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PARENTING AND ITS EFFECTS ON CHILDREN: On Reading and Misreading Behavior Genetics

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■ **Abstract** There is clear evidence that parents can and do influence children. There is equally clear evidence that children's genetic makeup affects their own behavioral characteristics, and also influences the way they are treated by their parents. Twin and adoption studies provide a sound basis for estimating the strength of genetic effects, although heritability estimates for a given trait vary widely across samples, and no one estimate can be considered definitive. This chapter argues that knowing only the strength of genetic factors, however, is not a sufficient basis for estimating environmental ones and indeed, that attempts to do so can systematically underestimate parenting effects. Children's genetic predispositions and their parents' childrearing regimes are seen to be closely interwoven, and the ways in which they function jointly to affect children's development are explored.

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INTRODUCTION

What are the forces that affect when and how children will change as they grow older? Can development be seen as a progressive process whereby children move toward a specifiable outcome or end state that we can call maturity? What conditions

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determine differences among children in their rates of development or their ultimate outcomes? These questions have been at the heart of much of the work in developmental psychology since the inception of the field. In pursuing the answers, the broad forces of nature and nurture, and the interplay between them, have been of central concern. It has long been clear that there are powerful maturational time-tables governing developmental change: e.g. the progression in infancy from sitting to crawling to standing to walking, or in the acquisition of language, the transition from rudimentary one-word utterances through intermediate phrases to the production of full, well-formed sentences. However, it has been equally obvious that children are learning many things through their daily experiences in interacting with the physical and social world, and that what is learned is not encoded in the genes. Some of the experiences children have are random—not planned or organized by any outside agency—but some occur according to what might be called a socialization time table. It is here that parenting has its place.

All societies prescribe certain characteristics that their members are expected to possess and certain things people must not do, if they are to function adequately as members of their society. Some of these prescriptions and proscriptions are nearly universal across cultures, such as the requirement for parents, or specified parent surrogates, to provide nurturance and protection for children. Other standards and values vary greatly from one cultural setting to another. In all societies, training of children occurs, and social controls are in place to ensure that children are socialized—that is, brought up in such a way that each new generation acquires the prescribed patterns of beliefs and behaviors. Of course, cultures do change, either slowly or rapidly, so that the cross-generational transmission is by no means absolute. A new generation may need to adapt to conditions that the parent generation did not face. And transmission of values, even when they continue to be appropriate for succeeding generations, is not always successful. Some children in every cohort may be seen to be inadequately socialized by the criteria that the society applies.

Not all socialization occurs in childhood. People are socialized into the customs and standards of an occupational culture when they take up an entry-level job. Socialization and resocialization occur when adults enter into new life roles (e.g. marriage, parenthood). In considering the role of parents, however, we are mainly concerned with childhood socialization. Some of the socialization that occurs throughout childhood is in a sense anticipatory, in that it functions to prepare children for adaptation to a fairly wide range of life roles and the various contexts children will encounter as they grow older. But childhood socialization also concerns the training of children in modes of behavior that are acceptable for the stage of childhood they currently occupy. Societies set different standards for people at different stages of their life cycle, and there are requirements that loom especially large in childhood. These include requirements for children to comply with adult demands, to avoid irritating adults or disrupting their activities, to accept age-appropriate responsibility, and to function as a pleasant, cooperative family member.

In modern societies, there are least three major contexts in which childhood socialization takes place: families, peer groups, and out-of-home contexts such as school

classrooms or day-care centers in which the daily experiences of children are structured and overseen by adults. The enormous body of literature on childhood socialization has strongly emphasized the role of parents. This emphasis has a long and deep tradition. The idea that ''as the twig is bent, so grows the tree'' can be traced at least as far back as Greek and Biblical times—(probably earlier), and in most societies parents are the ones assigned primary responsibility for ''bending'' the children in desirable directions, by supervising, teaching, and disciplining them as they grow up. Early childhood in particular has long been thought to be a period in the life cycle when humans are especially plastic—a time when children are especially open to social influences on characteristics they will carry with them long after they have left their family of origin. Things thought to be especially vulnerable to influence in the first 5–7 years of children's lives include the language they speak, their food preferences, their religious beliefs, and certain enduring personality traits.

In the twentieth century, assumptions about the importance of within-family childhood socialization have been part of the fabric of mainstream psychological theories. From roughly the 1920s through the 1960s, behaviorist learning theories held sway, emphasizing the ''blank slate'' status of infants and the power of adults to teach young children, for good or ill, what they must learn. Parents, of course, were seen as the most available teachers, and the ones responsible for carrying out the training of their children. The physiological drive states (hunger, fatigue) with which children are innately endowed were not ignored in the learning theories of the time, so there was some blending of nature and nurture, but the major emphasis was on the control of learning processes exercised by environmental inputs. Psychoanalytic theories of this period emphasized the importance of early in-family experience in determining subsequent inner conflicts, defense mechanisms, and internalization of values. In more recent decades, as the cognitive revolution took hold and learning theory (as it related to socialization) was reformulated as cognitive social learning theory, the active role of children as participants in their own socialization was increasingly stressed. Currently, there is increasing emphasis on the role of parents' and children's mutual perceptions and understandings about each other's dispositions and intentions as determiners of their influence upon one another. But none of these theoretical shifts has greatly affected the underlying assumption that parents have a powerful impact on the characteristics children develop and the directions their lives take. The child development research literature has continued to include a wide range of studies on such things as (*a*) familial risk factors (i.e. aspects of family functioning that are related to the subsequent development of externalizing or internalizing disorders in children); (*b*) social conditions that affect such parenting practices as how well parents are able to monitor their children, or how warm and responsive they are; and (*c*) parenting behaviors as mediators of the connection between societal risk factors (e.g. poverty or dangerous neighborhoods) and children's adjustment.

In recent decades, there has been a countervailing ground swell of research and theorizing about nature—the genetic endowment of parents and children—as exerting a powerful influence on the characteristics that children develop. Of course, for many decades, elementary psychology textbooks have carried tables comparing identical

and fraternal twins with respect to their degree of similarity on IQ or other traits. Studies of adopted children were also widely reported many years ago, and inferences were routinely drawn from both twin and adoption studies concerning the importance of genetic factors in development. Still, for many years, thinking remained largely compartmentalized, and readers continued to believe in both the importance of genetic factors and the importance of socialization factors as though they were in no way incompatible. In recent years, however, there has been more sophisticated work in behavior genetics, and there are insistent voices claiming that the findings from this work are indeed incompatible with many widely-held views about the power of within-family socialization.

These messages from behavior genetics have been picked up and synthesized with other misgivings about the weaknesses of socialization research into a more broad-based attack on traditional assumptions concerning parenting and its effects. Rowe's book, *The Limits of Family Influence* (1994), stated the case strongly, and Harris's more popular book *The Nurture Assumption* (1998) attracted a flurry of media attention to the issues. These authors have drawn together the findings from some well-known studies of parenting effects and findings from behavior genetics to make the following claims:

1. The connections that studies have found between the way parents deal with their children and how the children turn out are actually quite weak and have proved difficult to replicate. When parent ''effects'' are found, they tend to be effects on the way children behave at home and the relationships they develop with their parents. There is little carry-over from at-home experiences to the way children function in out-of-home contexts
2. When studies do establish connections between parenting and children's attributes, these are correlational findings. An example is Baumrind's early finding—now widely replicated—that the children of parents who are both responsive and firm tend to be more competent and cooperative than children of parents who are either authoritarian or permissive (Baumrind & Black 1967). Such findings have traditionally been interpreted as showing that authoritative parenting has beneficial effects on children, ignoring the possibility that the causal connection may run the other way—i.e. that competent, cooperative children may make it easier for their parents to be firm and responsive. In fact, the critics argue, parent behavior is substantially driven by the behavior of children, and much if not most of the parent/child correlation can be accounted for by the child's genetic predispositions.
3. Parental influence has been emphasized at the expense of sources of influence that in fact have great—or perhaps greater—importance in shaping children's development. Two kinds of influence which critics argue have been underemphasized are genetic predispositions and the influence of peers.

