RACIAL DIFFERENCES IN BIRTH HEALTH RISK: A QUANTITATIVE GENETIC APPROACH*

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In the United States the gap between black and white babies' birth weights has remained largely unexplained. Rather than trying to measure all relevant variables, we used a genetically informative design to study the relative importance of genetic and environmental factors. Employing multiple indicators of "birth health risk," we found that the racial differences increased with the magnitude of the shared environmental effects. This suggested that possible genetic effects would not pertain to fetal genes, although genes affecting the mother's physical or physiological characteristics could be important because they contribute to shared environment in our analysis.

In the United States the average birth weight and the prevalence of low birth weight (< 2,500 g) differ sharply among black and among white babies. Black newborns have about twice as great a risk of low birth weight as white newborns (12.7% vs. 5.9%; McCormick 1985). This gap persists even among the offspring of college-educated women (7% vs. 3%; Schoendorf et al. 1992). Because of the negative associations with infant health, infant mortality, IQ, and psychosocial adjustment (Brooks-Gunn et al. 1993; Klein, Hack, and Breslau 1989; Lagerstrom et al. 1994; McCormick 1985; Rose et al. 1992; Sappenfield et al. 1987; Tomchek and Lane 1993), tremendous effort has been devoted to discovering the determinants of these differences in birth weight.

Economic disparities between blacks and whites may explain the disparity. Cramer (1995), using data from the National Longitudinal Survey of Youth (NLSY), indeed found that birth weight was related to a group of "distant" variables such as income, years of education, and receipt of public aid. Cramer also examined a set of "immediate" variables including smoking cigarettes, drug use, perinatal care, and the mother's weight for height. His "medical model," combining distant and immediate variables, could account for about 10% of the variation in birth weight. Moreover, when a regression equation computed on whites was compared with one computed separately on blacks, the equations were equal. Therefore blacks and whites could be analyzed as a single group to determine whether the model would explain the racial difference. The difference in birth weight was reduced from 226 grams to 178 grams—still a substantial difference despite the predictive power of the model. Although blacks' greater poverty tended to reduce the black-white gap for birth weight, blacks had more favorable statuses on some of the immediate variables (e.g., they smoked less), which acted in the opposite direction.

Because Cramer's model failed to eliminate the gap, attention must be directed toward other possible explanations. It is easy to generate a long list of omitted variables such as sexually transmitted diseases, social support from family members, support from the child's biological father, nutrition, physical illnesses, and exposure to discrimination. The difficulty of measuring all these variables, however, hampers the resolution of the difference in mean birth weight between blacks and whites.

The Quantitative Genetic Approach

The explanatory factors in this study are latent variables representing three theoretical components of variation: heredity, shared environment, and nonshared environment. This quantitative genetic approach enables one to explain the difference between racial groups in terms of these components, without actually specifying and measuring all the variables. To perform the analyses, we used full and half-siblings from the same survey Cramer (1995) used, and analyzed a composite outcome variable labeled "birth health risk."

The shared environment component corresponds to those environmental influences that operate to make siblings alike. In this application, the shared environment effect is estimated from the resemblance of siblings' birth health risks. For example, if extreme poverty was characteristic of some mothers but not others, then it should induce some similarity of birth health risk across siblings. Maternal smoking and other substance use, so long as the mother continues her use from one pregnancy to another, also may act as a shared maternal environmental effect between pregnancies. Cramer (1995: 233–34) speculated that some mothers may be more conscientious than others during a pregnancy, and observed that "prenatal mothercraft might incorporate lifestyle and behavioral factors associated with nutrition, smoking, use of drug and alcohol, or proper medical care." Thus, insofar as some mothers excel more at "mothercraft" than others, this would be read into a behavior genetic model as a shared environmental effect.

The term *nonshared environment* refers to environmental effects that are unique to a particular child and uncorrelated across pairs of biological relatives. Nonshared environment always operates to make biological siblings dissimilar in a trait. Diseases that harm one pregnancy but not an-

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other will operate as nonshared influences. Because prevalence rates of such diseases could vary between groups, nonshared environmental effects also might contribute to racial differences in birth health risk. Measurement errors also have this consequence. Unless the reliability of a trait is considered, as we consider traits by using the "birth health risk" composite measure, measurement errors will be confounded in this parameter estimate.

In our research design comparing biological half-siblings with biological full siblings, genetic effects that operate in the fetus produce correlations that take the same rank order as the biological relatedness of the pairs: that is, from half-siblings to full siblings. If the correlations on birth health risk equal these genetic expectations, then "birth health risk" will have a heritability (the proportion of total variance due to genetic variation) of 1.0, or 100%. Insofar as the correlations are lower than these values, but follow the same rank order, the influences operating to produce variation in birth health risk will combine genetic and "nonshared environmental" effects.

In addition to fetal genes, maternal genes that influence the uterine environment may be important. The half-siblings in our study had the same biological mother but different fathers. For this reason, maternal genetic effects will not affect the resemblance between full and half-siblings differently, but simply will make siblings alike in birth health risk. As a result, the effect of maternal genes is not part of the genetic component but shows up as shared environment in our analysis.

Although previous studies have not examined racial mean differences in birth health risk, they have investigated the components of variation discussed above. These studies give reason to expect that genes could be important in addition to environmental effects (Magnus 1984; Vlietinck et al. 1989). Also, notably, a number of studies found substantial effects of a birth-weight-specific component labeled "maternal effects" (Boomsma, Orlebeke, and Van Baal 1992; Carr-Hill et al. 1987; Morton 1955; Nance et al. 1983). For instance, Morton (1955) reported that the birth weight correlation across the half-sibling pairs was .58 for maternal pairs but only .10 for paternal pairs. This showed that siblings who share the same uterine environment (e.g., maternal half-siblings) are more alike than those who do not (e.g., paternal half-siblings).

The Racial Variable

In this study, the racial variable is based on the mother's self-reported identification. Like most social science variables, racial identification contains measurement errors that would tend to attenuate its correlation with developmental outcomes such as "birth health risk." Racial categories in the United States also imply some degree of racial admixture between Caucasian- and African-origin genes. In a study of protein-coding genetic loci in American whites and blacks, it was estimated that 25% of the genes in blacks originated from racial admixture between these groups (Chakraborty et al. 1992). In this paper we use the generic terms *white* and *black* to describe these two broad racial categories within the United States.

Racial groups should be regarded as both genetic and social categories (Rowe, Vazsonyi, and Flannery 1994). They are genetic entities because generations of "reproductive isolation" have led to differences in gene frequency across racial groups. During prehistoric times, the physical barrier of the Sahara Desert reduced gene flow between Europeans and sub-Saharan Africans. For many traits, including those physical traits that visibly differentiate the groups (e.g., skin color and physical features), gene frequencies are sharply different. The same is true of some neutral genetic markers (e.g., Spurdle and Jenkins 1991).

Nonetheless, the degree of racial differentiation in gene frequencies is highly trait-dependent. For example, a gene that produces sickle cell anemia, a disorder of the red blood cells, is relatively common in blacks and extremely rare in whites (allele frequency = .10–.20 vs. < .001). Conversely, a gene causing cystic fibrosis, a disorder of ionic transport in the lung and other tissues, although rare in all populations, is substantially more frequent among whites than among blacks (.013–.022 vs. .0036; Weaver and Hedrick 1989:520). Although many gene frequencies are nearly identical in both racial groups, group differences may arise in part from genetic differences between groups whenever genes influence trait variation (i.e., whenever a trait is heritable).

