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Emergence of a Gene × Socioeconomic Status Interaction on Infant Mental Ability Between 10 Months and 2 Years

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Abstract

Recent research in behavioral genetics has found evidence for a Gene × Environment interaction on cognitive ability: Individual differences in cognitive ability among children raised in socioeconomically advantaged homes are primarily due to genes, whereas environmental factors are more influential for children from disadvantaged homes. We investigated the developmental origins of this interaction in a sample of 750 pairs of twins measured on the Bayley Short Form test of infant mental ability, once at age 10 months and again at age 2 years. A Gene × Environment interaction was evident on the longitudinal change in mental ability over the study period. At age 10 months, genes accounted for negligible variation in mental ability across all levels of socioeconomic status (SES). However, genetic influences emerged over the course of development, with larger genetic influences emerging for infants raised in higher-SES homes. At age 2 years, genes accounted for nearly 50% of the variation in mental ability of children raised in high-SES homes, but genes continued to account for negligible variation in mental ability of children raised in low-SES homes.

Keywords

gene-environment interaction, socioeconomic status, infancy, cognitive development, intelligence, behavioral genetics

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Socioeconomic status (SES) has robust relations with children's cognitive ability and academic achievement (McLoyd, 1998; Noble, McCandliss, & Farah, 2007; White, 1982), and SES-related disparities widen over the course of child development (Heckman, 2006; Tucker-Drob, 2010). Theoretical work in behavioral genetics has suggested that one major pathway through which SES-related disparities emerge may be an interaction between cumulative environmental disadvantage and genes: In more advantaged homes, children have the opportunity to evoke and select environmental experiences that allow them to maximize their genetic potential for cognitive development, whereas this process is stifled in disadvantaged homes (Bronfenbrenner & Ceci, 1994; Dickens & Flynn, 2001; Scarr & McCartney, 1983). Evidence supporting this hypothesis comes from recent empirical work indicating that the heritability of cognitive ability, although consistently estimated to be upward of 50% in the general population (McGue, 1997), is positively moderated by family SES. For example, Turkheimer, Haley, Waldron, D'Onofrio, and Gottesman (2003) found that the heritability of cognitive ability in 7-year-old twins was only 10% in low-SES families but was 72% in high-SES families. This Gene × SES interaction has been found across much of the life span, from middle childhood to middle adulthood (Harden, Turkheimer, & Loehlin, 2007; Kremen et al., 2005; Rowe, Jacobson, & Van den Oord, 1999), although some less conclusive findings have also been reported (Asbury, Wachs, & Plomin, 2005; van der Sluis, Willemsen, de Geus, Boomsma, & Posthuma, 2008).

This line of research suggests that the environment plays a substantial role in the expression of genetic variance in cognitive ability over the course of child development. However, it is not yet clear when in childhood this Gene × SES effect begins to emerge. The youngest children for whom a Gene × SES interaction on cognitive ability has been reported were 7-year-olds (Turkheimer et al., 2003), but it is possible that

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SES-related disparities in the realization of genetic potential begin much earlier in life. SES is, of course, associated with parents' ability to provide high-quality educational resources to their children, but SES-related disparities in children's life experiences precede the beginning of formal schooling (Bradley, Corwyn, McAdoo, & Coll, 2001). For example, compared with higher-SES parents, lower-SES parents spend less time with their children (Guryan, Hurst, & Kearney, 2008), are less able to allocate time spent with children in accordance with their children's developmental needs (Kalil, Ryan, & Corey, 2010), and are less sensitive in responding to their children's signals (Bradley & Corwyn, 2002; De Wolff & Ijzendoorn, 1997). As a result, children of lower-SES parents are less likely to "experience that their social initiatives are successful in establishing a reciprocal interchange with the mother" (De Wolff & Ijzendoorn, 1997, p. 571). Such proximal processes between child and parent are likely to be particularly important during infancy, when children cannot actively seek out interactions that fit their needs and must instead elicit experiences from their parents. Given the breadth of differences between the early experiences of children in high- and low-SES homes, SES-related disparities in the realization of children's genetic potentials for cognitive development may begin as early as infancy.

