIRE dummy variables were created for the ARM analyses: SIRE_Black was coded 1 for socially identified Black respondents and 0 otherwise in the African-ancestry sample; SIRE_Hispanic was coded 1 for socially identified Hispanic respondents and 0 otherwise in the Hispanic-ancestry sample. For the Hispanic-ancestry sample, additional dummy variables were created for self-reported Hispanic nationality: Mexican, including Chicano; Cuban; Puerto Rican; Central or South American; and Other Hispanic.

Nativity and language. Respondents were coded as U.S.-born (1 = U.S.-born, 0 = otherwise) based on Wave IV self-report (H4OD4 = 1) and as usually speaking English at home (1 = English, 0 = otherwise)

based on Wave I self-report (H1GI10 = 1). Parental foreign-born status was coded hierarchically: respondents were coded 1 if (a) the responding parental figure reported being foreign-born in Wave I (PA3), or, if that information was unavailable, (b) the adolescent reported either resident parent as foreign-born (H1RM2 or H1RF2), or, if that information was unavailable, (c) the adolescent reported either non-resident parent as foreign-born (H1NM6 or H1NF6). Otherwise, this variable was coded as 0.

Interviewer-rated skin color, hair and eye color, and interviewer ID. In Wave III, interviewers rated the respondents' skin color (H3IR17) on a 5-point scale (1 = black to 5 = white), hair color (H3IR18), and eye color (H3IR19). Hair color was recoded as a binary indicator: 1 ("light") if blond or red, 0 if black or brown, and missing otherwise. Eye color was recoded similarly: 1 ("light") if blue, green, or hazel, 0 if black or brown, and missing otherwise. The recoded hair and eye color variables were averaged to create a continuous light-pigmentation measure ranging from 0 to 1; the composite was set to missing only when both components were missing. Interviewer ID was retained as a factor so as to include interviewer fixed effects.

Interviewer-observed race. In Wave III, interviewers coded respondents' observed racial appearance (H3IR4) into one of four categories: White, Black, Native American, or Asian/Pacific Islander. Dummy variables were created for Black, Native American, and Asian/Pacific Islander.

Physical attractiveness. In Wave III, interviewers rated respondents' physical attractiveness (H3IR1) on a 5-point scale (1 = very unattractive to 5 = very attractive). Because physical attractiveness has been reported to covary with European ancestry in Add Health, it was included in supplemental models as an additional proxy for European-associated phenotype.

Self-reported racial discrimination. In Wave IV, participants were asked how often they felt treated with less respect or courtesy than other people (never, rarely, sometimes, often), followed by a question about the primary reason. Participants were coded as having experienced racial discrimination (1) if they reported experiencing less respect or courtesy sometimes or often and attributed this treatment to race, ancestry/national origin, or skin color; all others were coded 0.

Educational attainment. Educational attainment was measured using Wave V self-reports, with Wave IV educational attainment substituted when Wave V data were missing. Categorical responses were converted to approximate years of completed education (e.g., 8th grade or less = 8 years).

Parental SES. Parental SES was constructed as a Wave I composite of parental education and household income. Parental education was derived hierarchically to maximize coverage. The primary source was the

Parent Questionnaire, using the responding parent's self-reported education (PA12) and reported education of the spouse/partner (PB8). When those data were unavailable, adolescent reports of resident parents' education (H1RM1, H1RF1) were used; when resident-parent reports were unavailable, adolescent reports of non-resident parents' education (H1NM4, H1NF4) were used. Categorical responses were recoded into approximate years of schooling using standard Add Health conventions (e.g., high school graduate or GED = 12 years; college graduate = 16 years). Years of education were calculated separately for the mother- and father-figure and then averaged, with the composite set to missing if data were unavailable for both. Household income was obtained from the responding parent's report of total household income in the previous year (PA55). Missing household-income values were imputed using a linear regression model with average parental education and genetic ancestry-group indicators (African and Hispanic) as predictors, so that imputation reflected both education-linked and between-group differences in reported income. Predicted values were winsorized at the 1st and 99th percentiles of the observed distribution. The final composite was created by standardizing both the averaged parental education and imputed income measures and averaging the two, yielding a continuous, approximately normally distributed SES indicator with near-complete analytic coverage.

Cognitive measures. A picture vocabulary test was administered in Waves I and III. In Wave I, adolescents completed the Add Health Picture Vocabulary Test (PVT1), a 78-item computerized abridged version of the Peabody Picture Vocabulary Test–Revised. A second receptive vocabulary measure (PVT3) was administered in Wave III. The Add Health team computed age-standardized scores for both PVT measures, which were used in the current analysis. In Wave IV, participants completed a 15-item delayed recall test and a 7-item digit span backwards test.

General cognitive ability (*g*) scores. Multi-group confirmatory factor analysis (MGCFA) and local structural equation modeling (LSEM; [34]) was conducted using the four cognitive measures. We first assessed the impact of outliers and missing data and evaluated distributional assumptions. The cognitive measures were adjusted for centered age, centered age squared, and sex before the MGCFA and LSEM analyses. MGCFA was performed using full information maximum likelihood (FIML) to handle missing data. Maximum likelihood estimation with robust standard errors (MLR) was used as the estimator, given the modest multivariate non-normality of the data. Models were evaluated following the method reported by Lasker et al. (2019, [35]). In particular, invariance at each level was evaluated using the change in CFI criterion, where a CFI decrease exceeding .010 indicates meaningful deterioration. CFI increases

(indicating improved fit with added constraints) were not considered failures. Other fit indices (TLI, MFI, RMSEA, SRMR, AIC, and BIC) were taken into account.

First, we conducted multi-group CFA across the ancestry groups (European and African, and European and Hispanic) and across ancestry terciles (low, mid, high) within the African and Hispanic ancestry samples to test the standard invariance hierarchy: configural (same factor structure), metric (equal loadings), scalar (equal intercepts), and strict (equal residual variances).

Second, we applied local structural equation modeling (LSEM) with the progressive invariance testing procedure described by [36], implemented in the *sirt package* [37]. The single-factor model was estimated at focal points across the within-sample ancestry PC distribution using a Gaussian kernel (h = 1.5) and MLR estimation with FIML. Measurement invariance was tested progressively: a configural model allowing all parameters to vary across the moderator was compared to models imposing metric (equal loadings), scalar (equal loadings and intercepts), and strict (equal loadings, intercepts, and residual variances) constraints. At each level, bootstrap confidence bands (R = 500) were used to evaluate whether parameter trajectories fell within the range expected under the invariance constraint. This approach complements the discrete MGCFA by treating ancestry as a continuous moderator rather than imposing arbitrary tertile boundaries.

We saved g scores from the SIRE/ancestry-group based MGCFA analyses and used these in further analysis.

Polygenic *g*. A composite polygenic score for *g* was constructed using the SMTpred framework by combining three polygenic scores—educational attainment [38], cognitive ability [39], and intelligence [40]—with weights based on GWAS sample size, SNP heritability, and genetic correlations among the traits (r_g = .73–.87). The composite was standardized (M = 0, SD = 1) across the full genotyped sample.

Genetic principal components for PGS models. The first 10 within-ancestry genetic PCs were included as covariates to account for residual population stratification when examining the associations between polygenic *g* and cognitive or educational measures. These were precomputed and provided in the Add Health polygenic scores file. PCs were estimated separately within each genetic ancestry group using 609,130 genotyped SNPs. Because these PCs were computed separately within each genetic ancestry group, their axes and scores are not directly comparable across groups.

Ancestry PCs as measures of continental ancestry. In the full Add Health sample, the first two genetic PCs are strongly correlated with African and Amerindian ancestry, respectively [33]. However, the ARM

analyses use within-ancestry PCs, so we verified that the relevant within-group PC axes also reliably track continental ancestry proportions.