In the popular media, these critiques have been condensed into the oversimplified message ''Parents don't matter'' or ''matter very little''—news bites that, on their face, have little relation to reality as it is experienced daily in family life. Often,

reports in the popular media do not reflect what the cited authors actually said. For example, late in her book, Harris (1998) says she believes parents can foster the development of specific talents (e.g. by providing music lessons) and can influence such things as children's leisure time activities, their food preferences, their religious beliefs and practices, and the acquisition of knowledge and skills and preferences that will contribute to their ultimate choice of a profession. Yet, the burden of her book is to down-play such influences and stress the respects in which parents are not influential. Rowe says: " . . . parents in most working to professional-class families may have little influence on what traits their children may eventually develop as adults." (1994:7). His use of the word "may" does not greatly soften the import of his message. He goes on to say that he doubts whether any undesirable trait displayed by a child can be significantly modified by anything a parent does. Scarr (1992) expresses a similarly skeptical view about the possible effects of interventions. Such views, of course, when picked up and simplified in the popular press, can have serious implications for public policies concerning whether to invest in remedial or supportive programs for children and families.

These critiques constitute serious efforts to present a point of view that is clearly different from the traditional emphasis on the importance of parenting. They cite large bodies of data and have attracted the support of highly reputable psychologists. They deserve to be taken seriously. Nonetheless, I believe that they are out of date with respect to both the genetic studies and the parenting-effects studies they cite, and that they seriously misinterpret the pertinent body of research.

I turn first to the question of how strong the connections are between what parents do with their children and how the children turn out. I then turn to issues of genetic factors, in particular the ways in which these factors may determine or limit how we can interpret parenting effects.

HOW STRONG IS THE CONNECTION BETWEEN PARENT AND CHILD BEHAVIORS?

As noted above, critics charge that interpreters of traditional socialization studies have exaggerated the importance of parenting in children's lives—that in fact, the effect sizes reported in many widely-cited studies are really quite small. Indeed, reviews of research done before the mid-1980s did show weak correlations between parenting processes and children's characteristics (e.g. Maccoby & Martin 1983). Since then, many studies have come up with more robust findings, no doubt reflecting improvements in the ways in which parent and child characteristics are assessed. Leading researchers no longer rely on a single measure, such as a parent or child interview or a parent or child self-report scale, as a measure of parent or child attributes. Instead information is obtained from multiple sources—from parents, children, teachers, school records, sometimes from children's peers and police records as well—and importantly, from direct observation of parent-child interactions

and of children in out-of-home settings. When several measures such as these are aggregated, associations between parent attributes and children's behavior can be quite substantial. Parenting variables have typically accounted for 20% to 50% of the variance in child outcomes (Conger & Elder 1994, Reiss et al 1995). Exceptionally robust connections are reported in the recent large-scale study of adolescents in never-divorced and step-families, Hetherington and colleagues (Hetherington et al 1999). Using composite scores for both parenting styles and children's attributes, report a concurrent coefficient of 0.76 between mothers' ''authoritative parenting'' and adolescents' ''social responsibility'' (the coefficient for fathers is 0.49). Parental negativity has very strong connections for both parents with adolescents' depression and internalizing behavior.[1] Patterson and colleagues have also found substantial correlations between parental characteristics (e.g. disciplinary practices and monitoring) and children's antisocial behavior (Patterson & Forgatch 1995). They are able to show connections between parental behaviors and the children's negative, coercive behavior both at home and in out-of-home contexts.

Concurrent correlations are usually considerably larger than predictive ones. Longitudinal studies present the opportunity to examine the connections, if any, between child-rearing styles at one point in time and subsequent attributes of the child. The strength of the connections that have been found depends on many things, such as what ''packages'' or clusters of parent and child variables are considered, the way they are measured, the length of time between predictive and outcome measures, and whether background variables are statistically controlled. A few examples will illustrate the range of findings. Kochanska 1997b:94 has been able to show that aspects of early parenting account for a significant but moderate (Beta coefficient 0.29, F 9.96) portion of the variance in young children's self-regulation and internalization assessed a year later. Pettit and colleagues (1997:908) found some—but fewer and weaker—predictive relationships between parenting as assessed at the beginning of the kindergarten year and children's adjustment and academic performance seven years later, in the sixth grade. Strong predictive power of family interaction processes over much longer spans of time have been found in longitudinal studies of antisocial behavior (see Loeber & Dishion 1983). In current socialization studies, simple first-order correlations between parenting characteristics and child outcomes are seldom relied on. Indeed, sometimes they are not even reported. Instead, multivariate analyses are used to investigate such questions as whether a given aspect of parenting has different effects on different kinds of children or in families living in different circumstances; or whether different aspects of parenting have independent, addi-

[1]The coefficient connecting mothers' negative/conflictual behavior with children's externalizing behavior was 0.82; for fathers this coefficient was 0.79. These coefficients are path coefficients in structural models in which two aspects of parenting (authoritative parenting parental negativity/conflict) are considered simultaneously along with measures of sibling relationships. Scores derived from videotapes of parent-child interactions were a contributing element (though a minor one) in the composite scores of both parenting characteristics and child outcomes.

tive effects, whether they are interchangeable, or whether they interact so that the effects of one depend on the level of another.

In longitudinal work, the initial level of a child's characteristic at time 1 is sometimes statistically controlled to determine whether a time-l parent attribute is associated with subsequent *change* change in the child's behavior. As an example, Patterson & Bank (1989) studied families when their sons were in grade school, and again when the boys were adolescents. They found that changes in parenting during these years were strongly related to the chances of a boy's being arrested for delinquent activities in adolescence, even after the boy's anti-social tendencies at grade-school age were controlled. We see, then, that a variety of questions are being asked in current and recent research—questions to which simple parent/child correlations, either concurrent or time-lagged, will not provide answers.

A word should be said, too, about how large a correlation between some aspect of parenting and a child outcome is required for the relationship to be considered important or meaningful. Along with the rest of the psychological discipline, developmental psychologists are currently turning away from reporting the outcomes of studies primarily (or only) in terms of significance levels (*p* values) that indicate degree of departure from the null hypothesis. Instead, results are beginning to be reported in terms of effect sizes. For purposes of policy decisions in the medical arena, correlations as small as 0.03 between the use of a medication and reduction of disease have been considered strong enough to justify FDA approval of the drug (Rosenthal 1999). The importance of a medical intervention can be estimated in terms of such outcomes as the number of heart attacks averted or the number of people for whom a debilitating, chronic disease can be arrested or reversed. In the past, correlations in the 0.20s or 0.30s between aspects of family functioning and children's outcomes have often been dismissed as inconsequential. But when translated into the number of children who are at risk, for example, for failing in school or becoming delinquent or seriously depressed, predictive coefficients of this magnitude can be seen as by no means trivial. From the standpoint of social policy, the issue becomes one of how much importance a society attaches to social/behavioral outcomes, as compared with medical ones. This is obviously a matter of values, not statistics.

Studies continue to vary considerably with respect to the size of first-order correlations between parent and child characteristics. Clearly, a given parent behavior may have different effects on different children, depending on such things as age, sex, temperament, and distinctive prior experiences. If such differential effects exist, aggregating data across a whole sample of children will wash out parent/child effects—effects that might be quite robust within sub-groups of children. (See section on interactions, below.) It is not possible to arrive at any general rule as to when dividing by subgroups will increase or decrease a parent-child correlation. That will depend on the researcher's theoretical and empirical skill in identifying what the pertinent groupings might be. The use of more sophisticated statistical methods has contributed significantly to the ability of present-day researchers to identify parenting effects within the matrix of other factors with which they often co-vary.

Not only have methods of assessment been improved, but current socialization research includes a broader array of parenting attributes and focuses on a set of parenting processes that were not so clearly delineated in times past. One aspect of parental skill that has emerged in several recent studies as related to children's well-being is household organization; another concerns the ability of some parents to develop a reciprocal form of interaction with their children (e.g. shared positive affect, mutual responsivity). Studies of the predictive power of parent-child reciprocation in early childhood have yielded quite robust parenting effects (See Kochanska & Thompson 1997 for a review of this work). These examples illustrate the ways in which the field of family-impact studies has been growing in conceptual as well as methodological strength. Nevertheless we must be reconciled to the fact that there are important aspects of parenting that will never be revealed in studies that, by necessity, try to encapsulate parental characteristics into measurable clusters or traits. There are the memorable little socialization moments when the members of a parent/child dyad are, for some reason, especially attuned to one another—when the child, perhaps by virtue of having encountered a new and salient issue, is ready to both explain and listen. At such a moment, the parent may do or say something that makes a deep impression and can have a lasting influence. Conversely, a broken promise or a revealed deception may break the prevailing relationship of trust between the two, changing the nature of the influence that is possible between them. Such moments are unique to a dyad and may not be captured in socialization studies, even though our awareness of them is highlighted in biographies, autobiographies, and fiction.