The present study tested this hypothesis using longitudinal data on infant twins (tested at age 10 months and age 2 years) from the Early Childhood Longitudinal Study, Birth Cohort (ECLS-B). Results from three statistical models are presented. The first model examined SES-related disparities in phenotypic (i.e., observed) mental ability. On the basis of previous research (e.g., Heckman, 2006; Tucker-Drob, 2010), we predicted that SES would be positively correlated with developmental gains in mental ability, such that the difference in average mental ability between low-SES children and high-SES children would increase between 10 months and 2 years. The second model examined the population-average contribution of genes and environmental factors to mental ability at 10 months and to change in mental ability between 10 months and 2 years. We predicted, again on the basis of previous research (Davis, Haworth, & Plomin, 2009; Fulker, DeFries, & Plomin, 1988), that genes would influence change in mental ability, such that heritability would be higher at age 2 years than at age 10 months. Finally, the third model tested whether SES interacts with genetic and environmental contributions to mental ability at 10 months and to change in mental ability between 10 months and 2 years. We predicted that SES would moderate the genetic contribution to change in mental ability, such that the increasing heritability of mental ability in infancy would be most evident for children from high-SES families. In sum, we predicted that by late infancy, there would be significant SES-related disparities not only in average levels of mental ability, but also in the heritable variation in mental ability.

Method

Participants

Participants were approximately 750 pairs of twins drawn from the ECLS-B.[1] These were all the twins in the data set (out of 800 twin pairs in total) for whom zygosity information was collected. The ECLS-B was designed to be representative of the population of American children born in 2001. Therefore, children were sampled from a range of locations, ethnicities, and incomes. The first wave of ECLS-B data was collected when the children were approximately 10 months old (mean age = 10.4 months; range = 7.2–19.5 months), and the second wave of data was collected when they were approximately 2 years old (mean age = 24.4 months; range = 20.9–33.1 months). At each wave, children participated in several direct assessments of their abilities and behavior; cognitive-assessment data at 10 months were available for 98% of our sample, and cognitive-assessment data at 2 years were available for 94%. Sixty-one percent of the children were Caucasian; 16% were African American; 16% were Hispanic; 2% were Asian; less than 1% were Pacific Islander, American Indian, or Alaska Native; and 4% were of mixed race (Table S1 in the Supplemental Material available online reports race-ethnicity proportions by SES). Controlling for race and the interaction of race with genetic and environmental factors did not change the pattern of results we found throughout the study.

Zygosity

To assess the twins' zygosity, trained observers responded to six questions about the similarity of same-sex twins with regard to hair color, hair texture, complexion, facial appearance, and earlobe shape. Previous research has found such physical ratings to be highly predictive (i.e., greater than 90%) of objectively determined zygosity (Forget-Dubois et al., 2003; Goldsmith, 1991; Price et al., 2000). Observers' responses to each feature were coded as 1 (no difference), 2 (slight difference) or 3 (clear difference). We summed the scores for each twin pair, and the result was a bimodal distribution of scores ranging from 6 to 18. On the basis of the shape of this distribution, we classified twin pairs whose scores fell in the range from 6 to 8 as monozygotic (MZ). All other twin pairs were classified as dizygotic (DZ). Approximately 25% of twin pairs were classified as MZ, 35% as same-sex DZ, and 40% as opposite-sex DZ. To check the validity of these ratings, we computed a composite based on parents' responses to the same questions and dichotomized the scores in the same way. The correlation (φ) between the two dichotomized distributions was .80. We report the results of analyses that used the zygosity determined from observers' similarity ratings. Analyses that used the zygosity determined from parent ratings of their twins' similarity produced the same pattern of results, as did analyses in which twins with physical-similarity scores of 7, 8, and 9 were excluded.

Measures

Mental ability. At each of the two data-collection waves (10 months and 2 years), an experimenter administered the Bayley Short Form–Research Edition (BSF-R; for details, see Andreassen & Fletcher, 2007), which is a shortened version of the Bayley Scales of Infant Development, Second Edition (Bayley, 1993). The BSF-R has a Mental Scale (composed of up to 29 items administered at age 10 months and up to 33 items administered at age 2 years) and a Motor Scale (composed of up to 35 items administered at age 10 months and up to 32 items administered at age 2 years). Both scales have been extensively validated, using item response theory, for measurement invariance, unidimensionality, and discriminant validity relative to one another (Andreassen & Fletcher, 2007). Moreover, each scale has been placed on a vertical metric that is appropriate for assessing developmental change across time.

Our study used scores from the Mental Scale only. Example items from this scale include pulling a string to ring a bell, putting three cubes in a cup, repeating vowel-consonant combinations, matching pictures, and sorting pegs by color. At the first data-collection wave, the mean score was 71.14 (SD = 9.09, range = 33.84–112.58), and the reliability was .81. At the second data-collection wave, the mean score was 122.57 (SD = 10.80, range = 92.61–159.04), and the reliability was .88. The correlation between MZ twins was .80 at the first wave and .76 at the second wave. The correlation between DZ twins was .77 at the first wave and .68 at the second wave. All correlations were significant ($p < .001$).