To do so, we validated the within-ancestry PCs in a large independent American training sample using a four-step procedure:

1. We computed global ancestry proportions using tADMIXTURE and derived global PCs in the independent training sample, following the Add Health procedure described in [33].
2. We created ancestry-matched training subsets. The African training sample included self-identified African Americans falling within ±2 SD of the full-sample PC1 mean and ±1 SD of the full-sample PC2 mean. The Hispanic training sample included self-identified Hispanic Americans falling within ±1 SD of both PC1 and PC2 means.
3. Within these subsets, we computed new principal components using the same method.
4. We then correlated these within-ancestry PCs with ADMIXTURE-derived continental ancestry proportions.

In the African-ancestry training sample, the African-associated PC correlated extremely strongly with African ancestry proportions (r = .989). In the Hispanic-ancestry training sample, the African-associated PC correlated with African ancestry at r = .956, and the Amerindian-associated PC correlated with Amerindian ancestry at r = .933. These correlations increased further in the uncensored training data (r = .984 for African ancestry and r = .953 for Amerindian ancestry), indicating that the somewhat lower values in the Hispanic-ancestry sample were partly due to variance compression owing to the tighter selection criteria.

After this validation, we aligned the Add Health within-group PC axes with the corresponding training-sample PC axes by comparing the pattern of associations between polygenic scores (PGS) and PCs across the two datasets. This matching procedure was feasible because some PGS exhibit large ancestry-related differences due to ascertainment bias. For example, the Major Depressive Disorder PGS showed a 15.6 SD difference between European- and African-ancestry groups, while the Waist-to-Hip Ratio PGS showed a 1.21 SD difference between European- and Hispanic-ancestry groups [41].

In the main ARM tables, ancestry-PC predictors and outcomes are standardized within ancestry group, so the reported PC coefficients represent expected change in the outcome, in SD units, per one-SD change in the ancestry PC. Because the PCs are standardized to have a mean of zero and unit variance within each

ancestry subgroup, the regression coefficients represent the expected change in the outcome (in *SD* units) per one standard deviation change in the ancestry PC.

Tables also present rescaled ancestry coefficients (*B*), defined as the estimated outcome difference associated with a hypothetical shift from 0% to 100% of the relevant ancestry component. These *B* coefficients were obtained by rescaling the standardized PC coefficients using estimated within-sample standard deviations of ancestry proportions derived from truncated Beta distributions. Importantly, because the PSANCEST-defined samples are censored at the tails of the ancestry distributions, these full-range *B* coefficients represent model-based extrapolations rather than directly observed contrasts between individuals with 0% and 100% of the relevant ancestry. They should therefore be interpreted as approximations, not as precise estimates of effects at the unobserved extremes. Details of the rescaling procedure and simulation-based bias assessments are provided in the Supplemental Material.

2.3. Analyses

The rationale for ARM has been described in detail elsewhere [31]. Here, we summarize the analytic approach used in the present study.

Admixture regression. We estimated linear mixed-effects models in R using the lmer function from the lme4 package [42]; statistical tests for fixed effects used the lmerTest implementation with Satterthwaite degrees of freedom, as reported in the R-Notebook in the Supplemental Material. Primary models included random intercepts for family ID (FID) and geographic region to account for non-independence of observations within families and broad geographic areas. Models including interviewer-rated phenotype variables additionally included interviewer ID as a random intercept where applicable.

Fixed-effect covariates included age, modeled with restricted cubic splines, sex, English spoken at home, foreign-born parent status, and SIRE. Models for the Hispanic-ancestry sample additionally controlled for Hispanic nationality subtype (Mexican, Cuban, Puerto Rican, Central/South American, or Other). Ancestry PCs, dependent variables, and continuous covariates were standardized within each ancestry group prior to analysis; dichotomous indicators were left on their original 0/1 scale.

Separate models were estimated for latent *g*, each of the four individual cognitive tests (PVT1, PVT3, digit span backwards, and delayed recall), and educational attainment, with the relevant ancestry PC or PCs entered as the primary predictors of interest.

Subsequent models added parental SES and phenotype/discrimination variables to evaluate attenuation of the ancestry coefficients. These models included interviewer-rated skin color, hair/eye color, and self-reported racial discrimination where the measures had sufficient usable variation within the relevant ancestry sample. Due to limited variability or insufficient usable variation, hair/eye color was omitted from the African-ancestry models, and self-reported discrimination was omitted from the Hispanic-ancestry models.

We also estimated parallel within-ancestry models in which polygenic *g* replaced the ancestry PCs as the key predictor, with the first 10 within-ancestry genetic PCs included as covariates to adjust for residual population stratification. In addition, we modeled parental SES as an outcome predicted by polygenic *g*. For these models, one individual was randomly selected per family, and the family random intercept was omitted.

Mediation analysis. To estimate the extent to which cognitive ability statistically mediates the association between genetic ancestry and educational attainment, we used the mediation package in R [43]. We estimated total effects and proportions mediated using quasi-Bayesian confidence intervals based on 1,000 simulations, with robust standard errors. Two mediators were examined separately: adolescent verbal ability (PVT1) and the longitudinal latent *g* factor. Because the latent *g* factor includes measures from multiple waves, including adult cognitive measures, mediation models using PVT1 provide the clearest temporal ordering, while models using longitudinal *g* provide a broader estimate based on the more reliable latent cognitive factor. All mediation models controlled for SIRE, sex, age splines, English spoken at home, and foreign-born parent status. Mediation models in the Hispanic-ancestry sample additionally controlled for Hispanic nationality subtype.

Partial residual plots. To visualize the adjusted relationship between ancestry PCs and *g*, we generated partial residual plots using the *visreg* package [44]. These plots show the association between the ancestry PCs and *g* after holding all other covariates constant, with loess smoothing curves overlaid to assess potential non-linearity.

The primary models are those using family and region random intercepts with the baseline demographic covariates. SES-adjusted, phenotype/discrimination-adjusted, school-clustered, generation-restricted, and additional phenotype models are treated as robustness or attenuation analyses.

2.4. Code

The R notebook used to construct the analytic variables, estimate the MGCFA models, run the ARM and PGS regressions, conduct mediation analyses, and generate robustness checks is provided in the Supplemental Material.

3. Results

3.1. Measurement Invariance Testing

3.1.1. Measurement Invariance Between Ancestry Groups

Before extracting group-specific *g* scores, we assessed measurement invariance across ancestry groups (African- vs. European-ancestry and Hispanic- vs. European-ancestry) to ensure that the latent *g* scores were psychometrically comparable across groups. The results for the African–European comparison are shown in Table 1. The single-group baseline factor model showed acceptable fit, taking into account the low degrees of freedom (*df* = 2). The configural model confirmed that the same factor structure fit both groups acceptably (CFI = .970; RMSEA = .102). Full strict invariance caused meaningful model deterioration, with modification indices pointing to the residual variance of PVT3 as the primary source of misfit. A partial strict model freeing the PVT3 residual variance across groups recovered acceptable fit and was adopted for latent *g* extraction.

The procedure was repeated for the European–Hispanic comparison (Table 2). Again, the baseline model showed acceptable fit and the configural model confirmed that the same factor structure fit both groups acceptably (CFI = .969; RMSEA = .102). Full strict invariance again showed deterioration, while a partial strict model freeing the PVT3 residual variance fit well and was adopted.

We conclude that our longitudinal *g* measure is sufficiently psychometrically comparable across groups for the present analyses. On the latent *g* scale, African-ancestry participants scored 1.17 *d* below the European-ancestry mean (African M = 0.00, SD = 0.817; European M = 0.83, SD = 0.666). Hispanic-ancestry participants scored 0.76 *d* below the European-ancestry mean (Hispanic M = −0.560, SD = 0.873; European M = 0.00, SD = 0.717). These magnitudes of general ability differences are comparable to those typically reported in the literature for SIRE groups (e.g., [45]).

Model	χ^2	*df*	CFI	TLI	MFI	RMSEA	SRMR	AIC	BIC
Baseline (combined)	147.293	2	0.976	0.928	0.990	0.101	0.032	76084.588	76167.784
Configural	152.561	4	0.970	0.909	0.990	0.102	0.034	74377.739	74544.131
Metric	169.100	7	0.968	0.945	0.989	0.079	0.036	74382.810	74528.403
Scalar	196.676	10	0.963	0.956	0.988	0.071	0.039	74403.263	74528.057
Strict (full)	369.116	14	0.914	0.927	0.975	0.091	0.050	74600.629	74697.691
Strict (partial)	199.831	13	0.961	0.964	0.987	0.064	0.041	74410.915	74514.910

Table 1. Measurement Invariance: African–European Comparison

Note. Robust scaled fit indexes are reported. Partial strict model frees the PVT3 residual variances.

