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ORIGINAL RESEARCH

A Longitudinal Analysis of Gene x Environment Interaction on Verbal Intelligence Across Adolescence and Early Adulthood

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Abstract
The Scarr-Rowe hypothesis proposes that the heritability of intelligence is higher in more advantaged socioeconomic contexts. An early demonstration of this hypothesis was Rowe and colleagues (Rowe et al., Child Dev 70:1151–1162, 1999), where an interaction between the heritability of verbal intelligence and parental education was identified in adolescent siblings in Wave I of the National Longitudinal Study of Adolescent to Adult Health. The present study repeated their original analysis at Wave I using contemporary methods, replicated the finding during young adulthood at Wave III, and analyzed the interaction longitudinally utilizing multiple measurements. We examined parental education, family income, and peer academic environment as potential moderators. Results indicated increased heritability and decreased shared environmental variance of verbal intelligence at higher levels of parental education and peer academic environment in adolescence. Moreover, moderation by peer academic environment persisted into adulthood with its effect partially attributable to novel gene-environment interactions that arose in the process of cognitive development.

Keywords Cognitive development · Verbal intelligence · Heritability · Gene-environment interaction · Socioeconomic status · Academic environment

Introduction

The heritability of intelligence has been demonstrated to be substantial in twin studies (Bouchard & McGue 1981; Sundet et al. 1994) and, though somewhat smaller in magnitude, in genome-wide association studies (Davies et al. 2011; Plomin et al. 2013). Verbal intelligence, in particular, was found to be heritable (Rijsdijk et al. 2002). However, interpretation of a high heritability estimate is notoriously difficult (Devlin et al. 1997). Notwithstanding the heritability of intelligence, environmental factors also have undeniable influences on cognitive development (Bartels et al. 2002), as evidenced by the Flynn Effect—a substantial increase in intelligence scores across generations (Bouchard & McGue 2003)—and by compromised cognitive functions of children mistreated in Romanian orphanages (Morison & Ellwood 2000). Moreover, environmental factors must be taken into consideration when estimating the heritability of intelligence, as past twin studies have found that the heritability estimates are complicated by various gene-environment correlations and interactions (Manuck & McCaffery 2014).

One prominent consequence of gene-environment correlation and interaction on cognitive development is the Wilson Effect (Bouchard 2013), in which the heritability of intelligence increases across age. A meta-analysis of 6 twin studies by Haworth and colleagues (2010) demonstrated that the heritability of general cognitive ability

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increased from 41% in childhood, to 55% in adolescence, to 66% in young adulthood, and that the effects of shared environmental influences diminished across these developmental stages. The Wilson effect has been replicated in another meta-analysis of 11 unique longitudinal samples (Briley & Tucker-Drob 2013) and specifically for verbal intelligence in a longitudinal study (Van Soelen et al. 2011). These findings on the age-related changes in heritability estimates stress the importance of examining the heritability of intelligence longitudinally across different stages of cognitive development.

In addition to changes in relation to age, there is evidence that the heritability of intelligence varies across socioeconomic contexts (Bronfenbrenner & Ceci 1994; Scarr 1992). Named after Sandra Scarr and David Rowe (Turkheimer et al. 2009), the Scarr-Rowe interaction is another strong indication that heritability, rather than being a fixed parameter, is a relative quantity that fluctuates across different populations and different environments. Specifically, the Scarr-Rowe hypothesis proposes that with higher socioeconomic status, either the intelligence of monozygotic twins (MZ) becomes more correlated, or that dizygotic twins (DZ) become less correlated, or both. Recent work has suggested that changes in the DZ correlation are often more prominent (Giangrande et al. 2019; Turkheimer et al. 2017). Scarr-Salapatek first identified this phenomenon in a sample of twins from the Philadelphia school system, that socioeconomic status estimated from census-tract information modified the heritability of standardized ability test scores (Scarr-Salapatek 1971). After a considerable hiatus, Rowe, Jacobson and Van den Oord (1999) demonstrated an interaction between parental education and the heritability of verbal intelligence, as measured by Peabody Picture Vocabulary Test (PPVT), using Wave I of the National Longitudinal Study of Adolescent to Adult Health (Add Health), at which time the participants were adolescents. Heritability of verbal intelligence was higher for adolescents whose parents were more educated. Notably, only two waves of data and one PPVT were available in Add Health when Rowe et al. (1999) conducted their analysis. Add Health siblings have now been followed with later waves of data collection continuing into adulthood.

Studies since Rowe et al. (1999) have generally supported the Scarr-Rowe hypothesis in North America. Turkheimer et al. (2003) demonstrated a strong interaction effect in 7-year-old twins from the National Collaborative Perinatal Project. Several other studies have also generated results that add support to the Scarr-Rowe hypothesis for different cognitive aspects and across much of the life span, from childhood to middle adulthood (Bates et al. 2013; Friend et al. 2008; Harden et al 2007; Rhemtulla & Tucker-Drob 2012; Tucker-Drob et al. 2010). Moreover, the Scarr-Rowe interaction on cognitive abilities has been meta-analytically documented among samples of children in the United States (Tucker-Drob & Bates 2016).

Inconsistency in the Scarr-Rowe Literature

Some other studies, however, have obtained less conclusive or contradictory results (Asbury et al. 2005; Hanscombe et al. 2012; Kremen et al. 2005; Spengler et al. 2018; Van der Sluis et al. 2008). There have been several proposed explanations for replication failures in these studies. One is that most studies of the Scarr-Rowe effect have been underpowered (Van der Sluis et al. 2012). Many studies seeking to replicate the Scarr-Rowe hypothesis included only monozygotic and dizygotic twins, contributing to relatively small sample sizes. With a classical twin model, 2,500 pairs of monozygotic and dizygotic twins are needed to detect a typical medium-sized Scarr-Rowe effect with 80% power (Hanscombe et al. 2012). Very few studies that tested the Scarr-Rowe effect with twin pairs even approximated such sample sizes (Tucker-Drob & Bates 2016). Therefore, it is potentially beneficial to include more types of genetically-informative pairs (e.g., non-twin siblings, cousins, adoptees, unrelated coresidents, etc.) in analyses to amplify statistical power (Kirkpatrick et al. 2015; Rowe et al. 1999). Moreover, including these genetic pairs broadens the scope of the relationship to which the Scarr-Rowe can be applied: one recent study has included adoptive families as a way of examining the Scarr-Rowe interaction in contexts broader than only MZ and DZ twins (Loehlin et al. 2022). Siblings and adoptees have also provided valuable insights into other forms of gene-environment interplay in quantitative genetic and social genomic studies (Domingue & Fletcher 2020; Harden et al. 2008; Lau & Eley 2008; Visscher et al. 2006).

A second reason for variability in the Scarr-Rowe interaction is that gene × SES interactions may differ in strength across different populations or societies. Studies of the Scarr-Rowe effect have failed to replicate in Australian and European samples (Asbury et al. 2005; Bates et al. 2016; Spengler et al. 2018; Van der Sluis et al. 2008). The cross-national difference theory of gene × SES interaction posits that the difficulty replicating the Scarr-Rowe effect in these countries might be due to a higher availability of enriching or protective environmental factors (e.g., quality education, healthcare, social mobility) in their societies (Tucker-Drob & Bates 2016). However, this theory does not explain why some studies in the United States have also failed to replicate the Scarr-Rowe effect with sizeable or nationally-representative datasets (Figlio et al. 2017; Grant et al. 2010; Van den Oord and Rowe 1997). Further investigation of the Scarr-Rowe effect with U.S. samples is necessitated to better

understand the conditions under which socioeconomic environment moderates the heritability of intelligence.

A third explanation for the inconsistency in current literature is that replication of the Scarr-Rowe effect might depend on the age group of the population studied. While much evidence supporting the Scarr-Rowe hypothesis are from studies of children and adolescents, many analyses that found partial or no support for the Scarr-Rowe effect used samples that mainly consisted of adults (Kremen et al. 2005; Van der Sluis et al. 2008). As the heritability of cognitive functions increases and shared environmental influence decreases as individuals age (the Wilson Effect), developmental differences in gene-environment interactions might exist across age, such that the moderation effect by SES in adulthood is masked by the moderation by age. Indeed, it has been suggested that the heritability × age interaction and the heritability × SES interaction on cognition might share the same underlying transactional mechanism, in which genetically influenced differences within families select siblings into particular environments that further reinforce the genetic influence on their cognitive phenotypes (active gene-environment correlation; Tucker-Drob et al. 2013; Turkheimer & Horn 2014).

To better understand the processes underlying gene-environment interactions on cognition, more evidence is needed from studies that examine genetic and environmental contributions to intelligence longitudinally. Giangrande et al. (2019) studied the Scarr-Rowe effect with a sample of twins from middle childhood to early adolescence (age 7—15) in the Louisville Twin Study. Although a gene × SES interaction was observed cross-sectionally, no significant moderation effect on the heritability of longitudinal change in intelligence was found in that study. However, insignificant results might be due to limited statistical power afforded by a small sample of twins and their use of age-standardized scores to measure cognitive abilities. It would be meaningful to study the Scarr-Rowe effect longitudinally with a larger sample that captures a diverse range of family SES. Further investigation of the gene × SES interaction with longitudinal designs is warranted to elucidate the age-related stability of the Scarr-Rowe effect.

