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Poverty and Behavior: Are Environmental Measures Nature and Nurture?

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This article critiques the *Child Development* special issue on poverty. First, we argue that the special issue has understated the variation observed within social class groups. Second, we believe that a confounding of genetic and environmental effects in biological families weakens the case for environmental effects as presented in the special issue. Our conclusion is that behavior genetic research designs are required for studies of poverty.

In the April 1994 *Child Development* (*CD*), a distinguished group of scholars united to examine the influence of family poverty on children's developmental outcomes. The articles concluded that poverty is detrimental to children's intellectual and personality development. Children of poor parents had worse educational outcomes, higher rates of externalizing behavior, and more depressive symptoms than children of higher income parents (e.g., Connell, Spencer, & Aber, 1994; Dodge, Pettit, & Bates, 1994; Sampson & Laub, 1994; McLoyd, Jayaratne, Ceballo, & Borquez, 1994). Several articles also tried to find *mediators* of poverty's effects. That is, they measured developmental processes linking poverty with children's traits. For example, the relation of poverty to developmental outcomes was reduced when variation in home environments was controlled statistically (Duncan, Brooks-Gunn, & Klebanov, 1994). Other articles found that independently of some parental traits (e.g., maternal education), poverty still worsens home environments. Weakness in research design, however, compromises the contribution of the CD special issue to scientific knowledge about the effects of poverty.

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A mediator model can be used to describe the stated or implicit conceptual model of articles in the *CD* special issue as follows: *Family Poverty* causes *Family Socialization Practices*, which in turn cause *Children's Developmental Outcomes.* Although a special issue cannot be expected to consider all possible influences on poverty, we believe that this mediational model has neglected the possibility of biological influence on the attainment of socioeconomic status. Our critique is focused on the existence of genetic mediators of poverty effects.

First, we believe that the mediational model used in the special issue tended to exaggerate the effect sizes of poverty by downplaying the variation in developmental outcomes observed within social class groups. Second, we argue that the *CD* special issue's mediational model of poverty can lead to overestimates of the environmental effects of poverty. We do not argue that the effects of poverty necessarily vanish when biological influences are accounted for in a model; rather on both substantive and statistical grounds, we postulate that model-estimated effects would be biased without such a specification (Meehl, 1970). We advocate using behavioral genetic research designs when investigating poverty outcomes, and we illustrate the use of such designs with some findings from the *National Longitudinal Survey of Youth.*

The issues raised here are not new; the methodological consequences of ignoring genetic effects have been recognized for many decades. For example, Burks, in her pioneering studies of foster children, noted that some part of the family environment–child IQ association could arise from their shared association with parental and child heredity (Burks, 1928; 1938). Indeed, Burks' use of the method of path analysis, which had been invented not long before by the geneticist Sewall Wright (1923), was decades ahead of its time. Later in this article, Fig. 1 is a conceptual descendant of Burks' (1938) path model. Her conclusion, that 75–80% of IQ variance was due to innate and heritable causes and that family environmental effects were weak, did not lead social scientists, then or now, to routinely adopt behavior genetic strategies in the evaluation of family environmental effects.

In the postwar period, a considerable social controversy swirled around a publication by Jensen (1969a) in the *Harvard Education Review* that suggested possible genetic variation in racial and social class differences. Jensen was physically threatened for presenting these views; many criticisms of him by social scientists were *ad hominem* in content (Pearson, 1991). Despite a gradual accumulation of data on the heritability of most personality and intellectual traits (Plomin, DeFries, & McClearn, 1990), most critics did not respond by advocating that Jensen's hypotheses be taken seriously and that research be launched on group mean differences using research designs able to cope with genetic variation.

Jensen (1969b) coined the phrase ''sociologists' fallacy'' for the automatic assumption that all effects of social categories (e.g., race, ethnicity, social

class, intact vs two-parent families, and so on) must contain only environmental effects; he observed that

> . . . the statistical matching of racial groups on SES and other environmental factors is an invalid method in any of these studies, since it presumes that SES, etc., are entirely causal variables. Since there is substantial evidence that there are genetic as well as environmental differences between SES groups (within races), a matching procedure (statistical or actual) results in some degree of matching on the genetic as well as the environmental factors involved in development. (Jensen, 1969b, p. 220–221)

If these methodological cautions had been taken to heart, this article would be unnecessary. However, despite strong evidence that researchers need to be concerned with genetic variation in measures based on social categories (Jensen, 1973), it continues to be ignored. Indeed, the recent reawakening of the nature vs nurture debate about social class and racial differences, by the controversial book *The Bell Curve* (Herrnstein & Murray,1994), still has not led to methodological changes within the social sciences. Thus, this article will reiterate these themes in the context of critiquing the special issue of *Child Development*. But before discussing any genetic effects on measures of social class, the general issue of the magnitude of social class effects should be first examined.