Model	χ^2	*df*	CFI	TLI	MFI	RMSEA	SRMR	AIC	BIC
Baseline (combined)	132.473	2	0.971	0.912	0.990	0.102	0.034	63133.580	63214.879
Configural	134.335	4	0.969	0.908	0.990	0.102	0.034	62710.337	62872.936
Metric	136.835	7	0.969	0.946	0.990	0.078	0.035	62709.802	62852.075
Scalar	138.788	10	0.969	0.963	0.990	0.065	0.035	62705.762	62827.711
Strict (full)	202.805	14	0.944	0.952	0.983	0.074	0.042	62793.213	62888.062
Strict (partial)	153.235	13	0.966	0.969	0.989	**0.060**	0.040	62716.054	62817.678

Table 2. Measurement Invariance: European-Hispanic Comparison

Note. Robust scaled fit indexes are reported. Partial strict model frees the PVT3 residual variances.

3.1.2. Measurement Invariance Across Ancestry within the African and Hispanic Ancestry Groups

In the African ancestry sample, both ancestry tercile MGCFA and the LSEM progressive invariance tests indicated that strict invariance held across the African ancestry continuum (all ΔCFI < .010; parameter trajectories within bootstrap bands). The LSEM results for this sample are shown in Table 3.

Level	RMSEA [95% CI]	CFI [95% CI]	TLI [95% CI]	SRMR [95% CI]
Configural	.099 [.061–.137]	.961 [.930–.992]	.884 [.791–.976]	.032 [.020–.045]
Metric	.071 [.047–.095]	.956 [.923–.988]	.940 [.895–.984]	.037 [.025–.049]
Scalar	.054 [.036–.072]	.956 [.926–.986]	.965 [.941–.989]	.037 [.025–.049]
Strict	.049 [.033–.064]	.949 [.911–.988]	.972 [.950–.993]	.039 [.027–.052]

Table 3. LSEM Analysis of *g* across the African Ancestry Continuum in the African-ancestry Group

Note. Fit indices are averaged across focal points of the African ancestry PC (PC3) distribution in the African sample (N = 1912); 95% bootstrap confidence intervals in brackets (R = 500, h = 1.5).

In the Hispanic ancestry sample, results depended on the ancestry axis. For the African ancestry PC (PC5), scalar but not strict invariance held at all levels in both the discrete MGCFA and LSEM analyses. The configural-to-metric step shows no CFI deterioration (.942 to .941), and scalar invariance holds (.941). The strict step shows modest CFI decline (.941 to .929, ΔCFI = −.012), consistent with strict non-invariance. The African ancestry LSEM results for this group are shown in Tables 4.

Level	RMSEA [95% CI]	CFI [95% CI]	TLI [95% CI]	SRMR [95% CI]
Configural	.114 [.062–.166]	.942 [.887–.997]	.826 [.661–.991]	.037 [.020–.055]
Metric	.078 [.044–.111]	.941 [.882-1.000]	.919 [.839-1.000]	.038 [.022–.055]
Scalar	.059 [.036–.083]	.941 [.888–.994]	.953 [.911–.995]	.039 [.023–.054]
Strict	.054 [.036–.073]	.929 [.866–.992]	.961 [.925–.996]	.047 [.030–.063]

Table 4. LSEM Analysis of *g* across the African Ancestry Continuum in the Hispanic-ancestry Group

Note. Fit indices averaged across focal points of the African ancestry PC (PC5) distribution in the Hispanic sample (N = 813); 95% bootstrap confidence intervals in brackets (R = 500, h = 1.5).

For the Amerindian ancestry PC (PC3), Metric invariance does not hold: CFI drops from .944 (configural) to .925 (metric), ΔCFI = −.019 in the LSEM analysis. In the MGCFA analysis, factor loadings shift substantially across the Amerindian ancestry continuum, with PVT1 standardized loadings ranging from 0.73 to 1.01 across ancestry tertiles. The instability was concentrated in the PVT Wave 1 loading, which ranged from 0.73 (mid tertile) to 1.01 (low tertile, i.e., high Amerindian ancestry), with the latter exceeding the theoretical bound of 1.0 and producing a negative residual variance estimate. In the high-Amerindian tertile, *g* was disproportionately defined by PVT1 (loading = 1.01) while the remaining indicators contributed weakly (Digit Span = 0.26, Delayed Recall = 0.23). In the mid tertile, the factor was more balanced (PVT1 = 0.73, PVT3 = 0.81, Digit Span = 0.45, Delayed Recall = 0.36). This pattern suggests that among individuals with higher Amerindian ancestry, the common factor increasingly reflects verbal knowledge rather than a balanced composite of diverse cognitive abilities. The Amerindian ancestry LSEM results for this group are shown in Tables 5.

Level	RMSEA [95% CI]	CFI [95% CI]	TLI [95% CI]	SRMR [95% CI]
Configural	.113 [.067–.159]	.944 [.897–.991]	.832 [.690–.974]	.036 [.021–.051]
Metric	.088 [.056–.121]	.925 [.864–.986]	.898 [.815–.981]	.048 [.029–.067]
Scalar	.067 [.042–.092]	.925 [.862–.988]	.941 [.891–.991]	.048 [.030–.066]
Strict	.072 [.046–.097]	.878 [.775–.981]	.932 [.875–.990]	.061 [.039–.083]

Table 5. LSEM Analysis of *g* across the Amerindian Ancestry Continuum in the Hispanic-ancestry Group

Note. Fit indices averaged across focal points of the Amerindian ancestry PC (PC3) distribution in the Hispanic sample (N = 813); 95% bootstrap confidence intervals in brackets (R = 500, h = 1.5).

3.2. Global Ancestry and Cognitive Ability

Tables 6 and 7 report the admixture regression results with *g* as the dependent variable for the African- and Hispanic-ancestry samples, respectively. Table 8 summarizes the results across all five cognitive measures and educational attainment, and also reports the implied effects associated with a shift from 0% to 100% of the relevant ancestry component. Although the model-implied full-range effects (*B* coefficients) are large, these values are extrapolations based on the within-sample slopes. Because ancestry variation is restricted within the PSANCEST-defined samples, the actual observed differences between individuals at the extremes of the ancestry distributions in these samples were more modest (see Figures 1-3).

In the African-ancestry group, the African principal component was a significant negative predictor of *g*, PVT1, PVT3, delayed recall, and educational attainment, with implied African ancestry effects of $B = -1.36$ for *g* and $B = -0.84$ for education. The partial residual plot for the African PC in the African-ancestry sample (Figure 1) shows an approximately linear negative association across most of the observed range. However, the slope appears to flatten below approximately −1.5 SD on the African PC, although the wide confidence intervals at the extremes preclude a firm conclusion. One possible explanation for this apparent non-linearity is the inclusion of second-generation immigrants, who may fit the vertical-transmission assumptions less well because they are not multi-generational descendants of the admixed American populations. Consistent with this possibility, supplemental analyses excluding second-

generation immigrants produced a steeper ancestry slope. These individuals were nevertheless retained in the primary models for consistency with the Hispanic-ancestry analyses.