Finally, socioeconomic status (SES) is a very different construct in adulthood than in childhood. While childhood parental SES is a family-level exogenous variable imposed equally on siblings raised together, adulthood SES may partially be established and maintained by the intelligence and other personal characteristics of the adults themselves (Conger et al. 2010). Several studies of the Scarr-Rowe effect using attained SES in adulthood as the moderator (as opposed to rearing SES) have found the interaction to be insignificant or in the wrong direction (Van der Sluis et al. 2008; Zavala et al. 2018).

School environment as an alternative moderator

Another complication in the Scarr-Rowe literature is that the definition of both the outcome and the moderator in gene × SES interaction on intelligence has been variable. Although the Scarr-Rowe effect is usually defined as an interaction between socioeconomic status and intelligence, current literature has reported positive results with a range of cognitive measures (school achievement, reading ability, aptitude test scores) and environmental variables (parental education, environmental risk, family income, composite SES, home environment). Therefore, it is possible that the Scarr-Rowe effect are better characterized as an interaction with environmental aspects other than SES. Indeed, as Bates and colleagues (2016) have argued, causal factors underlying the Scarr-Rowe interaction in U.S. families are unclear, and the non-replication of the Scarr-Rowe effect in samples outside of the United States suggests that such causal environmental inputs might be decoupled from SES measures in non-U.S. populations. Identifying such non-SES aspects of the environment that magnifies genetic differences in intellectual development is apparently of importance.

School is a potentially important environmental factor that contributes to individual differences in cognitive development. Educational attainment is strongly and positively correlated with intelligence (Barber 2005; Neisser et al. 1996; Strenze 2007). Studies using quasi-experimental methodologies have shown the causal effect of education on cognition (Araujo et al. 2016; Brinch and Galloway 2011; Ritchie and Tucker-Drob 2018). Particularly, educational attainment affects cognitive abilities like verbal ability, verbal memory, and verbal fluency throughout the life course (Hatch et al. 2007). Findings by Tucker-Drob (2012) that attending pre-school reduces SES-linked achievement differentials suggest that differences in the expression of genetic potential in intellectual capacity could also be potentially attributable to schooling. Furthermore, as suggested by Hogan & Tudge (1999), peers play a significant role in one's educational achievement (Patacchini et al. 2017; Zimmer & Toma 2000). Classmate characteristics and achievements have specifically been found to affect cognitive development and performances (Bifulco et al. 2008; Hanushek et al. 2003; Hoxby 2000; Peetsma, 2007). Few studies have investigated the role of school/peer environment in the context of the Scarr-Rowe interaction. Hart et al. (2013) explored the moderation effect of a school-level environmental variable on cognitive development, but that variable still measured SES, rather than actual school provisions. To our knowledge, no

study has yet directly examined the effect of schoolmate characteristics on genetic and environmental influences on cognitive outcomes.

The current study

The current study examined gene-environment interactions on verbal intelligence in adolescence and early adulthood. Our analyses were based on a nationally representative sample of twins, siblings, cousins, and unrelated coresidents from the National Longitudinal Study of Adolescent to Adult Health, the dataset originally used by Rowe et al (1999) in their seminal study on the Scarr-Rowe interaction. First, using a cross-sectional model, we repeated the Rowe et al. (1999) analysis in adolescence using new methods and replicated it in early adulthood using data that have become available since Rowe et al. (1999). Second, a bivariate longitudinal model was specified to assess the Scarr-Rowe interaction on change in verbal intelligence from adolescence to early adulthood. Our analyses were based on a novel model, expanding on the Modified Twin Correlation Model (MTCM, Turkheimer et al. 2017) to include longitudinal designs in sibling pairs that are not limited to identical and fraternal twins, and to test for gene-environment interaction while controlling for heteroscedasticity in the outcome variable. By including pairs with varying degrees of genetic relatedness and analyzing multiple measurements in bivariate models, we expect the study to have improved statistical power to detect gene-environment interaction effects on verbal intelligence.

The present study investigated the moderation effects of three environmental indices that were measured in adolescence. First, parental education was studied as a replication of Rowe et al. (1999). Second, we examined the interaction with family income, as income level is putatively an important factor in the socioeconomic standing of a family. Third, we explored the role of peer academic environment as a factor in cognitive development that does not directly measure economic resources. We expect the environmental variables measured in adolescence to better reflect shared rearing environment that was imposed on the sibling pairs. Moreover, we hypothesized that an education-related environmental factor, as measured by the peer academic environment variable, will capture the gene-environment interaction on cognition equally well or even better in comparing to the measures of SES. The study design and all hypotheses were preregistered on Open Science Framework at https://osf.io/dwub5.

Methods

Samples

The sample was drawn from the genetic sample in the National Longitudinal Study of Adolescent to Adult Health (Add Health), a database originally used by Rowe et al. (1999), in which they found a significant Scarr-Rowe effect at Wave I. Add Health is a longitudinal study of a nationally representative sample of adolescents who were in grades 7–12 during the 1994–95 school year; the participants have been followed through adolescence into adulthood for five waves of data collection to date (Harris 2013). Schools are the basic sampling units in Add Health. Initially, a sample of over 90,000 adolescents was drafted and administered in-school surveys from a stratified set of qualifying high schools. 20,745 adolescents, including oversampled subgroups from underrepresented populations, were then randomly selected from the previous school sample and administered the detailed in-home interview at Wave I. In 2001 and 2002, Add Health conducted Wave III data collection on the original Wave I participants who were then of age 18–26 and transitioning into adulthood. 15,170 respondents completed interviews at Wave III, resulting in a 76% response rate. Cognitive ability of the participants was measured with an abridged version of the Peabody Picture Vocabulary Test at both Wave I and Wave III. The Add Health genetic sample is an oversample of more than 3,000 pairs of individuals of varying genetic relatedness, including monozygotic twins, dizygotic twins, full-siblings, half-siblings, cousins, and unrelated individuals living in the same household (Harris et al. 2013). The genetic sample has a high retention rate (90.3% at Wave III). Add Health sibling pairs are unique in that they are nationally representative of the United States, racially and ethnically diverse, and have comprehensive social, environmental, behavioral, and biological longitudinal data from early adolescence into adulthood (Harris et al. 2013).

The full genetic sample consists of 3,139 pairs with a mean age of 22.3 at Wave III. They are 49.8% female, 52.4% white, and include 289 monozygotic pairs, 452 dizygotic pairs, 1,251 full-sibling pairs, 442 half-sibling pairs, 201 cousin pairs, 408 unrelated pairs (i.e.: step siblings, adoptive/foster children, some not related individuals living in the same household), and 96 other pairs (i.e., aunt/uncle-niece/nephew pairs, group home pairs, spouse pairs, and in-law pairs). The 96 other pairs were excluded from the current study due to difficulty in accurately characterizing

Table 1 Demographics and Sample Statistics of the Full Genetic Sample and Three Subsamples

Variables	Full genetic sample	Parental education sample	Family income sample	Peer academic environment sample
n (pairs)	3139	2913	2322	2113
N (individuals)[a]	5512	5144	4121	3838
Mean Age at Wave III	22.29 (1.82)	22.27 (1.81)	22.24 (1.79)	22.46 (1.74)
Biological Sex				
Female	49.8%	49.5%	49.3%	49.0%
Male	49.4%	49.8%	50.2%	51.0%
Other/Missing	0.8%	0.7%	0.4%	0.0%
Race/Ethnicity				
Non-Hispanic White	52.4%	53.7%	56.4%	55.0%
Native/African American	24.2%	23.5%	23.1%	21.0%
Asian/Pacific Islander	6.3%	6.0%	6.3%	7.3%
Hispanic	15.2%	14.9%	15.2%	15.7%
Other/Missing	1.9%	1.8%	1.9%	1.1%
PPVT				
Wave I	97.19 (14.96)	97.79 (14.23)	98.59 (13.98)	98.05 (14.21)
Wave III	96.96 (17.03)	98.34 (13.50)	98.94 (13.24)	98.85 (13.39)
Sibling Pair Type				
MZ[b]	289	276	210	265
DZ[c]	452	433	351	381
FS[d]	1251	1214	973	894
HS[e]	442	419	336	250
CO[f]	201	184	141	95
UR[g]	408	387	311	228
Other[h]	96	N/A	N/A	N/A

[a]The number of individuals (N) is not exactly twice the number of pairs (n) because in families with multiple siblings, all possible sibling pairs were made, and some individuals were double-counted

[b]Monozygotic twins

[c]Dizygotic twins

[d]Full-siblings

[e]Half-siblings

[f]Cousins

[g]Unrelated coresident pairs

[h]Undetermined and other pairs

their genetic relatedness and family environment. Pairs with non-missing information on the moderator (parental education, family income, or peer academic environment) were selected from the full genetic sample for moderation analysis with that specific moderator, forming three distinct subsamples: one for each of the three moderators included in this study. Due to our focus on typical cognitive development, outliers with PPVT scores lower than 50 were dropped from our sample. This is because PPVT scores correspond to standard IQ scores with M = 100 and SD = 15, and scores lower than 50 represent non-ordinary cognitive performance. In families with multiple siblings, all possible sibling pairs were made (e.g., three siblings in a family form three pairs: 1–2, 1–3, and 2–3). Demographic information for the full genetic sample and three subsamples is reported in Table 1.

