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Genetic and Environmental Influences on Vocabulary IQ: Parental Education Level as Moderator

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This article examines how parental education level moderates the genetic and environmental contributions to variation in verbal IQ. Data are from 1909 non-Hispanic Whites and African American sibling pairs from the National Longitudinal Study of Adolescent Health, which obtained nationally-based samples of identical (MZ) twins, fraternal (DZ) twins, full and half siblings, cousins (in the same household), and biologically unrelated siblings. In the whole sample, the variance estimate for heritability ($h^2 = .57$, $SE = .08$) was greater than that for shared environment ($c^2 = .13$, $SE = .04$). Both heritability and the shared environmental estimate were moderated, however, by level of parental education. Specifically, among more highly educated families, the average $h^2 = .74$ ($SE = .10$) and the average $c^2 = .00$ ($SE = .05$). Conversely, among less well-educated families, heritability decreased and shared environmental influences increased, yielding similar proportions of variance explained by genetic and environmental factors, average $h^2 = .26$ ($SE = .15$), and average $c^2 = .23$ ($SE = .07$).

INTRODUCTION

Genetically influenced traits are sometimes expressed more in certain environments than in others. For example, in bacteria the presence of particular nutrients in the environment can regulate gene expression. Likewise, experiments on learning in rats have shown that enriched rearing can improve the performance of genetically dull animals to about the level of genetically bright ones (Cooper & Zubek, 1958). These two cases illustrate how the environment may moderate the genetic expression of certain traits. One way of testing this in humans is to examine whether the proportion of variance in phenotypes due to genetic differences among individuals (i.e., the heritability) varies across different environmental contexts.

Previous research suggests that variance in IQ is primarily attributable to genetic influences and not to environmental influences that are shared by children in the same family. By definition, common, or shared, environmental influences are those influences that are shared among family members and serve to make family members similar to one another. Shared environmental influences often include factors such as family structure and socioeconomic status (SES). In contrast, nonshared environmental influences are factors that are not shared among family members and contribute to differences among them. Examples of nonshared environmental influences among children and adolescents are peer groups, perinatal traumas or birth defects, and differential parental treatment. Scarr presented evidence that genetic influences on children's IQ were stronger than common family environmental influences when families were working class to affluent (Scarr, 1992, 1993). Likewise, in her adolescent adoption study (Scarr & Weinberg, 1978), the midparent-child correlation for IQ was .68 for biological offspring, but only .13 for adoptive offspring. This pattern of parent-child associations indicated a substantial genetic influence on variation in IQ (i.e., heritability) and only weak shared environmental influences. Moreover, the same study also examined shared environmental effects via correlations between biologically unrelated siblings. These correlations were close to zero for adolescent and young adult unrelated siblings, suggesting that a shared family rearing environment had little impact on sibling similarity. On the basis of these findings and other data on IQ, Scarr (1992) argued for *no* differential effect on IQ between average and above average family environments.

Nonetheless, Scarr expected environmental influences to be of greater importance at the harmful end of an environmental continuum, which would be rarely represented in adoptive families, stating, "Environments that fall outside of the species normal range will not promote normal developmental patterns" (1992, p. 5). Scarr's theory implies that the environment may moderate the relative genetic and environmental influences on variation in development, because heritability may decrease and shared environmental effects may increase if a sample includes individuals from these more harmful family environments.

Likewise, in their bioecological theory of nature-nurture effects, Bronfenbrenner and Ceci (1994) argued against the concept of a "single" heritability. Their argument was that proximal processes, mean-

ing the long-term interactions between children and their environments, are a necessary condition for the expression of any genetic trait. As they suggested, traits do not spring forth from genotypes fully formed—in the developmental course of any single individual, they must be nurtured. They further reasoned that heritability is an estimate of the expression of genetic potential for a given trait. Thus, one specific prediction from their bioecological theory was that better environments (i.e., enhanced proximal influences) should increase heritability because genetic potential would be more fully realized. At the same time, however, they took the view that environmental influences do *not* diminish in strength as environments improve. They stated,

> We take issue . . . with the prevailing conception of the reaction range simply as a curved plane, similar to a bent piece of chicken wire that quickly straightens out to become horizontal . . . This representation reflects the commonly held position among behavioral geneticists that environment exerts an important influence only in severely deprived environments . . . (p. 571)

This last argument presents something of a paradox, however. Given that the overall phenotypic variation is the sum of genetic variation and environmental variation, if heritability increases in enriched environments, then by definition, less of the remaining variability would be environmental in origin. This difference aside, Scarr (1992) and Bronfenbrenner and Ceci (1994) are in agreement that environments may moderate the expression of genetic dispositions.

Despite the conceptual allure of an environmental context by heritability interaction, only a handful of studies have found that heritability is moderated by context. Two early studies found support for the hypothesis that the heritability of IQ would be greater in more advantaged environments, and that shared environmental influences would be greater in less advantaged environments (Fischbein, 1980; Scarr-Salapatek, 1971). These studies, however, had important methodological limitations. Specifically, in the sample of twins from the Philadelphia area used by Scarr-Salapatek (1971), the zygosity of twins was not measured. Instead, Scarr-Salapatek calculated the correlations for identical (MZ) and fraternal (DZ) twins based on the estimated proportions of same-sex MZ and DZ twins. In addition, social class was measured at the census-tract level, not through actual measurements of family economic background. Although data from the Swedish twins used by Fischbein (1980) did include measured zygosity and family-level social class, social class was used as a categorical variable. Thus, the number of pairs in the lower social class was quite small (n = 14 MZ and n = 24 DZ pairs). Given the small sample size, it is possible that the correlations found for the lower social class in this particular study are unreliable.