OVERSTATING THE MAGNITUDE OF POVERTY EFFECTS

Table 1 shows representative *effect sizes* for associations between family income variables and children's behavioral traits drawn from articles in the *CD* special issue that provided correlations for income or socioeconomic status (SES) measures. The correlation coefficients (or standardized β weights) ranged from .38 in Walker, Greenwood, Hart, and Carta's (1994) small study of social class and language production ($N = 32$) to $-.02$ in Conger, Ge, Elder, Lorenz, and Simon's (1994) study of economic decline in the Midwest. Despite heterogenous populations and reasonable sample sizes, the study by Conger et al. produced small social class effects (see their articles for additional correlations). The small effect sizes in Leadbeater and Bishop (1994) and McLoyd et al. (1994) may reflect their homogeneous samples, which consisted primarily of disadvantaged mothers and their children. Three large and relatively heterogeneous samples produced effects sizes of about .30 for delinquency (Sampson & Laub, 1994), IQ (Duncan et al., 1994), and externalizing behaviors (Dodge et al., 1994). An effect size of about .30 appears to be an upper bound for social class effects on these behavioral traits in children, an estimate that is in good agreement with the effect size estimates from a classic meta-analysis of the relationship between parental SES and child's academic achievement (White, 1982) . In this meta-analysis, the correlation of SES averaged across studies was .33 with student's IQ and .27 with a composite of their academic achievement.

It is important to recognize that the use of mediational models cannot

TABLE 1
Direct Relationships between Socioeconomic Status Measures and Children's Traits

Outcome	r/β	Source
1. Income to needs–IQ age 5	.32	Duncan *et al.*
2. Income to needs–internalizing	−.15	
3. Income to needs–externalizing	−.16	
4. Economic risk–neg (pos) outcomes in New York	.16 (−.16)	Connell *et al.*
5. Economic risk–neg (pos) outcomes in Atlanta	.07 (−.22)	
6. Economic risk–neg (pos) outcomes in New York City/B/DC	.12 (−.13)	
7. Family poverty–delinquency	.33	Sampson & Laub
8. Income loss–antisocial	.06	Conger *et al.*
9. Income loss–trait depression	−.02	
10. Income loss–trait anxiety	−.07	
11. Financial strain–general anxiety	−.02	McLoyd *et al.*
12. Financial strain–depressive symptomatology	−.06	
13. Income–receptive language	.38	Walker *et al.*
14. Income–spoken language	.37	
15. Income–verbal ability	.11	
16. On welfare–child behavior problem	−.07	Leadbeater & Bishop
17. Social economic status and externalizing in Grade 3	−.34	Dodge *et al.*
18. Social economic status and peer nominated aggression in Grade 3	−.20	

change these effect sizes. A mediational model merely apportions the poverty–children's trait association into a part that is mediated through other measured variables and a part that is direct. The latter effect could be a direct one of poverty on the outcome, or it could represent effects of unmeasured mediational variables. However, the apportionment in a mediational model cannot increase the original effect size—rather, it would break an effect size into its component parts.

McLoyd's and Conger's studies serve to illustrate small effect sizes in mediational models. In summarizing their findings, McLoyd et al. actually implied the existence of rather strong effect sizes:

> Notwithstanding the modest relations between individual variables assessed in the study, a relatively impressive amount of the variance in mothers' punishment, and adolescents' general anxiety and depressive symptomatology was explained by equations that included both the hypothesized predictor and mediator variables. (p. 586)

Although this statement was true when applied to *all* variables in the statistical model, it was not true for those measures of socioeconomic status. In their Table 2, one finds only one significant correlation of 12 correlations computed between three socioeconomic status measures and four child behavior outcomes. Furthermore, one significant correlation, maternal unem-

ployment with general anxiety, accounted for just 2% of the variation in anxiety.

The effect sizes were also small in the Conger et al. (1994) study. Their structural equation model permits derivation of the effect of a latent Economic Pressure variable on a latent trait of Adolescent Externalizing Symptoms (see their Fig. 1, p. 556). Using the structural path coefficients for fathers, the total effect of Economic Pressure on Externalizing had the mathematical expectation that follows:

$$r = .10 = .32x - .01 + .59x.57x.26x.35 \quad (1)$$
$$+ .59x.57x.47x.45x.35 + .32x.45x.35$$

This .10 effect of Economic Pressure, which removed measurement unreliability and so was adjusted upward, was modest. These results would suggest that at least in a White population in rural Iowa, social class would leave substantial variability in children's externalizing behaviors unexplained.