Predictors	*B*	*SE*	*P-value*
Intercept	-0.748	0.879	0.395
European-African PC (std)	-0.147	0.023	**<0.001**
SIRE_Black	0.012	0.203	0.955
Age (linear spline)	-0.01	0.03	0.729
Age (cubic spline)	0.031	0.035	0.377
Sex	0.083	0.043	0.054
English at Home	1.011	0.294	**0.001**
Parent Foreign Born	0.484	0.111	**<0.001**
Random Effects			
σ^2	0.39		
$\tau_{00\ FID}$	0.55		
$\tau_{00\ Region}$	0.04		
ICC	0.6		
N_{FID}	1787		
N_{Region}	4		
Observations	1921		
Marginal R^2 / Conditional R^2	0.036 / 0.619		

Table 6. Regression Results for the Effect of Genetic Ancestry on *g* in the African-ancestry Sample

doi.org/10.32388/EO7DND

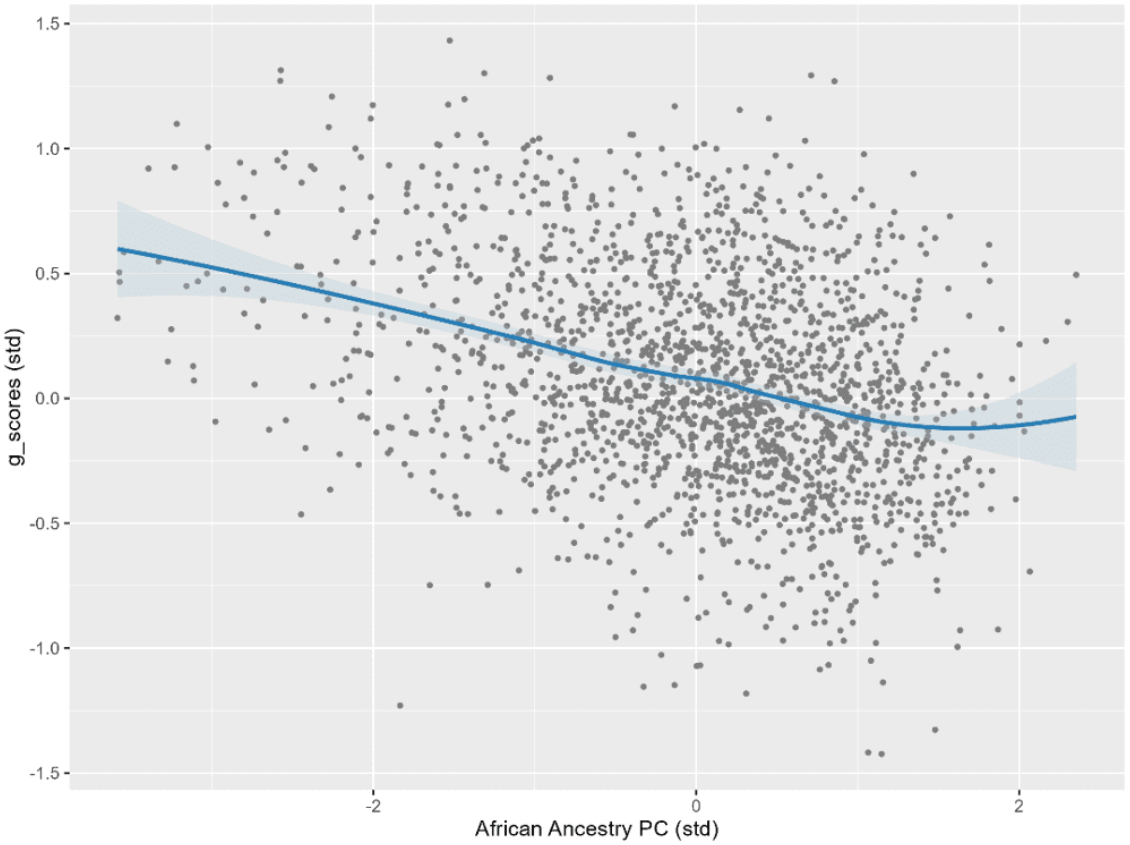

Figure 1. Partial Residuals and Estimated Regression Fits for African Ancestry PC in the African-ancestry Sample with *g* as the Dependent Variable (*N* = 1921). *Note: Loess line shown in dark blue with confidence interval shown in light blue.*

In the Hispanic-ancestry group, the African principal component was not a significant predictor of any cognitive or educational outcomes, likely due to limited statistical power stemming from low variability in African admixture within this sample, as the implied *B*s were nonetheless large. The Amerindian principal component was a significant negative predictor of *g*, PVT1, delayed recall, digit span backwards, and educational attainment, with implied effects of $B = -1.75$ for *g* and $B = -1.02$ for education. However, as noted in the measurement invariance section, metric invariance does not hold for *g* across the Amerindian ancestry continuum: the *g* factor becomes increasingly defined by vocabulary at higher levels of Amerindian ancestry, with the PVT1 standardized loading rising from 0.73 to 1.01 while non-verbal indicators weaken. The *g* estimate for the Amerindian PC should therefore be interpreted with caution. Notably, the Amerindian association was not restricted to verbal tests — delayed recall and digit span backwards also showed significant negative associations — suggesting that the pattern is not

entirely attributable to linguistic bias. Nonetheless, individual test results, which do not depend on factorial equivalence, are presented alongside *g* in Table 8 and should be considered the primary analysis for the Amerindian component.

Predictors	*B*	*SE*	*P-value*
Intercept	-0.739	1.268	0.56
European-African ancestry PC (std)	-0.084	0.052	0.107
European-Amerindian PC (std)	-0.206	0.052	**<0.001**
SIRE_Hispanic	-0.117	0.184	0.526
Mexican	-0.01	0.159	0.952
Cuban	-0.26	0.189	0.17
Puerto Rican	0.157	0.166	0.345
Other_Hispanic	0.243	0.133	0.069
Age (linear spline)	0.017	0.045	0.71
Age (cubic spline)	0.013	0.051	0.806
Sex	0.055	0.066	0.408
English at Home	0.327	0.089	**<0.001**
Parent Foreign Born	0.05	0.083	0.548
Random Effects			
σ^2	0.34		
$\tau_{00\ FID}$	0.56		
$\tau_{00\ Region}$	0.02		
ICC	0.63		
N_{FID}	752		
N_{Region}	4		
Observations	813		
Marginal R^2 / Conditional R^2	0.109 / 0.667		

Table 7. Regression Results for the Effect of Genetic Ancestry on *g* in the Hispanic-ancestry Sample (*N* =813)

doi.org/10.32388/EO7DND

Figures 2 and 3 display the partial residual plots for the African and Amerindian ancestry PCs, respectively, in the Hispanic-ancestry sample. The loess regression lines in both plots are nearly linear. As in the African-ancestry sample, the observed difference between the extremes of the ancestry distribution was somewhat greater than 0.5 standard deviations— smaller than the model-implied full-range *B* coefficients because the observed ancestry range in these samples is censored.