Other studies have found evidence for secular changes in heritability estimates. For example, the heritability of the age of first sexual intercourse has been found to depend on birth cohort (Dunne et al., 1997). Specifically, the heritability of sexual intercourse onset was considerably lower among Australian men and women older than 40 years than among younger Australian men and women. Thus, the shared environment effect (i.e., birth cohort) moderated genetic influence on age of first intercourse. One explanation is that strict social norms may have prevented the expression of any genetic propensity toward early sexual intercourse in the older cohorts, but not in the younger ones. Two additional studies found some evidence that the heritability of educational attainment (Heath et al., 1985) and intelligence (Sundet, Tambs, Magnus, & Berg, 1988) increased with more recent cohorts. Again, these studies suggest that the greater equality of educational opportunities available in more recent cohorts has augmented the proportion of individual differences that is due to genetic variation. Although these examples suggest that the heritability of certain phenotypes may vary across environments, these exceptions to the general rule are rare.

One reason for the general lack of evidence for moderating effects of the environment on estimates of heritability and shared environmental effects is that the standard behavioral genetic model uses latent, that is, unmeasured, variables as indicators of environmental influences (Anastasi, 1958). As the above examples serve to illustrate, measured environmental variables increase statistical power to detect moderating effects of the environment. Only a measured environment can specify the circumstances in which genetic and environmental effects may change. Thus, the prior absence of findings of environmental moderation may be partly a function of limitations of the research design.

A different possibility, however, is suggested by an alternative interpretation of genetic effects. As argued by many behavior geneticists, prominently by Scarr and McCartney (1983), many genetic effects may also occur through gene → environment (G→E) correlations, a theory that "genes drive experience." Such correlations lead to particular genotypes being nonrandomly distributed across different environments. In active G→E correlations, individuals may "pick" (consciously or unconsciously) those environments that most reinforce and sustain their genetic poten-

tials. For example, bright children might enjoy reading more than dull children. This process is often referred to as a "self-selection" effect. In the reactive form of G→E correlation, social reactions to peoples' phenotypes may create an association between genotypes and environments. For example, teachers might recognize a greater potential among bright children and may encourage them more strongly in their studies. Both of these G→E correlations serve to reduce the frequency of organism-environment "mismatches," such as bright children failing to read books. If bright versus dull children could be randomly assigned to high versus low book reading conditions, moderating effects of the environment (in this example, level of book reading) on IQ might emerge. In real life, there are too few children in two of the four possible experimental conditions: bright children with little reading experience, and dull children with voracious reading habits. In this way, the existence of a G→E correlation may greatly reduce the power to detect environmental moderation. In the standard behavioral genetic model G→E correlations are estimated as part of the genetic variation (Plomin, DeFries, McClearn, & Rutter, 1997).

In this article, we explore whether level of parental education (measured as a continuous variable) moderates the genetic and environmental contributions to variation in verbal IQ among adolescents. Although parental education may be related to children's IQ via shared genes, parental education can also be considered a measure of "environmental quality," because it is associated with the availability of intellectual stimulation and financial resources within the family. Moreover, because parental education is the same for siblings within a family, it is typically considered to be a shared environmental influence. Our expectation was that heritability estimates would be greater for adolescents from families with more parental education than from families with less parental education, because the former may typically provide a satisfactory context for intellectual development. Conversely, shared environmental influences should be greater among families with less parental education.

METHOD

Sample and Procedure

The National Longitudinal Study of Adolescent Health (Add Health) was designed to assess the health status of adolescents and explore the causes of adolescent health-related behaviors. Add Health began with a total sample of over 90,000 adolescents surveyed in school. The primary sampling frame was all high schools in the United States that had an 11th grade and had an enrollment of at least 30 students. A random sample of 80 high schools was selected from this sampling frame, taking into consideration enrollment size, region, school type, ethnicity, and urbanicity. The largest feeder school for each high school also was included in the sample. Seventy-nine percent of the schools initially contacted agreed to participate. Schools that refused to participate were replaced by another school in the same sampling stratum, resulting in a final sample of 134 schools. Within these schools, 90,118 of 119,233 eligible students (75.6%) in grades 7 to 12 completed a self-administered instrument for optical scanning.

Using both school roster information and information provided by adolescents during the school interview, a random sample of 15,243 adolescents also was selected for a detailed home interview. This sample was stratified by gender and age. The in-home interview was completed by 12,188 (79.5%) of these adolescents. In addition to this core sample, a number of subsamples also was selected for the home interview. These subsamples included samples of disabled adolescents, adolescents from well-educated African American families, adolescents from typically understudied racial and ethnic groups, and a special sibling pairs sample. Overall, 20,745 adolescents completed the in-home interview. Details on the Add Health study have been reported elsewhere (Resnick et al., 1997).

Included in the 20,745 adolescents is the Add Health *pairs sample* (N = 3,139 sibling pairs), which was selected using information from the in-school questionnaires and school rosters. Specifically, all adolescents who were identified as twin pairs, half siblings, or unrelated siblings raised together were selected for the home interview. Home interview data were obtained from both the target adolescent and his/her sibling. The sibling pairs were selected regardless of whether they were present on the day of the school interview, and regardless of whether the siblings attended the same school. A probability sample of full siblings also was drawn. The unrelated sibling pairs also included cousins who resided in the same household at the time of the survey. Thus, the pairs sample consists of all genetically informative sibling pairs. The average age in both the full sample and the sibling pairs sample is approximately 16 years (SD = 1.7).

The present sample contains 204 opposite-sex dizygotic twins. The majority of same-sex twins were diagnosed as either monozygotic (MZ, n = 247 pairs) or dizygotic (DZ, n = 200) on the basis of their self-reports of confusability of appearance. Confusability of appearance scales have been found to have greater

than .90 agreement with zygosity determination based on DNA evidence (Spitz et al., 1996). Eighty-nine twin pairs of uncertain diagnosis were further classified by their match on DNA genetic markers (47 DZ, 42 MZ). Twins were diagnosed as MZ if they were the same for five or more genetic makers (error rate approximately 4/1,000 or less) and DZ if they were different at one or more markers. Zygosity determination could not be ascertained for an additional 43 twin pairs. For the present analyses, these undecided (UD) twin pairs were combined with the DZ twins. Given that the UD group most likely contains a proportion of true MZ twin pairs, this may slightly inflate our estimate of shared environment. (All analyses were redone without the UD twin pairs, and results did not differ from those presented here.) Although the majority of households had just two siblings enrolled in the study, in households with multiple siblings, all possible pairs were made (e.g., siblings A, B, and C form pairings AB, AC, and BC). This use of all pairs makes the significance tests reported later slightly liberal.

