CHAPTER 5

UNITING NATURE AND NURTURE: THE GENETICS OF ENVIRONMENTAL MEASURES

In *Nature's Thumbprint*, a New York City psychiatrist and his son present a case history of early-separated identical twins who were raised apart:

> Identical twin men, now age thirty, were separated at birth and raised in different countries by their respective adoptive parents. Both kept their lives neat—neat to the point of pathology. Their clothes were preened, appointments met precisely on time, hands scrubbed regularly to a raw, red color. When the first was asked why he felt the need to be so clean, his answer was plain.
>
> "My mother. When I was growing up she always kept the house perfectly ordered. She insisted on every little thing returned to its proper place, the clocks—we had dozens of clocks—each set to the same noonday chime. She insisted on this, you see. I learned from her. What else could I do?"
>
> The man's identical twin, just as much a perfectionist with soap and water, explained his own behavior this way: "The reason is quite simple. I'm reacting to my mother, who was an absolute slob." (Neubauer & Neubauer, 1990, pp. 20-21)

In hindsight, how easily we can explain any behavior by drawing upon our experiences in childhood! To one twin, blissfully unaware of the other, his mother's obsessiveness had produced his own. To the other twin, blissfully unaware of the first, his mother's slovenly habits produced an opposing impulse in himself—a compulsion toward neatness and cleanliness. Neither twin thought to look inside himself for the causal influence—to the genes that instruct biological development, to

their similar minds, but we, as neutral observers, can forgive their common error of believing that what their parents did made them who they are. Rearing explanations may be seductive and flexible, but false.

This chapter discusses genetic variation in "environmental measures." We already understand that variation in rearing experiences, beyond rare extremes, has little influence on personality development. Nonetheless, rearing measures do possess statistical associations with personality and intellectual traits, as verified in hundreds of studies of biological families in the working-class to professional-class range. These associations are usually interpreted as "influence," although as social scientists we understand that correlation does not mean causation—a piece of advice often ignored in studies of childhood socialization. A lack of inferred rearing influence implies that these statistical associations cannot be causal ones; instead, they must be *spurious*, depending on genes shared by parents and children (in biological families) to create an appearance of causality. The insights needed to understand this phenomenon are simple. First, we need to recognize that variation in "environmental measures" may contain genetic variation; second, we need to see that this genetic variation may produce an appearance of rearing influences on children.

The Genetics of Social Class

Changes in Explanations for Class and Racial Differences

The most widespread explanations of behavioral differences among both children and adults are social class and culture. Socialization science relies on social class and culture for environmental explanations of behavioral pathologies (such as criminality and insanity), as well as of variation in IQ and scholastic achievement. During the period from 1900 to the beginning of World War II, class and cultural explanations replaced the formerly pervasive biological theories of racial and class differences in behavior.

Environmentalism prevailed for diverse reasons (Degler, 1991). One was that conceptions of inheritance changed. A Lamarckian could both believe in the genetic superiority of Caucasians and be a social reformer, because Lamarck's theory held that new traits acquired during one's lifetime could be passed on genetically to the next generation. In genetics,

when scientific advances showed that the Lamarckian doctrine was false, social reformers had to abandon it for some form of cultural influence if the "lower" races were to be raised, or the socially disadvantaged improved. The excesses of the eugenics movement also drove scholars away from biological explanations. In the United States, liberals vehemently opposed the political successes of the eugenics movement, which encouraged laws in many states permitting compulsory sterilization of the intellectually retarded. Geneticists, who formerly supported the movement, also abandoned it. One reason was scientific: For some traits, eugenics would be a slow and halting process, because deleterious, recessive genes respond to selective pressures only slowly.

In the period from 1900 to the 1930s, the anthropologists, psychologists, and sociologists who joined a movement toward cultural and class explanations expressed views that are now widely accepted (Degler, 1991). The psychologist Klineberg used a cultural explanation for the poor performance of Native Americans on speeded tests of intellectual ability (i.e., that their cultural values placed less emphasis on speed than did economically competitive American mainstream society), and he used both cultural and social class explanations for African-Americans' poorer test performance (i.e., their lack of educational and economic opportunity, surely evident in the United States in 1935). The anthropologist Kroeber assumed equal moralities and potentialities in all races; in his view, any observed difference could be attributed to a lack of exposure to rearing environments able to activate them. The sociologist Kelsey, once he had abandoned Lamarckianism, found that cultural inheritance made him more optimistic that racial and class differences in behavior could be eliminated, as soon as better environmental provisions were given to all. When Nazi racial theories furnished a final proof that biology could be used to justify the most horrendous acts of inhumanity, environmental explanations came to dominate the social sciences completely.

Today, socialization science depends, without much reflection or analysis, on variations in social class and culture as environmental explanations of the seemingly intractable class and racial differences seen in the United States—intractable because many additional years have not ended disparities in IQ and scholastic achievement favoring whites over blacks, and favoring professional occupations over working-class ones. Modern college textbooks commonly repeat the cultural and class explanations that first drove biology from social science in the 1920s and 1930s:

> Poor diets, poor health, poor schooling, and a way of life that does not require or reward abstract thinking, all can reduce intellectual capacities regardless of genetic potential. In this way, Sowell's careful study demolished notions of inborn [white over black] racial superiority. (Stark, 1985, p. 110)

> Racial and social-class differences in IQ test results are adequately explained by cultural factors. The problem is, however, that IQ tests are widely used as a basis for labeling and tracking students, providing yet another opportunity for the self-fulfilling prophecy of academic success or failure to occur. (Robertson, 1981, p. 393)

The raw emotion with which any challenges to class and cultural explanations are greeted reflects this historical fact: Such explanations freed socialization science, at least temporarily, from hereditarian arguments about class- and race-related developmental outcomes, and thus provided social scientists with a platform for social reform. But a disquieting threat to environmentalism lies in the idea that racial or class variation may itself be genetically based. This line of reasoning so threatens concerns for social welfare that its avoidance has undermined thorough research on sensitive topics such as race and class. It is one reason why some theories of socialization prefer to avoid genetics altogether.

Of course, "race" and "class" are not equivalent constructs. Social class levels are permeable to people of diverse ethnic backgrounds and individual characteristics; physical, racial characteristics are evolutionary legacies, and they are unchanging attributes ascribed to people. Specialized research designs can be applied to studying possible genetic bases of racial differences in behavior, including transracial adoption and genetic admixture research designs.[1] Although this research can be done, such studies are difficult to conduct, and data from them are sparse. Furthermore, standard behavior genetic research designs work well with social class variation, but poorly with racial variation: Among identical twins separated and raised apart, cases may be found where one twin is middle-class and another is lower-class, but one cannot find a case in which one twin is Caucasian and another is African-American. Because there is better evidence on class than on racial variation, the remainder of this section focuses on the former.

My thesis here is that social class may capture not variation in rearing and environmental social background, but instead variation in genes. This idea returns genes to socialization science by a back door—*by the very variable (social class) thought to have liberated social science from hereditarian thinking*! The present argument requires a somewhat dif-

ferent perspective: My question is not "What does social class predict?," but rather "What makes for social class differences in the first place?"

Social class can be measured by means of several popular indices: (1) years of education completed; (2) occupational prestige; and (3) family income. Unlike the violent storms that regularly hit Florida's coasts to wreck homes and property, class attainments do not represent environments imposed on adults by natural events beyond their control; rather, they represent what individuals *earn or find* through years of effort, mixed with good and bad luck. As was not the case in the rigid monarchies of pre-World War I Europe, social mobility between generations is a fact of life in the industrialized West today, nearly as persistent as death and taxes. Some children rise to a social class status above that of their fathers (and in this more liberated era, mothers); other children fall to a status below that of their parents; and still others remain in about the same place. If individuals' social class partly results from their behavior, then it can reflect genetic variation in the traits and abilities that may determine whether people rise, fall, or remain static. What we call "environment" can be, in part, genetic (Herrnstein, 1973).

Although these observations seem simple, social scientists have studiously avoided them. Apparently, we do not want to wrestle with the implications of admitting that 5%, 10%, or 25% of social class variation may be genetic. A common statement such as "the IQs of middle- and working-class children are more alike when equated for years of parental education" loses its cogency if variation in a social class measure is itself partly genetic. If so, equating groups on class matches them genetically as well as environmentally. And if genes can cause behavioral variation, then it may come as no surprise to us that genetically matched groups are no longer as different from one another as unmatched ones. This reasoning does not prove that class differences are genetic in origin, of course, but it does shake the habitual confidence that they are not.

Genetic Influence on Social Class Variation: Jencks's Model and an Alternative

Not all postwar scholars ignored their intellectual obligation to deal with how genetic variation may influence social class variation. In his seminal book *Inequality*, Christopher Jencks (1972) attempted to evaluate genes' contributions to social status. He used data on the genetic inheritance of IQ in his statistical models of income, concluding that the genes' contribution to men's income variation was a rather small one: "First, genes account for no more than 10 percent of the [income] difference. As usual, biological explanations for the inheritance of privilege do not take us very far" (p. 215).