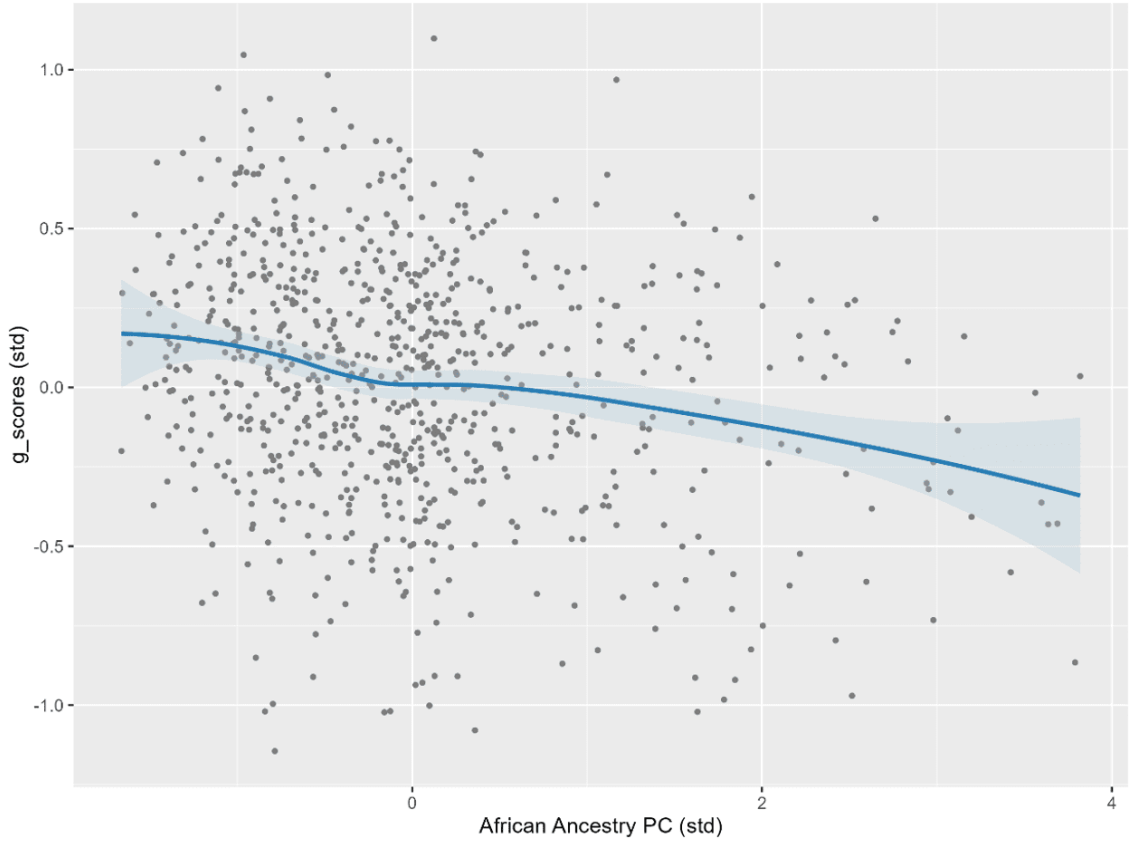

Figure 2. Partial Residuals and Estimated Regression Fits for African Ancestry PC in the Hispanic-ancestry Sample with *g* as the Dependent Variable. *Note: Loess line shown in dark blue with confidence interval shown in light blue.*

doi.org/10.32388/EO7DND

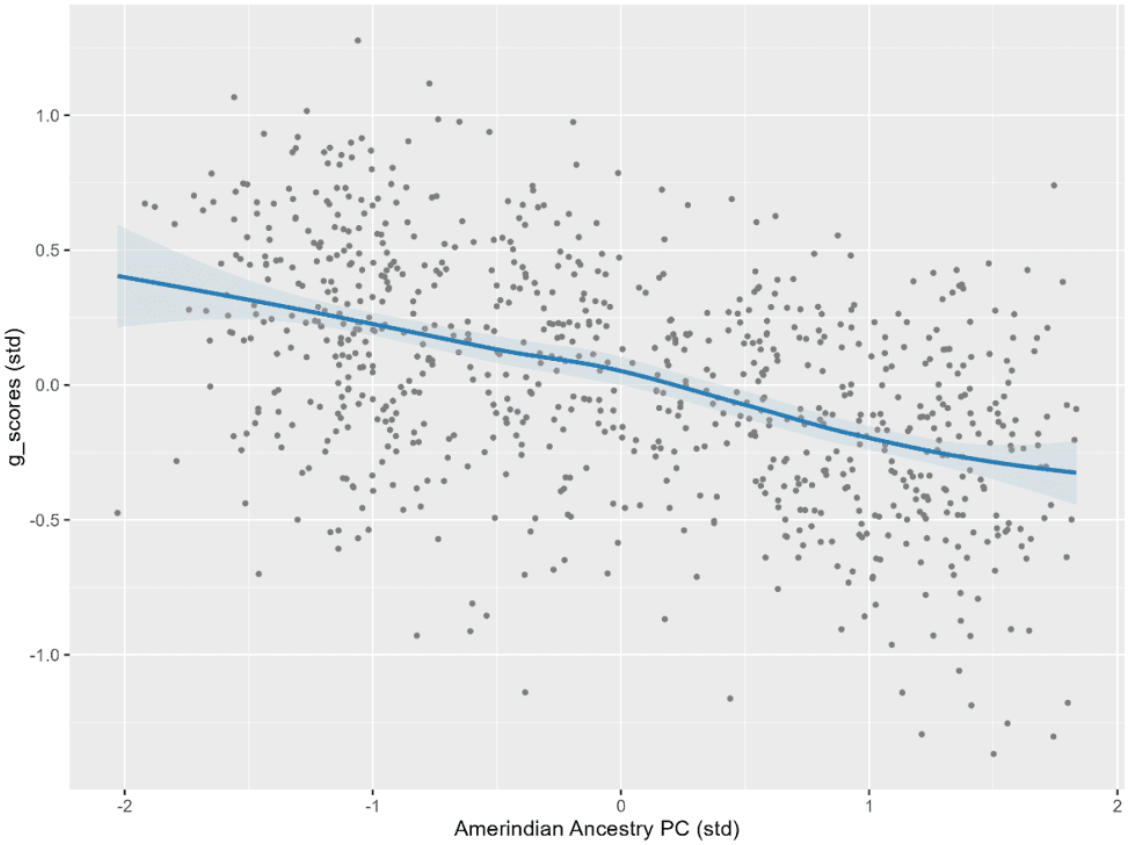

Figure 3. Partial Residuals and Estimated Regression Fits for Amerindian Ancestry PC in the Hispanic-ancestry Sample with *g* as the Dependent Variable (*N* =813). *Note: Loess line shown in dark blue with confidence interval shown in light blue.*

doi.org/10.32388/EO7DND

Ancestry group	Predictor	*g*	PVT1	PVT3	Digit Span	Delayed Recall	Education
African-ancestry	African ancestry PC (std.)	**-.147 (.023)**	**-0.131 (.023)**	**-0.150 (.026)**	-0.037 (0.023)	**-0.065 (0.023)**	**-0.091 (0.023)**
	N	1921	1847	1538	1914	1914	1921
	Implied African ancestry *B* (beta model)	**-1.36**	**-1.21**	**-1.39**	-0.34	**-0.60**	**-0.84**
Hispanic-ancestry	African ancestry PC (std.)	-0.084 (0.052)	-0.095 (0.053)	-0.112 (0.058)	0.072 (0.054)	-0.009 (0.054)	-0.033 (0.053)
	Amerindian ancestry PC (std.)	**-0.206 (0.052)**	**-0.191 (0.053)**	-0.062 (0.060)	**-0.211 (0.054)**	**-0.166 (0.054)**	**-0.120 (0.054)**
	N	813	788	656	812	811	813
	Implied African ancestry *B* (beta model)	-1.23	-1.39	-1.64	1.05	-0.13	-0.48
	Implied Amerindian ancestry *B* (beta model)	**-1.75**	**-1.62**	-0.53	**-1.79**	**-1.41**	**-1.02**

Table 8. Regression Coefficients from the Mixed-Effects Regression Models for Genetic Ancestry and Cognitive and Educational Variables for the African- and Hispanic-ancestry Samples

Note: The effects for the ancestry PCs are standardized, while the estimated ancestry B coefficients are rescaled, unstandardized estimates representing a model-implied shift from 0% to 100% ancestry. Metric invariance was not supported for the Amerindian PC, indicating that the g composite should be interpreted with caution as the meaning changed across the ancestry distribution. g scores were based on the ancestry group-based MGCFA model in which partial strict invariance across European and Hispanic groups held.

The European and African ancestry samples differed by approximately 81 percentage points in African ancestry (1% versus 82%) and by 1.17 *SD* in *g*. This observed difference is approximately 94% of what the within-group admixture regression would predict: given $B = -1.36$ for a shift from 0% to 100% African

ancestry, the expected difference is 0.81 × 1.36 = 1.10 SD. Similarly, the European and Hispanic ancestry samples differed by approximately 5 percentage points in African ancestry (1% versus 6%) and 25 percentage points in Amerindian ancestry (1% versus 26%), with an observed *g* difference of 0.76 SD. The admixture regression coefficients predict a difference of 0.25 × 1.75 + 0.05 × 1.23 = 0.50 SD, accounting for approximately 66% of the observed gap. Thus, the between-group differences in *g* across European and African ancestry samples are mostly accounted for by the ancestry–cognition associations estimated within the admixed samples, while this is only partially the case for the European and Hispanic ancestry samples.