The data reported here come from the in-home Wave I questionnaire. This in-home interview was completed between May and December 1995. All respondents were given the same interview, which took from one to two hours to complete. All data were recorded on laptop computers. Sensitive questions were asked via audio files drawn off the hard disk to coincide with presented questions. In addition to maintaining data security, this minimized the potential for interviewer bias.

Table 1 presents the percentages of adolescents in various racial and ethnic groups. Because variance heterogeneity increases with the large number of racial and ethnic groups included in the Add Health sample, the present analyses are restricted to adolescents pairs who belonged to one of the two largest racial and ethnic groups: non-Hispanic Whites (hereafter, Whites; n = 1,457 pairs, 46% of the sibling pairs sample) and African Americans (hereafter, Blacks; n = 661 pairs, 21% of the sibling pairs sample). We considered including siblings from the third-largest group, Hispanic Americans (n = 227 pairs, 7.2% of the sample); however, detailed analyses revealed that both siblings indicated that in only 44% of the Hispanic American families was English the primary language spoken at home. A one-way analysis of variance (ANOVA) revealed that Hispanic Americans from non-English-speaking homes scored significantly lower on the Verbal IQ test than adolescents from English-speaking home (M IQ = 88.5, SD = 14.2; M IQ = 93.8, SD = 11.0, respectively), $F(1, 394) = 16.5$, $p < .01$. Because this systematic variation meant that there was considerable heterogeneity within the Hispanic American group, a decision was made to exclude these individuals. Nonetheless, over two thirds (67%) of the pairs sample were either White or Black.

Table 1 Ethnic and Racial Characteristics of the Add Health Samples

Variable	Full Sample	Sibling Pairs Sample
N	20, 745	6,278
Mean age[a]	16.1 (1.7)	16.0 (1.7)
Ethnicity		
Non-Hispanic Whites[b]	50.2	49.1
African American[c]	20.8	22.8
Hispanic[d]	8.9	9.0
Asian[e]	2.3	2.0
Filipino	2.6	2.9
Cuban	2.2	1.0
Native American	.5	.5
Central/South American	1.4	1.3
Puerto Rican	2.1	1.7
Other	7.2	7.2
Missing	1.7	2.4

[a] Standard deviations are in parentheses.
[b] Percentages of Whites are somewhat lower than national averages due to the over-sampling of racial and ethnic minorities in the Add Health Study.
[c] Percentages of African Americans are greater than national averages, due primarily to the over-sampling of African American adolescents from college-educated families.
[d] Includes Mexican Americans and Chicanos.
[e] Includes Chinese, Japanese, Korean, and Vietnamese adolescents.

Some pairs were excluded for special reasons. Twenty-two pairs were deleted because their relationship was not exactly a "sibling" relationship. Specifically, these adolescents were identified as either aunt/uncle-nephew-niece pairs, boyfriend-girlfriend living together, or unrelated adolescents living in a group home. We reasoned that these individuals would be less likely to experience similar family environments; thus they were deleted from the present analyses. Further, 153 pairs were missing IQ data from one or both siblings. Thirteen pairs were outliers (i.e., one or both sibling scored less than 50 on the verbal IQ scale). They were also deleted. Finally, a small number of pairs (n = 23) was deleted because of missing data regarding parental education. In sum, the present analyses are conducted on 1909 sibling pairs (1,322 White pairs, 587 Black pairs; 60.8% of the pairs sample; 90.1% of pairs who were identified as Black or White).

Adolescents were from a variety of different living arrangements, with nearly one half (45.8%) living in two-parent, biological families. Almost one quarter (23.7%) lived in single-parent households (88% with biological mothers), and an additional 21.2% lived in

stepfamilies (78% with biological mothers and stepfathers). Just over 2% (2.1%) lived in adoptive homes (with over 75% in two-parent adoptive homes), and less than 1% (.4%) were in foster homes. Finally, an additional 6.8% lived in "other" arrangements, most often with other relatives (as in the case of cousins).

Measures

Parental education. Adolescents were asked to indicate the highest level of education completed by their residential mother (including biological mother, stepmother, adoptive mother, foster mother, and aunt) and residential father (including biological father, stepfather, adoptive father, foster father, and uncle). Of the 59% of individuals who reported information for both mother and father (n = 2,251), the correlation between residential mother and residential father education was .53, $p < .001$. Thus, reports of mother and father education were averaged for each sibling (with adolescents in single-parent families given the score for a particular parent). Likewise, the reliability of the educational reports is indicated by the high correlation between the siblings' independent ratings of parental education, $r(1741) = .81$, $p < .001$. Thus, sibling reports of parental education were averaged to create a single composite score for family educational level. The possible educational categories were: 0 = never went to school; 1 = eighth grade or less; 2 = more than eighth grade, but did not graduate high school; 3 = high school graduate or completed a GED or went to business, trade, or vocational school instead of high school; 4 = business, trade, or vocational school after high school or some college; 5 = graduated from college or university; and 6 = professional training beyond 4-year college. Educational levels for both mothers and fathers by race are reported in Table 2. The modal level of education for both residential mothers and residential fathers was 3, which corresponds to a high school level education, and the mean level of average parental education was 3.7 (SD = 1.1), representing some post-high school education.

Add Health Picture Vocabulary Test (verbal IQ). This test is an abridged version of the Peabody Picture Vocabulary Test–Revised (PPVT-R). It was administered at the beginning of the in-home interview. This test of vocabulary involved the interviewer reading a word aloud. The respondent then selected the illustration that best fit the word. Each word had four, simple, black-and-white illustrations arranged in a multiple-choice format from which the respondent indicated his or her choice. For example, the word "furry" had illustrations of a parrot, a dolphin, a frog, and a cat from which to choose. There were 78 items on the verbal IQ test, and raw scores have been standardized by age. Whites had a higher IQ mean than Blacks. This racial difference would tend to increase sibling correlations in all sibling groups, because siblings usually share the same racial status. For this reason, the IQ scores were corrected for their association with racial group by using residuals from the regression of IQ on a dummy variable representing racial group (0 = White, 1 = Black). The subsequent regression analyses used these residual scores.