More specifically, Jencks attributed 7–9% of income variation between men in the upper and lower social classes to IQ genes; 16–20% to IQ advantage attributable to the superior environment of the upper class; 24–29% to the extra schooling for those with equal IQs; 18% to higher-status occupations for those with equal IQs and equal schooling; and the 30% remaining to an additional income advantage of higher-status men after the men were equated on all other factors. Now even a 7–9% contribution of genes to income should deserve some consideration in socialization science, but Jencks's conclusion, and even his concern, are somehow absent from the pages of many social science textbooks. The feeling may be that although a genetic contribution exists, it is small enough to be neglected (although a correlation of .30 is a typical magnitude in socialization science and explains 9% of variation).

Matters become more serious, however, if flaws in Jencks's methods resulted in his underestimating genes' influence on social class differences. I can identify one such subtle flaw in Jencks's logic, and this must be understood before other data on the transmission of social advantage are considered. Table 5.1 presents correlations among genes, IQ at age 11 years, years of education, and adult income, provided by Jencks or derived from his data. Jencks inferred the genes' correlations with other variables from a particular statistical model—one based upon "causal chains". In such a model, one variable causes the next in a chain, along with new influences unrelated to the prior variables, which enter at each new place. With causal chain models, a well-established prin-

TABLE 5.1. Illustrative Correlations under Christopher Jencks's Model

	Genes	IQ, age 11 years	Years of education	Income (adult)
Genes	1.00			
IQ, age 11 years	.71[a]	1.00		
Years of education	.16[a]	.58[b]	1.00	
Income (adult)	.14[a]	.24[a]	.35[b]	1.00

[a]The correlations are derived from Jencks's (1972) data by means of various path-analytic models.
[b]The correlations are from Jencks (1972).

ciple is that the correlation of a variable at the chain's tail with any variable downstream is simply the product of the statistical associations linking them.

Figure 5.1 (top) illustrates the causal assumptions embodied in the more complex chain models that Jencks actually used. The "head" variable is IQ genes, which affect IQ at age 11, which in turn affects education, which then leads to the chain's "tail" (income). The path coefficient is the correlation of each variable with the next one in the chain. Each variable downstream from IQ genes is also affected by other influences, as represented by the vertical arrows. Since they are unmeasured influences, we do not need to concern ourselves with them.

Suppose we want to know the correlation between IQ genes and income. It is simply the product of the numbers along the chain: .71 ×

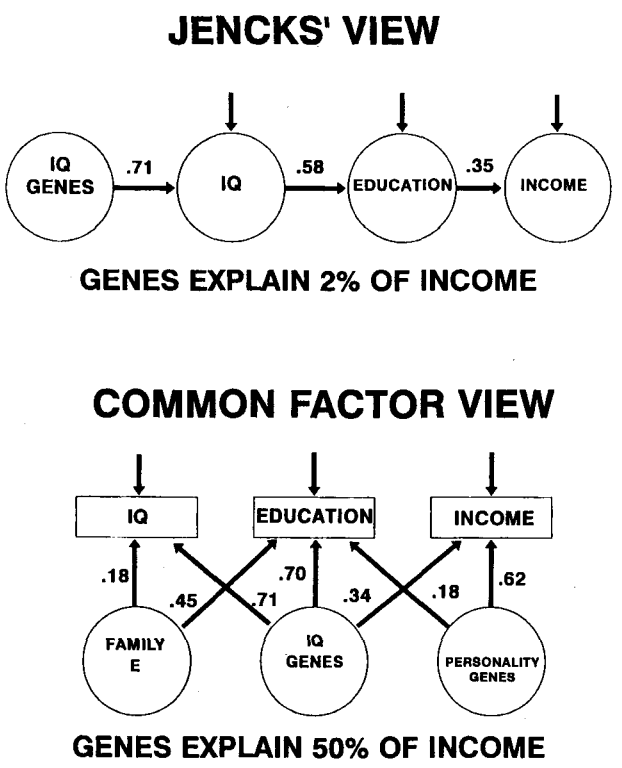

FIGURE 5.1. Genetic variation and income levels: two views.

.58 × .35 = .14. Thus, according to a chain model, genes explain just 2% of the variation in income ($.14^2 = .02$), an estimate actually below Jencks's 7–9%. His higher estimates came from a model containing more variables and pathways, but this model does reproduce the *logic* of Jencks's more complicated ones.

What is seriously wrong with Jencks's chain model? Its problem is that genes do not produce a test score at age 11, which next directly causes years of education, which next directly causes incomes. Rather, genes produce a phenotype—a person with particular intellectual abilities and weaknesses. Persons' strengths and weaknesses affect their encounters with the IQ test at age 11, the demands of schooling, the opportunities of the job market, and the rigors of succeeding in a job. Thus the influence of the genes is not mediated through the test score itself, and Jencks's model as a literal representation of genetic influence becomes misleading.

Figure 5.1 (bottom) shows an alternative representation of influences on social status. IQ genes, personality genes, and family environment influence IQ at age 11, education, and income simultaneously. Personality genes have been added because, as Jencks acknowledged, "Genes may . . . influence certain personality traits, and these may influence a man's earning power" (1972, p. 262). The figure neglects the temporal lags between events by assuming that the same IQ genes that affect adult income also affect IQ at 11 years. This assumption is warranted because considerable overlap exists between genes influencing intelligence in childhood and adulthood.[2] The figure also omits any correlation of family environment and genes, but we know that more intellectually capable parents will provide their children with more intellectually stimulating home environments. More complex behavior genetic models can estimate the effects on IQ of this genotype × environment correlation (Loehlin & DeFries, 1987), but for the present illustrative purposes this complication is omitted.

As before, the correlations among variables can be calculated by multiplying the statistical associations on the pathways that connect them. Figure 5.1 has been drawn to replicate the correlations among observable variables in Table 5.1. So income and IQ correlate .24 (.71 × .34), education and IQ correlate .58 (.71 × .70 + .45 × .18), and education and income correlate .35 (.70 × .34 + .18 × .62). Figure 5.1 shows the same correlation between IQ and IQ genes as Table 5.1 (r = .71). However, IQ genes correlate .70 with years of education and .34 with income,

as opposed to .16 and .14 in Table 5.1. The model in Figure 5.1 has been chosen as a conceptual illustration, not as a true partition of the variation in IQ, education, and income.

The common-factor world view depicted in Figure 5.1 departs from Jencks's original in one major respect. The influence of genes becomes much greater than before—the squares of the statistical associations on the arrows pointed at the measured variables. Thus IQ genes now explain 12% (.34 × .34) of variance in adults' incomes, as opposed to a mere 2% in the pure chain model (Figure 5.1, top). The addition of personality genes increases the genetic contribution to income variation by 38% (.62 × .62), so that gene substitutions, in total, explain about *50%* of income variation! The model allows rearing to influence education: One-fifth of education variation (.20 = .45 × .45) is attributable to rearing, whereas 49% is attributable to IQ genes (.49 = .70 × .70).

But which view of the world is more correct—Jencks's view that "biological explanations . . . do not take us very far" in the explanation of income, or the view that a major part of income variation is attributable to genes and that little is attributable to variation in family environments? The answer must come from behavior genetic studies that use social status itself as the outcome. Neither education nor income is a trait in the same sense as eye color or brain dopamine concentrations; however, heritable traits can create genetic variation in education and income through an influence on levels of accomplishment, in classroom learning and later in the workforce. Thus a behavior genetic analysis can be done on years of education or on income as though they were individual traits, and it can seek the degree of total genetic variation in them.

Behavior Genetic Studies of Social Status

An economist, Paul Taubman (1976), tried this approach with one of the largest and most representative American twin samples: the World War II Veterans Twin Panel. These adult male twins all served in the military during World War II and were identified through their military records. Except for the physically handicapped, felons, and people with serious mental or psychological handicaps, the population of World War II veterans spanned a wide cross-section of American society—with a wide range of years of completed schooling, and incomes from poverty to wealth. The twin registry included about 1,000 MZ twins and

1,000 DZ twins. Years of education yielded twin correlations of .76 for identical pairs and .54 for fraternal pairs (or, in terms of variance components, 44% for heritability, 32% for rearing environment, and 24% for unshared environment). For income, the results were even less encouraging to rearing influences. The MZ twin brothers correlated .52, whereas the DZ brothers correlated only .30 (or, in terms of variance components, 44% for heritability, 8% for rearing, and 48% for unshared environment). Ironically, Jencks's low estimate of family influence on attained income would be correct—but for *rearing* environments rather than for genes, as only 8% of variation in income in Taubman's study owed to the environmental advantages some families were able to confer on their children.