Race is also a social category. In the United States, large black-white differences exist in rates of teenage pregnancy and in the degree of poverty. Thus the mean differences between blacks and whites in "birth health risk" may arise not because of differences in gene frequency, but because of unequal exposure to environments harmful to prenatal development (e.g., poor nutrition, sexually transmitted diseases, lack of prenatal care).

In summary, we attribute racial differences in birth health risk to three components of variation: heredity, shared environment, and nonshared environment. Because race can be regarded as both a genetic and a social category, in principle each of these components could explain the race gap.

METHODS

Participants and Measures

The NLSY began in 1979 as a large sample of males and females age 14–21 in the United States (Center for Human Resource Research 1993). In 1992, 9,360 children had been born to 4,415 NLSY mothers. Because of the oversampling of minority groups, almost 29% of these children were black. White children constituted 53% of the total sample. The exact procedure used to determine race involved a combination of the mother's self-reported race and her country of origin. (For an exact account describing how the racial variable R2147 was constructed, see *NLS Users' Guide* 1994:357-65.) Elaborate demographic information contrasting the black and the white samples in the NLSY was reported by Cramer (1995).

Since 1983 the female respondents have been asked for information about their children's postnatal health history.

We use three variables: birth weight (measured in ounces), length of gestation (measured in weeks), and birth length (measured in inches). For a number of babies, birth length was not measured but was estimated by the mother. We excluded these estimated birth lengths from the analyses and regarded them as missing values. For the three variables, the respective percentages of missing values were 5.8%, 9.2%, and 50.9% for black babies, and 5.2%, 7.5%, and 29.7% for white babies. These figures indicated small proportions of missing values for birth weight and length of gestation, and many missing values for birth length. Calculations showed that with the possible exception of birth length, our results were not biased because the percentage of missing data was small and because most values seemed to be missing at random. (Details are available from the first author.)

Pairs of Relatives

Because most NLSY mothers have more than one child, the survey comprises a sizable number of siblings. We used a classification algorithm developed by Rodgers to divide these children into groups of full and half-siblings (Rodgers, Rowe, and Li 1994; Rodgers, Rowe, and May 1994). On the assumption that mothers cannot live simultaneously with different fathers, we classified children who lived with both biological parents as full siblings. If the father of one member of a sibling pair lived at home and the other sibling's father did not, they were classified as half-siblings. If neither father lived in the home, we used a second variable with four or five categories, which indicated how far away each child's father lived (within one mile, 1–10 miles..., more than 200 miles). If the two fathers' distances did not match, the children were classified as half-siblings; otherwise their status was considered indeterminate.

Six separate survey years provided information that allowed us to distinguish full and half-siblings. We first identified full and half-siblings for each year separately. Children who were classified as full siblings on at least two occasions and were never classified as half-siblings were considered to be full siblings. Children who were classified as half-siblings on at least two occasions and were never classified as full siblings were considered to be half-siblings.

To validate the classification algorithm for constructing groups of full and half-siblings, Rodgers and colleagues showed that it yields heritability estimates that are quite close to those reported in behavior genetic literature for frequently studied characteristics such as weight, height, and IQ. To check for possible errors, we also compared the sibling status obtained through our program with the sibling status obtained through the program followed by Rodgers (personal communication). Although our program was more conservative and more often classified sibling status as indeterminate, the number of misclassifications was nearly zero.

There is evidence that maternal half-siblings, who share the same uterine environment, are more alike than paternal half-siblings, who do not (Morton 1955). In principle, these uterine environmental differences should be taken into account in a genetic analysis that is based on the resemblance between relatives. Yet because the present sample consisted entirely of maternal half-siblings (the NLSY includes only the children of female respondents), this was not a problem in our analyses.

The generalizability of our results is not limited by the fact that our sample consisted entirely of maternal half-siblings. Thus effects of the uterine environment do not affect the resemblance between paternal half-siblings. As in the case of maternal half-siblings, however, the uterine environment remains a source of variation. Finally, because twins have identical gestational ages but often discrepant birth weights (Wilson 1984), we excluded them from our analyses.

Decomposition of Within-Group Variation

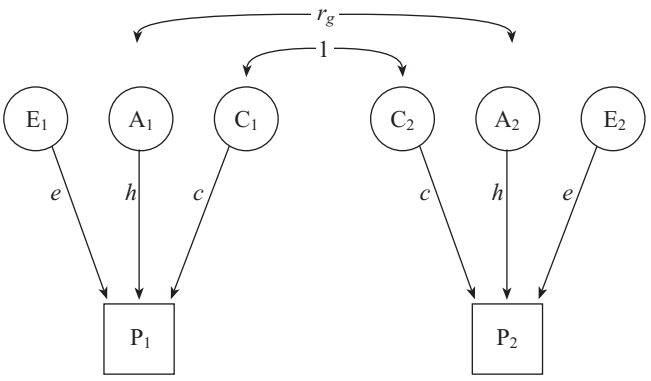
A first step in examining differences in birth health risk *between* racial groups is to decompose the variation *within* each group into genetic, shared environmental, and nonshared environmental contributions. Such a decomposition can be made using standard quantitative genetic techniques (Falconer 1989; Neale and Cardon 1992; Plomin, DeFries, and McClearn 1990). Because these techniques focus on differences, we examined causes of within- and between-group *variation;* we did not study the factors that determine the absolute levels of birth weight, length of gestation, and birth length.

A decomposition of the variation within groups can be made on the basis of the model depicted in Figure 1. The genetic component involves many different loci on the chromosomes; the additive genetic scores (A) are simply the sum of the effects across all those loci. On the assumption that mating in the population is random, the laws of segregation imply a correlation between the additive genetic scores equal to .5 for full siblings and .25 for half-siblings.

We distinguish two types of environmental influences. Shared environmental influences (C) are those that are identical for siblings; therefore they are correlated perfectly. Nonshared environmental influences (E) are unique for each sibling; thus they are uncorrelated. The model assumes that the genetic and environmental factors do not correlate or interact. If we determine the arbitrary scale of the latent variables by giving them variances of 1, the total phenotypic variance is the sum of the genetic, shared environmental, and nonshared environmental variance: $VAR(P) = h^2 + c^2 + e^2$. The proportion of variance explained by a component can be computed by dividing its explained variance by the total variance; this proportion is an indicator for the importance of that component for within-group variation. For instance, the proportion of genetic variance or heritability equals $h^2/(h^2 + c^2 + e^2)$; a heritability of .5 would imply that 50% of the differences between subjects within groups can be explained in terms of genetic influences.