Genetic relatedness

A sibling grouping variable in Add Health classifies sibling and non-sibling pairs into groups of monozygotic twins, dizygotic twins, full-siblings, half-siblings, cousins, and unrelated coresident pairs. A genetic relatedness variable (r_G) was assigned to twin, sibling, cousin, and unrelated pairs to express their average percentage of shared DNA:

MZ = 1.0, DZ = 0.5, FS = 0.5, HS = 0.25, CO = 0.125, UR = 0.00 (Lange 2002).

Verbal intelligence

Verbal intelligence was measured at Wave I and Wave III by the Add Health Peabody Picture Vocabulary Test (PPVT), an abridged version of the Peabody Picture Vocabulary Test–Revised (PPVT-R). Scores on PPVT correspond to scores on standard IQ tests with M = 100 and SD = 15. Among the complete Add Health sample, Wave III PPVT scores were available from 70.7% of individuals with valid Wave I scores. Wave II of Add Health is not utilized in this study, because PPVT was not administered as a part of Wave II data collection.

Parental education

Parental education was measured the same way as Rowe et al. (1999). In Wave I in-home interviews, participants were asked to indicate the highest level of education attained by their residential parents. Participants' responses were recoded into a factorial variable that represents different degrees of education with six possible levels. Because between-sibling and between-parent agreement on the measurement of parental education level was high, the responses were then averaged to estimate a family-level parental education indicator at Wave I. The final parental education variable has a mean of 3.51 and a median of 3.5, representing high school or some post-high school education experiences. The parental education levels in the genetic sample approximate a normal distribution.

Family income

Participants' total family income before taxes in 1994 was reported by their residential parents in Wave I in-home interviews. The reported income included parents' own income, the income of everyone else in the household, and income from welfare benefits, dividends, and all other sources. Income was measured in thousands of U.S. dollars and ranged from 0 to 999. The distribution of raw income was highly right-skewed (Mean = 134.96; Median = 41), which were then log-transformed to approximate a normal curve (M = 3.56, SD = 0.75).

Peer academic environment

Quality of the peer academic environment (PAE) was measured by average schoolmate cognitive performance on PPVT. School information was collected when Add Health participants were sampled from selected institutions. A school identification variable was assigned to each participant so that participants could be linked to their academic institution at Wave I. The school means of Wave I PPVT scores were then calculated for each school to represent the average cognitive ability of its students. Only pairs with both siblings enrolled at the same school (83% of the pairs) were included in PAE analysis, as our model assumes that the school environment is shared between siblings. The PAE variable used in the current study explains approximately 21% of the variance in PPVT scores, supporting the validity of the constructed PAE environmental measure. The PAE scores generally follow a normal distribution (Mean = 100.05, Median = 100.81, SD = 6.33). The correlations among PPVT scores and the three environmental moderators of interest are shown in Table 2.

Analysis

Basic data cleaning and descriptive statistics were completed in R (R Core Team 2022). Structural equation models were fit in Mplus (Muthén and Muthén, 2017) using full-information maximum likelihood estimation. In the current study, the Modified Sibling Correlation Model was adapted from the Modified Twin Correlation Model (MTCM; Turkheimer et al. 2017) to examine the Scarr-Rowe interaction at Wave I and III in Add Health. The MSCM is slightly different than the more common GxE model (Purcell 2002), in that

Table 2 Correlations among PPVT scores and the Three Environmental Moderators

Variable	1	2	3	4
1. Wave1 PPVT				
2. Wave3 PPVT	.61[a]			
	[.58, .63][b]			
3. Parental Education	.38	.29		
	[.35, .41]	[.26, .33]		
4. Family Income	.38	.32	.47	
	[.34, .41]	[.28, .36]	[.43, .50]	
5. Peer Academic Environment	.50	.44	.28	.35
	[.48, .53]	[.41, .47]	[.25, .32]	[.31, .38]

[a]All correlations are significant at $p < .001$

[b]95% confidence intervals are shown in square brackets

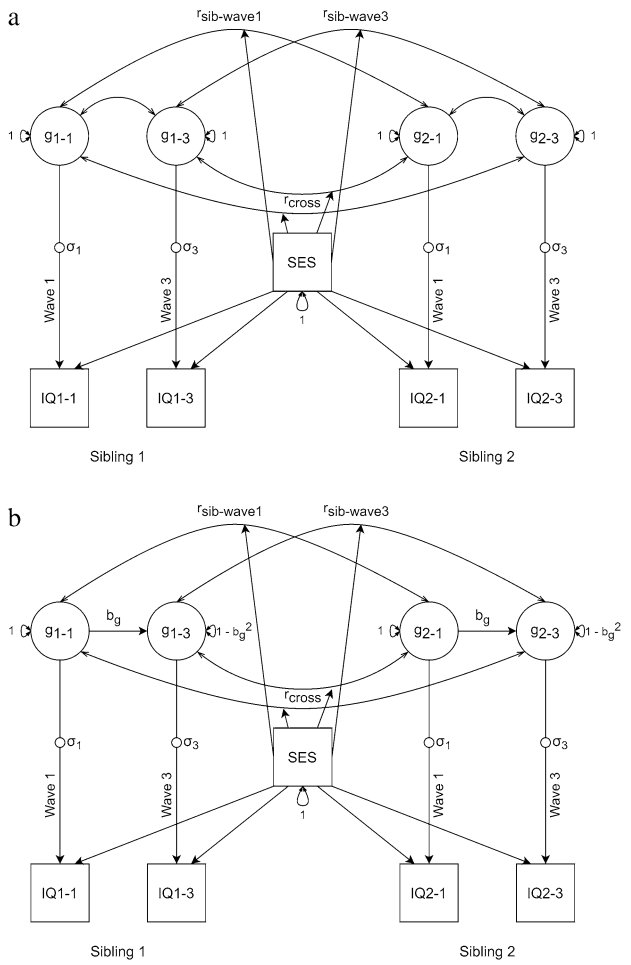

Fig. 1 **a** Represents the cross-sectional MSCM. **b** represents the longitudinal change MSCM. A separate model was fitted for each of the moderators of interest. SES in the diagrams serves as a placeholder for the actual moderator under inspection. IQ stands for PPVT scores

it models the Scarr-Rowe interaction basing on interactions between an environmental moderator and twin and sibling correlations, rather than on the genetic and environmental latent variables (ACE) that can be derived from sibling covariances. The MSCM also quantifies and controls for heteroscedasticity in the phenotype with respect to the moderator. Improving on the original MTCM, the MSCM allows for bivariate analysis of longitudinal data with genetically informative non-twin pairs, including siblings, half-siblings, cousins, and unrelated coresidents.

Cross-sectional model

First, bivariate MSCMs in which Wave III PPVT scores were not adjusted for Wave I scores were fit to examine gene-environment interactions cross-sectionally at the two waves (Fig. 1.a). Instead of testing for GxE interactions univariately, a bivariate model was applied here to boost the statistical power of our analyses (Schmitz et al. 1998). Separate models were fit for each moderator of interest. Before fitting the models, the linear effects of age and sex on verbal intelligence were partitioned out. Age was not significantly correlated with PPVT, whereas male sex was associated with scoring 1.64 points higher on PPVT. As recommended by Purcell (2002), the main effects of the moderators on PPVT scores were then removed by regressing the PPVT scores on each moderator of interest in the corresponding model, so to avoid inflating the estimates of the interaction effects. In the MSCM, the observed phenotypes at each wave were standardized as a part of the model by making them indicators of latent variables with M = 0 and SD = 1. The standardization process yields a factor loading weight that is equal to the standard deviation of PPVT scores of each wave, which was modeled for heteroscedasticity as an exponential function of the moderator. The sibling covariances between the standardized latent variables are then correlations, which were modeled as a function of zygosity (parameterized as degree of genetic relatedness, or r_G), the moderator, and the interaction between the two. This method allows the estimation of classical ACE parameters and their moderation according to the Scarr-Rowe interaction while separating effects on the phenotypic variance from effects on sibling pair correlations.