I do not want to claim too much for the strength of parental influence in children's lives. Critics are right in pointing out that we have overemphasized these influences at the expense of other kinds of environmental influences. To what extent early childhood is a time of especially great plasticity, during which environmental inputs will be more likely to have a lasting influence than inputs later in life is an open question. Probably the answer will vary, depending on what domain of children's development we are talking about. (See for example, Neville's finding [Neville 1995] that the openness to influence by early experience differs between the semantic and syntactic language systems). Because parents are usually the ones who spend the most time with young children over extended periods of time, these questions of changing plasticity do matter in our efforts to understand the parental realm of influence. Still, parents are never the only source of influence on children, and as children grow older, they are more and more subject to the influence of peers, of schools and teachers, and of television. Also, there are the random events—a serious illness or accident, an unexpected success, a residential move, an environmental catastrophe—that can alter the trajectory of a child's life in ways that have little to do with parenting.

Of course, when we do see robust correlations between parent and child attributes, the question of the direction of effects arises at once. In making their argument that we may be seeing child-to-parent effects rather than the reverse, critics

have relied heavily on the findings of behavior genetics[2], especially on studies of twins and adopted children. They have also relied on these findings to urge that nonparental aspects of a child's environment have greater weight than parental inputs in determining how a child will develop.

THE CHALLENGE FROM BEHAVIOR GENETICS

Some of the major findings of behavior genetics are powerful and require students of socialization to rethink some of their assumptions. Many of these findings are well known, and I do not summarize them in any detail here, but focus on the main lines of argument that bear on the issue of parenting effects.

The Focus on Variation

Behavior geneticists seek to understand the sources of variation in some human trait or characteristic. Their approach is to be distinguished from that of evolutionary psychologists, who seek to understand the genetic underpinnings of characteristics that are relatively uniform across a species.

There are important effects of both genes and environment that are overlooked in studies that focus on the variation of a characteristic within a given population. A human characteristic such as being born with two eyes is entirely genetic, yet its heritability would be computed as zero in a twin or adoption study since it is a characteristic that does not vary within the population studied. Similarly, there may be an environmental factor that affects the mean level of a characteristic—raising or lowering all scores to a similar degree—without greatly disturbing the rank-order of individuals on the characteristic. Thus, adoption studies have found that the correlation of adopted children's IQs with those of their biological parents can remain substantial, while at the same time the average IQ of the adopted children is higher than that of their natural parents, as though children receive an IQ bonus from being adopted into relatively stable, middle-class homes, while nevertheless continuing to differ from each other according to their genetic endowment. In a study of French children adopted at about the age of 5, it was found that the amount of increase in their IQs (assessed again in adolescence) was considerably greater for children adopted into affluent, well-educated families than for those adopted into underprivileged homes (Duyme et al 1999).

Secular trends illustrate the same point. The ''Flynn effect'' (Flynn 1987, 1999)—the substantial, monotonic rise in mean IQ scores over many decades in

[2]The term ''behavior genetics'' is a commonly used term for twin studies, adoption studies, and epidemiological studies of family resemblance. Currently, since molecular geneticists also study certain ''behavioral'' phenotypes in their relation to genes, the term quantitative genetics is sometimes used to distinguish studies that rely on statistical genetic analyses of family resemblance rather than on molecular gene identifiers. However the term ''behavior genetics'' is used here because it is more familiar to readers.

Western industrialized countries—is well known. There has been a substantial rise in the rates of smoking among American women in the last several decades, and the rates of drinking alcohol dropped during prohibition. These changes, of course, have occurred during periods of time that are much too short to reflect any genetic changes and they have occurred despite the fact that heritability estimates for IQ, drinking, and smoking have remained quite stable over the same time periods during which the average levels were changing. A similar phenomenon is seen in some migration studies, in which second-generation immigrants are on average quite different from their foreign-born grandparents, even on highly heritable traits such as height (Angoff 1988) or obesity (Price at all 1993, Ravussin et al 1994). The implication of these phenomena for parenting effects is this: There may have been secular changes in parenting—triggered perhaps by such things as changes in family structure or overall economic level—that have had widespread effects on children without affecting heritability estimates for the outcome characteristics being affected.

These powerful environmental effects are missed in the estimates of E (environment) derived from behavior genetics studies of twins and adopted children. Another way of putting this point is to note that high heritability of a trait does not imply that it is not also subject to the influence of environmental factors, or that it cannot be changed by alterations in environmental conditions. It is for this reason that, when comparing group means (by race, sex, or socioeconomic status) it is not legitimate to interpret any group differences in terms of estimates of genetic or environmental effects derived from quantitative behavior genetic studies.

Experimental Interventions with Parents If large-scale environmental events can change mean levels of a characteristic without greatly changing the rank-order of individuals, it follows that experimental interventions might do the same. It is difficult to change actual parenting practices through parent-training programs, and then to document that program-induced changes in parenting change the mean levels of children's characteristics. Such programs must be longitudinal, of course, and must have an untreated control group for comparison. Studies that intervene with the parents but do not simultaneously treat the children, and that have random assignment of families to treatment or control groups, are understandably rare, but several have clearly shown that when treatment is able to change parental behavior toward children in specified ways, the behavior of children changes correspondingly (e.g. Patterson & Forgatch 1995, Van den Boom 1994, Forehand et al 1980). Dishion et al (1992) were able to show that it was indeed the reduction of parent-to-child coercive behavior, brought about by a parent-training intervention with a randomly assigned experimental group, that produced declining levels of antisocial behavior in a group of aggressive children. An intervention program that changes the mean of a group of parents (and consequently, of their children's behavior as well) may or may not change the initial rank order of the children. Researchers commonly find that some parents are influenced more than others by an intervention, and some children are affected more than others by improvements in parental disciplinary or monitoring prac-

tices. These differential effects might either increase or decrease the range of outcome scores in the treatment group, depending on whether it was the initially better-functioning or poorly-functioning families who were most affected by the intervention. However, expanding or shrinking the range of outcome scores does not necessarily change the initial rank-order. The point here is that changes in a mean can be independent of any changes in rank order. Thus, changes in a mean can clearly demonstrate an environmental effect, quite apart from any correlational information (based on rank orders of individuals) that might be used to compute genetic or environmental effects in a genetic analysis. The environmental effects revealed by the mean change would go undetected in a correlational analysis.

The Claim for Substantial Genetic Effects

In traditional behavior genetic research, data from studies of twins and adopted children are used to compute heritability estimates (h^2), which are interpreted as estimates of the proportion of variance accounted for by genetic factors. Many such studies have yielded substantial heritability estimates. Identical twins have been found to be more similar to each other than are same-sex fraternal twins with respect to a wide range of characteristics, including susceptibility to certain diseases, intelligence, temperament, and a number of personality characteristics. The inference is that this must be due to their greater genetic similarity, because the important aspects of their environments—parenting received, neighborhood, presence of a same-age, same-sex sibling—are presumably equally similar for the two kinds of twin pairs. Adopted children have been found to be more similar to their biological parents than to their adoptive parents with respect to a selected set of characteristics for which researchers have been able to obtain measures from both biological and adoptive parents.

In a general sense, the behavior geneticists have made their case. Children's genetic endowments do clearly affect how individuals will develop—in comparison to other children—to a much greater extent than was thought to be the case during the years of the ascendancy of reinforcement learning theories and psychodynamic theories (the middle decades of the twentieth century.)

How substantial is this genetic contribution? Critics have argued that estimates derived from twin studies systematically overestimate the genetic contribution to a trait because identical twins in fact have more similar environments than do same-sex fraternal twins. Identical twins (compared with fraternals) are treated more similarly by their parents, spend more time together (and hence constitute a greater proportion of each other's social environment), and more often share the same friends (Dunn & Plomin 1986, Plomin et al 1988, Reiss et al 1999, Rowe 1983). Probably, the greater similarity in the environments of identical twins is not sufficiently strong to negate the findings on genetic effects, but it does weaken them. Very likely, it helps to account for the fact that heritability estimates are usually larger in twin studies than in adoption studies.

