Debates

Nature, nurture, and social development*

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Abstract

It is time to bring nature (genetics) together with nurture (environment) in the study of social development. Following a brief overview of behavioral genetic theory and methods, three examples are described of new genetic research especially relevant to social developmentalists. First, initial research findings on three key domains of social development (attachment, empathy, and social competence) suggest that genetic factors contribute to individual differences in social development. Second, research on widely used measures of social environment implicates a genetic contribution, which opens up new directions for research at the interface of nature and nurture in social development. Third, by the turn of the century, it is predicted that behavioral genetic research will be conducted using DNA markers that assess genetic variation among individuals directly rather than resorting to indirect estimates based on twin and adoption methods. This will revolutionize behavioral genetic research and make it more accessible and applicable to developmentalists. As a first step in the direction of behavioral genetics, social developmentalists are encouraged to include siblings in their research.

Keywords: Behavioral genetics; molecular genetics; environment; relationships.

The biennial conferences of the Society for Research in Child Development can be used as a gauge of trends in developmental research. As usual, social development was well represented at the Sixtieth Anniversary Meeting, 25–28, 1993, in New Orleans, Louisiana. The index to the meeting's program serves as a rough guide to topical coverage. In the index, 22 topics had four or more lines (at least 30 entries) which represent major research topics. Half of these topics are central to social development: attachment, conflict, family, interaction, parent–child, parenting, peer relations, peers, relationships, siblings, and social cognition. Other major topics are also related to social development such as affect, emotion, and temperament.

Comparing the 1991 and 1993 meetings, the most striking trend that I detected from the 1991 to the 1993 meeting was a sharp rise in the number of presentations

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related to genetics. At the 1991 meeting, there were only a handful of papers or posters on genetics, other than the presidential address given by Sandra Scarr and published in *Child Development* (Scarr, 1992). In contrast, more than 35 papers and posters on genetic research were presented at the 1993 meeting. These presentations included symposia on genetics and activity level (Saudino, 1993), competence (Neiderhiser & McGuire, 1993), the Minnesota adoption study (DeBerry & Waldman, 1993), and nonshared environment (McGuire, 1993). Two of the four invited Master Lectures focused on nature and nurture – in language development (Bates, 1993) and developmental behavioral genetics (Plomin, 1993). In addition, genetics played a prominent role in the presidential address by Robert Emde (1993), whose own work includes a longitudinal twin study of socioemotional development during the second year of life (Emde *et al.*, 1993).

A watershed for genetic research in development was an invited symposium, with an audience approaching a thousand, on the topic of genetic and environmental influences in development (Garcia-Coll, 1993). Frances Horowitz (1993) began by describing the history of genetic research in development. Sandra Scarr (1993) responded to criticisms of her 1991 presidential address. Felton Earls (1993) emphasized the need to understand the neurobiological processes that link genes and developmental outcomes. Michael Rutter (1993) ended the symposium with a plea to put the nature vs. nurture controversy behind us and to 'bring nature and nurture together in the study of development.'

Genetics seems to be accepted increasingly not only by developmentalists but also by psychologists in general. For example, as part of the centennial conference of the American Psychological Association in 1992, a committee was formed under the auspices of the APA Board of Convention Affairs to select two themes that would best represent the past, present, and future of psychology. One theme was consciousness, which encompasses the past century's shift from philosophy to psychology, the past decade's cognitive revolution, and the present 'Decade of the Brain.' The other theme was behavioral genetics. A series of symposia were convened at the centennial conference to provide an overview of the past, a summary of the present, and a glimpse of the future of genetic research in psychology and has been organized as a book (Plomin & McClearn, 1993).

As documented in this volume, the vast majority of genetic research in psychology has focused on three domains: cognitive abilities, personality, and psychopathology. The purpose of this article is to suggest that social development represents an exciting new area for consideration of nature as well as nurture, as illustrated by three recent developments. First, initial attempts to apply behavioral genetic strategies to key domains within social development suggest genetic influence. Second, recent research implicates genetic involvement in measures of psychological environments which largely involve social relationships and interactions. The third example seems far-fetched but will, I predict, be part of the armamentarium of researchers in social development by the turn of the century: molecular genetics. Before describing these three examples, a few preliminary words about behavioral genetic theory and methods is in order.