Some children, of course, do inherit fortunes from their parents. However, this is true in only a very small percentage of cases, so that monetary inheritances fail to alter the picture of little overall family environmental advantage for children's incomes in adulthood in the population at large. Indeed, years of education is a better "environmental" variable than is income, because at least some variation in years of education can be attributed to environmental advantages conferred by rearing. In Norway, however, even variation in educational attainment is primarily genetic rather than attributable to rearing environment (allowing for assortative mating, h^2 = .60; Tambs, Sundet, Magnus, & Berg, 1989). And as the last two chapters have shown, these rearing differences, though effectively influencing years of schooling completed in the United States, lack influence on most personality or intellectual capacity traits, because rearing influences on these traits are for the most part nil. Behavior genetic studies conducted in other countries also demonstrate genetic variation in standard social class measures (Tambs et al., 1989; Teasdale, 1979; Teasdale & Owen, 1981).

As I have noted already, social class measures thus contain genetic variation because heritable traits are associated with life accomplishments. Richard Herrnstein proposed the following syllogism relating abilities to social standing (1973, pp. 197–198):

1. If differences in mental abilities are inherited, and
2. If success requires those abilities, and
3. If earnings and prestige depend on success,
4. Then social standing (which reflects earning and prestige) will be based to some extent on inherited differences among people.

Mobility effects—"success," in Herrnstein's syllogism—can be seen in the fate of children who move up and down the occupational ladder, relative to the status of their families of origin. In each generation in industrialized societies, about 30% of children move upward in social class (relative to their class of origin), about 30% move downward, and the remainder stay in place. In light of the data summarized here and in previous chapters, each statement in the syllogism is noncontroversial. In presenting them, however, Herrnstein offered no evidence for the influence of heritable traits on social mobility, other than the general correlation of IQ and social status.

A more direct demonstration of the influence of heritable IQ variation on social mobility comes from *within*-family comparisons. In the quiet suburbs of an English city, the IQs of upwardly mobile sons averaged 7 points higher than those of their fathers, whereas those of downwardly mobile sons averaged 8 points lower (Mascie-Taylor & Gibson, 1978). In Minnesota, the IQs of downwardly mobile sons were consistently lower than those of their fathers, and those of the upwardly mobile sons, were consistently higher. Moreover, as shown in Figure 5.2, the proportion of sons rising or falling in social class increased systematically with the departure of their IQs from their fathers' (Waller, 1971). About 40% of those sons with IQs 15 points below their fathers' fell in social standing, whereas an equal number of those with IQs 15 points above their fathers' rose in social standing. The more discrepant a son's IQ from his father's, the more likely the son was to fall or rise in social standing. Herrnstein's (1973) syllogism has thus received empirical support in studies conducted in England and the United States. Although one might prefer larger and more representative studies than these two, I think it unlikely that a massive National Institute of Health study would discover that children duller than their parents tend to rise in social class, or that the brighter ones tend to fall.

In summary, social mobility explains why genes in a professional-class person differ, on average, from those in a working-class person. If people were randomly allocated to social class levels, then no systematic genetic differences would exist among them. However, this is not the case: It is by dint of individual effort, and by the presence or absence of favorable traits, that people with different genotypes become reassorted into different social classes. The situation is like that of a species of mollusks that find different depths in a tidal plain. Genotypic variants able to survive better in deeper, cooler waters become more common there, whereas in the shallower, warmer waters, other genotypes

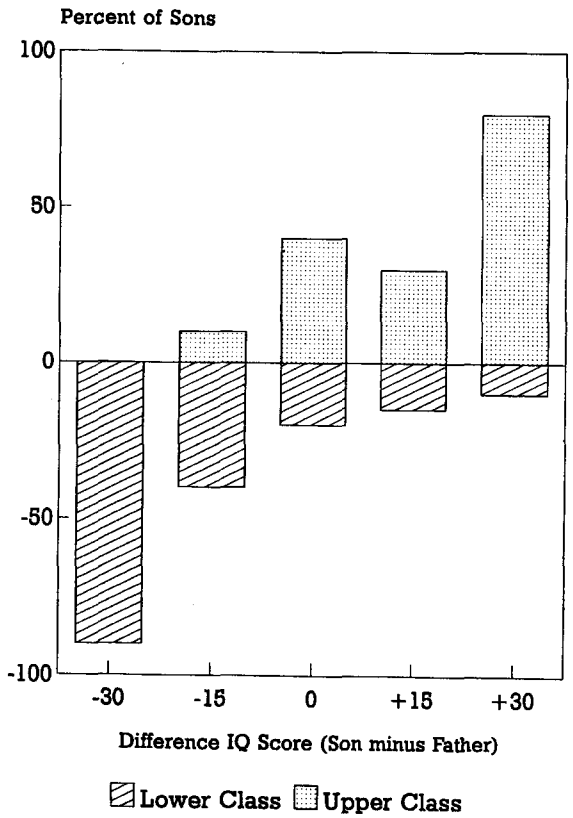

FIGURE 5.2. Social class mobility based on sons' IQs. Adapted from Waller (1971). Copyright 1971 by the Society for the Study of Social Biology. Adapted by permission.

are favored. Through this selective action, genetic variation may arise between mollusks in deep and shallow waters. That is, the existence of environmental "niches," each more compatible with one particular genotype than with another, supports gene variation at particular loci because one genotype (AA) thrives in one niche, whereas another (aa) thrives in a different niche (Kari & Avise, 1992).

In each generation, countervailing forces try to eliminate the genetic basis of social class. One force is the reassortment of genes occurring within marriages. Although a college student is more likely to marry another college student than to marry a laborer, mate choice is far from perfect for traits such as IQ (with a strong influence on social success),

and imperfect for other traits as well. The husband–wife correlation on IQ is only about .35 (Johnson, Ahern, & Cole, 1980). Children, receiving genes from the parent with the genotype more favorable to mobility as well as from the other parent, usually enter social competition with a more average genotype than that of the better parent's. For a typical family at the upward social class extreme, downward social mobility (toward the population mean) is to be expected, because less favorable genes tend to be received from the more average parent. For a typical family at the social bottom, upward social mobility (toward the population mean) is to be expected, because more favorable genes tend to be received from the more average parent.

This reassortment, however, fails to imply a continuing progression toward the average. The loss of very favorable and very unfavorable genotypes from extreme families is compensated for in their recreation in the children of average parents. The real world is unlike Garrison Keillor's Lake Wobegon, where all the women are strong, all the men are good-looking, and all the children are above average. Truly average people—neither grossly underachieving nor outstanding, and existing in great numbers—are the reservoir from which most very good and poor genotypes are randomly reformed through genetic recombination.

Another force also encourages social mobility: environmental regression toward the mean. The lucky and unlucky events that propel parents to high or low social statuses are not recreated in a child. So neither lucky events nor the exact genotypes that produced parental fates are likely to be found in a family's offspring, who must then seek their own fortunes. If husband–wife matching for status-relevant traits were more nearly perfect, of course, the pressure on social mobility to recreate a genetically based social status hierarchy in each generation would be eased. As Romeo and Juliet illustrate, however, love often ignores social convention, and mating systems in Western democracies are not strong engines of social status.

The driving power of effort and merit can be overemphasized, of course. The unshared environmental influences—already acknowledged in the elements of fortune and luck—account for about half the variation in income and one-quarter of the variation in years of education. Moreover, macroeconomic events can devastate the economy. Some years ago, a chief engineer in the aerospace industry was heralded for helping to land men on the moon. His achievements brought him little comfort when, shortly thereafter, an economic recession in the aerospace industry cost him his job; even highly praised engineers are sent pink slips. But in social class variation within the economy at a particular time, a major part of its origin is in the genes.

Social Class and Behavioral Outcomes

The Confusion of Cause and Effect

Even if the variation in an environmental measure is genetic, it is still possible for its association with behavioral outcomes to be environmentally mediated, but it remains unlikely. For the most part, we can expect that the statistical association between social class and any one of these outcomes will be genetically based—a result of shared genes that in a parent may affect income or years of education, and that in a child may affect a particular trait. An edition of the *World Book Encyclopedia* does not appear on a family's book shelf by magic; the parents must want to buy it. Who will so decide? Parents who are bright and intellectually curious, and who wish their children to excel at school. The same genes affecting these parental traits can influence children's IQs. Thus a genetic confound in an environmental measure is not difficult to spot; unfortunately, however, this concept eludes the grasp of many social scientists, who insist on reading causation into the statistical association of social class with children's behaviors.

Abundant data on children's IQs demonstrate this confusion of cause and effect. In biological families, the association between a measure of parental social class (say, income or years of education) or one of rearing environment (say, books in the home) can capitalize on genes shared by parent and child. In adoptive families, by contrast, the opportunity to capitalize on this association has been removed (although selective placement, as noted in Chapter 2, can induce an artificial association between genotypes of the adoptee and adoptive parent). The extent of genetic influence in the association is contained in the difference of the biological parent–child correlation (heredity + rearing) and the adoptive parent–adoptee correlation (rearing alone).