Estimation of the model differs from the standard computational approach in that it is modeled as a single group, with the pair correlations of verbal intelligence (standardized latent variable for PPVT scores) regressed on a variable representing genetic relatedness (r_G). According to the model, the sibling pair correlations at Wave I and Wave III were modeled as linear functions of genetic relatedness, the moderator, and their interaction:

$$r_{sib\_waveX} = C_{waveX} + C'_{waveX} \times SES + A_{waveX} \times r_G + A'_{waveX} \times r_G \times SES$$

Similarly, because the sibling cross correlations (i.e.: sibling 1 at Wave I with sibling 2 at Wave III) also partially reflect pair covariance, they were specified the same way in the model as:

$$r_{cross} = C_{cross} + C'_{cross} \times SES + A_{cross} \times r_G + A'_{cross} \times r_G \times SES$$

The intercept in this model (C), equal to the expected correlation between a sib-pair when genetic relatedness is 0, corresponds to the shared environmental term in the classical ACE model. The slope of regression of the pair correlation on the moderator (C') represents changes in the intercept as a function of the moderator, which corresponds to the shared environmental portion of the Scarr-Rowe interaction. The slope of regression of the pair correlation on r_G (A) is equal to the additive genetic term from the classical model. The interaction between the r_G term and the moderator (A') represents changes in the A parameter as a function of the moderator, and thus estimates the genetic aspect of the Scarr-Rowe effect. In addition, we modeled the phenotypic standard deviation at each wave as a function of the moderator to test

for heteroscedasticity in PPVT across SES contexts. In the expression, b_{SD1} represents the change in the standard deviation of PPVT scores as the moderator increases by 1 SD. The exponential function was utilized because the standard deviation of verbal intelligence is defined as positive:

$$\sigma_{waveX} = exp(b_{SD0\_waveX} + b_{SD1\_waveX} \times SES)$$

Longitudinal change model

Next, a longitudinal bivariate MSCM (Fig. 1.b) was fitted to examine potential gene-environment interaction on change in verbal intelligence from adolescence to early adulthood. Similar to the bivariate cross-sectional model, age, sex, and the moderator's main effects on PPVT scores were removed, and PPVT scores were standardized onto latent variables representing verbal intelligence at each wave. The main difference in the longitudinal model is that the Wave III latent variables are now regressed onto Wave I variables, where the Wave III residuals represent the relative change in verbal intelligence from Wave I to Wave III (the variance of Wave III residuals is thus constrained to $(1 - b_g^2)$. Residualizing the Wave III ability variable allows us to estimate the genetic and shared environmental effect on cognitive plasticity between adolescence and adulthood in different environmental contexts. Sibling correlations of the Wave III residuals were thus modeled as functions of the degree of genetic relatedness, the moderator of interest, and their interaction, from which ACE parameters can be derived to test for the Scarr-Rowe effect on the heritability of change in verbal intelligence. Sibling pair correlations of Wave III residuals can be expressed as:

$$r_{sib_{wave3}} = C_{change} + C'_{change} \times SES + A_{change} \times r_G + A'_{change} \times r_G \times SES$$

Accordingly, A and C parameter in the expression stand for additive genetic and shared environmental effect. A' and C' represent the moderation on A and C by the moderator. The ways that the sibling pair correlation of Wave I verbal intelligence, the sibling cross correlation, and heteroscedasticity were modeled are essentially the same as in the cross-sectional model.

Results

Missing information on the moderators

Table 3 compares the mid-pair PPVT scores for sibling pairs with valid versus missing information on parental education, family income, and peer academic environment. Overall, sibling pairs with missing information on the moderators scored lower on PPVT. Pairs with missing information on parental education scored much lower on PPVT than pairs with valid information (a difference of 8—10 points), but such pairs are rare (n = 52, 1.8% of all pairs). Missing information on family income or peer academic environment predicts a 2-to-4-point decrease in PPVT score. Sibling type composition is not significantly different between pairs with valid versus missing information on the moderator. However, certain pair types were prone to be excluded from the peer academic environment analysis, as half-siblings and cousins were less likely to be enrolled at the same school.

Missing information on wave III PPVT scores

Table 4 compares the sample characteristics for individuals who have valid versus missing PPVT scores at Wave III. Individuals who are missing Wave III data were slightly older, less likely to be female, and less likely to be White when compared to individuals with valid Wave III data. However, there are no significant differences between the groups in parental education, family income, or peer academic environment. This suggests that attrition from cognitive testing at Wave III did not affect the primary moderators of interest in the current study, and thus is unlikely to bias the results of our moderation analysis with these variables. Sibling type composition is significantly different between pairs with valid versus missing PPVT scores at Wave III. Cousins and unrelated pairs are less likely to have complete data at Wave III than other sibling types. The proportion of cousin pairs with complete data at Wave III is 72% and that of unrelated pairs is 63.2%, compared to 78 to 82% among the MZ twin, DZ twin, and full-sibling pairs.

Sibling correlations

Sibling correlations of PPVT scores at Wave I and Wave III are reported in Table 5. The sibling correlations at Wave I are roughly proportional to genetic relatedness. Dizygotic twin and full-sibling correlations are roughly equal in magnitude, suggesting no specific twin effect. Notably, in violation of quantitative genetic expectations, Wave III correlations for unrelated pairs are often as high or higher than the corresponding correlations for half-siblings and cousins across the full genetic sample and all three subsamples. The pattern of uncommonly high r_{UR} is observed only at Wave III, suggesting an attrition effect. This anomaly warrants our attention, because abnormality in r_{UR} could substantially inflate our estimation of the shared environmental effect, thereby biasing the results of moderation analyses.

Table 3 PPVT Scores for Sibling Pairs with Valid versus Missing Information on the Moderators[a]

	Parental education			Family income			Peer academic environment		
	Valid	Missing	t-value[b]	Valid	Missing	t-value	Valid	Missing	t-value
n pairs	2918	52		2324	646		2115	855	
W1 PPVT Score	97.29 (12.56)	88.76 (10.51)	4.867***	98.13 (12.25)	93.53 (13.08)	8.317***	97.66 (12.64)	95.83 (12.33)	3.598***
W3 PPVT Score	98.03 (12.17)	91.37 (12.69)	3.909***	98.54 (11.99)	95.61 (12.75)	5.418***	98.58 (12.09)	96.33 (12.36)	4.563***

[a]Comparisons were based on a working sample (n = 2970) with "other" sibling pairs (i.e., aunt/uncle-niece/nephew pairs, group home pairs, spouse pairs, and in-law pairs) and individuals with non-ordinary cognitive development (PPVT scores < 50) excluded

[b]t-values were calculated from two-sample t-tests. ***p < .001, two-tailed

Moderation results

An MSCM was fitted for each moderator of interest (parental education, family income, or peer academic environment). Parameter estimates from MSCMs are shown in Table 6. The main effects on PPVT were significant for all three moderators at both waves and were removed from PPVT scores, following the recommendation by Purcell (2002). Heteroscedasticity in PPVT with respect to SES was generally suggested (Fig. 2). As shown by $b_{SD1\text{-}wave1}$ and $b_{SD1\text{-}wave3}$ in Table 6, the standard deviation of PPVT scores was lower at higher levels of parental education, family income, and peer academic environment.

At Wave I, Scarr-Rowe interactions were observed for parental education and peer academic environment, but not family income. Higher levels of parental education and peer academic environment predicted both an increased additive genetic effect (A'_{wave1}) and a decreased shared environmental effect (C'_{wave1}; as reported by Rowe et al. 1999). Across the range of parental education, the MZ correlation increased, and correlations of all other groups decreased, resulting in an increase in heritability from 22 to 62% and a decrease in the proportion of shared environmental variance from 40 to 15%. The MZ correlation was stable across the range of peer academic environments, while correlations of all other pair-types decreased. The heritability estimate rose from 23 to 67% and the shared environmental effect dropped from 46 to 1%.

A Scarr-Rowe effect with peer academic environment was observed at Wave III in the cross-sectional model, when participants were young adults. Similar to Wave I results, better peer academic environment was associated with an increase

Table 4 Sample characteristics by Wave III attrition status

	Wave 1 valid	Wave 3 valid	Wave 3 missing	W3 group difference test[a]
N individuals	4959	3826	1133	
Age at wave 3	22.27 (1.80)	22.25 (1.80)	22.82 (1.75)	t = − 9.422***
% Female	50.2	52.1	43.7	χ^2 = 28.19***
% White	53.9	54.6	51.3	χ^2 = 7.38**
W1 PPVT score	97.70 (14.22)	97.99 (14.17)	96.70 (14.34)	t = 2.684**
W3 PPVT score	98.27 (13.51)	98.27 (13.51)		
n pairs	2723	2099	624	
Parental education	3.56 (1.16)	3.55 (1.16)	3.59 (1.18)	t = − 0.753 (ns)
Family income	3.58 (0.76)	3.57 (0.77)	3.60 (0.72)	t = − 0.867 (ns)
Peer academic environment	99.78 (6.43)	99.90 (6.39)	99.37 (6.54)	t = 1.809†
Sibling pair type				χ^2 = 58.74***
MZ	255	203	52	79.6% complete
DZ	414	322	92	77.8% complete
FS	1147	940	207	82.0% complete
HS	374	282	92	75.4% complete
CO	174	125	49	71.8% complete
UR	359	227	132	63.2% complete

Attrition analyses were based on a working sample ($N_{individual}$ = 5232) with "other" sibling pairs (i.e., aunt/uncle-niece/nephew pairs, group home pairs, spouse pairs, and in-law pairs) and individuals with non-ordinary cognitive development (PPVT scores < 50) excluded