Table 2 Educational Levels of Residential Mothers and Residential Fathers, by Racial Group

	Mother		Father	
	White	Black	White	Black
N	2,415	1,039	2,004	443
No school	.0	.1	.0	0
Eighth grade or less	1.6	2.4	2.5	5.6
More than eighth grade, but less than high school	8.9	14.0	7.2	7.9
High school equivalent[a]	38.6	39.7	35.5	41.1
Some college	21.4	21.8	19.7	19.6
4-Year college degree	21.7	17.3	24.4	19.9
Graduate or professional training	7.7	4.6	10.8	5.9

[a] Includes high school graduate, GED, and business or trade school instead of high school.

RESULTS

Table 3 presents the sibling correlations for the full sample of Black and White adolescents. In general, the correlations order as expected by genetic theory. In other words, sibling correlations increase according to the level of genetic relatedness, from .07 for biologically unrelated sibling pairs to .73 for MZ twins. There is no suggestion of a specific twin effect because DZ twins and full-siblings were equally alike in IQ. Some correlations, however, suggested shared environmental effects. In particular, the half sibling and cousin correlations are greater than their theoretical

Table 3 Sibling Correlations for Peabody Picture Vocabulary IQ

Sibling Group	No. of Pairs	*r*
MZ twins	176	.73***
DZ twins	347	.39***
Full siblings	795	.39***
Half siblings	269	.35***
Cousins	118	.21*
Unrelated siblings	204	.07***

* $p < .05$; *** $p < .001$.

expectations based on a solely genetic model. Specifically, if genetic factors were the only influences on sibling similarity, then the half sibling correlation should be one half the full sibling correlation, and the cousin correlation should be one quarter the full sibling correlation ($r_{\text{full sibling}} = .39$). As can be seen from Table 2, the half sibling correlation of .35 and the cousin correlation of .21 exceeded the .195 and .098, respectively, that would be expected under a purely genetic model. Moreover, the twin groups indicated a small shared environmental effect (using the common formula for shared environment, $2r_{DZ} - r_{MZ} = .05$).

To explore the moderating effect of parental education, a DeFries-Fulker (DF) regression equation was used to estimate genetic and environmental parameters (DeFries & Fulker, 1985). The DF equation is an unstandardized regression equation. The phenotype of sibling 1 (P_1) is predicted from the phenotype of sibling 2 (P_2), the coefficient of genetic relatedness (R; with R = 1.0 for MZ twins, R = .5 for DZ twins and full siblings, R = .25 for half-siblings, R = .125 for cousins, and R = .0 for unrelated siblings), and the interaction of the latter two variables ($R \times P_2$). In this form, the unstandardized regression coefficient on P_2 is the estimate of the proportion of variance due to shared environmental effects (c^2) and the regression coefficient on the interaction term is the heritability coefficient (h^2; for details of how the unstandardized estimates translate into c^2 and h^2, see Cherny, DeFries, & Fulker, 1992; Rodgers & McGue, 1994). The DF model was run for the full sample, for Whites only, for Blacks only, and then for low and high parental education groups using high school graduate as the cutoff (i.e, low education = high school education or less, high education = more than a high school education). The parental educational groups differed in average adolescent (unadjusted) IQs: high education, *M* IQ = 103.0, *SD* = 13.0; low education, *M* IQ = 95.4, *SD* = 12.6, $F(1, 3816) = 257.6$, $p < .001$.

Table 4 summarizes the heritability and shared environment variance estimates obtained from the DF models. In the full sample, the heritability of IQ was .57, and the shared environmental effect was .13. Estimates of shared environmental (c^2) and genetic effects (h^2) were similar for Black and White adolescents (.13 versus .12, and .57 versus .58, respectively). The adjusted R^2 from this model was .17, $F(3, 3814) = 257.0$, $p < .001$. The similarity of the variance estimates for Blacks and Whites indicates that the two groups can be safely combined for further analyses. In the regressions that divided adolescents on the basis of their parents' education, we found a powerful moderation effect for both genetic and shared environmental influences. Heritability was greater for adolescents in homes with more educated parents (.74 versus .26), whereas the shared environmental estimate was lower (.00 versus .23).

To test whether these differences were significant, we used an extension of the DF analysis that added further interaction terms (for details, see LaBuda & DeFries, 1990). The full, extended equation is:

$$P_1 = b_o + b_1P_2 + b_2R + b_3ED + (b_4R{\cdot}P_2) + (b_5R{\cdot}ED) + (b_6P_2{\cdot}ED) + (b_7P_2{\cdot}R{\cdot}ED)$$

where P is verbal IQ (the subscripts 1 and 2 designate each sibling), R is the coefficient of genetic relatedness, and ED represents level of parental education, coded as a continuous variable. If level of parental education moderates the c^2 estimate, then the unstandardized regression coefficient on the interaction term $P_2{\cdot}ED$ (i.e., the b_6 coefficient) would be significant. Similarly, the unstandardized regression coefficient for the $P_2{\cdot}R{\cdot}ED$ term (b_7) is the test of whether the heritability of verbal IQ differs across education levels.

Table 4 DeFries-Fulker Regression Equation Heritability and Shared Environmental Variance Estimates

	No. of Pairs	c^2	$(\sigma_{SE})^a$	*t* Value	h^2	$(\sigma_{SE})^a$	*t* Value[b]
Full sample	1,909	.13	.04	3.25***	.57	.08	7.13***
Racial groups							
Whites	1,322	.12	.05	2.40**	.58	.10	5.80***
Blacks	587	.13	.07	1.86*	.57	.15	3.80***
Educational groups							
Low education	753	.23	.07	3.29***	.26	.15	1.73*
High education	1,156	.00	.05	.00	.74	.10	7.40***

Note: c^2, shared environmental effects; h^2, heritability; σ_{SE} = standard error. High education reflects greater than a high school education; low education reflects a high school education or less.

[a] Standard errors corrected for double-entry of data.