What does the research say? In Leahy's (1935) adoption study, economic class correlated .37 with children's IQs in biological families ($p < .05$), whereas its correlation in adoptive families was only .12. In another early adoption study, family quality correlated about twice as strongly with children's IQs in biological as in adoptive families (about .40 vs. .20, respectively; Burks, 1928). In the Colorado Adoption Project

(Plomin, Loehlin, & DeFries, 1985), the general score on the Home Observation for Measurement of the Environment (HOME; Caldwell & Bradley, 1984)—a widely used assessment of the intellectual qualities of the rearing environment—correlated .44 with infants' IQs in biological families, but only .29 in adoptive families. In one of the Minnesota adoption studies, a combination of mother's education and father's occupation and income correlated .33 with children's IQs in biological families, but only .14 in adoptive families (Scarr & Weinberg, 1978). This last result was published in the prestigious *American Sociological Review*, where it stimulated one round of debate in the same journal in 1980, but has since been mainly ignored by a field unwilling to deal with scientific anomalies. Other studies could be added here, but the trend is already clear: *Environmental* social status variation has been greatly overstated, and *genetic* social class variation has been greatly understated, whenever socialization science has presented data from biological families.

A Case Example: Asian Refugees

Academic achievements of the children of newly arrived Asian refugees may be used to illustrate these principles. Despite coming from illiterate and poor backgrounds in their homelands and arriving in America economically destitute, the refugees' children have performed outstandingly in the inner-city schools of five urban areas—in precisely those schools in poor neighborhoods that are thought to be unable to educate our youth (Caplan, Choy, & Whitmore, 1992). In a randomly chosen sample of 536 school-age refugee Asian children, *one-third* scored above the 90th percentile in mathematics on a standardized test. Their overall test average was at the 54th percentile, but their performance was handicapped on the more language-intensive subtests (the children's parents were non-English-speaking, and English was for many children a second language).

The University of Michigan research team responsible for the study reflexively turned to the families, and to rearing, for an explanation of the ability of these children to thrive academically in an environment thought to be implacably hostile to intellectual pursuits. They identified three possible rearing influences responsible for the "pivotal role of the family in the children's academic success" (p. 36). The first was Asian values, which encouraged a family-based orientation toward achievement, hard work, perseverance, and pride. The second was the tremendous effort devoted to school work: Whole families would gather around the dining room table to spend 3 hours per night on school work (about twice the hours put into school work by the average American child). Third, the siblings taught one another, so that families with more children actually had enhanced levels of academic achievement.

Throughout this book, we have seen a failure of rearing experiences to account for children's traits, and yet this would appear to be a dramatic case of academic achievement, accompanied by patterns of parenting sharply different from those in most poor urban families. Are we now to believe that rearing matters? Are we to accept that the causal influences on these Asian children's achievement were sibling tutors and Asian family values? No, at least not in the sense implied by the Michigan research team. The Michigan researchers imagined that good rearing is like an experimental treatment that can be applied to anyone with equal success: Put other children into an Asian family, with its values and emphasis on achievement, and their test scores should bloom as well. In an experimental treatment, plants given more fertilizer grow larger and produce a more abundant crop than plants given less: Double the fertilizer given to the crop, and its agricultural yield should double. Such experimental results are true when a manipulated variable has produced an outcome, but they are false and profoundly misleading when little *causal* determination lies within rearing environment.

Previous chapters have shown that traits emerge through a process of gene × environment correlation—through Dawkins's (1982) "extended phenotype." In this view, the supportive environments of Asian families *and* a set of genetically based traits lead together to high levels of academic achievement. The Asian children's long attention span, greater self-control, and large working memory capacity constitute a recipe for academic success through self-directed study. Were these traits lacking, the long hours around a dining room table would erupt into family arguments, with jumpy children anxious to break away from the unpleasant duty imposed on them. Even if more average children, lacking the persistence of these Asian boys and girls, could be handcuffed and chained to their books, would not the lesser absorption of academic material make learning less satisfying? In a nutshell, an alternative hypothesis is that these Asian children were indeed different in (genetically based) temperamental and intellectual traits from the other inner-city children with whom they were compared.

Studies of transracially adopted Asian children diminish the argument for the *necessity* of Asian values and family life. One study followed

12 children from Vietnam, 8 from Korea, 3 from Cambodia, and 2 from Thailand, all adopted into American homes prior to 36 months of age (Clark & Hanisee, 1982). About half the babies required hospitalization for malnutrition in the United States prior to their adoptive placements. There was little screening of the babies as adoptable or unadoptable at the time of their placements, and most had had checkered histories in orphanages, foster homes, hospitals, or combinations of these. Like the larger sample of Asian children reared by their natural parents, these adoptees, even as infants, excelled in showing academic ability. Their mean score on the Peabody Picture Vocabulary Test was an IQ of 120, as opposed to national norms of 100. They also excelled on a test of social competence. Two studies of transracially adopted Korean children replicate these results (Frydman & Lynn, 1989; Winick, Meyer, & Harris, 1975). Provided with an environment generally supportive of intellectual work, Asian children seem to find their own ways to thrive.

Indeed, soft-pedaled in the Michigan report was a mention of the Asian children's own satisfaction with studying: "... the children experienced intrinsic gratification when they correctly worked a problem through to completion. The pleasure of intellectual growth, based on new knowledge and ideas and combined with increased competence and mastery, was considered highly satisfying" (Caplan et al., 1992, p. 40). Although the Michigan team attributed this intrinsic response to the children's cultural identity, I am convinced that it would be more correctly attributed to their genes and to their reactive and active responses to this genetic endowment. Such gene × environment correlation meant that "no damaging manipulation of their [the children's] lives" (p. 41) was made by their parents and that a "love of learning sustained their academic pursuits" (p. 41). For other children, an educational intervention similar to long, unbroken study periods and difficult mathematical materials, as practiced by these Asian children, would fail miserably. By analogy, putting down a second bag of fertilizer would fail to transform a cherry tomato plant into a beefsteak tomato plant.[3]

The Genetics of Child-Rearing Styles

Acceptance of genetic variation in social class measures is just the peak of a large iceberg, because the same logic applies compellingly to other measures labeled in socialization science as "environmental" (Plomin & Bergeman, 1991; Scarr, 1992).

For decades, socialization science has sought connections between variation in child rearing and behavioral outcomes. Responses to questionnaires on rearing style can be factored mathematically into two broad dimensions: "parental warmth" and "parental control." The former dimension refers to the degree to which parents show their children concern and love; the latter refers to the degree to which they impose on them rules and restrictions. As noted in Chapter 1, the ideal rearing style has been described as a combination of high parental warmth and appropriate parental control (i.e., a degree of control that is tailored to children's maturity and skills)—a rearing style called "authoritative." On the other hand, like social class attainment, rearing styles are no more than parental behaviors; as such, they can be regarded as "phenotypes" of the parents as well as "environments" for children. And as relatively stable parental traits, the rearing practices by which parents raise children can be themselves analyzed for genetic variation.

I first attempted this kind of analysis with adolescent twins' and siblings' self-reports of what kind of parenting they had received (Rowe, 1981, 1983). Perceptions of whether one is loved or controlled, of course, are filtered through each individual's psychology, and so may not exactly match the parents' rearing style as seen by outside observers; on average, one family member's report of rearing explains about 10% of the variation in another's report. Children's reports are one source of information about rearing whose importance cannot be ignored, however, because these perceptions are associated with such developmental outcomes as self-esteem and delinquency.

I found that rearing styles were not innocent of genetic variation. On two different measures of rearing, identical twins reported more similar perceptions of parental love than either DZ twins or nontwin siblings. The traditional twin analysis—greater MZ twin than sibling resemblance—suggests that perceived love is heritable. The results for control were different. As shown in Table 5.2, DZ twins were about as similar as MZ twins in perceptions of parental control (correlations averaging about .45). MZ twins may see more similarity in affection than DZ twins, because their greater behavioral similarity may tend to elicit similar parental treatments. If for reasons of fairness, parents place similar restrictions on both twins (who are, after all, the same age), then the DZ and MZ twins may experience a similar degree of parental control, and this latter dimension of rearing cannot be regarded as heritable when assessed through the eyes of adolescent children.