In summary, the effects of social class on children's behavioral outcomes were quite modest in most studies. If an effect size of about .30 is accepted for IQ and externalizing behaviors (from the large studies based on heterogeneous populations), then several implications follow. The first is that a 1 *SD* increase in social economic status would yield a .3 *SD* improvement in children's traits, *if the entire association was causally environmental.* We examine this assumption in a later section. The second implication is that the middle class population would contribute the majority of children with serious behavioral problems. Although the incidence of low IQ and behavior problems would be greater among the poor, the three-fifths of the population that is middle class would contribute the majority of new incidences, simply because they are more numerous than the bottom one-fifth. If, by some powerful social intervention, no poor families had offspring with behavior problems, the numbers of troubled children in the United States would still be substantial because so many are born into middle class homes. The articles in the *Child Development* special issue, by focusing solely on poverty, underemphasized this important fact.

CAUSAL MODELS OF SOCIAL CLASS

In their introduction to the special issue, Huston, McLoyd, and Coll (1994) emphasized that understanding the effects of poverty requires an attack on the root problem of poverty, stating that ''. . . interventions continue to focus on changing the child or family without addressing the root of the problem, namely, poverty'' (p. 281). Of course, this latter approach requires that social scientists make the indices of poverty, such as low incomes and low levels of education, the *dependent* variables and then develop causal models of them. A weakness of the articles in the special issue is that few of them devoted attention to the causes of poverty, the actual variable of interest.

As Huston et al. observed, poverty can be regarded from both relative and absolute standpoints. The relative meaning of socioeconomic status is in the ranking of adults within a population according to earned income, education, and occupational prestige. The absolute meaning of poverty is a threshold below which a family lacks the cash income to purchase essential goods (e.g., food).

The causes of poverty are extremely multidimensional and complex (Wilson, 1987). Further, absolute and relative poverty may have different determinants. The ability of a nation to generate wealth through its economic activities plays a major role in determining absolute poverty. According to Jencks (1992, p. 146), if the noncash welfare benefits are considered, about 10.5% of the U.S. population fell below the poverty line in 1988, in comparison with 23.1% in 1959. Yet by comparison to developing countries, our rates of absolute poverty are extremely low, and malnutrition is rare. Absolute poverty rates in the United States, however, are substantially above those in other industrialized countries. Structural changes also can redistribute wealth; for instance, in the logging economies of the Northwestern states, family incomes were devastated by changes in public policy regarding the clear cutting of trees on federal lands.

Although economic structural conditions can create poverty, we theorize that individual-level personality and cognitive traits also contribute. Because they are individual difference variables, they and indices of relative poverty (e.g., income, years of education) are more amenable to causal modeling. Of course, poverty levels, however defined, can be related to incomes and education levels so that the relative and absolute measures of poverty can be bridged. Nonetheless, absolute poverty could be eliminated without reducing substantially the social inequalities represented by different levels of incomes, educations, and technical skills.

HERITABLE TRAITS AND SOCIOECONOMIC STATUS

To have a causal model of socioeconomic status, then, one must switch its role from an independent to a dependent variable. In this approach, childhood traits become *antecedents* of adult socioeconomic statuses. For example, second grade IQ can anticipate adult years of both education and occupational status (McCall, 1977). Social mobility occurs to the extent a child achieves an adult status above or below that of his parents. In each generation, about 30% of children move upward in social status (relative to their parents), about 30% move downward, and the remainder stay in place.[1] Upward and downward mobility also may depend on IQ (Waller, 1971). To the

[1] Each child receives a random sample of maternal and paternal genes—thus, within-family genetic reassortment can lead a child to possess genes more favorable to high IQ than possessed by either parent, or conversely, more genes unfavorable to high IQ than possessed by either parent.

extent that heritable traits such as IQ and conscientiousness (Loehlin, 1992) contribute to upward and downward social mobility, measures of SES should themselves be heritable. That is, the genetic variation in these heritable traits is part of the total variation of SES measures.[2]

In a review of the genetics of environmental measures Plomin and Bergeman (1991, p. 382) concluded that both twin and adoption studies indicate genetic variation in socioeconomic status measures. On the basis of the few studies directly examining social class measures, heritability estimates for SES fell into the 40–50% range. In behavior genetics, correlations on first degree relatives would be doubled to estimate heritability, because a mother or father and a child share only half their genes, not all their genes. For occupational status, doubling the correlation between biological fathers and their adopted away sons (.20) yields a heritability of 40% ($N = 2467$ pairs; Teasdale, 1979). In a twin study done in the United States, ''years of education'' gave a heritability of 50% (Taubman, 1976).