[b] Significance of *t* tests are based on one-tailed tests.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 5 Results from the Extended DF Regression to Test Whether Parental Education Moderates c^2 and h^2

	B	σ_{SE}[a]	*t* Value[b]
Intercept (b_0)	−9.78	1.87	−5.23***
c^2 (b_1)	.50[c]	.15	3.33***
R (b_2)	4.79	3.90	1.23
ED (b_3)	2.73	.49	5.57***
h^2 (b_4)	−.12[c]	.30	−.40
R·ED (b_5)	−1.25	.99	−1.26
c^2·ED (b_6)	−.12	.04	−3.00***
h^2·ED (b_7)	.19	.08	2.38***

Note: c^2, shared environmental effects; h^2, heritability; σ_{SE} = standard error; R, coefficient of genetic relatedness.
[a] Standard errors corrected for double-entry of data.
[b] Significance is based on one-tailed *t* tests.
[c] The estimates of c^2 and h^2 in these equations represent the shared environmental effects and heritability when ED = 0, that is, when parents received no formal education.
*** $p < .001$.

Table 5 shows that, as expected, both environmental (c^2) and genetic (h^2) interaction effects were statistically significant: $b_6 = -.12$, adjusted $SE = .04$, $t(3810) = -3.00$, $p < .001$; and $b_7 = .19$, adjusted $SE = .08$, $t(3810) = 2.38$, $p < .001$, respectively. The negative sign on the b_6 term indicates that shared environmental effects decrease with increasing levels of parent education. Likewise, the positive sign of the b_7 term indicates that as parental education rises, the heritability of verbal IQ also rises. Moreover, the fact that the coefficient for c^2 was significant, $b_1 = .50$, adjusted $SE = .15$, $t(3810) = 3.33$, $p < .001$, yet the coefficient for h^2 was not, $b_4 = -.12$, adjusted $SE = .30$, $t(3810) = -0.40$, $p > .50$, indicates that at the very extreme level of parental education (i.e., when parental education = 0, representing no formal education), sibling resemblance for verbal IQ would be due entirely to shared family environments. Finally, the adjusted R^2 for this model was .21, compared to the $R^2 = .17$ obtained from the model without interactions, indicating that the full model explained an additional 4% of the variance. Because we suspect that the group of cousins living in the same household could have been living there for only a short period, and because they were selected to be in the same household (typically with an aunt or uncle) by an unknown process, this DF regression was repeated with the cousin group omitted. In the new DF regression, the interaction terms were again statistically significant and the parameter estimates were similar to those shown in Table 5 (results not shown). Moreover, the pattern of regression coefficients also held when the DF equation was repeated for Whites and Blacks separately (results not shown). Thus, the findings reported in Table 5 appear to be robust.

The precise form of the interaction effect can be estimated from formulas that combine the main effect and interaction regression coefficients. For the estimation of the shared environmental effect, the formula is:

$$c^2 = .50 - .12(\text{ED}),$$

where ED is level of parental education and .50 and −.12 are the unstandardized regression coefficients for the estimate of c^2 when parental education is zero (b_1) and the interaction of c^2 with level of parental education (b_6), respectively (see Table 5). Similarly, the corresponding formula for heritability is:

$$h^2 = -.12 + .19(\text{ED}),$$

where −.12 is the unstandardized regression coefficient for the estimate of heritability when parental education is zero (b_4), and .19 is the unstandardized regression coefficient for the heritability by parental education interaction (b_7; see Table 5).

Figure 1 takes the above equations and plots the values of c^2 and h^2 across varying levels of parental education. As can be seen from this figure, when parents have had at least a high school education (on average; 60.6% of the sample), shared environmental effects were always below 20% of the total variance and genetic influences came to predominate. The converse held when the average parental education was less than a high school degree. In this range, the influence of shared environment was actually greater than that of genes.

At the extremes of parental education, variance estimates become less accurate because fewer families are found (less than 1.1% at the lower extreme and less than 10% at the upper extreme) and because the function form in Figure 1 was necessarily linear, which prevented the function from tapering off at the extremes. This explains why the estimate of c^2 for those adolescents whose parents were college graduates was less than zero, and why the estimate for h^2 was less than zero for adolescents whose parents received no formal schooling.

The moderating effect of the environment on genetic and shared environmental influences on verbal IQ also can be seen in the pattern of sibling correlations when adolescents are divided into two groups on the basis of parental education (see Table 6). To reduce sampling variation, the less related sibling groups were combined into a single group (unrelated siblings, cousins, and half siblings), as were the moderately related sibling groups (DZ twins and full siblings). A third group consisted of the MZ twins only. As shown in Table 6, the correlational patterns are consistent with the interactions found by the DF anal-