SES. SES was computed using parental survey data from the first data-collection wave. SES was a composite of five variables: paternal education, maternal education, paternal occupation, maternal occupation, and family income (Hollingshead, 1975). Each of these variables was standardized in the larger ECLS-B sample to a mean of 0 and a standard deviation of 1. In the larger ECLS-B sample, the SES composite had a mean of –0.05 and a standard deviation of 0.86; in the twin subsample, its mean was 0.13 and its standard deviation was 0.87. SES ranged from –2.13 to 2.12 in the twin subsample. The correlation between SES and BSF-R Mental Scale scores was .05 (n.s.) at age 10 months and .32 ($p < .001$) at age 2 years. Figure S1 in the Supplemental Material is a histogram of the distribution of SES scores.

Analytical methods

We fit three structural equation models using full-information maximum-likelihood estimation in MPlus statistical software (Muthén & Muthén, 2010). Each model specified two latent factors per twin: a factor representing initial BSF-R performance at age 10 months (y_0) and a factor representing the change in performance between age 10 months and age 2 years (y_Δ). Both the initial-performance factor and the change factor were regressed onto the SES variable. In all models, BSF-R_{10m} represents mental-ability scores at the 10-month wave, BSF-R_{2y} represents mental-ability scores at the 2-year wave, t stands for twin, p represents twin pair, and s equals the main effect of SES.

Model 1: main effect of SES on change in mental ability. To begin our analysis, we estimated a model for phenotypic change in mental ability. This model was written as follows:

$$\text{BSF-R}_{10m,t,p} = y_{0,t,p}$$
$$\text{BSF-R}_{2y,t,p} = y_{0,t,p} + y_{\Delta,t,p}$$
$$y_{0,t,p} = s_0 \times \text{SES}_p + u_{0,t,p}$$
$$y_{\Delta,t,p} = s_\Delta \times \text{SES}_p + u_{\Delta,t,p}$$

In this model, u_0 and u_Δ are residual terms that respectively represent variation in initial performance and change that is unaccounted for by SES.

Model 2: genetic and environmental influences on change in mental ability. Next, we used a biometric model for twin data to identify the relative contributions of genes and environment on infant mental ability at age 10 months and on the developmental change in infant mental ability between 10 months and 2 years. Each latent factor (y_0 and y_Δ) was modeled as a linear combination of three standardized (z-scored) biometric components: an additive genetic component (A), a shared-environmental component (C, i.e., all environmental influences that make twins similar), and a nonshared-environmental component (E, i.e., all environmental influences that make twins less similar, plus measurement error).

Consistent with genetic theory, the correlation between temporally corresponding A components in the first and second members of each twin pair was fixed to 1.0 in MZ twin pairs and to .5 in DZ twin pairs (DZ pairs, like regular siblings, share approximately 50% of their genetic variance).[2] The temporally corresponding C components were, by definition, perfectly correlated across twin pairs, whereas the temporally corresponding E components were, by definition, uncorrelated across twin pairs. All regression coefficients in the model were constrained to be the same for both twins in each pair, and for both MZ and DZ twins. SES was included as a covariate to account for its main effect on mental-ability scores. Thus, mental ability at age 10 months and at age 2 years was predicted as follows:

$$\text{BSF-R}_{10m,t,p} = y_{0,t,p}$$
$$\text{BSF-R}_{2y,t,p} = y_{0,t,p} + y_{\Delta,t,p}$$
$$y_{0,t,p} = s_0 \times \text{SES}_p + (a_0) \times A_{0,t,p} + (c_0) \times C_{0,t,p} + (e_0) \times E_{0,t,p}$$
$$y_{\Delta,t,p} = s_\Delta \times \text{SES}_p + (a_\Delta) \times A_{\Delta,t,p} + (c_\Delta) \times C_{\Delta,t,p} + (e_\Delta) \times E_{\Delta,t,p}$$

The effects of the additive genetic, shared-environmental, and nonshared-environmental components on initial level and change in mental ability are represented with the a, c, and e regression coefficients, respectively.