3.3. Global Ancestry and Cognitive Ability with SES controls

Table 9 summarizes the same analyses presented in Table 8, this time with parental socioeconomic status (SES) included as a covariate. Full model results are available in the Supplemental Material. Among the African-ancestry sample, the African principal component remained a statistically significant negative predictor of *g*, PVT1, PVT3, and delayed recall. However, the association between African ancestry and educational attainment was attenuated to statistical non-significance by family SES. Compared to the model estimated on the same subset of participants, the inclusion of parental SES attenuated the African ancestry–*g* association by 38% (from $B = -0.144$ to $B = -0.089$; $N = 1{,}908$). These results are consistent with previously reported findings (e.g., [31]). Among Hispanic-ancestry participants, the Amerindian principal component remained a significant negative predictor of *g*, PVT1, digit span backwards, delayed recall, and educational attainment. The inclusion of parental SES attenuated the Amerindian ancestry–*g* association by 20% (from $B = -0.202$ to $B = -0.161$; $N = 805$).

Ancestry	Predictor	*G*	PVT1	PVT3	Digit Span	Delayed Recall	Education
African-ancestry	African ancestry PC (std.)	**-0.089 (0.022)**	**-0.075 (0.022)**	**-0.102 (0.025)**	-0.002 (0.023)	**-0.047 (0.023)**	-0.024 (0.021)
	Parental SES	**0.348 (0.022)**	**0.329 (0.022)**	**0.278 (0.025)**	**0.210 (0.023)**	**0.125 (0.023)**	**0.401 (0.021)**
	N	1908	1835	1526	1901	1901	1908
	Implied African Ancestry *B* (beta model)	**-0.82**	**-0.69**	**-0.94**	-0.02	**-0.43**	-0.22
Hispanic-ancestry	African ancestry PC (std.)	-0.049 (0.051)	-0.061 (0.052)	-0.077 (0.058)	0.087 (0.054)	-0.005 (0.054)	0.006 (0.053)
	Amerindian ancestry PC (std.)	**-0.161 (0.052)**	**-0.153 (0.052)**	-0.015 (0.060)	**-0.186 (0.054)**	**-0.162 (0.054)**	-0.080 (0.053)
	Parental SES	**0.263 (0.040)**	**0.244 (0.040)**	**0.226 (0.045)**	**0.118 (0.041)**	0.040 (0.042)	**0.266 (0.041)**
	N	805	780	649	804	803	805
	Implied African Ancestry *B* (beta model)	-0.72	-0.89	-1.12	1.27	-0.07	0.09
	Implied Amerindian Ancestry *B* (beta model)	**-1.37**	**-1.30**	-0.13	**-1.58**	**-1.38**	-0.68

Table 9. Regression Coefficients from the Mixed-Effects Regression Models for Genetic Ancestry and Cognitive and Educational Variables for the African- and Hispanic-ancestry Samples with SES Included as a Covariate

Note: The effects for the ancestry PCs are standardized, while the estimated ancestry B coefficients are rescaled, unstandardized estimates representing a model-implied shift from 0% to 100% ancestry. Metric invariance was not supported for the Amerindian PC, indicating that the g composite should be interpreted with caution as the

meaning changed across the ancestry distribution. g scores were based on the ancestry group-based MGCFA model in which partial strict invariance across European and Hispanic groups held.

3.4. Global Ancestry and Cognitive Ability with Color Phenotype and Discrimination

Table 10 extends the analysis by adding interviewer-rated appearance and discrimination variables: skin color and self-reported experiences of racial or color discrimination for the African-ancestry sample, and skin, hair, and eye color for the Hispanic-ancestry sample. Among African-ancestry sample participants, lighter skin color was a significant positive predictor of PVT3 scores, and self-reported racial discrimination was a significant predictor of lower digit span backwards scores, even after adjusting for ancestry. Inclusion of these variables attenuated the African ancestry–*g* association by 8% (from $B = -0.123$ to $B = -0.113$; $N = 1{,}604$). This attenuation of the African ancestry–*g* coefficient was not statistically significant.

Among Hispanic-ancestry participants, hair and eye color were not significantly related to any cognitive or educational outcomes. However, lighter skin color was both a statistically and practically significant positive predictor of *g*, PVT1, PVT3, digit span backwards, and educational attainment. Inclusion of the skin, hair, and eye color variables attenuated the Amerindian ancestry–*g* association by 9% (from $B = -0.175$ to $B = -0.160$; $N = 679$). This attenuation of the Amerindian ancestry–*g* coefficient was statistically significant.

Overall, the results regarding color-related effects are mixed. Skin color showed independent associations with cognitive outcomes (particularly in the Hispanic-ancestry sample), but the ancestry effects remained largely intact in both groups. These findings thus do not provide strong support for the hypothesis that phenotype-related effects (which could include discrimination) account for ancestry–cognition associations.

Ancestry	Predictor	*G*	PVT1	PVT3	Digit Span	Delayed Recall	Education	
African-ancestry	African ancestry PC (std.)	**-0.113 (0.026)**	**-0.102 (0.027)**	**-0.109 (0.026)**	-0.029 (0.027)	-0.056 (0.026)	**-0.074 (0.026)**	
	Skin Color	0.042 (0.026)	0.020 (0.027)	**0.079 (0.026)**	0.021 (0.027)	0.017 (0.026)	0.028 (0.026)	
	Self-reported discrimination	0.012 (0.022)	0.015 (0.023)	0.020 (0.022)	**-0.061 (0.025)**	0.027 (0.024)	-0.008 (0.023)	
	N	1604	1530	1537	1599	1600	1604	
	Implied African Ancestry *B* (beta model)	**-1.04**	**-0.94**	**-1.01**	-0.27	-0.52	**-0.68**	
Hispanic-ancestry	African ancestry PC (std.)	-0.077 (0.057)	-0.089 (0.058)	-0.071 (0.059)	0.090 (0.059)	0.018 (0.059)	-0.014 (0.058)	
	Amerindian ancestry PC (std.)	**-0.160 (0.058)**	**-0.155 (0.059)**	-0.043 (0.060)	**-0.176 (0.059)**	**-0.155 (0.060)**	-0.107 (0.060)	
	Skin Color	**0.093 (0.039)**	**0.096 (0.040)**	0.056 (0.041)	**0.101 (0.041)**	0.052 (0.041)	**0.118 (0.040)**	
	Hair / Eye Color	0.014 (0.039)	-0.014 (0.040)	0.060 (0.041)	0.032 (0.040)	0.025 (0.041)	0.027 (0.040)	
	N	679	654	655	678	677	679	
	Implied African Ancestry *B* (beta model)	-1.12	-1.30	-1.04	1.31	0.26	-0.20	
	Implied Amerindian Ancestry *B* (beta model)	**-1.36**	**-1.32**	-0.37	-1.50	-1.32	-0.91	

Table 10. Regression Coefficients from the Mixed-Effects Regression Models for Genetic Ancestry and Cognitive and Educational Variables for the African- and Hispanic-ancestry Samples with Skin, Hair, Eye

Color and Self-reported Experiences of Discrimination as Covariates

Note: The effects for the ancestry PCs are standardized, while the estimated ancestry B coefficients are rescaled, unstandardized estimates representing a model-implied shift from 0% to 100% ancestry. Metric invariance was not supported for the Amerindian PC, indicating that the g composite should be interpreted with caution as the meaning changed across the ancestry distribution. g scores were based on the ancestry group-based MGCFA model in which partial strict invariance across European and Hispanic groups held.

3.5. Mediation of the Ancestry–Education Association by Cognitive Ability

An important question is whether cognitive ability mediates the association between genetic ancestry and educational attainment. The longitudinal design of Add Health provides a rare opportunity to test this pathway with partly prospective temporal ordering, especially for adolescent verbal ability measured at Wave I. Adolescent verbal ability (Wave I PVT) and the longitudinal general cognitive ability factor (*g*, spanning Waves I, III, and IV) were tested as mediators of the ancestry–educational attainment association (education measured primarily at Wave V, with Wave IV substituted when Wave V was missing). Results are summarized in Table 11, with full model output available in the Supplemental Material.