Hence, labeling a measure ''environmental'' does not mean that all components of its variation are environmental (Ge, Conger, Cadoret, Neiderhiser, Yates, Troughton, & Stewart, 1996; McGue, Sharma, & Benson, 1996; Perusse, Neale, Heath, & Eaves, 1994; Plomin, 1994; Rowe, 1994). Indeed, any ''environmental'' mediator variable in the *CD* special issue, such as the HOME measure of family environmental quality, can be regarded as a dependent variable in a behavior genetic study. Because individual differences in heritable traits can influence parenting styles, the latter also may be as heritable as ordinary traits. In Plomin's *Genetics and Experience* book and in Plomin and Bergeman's review article, parenting styles, amount of television viewing, peer group orientations, perceptions of social support, and reports of negative life events were all shown to be heritable.

The HOME measure of family environmental quality was used as the dependent variable in several special issue articles. This measure has been subject to behavior genetic analysis in the Colorado Adoption Project (Braungart, Fulker, & Plomin, 1992). HOME sibling correlations were compared for 105 biological and 85 adoptive pairs of infant siblings. As these correlations were greater for the biological pairs, model-fitting procedures gave positive heritability estimates (h^2 = about .40 when the infants were both 1 and 2 years). Therefore, although one can attribute the majority of variation in HOME scores to environmental variation plus error (60%), genetic variation is still a substantial component. Plomin and Bergeman cautioned that ''even though these are very early days in research on the nature of nurture,

[2] Udry (1994) built his presidential address to the *Population Association of American* around the theme that demographers have neglected biological processes in their modeling. Because the variables used in the *CD* special issue—such as gender and SES—are strongly demographic in origin, Udry might just as well have been addressing child development experts. The importance of biological modeling is a cross-discipline issue.

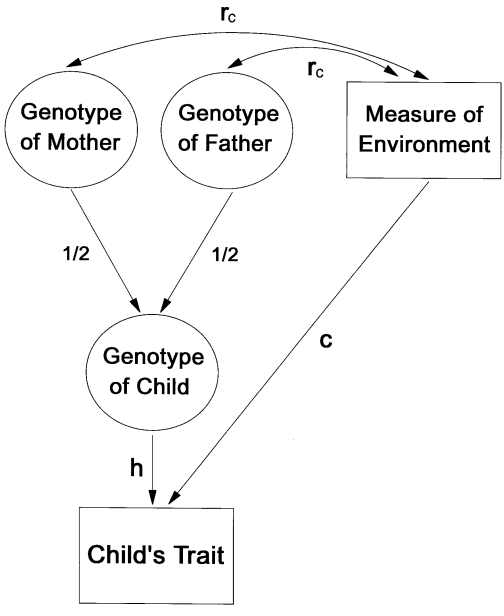

FIG. 1. Relation of environmental measure to child trait scores in biological families (*h*, genetic path coefficient; *c*, environmental path coefficient; r_c, correlation of family environment with parental genotype).

the results would so far seem to shift the burden of proof to those who continue to assume that environmental measures are free of genetic influence'' (p. 384).

THE CONFOUNDING OF GENETIC AND ENVIRONMENTAL INFLUENCES IN STUDIES OF POVERTY

The confound of genetic variation in environmental measures renders most findings in the *CD* special issue on poverty ambiguous. Genetic influences must be controlled when the influence of family environmental measures on child developmental outcomes are assessed (Plomin, Loehlin, & DeFries, 1985; Rowe, 1994). If genetic influences are likened to a drug therapy and environmental ones to a psychosocial therapy, most social scientists would recognize limitations of a research design with just two experimental conditions: a control condition and an experimental condition in which both therapies were received by patients. Without other combinations of placebo therapy and drug therapy, or placebo drug and psychosocial therapy, the effect of one form of intervention could never be separated from that of the other. Unfortunately, studies of biological families present just such poor research designs (which would likely not be tolerated in the evaluation or treatment intervention literature).

Figure 1 clarifies these limitations of the family study research design.