[a]t-values and χ^2 values were calculated from ANOVAs and Chi-Square Tests between the Wave III valid group and the Wave III missing group. ***p < 0.001, **p < 0.01, †p < 0.1

Table 5 Sibling Correlations of PPVT Scores in the Full Genetic Sample and the Three Subsamples

	Full genetic sample		Parental education		Family income		Peer academic environment	
	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III
r_{MZ}	.75[a]	.68	.75	.68	.75	.71	.75	.68
r_{DZ}	.55	.57	.55	.57	.56	.54	.55	.57
r_{FS}	.57	.49	.57	.49	.54	.50	.57	.49
r_{HS}	.46	.32	.46	.31	.43	.28	.46	.31
r_{CO}	.31	.33	.28	.33	.27	.33	.28	.33
r_{UR}	.27	.40	.26	.40	.20	.39	.26	.45

[a]All correlations are significant at $p < .001$

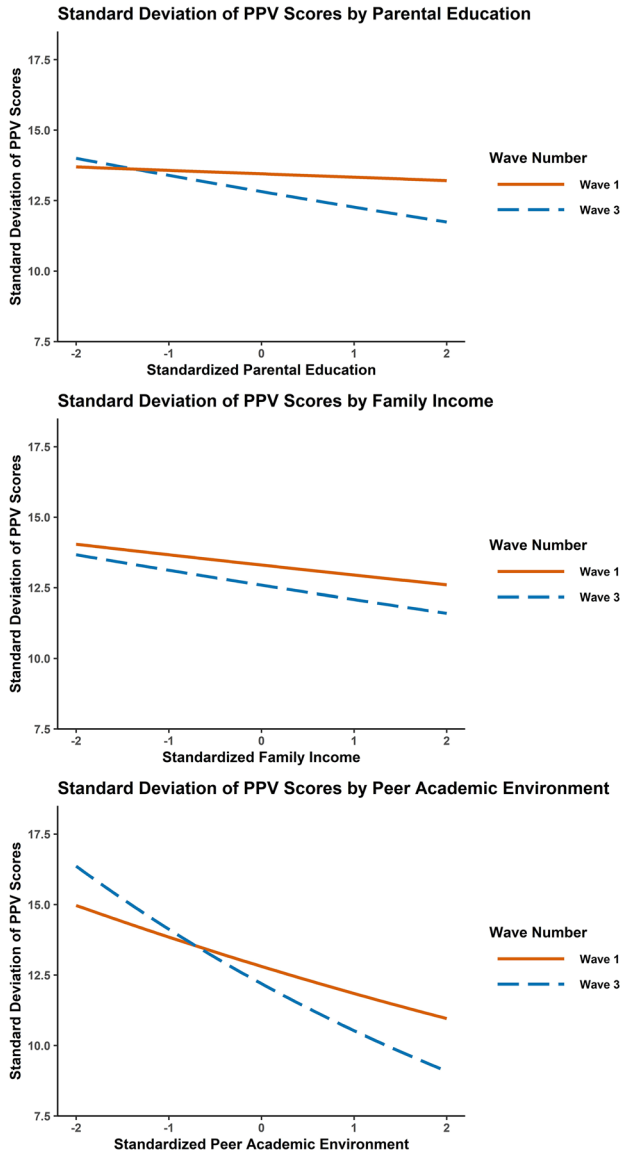

Fig. 2 Heteroscedasticity in PPVT scores at Wave I and Wave III was shown separately for parental education, family income, and peer academic environment. See legends

in A'_{wave3} from 2 to 88% and a decrease in C'_{wave3} from 51 to 0%. The effect was attributable to both an increasing MZ correlation and decreasing correlations of other pairs. For parental education, however, the Scarr-Rowe interaction was in the predicted direction, but not statistically significant. Diminished magnitude of the effect size and large standard errors around the parameter estimates were possibly due to anomalous correlation of unrelated pairs at Wave III (see discussion).

A Scarr-Rowe interaction on the development of verbal intelligence across adolescence and young adulthood was suggested by results from the longitudinal change model. Better peer academic environment predicted a lower shared environmental effect (C'_{change}) on the residualized Wave III latent variable that represents change in verbal intelligence from Wave I to Wave III. An increase in the additive genetic effect (A'_{change}) was also observed, but was only marginally significant ($p = 0.064$). The moderation of the heritability of cognitive change between Wave I and Wave III by parental education was once again in the predicted direction but did not reach conventional statistical significance ($p = 0.058$). The magnitude of sibling correlations of change in verbal intelligence was low (approximately 0.12) and sometimes negative, suggesting substantial nonshared environmental effect. The relations between the moderators of interest and sibling correlations of PPVT scores are shown in Fig. 3.

Lastly, the heteroscedasticity models of phenotypic variances were combined with the standardized twin correlations in the MSCMs to obtain estimates of the moderation of the unstandardized ACE variance components by the three moderators. Results are illustrated in Fig. 4. The top black line in each panel is the modeled phenotypic variance as a function of the moderator. The red, blue and green lines represent the moderated unstandardized A, C and E variances, respectively, with the sum of the ACE variances equal to the total phenotypic variance. Overall, in more advantaged environments, total phenotypic variance decreases, additive genetic variance increases, and shared and non-shared

Table 6 Model Parameter Estimates

A Results from the Cross-Sectional MSCM[a]

	Parental education			Family income			Peer academic environment		
	Estimate	S.E	t-value	Estimate	S.E	t-value	Estimate	S.E	t-value
Wave 1									
C_{wave1}	0.278	0.026	10.633***	0.224	0.029	7.683***	0.233	0.033	7.039***
C'_{wave1}	**− 0.066**	**0.024**	**− 2.718****	− 0.031	0.026	− 1.203	**− 0.114**	**0.031**	**− 3.654*****
A_{wave1}	0.428	0.040	10.634***	0.511	0.042	12.115***	0.449	0.048	9.400***
A'_{wave1}	**0.100**	**0.037**	**2.744****	− 0.011	0.040	− 0.290	**0.111**	**0.042**	**2.608****
$b_{SD0\text{-}wave1}$	2.599	0.010	250.2***	2.588	0.012	224.5***	2.550	0.012	210.9***
$b_{SD1\text{-}wave1}$	0.009	0.009	0.991	**− 0.027**	**0.010**	**− 2.752****	**− 0.078**	**0.011**	**− 7.116*****
Wave 3									
C_{wave3}	0.275	0.033	8.414***	0.246	0.036	6.894***	0.190	0.042	4.478**
C'_{wave3}	− 0.022	0.032	− 0.672	0.013	0.032	0.412	**− 0.161**	**0.045**	**-3.594*****
A_{wave3}	0.346	0.057	6.113***	0.393	0.059	6.708***	0.454	0.065	7.023***
A'_{wave3}	0.082	0.054	1.539	0.049	0.055	0.885	**0.219**	**0.075**	**2.933****
$b_{SD0\text{-}wave3}$	2.551	0.011	228.1***	2.533	0.012	204.5***	2.501	0.013	190.3***
$b_{SD1\text{-}wave3}$	**− 0.044**	**0.010**	**− 4.508*****	**− 0.041**	**0.010**	**− 4.062*****	**− 0.147**	**0.012**	**− 12.068*****
Cross Correlations									
C_{cross}	0.222	0.024	9.436***	0.187	0.026	7.229***	0.177	0.030	5.939***
C'_{cross}	− 0.017	0.022	− 0.790	− 0.023	0.024	− 0.985	**− 0.104**	**0.029**	**− 3.520*****
A_{cross}	0.414	0.036	11.646***	0.479	0.036	13.215***	0.444	0.042	10.612***
A'_{cross}	0.017	0.032	0.548	0.007	0.040	0.172	**0.102**	**0.042**	**2.428***

B Results from the Longitudinal Change MSCM[b]

	Parental Education			Family Income			Peer Academic Environment		
	Estimate	S.E.[c]	t-value[d]	Estimate	S.E	t-value	Estimate	S.E	t-value
Wave 3 Residualized									
C_{change}	0.105	0.027	3.926***	0.098	0.028	3.479**	0.059	0.036	1.652
C'_{change}	-0.026	0.025	− 1.042	0.030	0.021	1.418	**− 0.076**	**0.037**	**− 2.043***
A_{change}	− 0.007	0.056	− 0.124	− 0.014	0.058	0.239	0.073	0.068	1.082
A'_{change}	**0.101**	**0.053**	**1.912**†	0.035	0.043	0.820	**0.135**	**0.073**	**1.850**†
Cross Correlations									
C_{cross}	0.045	0.019	2.323*	0.042	0.021	1.997*	0.031	0.025	1.237
C'_{cross}	0.025	0.036	1.408	− 0.003	0.018	− 0.164	− 0.032	0.024	− 1.308
A_{cross}	0.141	0.018	3.875***	0.148	0.038	3.930***	0.162	0.042	3.803***
A'_{cross}	− 0.046	0.032	− 1.431	0.014	0.037	0.389	0.032	0.041	0.770

[a]Table 6 A presents the parameter estimates of the cross-sectional MSCM.