Rough estimates of the proportions of genetic and environmental variance can be derived from the sibling correlations. Using the above-mentioned assumptions, we can express these correlations as $r_{\text{sibling}} = r_g h^2 + c^2$. This formula shows that the correlation between siblings is a function of the heritability and the degree of genetic relatedness. The

FIGURE 1. A STANDARD BEHAVIOR GENETIC MODEL FOR DECOMPOSING WITHIN-GROUP PHENOTYPIC VARIATION INTO GENETIC AND ENVIRONMENTAL INFLUENCES



Notes: The "observed" phenotypic scores (P) of siblings are modeled as a weighted sum of "unobserved" or "latent" additive genetic (A), shared environmental (C), and nonshared environmental scores (E). Subscripts 1 and 2 refer to the first and the second sibling respectively. Parameters *h*, *c*, and *e* are the weights or the effects of the latent factors on the phenotype.

implication is that for heritable phenotypes, the correlation will be larger for full (r_g = .5) than for half-siblings (r_g = .25). Furthermore, all shared environmental variance affects the sibling correlation because these influences, by definition, are equal for siblings. Nonshared environmental effects are unique for each child and do not contribute to the resemblance between siblings.

To obtain an indication of the shared environmental variance, one can subtract the full-sibling correlation from two times the half-sibling correlation: $c^2 = 2 \times (.25h^2 + c^2) - (.5h^2 + c^2)$. The genetic variance can be estimated by multiplying by 4 the difference between the full-sibling and the half-sibling correlation: $h^2 = 4 \times ((.5h^2 + c^2) - (.25h^2 + c^2))$. Finally, an impression of the nonshared environmental variance can be obtained by subtraction. For instance, if the phenotypic variance equals 1, then $e^2 = 1 - h^2 - c^2$.

The above discussion shows how the phenotypic variance can be decomposed into genetic and environmental contributions by using sibling correlations that involve the same variable. Analogously, the phenotypic covariances can be decomposed into genetic and environmental contributions by using sibling correlations that involve different variables. For instance, the covariance between birth weight in sibling A and birth length in sibling B can be used to decompose the phenotypic correlation between birth weight and birth length. Formulas similar to those discussed above can be used; these will yield estimates of the contribution of genetic and environmental factors to the covariances.

In our study we use a 3 × 3 phenotypic covariance matrix between birth weight, length of gestation, and birth length. Each element in this matrix can be decomposed by using the corresponding sibling correlations. Although the simple formulas discussed above are useful for descriptive purposes, it is preferable to make a simultaneous decomposition of the whole covariance matrix using structural equation modeling. This approach employs the same principles as described above, but has these advantages: The genetic and environmental contributions can be estimated in a statistically efficient way, and the approach yields fit indices that can be used to test hypotheses concerning within- and between-group differences. This method can be compared to a confirmatory factor analysis because a model must be specified in advance, comprising both common and unique factors.

The model we used is a multivariate extension of Figure 1, and is displayed in Figure 2. It consists of one common genetic factor, one common shared environmental factor, and one common nonshared environmental factor. Because these common factors affect all three observed variables, they explain the covariance or the shared variance among birth weight, length of gestation, and birth length. This shared variance is associated with postnatal health; therefore we may refer to the common factors as birth health risk factors. The unique genetic and environmental factors represent the variance that is unique for each observed variable, and resemble the error variances in a confirmatory factor analysis.

This model does not assume that all factors that are correlated with the indicator variables are risk factors for birth health. For instance, birth length probably is affected by the mother's height. If the mother's height does not affect birth weight and length of gestation, however, these effects would be incorporated into the unique part of the model. On the other hand, if the mother's height affects birth weight and length of gestation as well, it would be a common factor; in such a case, it would seem reasonable to view this as a "risk" factor for birth health.

Decomposition of Between-Group Variation

To study genetic and environmental contributions to between-group variation, we also must include the means of birth weight, length of gestation, and birth length in the model. Several authors have shown how this can be done in structural equation models (e.g., Bollen 1989; Jöreskog and Sörbom 1989; Sörbom 1974). Methodological extensions of this approach to genetically informative designs have been described by Dolan, Molenaar, and Boomsma (1989, 1991, 1992, 1994); actual applications of genetic models involving both covariances and means can be found in Dolan (1992) and Rowe and Cleveland (1997).

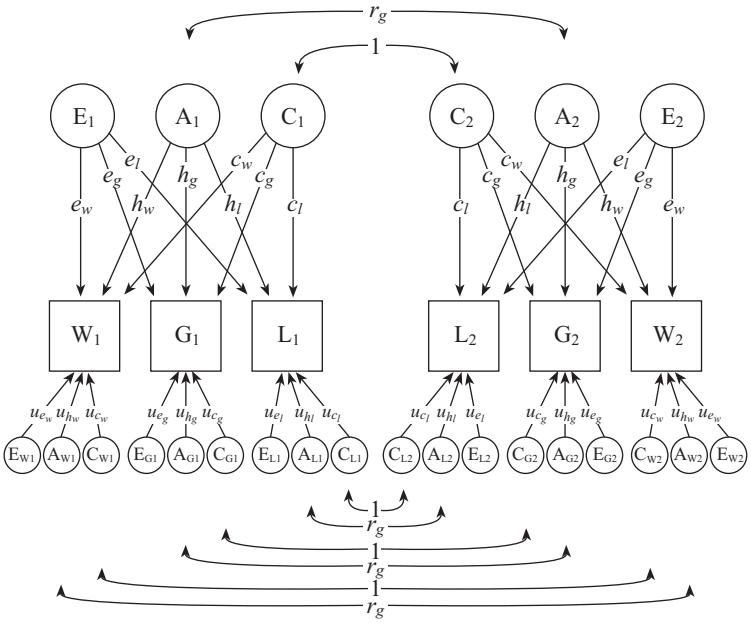
By taking the expected value E, we obtained the means of the three variables for the model presented in Figure 2. (For simplicity we omitted the subscripts for first and second sibling.) Because estimates of *h*, *c*, and *e* may vary across racial groups, we assigned them an additional subscript: *b* for black babies and *w* for white.

$$E(W) = o_w + h_{ww}\,E(A) + c_{ww}\,E(C) + e_{ww}\,E(E)$$

$$E(G) = o_g + h_{gw}\,E(A) + c_{gw}\,E(C) + e_{gw}\,E(E)$$

$$E(L) = o_l + h_{lw}\,E(A) + c_{lw}\,E(C) + e_{lw}\,E(E)$$

FIGURE 2. A MULTIVARIATE GENETIC MODEL FOR BIRTH WEIGHT, LENGTH OF GESTATION, AND BIRTH LENGTH



Notes: Capital letters W, G, and L represent birth weight, length of gestation, and birth length respectively. A is the common additive genetic factor, E the common nonshared environmental factor, C the common shared environmental factor, and r_g the genetic correlation that is .5 for full siblings and .25 for half-siblings. The factors A, C, and E subscripted with W, G, and L are the unique genetic or environmental factors. The factors are subscripted 1 and 2 to distinguish the factors for the first and the second sibling. Parameters *h*, *e*, and *c* are the effects of the common factors; parameter *u* represents the effect of the unique factors. They are subscripted *w* for birth weight, *g* for length of gestation, and *l* for birth length.

$$\begin{aligned} E(W) &= o_w - h_{wb}\,E(A) - c_{wb}\,E(C) - e_{wb}\,E(E) \\ E(G) &= o_g - h_{gb}\,E(A) - c_{gb}\,E(C) - e_{gb}\,E(E) \\ E(L) &= o_l - h_{lb}\,E(A) - c_{lb}\,E(C) - e_{lb}\,E(E) \end{aligned} \quad (1)$$

The formula indicates that the group means involve a variable-specific overall mean *o* subscripted *w* for birth weight, *g* for length of gestation, and *l* for birth length. These overall means are identical in the two racial groups. In addition, there are deviations from the overall means that involve the genetic E(A), shared environmental E(C), and nonshared environmental E(E) factors. These deviations are added to the overall mean in the groups of white babies, and are subtracted from the overall mean in the group of black babies. The unique genetic and environmental "error" effects are assumed to cancel each other out; thus their means are zero and they do not contribute to between-group variations.