Parental genotypes may influence both a measure of family environment and children's trait scores. This confound means that in biological families, the environmental pathway (c) would be confounded with a genetic influence pathway (hr_c) as follows:

$$r_{\text{environment-child}} = h(.5r_c + .5r_c) + c = hr_c + c, \quad (2)$$

where c is the direct effect of the environment on the children's trait scores, h is related to heritability, r_c is the correlation of a parental genotype and the environmental measure, and the genetic correlation of parental and child genotypes equals 0.50 because genetically related parents and offspring share one-half their genes on average. In families in which family members were biologically unrelated, however, this confound fails to occur because the correlation of parental and children's genotypes would be zero instead of one-half (in the absence of selective placement). Comparison groups formed from unrelated family members (e.g., adopted and stepchildren) may provide one means of separating these genetic and environmental confounds. In this latter family type, only the environmental path c would link the environmental measure and children's traits.

In general then, the biological family design would overestimate environmental effects, if any genetic effects act commonly on the environmental measure and children's traits. This exaggeration of environmental effects can be demonstrated by Scarr and Weinberg's (1981) Minnesota Adoption Study. Children 16 to 22 years old in 104 adoptive and 120 biological families were compared. All the adoptive children had been placed before 12 months of age. The predictive power of four SES measures on adoptive children's IQs (father's education, mother's occupation, father's occupation, and family income) was greater in biological families ($R = .33$) than in adoptive families ($R = .14$). The zero order correlation of rearing mother's education and children's IQ was .28 ($N = 262$, $p < .05$) in the biological families and .09 ($N = 188$, ns) in the adoptive families (Scarr & Weinberg, 1980). Furthermore, the adoptive children's IQs correlated .28 ($N = 184$, $p < .05$) with their nonrearing, biological mother's IQs. These numbers clearly show estimates of the environmental effect of social class would be greatly exaggerated if data from biological families were relied on. The environmental estimates would fall downward by 58% ([.33−.14]/.33) for the overall regression equation and 68% ([.28−.09]/.28) for mother's years of education.

Despite the availability of this evidence as long ago as Burks (1928), it has had little influence on the interpretations given for studies of SES. For the most part, the articles in the special issue chose specifications that ignored the possibility that their estimates of environmental effects may have been enhanced by genes shared by parents and their children. However, Dodge et al. (1994) did acknowledge the possibility of genetic effects, but only in the remainder of covariation between SES and externalizing behavior (once quality of parenting had been partialled away):

> ''The possibility also remains that factors other than family social experiences that might be associated with SES might also contribute to antisocial development (such as *genetically* endowed factors. . . .'' (p. 662, italics added)

Although appropriate, Dodge et al.'s caveat misses the main implication of the behavior genetic findings: genetic variation exists in the variance of measurements labeled as environmental, not just in the residual variance. Although Scarr and Weinberg (1978, reprinted, 1981) set this direction 16 years ago as one needed in research, their call for a truce between ''camps'' and for research on SES exploring possible genetic effects has gone unheeded. As they observed,

> Although it may be distasteful to some social scientists to acknowledge genetic sources of individual and SES differences, it appears to us important to recognize the biosocial nature of human variation. (p. 397, Scarr & Weinberg, 1981)

CONTROLLING FOR GENOTYPES USING MEASURED PHENOTYPES

Although a concern for heritable traits was absent in most *CD* special issue articles, an exception was Sampson and Laub's (1994) reanalysis of the Gluecks' (1950) classic study of delinquency in Boston males born between 1924 and 1935. They proposed a latent trait model in which a latent variable labeled *Informal Social Control* (i.e., three parent–child interaction variables) directly causes delinquency. In turn, the *Social Control* variable had as determinants *Parent/Child Dispositions* and *Structural Context* (e.g., poverty, family size, residential mobility). Their overall model gave an excellent statistical fit (i.e., a χ^2 of 30, almost equal to the 28 degrees of freedom in the model, $p = .35$). Thus, their inclusion of Child Dispositions was an attempt to evaluate the effects of poverty (and related variables), independently of ''child effects,'' which might relate to children's heritable traits:

> Although difficult children who display early antisocial tendencies do appear to self select or sort themselves into later states of delinquency, family processes of informal social control still explain a significant share of variance in adolescent delinquency . . . analyses further suggest that the effect of childhood antisocial/difficult behavior is mediated by family process. (p. 538)

Although this overt consideration of ''child effects'' is a great advance over studies that ignore the issue, the methods of statistical control proposed by Sampson and Laub would be weak attempts to deal with genetic variation. Their statistical model failed to represent genetic influences as possible latent variables that could influence both parenting and children's outcomes. In a study with, for example, an adoptive comparison group, the correlation of parenting variables with children's delinquency might be greatly reduced, if these associations partly reflected correlated genetic variation (although they would not necessarily be reduced to zero, as parents may be responding to children's characteristics). Having data from such an quasi-experimental

comparison group would impose many additional constraints for a structural equation model to meet. It is in these added constraints that a resolution of genetic and environmental influences can occur— statistical controls for measured phenotypes alone would be inadequate for this purpose.