Behavioral Genetics

It is critical to recognize that behavioral genetic research is limited to the investigation of individual differences within a species, not species-typical development.

Its focus is on genetic and environmental factors that make a difference in behavioral phenotypes. That is, its genetic focus is on DNA differences among individuals, not the vast majority of DNA that is the same for all members of a species. Its environmental focus is on environmental differences, not the many critical environmental factors such as nutrients, light, and oxygen that do not vary or are functionally equivalent among members of a species. Although it is important to consider species-wide themes as well as variations on these themes (Mayr, 1982; McCall, 1981; Scarr, 1992), it is also important to emphasize the distinction between them because the processes involved in the development of individual differences can differ from the processes responsible for species-typical development. For example, the young of mammalian species require caregivers and form special relationships with them, a universal feature of our species and other mammals that is surely the result of evolution. However, there are individual differences in attachment relationships within our species. These individual differences may be influenced by genetic differences between infants or between caregivers, but it is also possible that they are due entirely to nongenetic factors. In summary, behavioral genetics only addresses phenotypic differences among individuals in a population; its goal is to ascribe phenotypic differences to genetic and environmental sources of variance.

Behavioral genetic theory and methods may now be sufficiently familiar to developmentalists that it is no longer necessary to describe them in detail. Behavioral genetics consists of quantitative genetic theory and methods (Falconer, 1989) as applied to behavior (Plomin *et al.*, 1990). The methods decompose phenotypic variation in a population into genetic and environmental components of variance. Quantitative genetic methods are quasi-experimental designs that control genetic relatedness while studying environmental influence and that control environmental relatedness while studying the effects of genetic relatedness. They can be viewed as simple natural experiments. For example, identical twins are twice as similar genetically as fraternal twins. Shared environmental influences are roughly similar for the two types of twins: Both identical and fraternal twins are born from the same womb, they are the same age and gender (when same-gender fraternal twins are studied), and they grow up in the same family. If heredity is important for a particular trait, identical twins must be more similar than fraternal twins because of the twofold greater genetic similarity of identical twins. Twin resemblance not explained by heredity is attributed to shared rearing environment. Another quantitative genetic method is the adoption design which assesses genetic influence from resemblance between genetically related individuals adopted apart (such as siblings adopted apart) and from comparisons between genetically related relatives versus adoptive relatives (such as nonadoptive siblings versus adoptive siblings). The correlation for genetically unrelated children adopted into the same adoptive homes provides a direct test of the influence of shared rearing environment.

As in any experiment, and especially in quasi-experimental designs, possible confounding factors must be considered. For the twin method, a possible confounding factor is the so-called equal environments assumption – that is, the assumption that the two types of twins share environmental influences to a similar extent. However, research on the equal environments assumption suggests that it is a reasonable assumption (Plomin *et al.*, 1990). For the adoption design, selective placement may be a confounding factor. That is, adopted children may be

placed with adoptive parents who are similar to their birth parents. If selective placement occurs, it can be taken into account in adoption study estimates of genetic and environmental influence.

The theory and methods of behavioral genetic are presented elsewhere in overview (Plomin, 1990a) and in detail (Plomin *et al.*, 1990).

Genetics and Social Development Phenotypes

Most genetic research in psychology continues to focus on cognitive abilities, personality, and psychopathology. Not coincidentally, these are the three domains of psychology that have always shown the most interest in individual differences. Nearly ubiquitous evidence for modest genetic influence continues to emerge from these fields (Plomin & McClearn, 1993). In contrast, little is known about the origins of individual differences in traditional areas of social development. In part, this lack of genetic research in social development is due to the fact that, unlike the domains of cognitive abilities, personality, and psychopathology, much research and theory on social development has focused on species-typical developmental themes rather than individual variations on these normative themes. For reasons described earlier, unless a field displays an interest in individual differences, genetic research on the origins of individual differences seems irrelevant.