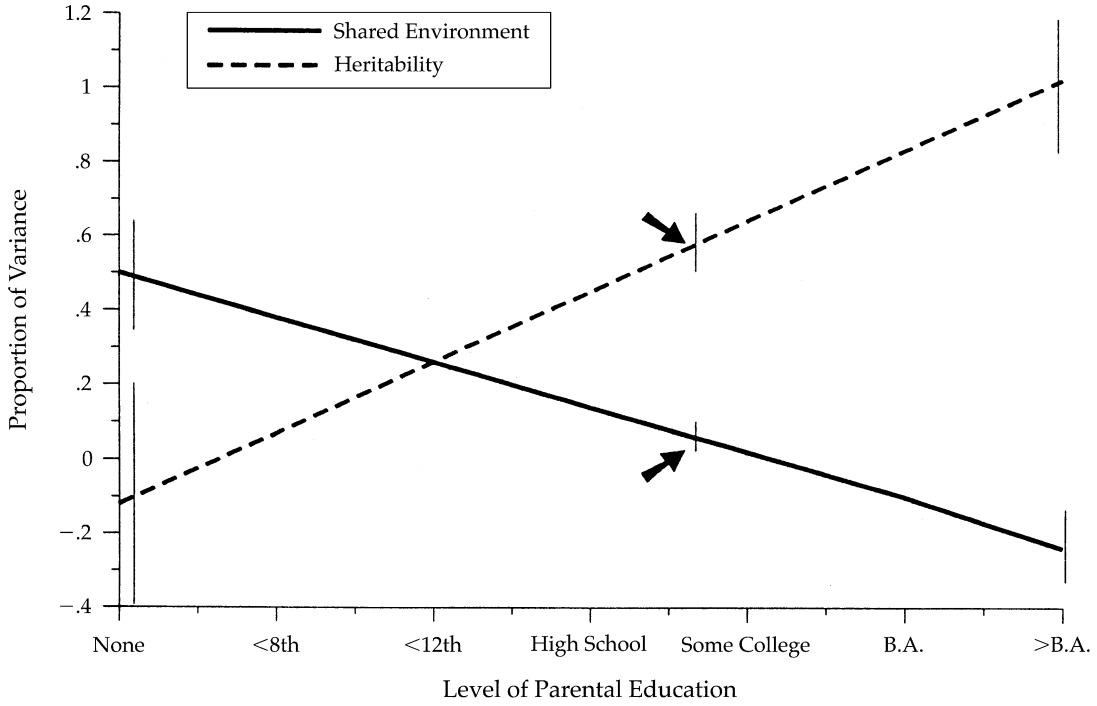

Figure 1 Relations between genetic and shared environmental variance components by level of parental education. Standard errors at the means and the extremes are indicated by vertical lines. Arrows point to the estimates of heritability and shared environmental influences at the sample mean.

yses. In the low relatedness group, the sibling correlation was greater in the offspring of low educated parents (.32) than those offspring of highly educated parents (.10), indicating greater shared environmental influences on sibling similarity in less educated families, $Z_{\text{difference}} = 2.79$, $p < .01$. In contrast, weaker genetic effects in less educated families were indicated by a smaller MZ twin correlation in the low education group: .75 in the high education group versus .55 in the low education group, $Z_{\text{difference}} = 2.09$, $p < .05$. Although the sibling correlations in Table 6 are given for illustrative purposes, they represent an arbitrary slice of the sample, whereas the DF analysis estimated c^2 and h^2 using education as a continuous variable.

Table 6 Sibling Correlations by Level of Parental Education

	Low Education		High Education	
	No. of Pairs	*r*	No. of Pairs	*r*
High relatedness	52	.55***	124	.75***
Moderate relatedness	424	.33***	718	.37***
Low relatedness	277	.32***	314	.10#

Note: High education reflects greater than a high school education; low education reflects a high school education or less. High relatedness = MZ twins; moderate relatedness = DZ twins and full siblings; low relatedness = half siblings, cousins, and unrelated siblings.
#$p < .10$; ***$p < .001$.

DISCUSSION

Similar to results from two earlier studies (i.e., Fischbein, 1980; Scarr-Salapatek, 1971), the present study adds support to the hypothesis that the heritability of IQ varies across social class levels. This study found that level of parental education moderated both the genetic and shared environmental influences on Peabody Picture Vocabulary IQ. Based on significant interaction terms in an extended DeFries-Fulker (DF) model, heritability increased from about 25% in the offspring of parents with less than 12 years of education to about 74% in the offspring of parents with greater than a high school education. In contrast, the shared environmental effect decreased from 20–40% of IQ variation in the offspring of parents without high school degrees to about zero for more-educated parents.

Our findings confirmed hypotheses stemming from bioecological theory of Bronfenbrenner and Ceci (1994) and the theory of Scarr (1992), namely, that trait

heritabilities will increase with improvement in environmental conditions. When the Add Health sample was split between parents with a high school diploma (or less) versus some education beyond high school, the heritability of verbal IQ was much greater for the children of more educated parents (h^2 = .74 versus .26). Thus, the genetic potential for learning vocabulary was expressed more fully when parents were better educated.

Although no exact threshold may exist for shared family environmental effects, it is clear that shared environmental effects became significant only among relatively uneducated parents. This finding corresponds to the hypothesis that environmental effects on IQ may be nonlinear. According to this theoretical model, when family environments are average or better, the "reaction surface" (Turkheimer & Gottesman, 1991) of the IQ phenotype to environmental variation is fairly flat. In the region of poor quality family environments, however, the IQ phenotype could be highly responsive to environmental variation.

One important implication from the present study is that results bear on the responsiveness of IQ to variation in normal family environments. In the group of parents with higher levels of education, family environmental influences were not contributing to individual variation in IQ. They may, however, have raised the mean IQ of the whole group. Shared environmental effects were most evident when the parents had less than a high school education. The children of these parents also had lower IQs. Thus, the provision of better family environments could raise IQs, and should decrease the IQ gap between socioeconomic groups.

The magnitude of IQ gain would depend on the number of children who could be influenced and the strength of any likely change. For example, at one extreme, in 8.5% of the sample *neither* parent had more than an 11th grade education. For this level of parental education, the estimate of shared environmental effect was approximately .40. The average IQ (unresidualized on race) was 90.8, with a standard deviation of 11.9. Hence, a 1 *SD* improvement of their family environments could increase their IQs from the 90.8 to approximately 98, that is, a gain of (.40 $\times$ 11.92),[5] which equals 7.5. A 2 *SD* change could yield a gain of approximately 15 IQ points. These values lie within the range observed in a cross-fostering study of IQ done in France, where children born into poor families were adopted by affluent families (Capron & Duyme, 1989; McGue, 1989). The children's IQ gain was 12 points (with a large standard error, because of the small number of children sampled). At higher levels of parental education, however, IQ should be less responsive to variation in family environments because shared environmental effect was weaker. Overall, the reaction range of IQ had the property envisioned by Scarr (1992, 1993)—increasing environmental effects in more deprived environments.

Although encouraging, these results bear further replication. For example, Capron and Duyme (1989) lacked the sample size to investigate any moderating effects of parental social class on the magnitude of genetic and shared environmental influences. In another study, moderating effects of the environment on children's academic abilities were not found (Van den Oord & Rowe, 1998). In this study of children of mothers in the National Longitudinal Survey of Youth (NLSY), family environmental quality failed to moderate estimates of either the shared environmental effect or the genetic effect. Our failure to replicate these earlier results was unexpected, given that the NLSY, just as the Add Health study, was a large sample of children from a broad range of socioeconomic statuses.