Another approach lets parents tell us about their rearing styles. In

TABLE 5.2. Twin Correlations for Ratings of Parental Control and Affection

Measures	Twin correlation MZ	DZ
Control scales		
Control–autonomy of mother[a]	.44	.47
Control–automony of father[a]	.43	.46
Firm–lax control of mother[a]	.55	.46
Firm–lax control of father[a]	.43	.45
Restrictiveness–permissiveness[b]	.44	.45
Warmth/love scales		
Acceptance–rejection of mother[a]	.54	.17
Acceptance–rejection of father[a]	.74	.21
Accepance–rejection[b]	.63	.21

[a]The data are from Rowe (1981) for a sample of 89 twin pairs.
[b]The data are from Rowe (1983) for a sample of 90 twin pairs.

this research design, the subjects are now *adult* twins or siblings who are reporting on how they treat their own children. It may be that adult twins hold in mind an image of how their parents have treated them as children, and that this recollection guides and shapes their rearing practices. If so, we should find some shared childhood rearing influence on adult child rearing, and also little genetic influence. Alternatively, the lessons of the twins' own childhoods may have been long-forgotten casualties of time and maturation, so that rearing may reflect more heritable dispositions.

Although evidence comes from only a few studies, the findings indicate that rearing is like any other behavior—genetically influenced. In Sweden, researchers from the University of Pennsylvania and their Swedish collaborators identified twins who had been raised apart, mainly in the 1920s and 1930s, because of poor economic conditions and epidemic diseases in Sweden (Plomin, McClearn, Pedersen, Nesselroade, & Bergeman, 1989). The separated twins had been placed with different families at an average age of 2.8 years, and about half (48%) had been separated at less than 1 year of age. The separated twins were compared to unseparated twins born during the same historical period. When surveyed by mailed questionnaires, the twins who were now in their 50s and 60s, completed the Family Environment Scale (FES), a widely used measure of child rearing in the family.

Table 5.3 presents the *mean* rearing correlation for the adult twins over the eight scales comprising the FES. The correlations were low, but then the twins were raising different children (who possessed different traits) and had different spouses. One impressive result was that the families of origin lacked an influence on rearing practices, because twins raised together or raised apart in separate adoptive families were equally alike in their rearing styles. Too, with a mean correlation of only .09, the rearing practices of DZ twins were only slightly more alike than those that would be found for randomly paired adults. Genetic influences were confirmed, with the adult MZ twins who had been raised apart proving to be alike in their child-rearing practices. From the MZ twins' correlation, the estimated heritability of child rearing would be .245; from the DZ twins', it would be .18 (i.e., twice .09). In a more complex model-fitting analysis, the twin study team arrived at the following average estimates: heritability, .26; childhood rearing environment, .03; and nonshared environment, .72. Although the degree of genetic influence varied from one scale to another, seven of the eight FES scales showed statistically significant genetic influence. For no scale was the influence of childhood rearing environment statistically significant by a chi-square test. As was not the case for adolescent twins' perceptions of rearing, genetic influence was statistically significant both for control dimensions of rearing (FES organization and control) and for warmth dimensions of rearing (e.g., FES conflict and expressiveness).

In unpublished work, I surveyed a fifth kinship group: 20 pairs of unrelated children reared together. The unrelated children were usually both adopted into the same adoptive family; their average age at placement was under 2 years. Now adults with children 9 years of age

TABLE 5.3. Mean Child-Rearing Correlations for Adult Twins with Families

Group	Rearing *r*	No. of pairs
Adult MZ twins reared apart	.21	40–50
Adult MZ twins reared together	.28	82–90
Adult DZ twins reared apart	.10	120–129
Adult DZ twins reared together	.09	104–115

Note. Mean correlations averaged over eight subscales in the Family Environment Scales (measuring cohesion, expressiveness, conflict, achievement, culture, activity, organization, and control). The data are from Plomin, McClearn, Pedersen, Nesselroade, & Bergeman (1989).

and under, this group provides another check on the influence of family background on adult rearing correlations. Their child-rearing correlations for both warmth (r = .00) and control (r = .18) were statistically nonsignificant. Of course, perhaps some effect of childhood environment would emerge in a larger sample of unrelated siblings. But I expect that a larger sample would confirm this discovery—that no two children learn the same things about parenting from their own parents.

Table 5.4 presents correlations of the adults' rearing styles with their self-reports of personality on the Big Five personality traits, discussed in Chapter 3. In particular, the warmth dimension has personality correlates in the domains of extraversion and intellectual openness. The control dimension has such correlates as emotional stability and agreeableness. Of course, in standard personality inventories, some sources of heritable variation in child-rearing styles may be missed. One interesting avenue, which can be pursued in new research, is whether rearing practices contain unique genetic variation, separable from standard traits. The emotional depth we feel in our relationship with our own children suggests that here is a special domain for revealing the inner qualities of human character.

In the association of children's IQ scores and their families' social class, we have already seen causal confounds. But few studies have shown them directly in the more emotional domains of family life. One study to make this demonstration nicely is the Colorado Adoption Project. In the Colorado Adoption Project, as noted in earlier chapters, adoptive families were compared with matched, nonadoptive families. In the former families, the lack of association of parental and child genes meant that heredity would be unable to mediate associations between measures

TABLE 5.4. Correlations between the Parenting Composites and the "Big Five" Self-Descriptions of Personality

Dimensions	Warmth	Control
Extraversion	.35*	.07
Agreeableness	.38*	−.28*
Conscientiousness	.29*	−.15*
Emotional stability	.28*	−.35*
Intellectual openness	.45*	−.14

Note. n = 186. Control dimension includes strictness and negative emotions.
*$p < .05$.

of family environment and children's outcomes. And, as expected, greater correlations between rearing and child outcomes were found in the biological than in the adoptive families. For infants' behavioral problems, the mean environment–behavior correlation was .07 in adoptive families and .23 in biological families; for infants' temperament, the mean correlations were .06 and .20, respectively (Plomin et al., 1985). Primarily, what seems to be a causal association is just the happenstance of similar genes shared by biological parent and child.

The Genetics of Other Environmental Variables

Parental Divorce

Genetic self-selection processes may extend to many other "environmental" variables favored in socialization science. Parental divorce is one important example. Although divorced children suffer worse outcomes for some behavioral traits than children of nondivorced parents, the causality is ambiguous. Divorced and nondivorced parents are not random samples of a population, assigned by some impartial decision maker to two different social statuses; rather, they are people who have elected either to dissolve their marriages or to remain married. Simply put, people who divorce may be different from people who do not.

And once again, an "environmental" variable can reveal genetic variation secreted within its categories. In Minnesota, the divorce status of 1,516 same-sex twin pairs, their parents, and their spouses' parents was studied through a mail survey (McGue & Lykken, 1991). Twin samples in Minnesota are noteworthy for their representativeness of white Americans from northern Europe as such, they are the closest American approximations to the nationally complete data banks of the Scandinavian countries. From the twin and parent–child data, the heritability of divorce can be estimated. The correlations were as follows: for MZ twins, .55; for DZ twins, .16; and for parents and offspring, .17 for the twins and .27 for their spouses. The heritability of divorce was then estimated as 52%. McGue and Lykken found no evidence for a rearing influence on the risk of divorce: The likelihood of divorce did not come from the social example of divorced parents, spouses' parents, or cotwins, according to their mathematical model.

In the case of divorce, the degree of risk can arise from genetic influences brought into the relationship from either the spouses' or

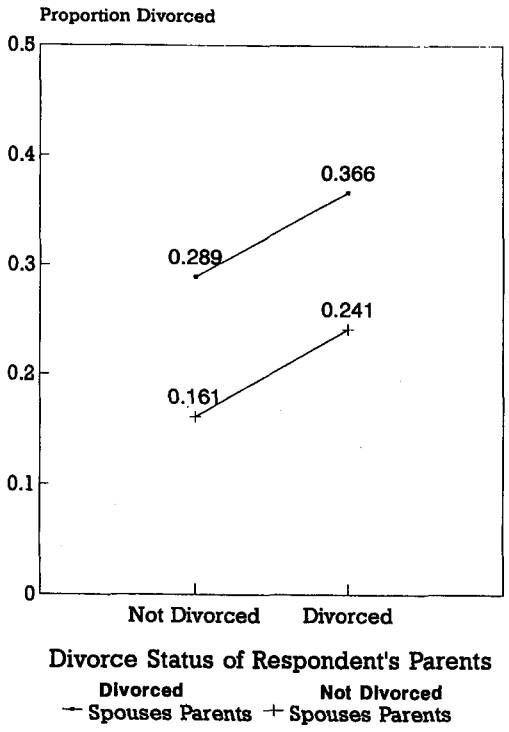

FIGURE 5.3. Parental divorce and the risk of divorce. Adapted from McGue & Lykken (1991). Copyright 1991 by the American Psychological Society. Adapted by permission.

respondents' biological families. As shown in Figure 5.3, divorce risk in this sample increased additively with divorce in (1) neither family of origin of the couple; (2) divorce on the spouse's side alone; (3) divorce on the respondent's side alone; or (4) divorce on both sides. Odds increased even more extremely with information on an MZ twin. In the news, we sometimes read about an MZ twin man taking an MZ twin woman's hand in marriage. The risk for a hypothetical marriage of an MZ twin man to an MZ twin woman with no family history of divorce (5.3%) was 15 times smaller than that for the same type of marriage in which the married MZ twins' biological parents, and both their cotwins, were divorced (77.5%). Given these circumstances, a minister might do well to stop the marriage!