[b]Table 6 B presents the parameter estimates of the longitudinal change MSCM.

[c]Standard error.

[d]Significance is based on two-tailed p-values. ***p < .001, **p < .01, *p < .05, †marginally significant.

environmental variances decrease. Note that the shared environmental variance and the phenotypic variance are underestimated in the figure, as the main effect of the moderator on PPVT scores was removed in each of the MSCMs, as is standard in Scarr-Rowe models (Purcell 2002). Note also that the MSCM allows the ACE variances to become negative at the extremes. Such a result is anomalous substantively, as variance cannot be negative by definition, but it is nevertheless what is frequently implied by twin correlations when r_{DZ} becomes less than half of r_{MZ}, which would lead to a negative estimate of the shared environment effect.

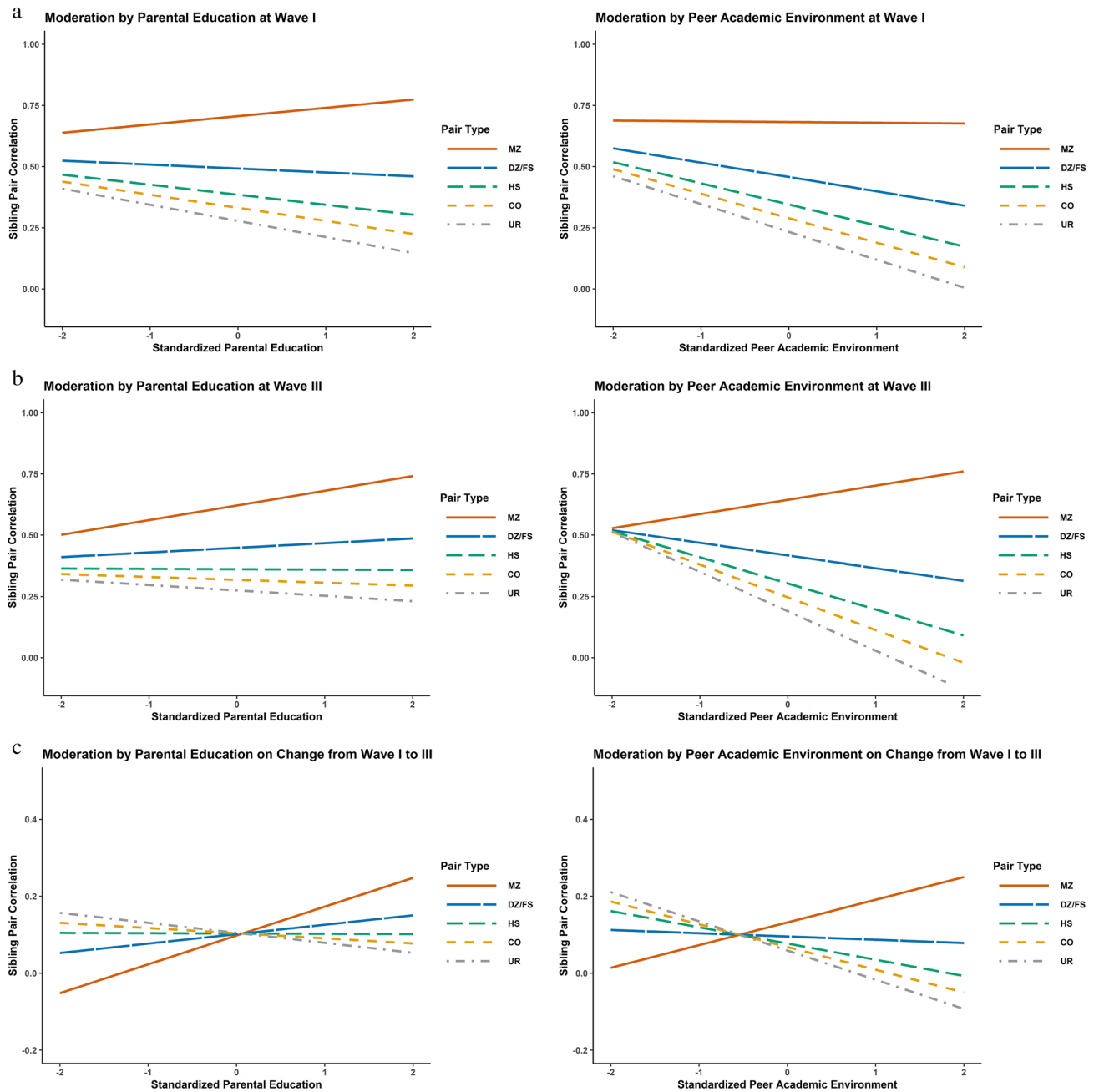

Fig. 3 **a–c** Represent the moderation on sibling pair correlations of PPVT at Wave I, at Wave III, and longitudinally. Visual representation was based on MSCM model estimation instead of actual data

Discussion

In this study, we longitudinally examined the interactions between the heritability of intelligence and three potential environmental moderators (parental education, family income, and peer academic environment) in a developmental context. Our longitudinal analyses started with the Scarr-Rowe interaction in adolescence, as detected by Rowe et al. (1999) in Wave I of Add Health, and tested whether this effect extended into Wave III, when the participants were young adults. Overall, two of the three environmental factors of interest – parental education, used by Rowe et al., and peer academic environment – moderated sibling correlations of verbal intelligence at Wave I and Wave III.

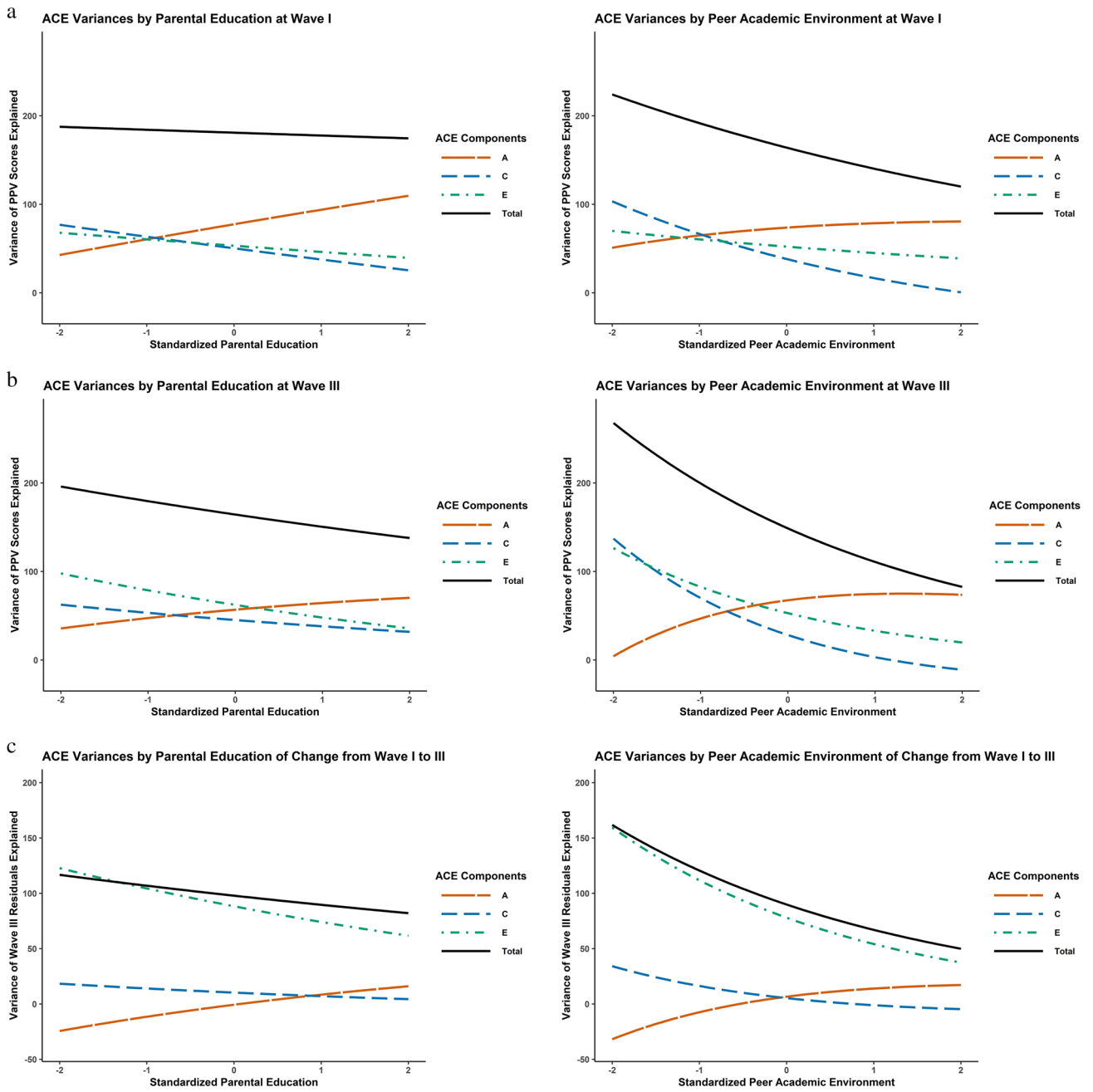

Fig. 4 A, Additive genetic variance; C, shared environmental variance; E, non-shared environmental variance; Total, total phenotypic variance. **a**, **b** represent how ACE variances vary by the moderator at Wave I and Wave III. **c** shows the moderation on ACE variances of change in verbal intelligence from Wave I to Wave III

Our results again suggest that rather than being a fixed characteristics of a trait, the heritability of intelligence is a population-specific estimate that varies, among other things, across the range of socioeconomic contexts. Therefore, in order to interpret any heritability estimate, the genetic and environmental effects on intelligence must be contextualized within specific environments that provide different levels of resources and support for cognitive development.