One explanation may lie in the age difference among the two samples: The NLSY children averaged about 9 years of age, whereas the average age in the Add Health sample was approximately 16, with 80% of adolescents between 13.5 and 18 years old. If harmful environmental effects accumulate over time, then perhaps the moderating effect of environment may be more detectable in a sample of older children. Although the age ranges in the two samples do not overlap, thus no direct comparison can be made, we did examine whether moderating effects could be found in a subsample of adolescents where both siblings were younger than 14 years old (results not shown). Although the sample size was quite small (n = 336 pairs), the moderating effects of parent education on heritability and the shared environmental influence were in the same direction as results from the whole sample, and were statistically significant. Thus, moderating effects of parental education were found even among the youngest adolescents in our sample.

A more likely explanation lies in an important design difference between the studies. Specifically, the NLSY sample had only three levels of genetic kinship: full siblings, half siblings, and cousins, whereas this study also included twins and biologically unrelated siblings. To evaluate the effect of restricting our data analyses to the former groups, the DeFries-Fulker regression was repeated omitting the twins and unrelated siblings (a loss of almost 40% of the sample). Although the pattern of results remained the same (i.e., a negative coefficient for the $c^2 \times$ ED interaction and a positive coefficient for the $h^2 \times$ ED interaction), the

deletion of the twins and unrelated siblings in our sample caused the interaction terms to become nonsignificant. Thus, it is possible that large, multiple kinship samples are necessary to have sufficient statistical power to detect moderating effects of the family environment.

Moreover, it is possible that our results reflect something other than a moderating effect of parental education on the genetic and environmental contributions to sibling similarity in IQ. For example, heritability may increase or decrease as phenotypic scores move toward an extreme. In the present study, adolescents from the least educated families also have the lowest IQs, and adolescents from the most educated homes have the highest IQs, $F(2, 3815) = 97.5$, $p < .001$. The effect of extreme scores on heritability has been investigated using squared-phenotype terms in DF regression analyses, or by regressing within-sibling pair phenotypic differences on phenotypic sums (Bailey & Revelle, 1991; Detterman, Thompson, & Plomin, 1990). Some twin studies have found no evidence of differential heritability (on infants 1–3 years, Cherny, Cardon, Fulker, & DeFries, 1992; on older adolescents, Sundet, Eilertsen, Tambs, & Magnus, 1994). Two twin studies were supportive of our findings, however. One found greater IQ heritability among high IQ twin children, who were most likely the children of better educated parents (Bailey & Revelle, 1991). The other study, of sixth grade twins in Cleveland, Ohio, found interaction effects most comparable to ours: an increase of shared environmental influences when children had lower IQs *and* a trend toward greater heritability when they had higher IQs (Thompson, Detterman, & Plomin, 1993). It should be noted, however, that in an earlier report using fewer twin pairs from the same Western Reserve Twin Sample, Detterman et al. (1990) found the reverse: the heritability of IQ was greater among *lower* IQ children. Findings for such interaction effects might become more consistent if studies systematically over-sampled individuals at IQ extremes.

The present study found that for the offspring of parents with more than a high school diploma, verbal IQ was highly heritable. It is not the case, however, that all working-class to affluent families provide the same degree of intellectual stimulation. To explain a high heritability of IQ despite unequal environments across families, a process of G→E correlation can be invoked. Brighter children may extract information from their environments more rapidly, handle complex information better, and expose themselves to information at a greater rate than do less able children (Carroll, 1997; Gottfredson 1997). The same objective environment is, in actuality, different environments for children with disparate intellectual abilities. Furthermore, intellectual simulation within families can be compensated for, or complimented by, stimulation in schools and in peer groups. With their active G→E effects, bright children can accelerate intellectual development, regardless of their immediate family circumstances. In contrast, children with a potential for higher IQ in lower SES environments may not be able to find the intellectual stimulation they need, given the lower quality of schooling often found in less well-educated communities. Thus, the apparent moderating effect found in the present study may represent a G→E correlation.

Furthermore, level of parental education, in and of itself, is also likely to be influenced by genetic factors. Thus, more highly educated parents are more likely to pass on genes related to higher IQ as well as provide more intellectually stimulating environments. There is evidence, at least among infants and children, that part of the correlation between the home environment and offspring intelligence is mediated genetically (Braungart, Fulker, & Plomin, 1992; Coon, Fulker, DeFries, & Plomin, 1990). Therefore, continued investigations of the genetic and environmental influences on the relationship between family environment and IQ are warranted.

Finally, this study had several limitations. One was the use of just a single measure of IQ, verbal IQ, from the Peabody Picture Vocabulary test. Because Add Health did not include a measure of nonverbal IQ, or nonvocabulary, verbal IQ, we cannot demonstrate conclusively that the genetic and environmental etiology of general intelligence (g), rather than just vocabulary, is moderated by the environment. Vocabulary IQ, however, loads strongly on g in factor-analytic studies of IQ, which makes the moderating effects more plausible. An additional limitation is that our findings were restricted to Non-Hispanic Whites and Blacks, because the other ethnic and racial groups within Add Health were too few in number (or too heterogeneous) to permit separate analyses. Although in many cases racial and ethnic groups show similar developmental processes (Rowe, Vazsonyi, & Flannery, 1994), the results presented here may not generalize to other racial or ethnic groups. The DF regression also assumed that the equal environments assumption was not violated in any sibling group. This assumption is that the environmental influences that affect a phenotype correlate equally between siblings for each type of sibling pair. In other words, it is assumed that MZ twins do not experience more similar environments than DZ twins, full siblings, or any other sibling group and/or that more similar treatment does not systematically predict sibling similar-

ity. Data on this assumption for twins is presented by Rowe (1994; also, Hettema, Neale, & Kendler, 1995), but this assumption was not tested explicitly in the present study. Finally, our model incorporated neither assortative mating, which could increase the correlation of all siblings except MZ twins, nor genetic dominance, which could increase the correlation of DZ twins and biological full siblings, but not of the other sibling types. Although our simpler model is a limitation, it probably captures the major sources of variance, which were additive genetic influences and shared environmental influences, satisfactorily.