Eq. (1) shows that observed group differences can be broken down into genetic, shared environmental, and nonshared environmental contributions. The contributions are the product of path coefficients *h*, *c*, and *e* and the corresponding genetic or environmental deviation from the overall mean E(A), E(C), and E(E). In the previous section we indicated how the path coefficients *h*, *c*, and *e* can be estimated by using the full- and half-sibling correlations. Here we use an example to show how the deviations E(A), E(C), and E(E) can be estimated.

Assume that the genetic path coefficients are identical in both racial groups and that they equal .8 for birth weight and .5 for length of gestation, and that the genetic deviation from the overall mean E(A) is .5. Because the deviations from the overall mean involve the product of the genetic path coefficient and the genetic deviation, they will be .8 × .5 = .4 for birth weight, .5 × .5 = .25 for length of gestation, and .2 × .5 = .1 for birth length. This example demonstrates that the observed group differences covary with the size of the genetic path coefficients because the path coefficients differ for the three variables. The genetic deviations, however, are identical because each variable is affected by the same common genetic factor. More generally, genetic and environmental deviations of zero will be estimated if the corresponding path coefficients are unrelated to observed group differences; nonzero deviations will be estimated for the components where the path coefficients covary with the observed group differences in means.

In estimating the deviations from the overall means, a necessary condition is that the number of common genetic and environmental factors should be at least equal to the number of observed means. For instance, assume that there is one observed variable with an observed group deviation of .5, a genetic path coefficient of .8, and a nonshared environmental path coefficient of .5. In this situation it is impossible to estimate the genetic and nonshared environmental deviations because we have only one equation for two unknowns:

.5 = .8 × E(A) + .5 × E(E). The deviations E(A) and E(E) therefore are not identified, and an infinite number of solutions is possible (e.g., E(A) = .6 and E(E) = .04, or E(A) = 0 and E(E) = 1). Next, suppose that there is a second variable with this equation: .3 = .4 × E(A) + .5 × E(E). With two equations for the same two unknowns, E(A) and E(E) would be identified exactly, and there would be a unique solution: E(A) = .5 and E(E) = .2.

In our study there were three observed variables: birth weight, length of gestation, and birth length. Except for the signs, in the case of two groups the deviations are the same in both groups (because with two means, the absolute deviation from each mean to the overall mean is identical). Therefore there were also three deviations E(A), E(C), and E(E), so that our model was identified exactly.

Equal numbers of common factors and observed means are a necessary but not a sufficient condition for identifying the genetic and environmental deviations. Another requirement is that the proportions of genetic and environmental variance explained by the common factors must be different for the three variables. The reason is that if these common effects are identical except for differences in scale, they cannot covary with the observed group differences in means. Instead, the common factor could affect the three variables via an intermediate latent psychometric factor (Neale and Cardon 1992:231–59).

In this latter model, known as the "common pathway" or "psychometric" model, differences between genetic and environmental sources cannot be distinguished, and they can be ascribed only to the latent psychometric factor. The common pathway model can be viewed as a submodel of the independent pathway model, in which the relative importance of genetic and environmental effects is identical for all observed variables. Thus, by comparing the fit of both models, one can perform a test to study whether the factor deviations are identified and can ascribe racial differences in means to genetic or environmental sources.

An assumption underlying this decomposition of the means is that the same factors that cause within-group variation also cause between-group variation. From a theoretical perspective, this assumption of common causation may be plausible (Dolan 1992; Dolan et al. 1994). An empirical check is also possible because it implies that the differences between racial groups depend on the magnitude of the path coefficients of the common factors. To examine this assumption, one can fit a model in which the means are estimated freely and do not depend on the path coefficients. By comparing the fit of the two models, the observer obtains an indication about the plausibility of the common causation assumption. Thus, if the deterioration in fit is substantial, the assumption may be unrealistic. If the difference in fit is very small, however, this finding supports the assumption that the same factors that cause within-group variation also cause between-group variation.

Model Fitting

We used the computer program Mx (Neale 1994) to analyze the covariance matrices and means in the groups of black and white siblings. To evaluate our null hypotheses pertaining to differences between black and white babies, we used several criteria.

First, we employed the chi-square difference test to test whether the target models (i.e., the various biometric and psychometric models) fit significantly less well than a saturated model. The test statistic equals the difference between the chi-squares of the saturated model and target model, and is chi-square distributed with the degrees of freedom equal to the difference in degrees of freedom of both models.

The second index was Akaike's Information Criterion (*AIC;* Akaike 1987), which has proved to be an effective index in many situations (Williams and Holahan 1994). Larger values indicate a poorer fit.

Finally, we used three incremental fit indices that reflect the improvement in fit compared with a baseline model: the Tucker-Lewis Index (TLI), the Incremental Fit Index (IFI), and the Comparative Fit Index (CFI). Marsh, Balla, and McDonald (1988) favor the TLI because it was relatively independent of sample size. Bentler (1990) prefers the IFI and the CFI because of the smaller sampling error. These incremental fit indices usually range from 0 to 1; higher values imply a better fit.

For birth weight and length of gestation, the total numbers of pairs and of families were 263 and 487 for black full siblings, 231 and 452 for black half-siblings, 907 and 1,609 for white full siblings, and 183 and 275 for white half-siblings. For birth length, the total numbers of pairs and of families were 183 and 341 for black full siblings, 148 and 295 for black half-siblings, 787 and 1,435 for white full siblings, and 159 and 244 for white half-siblings. These smaller sample sizes are the result of the larger percentage of missing values for birth length. To perform the analyses we further divided the siblings according to whether information on birth length was available; this operation resulted in 2 (full/half) × 2 (black/white) × 2 (birth length missing/present) = 8 different sibling groups. In the groups where information on birth length was missing, we specified the same model except that the part pertaining to birth length was omitted. In addition, if some values still were missing within the eight groups, we used pairwise deletion. Thus we obtained parameter estimates using all available information.

The sibling pairs in our analyses are not independent observations: For example, one family may yield multiple sibling pairs. For this reason we used the number of families, not the number of pairs, as the number of observations. This is a somewhat conservative approach, which tends to yield too few rejections of the null hypotheses.

RESULTS

Descriptive Statistics

We first examined whether our subsample of classified siblings was representative of the total sample. Eq. (1) indicates that the relevant statistics for our study are (on the one hand) the differences in means for black and for white babies, and (on the other) the genetic and environmental effects within

the groups. For this reason, in Table 1 we show the means, the standard deviations, and the correlations among birth weight, length of gestation, and birth length for both samples.