Adolescent verbal ability mediated 68% of the African ancestry–education association in the African-ancestry sample and 47% of the Amerindian ancestry–education association in the Hispanic-ancestry sample. Longitudinal *g* mediated 83% and 60% of the respective associations. These results indicate that ancestry-associated differences in adult educational attainment are substantially statistically mediated by differences in cognitive ability, including ability measured as early as adolescence.

Predictor	Mediator	Criterion	African-ancestry sample			Hispanic-ancestry sample		
			N	Total Effect	% Mediated	*N*	Total Effect	% Mediated
African Ancestry PC (std.)	PVT1	Education	1847	**-0.096**	**68.21%**			
African Ancestry PC (std.)	*G*	Education	1921	**-0.093**	**83.13%**			
African Ancestry PC (std.)	PVT1	Education				788	-0.030	49.31%
African Ancestry PC (std.)	*G*	Education				813	-0.032	52.55%
Amerindian Ancestry PC (std.)	PVT1	Education				788	**-0.144**	**47.31%**
Amerindian Ancestry PC (std.)	*G*	Education				813	**-0.138**	**59.90%**

Table 11. Summary of the Mediation Results for the African- and Hispanic-ancestry samples

Notes: Total effects are standardized. Bolded entries indicate statistically significant mediation estimates (in this case, p < .001); exact p-values and confidence intervals are reported in the Supplemental Material.

3.6. Genetic–Environmental Uncertainty

Since ancestry-associated genetic effects depend on within-ancestry-group heritability [26], we can evaluate whether a partial genetic model is plausible by assessing if our outcomes are genetically influenced. PVT1 and PVT3 have been reported to have a similar kinship-based heritability in both the Black and the White Americans Add Health samples [46] and so we would expect polygenic effects to be present. We ran a series of regression models with genetic *g* as a predictor of outcomes. Results are summarized in Table 12. Genetic *g* significantly predicted all outcomes except delayed recall in the African-ancestry sample and all outcomes except parental SES in the Hispanic-ancestry sample.

In the African-ancestry sample, genetic *g* predicted parental SES about as strongly as it predicted *g*, suggesting that controlling for SES in this group partly captures genetic variance. This was not the case in the Hispanic-ancestry sample, where genetic *g* was unrelated to parental SES. These results show that

parental SES is itself predicted by both ancestry and—in the African-ancestry sample—polygenic *g*, meaning parental SES cannot be automatically assumed to be an exogenous environmental variable. The SES-adjusted ancestry–cognition coefficients in the main text should therefore be interpreted as conservative lower-bound estimates, with the unadjusted coefficients representing the total association across all pathways, including those operating through intergenerationally accumulated socioeconomic position.

	African-ancestry sample		**Hispanic-ancestry sample**	
Dependent	*N*	*β*	*N*	*β*
G	1921	**0.149**	813	**0.163**
PVT1	1847	**0.161**	788	**0.138**
PVT3	1538	**0.094**	656	**0.133**
Digit Span	1914	**0.090**	812	**0.153**
Delayed Recall	1914	0.018	811	**0.095**
Education	1921	**0.094**	813	**0.161**
Parental SES	1776	**0.130**	745	0.013

Table 12. Mixed-Effects Regression of Genetic *g* on Cognitive Ability, Educational Attainment, and Parental SES in the African- and Hispanic-ancestry Samples

Notes: Statistically significant effects reported in bold.

3.7. Supplemental results

We conducted several robustness checks, rerunning the *g* models with the following modifications: (1) restricting the sample to third-generation-or-later respondents; (2) using school ID rather than geographic region as the clustering variable; and (3) adding physical attractiveness and, for the Hispanic-ancestry sample, interviewer-observed race to the color and discrimination models. Results are summarized in Table 13.

Excluding second-generation immigrants slightly increased the ancestry PC–*g* associations. Substituting school for geographic region had no substantial effect on results in the Hispanic-ancestry sample, but doing so reduced the African PC–*g* association by 27% in the African-ancestry sample, though interpretation is complicated by the non-random sorting of students into schools in the United States. Adding physical attractiveness and observed race further attenuated the ancestry associations by 4% (African PC in the African-ancestry sample: from B = -0.113 to -0.109) and 2% (Amerindian PC in the Hispanic-ancestry sample: from B = -0.160 to -0.157) relative to the model results with discrimination and hair, eye, and skin color. Thus, although physical attractiveness was itself strongly related to *g*, its inclusion had minimal additional impact on the ancestry–cognition association.

Ancestry	Model	Amerindian *β*	*SE*	African *β*	*SE*	*N*
African-ancestry	3rd+ generation			-0.152	0.024	1,825
African-ancestry	School (PSUSCID)			-0.107	0.022	1921
African-ancestry	Add Physical attractiveness			-0.109	0.025	1604
Hispanic-ancestry	3rd+ generation	-0.200	0.074	-0.116	0.085	367
Hispanic-ancestry	School (PSUSCID)	-0.200	0.052	-0.078	0.051	813
Hispanic-ancestry	Add Physical attractiveness + observed race	-0.157	0.058	-0.085	0.056	679

Table 13. Mixed-Effects Regression of Genetic Ancestry on *g* Robustness Results

3. Discussion

Several previous admixture regression studies have reported that African and Amerindian ancestry tend to be negatively associated with cognitive ability in American admixed populations such as African Americans and Hispanic Americans [31]. These studies report that the associations between genetic ancestry and cognitive ability are robust to controls for parental SES and for genetically predicted skin, eye, and hair color. Using data from Add Health, we replicate and extend these past results.

We first evaluated whether at least scalar measurement invariance held across genetic ancestry samples and across the ancestry continuum within admixed samples. Between-group MGCFA across SIRE-

defined ancestry groups indicated partial strict invariance, with only PVT residual variances requiring release. We then tested whether measurement properties were stable across the continuous ancestry gradient within each admixed sample, using both discrete MGCFA across ancestry PC tertiles and LSEM progressive invariance testing with bootstrap confidence bands.

These within-group analyses revealed an asymmetry across ancestry components. In the African-ancestry sample, all invariance levels held across the African ancestry continuum: factor loadings, intercepts, and residual variances were stable, confirming that *g* has equivalent measurement properties across the full range of African admixture. In the Hispanic-ancestry sample, invariance likewise held across the African ancestry PC. However, metric invariance failed across the Amerindian ancestry PC in both analytic approaches. The instability was concentrated in the vocabulary measure, whose loading on g increased substantially at higher levels of Amerindian ancestry, while non-verbal indicators contributed less. At the high-Amerindian end of the continuum, the *g* factor was increasingly defined by vocabulary rather than a balanced composite of diverse cognitive abilities. Intercepts, by contrast, did not shift — the problem was one of differential test weighting rather than systematic item bias.

This finding complicates the interpretation of *g* score associations with Amerindian ancestry, though the *g* scores used in the regression models were extracted from the between-group MGCFA, for which partial strict invariance held. The Amerindian ancestry association was also not limited to verbal tests — delayed recall and digit span backwards showed significant negative associations as well — suggesting the pattern is not entirely attributable to the loading instability. Nevertheless, individual test results are presented alongside *g* for the Amerindian component and should be considered the primary analysis.

The metric invariance failure is surprising given that strict invariance held between the European and Hispanic SIRE groups and that all individuals were US-born. One possible explanation is that the ancestry group-based test compares broad racial/ethnic groups that differ in many measured and unmeasured ways, allowing compensating biases to cancel at the group level. The within-group test, by contrast, isolates genetic ancestry variation within a single ethnic group while holding most cultural and demographic factors roughly constant, providing a more sensitive test of whether measurement properties track ancestry itself. These results demonstrate that testing measurement invariance between SIRE groups alone is not sufficient when conducting admixture regression with general cognitive ability; invariance must also be evaluated across the continuous ancestry gradient within admixed samples.

With the exception of delayed recall in the African ancestry sample, our cognitive and educational measures were significantly predicted by polygenic *g* within both the African- and Hispanic-ancestry

samples and parental SES was also predicted by polygenic *g* in the African-ancestry sample, consistent with the within-group heritability that a partial genetic model requires.