Of course, the genetic contribution might be expected to be greater for some human attributes than others. It appears to be more substantial for measures of intelfjlectual abilities than for social or personality attributes. However, it is difficult to establish a reliable, generalizable estimate for any given trait. For one thing, estimates

vary depending on the source of information for measuring a trait. When children's characteristics are assessed through parents' ratings, heritability estimates are often considerably higher than when assessments are derived from behavioral observations of the children, from children's self-reports, or from teacher ratings. It appears that parents see their children as more different from one another than other sources of information find them to be (a contrast effect). In a recent review of studies of the heritability of aggressive behavior, Cadoret and colleagues (Cadoret et al 1997) report a very wide range of heritability coefficients, (from near zero to over 0.70), with the higher figures coming from studies using parent report measures, and the lower ones from observational studies. Miles & Carey (1997), in a meta-analysis of 24 twin and adoption studies, report substantially greater values for h^2 based on parent reports than for those based on adolescent self-reports.

Especially important is the fact that the size of a heritability coefficient depends greatly on the range of both genetic and environmental factors in the population being studied (G. Patterson, under review). Estimates of the heritability of a given trait can change considerably when a new estimate is based on a culturally different population, or especially when a new estimate includes families from a wider range of subcultures and socioeconomic levels.

All this means that while the fact of a genetic contribution to human variability is not in doubt, the size of this contribution is indeterminate for any given trait. More specifically, the size of a heritability estimate cannot be generalized from the specific population—in its specific environment—assessed with the specific set of measures used in a given study.

Estimating the Size of Environmental Effects

In twin and adoption studies, estimates of the power of environmental factors are derived by adopting the additive assumption, i.e. by assuming that that the sources of variation in a trait can be separated into independent genetic (G) and environmental (E) components that together (along with error variance) add to l00% of the variance to be accounted for. On the basis of this assumption, the heritability coefficient can be subtracted from 100% to yield an estimate of the environmental contribution to variance. Estimating E in this way can be done without utilizing any direct measures of environmental factors. Obviously, if the estimates of h^2 are indeterminate, so are the estimates of E derived by subtracting h^2 from 100%.

The validity of the additive assumption has been widely challenged (Feldman & Lewontin 1975, Gottlieb l995, Block 1995, Rose 1995, Turkheimer 1998) A number of these critiques have appeared in connection with the controversy over Hernstein & Murray's book *The Bell Curve* (1994), but they are equally pertinent to the current debate over parenting effects. If one adopts the additive assumption, it follows that when h^2 is large, the effects of all environmental factors—including parenting—must be correspondingly small. A major counter-argument has been that in fact, everything that human beings are or do must be a joint function of both their genes and their life experiences. The pathway between genes and phenotypes is a long one,

with G and E being interwoven all along the way (see Elman et al 1996). The effects of genes depend on environmental triggers or enabling conditions, and the effects of different environments depend on the genetic characteristics of the individuals encountering an environment. When genes and environment act jointly, this can emerge empirically in behavior genetics studies in the form of either G × E correlations or G × E interactions. In estimating environmental effects, much depends on how these joint processes are handled (or not handled). Both kinds of coaction are considered below, but the main point here is that neither G × E covariances nor interactions fit into an additive model

Shared and Unshared Environmental Effects

In twin and adoption studies, once an overall estimate of E has been derived by subtracting h^2 from 100%, E can be further subdivided into two environmental components: Es (shared environment) and Eus (unshared environment). Once again, this can be done without utilizing direct measures of either. If fraternal twins are quite similar—more similar than would be expected from their shared genetics alone—or if adopted children are more similar to the parents or siblings in their adopted families than they are to adults or children in other households, this would imply an effect of their rearing environment, including of course the parents' child-rearing methods. Es is estimated from sibling similarities, and any variance still unexplained after the effects of G and Es have been accounted for are attributed to unshared environment or error of measurement.

An especially surprising finding emerging from the body of behavior genetics work has been that the effects of nonshared environment appear to be much greater than those of shared environment (see Plomin & Daniels 1987 and Plomin et al l994). Recent estimates of nonshared environmental effects are much reduced when measurement error is taken into account (Rutter et al 1999). And a number of studies of social behavior or pathology have found substantial shared-environment effects. Nevertheless, shared environmental effects consistently emerge as small, and indeed are often reported as being close to zero (Plomin & Bergeman 1991). Adopted children do not appear to resemble their adoptive siblings or parents any more closely than they resemble children growing up in different households. Also,in many respects fraternal twins—or ordinary siblings, for that matter—do not greatly resemble each other or their parents.

Critics have urged that it is not valid to estimate environmental effects, either shared or unshared, without measuring them (Goodman 1991, Hoffman 1991, Rose 1995, Stoolmiller 1999, Patterson 1975). Recent work has involved designs in which both genetic and environmental factors have been directly assessed. For example, a group of leading behavior geneticists and leading students of parent-child interaction collaborated in a study comparing children of different degrees of genetic relatedness (twins, full siblings, half siblings, step siblings), in which parental child-rearing inputs were assessed through observations of parent-child interactions, as well as through parent and child reports (Reiss 1997, Reiss et al 1999, Hetherington et al 1999).

Relying on the additive assumption, these investigators have partitioned the variance in child adjustment outcomes into the three components: G, Es, and Eus, reporting substantial contributions from genetics. Effects of shared environment are variable, making clear contributions to some outcomes but not others; in general, though, they are considerably smaller than the substantial contributions from unshared environment. Unfortunately the design of this study confounds genetic similarities and family structures: the group in which siblings are most genetically unlike (stepsiblings) is also the group in which the two siblings receive the most discordant parenting (Hetherington et al 1999). It should be noted, too, that the range of environmental variation is restricted in this study. Thus, many of the reported findings of this important study are difficult to interpret.

The inference of behavior geneticists' claims concerning shared and unshared environments might be that children are not greatly affected by the characteristics of the household in which they are growing up. The weak shared-environment effects have been interpreted to mean that such factors as the parents' income or education, parental pathology, the level of harmony or conflict between the parents, or the neighborhood where the family lives must have little impact on how well the child will do in school, how socially competent the children will be, and so forth (Plomin et al 1994, Scarr & Grajek 1982).

These findings on weak shared-environment effects are startling, considering how consistently studies of parenting effects have found substantial relationships between these family characteristics and child outcomes. As an example, McLoyd (1998) made a strong case for the mediating role of parenting in the deleterious effects of poverty. In McLoyd's analysis, it emerges that the great stresses on impoverished parents—stresses stemming from the day-to-day struggle to find the resources to pay for food and rent, and the stresses of trying to cope with living in crowded housing and deteriorated, dangerous neighborhoods—bring about a weakening of parenting skills and a disorganization of family life. It is the deterioration of parenting, McLoyd found, that in its turn is responsible for many of the adjustment difficulties of children growing up in impoverished families. (See also Conger et al 1994 and Pettit et al 1997 for findings supporting this conclusion.)

It is difficult to reconcile findings such as these with the claim that the aspects of family environments that are shared by siblings do not affect their development. An obvious possibility is that while the family environment does have an effect on each child, its effects are different for different children. There probably was an unspoken assumption, in traditional socialization work, that the effects of shared environments would be to make siblings similar to one another. What the behavior geneticists are telling us is that any influences of familial circumstances—such as parental illness or health, economic prosperity or adversity, good or poor parenting—often function to make siblings different rather than similar. It is possible that a dysfunctional family environment may have effects on both members of a sibling pair, but that the effects are not such as to make siblings more alike, but indeed might function to make them more different. We know from Elder's work on effects of the Great Depression (Elder 1974) that when a father loses his job, the effects on the child will depend on the

age and sex of the child at the time that this stressful event occurs. Even for same-sex twins, we can imagine that if they were adolescents at the time, one might react to a father's job loss by going out to get an after-school job to help support the family while the other might distance himself from the family and spend more time "hanging out" with friends. Both children would be affected by the change in the family environment, but differently.