In the total sample, the means were consistently lower for black than for white babies. To quantify the differences, we used Cohen's (1988:40) criterion, which classifies an effect size of .2 as a small difference, .5 as a medium difference, and .8 as a large difference. The effect sizes for birth weight, length of gestation, and birth length were .395, .148, and .203 respectively, indicating nearly medium effects for birth weight, and small effects for length of gestation and birth length. The effect sizes in the subsample of classified siblings, .398, .137, and .255, showed a similar magnitude and an identical ranking to the total sample. This indicated that our subsample was representative regarding the magnitude of differences between black and white babies.

Although it is not possible to determine the size of the genetic and environmental effects in the total sample, we can obtain an impression of the representativeness of our subsample of classified siblings by comparing the phenotypic correlations and the standard deviations that are a function of these effects. Table 1 shows that for black babies the largest correlation involved birth weight and length of gestation; for white babies it involved birth weight and birth length. Furthermore, for blacks the correlations involving birth length were smaller than for whites. Finally, all standard deviations were larger for blacks. This was especially the case for birth length, for which the standard deviation was about 20% larger than among whites. We also found this pattern of correlations and standard deviations in the subsample of classified siblings; again, these findings supported the representativeness of our subsample.

The fact that trends and patterns in the total sample were consistent with those found in the subsample of classified siblings does not exclude the possibility of some variation within the total sample. To examine this possibility, we contrasted our subsample of classified siblings with siblings who could not be classified and with singletons. Effect sizes for unclassifiable siblings were .415, .147, and .209 respectively. These values corresponded closely to the effect sizes of .398, .137, and .255 found in the subsample of classified siblings. A chi-square test showed that in comparison with a saturated model (which gives a perfect fit so that the chi-square and the AIC are both zero), constraining these effect sizes to be equal did not result in a decrease in fit (chi-square difference = 6.17, $df = 3$, $p = .10$). Although the pattern of correlations and standard deviations was very similar to those described above for the subsample of classified siblings, the covariance matrices did not appear to be identical (chi-square difference = 76.04, $df = 12$, $p = .00$).

In the groups of singletons, the effect sizes of .225, .086, and .001 respectively were somewhat smaller than in classified siblings. When we tested for equal effect sizes using our subsample of classified siblings, the difference was not significant (chi-square difference = 7.01, $df = 3$, $p = .07$). It became significant, however, when we contrasted the singletons with the whole group of siblings. We also found differences with respect to the covariance matrix (chi-square difference = 100.53, $df = 12$, $p = .00$). The conclusion therefore seems to be that although our subsample was fairly repre-

TABLE 1. DESCRIPTIVE STATISTICS FOR TOTAL SAMPLE AND SUBSAMPLE OF RELATIVES

	Total Sample			Subsample of Siblings		
Statistic	Birth Weight	Length of Gestation	Birth Length	Birth Weight	Length of Gestation	Birth Length
Blacks						
Correlations	1	—	—	1	—	—
	.541	1	—	.490	1	—
	.459	.286	1	.335	.187	1
Mean	109.97	38.46	19.91	111.55	38.59	19.94
Standard deviation	22.23	2.44	1.73	21.27	2.32	1.69
Number of children	2,510	2,419	1,307	1,016	994	541
Number of families	870	961	714	415	412	304
Whites						
Correlations	1	—	—	1	—	—
	.493	1	—	.446	1	—
	.584	.387	1	.584	.381	1
Mean	118.54	38.81	20.22	119.93	38.88	20.30
Standard deviation	20.78	2.21	1.45	20.48	2.01	1.32
Number of children	4,678	4,564	3,469	2,421	2,401	1,874
Number of families	2,156	2,150	1,869	999	1,003	921

sentative of the total sample, there was some variation within the total sample, which mainly involved siblings versus singletons.

Alternative Models

The model displayed in Figure 2 assumes that birth weight, length of gestation, and birth length are indicators of the same set of common genetic and environmental factors. Although there are theoretical reasons supporting the notion of such a factor model (such as ample evidence that these indicators share the same sociodemographic risk factors; see Institute of Medicine 1985), alternative explanations for the correlations also may exist. For instance, gestational length could be the *cause* of differences in birth weight and birth length.

To examine this possibility we used the phenotypic correlations between birth weight, length of gestation, and birth length reported in Table 1. For a causal model, the rules of path analysis imply that the observed correlation between birth weight and birth length should equal the product of the "causal" effects of length of gestation on birth weight and the "causal" effects of length of gestation on birth length. Thus, for black babies, the predicted correlation would be .541 × .286 = .155; for white babies the predicted correlation would be .493 × .387 = .191. Yet the actual correlations of .459 for blacks and .584 for whites were more than three times as large as expected for a causal model. Furthermore, chi-square tests indicated that the model did not fit for black (chi-square = 16.34, $df = 1$, $p = .00$) nor for white babies (chi-square = 13.07, $df = 1$, $p = .00$). These findings are clearly inconsistent with a causal model, and supported the plausibility of our factor model.

In the analyses described here, we assume that the population mean for birth weight is lower among black than among white babies. Under a "birth trauma model," however, it would be possible to have equal means for blacks and for whites but an excess of black low-birth-weight or preterm babies. This excess could be due to genetic and environmental hazards that are qualitatively distinct from the genetic and environmental factors that describe continuous variation and that are responsible for a general shift in group means.

To examine the plausibility of this alternative model, we categorized birth weight into 19 categories (< 20, 20–30, 30–40,…,180–190, > 190 oz.); the frequency distributions for blacks and whites are displayed in Figure 3. The figure shows that birth weight was distributed normally (the skewness index of –.71 for blacks and –.38 for whites suggested that the distributions were skewed slightly to the left), and that the whole distribution of blacks was shifted to the left in relation to the distribution of whites. Furthermore, when we computed the effect size for birth weight with the exclusion of low-birth-weight babies (< 2,500 g), we obtained an effect size of .372. This was almost identical to the effect size of .390 reported previously for the whole sample. This finding indicated that the exclusion of low-birth-weight babies did not change the difference in means, either for black or for white babies. These findings supported the validity of our model. They showed that differences in birth weight cannot be explained by an extreme group but are a characteristic of the whole population.

We obtained similar results for length of gestation and birth length. For instance, when we computed the effect size for length of gestation with the exclusion of preterm babies (< 37 weeks), it equaled .127. Again, this was close to the effect size of .139 that we found in the whole sample.

Genetic Analyses

Table 2 shows the correlations for pairs of full and half-siblings. For different variables there are two sibling correlations (e.g., birth weight in sibling 1 with birth length in sibling 2, and birth weight in sibling 2 with birth length in sibling 1). To obtain a single correlation, we entered the measurement of each child one time as the measurement of the first sibling and one time as the measurement of the second sibling. We used this double entry only for the presentation of the descriptive statistics, and not in the significance tests.