Model 3: socioeconomic differences in genetic and environmental influences on change in mental ability. In our final model, the effects of *A*, *C*, and *E* were allowed to vary as functions of SES (Purcell, 2002). That is, each regression path was modeled as a combination of a main effect (*a*, *c*, and *e*) and an interaction with SES (*a*′, *c*′, and *e*′). This model is written as follows:

$$\text{BSF-R}_{10\text{m},t,p} = y_{0,t,p}$$
$$\text{BSF-R}_{2\text{y},t,p} = y_{0,t,p} + y_{\Delta,t,p}$$
$$y_{0,t,p} = s_0 \times \text{SES}_p + (a_0 + a'_0 \times \text{SES}_p) \times A_{0,t,p} + (c_0 + c'_0 \times \text{SES}_p) \times C_{0,t,p} + (e_0 + e'_0 \times \text{SES}_p) \times E_{0,t,p}$$
$$y_{\Delta,t,p} = s_\Delta \times \text{SES}_p + (a_\Delta + a'_\Delta \times \text{SES}_p) \times A_{\Delta,t,p} + (c_\Delta + c'_\Delta \times \text{SES}_p) \times C_{\Delta,t,p} + (e_\Delta + e'_\Delta \times \text{SES}_p) \times E_{\Delta,t,p}$$

Model 3 is depicted as a path diagram in Figure 1. Note that when the interaction parameters (*a*′, *c*′, and *e*′) are fixed to 0, Model 3 is identical to Model 2.

Results

Model 1: main effect of SES on mental development

The three major results from the phenotypic analyses are shown in Table 1. First, at 10 months, SES was not related to mental ability. Second, as would be expected, mental ability increased dramatically between age 10 months and age 2 years. The average change was 50.85 points, which corresponds to about 5 standard-deviation units relative to the variation observed at 10 months. Third, there was a statistically significant relation between SES and the magnitude of this change. An increase of 1 standard deviation of SES was associated with about 3.6 points more developmental gain between 10 months and 2 years. Although this effect may appear to be small relative to the overall magnitude of gain over the study period, it is a moderate effect relative to the individual differences in mental ability at 10 months: 3.6 points of gain is more

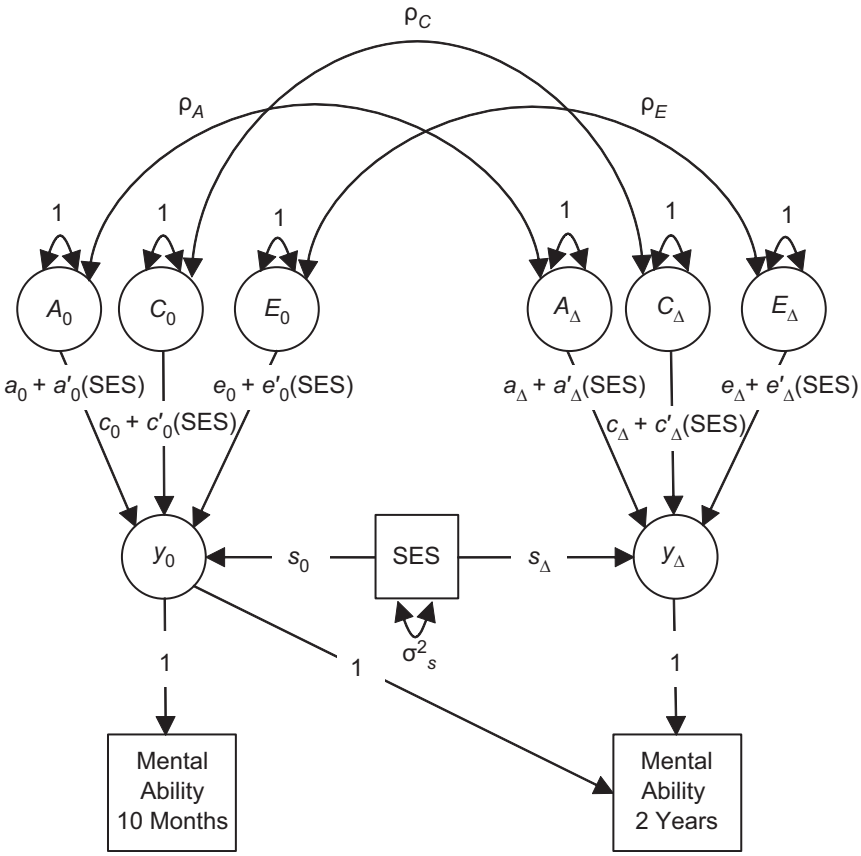

Fig. 1. Path diagram of the behavioral genetic model (Model 3) fit to mental-ability scores at age 10 months and age 2 years. Participants were tested with the Bayley Short Form–Research Edition (Andreassen & Fletcher, 2007). This diagram represents one half of the model (i.e., one twin in each pair). Single-headed arrows represent regression relations, and curved double-headed arrows represent variances and covariances. The factors y_0 and y_Δ represent baseline mental ability and change in mental ability, respectively. The factors *A*, *C*, and *E* represent additive genetic influences, shared-environmental influences, and nonshared-environmental influences, respectively. Each biometric path was modeled as a combination of a main effect (*a*, *c*, and *e*) and an interaction with socioeconomic status (SES; *a*′, *c*′, and *e*′). The regression of the factor on SES is denoted by *s*.