The Scarr-Rowe interaction in adolescence

Our findings add to the existing literature in support of the Scarr-Rowe interaction in American adolescents. Using more sophisticated models than were available to Rowe et al. (1999), the current study confirms that parental education moderates the heritability of verbal intelligence at Wave I. The heritability estimate varied from 22 to 62%

across the range of parental education. Moreover, an interaction between heritability and quality of peer academic environment was found, in which the heritability estimate increased from 23 to 67%. Our effect sizes align well with previous studies on gene × SES interaction in adolescence (Gottschling et al. 2019; Harden et al. 2007; Kirkpatrick et al. 2015) and with a meta-analysis by Tucker-Drob and Bates (2016) that documented a 30–40% increase in the heritability of intelligence from low- to high-SES homes in average U.S. non-adult samples.

The Scarr-Rowe interaction in adulthood

The heritability of verbal intelligence in early adulthood was found to be strongly moderated by peer academic environment measured in adolescence. In contrast to this finding, two prior studies of the U.S population have found no evidence of Scarr-Rowe effects in adults (Van der Sluis et al. 2008; Zavala et al. 2018). One reason previous studies may have failed to detect the Scarr-Rowe effect in adulthood is that instead of measuring parental SES in childhood, previous analyses used adult SES as the moderator. Van der Sluis et al. (2008) found no gene × SES effect using partner education and urbanization level reported by young adults at age 26 and middle-aged adults at age 49. In a sample of adults aged 27 to 98 years old, Zavala et al. (2018) found an interaction in the reversed direction between occupation status in adulthood and the heritability of verbal ability.

SES reported in adulthood is conceptually very different from SES measured at the parental level in childhood or adolescence. Whereas SES in childhood is imposed by one's parents and strictly shared among siblings, adult SES varies within sibling pairs, is at least partially determined and maintained by the individual, and presumably depends in part on cognitive ability. In support of this hypothesis, attained SES in adulthood was found to moderate the genetic and environmental influences of cognitive abilities through a different mechanism than rearing SES in childhood (Zavala et al. 2018). Examining early-life SES might be key to studying the Scarr-Rowe interaction in adulthood. We are currently involved in an extensive data collection of adult cognitive ability scores in a twin sample whose rearing environments were studied intensively when the participants were children (Davis et al. 2019).

One anomaly in Add Health is that the group of unrelated pairs is as similar or more similar in verbal intelligence at Wave III than either the half-siblings or the cousins, a phenomenon that was not originally observed at Wave I. There is no reason in the quantitative genetic model why this would be the case. An attrition analysis was thus performed on unrelated pairs that reveals systematic differences in the pair correlations in the unrelated group in relation to attrition status. Unrelated pairs with missing Wave III data were correlated 0.19 in their verbal intelligence at Wave I, while pairs who were retained in the study at Wave III were correlated 0.36. We thus decided to rerun a post-hoc, exploratory analysis excluding the UR pairs (see Table 7 in the appendix for results). The results did not change substantially for Wave I. However, the effect sizes were generally augmented in the cross-sectional analysis at Wave III and in the longitudinal analyses. Specifically, with the unrelated group excluded, moderation of the A term by parental education was in the expected (positive) direction and statistically significant ($p = 0.046$) at Wave III in the cross-sectional model. In the longitudinal change model, moderation of both A and C by parental education were in the expected direction and statistically significant ($p = 0.002$ and 0.017 for A and C, respectively). Moderation effects by peer academic environment were also larger in magnitude in both the cross-sectional and the longitudinal models with the unrelated group excluded. We theorize that the insignificant results for A and C were potentially the result of the unusually high correlations in the unrelated group at Wave III. These correlations, which occur for a substantial number of phenotypes, are worthy of further investigation. It is possible that some unrelated pairs were miscategorized and in fact genetically related, but this hypothesis is hard to test because few unrelated pairs in the sibling sample were genotyped (McQueen et al. 2015). It is also possible that non-ordinary characteristics about step siblings and adoptive/foster pairs prevent the Scarr-Rowe effect from being generalized to these families. In fact, the Scarr-Rowe effect was also absent in a recent family study of adoptees (Loehlin et al. 2022).

The Scarr-Rowe interaction on longitudinal change in verbal intelligence

The current study is the first to analyze the Scarr-Rowe interaction longitudinally across adolescence and early adulthood. The non-shared environmental effect on change in verbal intelligence between the two developmental stages was universally high across the range of parental education and peer academic environment, suggesting that cognitive changes between adolescence and early adulthood is either measured with substantial error, or mainly influenced by environments that are unique to each individual, or both. Peer academic environment was found to moderate the shared environmental effect on change in verbal intelligence. Moderation of the additive genetic effect by both parental education and peer academic environment was observed, but only marginally significant. When the UR pairs were not considered, moderation of the additive genetic effect became robust and substantially larger in its effects. These results suggest the possibility that gene-environment interaction affecting intelligence in adulthood was not fully accounted for by the pre-existing interaction effect in adolescence, but

was partially attributable to novel gene-environment interactions that arise in the process of cognitive development. Our findings again highlight the importance of the environment in which cognitive development takes place, as the quality of this environment has implications on the genetic and environmental etiology of cognitive plasticity across developmental stages.

The role of academic environment

The current study also explored the possibility that quality of the academic environment might moderate the heritability of intelligence. Cognitive competency of schoolmates has been shown to facilitate learning and the acquisition of various cognitive skills (Bifulco et al. 2008; Hanushek et al. 2003; Hoxby 2000; Peetsma, 2007). Through measuring cognitive competency of the students, we used the peer academic environment variable to approximate school environments that facilitate cognitive development. Echoing Taylor et al. (2010), our results suggest that the quality of school environment interacts with genetic and environmental factors of intelligence. In both adolescence and early adulthood, the additive genetic effect is lower while shared environmental effect is higher in schools where students are less cognitively advanced. The interaction effects appear to be stronger in adulthood than in adolescence, suggesting that education might have a cumulative effect on cognitive development. Notably, the effect size of moderation by parental education seems to decrease in adulthood. This observation, along with the strong moderation in adulthood by peer academic environment, may reflect typical developmental shifts in adolescence whereby family factors become less important relative to peer effects. Although cognitive development in childhood may be subject to fluctuations within the home environment, cognitive development from adolescence to adulthood may be more sensitive to peer norms or school resources.

Our novel analysis of peer academic environment has established school context as an environmental moderator of the heritability of intelligence that is not directly related to strictly economic differences among families. The correlations between peer academic environment and traditional SES variables like parental education and income are relatively low, differentiating the peer academic environment measure from pure socioeconomic indices. The Scarr-Rowe moderation effects of peer academic environment are quite strong: most notably, from the lowest to the highest levels of peer academic environment, heritability of verbal intelligence at Wave III increased from 2 to 89%. The strength of moderation by peer academic environment suggests that the tendency for the Scarr-Rowe effect to be more robust in North America than in Europe or Australia may be related to greater degrees of variation in educational quality in the United States. Future studies should investigate other environmental factors that are not directly linked to economic resources that might reproduce the Scarr-Rowe effect, including contextual aspects of neighborhoods, schools, and homes, caregiver behaviors and personalities, and peer interactions. Identification of such variables can inform public policy and intervention programs that could systematically improve aspects of the environment that are independent from rearing SES to maximize individual potential in cognitive development (Bates et al. 2016).

The absence of moderation by family income was unexpected, considering that the family income variable is highly correlated with parental education, which was already known to have produced a significant interaction at Wave I. Although past research has yielded positive results on gene × SES interactions with parental/family income as an independent moderator or as a part of a composite SES measure (Bates et al. 2013; Harden et al. 2007; Tucker-Drob et al. 2010), our analysis showed no such evidence in either adolescence or adulthood. This further implies that the Scarr-Rowe effect may not be socioeconomic in nature. Its underlying mechanism could be less related to monetary resources in a family like family income. Rather, it may be caused by differential educational resources that would facilitate cognitive development in children and adolescents, which could potentially be decoupled from SES variables like family income.