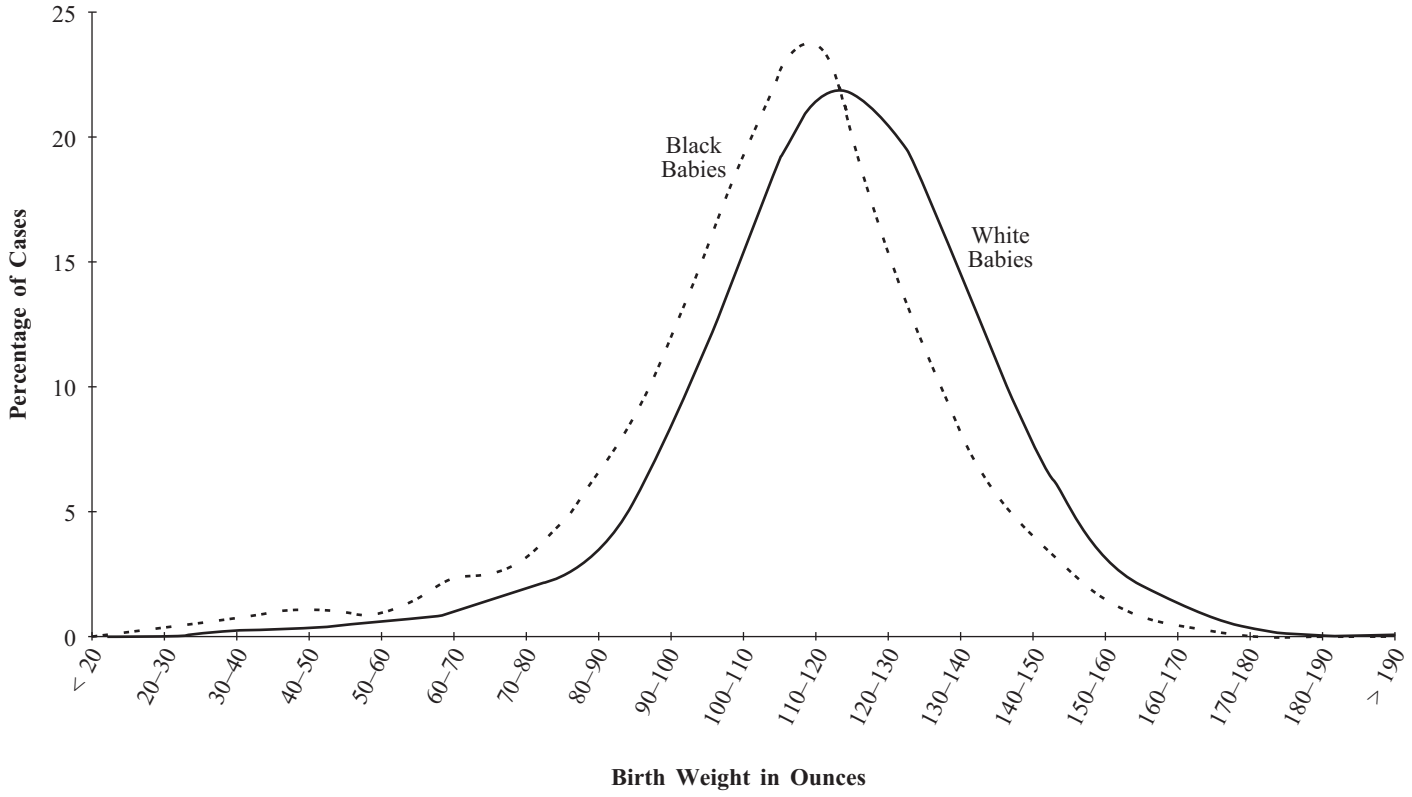
Twice the half-sibling correlation minus the full-sibling correlations yields an estimate of shared environmental influences. This formula suggested the largest shared environmental influences for birth weight and the smallest shared environmental influences for length of gestation. We confirmed this possibility with the results of univariate model fitting, which showed that shared environmental influences accounted on average for 30.7%, 0%, and 6.5% of the variance in birth weight, length of gestation, and birth length. These magnitudes of shared environmental effects were ranked identically to the effect sizes for birth weight, length of gestation, and birth length: .398, .137, and .255 respectively. Thus the magnitude of the difference in means between blacks and whites covaried with the size of the shared environmental effects. This finding suggested that shared environment may explain the differences in birth health risk between black and white babies.

The results of univariate model fitting showed that nonshared environmental influences accounted on average for 65.1%, 65.9%, and 37.1% of the variance in birth weight, length of gestation, and birth length. This ranking suggested that the magnitude of the nonshared environment effects was unrelated to the effect sizes, and that nonshared environment may not contribute to differences in means for black and for white babies.

Genetic effects are suggested by full-sibling correlations larger than half-sibling correlations. The sibling correlations indicated such a trend: This was confirmed by the results of the univariate model fitting, which showed that genetic influences accounted on average for 4.2%, 34.1%, and 56.5% of the variance in birth weight, length of gestation, and birth length. The genetic effects were smallest for birth weight, whereas the effect size was largest for birth weight. This finding suggested that the magnitude of the genetic effects did not covary with the effect sizes and that genetics may not contribute to the differences between black and white babies.

A final remark with respect to Table 2 is that among blacks, but not among whites, some of the half-sibling correlations were negative or larger than the full-sibling correla-

FIGURE 3. BIRTH WEIGHT DISTRIBUTION IN BLACK AND WHITE BABIES



tions. Sample fluctuations are a plausible explanation. That is, if the genetic effects are small (as suggested, for example, by the univariate results for birth weight), the half-sibling correlations will be only slightly smaller than the full-sibling correlations (the expected difference equals half the heritability). Furthermore, if the shared environmental effects are small (as suggested, for example, for length of gestation and birth length), the expected half-sibling correlations, which reflect only one-quarter of the heritability, will be close to zero. Thus, because of sample fluctuations in both half- and full-sibling correlations, in these situations half-sibling correlations quite possibly could be larger than full-sibling cor-

TABLE 2. CORRELATIONS BETWEEN SIBLINGS FOR BIRTH WEIGHT, LENGTH OF GESTATION, AND BIRTH LENGTH

Variable	Full Siblings			Half-Siblings		
Black Babies						
	1.	2.	3.	1.	2.	3.
1. Birth weight	.222	—	—	.281	—	—
2. Length of gestation	.066	.148	—	.098	.026	—
3. Birth length	.140	.069	.432	.083	–.005	–.075
White Babies						
	1.	2.	3.	1.	2.	3.
1. Birth weight	.403	—	—	.378	—	—
2. Length of gestation	.179	.222	—	.071	.045	—
3. Birth length	.279	.122	.307	.262	.070	.243

relations or close to zero. This explanation also seemed to be supported by the fact that such half-sibling correlations did not occur in the sample of white babies, which is about 2.5 times larger than the sample of black babies.

Before evaluating our hypotheses pertaining to differences between black and white babies, we performed a number of preparatory tests. Fit indices showed that our independent pathway model (depicted in Figure 2) fit better than the common pathway. That is, the difference in chi-square was significant (chi-square difference = 13.45, $df = 6$, $p = .04$), and the AIC was lower for the independent pathway model, another indication of a better fit. This finding was consistent with the univariate results reported above, showing that the relative importance of genetic and environmental effects differed considerably for the three observed measures of birth health risk. This variation in relative importance suggested that it is possible to attribute mean differences to separate genetic and environmental sources rather than to a common psychometric factor.