Adolescent Peer Groups

The choice of friends is also a selective process: The Republican avoids the Democrat; the drinker, the teetotaler; the daredevil, the sissy; and the selfish, the altruistic. In light of this self-selection process, friends should be more genetically alike than randomly paired individuals (Rushton, 1988), and peer group choice should show genetic variation.

The Sibling Inventory of Differential Experience (SIDE) nicely demonstrates the component of genetic variation in adolescents' peer group choices (Baker & Daniels, 1990). The inventory requests from a sibling respondent a relative judgment of peer group popularity, achievement, and delinquency. That is, the respondent indicates whether his or her peer group has more of the characteristic than that of his or her sibling, and the sibling makes the same judgment in reverse. Absolute differences on these scales are scored as follows: A score of 2 means that the siblings' peer groups are very different; a score of 1, that they are somewhat different; and a score of 0, that they are exactly alike.

Figure 5.4 presents the absolute differences on the SIDE for kinship groups that differed in genetic and social relatedness. As shown, the family members' adolescent peer groups became increasingly dissimilar in their achievement, delinquency, and popularity as the siblings became less genetically alike—from MZ twins, to DZ twins and nontwin siblings, to adoptees. A simple interpretation can be offered: Siblings select peers partly on the basis of matching personality traits and social interests. Because both traits and interests are heritable, the more genetically dissimilar siblings select different peer groups, which then reinforce their trait dispositions. In Scarr and McCartney's (1983, p. 433) phrase, "genes direct the course of human experience" to the point that the character of people and the environments they choose to inhabit are inseparable.

Differential Treatment within Families

As discussed several times in this book, the evidence against shared environmental effects does not apply to parents' differential treatments of children. Instead, an explanation in terms of shared environments applies to parent–child resemblance. If parents resemble children in psychopathology, personality, or intellect, then it is natural to attribute this

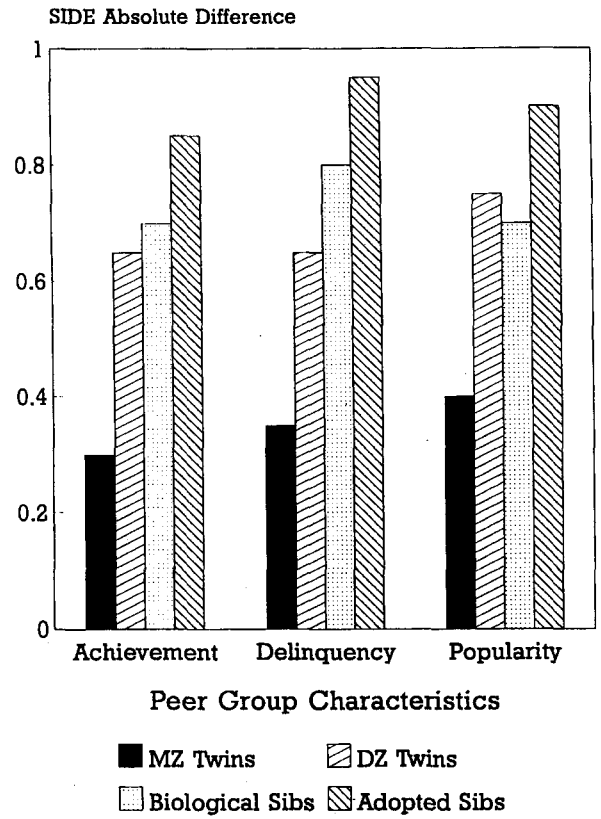

FIGURE 5.4. Peer group dissimilarity and biological relatedness. Adapted from Baker & Daniels (1990). Copyright 1990 by the American Psychological Association. Adapted by permission.

resemblance to the causal influence of experiences shared by parents, or in the case of sibling resemblance, the siblings' common experiences. But this book's thesis has been that the correct causal attribution for these resemblances is genetic inheritance, not shared family environment. Nonetheless, behavior genetic models also estimate another environmental term—the nonshared environment that differs among siblings (or between parent and child) and operates to make them different in their behavioral traits. Could parenting practices contribute to family members' behavioral dissimilarities? Are parenting influences more important than low estimates of shared family effects would suggest?

For example, variations in emotional climate among families (with the exception of severe abuse or neglect) are discounted as causal influences by this book's accumulated evidence. But what about variations in the microemotional climate within families as seen from the vantage point of each child? Children may be exquisitely sensitive to unevenness of parental treatments, as when one sibling is favored over another.

In their book *Separate Lives* (1990), the husband-and-wife team of Dunn and Plomin cite the enormous personality differences between two brothers: the gifted novelist Henry James, and the famous psychologist William James. Henry was aloof, quiet, and unsociable, but he was his mother's favorite child. William had an easy gregariousness; he was energetic and vibrant. Although William's traits would seem more likely to be a result of maternal favoritism than Henry's, sometimes true causal influences work in ways different from what common sense would dictate.

Given the possibility of subtle within-family parenting effects, behavior geneticists have called for increased research on the differential treatments of siblings (Rowe & Plomin, 1981; Plomin & Daniels, 1987). A behavior geneticist writing with two environmentally oriented social scientists commented:

> Although differences between siblings in normal and pathological outcomes are beginning to be delineated, we know far less about environmental differences between them. The genetic data suggest that only environmental variables that are significantly different between siblings are likely to be important in developmental differences. (Reiss, Plomin, & Hetherington, 1991, p. 285)

Of course, "environmental differences between siblings" include far more than unequal parental treatments. Isolating what particular nonshared environmental influences are responsible for an observed behavioral difference is a daunting task, all the more so because such influences can be almost anything under the sun. Nonshared environmental influences range from intrauterine environments to attending different colleges. To show that parental treatments are effective nonshared environmental influences, several tests must be passed: (1) The differential treatment must be associated with sibling differences in normal or pathological traits; and (2) the direction of causality must be from parental treatments to the observed behavioral difference between the siblings, rather than vice versa. Genetic influences may reverse the directionality of effects in the second pathway, if nonshared genetic

effects produce different phenotypes in siblings, which are then reacted to differently by parents.

I agree with encouraging work aimed at identifying various nonshared environmental influences, including parental favoritism toward one child versus another. Without a large existing body of evidence, any conclusion about the strength of specific nonshared influences must be regarded as tentative. Nonetheless, I am doubtful that new discoveries about nonshared parental treatments will upset this book's thesis that family influences on children's developmental outcomes are limited.

One reason for caution is a distinction between components of variance and developmental processes. As discussed in Chapter 2, components of variance are the results of calculations made for estimating and testing models of environmental and genetic inheritance. One way to understand these components is to say that variance is apportioned to differences among family means (i.e., the shared or between-family component) and to siblings' differences from their respective family means (i.e., the nonshared or within-family component). Let us suppose that in the Smith family John scores 90 on an IQ test and his sister Mary scores 100. The family mean (the average of John's and Mary's IQs) is 95. This mean represents the "shared" component of IQ—an IQ level common to the siblings. The nonshared component of IQ is the 10-point difference between them—John is 5 IQ points below the family mean; Mary is 5 IQ points above it. Thus mathematically, a shared component (the siblings' overall mean of 95) can be distinguished from a nonshared component (the siblings' 10-point IQ difference). As noted in Chapter 2, a larger shared component would mean greater family-tied genetic or environmental effects.

Most developmental processes, however, do not map exactly onto this convenient mathematical distinction. Development proceeds, regardless of how variation in trait scores is later apportioned. Suppose, for example, that the book's thesis had proved wrong—that the majority of IQ variation was correctly attributed to intellectual stimulation in families. Over her childhood, Mary's exposure to intellectual stimulation would lead to her IQ level—an IQ of 100. Furthermore, Mary must have received somewhat more intellectual stimulation than her brother John; hence her higher IQ score. If intellectual stimulation by Mary's parents has been responsible for the development of Mary's IQ, then its effects will necessarily appear both in the shared component (the family mean IQ) and in the nonshared component (the siblings' IQ difference). "Intellectual stimulation" does not recognize this distinction of "shared" versus "nonshared" variation. This distinction arises only after development has occurred, when a researcher decides to use statistical procedures by which IQ variation is apportioned to that which is shared by siblings and to that which makes them different.

A special developmental process that makes siblings different could conceivably have a mainly nonshared influence. Let us suppose that Mary is the first-born child and John is the second-born. Let us imagine further that parents typically put most of their intellectual stimulation into a first-born child and less into a second-born child. To complete the picture, all parents use *exactly* the same amount of intellectual stimulation (there are no differences among families), but the first-born child always receives twice the intellectual stimulation of the second-born child. Under these restrictive conditions, the environmental influence would operate entirely within families—as a nonshared influence making siblings different. I do not see why we should expect to find developmental processes with such restrictive effects. In general, developmental processes are seamless; they are not isolated from other processes just to make siblings different from one another.