Utility of the MSCM

The current study has also reinforced the utility of the longitudinal Modified Sibling Correlation Model in investigating gene-environment interactions on cognition and other phenotypes. The bivariate adaptation of the MTCM is useful for longitudinal modeling of genetic and environmental effects on the change in a phenotype between two timepoints. The MSCM utilizes genetically-informative pairs of different types (i.e.: twins, siblings, cousins), thereby increasing the sample sizes and statistical power of prospective gene-environment interaction studies. Another feature of the MSCM is that it separates heteroscedasticity in the phenotype as a function of the moderator from the main objects of interest, moderation of the pair correlations. We have found that phenotypic heteroscedasticity is common in data of this kind (Turkheimer et al. 2017). Moreover, as shown in previous studies (Turkheimer et al. 2017; Gottschling et al. 2019), shared environmental estimates can become negative under moderation when the dizygotic twin correlation falls to less than half of the monozygotic twin correlation. In our analyses, we were able to model the additive genetic effects and shared environmental effects as negative at extreme values of SES. The MSCM is thus more flexible than the classical Purcell model, since focusing exclusively on ACE components

of variance cannot successfully capture the dynamics of actual twin analyses on gene-environment interaction, in which it is common for DZ correlations to be less than half of the corresponding MZ correlations.

Limitations

The current study has several notable limitations. Like Rowe et al. (1999), the cognitive ability data was limited to verbal intelligence, as measured by the Add Health Picture Vocabulary Test. Recognized as an important aspect of intelligence from the earliest days of intelligence testing, vocabulary score has a high factor loading on general intelligence (Jensen 1998; Terman 1911). Moderate to high correlations between earlier versions of PPVT and performance, verbal, as well as full-scale intelligence in Wechsler Intelligence Scale for Children were documented, which makes the same moderation effects on general intelligence plausible (Anderson & Flax 1968; Hodapp & Gerken 1999). Although the Add Health sample was designed to be representative of the U.S. population, attrition between Wave I and III was notably more severe for non-white participants, reducing the representativeness of results from Wave III. The measurement of peer academic environment in this study is relatively coarse, informed solely by schoolmate intelligence scores. Future studies should measure school and peer environments with more comprehensive behavioral and ecological information. As is always the case in Scarr-Rowe analyses of intelligence, measures of parental education and peer academic environment are not purely environmental; they also reflect genetic differences associated with intelligence (Calvin et al. 2012; Marioni et al. 2014; Rietveld et al. 2014; Smith-Woolley et al. 2018). In our study, correlations of verbal intelligence were modeled as linear functions, so that the parameter estimates can be straightforwardly interpreted as moderation of genetic and environmental variances. Considering the asymmetrical sampling distribution of correlation coefficients, we also ran the models using a Fisher-Z transformation and the results were not substantially different. Future work should explore how models like MSCM could better handle significance testing with correlation coefficients (i.e., using a Bayesian estimator). Finally, high correlations were observed in the group of unrelated pairs at Wave III. To some extent, correlations among unrelated family members are an indication of shared environmental effects, but there is no reason we are aware of for the unrelated group to be more correlated than cousins or half-siblings. Although we suggested that differential attrition might have contributed to this unexpected pattern and found that interactions with parental education were more typical when the unrelated pairs were excluded, future attempts should be made to explain the pattern of attrition and its influence on pair correlations.

Conclusion

The current study examined the Scarr-Rowe interaction in a longitudinal sample of twins, siblings, cousins, and unrelated coresidents from the National Longitudinal Study of Adolescent to Adult Health. Overall, our results mirror findings in prior studies that the heritability of intelligence is not a static characteristic of a trait but a populational estimate that varies in predictable ways across environmental contexts. This study provides evidence that the moderation of the heritability of intelligence by childhood SES persists into adulthood, and the effect sizes of the interactions vary across developmental stages. Moreover, the Scarr-Rowe interaction in adulthood is partially due to novel gene-environment interaction effects on change in verbal intelligence from adolescence and early adulthood. Peer academic environment was found to strongly moderate the heritability of intelligence, while income did not, implying that the environmental input causing the Scarr-Rowe effect could potentially be decoupled from specifically socioeconomic measures. The study also demonstrates the utility of the Modified Sibling Correlation Model in studying gene-environment interactions longitudinally.

Appendix

See Table 7

Table 7 Model Parameter Estimates After Removing UR Pairs

Results from the Cross-Sectional MSCM, Excluding UR Pairs

	Parental education			Family income			Peer academic environment		
	Estimate	S.E	t-value	Estimate	S.E	t-value	Estimate	S.E	t-value
Wave 1									
C_{wave1}	0.293	0.031	9.559***	0.247	0.034	7.196***	0.262	0.039	6.677***
C'_{wave1}	**− 0.071**	**0.029**	**− 2.485***	− 0.030	0.030	− 1.002	**− 0.090**	**0.036**	**− 2.493***
A_{wave1}	0.407	0.047	8.755***	0.481	0.049	9.727***	0.414	0.057	7.302***
A'_{wave1}	**0.113**	**0.042**	**2.693****	− 0.017	0.044	− 0.383	0.079	0.049	1.606
$b_{SD0\text{-}wave1}$	2.602	0.011		2.590	0.013		2.555	0.013	
$b_{SD1\text{-}wave1}$	0.009	0.010	0.874	**− 0.033**	**0.011**	**− 3.076****	**− 0.086**	**0.012**	**− 7.251*****
Wave 3									
C_{wave3}	0.240	0.038	6.377***	0.226	0.041	5.495***	0.149	0.047	3.193**
C'_{wave3}	− 0.040	0.036	− 1.097	0.023	0.038	0.603	**− 0.173**	**0.050**	**− 3.492*****
A_{wave3}	0.398	0.061	6.513***	0.426	0.064	6.607***	0.521	0.066	7.933***
A'_{wave3}	**0.110**	**0.055**	**1.992***	0.032	0.068	0.461	**0.242**	**0.077**	**3.137*****
$b_{SD0\text{-}wave3}$	2.553	0.012		2.537	0.013		2.509	0.014	
$b_{SD1\text{-}wave3}$	**− 0.052**	**0.011**	**− 4.919*****	**− 0.044**	**0.011**	**− 4.025*****	**− 0.153**	**0.013**	**− 11.884*****
Cross Correlations									
C_{cross}	0.234	0.027	8.686***	0.197	0.030	6.660***	0.198	0.034	5.754***
C'_{cross}	0.000	0.025	− 0.009	− 0.027	0.027	− 0.982	− 0.085	0.034	− 2.509*
A_{cross}	0.390	0.040	9.848***	0.462	0.041	11.288***	0.413	0.048	8.691***
A'_{cross}	− 0.013	0.036	− 0.354	0.012	0.044	0.275	0.073	0.048	1.532

Results from the Longitudinal Change MSCM, Excluding UR Pairs

	Parental Education			Family Income			Peer Academic Environment		
	Estimate	S.E	t-value	Estimate	S.E	t-value	Estimate	S.E	t-value
Wave 3 Residualized									
C_{change}	0.062	0.031	1.997*	0.074	0.033	2.228*	0.004	0.042	0.098
C'_{change}	**− 0.067**	**0.028**	**− 2.393***	0.045	0.026	1.730	**− 0.102**	**0.043**	**− 2.358***
A_{change}	0.069	0.062	1.112	0.031	0.066	0.465	0.166	0.075	2.217*
A'_{change}	**0.171**	**0.054**	**3.144****	0.009	0.054	0.166	**0.181**	**0.081**	**2.245***
Cross Correlations									
C_{cross}	0.372	0.214	1.735	0.282	0.210	1.341	1.021	5.577	0.183
C'_{cross}	0.330	0.184	1.790	− 0.053	0.154	− 0.343	− 0.874	4.355	− 0.201
A_{cross}	0.800	0.519	1.541	1.253	1.532	0.818	0.584	0.299	1.952†
A'_{cross}	− 0.500	0.309	− 1.621	0.190	0.373	0.508	0.090	0.192	0.469

Table 7 presents the parameter estimates of the cross-sectional MSCM and the longitudinal change MSCM with unrelated pairs excluded. Significance is based on two-tailed p-values. ***p < .001, **p < .01, *p < .05, †marginally significant.

Authors' Contributions LiChen Dong conducted the main analyses and took primary responsibility for writing the manuscript, under Eric Turkheimer's direct supervision. Christopher Beam, Evan Giangrande, Sean Womack, and Kristy Yoo are members of the Scarr-Rowe analysis team. Kristen Jacobson provided continuity with Wave I analyses.

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Data Availability Data can be accessed through University of North Carolina—Chapel Hill at https://addhealth.cpc.unc.edu/.

Code Availability Mplus codes are made available on OSF at https://osf.io/mxd2h/.

Declarations

Conflicts of interest The LiChen Dong, Evan J. Giangrande, Sean R. Womack, Kristy Yoo, Christopher R. Beam, Kristen C. Jacobson, and Eric Turkheimer2 declare no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

This research project was approved by University of Virginia Institutional Review Board for the Social and Behavioral Sciences (IRB-SBS).

Consent to Participate This research project was a secondary analysis. Consent to participate was obtained as a part of the National Longitudinal Study of Adolescent to Adult Health.

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