Table 1. Parameter Estimates for Model 1

Parameter	Estimate	95% CI
s_0	0.53	[–0.19, 1.26]
s_Δ	3.56*	[2.63, 4.50]
μ_0	71.06*	[70.43, 71.69]
μ_Δ	50.85*	[50.04, 51.66]
σ^2_{u0}	82.14*	[74.56, 89.73]
$\sigma^2_{u_\Delta}$	142.90*	[130.29, 155.52]
σ_{u0, u_Δ}	–60.35*	[–68.28, –52.43]

Note: The –2 log likelihood of this model was 21,419.60. The model included 13 free parameters. The subscript 0 represents the baseline (age 10 months) wave of data collection. The subscript Δ represents the change from the baseline wave to the follow-up (age 2 years) wave of data collection. CI = confidence interval; s = regression on socioeconomic status; μ = mean; σ^2 = variance; σ = covariance; u = variation unaccounted for by socioeconomic status.
*$p < .05$.

than one third of a standard deviation of baseline mental ability. Overall, the phenotypic results indicate that higher-SES children experience more rapid developmental gains in mental ability than lower-SES children do, such that SES-related disparities in mental ability are evident by the time that children are 2 years old. These findings are illustrated in Figure 2, which plots longitudinal age trends in mental ability for children with low, average, and high levels of SES.

Model 2: genetic and environmental influences on mental development

Model 2 estimated the relative contributions of genes, shared environment, and nonshared environment to infants' initial level of mental ability and to developmental change in mental ability between age 10 months and age 2 years. Parameter estimates from Model 2 are presented in Table 2. Note that adding biometric components to Model 1 did not change the model's fit. Model fit was excellent for both models, $\chi^2(23) = 21.77$, $p = .53$.

On the basis of the parameters of Model 2, we computed heritability coefficients for mental ability at age 10 months and at age 2 years, as well as the heritability of change in mental ability. These heritability coefficients represent the variability accounted for by the additive genetic component (A) as a proportion of the total variability accounted for by all three of the biometric components (A, C, and E). The heritability of mental ability at 10 months was 2% ($p = .67$), the heritability of the change in mental ability between 10 months and 2 years was 23% ($p = .003$), and the heritability of mental ability at 2 years was 23% ($p = .001$). Thus, the effect of genes on mental ability increases over infant development (see Fig. S2 in the Supplemental Material for a plot of this result).

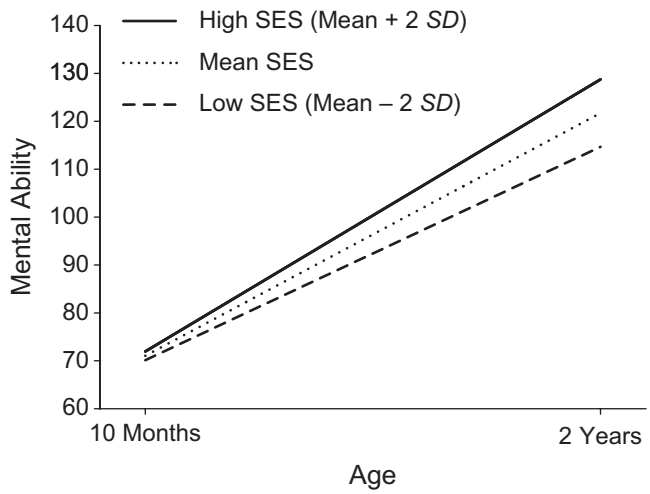

Fig. 2. Mean longitudinal age trends in mental-ability scores for children being raised in homes with low, mean, and high levels of socioeconomic status (SES). Participants were tested with the Bayley Short Form–Research Edition (Andreassen & Fletcher, 2007). These trends are based on parameter estimates from Model 1.

Model 3: socioeconomic differences in genetic and environmental influences on mental development

Model 3 tested whether the additive genetic, shared-environmental, and nonshared-environmental influences on change in mental ability were moderated by SES. Parameter estimates from Model 3 are shown in Table 2. The pattern of significant main effects of A, C, and E was virtually identical to results obtained from Model 2. Model 3 fit the data significantly better than Model 2 did ($p < .001$). None of the interactions between SES and the effects of A, C, and E on mental ability at 10 months (a'_0, c'_0, e'_0, respectively) was significant. For developmental change in mental ability, however, there were significant interactions between SES and genes (a'_Δ) and between SES and shared environment (c'_Δ). Figure 3 illustrates these interaction effects by plotting the amounts of variance in change in mental ability accounted for by genetic, shared-environmental, and nonshared-environmental influences as functions of SES. For children of low SES, almost all change is due to the shared environment, and genes play a negligible role in the development of mental ability. For children of high SES, genes play a substantial role in the development of mental ability, whereas less change is due to the shared environment.