Consider, for example, that a large scientific literature on birth order has not produced an explanation for sibling differences in behavior. Variation in IQ, in particular, has been attributed in both textbooks and in the popular press to birth order differences. I think that under the scrutiny of carefully collected data, most birth order theories of IQ variation simply collapse (Schooler, 1972). True, only children have higher mean IQs than last-born children in large American families—but this effect may be easily explained as one of selection, because parents with high IQs also often restrict their family sizes more (since the baby-boom generation) than parents with low IQs. When birth orders are compared within families, mean IQ differences are rarely found, and children of different birth orders are equally *similar* to one another in their IQs (Rodgers, 1984; Rodgers & Rowe, 1985).

As in the example above of Henry and William James, another hypothesis of purely nonshared effects is parental favoritism. If this effect arises as one sibling senses how much he or she is favored relative to another sibling, then favoritism would truly create differences mainly between siblings, without any reference to a family's overall level relative to a population. In other words, both siblings may be terribly mistreated (compared to how children in general are treated), but a better-treated sibling may still feel well psychologically, if he or she has received the more favorable treatment of two siblings.

Although this argument is logically consistent, I am again doubtful—merely because children make social comparisons not only to their own brothers or sisters, but also to children in other families, and (through media portrayals) even to children in other social classes and countries. Eventually, I believe that two children with little parental affection or interest would come to know their status relative to other children, making parental treatment a shared influence (relative to the general population) instead of a purely nonshared influence (relative to a sibling).

Despite these cautions, it is certainly possible that "nonshared" parenting effects exist, and they may be stronger than I have anticipated. The best way to assess the strength of nonshared parental treatments is through empirical studies designed for this purpose.

In a study of 5- to 11-year-old children in the National Longitudinal Study of Youth (NLSY), my colleagues and I evaluated nonshared environmental influences on childhood problem behaviors (Rodgers, Rowe, & Li, in press). The NLSY data source is a representative sample of American families stratified to contain more poor families than there are in the general population. To vary genetic resemblance, four types of related child pairs were identified from over 7,000 children in the NLSY: twin pairs, full-sibling pairs, half-sibling pairs, and cousins. The statistical analyses used a regression equation technique to control statistically for both genetic and environmental *shared* influences. With this technique, the nonshared effect of specific differences in parental treatments could be estimated. The twin, sibling, half-sibling, or cousin who was spanked more, read to less, and had a poorer quality of home environment (as rated on the HOME; Caldwell & Bradley, 1984)—*relative to the child with whom he or she was being compared*—tended to have a greater number of problem behaviors. For some variables, the within-family treatment differences seemed to be attributable to nonshared genetic influences; in other cases, they seemed to be "pure" nonshared environmental effects.

However, one caveat is in order. The full regression equations accounted for some 10–24% of variance in problem behavior. The nonshared variable contributed 1% or even less to the variance explained. Given our large sample sizes, this was often a highly reliable addition statistically; however, in absolute terms, only a small part of the total variance in problem behavior was explained. If measurement error and nonshared genetic influences are excluded, other *nonparental*, nonshared environmental influences must explain the remainder of nonshared differences in problem behavior. If other research programs yield results like ours, this book's thesis that family environments—and child-rearing styles in particular—are limited in the extent of their influence on developmental outcomes will not require modification in light of nonshared parental treatments.

Finding the Thresholds

In this book, I have argued that variation in most rearing experiences does not matter for most developmental outcomes. Yet, as readers have surely noticed, some adoption and twin studies fail to include great numbers of the most seriously disadvantaged children. For instance, most parents in the Texas Adoption Project (described in earlier chapters) had at least a high school education. Such a range covers about 60–80% of the current American population, depending on ethnic group and geographic location, because today a majority of teenagers complete at least 12 years of education. Twin studies are typically more representative than adoption ones, but even in twin studies, parents who physically abuse or who severely neglect their children are rare. Because genetic variation in environmental measures does not preclude their *environmental* effects on children, one should not extrapolate my conclusions about rearing influences to environmental extremes, any more than one should be assured that equipment able to work in Maine's winters can be entrusted with human lives and safety in Antarctica.

Of great concern to Americans are the very poor—the lowest 10% of the population in terms of income and education levels, the families on welfare, the families who are disproportionately black and living in America's inner cities. It is in this group that the rates of child abuse and neglect are greatest, as are the rates of births to teenagers and to single women of all ages. Moreover, among the urban poor, a wave of violence among teenagers has made homicide a leading cause of death. Are we to think that rearing variation makes no difference here?

Unquestionably, these children's lives would be improved if they were not placed in the physical danger and psychological stress of neglectful or abusing parents and neighborhood violence. At some intensity, poor rearing must affect children's development—leaving emotional and physical scars, and actually leaving some children dead. But whether poor rearing is generally the culprit behind the problems of the nation's most disadvantaged children is unclear.

In rearing explanations, one weakness is that the psychological processes invoked to explain extreme outcomes are often identical to those used to explain more normal ones. But why should psychological processes hold only in the extremes? If mothers who hate their children damage them, shouldn't mothers who are just cold create more minor harm? If physical abuse leaves scars, shouldn't overly strict rearing also misdirect personality development?

Thus, we must explain unexpected rearing effects as threshold ones: Previously impotent factors may acquire power once they reach a high level of intensity, but not before. Threshold principles hold in many domains. In the mathematics of catastrophe theory, equations are well behaved up to a certain point, when suddenly a smooth trend breaks apart and predicted values fall sharply, like a stream thundering into a waterfall. A metal bar under stress may fail all at once, snapping in two, rather than bending slowly. In our diets, trace amounts of many vitamins are required for health; let their quantities fall below a threshold, and physical illness and death may result. A person's resentments and hatreds may be held within for years before they explode as murderous revenge. Rearing may have such threshold properties: At some intensity of abuse, neglect, poverty, and poor nutrition, the feelings that bind most people to others may fail to develop, and so unsocialized children become threats to others as well as to themselves.

But threshold principles may be insufficient. To explain the plight of the most disadvantaged Americans, we may need to admit genetic influences into our explanatory framework. After all, if genes account for half the variation in income over a wide range of American families, is it realistic to believe that only the poorest ones lack those genetic disadvantages affecting lower class individuals in general?

With the evidence reviewed above of genetic variation in "environmental" measures, we must, at least be cautious about expecting family environmental change to offer panaceas for children's behavior problems. Consider, for instance, average developmental outcomes for children adopted as infants. Although most adoptive children fare very well, their behavioral outcomes have been, on average, worse than those of nonadoptive comparison children.[4] In childhood, adoptees are statistically overrepresented in clinic referrals for externalizing behavior disorders. Adoptees' rates of delinquency are also higher than those of comparison nonadoptees. Finally, adoptive children have no less serious psychopathology than other children. Worse outcomes, despite materially and socially advantaged upbringings, bode ill for the idea that even

a massive redistribution of economic wealth would produce problem-free children or crime-free communities. Although social conditions would be improved for these children raised in relatively economically advantaged situations, their behavior problems would persist.

A nagging fear of a hereditary basis to racial and social class differences is but one reason why socialization science has gone astray, and perhaps not the main one. In seeking an understanding of behavioral traits, we look too closely to ourselves, to the history of just a single ontogeny. By analogy, one might seek the source of the Nile at the Aswan Dam, forgetting entirely the more than 3,500 miles of river further upstream, reaching into the African continent into what the colonial explorers named the Mountains of the Moon and Lake Victoria. The fallacy is in believing that what forms human nature is a 14-year period of rearing, rather than a heavier weight of cultural history, and ultimately human evolutionary roots. In broader terms, cultural traditions can be passed in many ways other than exposure to idealized nuclear families. The adolescents who signed up enthusiastically for Nazi youth groups before World War II did not have souls bent and torn by poor rearing in early childhood; indeed, their families were stolidly middle-class and emotionally supportive. If a nation's youth can be changed by a few years of great cultural change, why emphasize childhood? And more deeply still, what genes has nature selected for us? In the next chapter, I apply ideas from evolutionary biology to behavioral sex differences. In the last chapter, these themes reappear as I discuss alternative routes by which traits may move from one generation to the next.

Notes

[1]Some scholars may ask, "Why look for genetic bases of racial differences at all?" As in other applications presented in this book, the best answer is that genetic influences must be considered if we are to estimate environmental ones accurately. Clearly, studies of racial differences must be carried out with great sensitivity to their potential for social harm (Loehlin, 1992). If a result supports a genetic basis of racial differences, care should be taken neither to exaggerate its strength nor to overgeneralize it to other traits where it may not apply. The issue of genetic differences in racially linked behavioral traits is further discussed by Loehlin, Lindzey, and Spuhler (1975) and by Mackenzie (1984). Turkheimer (1991) discusses reasons for keeping mean differences and individual differences within a single explanatory framework. Transethnic adoption studies include studies of American black children adopted by white adoptive parents and Japa-

nese children adopted by Chinese adoptive parents (Weinberg, Scarr, & Waldman, 1992; Tseng, Ebata, Miguchi, Egawa, & McLaughlin, 1990).