Next we included the means in our independent pathway. We examined whether the means were identical for the first and the second sibling, and similar in groups with and without birth length measurements. This test did not suggest differences (chi-square difference = 31.34, $df = 28$, $p = .30$). A significant difference resulted, however, from the inclusion of the additional constraint that the means were to be equal for full and for half-siblings (chi-square difference = 62.65, $df = 34$, $p = .00$). This indicated that the means were somewhat lower for half-siblings than for full siblings. The effect sizes for birth weight, length of gestation, and birth length, however, were .29, .07, and .21 for blacks and .20, –.04, and .10 for whites. These values showed that the differences between full and half-siblings were small and were similar for blacks and for whites.

In a final preparatory test we modeled the means in both racial groups as a function of common genetic and environmental factors, as in Eq. (1). To let this model converge to a proper solution, we imposed the boundary constraint that the means for black babies could not be higher than for white babies. The difference in fit was very small (chi-square = 268.599, $df = 122$, $p = .00$ versus chi-square = 269.00, $df = 122$, $p = .00$). This finding suggested that the means could very well be modeled as a function of the common genetic and environmental factors, and it supported the assumption of common causation.

Table 3 shows the fit indices used to evaluate our hypotheses. The baseline for the chi-square difference tests was the *saturated* independent pathway model described above, in which the means were modeled as a function of the common genetic and environmental factors (chi-square = 269.00, $df = 122$, $p = .00$). The null model used to compute the TLI, the IFI, and the CFI differed for each set of hypotheses. For the tests that examined whether the genetic and environmental effects differed from zero, the model assumed that all phenotypic correlations were zero. For the tests that assumed no differences in path coefficients, it assumed equal phenotypic correlations. For the tests examining differences in factor means, it assumed equal phenotypic means for black and for white babies. The hypothesis was rejected if the model incorporating the constraints implied by the hypothesis resulted in a poorer fit: that is, if the chi-square difference test indicated a significant decrease in fit in comparison with the saturated model, if the AIC was larger, and if the TLI, the IFI, and the CFI were smaller.

Table 3 shows that the five fit indices yielded almost exactly the same conclusions. The only exception was the IFI, which deviated from the other four indices in two conditions. The major conclusions were that in contrast to the nonshared environmental effects and the effects of the common shared environmental factor, the genetic and unique shared environmental effects did not differ from zero. Only the effects of the common nonshared environmental factor differed for black and for white babies. In addition, the difference in means seemed to be caused by the common shared environmental factor because a poorer fit resulted when the means of this factor were constrained to be equal for black and for white babies.

On the basis of the results from Table 3, we specified a reduced parsimonious model in which there were no genetic effects, and that allowed only group differences in common nonshared environmental path coefficients and shared environmental factor means. Table 4 shows the parameter estimates for the saturated independent pathway model as well as for this reduced model. We computed the proportion of variance explained by a certain factor by dividing the squared path coefficient of that component by the sum of the squared path coefficients of all components. In the initial model, for instance, the common shared environmental factor explained $(9.81)^2/((3.61)^2 + (9.81)^2 + (13.09)^2 + (7.78)^2 + (0.00)^2 + (11.35)^2) = 20.5\%$ of the total variance in birth weight for blacks. Similar calculations showed that on average in both racial groups, the common genetic, shared environmental, and nonshared environmental influences respectively explained 22.2%, 11.3%, and 20.8% of the total within-group variance of the three observed variables.

We found considerable differences, however, between the three indicators of birth health risk. For instance, genetic factors were much more important for birth length than for birth weight or length of gestation. The unique genetic, shared environmental, and nonshared environmental factors explained on average 14.2%, 0.3%, and 31.2% of the total variance. This relatively large contribution by nonshared environmental factors may be explained in part by the fact that in contrast to the common nonshared environmental effects, the unique nonshared environmental influences are confounded with errors of measurement.

Table 4 shows for each variable the total genetic and environmental contribution to the racial differences in means. We computed these deviations by multiplying in each group, for a given component, the path coefficient and the deviation from the overall mean (also see Eq. (1)). For birth weight among black babies, for example, the path coefficient for the common shared environmental factor was 9.81, and the factor deviation was .42. Therefore the total contribution of the

TABLE 3. FIT INDICES TESTING THE SIGNIFICANCE AND EQUALITY OF GENETIC AND ENVIRONMENTAL CONTRIBUTIONS

Model	Chi-Square Difference Test			Stand-Alone Fit Index	Incremental Fit Indices		
	χ^2	*df*	*p*	AIC	TLI	IFI	CFI
Path Coefficients Different From Zero							
Baseline	1,444.62	18	0.00	1,438.56	—	—	—
Saturated	—	—	—	29.94	0.908	0.925	0.924
Common path coefficients							
Genetic	1.88	6	0.93	19.83	0.917	0.927	0.926
Shared env.	17.10	6	0.01[a]	35.05[a]	0.906[a]	0.917[a]	0.916[a]
Nonshared env.	27.42	6	0.00[a]	45.36[a]	0.898[a]	0.910[a]	0.910[a]
Unique path coefficients							
Genetic	4.49	6	0.61	22.44	0.915	0.925	0.925
Shared env.	0.00	6	1.00	17.94	0.918	0.928	0.928
Nonshared env.	240.97	6	0.00[a]	258.92[a]	0.742[a]	0.773[a]	0.772[a]
Equal Path Coefficients in Black and White Babies							
Baseline	312.56	18	0.00	306.50	—	—	—
Saturated	—	—	—	29.94	0.656	0.726	0.714
Common path coefficients							
Genetic	1.33	6	0.97	25.27	0.672	0.728	0.718
Shared env.	2.69	6	0.85	26.63	0.668	0.725[a]	0.715
Nonshared env.	28.24	6	0.00[a]	52.18[a]	0.596[a]	0.665[a]	0.653[a]
Unique path coefficients							
Genetic	0.83	6	0.99	24.77	0.673	0.729	0.719
Shared env.	0.00	6	1.00	23.94	0.675	0.731	0.721
Nonshared env.	0.27	6	1.00	24.22	0.675	0.730	0.721
Equal Means in Black and White Babies							
Baseline	99.42	3	0.00	118.41	—	—	—
Saturated	—	—	—	25.00	0.381	0.403	0.396
Means							
Genetic	1.14	1	0.29	22.14	0.395	0.402	0.400
Shared env.	6.45	1	0.01[a]	27.44[a]	0.373[a]	0.380[a]	0.378[a]
Nonshared env.	0.00	1	1.00	21.00	0.400	0.407	0.404

[a]Null hypothesis must be rejected on the basis of fit index.

common shared environmental factor to the deviation from the overall birth weight mean was 9.81 × –.42 = –4.14. Analogously, the total contribution of the common shared environmental factor to the deviation from the overall birth weight mean for white babies was 11.52 × .42 = 4.86. Because the overall mean was 114.87, the mean for black babies equaled 114.87 – 4.14 = 110.73, and the mean for white babies equaled 114.87 + 4.86 = 119.73.

Finally, we comment on the relative power to detect genetic and environmental effects. Results for the initial model reported in Table 4 suggested that common genetic effects were relatively important for birth length but not for the other two variables. In contrast, common shared environmental effects were relatively important for birth weight but not for the other two variables. Table 3 shows, however, that the test for common shared environmental effects was significant but that the test for genetic effects was not significant. The explanation is that our design had less power to detect genetic effects than to detect shared environmental effects. Thus the nonsignificance of these results does not necessarily imply that genetic effects were not important for birth health risk; it also could reflect the lesser power to detect these effects.