Any familial or parental factors that serve to make siblings different rather than similar to one another are assigned, in behavior genetics, to the unshared rather than the shared environmental component when computing environmental effects. Behavior geneticists have never said that estimates of unshared environments did not include parent effects, but they argue that if parenting does have effects it must take one of two forms: parents must be treating different children in their families differently (or providing different environments for them), or different children in the same family who are exposed to similar parenting must react to the same parental inputs differently.

A considerable body of recent work has focussed on the question, What is it that makes siblings different from one another? (see Hetherington et al 1994). In these studies evidence is presented that siblings tend to join different peer groups and that siblings have considerably different experiences within the context of the sibling relationship itself. The question of how differently they are treated by their parents remains open. Studies done during a single time period often show that two siblings are treated differently by their parents (see summary by Brody & Stoneman 1994) However, in a longitudinal study Dunn found that parents were fairly consistent in how they treated children at a specific age. That is, a second child, when reaching the age of four, is treated in a similar way to the way his/her older sibling was treated at that age, even though the older sibling may now be receiving different treatment. Thus, over the span of the "growing up" years, different children in the same family received comparable treatment. This fact, of course, would be missed in any study that did not look for it longitudinally; the extent of differential treatment is likely to be overestimated in cross-sectional studies (except in the case of twins). Whether or not children actually are treated differently over the whole span of childhood, there is reason to believe that children's perceptions of how differently they are treated may be of considerable importance in children's development, so concurrent differences are important in their own right (Dunn & McGuire 1994).

In general, the exploration of siblings' unshared environments has been a productive and instructive enterprise. We now know that the environments of children growing up in the same family can indeed be different. But this does not solve the problem of how to interpret aspects of the environment that are truly shared, such as a parental illness, family income, parents' education, or the neighborhood where the family lives—factors that have an impact even when they function to make siblings different rather than alike. As noted above, behavior geneticists tend to conclude that, since it is clear that these aspects of environment are truly shared, they must not be having an effect because Es effects are negligible. As Plomin and colleagues say, "So often, we have assumed that the key influences on children's development are

shared: their parents' personality and childhood experiences, the quality of their parents' marriage relationship, children's educational background, the neighborhood in which they grow up, and their parents' attitude to school or to discipline. Yet to the extent that these influences are shared, they cannot account for the differences we observe in children's outcomes'' (Plomin et al 1994:23).

On the contrary, it seems plausible that these shared factors may indeed have powerful effects that do not show up in computations of shared environmental effects because of the requirement that only an environmental factor that makes siblings more similar can be called ''shared.'' A behavior geneticists might say about the effect of a shared environmental factor that makes siblings different, ''Oh, but we are calling those unshared effects.'' But to call an environmental input unshared even though it is experienced by all children in a family (e.g. a father's job-loss, a mother's depression, a move to a better neighborhood) is an unfortunate distortion of the simple meaning of the word ''shared.'' We could see this as only a trivial matter of terminology choice, but it can lead to serious misunderstandings of behavior geneticists' findings. By definition, they have ruled out the possibility that a truly shared aspect of the environment could have a significant effect on at least one child, when the effects on different children are not the same.

When we deal with a shared environmental factor that impacts different children in the family differently, it could be argued—and behavior geneticists do so argue—that the effect stems from the fact that some children are more genetically vulnerable to an environmental event than others. In the usual computations of heritability, such an effect would then be assigned to the G component of the equation, rather than to the environmental one. Surely, it is equally plausible that both G and E are important here. Risk factors, such as poverty, a father's unemployment, or a mother's depression, are indeed environmental conditions that are shared by all the children in a family. In large population studies they will rightly emerge as having a negative impact on children, even though some children are more vulnerable to them than others. In the extreme case, we could imagine that in every two-child family, one of the children would show the deleterious effects of poverty and the other would not (perhaps because of genetic differences between them). Across many families, there would be a very powerful effect of poverty and it would be rightly identified as a strong risk factor, even though the shared environment effect would be computed at zero. The obvious danger here is that low estimates for Es can be interpreted as meaning that family environmental conditions that children share do not have an impact on their development, whereas in fact the opposite can be true, and often is.

The findings from behavior genetics on shared and unshared environments have profound implications for the way we think about child-rearing practices and their effects. For one thing, they focus attention on sibling differences. This is something that traditional research on child-rearing—almost always involving only one child per family—did not deal with. It should be noted that there is nothing about the findings of these traditional studies that is invalidated by their having studied only one child. The connections identified between the parental inputs to this child and the child's characteristics can be reliable, replicable ones, even though if we had

studied a different parent-child pair in the same family we might have gotten a different constellation of parenting and outcomes. The picture emerging from aggregating data across a set of one-child cases is valid as well, though the findings are surely attenuated by the within-family sibling variation. Still, we get a less differentiated picture than the one that emerges from the study of siblings. Family systems theorists have alerted us to ''niche-picking'' by different children in a family—the effort of children to find distinctive roles. Evolutionary theorists have argued that there is natural competition among siblings for parental attention and other resources provided by parents. In short, there is reason to believe that there are forces motivating children to differentiate themselves from their siblings, and these may counterbalance, or transform, the effects of parental inputs that might otherwise function to make them the same. Of course, some of the differentiation between siblings can come directly from differential treatment by the parents, or it can stem from differential reactions by different children to the same parental inputs.

INTERPRETING PARENT-CHILD COVARIANCE

As noted above, quantitative geneticists have raised serious questions concerning the direction of effects when parental behaviors and child characteristics are found to be correlated. They point out that parent-child correlations could stem from genetic predispositions shared by parents and children that are directly transmitted from one generation to the next. In addition, evocative covariance occurs when children with different genetic predispositions elicit correspondingly different reactions from their parents. Thus, when a child is predisposed to be resistive or distractible and does not pay attention to the parent, the parent reacts by becoming more authoritarian, whereas a cooperative child will evoke a different reaction. (See Ge et al 1996, which shows clearly how the parenting by adoptive parents is affected by the predispositions of their adopted children.) Active covariance occurs when children select from a range of potential environmental influences only certain features with which to engage—certain TV programs, certain friends, certain sports—presumably on the basis of their own predispositions. Although children do not have the freedom to choose their parents, they do have some power to select which aspects of parental inputs they will attend to. Children with different genetic predispositions no doubt react differently to the same parental input, depending either on what they attend to, how they interpret their parents' actions, or what behavioral predisposition of their own has been triggered. In twin and adoption studies, all these forms of covariance between parent and child are thought to imply that genetics—either the child's own or the genes shared with parents—are driving the parental behaviors. For these reasons, it has seemed reasonable, in behavior genetic analyses, to assign parent-child covariances to the genetic component in the G + E = l00% equation.

I would argue that to assign parent-child covariance to G systematically underestimates the strength of parenting effects. It does so by ignoring the feed-back

loop whereby parents, in reacting to a given child's distinctive input, reciprocate with counter influences of their own.

The fact that parents respond differently to children with different predispositions is not in doubt, and it has been one of the contributions of behavior genetics to bring this fact into the foreground of our thinking. Socialization researchers, too, have for some time been centrally aware of this issue and in the past several decades have by no means ignored the problem of direction of effects. A great deal of effort has been devoted to examining the processes whereby parents and children influence one another. The predominant modern viewpoint among students of socialization is an interactionist one, in which it is assumed that in any ongoing relationship, each member of an interacting pair is a significant feature of the other's environment to which each must adapt. In addition, it has become clear that the developmental level of a child is a powerful determiner of what kind of socialization inputs a parent will provide and what kind of receptiveness, resistance, or negotiation the child will bring to a parent-child encounter. We cannot expect to find generalizations about the nature and effects of specific parent/child interactions that will span all the ages and stages of a child's development.

From an interactionist perspective, the idea that in a long-standing relationship such as the one between a parent and child, the child would be influencing the parent but the parent would not be influencing the child is absurd. While it is entirely reasonable to assign the child's part in parent-child covariance (i.e. evocative effects) to the genetic component, it is not reasonable to assign the reciprocal parent contribution to the child's genetics. The parent's response is surely a function not only of the child's initiative but also of the parent's genetics, learned modes of behavior, perceptions of the child's needs and characteristics, and socialization objectives. And, just as surely, the parent's response to the child's initiatives is a central element in the child's environment. Thus, to assign the whole of parent-child covariance to G is surely to overestimate G and underestimate E.