THE RELATION OF ETHNIC AND RACIAL GROUP MEMBERSHIP TO POVERTY

The overrepresentation of minority groups (and some recent immigrants to the United States) among the United States poor is well known. For this reason, the special issue included samples in which blacks, Hispanics, and other minorities were overrepresented in comparison to their share in the general population.

The articles, however, did not clearly enough focus on an essential question in understanding poverty; namely, whether the *causal models* of poverty differ by ethnic or racial group. The term ''causal model'' refers here to an understanding of the processes leading to, away from, and maintaining poverty in a particular population. In practical terms, a causal model describes the correlations (or covariances) among putative influence variables (e.g., needs relative to income) and children's developmental outcomes.

A common misunderstanding is to believe that group mean differences necessarily imply the need for group-specific causal models. This is not so. Group means may differ for two reasons. On the one hand, the causal processes may differ between Group A and B. For example, in Group A, teenage pregnancy may be the result of peer influence. In Group B, teenage pregnancy may arise from permissive parenting. In this situation, different group means (e.g., 10% pregnancy rate in group A, 17% in group B) may have arisen through different social processes. On the other hand, group mean differences can arise through common processes, if the groups differ in mean levels on the antecedent variables. For example, in both Groups A and B, teenage pregnancy may have arisen from peer influence in various ways, but the peers in Group A may have a view of teenage pregnancy that is less tolerant than those teenagers in Group B.

In research not directed in particular at poverty—although including many of the kinds of children's traits as emphasized in the *CD* special issue— Rowe, Vaszonyi, and Flannery (1994) found strong support for the hypothesis that developmental processes were generally similar across ethnic and racial groups. Applied to the question of poverty, there is evidence in the *CD* special issue for ethnic/racial similarity in developmental processes, despite differences in either the prevalence or mean-level of children's traits. For example, Dodge et al., despite finding higher mean-levels of externalizing in black than white children, concluded that their models of developmental processes were ''. . . not substantively moderated by race'' (p. 663). That is, independent variables such as mother's aggressive values, peer stability, and cognitive stimulation interrelate with each other and the outcome vari-

able in much the same way across racial groups. Similarly, Duncan et al. (1994) observed, in their footnote 12, that their regression equations involving poverty were similar for blacks and whites. In the effort to see differences among ethnic and racial groups, these important and broad domains of ethnic and racial group similarity are often missed.

GENETIC VARIATION IN ENVIRONMENTAL MEASURES AS CONFOUNDS FOR ETHNIC AND RACIAL COMPARISONS

A conventional assumption in the *CD* articles is that controlling for socioeconomic differences would equate racial and ethnic groups *environmentally*. Consider these illustrative quotes:

> At all ages, African–American boys and girls were rated by teachers and peers as higher in conduct problems than white children. However, these differences were fully accounted for by SES; that is, once SES was taken into account, race differences in both teacher-rated and peer-nominated conduct problems became nonsignificant. (Dodge et al. 1994, p. 663)

Or, consider the views of Duncan et al. on reducing IQ differences between black and white children: ''Family and neighborhood income differences go a long way in accounting for the differences in IQ scores of black and white children'' (p. 314).

In their introductory article for the volume, Huston et al. (1994) also emphasize the practice of equating racial and ethnic groups for any SES differences: ''It is now standard practice to introduce statistical sampling controls for SES when assessing the effects of race/ethnicity'' (p. 277).

However, they were cautious about the ability of equalizing on SES to account fully for social context differences between blacks and whites.

Our criticism is that equating groups for SES controls for both environmental and genetic variation. The existence of genetic variation in measures such as the HOME, years of education, and income indicate that an interpretation of these statistical controls as purely environmental effects would be unwarranted. At best, they are ambiguous as to the processes reducing the group differences: a greater equality of genetic variation, a greater equality of environmental variation, or a combination of both. Social scientists should acknowledge the causal ambiguity of such statistical analyses.