Further illustrating these findings, Figure 4 displays the Model 3–implied amounts of variance in mental ability at age 10 months and age 2 years that were accounted for by genes, the shared environment, and the nonshared environment at three levels of SES: 2 standard deviations below the mean SES, the mean SES, and 2 standard deviations above the mean SES (Fig. S3 in the Supplemental Material plots these variance components as proportions). This figure clearly shows the three-way interaction of age, SES, and genes on mental ability. Note in particular the effects of genes. At 10 months, there was very little genetic variance in mental ability at any

Table 2. Parameter Estimates for Model 2 and Model 3

	Model 2		Model 3	
Parameter	Estimate	95% CI	Estimate	95% CI
a_0	1.34	[–1.76, 4.44]	0.98	[–2.73, 4.68]
a'_0			0.90	[–1.05, 2.84]
c_0	7.91*	[7.32, 8.50]	7.97*	[7.41, 8.52]
c'_0			–0.49	[–1.05, 0.06]
e_0	4.22*	[3.80, 4.63]	4.25*	[3.90, 4.61]
e'_0			–0.37	[–0.78, 0.04]
a_Δ	5.69*	[3.82, 7.55]	5.18*	[0.13, 10.23]
a'_Δ			2.28*	[0.20, 4.37]
c_Δ	8.38*	[7.25, 9.50]	8.49*	[7.34, 9.63]
c'_Δ			–1.16*	[–2.26, –0.07]
e_Δ	6.36*	[5.72, 7.00]	6.28*	[5.67, 6.89]
e'_Δ			–0.34	[–0.91, 0.24]
ρ_A	–0.67	[–1.73, 0.40]	–0.69	[–1.89, 0.51]
ρ_C	–0.59*	[–0.69, –0.50]	–0.60*	[–0.74, –0.45]
ρ_E	–0.60*	[–0.69, –0.50]	–0.60*	[–0.74, –0.45]
s_0	0.53	[–0.19, 1.26]	0.54	[–0.19, 1.27]
s_Δ	3.56*	[2.63, 4.50]	3.59*	[2.64, 4.54]
μ_0	71.06*	[70.43, 71.69]	71.06*	[70.42, 71.70]
μ_Δ	50.85*	[50.04, 51.66]	50.85*	[50.03, 51.66]

Note: The –2 log likelihood of Model 2 was 21,419.60 and of Model 3 was 21,449.08. Model 2 included 13 free parameters; Model 3 included 19 free parameters. Model 3 fit the data significantly better than Model 2 did, $\chi^2(6) = 29.48$, $p < .001$. The subscript 0 represents the baseline (age 10 months) wave of data collection. The subscript Δ represents the change from the baseline to the follow-up (age 2 years) wave of data collection. *A*, *C*, and *E* are latent factors corresponding to additive genetic influences, shared-environmental influences, and nonshared-environmental influences, respectively. *a* = regression on genetic factor; *c* = regression on shared-environment factor; *e* = regression on nonshared-environment factor; *a′* = regression on Gene × Socioeconomic Status (SES) interaction; *c′* = regression on Shared Environment × SES interaction; *e′* = regression on Nonshared Environment × SES interaction; CI = confidence interval; ρ = factor intercorrelation; *s* = regression on SES; μ = mean.
*$p < .05$.

level of SES. At 2 years, however, socioeconomic differences in genetic influences on mental ability were quite large. For low-SES infants, genes played virtually no greater role at 2 years than at 10 months, whereas for high-SES infants, genes accounted for nearly 50% of the variability in mental ability at 2 years.

Discussion

This article reports three main findings. First, SES-related disparities in mental ability emerged over the course of infant development: SES was unrelated to mental ability at 10 months, but it was related to change in mental ability between age 10 months and age 2 years, such that by 2 years, each standard deviation of SES was associated with approximately one third of a standard deviation of mental ability. Second, at the population level, genes began to play a role in the development of mental ability between 10 months and 2 years. Third, the extent to which genes influenced mental development differed according to SES, such that by 2 years, genetic influences on mental ability were larger for children being raised in higher-SES homes than for children being raised in lower-SES homes.