A recent study from the Rutter-Plomin research group in London (O'Connor et al 1998) beautifully identifies the contributions of correlated G and E factors to developmental outcomes. Using longitudinal data from the Colorado Adoption Study, these researchers identified two groups of adoptees: one at genetic risk for anti-social behavior (i.e. a history of anti-social behavior in the biological mother) and the other not at risk. At several points during the adoptees' childhood, both the children's characteristics and the adoptive parents' child-rearing methods were assessed. Findings were that children carrying a genetic risk for antisocial behavior were more likely to receive negative socialization inputs from their adoptive parents—an evocative effect. But parental negative behavior made an independent contribution to children's externalizing, over and above the children's genetic predispositions.

This study illustrates what an interactionist perspective would lead us to expect: Parent-child covariance reflects the reciprocal effects of both parent and child inputs to a relationship. The issue here is not to compare G and E effects to see which is stronger. Instead, it is to explore how they intersect or how one mediates the effect of the other. Such issues remain largely unexplored. The relative strength of each con-

tribution is difficult to assess and is almost entirely unknown in the large body of research literature on within-family socialization. The study by O'Connor et al illustrates the futility of efforts to compartmentalize the variance in children's characteristics into separate G and E components without getting independent measures of each. What this study shows is that G and E operate jointly to produce an outcome.

THE INTERACTION OF GENETIC AND ENVIRONMENTAL FACTORS

Interactions are found when a given environment has different effects on an organism, depending on the organism's genetic traits. Interactions are also seen when organisms with a given set of genetic traits react in one way under one set of environmental conditions, but another way under different environmental conditions. Plant biologists are able to point to dramatic examples, such as when there are two genetic strains of a grain, and strain 1 grows taller than strain 2 at high altitudes and shorter than strain 2 at low altitudes.

G × E Interactions in Animal Studies

A careful review of animal studies that looked for G × E interactions (Plomin 1986) reported that though interactions were sometimes found, they were not consistent within or across studies and accounted for only a small portion of variance. Since that review, some progress has been made in the difficult enterprise of mapping the complex processes that intervene between genotype and phenotype, and recently there has been some success in uncovering interactions with respect to these better-defined processes. In several mammalian species, it is now known that there are genetic factors underlying variation in ''reactivity,'' that is, in the tendency to become emotionally aroused and fearful. Different levels of reactivity in rats are associated with both neuroendocrine and behavioral functioning (Caldji et al 1998, Liu et al 1997). Reactive animals appear jittery and hesitate to explore novel environments. In Rhesus monkeys, a gene has been isolated one of whose alleles is associated with the emergence of a reactive temperament (Suomi 1999). It has been found that young animals carrying the ''reactive'' allele are particularly vulnerable to variations in early rearing experience. If they are subjected to maternal deprivation during their first six months (reared with peers but no adult females) their neuroendocrine functioning is affected and they display a variety of pathological symptoms into adulthood, including incompetence in social interactions, low status in peer groups, and incompetence in mothering their own offspring (Suomi 1997). By contrast, young animals who do not carry the genetic risk factor are much less affected by maternal deprivation. In current work, genetically reactive newborn monkeys are being cross-fostered to non-reactive mothers, and preliminary observations indicate that calm mothering does indeed buffer them from the development of strongly reactive behavior. Cross-fostering work with rodents is also showing the positive effects of rearing

genetically at-risk infants by a nurturant mother (Anisman et al 1998).We see here that the effects of a genetic predisposition are strongly seen under one set of environmental (rearing) conditions but not another.

G × E Interactions in Adoption Studies

Of course it is not possible to carry out systematic experiments of this sort with humans, but quantitative genetic studies can be used to test for G × E interactions. However, in such studies it is no longer possible to bypass measures of the environment and estimate E effects only as a residual after G effects have been estimated and subtracted out. Instead, there must be direct measures of both G and E. In most twin studies, the environments of twin pairs are too homogeneous to permit good estimates of G × E interactions, and there are difficulties in interpreting the differences between identical and fraternal twins in interaction terms. As Plomin said, " . . . it is difficult if not impossible to use the twin design to estimate the overall contribution of genotype-environment interaction to phenotypic variance" (Plomin 1986:96).[3] Since that time, there have been some innovations in utilizing twin studies to study interactions. One method is to use one twin's characteristic as an index to the co-twin's genetic risk; when the two are not highly concordant for the trait, their respective environments can then be examined for clues as to the origins of their non-genetic differences. Another method is simply to compare the heritability estimates found in two different environments.

In studies of adopted children, adoptive families vary with respect to the kind of environments they provide (though the range of environmental variation is usually consistently narrower than in unselected populations), and interactions can be effectively studied. In a large-scale study of adopted children in Finland (Tienari et al 1994), children with a schizophrenic biological parent were contrasted with adopted children who did not carry this genetic risk factor. It was found that the at-risk children were more likely to develop a range of psychiatric problems, but only if they were adopted into dysfunctional adoptive families. A study of adopted children whose biological parents did or did not have a history of criminality (Bohman 1996) yielded similar results: Among adoptees who carried a risk factor from their biological parents, those who had been adopted into dysfunctional homes were over three times more likely to become petty criminals than those whose adoptive parents had provided a stable, supportive environment.

These findings from adoption studies are consistent with studies of cross-generational transmission of psychiatric disorders (Ge et al 1996, Downey & Walker 1992). These studies point to a mediating role of parenting: Children whose parents suffer from a psychiatric disorder are usually no more likely than children with normal parents to develop psychiatric disorders, unless the children are exposed directly

[3]In studies of twins reared apart more variation in environments is of course usually present but the Ns for such studies are small and environmental information fragmentary.

to harsh parenting and/or an otherwise dysfunctional family environment by the parents who are rearing them.

Taken together, these studies indicate that genetic risks may or may not become manifest, depending on the quality of the parenting children receive. In other words, whatever genetic risks a child carries can require an environmental trigger to emerge into phenotypic expression. Well-functioning parents can buffer children against the emergence of negative genetic potentials.

Studies of Interactions with Temperament

It is possible to approximate the study of G × E interactions even when no direct information on children's genetics is available. Since several dimensions of temperament are known to have a significant genetic component,[4] researchers have identified children with different temperaments, and studied how they differ in the way they interact with their parents and in the impact parental inputs have on them. Children's temperamental characteristics appear to set the stage for the kind of bi-directional processes that will emerge between them and their parents (Collins et al 1999). Evidence has been emerging that a given parental practice can have different effects on children with different temperaments. Kochanska (1995, 1997a) studied the development of conscience in young children. She reported that for shy, temperamentally fearful children, parental power-assertion does not appear to promote conscience development—gentler techniques are called for. But with bold assertive children, effective parenting involves firmness, along with maternal responsiveness and the formation of a close emotional bond with the child. In a similar way, it has been found that for children who are initially difficult, impulsive, and/or resistive, parental firmness and restrictiveness are more important ingredients in preventing the subsequent development of externalizing behavior than is the case for children with easier temperaments (Bates et al 1998). Other studies finding interactions between children's temperament and parenting effects are those by Belsky et al (1997) and Deater-Deckard & Dodge (1997).

In Plomin's (1986) review of the studies on G × E interactions in children that were available at that time, significant interactions were found to be quite rare. It is possible at this time to be more positive—though guardedly so—concerning the prevalence and power of these interactions. They may be more prevalent with respect to personality dimensions and psychopathology than they are with respect to cognitive dimensions, but they obviously cannot be detected by using the traditional additive approach to partitioning variance between G and E. Indeed, the presence of interactions constitutes strong evidence against the validity of this approach. In molecular genetics, it is axiomatic that interactions are the rule, not the exception and that efforts to partition variance into the two traditional components are counterproductive.

[4]Temperament is currently defined as " . . . constitutionally based individual differences in reactivity and self-regulation . . . " (Rothbart & Ahadi 1994:54).

OVERVIEW

Behavior genetics studies have made substantial contributions to our understanding of the factors that underlie the variation among children in their intellectual and personality characteristics. Studies of twins and adopted children have shown beyond reasonable doubt that a wide range of children's attributes are influenced substantially by the genes they inherit from their biological parents. These studies first began to appear in the 1930s, and work done since that time has continued to confirm the power of genetic factors. The precise magnitude of the genetic contribution to a given trait, however, has proved to be difficult to establish: Heritability estimates vary widely, and indeed there is no reason to expect that there exists any one valid number for any given trait. Instead, heritability inevitably depends on the range of variation within a given sample being studied, and on the socio-cultural milieu in which the studied population lives. No single estimate can ever be taken as definitive.