Within the African-ancestry sample, the African ancestry PC predicted PVT1 (adolescence), PVT3 (adulthood), delayed recall (adulthood), educational attainment (adulthood), and longitudinal *g*. Within the Hispanic-ancestry group, the Amerindian ancestry PC predicted PVT1, digit span backwards, delayed recall, and *g*. The African ancestry PC among Hispanics was not a statistically significant predictor of cognitive or education outcomes, owing to the minimal African variation in this sample (*SD* = 0.069). Although the model-implied full-range effects (*B*) are typically large (e.g., *B* = -1.75 for Amerindian ancestry in the Hispanic-ancestry sample), the actual observed differences between individuals at the extremes of the (restricted) ancestry distributions in these samples were more modest (see Figures 1-3).

Parental SES accounted for 38% of the African PC–*g* association in the African-ancestry sample and 20% of the Amerindian PC–*g* association in the Hispanic-ancestry sample. Among the Hispanic-ancestry sample, parental SES was unrelated to polygenic *g*, suggesting it may function as a more nearly exogenous environmental variable in this group. The picture is less clear among the African-ancestry sample, where polygenic *g* predicted parental SES about as strongly as it predicted cognitive ability, raising the possibility that SES controls partly absorb genetic variance.

Neither interviewer-rated skin color nor self-reported discrimination was significantly related to *g* in the African-ancestry sample, though skin color was related to PVT3 and self-reported discrimination was related to delayed recall. Including these variables attenuated the ancestry–*g* association by only 8%. In the Hispanic-ancestry sample, hair and eye color were unrelated to outcomes, but skin color was a significant predictor of most cognitive and educational measures, including g. Nonetheless, including color variables attenuated the Amerindian ancestry–g association by only 9%. Since the PC-based ancestry index is a noisier measure of true ancestry in the Hispanic sample than in the African-ancestry sample, some genuine ancestry variance may have been captured by the color variables, meaning that the 9% attenuation possibly overstates the independent contribution of color-related effects.

In short, the ancestry–*g* associations were largely unaffected by either phenotypic color or self-reported racial discrimination, providing limited support for a colorism model in which phenotype-based discrimination is the primary driver of the ancestry–cognition association.

Mediation analyses demonstrated that cognitive ability accounted for 68-83% of the African ancestry–education association among the African-ancestry sample and 47-60% of the Amerindian ancestry–education association among the Hispanic-ancestry sample. These results provide, to our knowledge, the

first longitudinal evidence that cognitive ability statistically mediates a substantial share of the association between ancestry and educational attainment.

Supplemental analyses indicate that results are largely stable when restricting the sample to third-generation-or-later individuals, when using school ID as a clustering variable, and when including physical attractiveness and, for the Hispanic-ancestry sample, overall racial appearance as additional covariates, with the exception that using school clustering instead of geographic clustering attenuated the association between African PC and *g* in the African-ancestry sample by around 30%. Interpretation is complicated, as school attendance in the United States is socioeconomically assortative. Using a larger number of demographic controls, Guo et al. report, in their preprint, associations between the African and Amerindian ancestry PCs and verbal ability among African, Hispanic, and White Americans [32]. Therefore, it seems unlikely that adding further demographic controls would substantially change the results.

These findings extend previous work by demonstrating that the ancestry–cognition association persists into adulthood and that cognitive ability substantially statistically mediates ancestry-associated differences in later-life educational attainment. Given the robust cognitive epidemiology literature linking cognitive ability to health outcomes, it may be worthwhile to investigate whether cognitive ability also mediates ancestry-associated differences in disease risk — a hypothesis with clear potential implications for health disparities research.

In genetic epidemiology, a robust association between genetic ancestry and a heritable trait is commonly interpreted as suggestive evidence of genetically based group differences. However, we frame our findings within a more general vertical transmission model, which encompasses both genetic and cultural inheritance, and leave the decomposition of transmission mechanisms to future work. Local ancestry analyses — which can distinguish genomic segments by ancestral origin and test whether ancestry effects are distributed across the genome — offer a promising avenue for adjudicating between genetic and non-genetic vertical-transmission explanations.

An alternative interpretation to the vertical transmission model is that contemporaneous phenotypic discrimination accounts for the observed continental ancestry–cognitive ability associations. Proponents of the colorism hypothesis "place primacy on the causal role of skin tone" [47], and so we focused on this variable. However, it is possible that our measures incompletely capture the full range of color-based discrimination. Discrimination may be based on a gestalt phenotype that closely tracks genotype. For example, Taddess et al. [48] report that genetic ancestry predicts physical attractiveness in

the Add Health sample, even controlling for skin color. We consider a strong colorism hypothesis — according to which most of the ancestry–cognitive ability association is accounted for by phenotype-based discrimination—unlikely for several reasons, including that ancestry is associated with cognitive and SES outcomes at the national and regional level and that the associations are evident at young ages (e.g., age 10 in the Adolescent Brain Cognitive Development Study sample [31]). Nonetheless, if phenotypic discrimination closely tracked continental genetic ancestry rather than stereotypical race-associated phenotypes such as skin color, this would be an important finding for the colorism research program. This hypothesis could be investigated more rigorously with better composite scales of racial phenotype designed for tri-racially admixed populations, as well as through local ancestry analyses—specifically, by testing whether local ancestry at particular genomic regions contributes disproportionately to the association between global ancestry and cognitive or educational outcomes.

In this paper, we use genetic ancestry to index persistent intergenerational transmission. A growing body of literature examines family-lineage effects on socioeconomic outcomes within populations (e.g., [24] [25]). We extend this approach to admixed populations, where continental ancestry proportions provide a natural marker of lineage depth — individuals with higher African or Amerindian ancestry have, on average, more generations of ancestors drawn from those source populations and fewer from the European source population. Our theoretical model does not assume a specific mechanism of transmission; the ancestry gradient may reflect genetic, cultural, epigenetic, or other intergenerational pathways, and disentangling their relative contributions is a task for future research. Critically, ancestry in this framework does not index continental populations in the abstract — it indexes lineage within a specific admixed American population with a particular historical trajectory. The results reflect whatever has been persistently transmitted across generations within the African American and Hispanic American communities, shaped by the specific conditions of those communities′ formation and continuation. A sample drawn from a different population with similar continental ancestry proportions but a different historical trajectory could yield entirely different results. The findings therefore do not generalize beyond these admixed American populations.

A key limitation is the study′s dependence on ancestry PCs from the Add Health subsample with precomputed polygenic scores. The PSANCEST classification procedure, which selects individuals falling within a narrow PC-space window of ±1/2 *SD* around each SIRE groups mean, excluded a large share of self-identified Hispanic respondents whose PC values fell outside this window, substantially reducing the Hispanic-ancestry analytic sample. In addition, because within-group PCs were computed separately for

each PSANCEST-defined ancestry group, the resulting PC axes are not directly comparable across groups, precluding the merged-sample analytic strategies employed in previous admixture regression studies. Finally, while the within-group PCs are very strongly correlated with ADMIXTURE-derived continental ancestry proportions (r = .933–.989), they are not exact measures of ancestry. As detailed in the Supplemental Material, this imperfection introduces offsetting biases in the rescaled ancestry coefficients — a modest net overestimation of approximately 6% for the Amerindian component and negligible net bias for the African component. Moreover, the rescaling to B is dependent on a derived estimate of the ancestry SD, potentially introducing inaccuracies. While examining the relationship between ancestry and outcomes within relatively ancestrally homogeneous groups offers some analytic benefits — in particular, narrowing phenotypic and likely sociocultural variation — analyzing the full spread of ancestry variation in the combined samples would allow for more thorough analyses, including nonlinear modeling and direct comparisons of within- and between-group ancestry gradients.

Supplementary Material

A supplemental file (including full model output, robustness checks, R code, and additional tables) is available online with this article: here.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability

The data used in this study are available from the Carolina Population Center at the University of North Carolina at Chapel Hill.

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