These findings are consistent with the hypothesis that the emergence of genetic variation in complex behavioral phenotypes depends on reciprocal interactions between the child and his or her environment (Bronfenbrenner & Ceci, 1994; Dickens & Flynn, 2001; Scarr & McCartney, 1993). According to this perspective, poor socioeconomic contexts constrain children's opportunities to engage with supportive environments that foster cognitive growth, and this constraint results in the suppression of genetic influences on mental ability. In particular, socioeconomic disadvantage is likely to impair an infant's ability to elicit responsive and developmentally appropriate stimulation from caregivers (i.e., evocative processes). However, later in childhood, the role of SES likely shifts, such that socioeconomic disadvantage restricts genetic variation in cognitive ability by limiting opportunities for individuals to actively seek out educational and social experiences that are congruent with their own genetically influenced interests and motivations (Scarr & McCartney, 1983). We should emphasize that this specific mechanism may not generalize to all psychological outcomes and that there are likely to be some outcomes (e.g., attention-deficit/hyperactivity disorder) that are actually more heritable in higher-risk social environments than in lower-risk social environments (Pennington et al.,

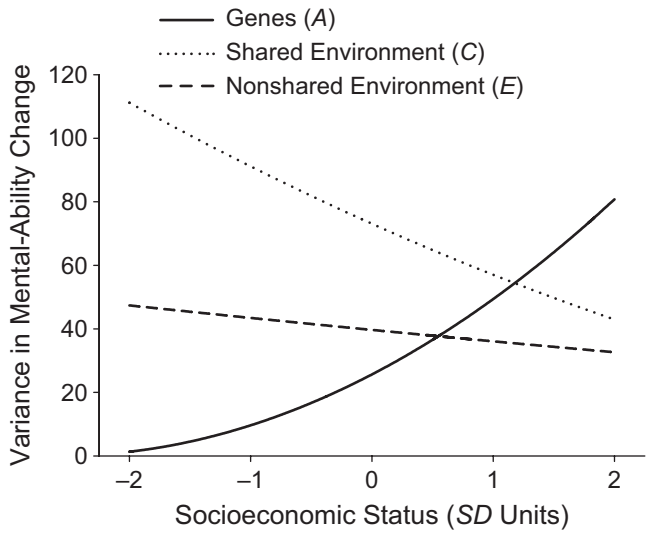

Fig. 3. Amounts of variance in longitudinal change in infant mental-ability scores accounted for by genes (*A*), the shared environment (*C*), and the nonshared environment (*E*) as functions of socioeconomic status. Participants were tested with the Bayley Short Form–Research Edition (Andreassen & Fletcher, 2007) at ages 10 months and 2 years. These trends are based on parameter estimates from Model 3.