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REFERENCES

Anastasi, A. (1958). Heredity, environment, and the question "How?" *Psychological Review, 65*, 197–208.

Bailey, M. J., & Revelle, W. (1991). Increased heritability for lower IQ levels. *Behavior Genetics, 21*, 397–404.

Braungart, J., Fulker, D. F., & Plomin, R. (1992). Genetic mediation of the home environment during infancy: A sibling adoption study of the HOME. *Developmental Psychology, 28*, 1048–1055.

Bronfenbrenner, U., & Ceci, S. J. (1994). Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review, 101*, 568–586.

Capron, C., & Duyme, M. (1989). Assessment of the effects of socio-economic status on IQ in a full cross-fostering study. *Nature, 340*, 552–554.

Carroll, J. B. (1997). Psychometrics, intelligence, and public perception. *Intelligence, 24*, 25–52.

Cherny, S. S., Cardon, L. R., Fulker, D. W., & DeFries, J. C. (1992). Differential heritability across levels of cognitive ability. *Behavior Genetics, 22*, 153–162.

Cherny, S. S., DeFries, J. C., & Fulker, D. W. (1992). Multiple regression of twin data: A model fitting approach. *Behavior Genetics, 22*, 489–497.

Coon, H., Fulker, D. W., DeFries, J. C., & Plomin, R. (1990). Home environment and cognitive ability of 7-year-old children in the Colorado Adoption Project: Genetic and environmental etiologies. *Developmental Psychology, 26*, 459–468.

Cooper, R. M., & Zubek, J. P. (1958). Effects of enriched and restricted early environments on the learning ability of bright and dull rats. *Canadian Journal of Psychology, 12*, 159–164.

DeFries, J. C., & Fulker, D. W. (1985). Multiple regression analysis of twin data. *Behavior Genetics, 15*, 467–473.

Detterman, D. K., Thompson, L. A., & Plomin, R. (1990). Differences in heritability across groups differing in ability. *Behavior Genetics, 20*, 369–384.

Dunne, M. P., Martin, N. G., Statham, D. J., Slutske, W. S., Dinwiddie, S. H., Bucholz, K. K., Madden, P. A. F., & Heath, A. C. (1997). Genetic and environmental contributions to variance in age at first sexual intercourse. *Psychological Science, 8*, 211–216.

Fischbein, S. (1980). IQ and social class. *Intelligence, 4*, 51–63.

Gottfredson, L. S. (1997). Why *g* matters: The complexity of everyday life. *Intelligence, 24*, 79–132.

Heath, A. C., Berg, K., Eaves, L. J., Solaas, M. H., Corey, L. A., Sundet, J., Magnus, P., & Nance, W. E. (1985). Educational policy and the heritability of educational attainment. *Nature, 314*, 734–736.

Hettema, J. M., Neale, M. C., & Kendler, K. S. (1995). Physical similarity and the equal environments assumption in twin studies of psychiatric disorders. *Behavior Genetics, 25*, 327–335.

LaBuda, M. C., & DeFries, J. C. (1990). Genetic etiology of reading disability: Evidence from a twin study. In G. T. Pavlidis (Ed.), *Perspectives on dyslexia* (Vol. 1, pp. 47–76). New York: Wiley.

McGue, M. (1989). Nature-nurture and intelligence. *Nature, 340*, 507–508.

Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (1997). *Behavioral genetics* (3rd Ed.). New York: W. H. Freeman.

Resnick, M. D., Bearman, P. S., Blum, R. W., Bauman, K. E., Harris, K. M., Jones, J., Tabor, J., Beuhring, T., Sieving, R. E., Shew, M., Ireland, M., Bearinger, L. H., & Udry, J. R. (1997). Protecting adolescents from harm: Findings from the National Longitudinal Study of Adolescent Health. *Journal of the American Medical Association, 278*, 823–832.

Rodgers, J. L., & McGue, M. (1994). A simple algebraic demonstration of the validity of DeFries-Fulker analysis in unselected samples with multiple kinship levels. *Behavior Genetics, 24*, 259–262.

Rowe, D. C. (1994). *The limits of family influence: Genes, experience, and behavior.* New York: Guilford Press.

Rowe, D. C., Vazsonyi, A. T., & Flannery, D. J. (1994). No more than skin deep: Ethnic and racial similarity in developmental process. *Psychological Review, 101*, 396–413.

Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development, 63*, 1–19.

Scarr, S. (1993). Reply: Biological and cultural diversity: The legacy of Darwin for development. *Child Development, 64*, 1333–1353.

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype→environment effects. *Child Development, 54*, 424–435.

Scarr, S., & Weinberg, R. A. (1978). The influence of "family background" on intellectual attainment. *American Sociological Review, 43*, 674–692.

Scarr-Salapatek, S. (1971). Race, social class, and IQ. *Science, 174*, 1285–1295.

Spitz, E., Moutier, R., Reed, T., Busnel, M. C., Marchaland, C., Roubertoux, P. L., & Carlier, M. (1996). Comparative diagnoses of twin zygosity by SSLP variant analysis, questionnaire, and dermatoglyphic analysis. *Behavior Genetics, 26*, 55–63.

Sundet, J. M., Eilertsen, D. E., Tambs, K., & Magnus, P. (1994). No differential heritability of intelligence test scores across ability levels in Norway. *Behavior Genetics, 24*, 337–339.

Sundet, J. M., Tambs, K., Magnus, P., & Berg, K. (1988). On the question of secular trends in the heritability of intelligence test scores: A study of Norwegian twins. *Intelligence, 12*, 47–59.

Thompson, L. E., Detterman, D. K., & Plomin, R. (1993). Differences in heritability across groups differing in ability, revisited. *Behavior Genetics, 23*, 331–336.

Turkheimer, E., & Gottesman, I. I. (1991). Individual differences and the canalization of human behavior. *Developmental Psychology, 27*, 18–22.

Van den Oord, E. J. C. G, & Rowe, D. C. (1998). An examination of genotype-environment interaction for academic achievement in an U.S. National Longitudinal Survey. *Intelligence, 25*, 205–228.