Advances in research design, and in statistical models, however now make the group difference question empirically approachable. The group difference question is one of genetic and environmental components of group means, where the groups may be defined by socially achieved categories such as single-parent vs two-parent families, high vs low social class, divorced vs married, and so on, or on the basis of ascribed categories such as sex, race, and ethnicity. These methods require two basic elements in their research design. First, latent variables must be used (e.g., IQ, disruptive behavior disorders), where a latent variable has a measurement model with three or

more indicators. Second, the research design must be a genetically informative one in which kinship types are nested within the larger groups. For example, suppose the question is one of the means of two-parent vs single-parent families. To make this a behavior genetic study, four groups would be needed: MZ twin children of single parents, DZ twin children of single parents, MZ twin children of two parents, and DZ twin children of two parents. When both conditions have been met, the genetic and environmental components of group means can be estimated (Dolan, Molenaar, & Boomsma, 1992; Rowe & Cleveland, 1996). A detailed description of the biometric *model of means* is beyond the scope of this article. As a statistical approach, however, it offers one important property; it can be used to check statistically whether group means and variation within groups have the *same causal determinants.* Only if this check is passed would a model then be used to estimate genetic and environmental components of group mean differences. With these advances in the statistical modeling of means, there is little reason for continuing to avoid a genetically informed approach to the study of group means.

FURTHER DIRECTIONS FOR FUTURE RESEARCH

At present, we have been investigating genetic and environmental influences in one sample used in studies of poverty: The National Longitudinal Survey of Youth (Rodgers, Rowe, & Li, 1994a; Rodgers, Rowe, & May, 1994b; see in special issue, Garrett, Ng'andu, & Ferron, 1994; Caughy, DiPietro, & Strobino, 1994). The NLS-Youth began in 1979 as a household probability sample of 11,406 civilian respondents aged 14–21 years in the United States. Since then, the respondents have been resurveyed at regular intervals. The NLS-Youth was the sample used by Herrnstein and Murray (1994) in *The Bell Curve.*

A computer algorithm identified different kinships among the NLS- Youth (Rodgers, Rowe, & Buster, 1996). This algorithm identified 3890 kinship pairs for possible classification. Cousins were unambiguously identified from the files as such. If both members of a kinship pair agreed that they were cousins, they were classified as such. Probably some non-first cousins called themselves cousins. However, as this was probably a rare error, our estimate of a genetic relatedness for this group (coefficient of relatedness, $r_g = .125$) is probably only a slight overestimate. There was no information in the files to allow the classification of twins. Because opposite-sex twins must be fraternal, they were assigned an $r_g = .50$. Of the remaining same-sex twin pairs, about half will be monozygotic and half dizygotic. They were given a genetic relatedness of .75.

The sibling pairs were most critical because of the large sample size. In 1988 (when they were 23 to 30 years old), each respondent created a retrospective time line from 0 to 18 years of age of whether they lived with their biological father and their biological mother in each year. The critical target

year was the year in which the sample was drawn, 1979, the only year in which we know for certain that a given sibling pair was living together in the same household. By linking respondent's ages to 1979, an ordered quadruple was constructed indicating whether sibling 1 lived with biological mother and/or father in 1979, and whether sibling 2 did the same. Thus, a (1,1,1,1) response pattern would indicate two siblings, both of whom were living in the same household, and both of whom were living with their biological mother and father. These siblings must be unambiguously full siblings ($r_g = .50$). Similarly, a response pattern of (1,0, 1,1) indicates two siblings living together in 1979, both of whom were living with their biological mother, but only one of whom was living with their biological father. Such pairs were classified as half-siblings, as was any other pattern of three 1's and one 0. Furthermore, response patterns of (0,0,1,1) and (1,1,0,0) were classified as adoptive/stepsiblings ($r_g = 0$).

The remaining pattern was ambiguous. A (1,0,1,0) or (0,1,0,1) indicates two siblings who share one biological parent, and were therefore at least half-siblings. For those whose fathers were in question, the algorithm used another variable indicating how far away the fathers lived to help resolve those ambiguities. Some respondents were older than 18 in 1979; many of them could not be classified because there was no age to definitely tie them to both their sibling and their biological parents (because their response to whether they were living with biological mother or father stopped at age 18, before the year 1979 was reached). A total of 2338 kinship links were defined by this procedure of the original 3890 pairs (a 60% classification rate). This algorithm identified a fewer percentage of matches than the one applied to the NLS-Children (see Rodgers et al., 1994a; 1994b). The identified pairs broke down into 32 twin pairs of unknown zygocity, 1877 full sibling pairs, 43 half-sibling pairs, and 76 cousin/adoptive sibling pairs. Another 310 pairs were ambiguous, either full or half silbings; they were given a genetic relatedness of $r_g = .375$.