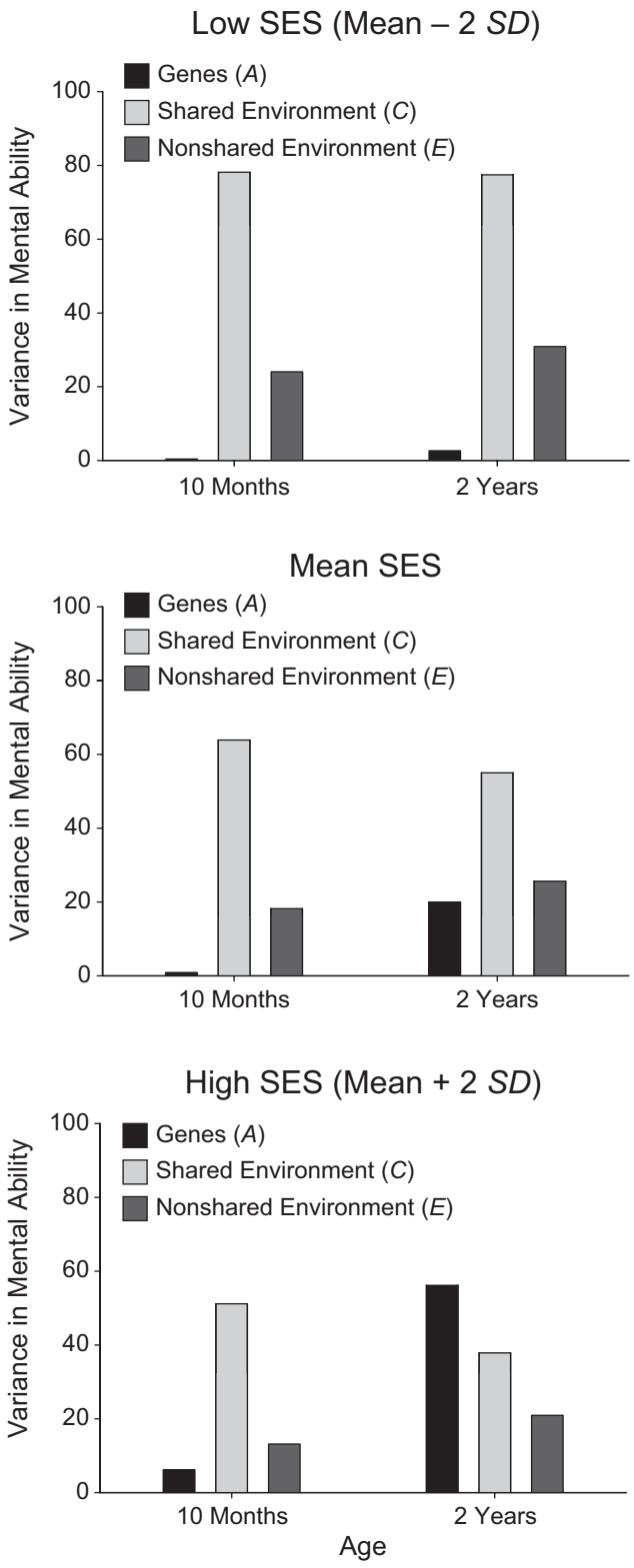

Fig. 4. Amounts of variance in mental-ability scores accounted for by genes (*A*), the shared environment (*C*), and the nonshared environment (*E*) at ages 10 months and 2 years. Results are shown separately for children at low, mean, and high levels of socioeconomic status (SES). Participants were tested with the Bayley Short Form–Research Edition (Andreassen & Fletcher, 2007). These trends are based on parameter estimates from Model 3.

2009). Additionally, although SES is often conceived of as a purely environmental variable, socioeconomic groups may differ in the frequencies of specific genetic polymorphisms.

These findings build on a growing body of literature that highlights the importance of early life experiences for cognitive development (e.g., Nelson et al., 2007). Current evidence suggests that, although children maintain a great deal of neurobiological and behavioral plasticity well past infancy (Brehmer, Li, Müller, von Oertzen, & Lindenberger, 2007; Garlick, 2002), the predictive validity of infant mental ability for later cognitive ability is moderate (Bornstein & Sigman, 1986; Rose & Feldman, 1995). We agree with Bornstein and Sigman (1986), who have strongly argued against the perspective "that infancy might play little or no role in determining the eventual cognitive performance of the child and, therefore, that individuals could sustain neglect in infancy if remediation were later made available" (p. 269). Heckman (2006) has recently taken an economic perspective on this topic. He argued that prophylactic interventions for disadvantaged younger children produce much higher rates of return on what he termed "human skill formation" than later remedial interventions for older children and adults do. On the basis of this perspective, Heckman concluded that "at current levels of funding, we overinvest in most schooling and post-schooling programs and underinvest in preschool programs for disadvantaged persons" (p. 1901).

Our research makes an important contribution to the literature by establishing the developmental timing of Gene × SES effects on mental ability. However, future research will be necessary to address three remaining issues. First, as have previous studies, our study examined the moderation of genetic influences by only an omnibus index of SES. In order to translate the current findings into useful recommendations for policy and intervention, it will be important for future research to examine the specific aspects of SES that potentially contribute to Gene × SES effects. These range from contextual aspects of neighborhoods, schools, and homes to more proximal aspects of caregiver behavior. Second, it will be important to further investigate the developmental patterns of gene-environment correlation that have been hypothesized to underlie Gene × SES effects on mental ability and to identify specific child and caregiver characteristics that become matched to one another over time. Third, it will be important to identify the neurobiological foundations of Gene × SES effects on mental ability. There is evidence that genetic differences in cognitive ability are strongly related to genetic differences in brain volume and cortical thickness (Posthuma et al., 2002; Toga & Thompson, 2005) and that the population-level heritabilities of volumes of brain regions that have been linked with mental ability increase over child development (Lenroot & Giedd, 2008). This suggests that Gene × SES effects on mental ability in early childhood may be mediated by Gene × SES effects on measures of regional brain volumes.

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The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information may be found at http://pss.sagepub.com/content/by/supplemental-data

Notes

1. Sample sizes are rounded to the nearest 50 in accordance with ECLS-B data-security regulations.

2. The assumed values will be incorrect under conditions of assortative mating or when twin-pair zygosity is misclassified. Loehlin, Harden, and Turkheimer (2009) demonstrated that although varying the assumed genetic correlation between DZ twins may alter the magnitude of the estimated main effects of genes and environments, it does not appreciably alter the magnitude or significance level of the Gene × Environment interaction. We confirmed that this was the case in the analyses reported here by fitting models in which the MZ and DZ correlations deviated from 1 down to .85 and from .5 up to .65, respectively.

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