I have argued that when genetic factors are strong, this does not mean that environmental ones, including parenting, must be weak. The relation between the two is not a zero-sum game, and the additive assumption is untenable. There are environmental factors that can affect a group or population without greatly rearranging the rank order of individuals within that group. In such a case, estimates of heritability can remain high while at the same time powerful environmental forces are at work. For this reason, it is not legitimate to extrapolate G or E estimates derived from a behavior genetic analysis to differences between groups (e.g. between races, social classes, or genders) that differ in their environmental milieu.

Experimental interventions have been designed to change children's behavior by means of changing the child-rearing practices of their parents. These intervention programs have amply demonstrated that parenting does have direct effects on how children behave, both inside and outside the home. When families are randomly assigned to an intervention group, the children show a reduction in problem behaviors by comparison with an untreated control group, and these effects are clearly independent of any genetic contribution to the outcome behavior being studied. Equally important is the presence of interactions between genes and environment, such that an environmental trigger is needed to evoke a genetic predisposition. Included here would be instances in which competent, supportive parenting protects a child from developing a dysfunction for which he or she is genetically predisposed. Such interactions have been largely ignored in traditional behavior genetic studies. What I argue here is that while the contribution of genetic factors to children's characteristics has been solidly documented in behavior genetics work, the contribution of environmental factors as derived from these studies has not.

A crucially important contribution of behavior genetics has been to draw our attention to the unlikeness of siblings. While we may have been marginally aware of sibling disparities, the traditional studies of childhood socialization included only one child per family, and there was an implicit assumption that parents treated their various children much alike and that the effects of what they did would be similar

for all their children. We must now seriously reexamine these assumptions. We now know that the correlations between siblings with respect to many of their characteristics are very low—indeed, sometimes lower than their genetic relatedness would predict. Is the unlikeness of siblings due to their being treated differently by their parents? To some extent, yes, though findings are not consistent across studies. What the behavior geneticists have shown is that the genetic predispositions of different children often drive the responses of parents, determining to some degree the kind of parenting a child will receive. Understandably then, behavior geneticists have assigned correlations between parent and child behaviors to the child's genetics, but I argue that this is a mistake, in that it ignores the return feed-back loop whereby a parent, whose behavior has been triggered by the child, responds with actions which in their turn influence the child. To ignore this reciprocal influence is to seriously underestimate parenting effects.

The unlikeness of siblings continues to be something we do not fully understand. It has been interpreted to mean that aspects of environment which siblings share—amount of inter-parental conflict, good or poor neighborhoods, poverty or affluence, level of parental education or the ''cultural'' level of the home environment, household organization or disorganization, the amount of good humor characterizing the family atmosphere—all these things must have very little influence on children's development. This interpretation flies in the face of the large body of research on risk factors, which repeatedly finds strong relationships between these aspects of family functioning and children's outcomes. I argue that the risk-factor findings are indeed valid, but that they need not have the same effects on all children in a family nor function to make siblings more alike. It begins to seem likely that there are strong factors pushing siblings toward differentiation from one another, including perhaps competition for parental attention or other resources, ''niche picking,'' counteridentification, and differential perceptions of the sibling relationship on the part of the participants in it. Such factors could function as counter forces, working against parental inputs that might otherwise make siblings more alike. But this is speculation. Much remains to be learned about this complex matter.

Many factors other than parents' actions influence how children grow and develop. As children grow beyond the preschool years, they are exposed more and more to other adult socialization agents (teachers, coaches) and, of course, to individual friends and larger peer groups. Within the matrix of factors that affect children's development, it is clear that parenting effects are real, though they often combine with genetic effects in influencing an outcome. Along with many other students of these phenomena, I urge that we give up the effort to partition the causal factors influencing children's development into two separate ''nature'' and ''nurture'' components, and that we abstain from asking ourselves which is more important. The two are inextricably interwoven all along the pathway from birth to maturity. So be it. Let us not underestimate either, but concentrate on the ways in which they function jointly.

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LITERATURE CITED

Angoff WH. 1988. The nature-nurture debate, aptitudes and group differences. *Am. Psychol.* 43:713–20

Anisman H, Zaharia MD, Meaney MJ, Merali Z. 1998. Do early-life events permanently alter behavioral and hormonal responses to stressors. *Int. J.Dev. Neurosci.* 16:149–64

Bates J, Pettit G, Dodge K, Ridge B. 1998. Interaction of temperamental resistance to control and restrictive parenting in the development of externalizing behavior. *Dev. Psychol.* 34:982–95

Baumrind D, Black AE. 1967. Socialization practices associated with dimensions of competence in preschool boys and girls. *Child Dev.* 38:291–327

Belsky J, Hsieh K, Crnic K. 1997. Mothering, fathering and infant negativity as predictors of boys' externalizing problems and inhibition. *Dev. Psychopathol.* 10:301–19

Block N. 1995. How heretability misleads about race. *Cognition* 56:99–128

Bohman M. 1996. Predispositions to criminality: Swedish adoption studies in retrospect. In *Genetics of Criminal and Anti-Social Behavior, Ciba Found. Symp. 194,* ed. GR Bock, JA Goode, pp. 99–114. Chickester/ New York: Wiley. 283 pp.

Brody G, Stoneman Z. 1994. Sibling relationships and their association with parental differential treatment. See Hetherington et al 1994, pp. 129–42

Cadoret RJ, Leve LD, Devor E. 1997. Genetics of aggressive and violent behavior. *Psychol. Clin. N. Am.* 20:301–22

Caldji C, Tannenbaum B, Sharma S, Francis D, Plotsky PM, Meaney MJ. 1998. Maternal care during infancy regulates the development of neural systems mediating the expression of fearfulness in the rat. *Proc. Nat. Acad. Sci.* 95:5335–40

Collins WA, Maccoby EE, Steinberg L, Hetherington EM, Bornstein M. 1999. Contemporary research on parenting: the case for nature *and* nurture. *Am. Psychol.* In press

Conger RD, Elder GH. 1994. *Families in Troubled Times: Adapting to Change in Rural America.* Hawthorne, NY: Aldine

Conger RD, Ge X, Elder GH, Lorenz FO, Simons R. 1994. Economic stress, coercive family process, and developmental problems of adolescents. *Child Dev.* 65:541–61

Deater-Deckard K, Dodge K. 1997. Spare the rod, spoil the authors: emerging themes in research on parenting. *Psychol. Inq.* 8:230–35

Dishion TJ, Patterson GR, Kavanagh K. 1992. An experimental test of the coercion model: linking theory, measurement, and intervention. In *The Interaction of Theory and Practice: Experimental Studies of Interventions,* ed. J McCord, R Trembly, pp. 253–82. New York: Guilford. 29 pp.

Downey G, Walker E. 1992. Distinguishing family-level and child-level influences on

the development of depression and aggression. *Dev. Psychopathol.* 4:81–96

Dunn J, McGuire S. 1994. Young children's nonshared experiences: a summary of studies in Cambridge and Colorado. See Hetherington et al 1994, pp. 111–28

Dunn J, Plomin R. 1986. Determinants of maternal behavior toward three-year-old siblings. *Br. J. Dev. Psychol.* 57:348–56

Duyme M, Dumaret AC, Stanislaw T. 1999. How can we boost IQs of ''dull'' children?: a late adoption study. *Proc. Nat. Acad. Sci.* In press

Elder GH. 1974. *Children of the Great Depression.* Chicago: Univ. Chicago Press. 400 pp.