[2]Consider that in Table 4.1, the correlation for parent and child reared apart (.24) is the same as that for biological siblings reared apart (.24). If different genes were to influence IQ in adulthood versus childhood, then the parent–child (far apart in age) would be much weaker than the sibling association (close in age).

[3]The observation that immigrant Asian children are outscoring their American compatriots raises the reasonable possibility that some racial differences in IQ may be attributable to different genes. But evidence on immigrants is not strong. Immigrant people may fail to represent a random draw of their original populations; for example, the Southeast Asians who manage to reach American shores may be more ambitious, determined, and intelligent than those who choose not to migrate or fail in their efforts to do so. If the children of some immigrants are genetically smarter than other Americans, we still cannot be sure that this generalization would hold for their home populations.

[4]Two studies have reported on representative samples of infant adoptees, but are unpublished (Sharma & Benson, 1992; Warren, 1992). A recent book by Brodzinsky and Schechter (1990) also discusses outcomes of adoption.

References

Baker, L. A., & Daniels, D. (1990). Nonshared environmental influences and personality differences in adult twins. *Journal of Personality and Social Psychology, 58,* 103–110.

Brodzinsky, D. M., & Schechter, M. D. (1990). *The psychology of adoption.* New York: Oxford University Press.

Burks, B. S. (1928). The relative influence of nature and nurture on mental development: A comparative study of foster parent–foster child resemblance and true parent–true child resemblance. *27th Yearbook of the National Society for the Study of Education* (Part 1, pp. 219–316).

Caldwell, B. M., & Bradley, R. H. (1984). *Home Observation for Measurement of the Environment* (HOME). Little Rock: University of Arkansas Press.

Caplan, N., Choy, M. H., & Whitmore, J. K. (1992). Indochinese refugee families and academic achievement. *Scientific American, 267,* 36–42.

Clark, E. A., & Hanisee, J. (1982). Intellectual and adaptive performance of Asian children in adoptive American settings. *Developmental Psychology, 18,* 595–599.

Dawkins, R. (1982). *The extended phenotype: The gene as the unit of selection.* Oxford: Oxford University Press.

Degler, C. N. (1991). *In search of human nature: The decline and revival of Darwinism in American social thought.* New York: Oxford University Press.

Dunn, J., & Plomin, R. (1990). *Separate lives: Why siblings are so different.* New York: Basic Books.

Frydman, M., & Lynn, R. (1989). The intelligence of Korean children adopted in Belgium. *Personality and Individual Differences, 12,* 1323–1325.

Herrnstein, R. J. (1973). *I.Q. in the meritocracy.* Boston: Little, Brown.

Jencks, C. (1972). *Inequality: A reassessment of the effect of family and schooling in America.* New York: Basic Books.

Johnson, R. C., Ahern, F. M., & Cole, R. E. (1980). Secular change in degree of assortative mating for ability. *Behavior Genetics, 10,* 1–8.

Kari, S. A., & Avise, J. C. (1992). Balancing selection at allozyme loci in oysters: Implications from nuclear RFLPs. *Science, 256,* 100–102.

Leahy, A. M. (1935). Nature–nurture and intelligence. *Genetic Psychology Monographs, 17,* 237–308.

Loehlin, J. C. (1992). Should we do research on race differences in intelligence? *Intelligence, 16,* 1–4.

Loehlin, J. C., & DeFries, J. C. (1987). Genotype–environment correlation and IQ. *Behavior Genetics, 17,* 263–277.

Loehlin, J. C., Lindzey, G., & Spuhler, J. N. (1975). *Race differences in intelligence.* San Francisco: W. H. Freeman.

Mascie-Taylor, C. G. N., & Gibson, J. B. (1978). Social mobility and IQ components. *Journal of Biosocial Science, 10,* 263–276.

Mackenzie, B. (1984). Explaining race differences in IQ: The logic, the methodology, and the evidence. *American Psychologist,* 39, 1214–1233.

McGue, M., & Lykken, D. T. (1991). Genetic influence on risk of divorce. *Psychological Science, 3,* 368–373.

Neubauer, P. B., & Neubauer, A. (1990). *Nature's thumbprint: The new genetics of personality.* Reading, MA: Addison-Wesley.

Plomin, R., & Bergeman, C. S. (1991). The nature of nurture: Genetic influences on "environmental" measures. *Behavioral and Brain Sciences, 14,* 373–427.

Plomin, R., & Daniels, D. (1987). Why are children in the same family so different from one another? *Behavioral and Brain Sciences,* 10, 1–16.

Plomin, R., Loehlin, J. C., & DeFries, J. C. (1985). Genetic and environmental components of "environmental" influences. *Developmental Psychology, 21,* 391–402.

Plomin, R., McClearn, G. E., Pedersen, N. L., Nesselroade, J. R., & Bergeman, C. S. (1989). Genetic influence on adults' ratings of their current family environment. *Journal of Marriage and the Family, 51,* 791–803.

Reiss, D., Plomin, R., & Hetherington, E. M. (1991). Genetics and psychiatry: An unheralded window on the environment. *American Journal of Psychiatry, 148,* 283–291.

Robertson, I. (1981). *Sociology* (2nd ed.). New York: Worth.

Rodgers, J. L. (1984). Confluence effects: Not here, not now! *Developmental Psychology, 20,* 321–331.

Rodgers, J. L., & Rowe, D. C. (1985). Does contiguity breed similarity?: A

within-family analysis of nonshared sources of IQ differences between siblings. *Developmental Psychology, 21,* 743–746.

Rodgers, J. L., Rowe, D. C., & Li, C. (in press). Beyond nature vs. nurture: DF analysis of nonshared influences on problem behaviors. *Developmental Psychology.*

Rowe, D. C. (1981). Environmental and genetic influences on dimensions of perceived parenting: A twin study. *Developmental Psychology, 17,* 203– 208.

Rowe, D. C. (1983). A biometrical analysis of perceptions of family environment: A study of twin and singleton sibling kinships. *Child Development, 54,* 416–423.

Rowe, D. C., & Plomin, R. (1981). The importance of nonshared (E_1) environmental influences in behavioral development. *Developmental Psychology, 17,* 517–531.

Rushton, J. P. (1988). Genetic similarity in male friendships. *Ethology and Sociobiology, 10,* 361–373.

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

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

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

Schooler, C. (1972). Birth order effects: Not here, not now! *Psychological Bulletin, 78,* 161–175.

Sharma, A. R., & Benson, P. L. (1992, March). *A comparison of adopted and nonadopted adolescents on psychological at-risk indicators.* Paper presented at the meeting of the *Society for Research on Adolescence,* Washington, DC.

Stark, R. (1985). *Sociology.* Belmont, CA: Wadsworth.

Tambs, K., Sundet, J. M., Magnus, P., & Berg, K. (1989). Genetic and environmental contributions to the covariance between occupational status, educational attainment, and IQ: A study of twins. *Behavior Genetics, 19,* 209–222.

Taubman, P. (1976). The determinants of earnings: Genetics, family and other environments: A study of white male twins. *American Economic Review, 66,* 858–870.

Teasdale, T. W. (1979). Social class correlations among adoptees and their biological and adoptive parents. *Behavior Genetics, 9,* 103–114.

Teasdale, T. W., & Owen, D. R. (1981). Social class correlations among separately adopted siblings and unrelated individuals adopted together. *Behavior Genetics, 11,* 577–588.

Tseng, W., Ebata, K., Miguchi, M., Egawa, M., & McLaughlin, D. G. (1990). Transethnic adoption and personality traits: A lesson from Japanese orphans returned from China to Japan. *American Journal of Psychiatry, 147,* 330–335.

Turkheimer, E. (1991). Individual and group differences in adoption studies of IQ. *Psychological Bulletin, 110,* 392–405.

Waller, J. H. (1971). Achievement and social mobility: Relationships among IQ score, education, and occupation in two generations. *Social Biology, 18,* 252–259.

Warren, S. B. (1992, March). *Types and prevalence of behavior problems among adopted and nonadopted adolescents: An epidemiological study.* Paper presented at the meeting of the *Society for Research on Adolescence,* Washington, DC.

Weinberg, R. A., Scarr, S., & Waldman, I. D. (1992). The Minnesota transracial adoption study: A follow-up of IQ test performance at adolescence. *Intelligence, 16,* 117–135.

Winick, M., Meyer, K. K., & Harris, R. C. (1975). Malnutrition and environmental enrichment by early adoption. *Science, 190,* 1173–1175.