DISCUSSION

Our results suggested that the gap between black and white babies' birth weights may be explained by the shared environment of the fetuses, not by fetal genes nor by environmental influences that affect one pregnancy but not another. This finding implies that the relevant environmental factors

TABLE 4. PARAMETER ESTIMATES FOR INITIAL AND REDUCED MODEL[a]

	Common Factors			Unique Factors			Contribution to Mean Difference		
Variable	Genetic h	Shared Env. c	Nonshared Env. e	Genetic u_h	Shared Env. u_c	Nonshared Env. u_c	Genetic $h \times E(A)$	Shared Env. $c \times E(C)$	Nonshared Env. $e \times E(E)$
Black Babies									
Birth weight	3.91	9.81	13.09	7.78	0.00	11.35	–0.29	–4.14	0.00
	—	12.33	16.13	—	—	9.21	—	–4.85	—
Length of gestation	0.23	0.36	1.53	0.99	0.00	1.35	–0.02	–0.15	0.00
	—	0.48	1.30	—	—	0.06	—	–0.19	—
Birth length	1.55	0.28	0.23	0.00	0.00	0.86	–0.13	–0.12	0.00
	—	0.66	0.16	—	—	1.20	—	–0.26	—
White Babies									
Birth weight	7.85	11.52	9.11	0.00	0.00	11.24	0.64	4.86	0.00
	—	12.33	12.79	—	—	9.21	—	4.85	—
Length of gestation	0.35	0.51	0.65	0.00	0.00	1.40	0.03	0.22	0.00
	—	0.48	0.83	—	—	1.77	—	0.19	—
Birth length	1.27	0.16	0.55	1.55	0.28	0.23	0.10	0.07	0.00
	—	0.66	0.51	—	—	1.20	—	0.26	—

[a]The first line for each variable gives the results for the saturated model; the second line gives the results for the reduced model. For the saturated model, the respective overall means for birth weight, length of gestation, and birth length were 114.87, 38.73, 20.06; the deviations from the factor means E(A), E(C), and E(E) were .08, .42, and .00. For the reduced model, the overall means were 115.48, 38.72, 20.05; the deviations from the factor means were .00, .39, and .00.

are correlated with birth health risk, are stable across pregnancies, and have different average levels among blacks and among whites. It is clear that traditional socioeconomic variables such as poverty and years of education fall into this category. A number of authors, however, showed that these variables explain only part of the racial gap (Cramer 1987, 1995; Gortmaker 1979; Kallan 1993; McCormick 1985; Sappenfield et al. 1987). What other variables, then, satisfy these criteria and have additional explanatory power?

The shared environmental effects on birth weight, the indicator of birth health risk that differed most between black and white babies, was clearly larger in this study than is usually found for human phenotypes (Plomin and Daniels 1987). This finding does not seem to be a matter of chance, and it is consistent with other studies demonstrating the importance of these effects for within-group variation in birth weight (Boomsma et al. 1992; Carr-Hill et al. 1987; Morton 1955; Nance et al. 1983). Shared environment usually consists of family or cultural factors. For birth weight, however, it has a somewhat different meaning, referring to characteristics of the fetal uterine environment that are constant across pregnancies. Therefore the relatively large shared environmental influences may be explained by a birth-weight-specific component that is labeled "maternal effect" because the uterine environment is provided by the mother.

In our sample of siblings, the importance of maternal effects could not be assessed directly. Yet because the NLSY sample includes cousins, we could compute birth weight correlations between cousin pairs. These correlations equaled .091 for blacks (528 pairs, 90 families) and .032 for whites (746 pairs, 171 families); for half-siblings these correlations were .281 and .378 respectively. This finding suggested that babies who share the same uterine environment are much more alike than babies who do not, and provided an additional indication for maternal effects.

It is unlikely that such a large difference between half-sibling and cousin correlations can be explained by fetal genetic effects, because the genetic factors imply a difference equal to one-eighth of the heritability of birth weight (the difference in the genetic correlations for these groups is .25 – .125 = .125). Because the heritability of birth weight appeared to be low (e.g., the univariate analyses indicated an average heritability of .04), it is unlikely that genetic factors account for the relatively large difference between half-sibling and cousin correlations.

Given the possible importance of maternal effects for the fetal shared environment, the determinants of maternal effects could be a second set of variables that contribute, in addition to the traditional socioeconomic variables, to the racial gap in birth weight. To study these determinants and to decompose the maternal effects into genetic, shared environmental, and nonshared environmental contributions, groups of genetically related mothers would be necessary (e.g., mothers who are full or half-siblings).

Although Nance et al. (1983) did not have such a design, they commented "At all birth ranks, factors that make MZ twins alike, presumably mostly genetic, accounted for 72% of the maternal variation" (pp. 1221–22). These authors did not make explicit why they regarded the "common maternal" effect as mostly genetic, but they noted that it makes

evolutionary sense for maternal biology to exert some control over birth weight:

> The ability of the mother to constrain the growth of the conceptus regardless of the fetal genotype has obvious survival value. By allowing parents discordant for body size to reproduce freely, the maternal control of fetal growth removes what might otherwise constitute a formidable obstetrical barrier to panmixia. (1983:1217)

This remark seems to indicate constant physical or physiological characteristics of the mother. For such factors, which last for years in adult life and create birth weight similarities in siblings, it would seem reasonable to suppose a strong genetic component rather than assuming that they arose merely because the mothers were raised in the same household. Furthermore, for statistical reasons the relevant determinants of maternal effects should themselves be stable so as to appear as fetal shared environment in our study. Because it is unlikely that a woman's genetic effects on the uterine environment change substantially within the few years that pass between the births of her children, a genetic explanation satisfies this requirement.

The above comments suggested that in addition to the traditional socioeconomic variables, the fetal shared environment could be determined by maternal effects, possibly with a strong genetic component, that are related to physical or physiological characteristics of the mother. To explain the racial gap, these factors also should differ systematically between blacks and whites. There is evidence that this may indeed be the case. Rushton and Bogaert (1987), for example, reported a large number of physical characteristics and physiological processes, such as gamete production and twinning rates, that are related to reproduction and differ across racial groups.

A tremendous research effort has been made to explain the gap between black and white babies' birth weights. This research is important because of the negative association between low birth weight and health or developmental outcomes. Even though the traditional "environmental" variables provide only a partial explanation, genetic factors are rarely considered. There are no a priori reasons, however, to assume that such effects are unimportant. That is, in addition to the obvious environmental differences, there are demonstrable genetic differences consisting of variations in gene frequencies across racial groups. If these genes influence birth weight, they will contribute to the racial gap. In fact, some authors even have argued that racial differences may originate largely from genetic factors (Rushton 1995: chaps. 7, 11; Rushton and Bogaert 1987).

Therefore, to produce a comprehensive picture, the possible role of genetic factors should be evaluated empirically, or at least considered. In the present study we make a first attempt in that direction. Our findings suggest that the differences can be traced to the shared environment, which can be produced socially or genetically in the case of birth weight. This genetic effect would pertain not to fetal genes but to the mother's genes, possibly those affecting her physical or physiological characteristics.

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