Using the data in the NLS-Youth (now adults), we can focus on genetic and environmental components of ''environmental'' variation. Table 2 presents correlations for the pairs of siblings sharing a childhood environment: half, mixed half and full, full, and same-sex twins. The ''environmental'' measures were income (dollars earned per hour) and years of education. Although some correlations had wide confidence intervals because of the small number of pairs, it is clear that, except for twins' income, the correlations between adult siblings' SES increased linearly with genetic relatedness. The income correlations increased from .11 for half-siblings to .30 for full siblings; the education ones, from $-.22$ to .77 in twin pairs. In the 17 pairs of half-siblings, the negative sibling correlation of $-.22$ probably reflected the instability of correlations computed on small samples; we expected that the half-sibling correlation on income would be positive. Similarly, the 90% confidence interval for the twin's income correlation extended as high as

TABLE 2
Sibling Correlations for ''Environmental Measures'' in the NLSY-Youth

Environmenal measure	Sibling *r*	90% confidence interval
Half-siblings (r_g = .25, N = 17 pairs)		
Education	−.22	−.61 to .17
Income	.11	−.29 to .52
Mixed full and half-siblings (r_g = .375, N = 156 pairs)		
Education	.39	.32 to .46
Income	.17	.04 to .30
Full siblings (r_g = .50, N = 169 pairs)		
Education	.53	.49 to .57
Income	.30	.25 to .35
Same-sex twins (r_g = .75, N = 12 pairs)		
Education	.77	.57 to .97
Income	.12	−.37 to .61

Note: Income less than $1 per hour and $250 per hour were removed as outliers. Income was then log-transformed before computing the sibling correlations. Standard errors estimated from $1.65 \times (1 - r^2)/\text{sqr}\ (N - 1)$.

r = .61. In overview, the sibling correlations were consistent with a hypothesis of genetic influences on variation of both environmental measures. Certainly, Herrnstein and Murray's thesis (1994) in the *Bell Curve* that life outcomes may be substantially shaped by genetic variation can be supported with these findings from the same data set as they had used. Genetic influence cannot be ignored in studies of the relation of SES measures to children's behavioral traits because genes constitute a major source of variation in the former.

Although we were able to capitalize on the NLS-Youth household sampling frame, other large scale studies of poverty lack informative kinships for behavior genetic analyses. We strongly encourage future data collections on poverty to include genetically and environmentally informative kinships. Methods exist for the analysis of trait variance components, such as heritability, or of greater relevance to environmentally oriented researchers, for the analysis of environmental measures within genetically informative research designs (see Rodgers et al. 1994a; Rowe & Waldman, 1993; Neale & Cardon, 1993).

CONCLUSIONS

Behavior genetic studies should be used when the environmental effects of poverty are the target of investigation. The flawed design of studying biological families (Rowe, 1994), which inevitably would confound parental heredity with family nurture, obfuscates rather than illuminates the environ-

mental effects of family socioeconomic status and poverty. This observation is not a new one; it was made by Barbara Burks (1928; 1938) prior to World War II. Although she was one of child development's most important methodologists, her contribution has been ignored by a discipline often unwilling to take the consequences of genetic variation seriously.

No single behavior genetic research design can simultaneously answer all the research questions about poverty. Research designs that include group means (e.g., Dolan et al., 1992; Rowe & Cleveland, 1996) offer a potentially powerful method for estimating genetic and environmental components of group *mean* differences related to poverty. In these designs, a shared environmental factor (which makes siblings alike) can represent environmental socioeconomic status effects, whereas a genetic factor can represent genetic effects attributable to people self-selecting into different social groups. Some difficult issues require special research designs that may be difficult to implement in practice. For example, gene *x* environment correlation of the active type can be estimated in a full adoption design in which the parenting practices of adoptive parents are correlated against the biological traits of their adoptive children as estimated from the traits of the adoptive children's biological parents (but for an example, see Ge et al., 1996). Although the requirements of behavior genetic research design are always more stringent than simply collecting information on biological families (Neale & Cardon, 1992), they represent the only methodology able to disentangle poverty's environmental effects from genetic ones that may masquerade as environmental effects. Although today, latent variables are used to represent genes, molecular genetics may lead to the identification of behaviorally relevant genes (Plomin, Owen, & McGuffin, 1994). If this effort succeeds, then genetic differences among people in different social categories will be directly observable at the DNA-level, and new methodologies for discovering gene–environment correlations and interactions will be created for developmental researchers.

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