Elman JL, Bates EA, Johnson MH, Karmiloff-Smith A, Parisi D, Plunkett K. 1996. *Rethinking Innateness: A Connectionist Perspective on Development.* Cambridge, MA: MIT Press

Feldman MW, Lewontin RC. 1975. The heritability hangup. *Science* 190:1163–68

Flynn JR. 1999. Searching for justice: the discovery of IQ gains over time. *Am. Psychol.* 54:5–20

Flynn JR. 1987. Massive IQ gains in 14 nations: what IQ tests really measure. *Psychol. Bull.* 101:171–91

Forehand R, Wells KC, Griest DL. 1980. An examination of the social validity of a parent training program. *Behav. Ther.* 11:488–502

Ge X, Conger R, Cadoret R, Neiderhiser J, Yates W, et al. 1996. The developmental interface between nature and nurture: a mutual influence model of child antisocial behavior and parent behavior. *Dev. Psychol.* 32:574–89

Goodman R. 1991. Growing together and growing apart: the non-genetic forces on children in the same family. In *The New Genetics of Mental Illness,* ed. R McGuffin, R Murray, pp. 212–24. Oxford: Oxford Univ. Press

Gottlieb G. 1995. Some conceptual deficiencies in ''developmental'' behavior genetics. *Hum. Dev.* 38:131–41

Harris JR. 1998. *The Nurture Assumption: Why Children Turn Out the Way They Do.* New York: Free Press. 462 pp.

Hernnstein RJ, Murray C. 1994. *The Bell Curve: Intelligence and Class Structure in American Life.* New York: Free Press

Hetherington EM, Reiss D, Plomin R, eds. 1994. *Separate Social Worlds of Siblings.* Hillsdale, NJ: Erlbaum. 232 pp.

Hetherington EM, Henderson SH, Reiss D. 1999. *Adolescent Siblings in Stepfamilies: Family Functioning and Adolescent Adjustment.* Monog. Soc. Res. Child Dev. In press

Hoffman LW. 1991. The influence of the family environment on personality: accounting for sibling differences. *Psychol. Bull.* 110:187–203

Kochanska G. 1995. Children's temperment, mothers' discipline, and security of attachment: multiple pathways to emerging internalization. *Child Dev.* 66:597–615

Kochanska G. 1997a. Multiple pathways to conscience for children with different temperaments: from toddlerhood to age five. *Dev. Psychol.* 33:228–40

Kochanska G. 1997b. Mutually responsive orientation between mothers and their young children: implications for early socialization. *Child Dev.* 68:908–23

Kochanska G, Thompson RA. 1997. The emergence and development of conscience in toddlerhood and early childhood. In *Parenting and Children's Internalization of Values,* ed. JE Grusec, L Kuczunski pp. 53–77. New York: Wiley

Liu D, Diorio J, Tannenbaum B, Cladji C, Francis D, et al. 1997. Maternal care, hippocampal glucocorticoid receptors and hypothalamic-pituitary-adrenal responses to stress. *Science* 277:1659–62

Loeber R, Dishion TJ. 1983. Early predictors of male delinquency: a review. *Psychol. Bull.* 94:68–99

Maccoby EE, Martin JA. 1983. Socialization in the context of the family: parent-child interaction. In *Handbook of Child Psychology,* Vol. 4, ed. PH Messen, EM Herrington, 4:1–102. New York: Wiley. 4th ed. pp.1–102

McLoyd VC. 1998. Socioeconomic disadvantage and child development. *Am. Psychol.* 53:185–204

Miles D, Carey G. 1997. Genetic and environmental architecture of human aggression. *J. Pers. Soc. Psychol.* 72:207–17

Neville HJ. 1996. Developmental specificity in neurocognitive development in humans. In *The Cognitive Neurosciences,* ed. M Gazzaniga, pp. 219–31 Cambridge, MA: MIT Press

O'Connor TG, Deater-Deckard K, Fulker D, Rutter M, Plomin R. 1998. Genotype-environment correlations in late childhood and early adolescence: antisocial behavioral problems and coercive parenting. *Dev. Psychol.* 34:970–81

Patterson GR. 1975. Multiple evaluations of a parent-training program. In *Applications of Behavior Modification,* ed. T Thompson, W Dockens, pp. 299–322. New York: Academic

Patterson GR, Bank LI. 1989. Some amplifying mechanisms for pathologic processes in families. In *Systems and Development: The Minnesota Symposium on Child Psychology,* ed. MR Gunnar, E. Thelen pp. 167–209. Hillsdale, NJ

Patterson GR, Forgatch M. 1995. Predicting future clinical adjustment from treatment outcome and process variables. *Psychol. Assess.* 7:275–85

Pettit GS, Bates JE, Dodge KA. 1997. Supportive parenting, ecological context, and children's adjustment: a seven-year longitudinal study. *Child Dev.* 68:908–23

Plomin R. 1986. *Development, Genetics, and Psychology.* Hillsdale, NJ: Erlbaum. 372 pp.

Plomin R, Bergman CS. 1991. The nature of nurture: genetic influences on "environmental" measures. *Behav. Brain Sci.* 14:1–15

Plomin R, Chipuer HM, Neiderhiser JM. 1994. Behavioral genetic evidence for the importance of nonshared environment. See Hetherington et al 1994, pp. 1–31

Plomin R, Daniels D. 1987. Why are children in the same family so different from each other? *Behav. Brain Sci.* 10:1–16

Plomin R, DeFries J, Fulker D. 1988. *Nature and Nurture During Infancy and Early Childhood.* Pacific Grove, CA: Brooks-Cole

Price RA, Charles MA, Pettit DJ, Knowler WC. 1993. Obesity in Pima Indians: large increases in post- World War II birth cohorts. *Am. J. Phys. Anthropol.* 92:473–79

Ravussin E, Bennett PH, Valencia ME, Schulz LO, Esparaz J. 1994. Effects of a traditional lifestyle on obesity in Pima Indians. *Diabetes Care* 17:1067–74

Reiss D. 1997. Mechanisms linking genetic and social influences in adolescent development: beginning a collaborative search. *Curr. Dir. Psychol. Sci.* 6:100–6

Reiss D, Hetherington EM, Plomin R, Howe GW, Simmens SJ, et al. 1995. Genetic questions for environmental studies: differential parenting and psychopathology in adolescence. *Arch. Gen. Psychol.* 52:925–36

Reiss D, Neiderhiser J, Hetherington EM, Plomin R. 1999 *The Relationship Code: Deciphering Genetic and Social Patterns in Adolescent Development.* Cambridge, MA: Harvard Univ. Press. In press

Rose R. 1995. Genes and human behavior. *Annu. Rev. Psychol.* 46:625–54

Rosenthal R. 1999. *Discussion on effect sizes at symposium on, "Does Child Care Quality matter?"* Presented at Biennial Meet. Soc. Res. Child Dev., April, Albuquerque, NM

Rothbart M, Ahadi S. 1994. Temperament and the development of personality. *J. Abnorm. Psychol.* 103:55–66

Rowe D. 1994. *The Limits of Family Influence: Genes, Experience, and Behavior.* New York: Guilford

Rowe D. 1983. A biometrical analysis of perceptions of family environment: a study of twin and singleton sibling kinship. *Child Dev.* 54:416–23

Rutter M, Silberg J, O'Connor T, Simonoff E. 1999. Genetics and child psychiatry: I. Advances in quantitative and molecular genetics. *Child Psychol. Psychol.* 40:3–18.

Scarr S. 1992. Developmental theories for the 1990's: development and individual differences. *Child Dev.* 63:1–19

Scarr S, Grajek S. 1982. Similarities and differences among siblings. In *Sibling Relationships: Their Nature and Significance Across the Lifespan,* ed. ME Lamb, B Sutton-Smith, pp. 357–82. Hillsdale, NJ: Erlbaum

Stoolmiller M. 1999. Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior genetic adoption studies. *Psychol. Bull.* In press

Suomi SJ. 1999. A biobehavioral perspective on developmental psychopathology: excessive aggression and serotonergic dysfunction in monkeys. In *Handbook of Developmental Psychopathology,* ed. AJ Samaroff, M Lewis, S Miller. New York: Plenum. In press

Suomi SJ. 1997. Long-term effects of different early rearing experiences on social, emotional and physiological development in non-human primates. In *Neurodevelopmental Models of Adult Psychopathology,* ed. MS Kesheven, RM Murra, pp. 104–16. Cambridge: Cambridge Univ. Press

Tienari P, Wynne LC, Moring J, Lahti I, Naarala M, et al. 1994. The Finnish adoptive family study of schizophrenia: implications for family research. *Br. J. Psychiatry* 23(Suppl. 164):20–26

Turkheimer E. 1998. Heritability and biological explanation. *Psychol. Rev.* 105:1–10

Van den Boom DC. 1994. The influence of temperament and mothering on attachment and exploration: an experimental manipulation of sensitive responsiveness among lower-class mothers with irritable infants. *Child Dev.* 65:1457–77