Social development is not alone in this respect. For example, the current edition of the *Handbook of Child Psychology* (Mussen, 1983) presents a representative sampling of theory and research in the field of developmental psychology. Of the 2,926 pages of text (excluding references, outlines, and notes), 78% are devoted predominantly (i.e., more than half the page) to normative or group difference approaches. Of the 48 chapters, 41 include more pages on group differences than on individual differences. Moreover, 19 chapters include not a single page on individual differences, and 4 other chapters consider individual differences on 2% or fewer of their pages. The 23 chapters primarily include neuroscience, perception, learning, cognition, language, and intervention.

Why study individual differences? First, individual differences exist and thus theories must be able to account for the 'very standard deviation' as well as averages. Second, it can be argued that developmental issues of greatest relevance to society are issues of individual differences (Plomin, DeFries, & Fulker, 1988). Third, the description and explanation of group differences bear no necessary relationship to those of individual differences, as discussed earlier.

Why is the study of individual differences neglected? One reason is historical happenstance. William James' (1990) *Principles of Psychology* has had a strong influence on psychologists' categories of thought. Unfortunately, James seldom wrote about individual differences. His 1,278-page tome ranges across most of the domains of psychology, but other than a couple of scattered bits James essentially ignored individual differences. A practical reason for the neglect of individual differences is that the study of individual differences is more demanding in terms of psychometrics and statistics than is the study of normative development or group differences. The major psychometric issue is reliability of measurement. Because calculation of an average dilutes the impact of errors of measurement, reliability is not of major concern in studies of group differences. In contrast, reliability of measurement is of paramount concern in the study of individual differences. A more subtle, and paradoxical, obstacle to the study of

individual differences is that the statistics of individual differences are readily translated into the amount of variance explained. In contrast, analyses of group differences focus on statistical significance. The focus on variance explained is often depressing because it rudely reminds us, for example, that the ubiquitous correlation of .30 explains less than 10% of the variance. However, despite its bitter taste, this is important medicine. Psychology's preoccupation with statistical significance has left a mountain of statistically significant results that are insignificant in terms of effect size. The emphasis on variance explained is a virtue of individual differences analyses: Any mean group difference can also be converted to a statement of effect size (Cohen, 1988) and group differences rarely account for as much as 10% of the variance.

Another barrier to the study of individual differences is the concern that individual differences violate the fundamental belief that all persons are created equal. By 'equality,' John Locke clearly meant political equality – equality in opportunity and before the law – not an absence of individual differences (Loehlin, 1983). Individuality is a fact. It is also the foundation for the dignity of humankind: We are not interchangeable. The real danger lies in treating people on the basis of group membership – ethnic group, gender, age – when individual differences within these groups far exceed average differences between the groups. A final impediment to studying individual differences is that individual differences research often seems atheoretical. Without worrying about the philosophical intricacies of the word *theory*, from the pragmatic view of a behavioral researcher theories should clarify our thinking by describing, predicting and explaining behavior. At the very least, theories should be descriptive, organizing and condensing existing facts in a reasonable, internally consistent manner. Theories should also make predictions concerning phenomena not yet investigated and allow clear tests of these predictions to be made. At their best, theories explain phenomena as well as describe and predict them. In all of these senses, quantitative genetics can be seen as the basis for a powerful theory of individual differences (Plomin *et al.*, 1988).

Once a researcher's focus shifts to individual differences, descriptive and predictive questions invariably lead to questions about the origins of individual differences. Such questions put a researcher at risk for behavioral genetics. Three examples of domains within social development that have traditionally been approached from a normative perspective but have recently begun to consider individual differences are attachment, empathy, and social competence. Not coincidentally, the first genetic studies in each of these domains have recently been reported. In each case, some genetic influence has been suggested.

Attachment

Bowlby (1969), influenced by Lorenz' (1935/1970) work on imprinting, originally discussed attachment as a species-typical developmental process. A first step in the direction of individual differences is often typological and only later are quantitative dimensions recognized. For attachment, typologies still predominate – avoidant, secure and resistant types (Ainsworth, Blehar, Waters, & Wall, 1978).

Although these attachment types are generally viewed as an index of the parent–child dyad, the contribution of child temperament has been investigated (e.g. Chess & Thomas, 1982; Goldsmith & Campos, 1982). Several studies have

suggested that security of attachment *per se* is not influenced by temperament but that temperament is related to the child's level of emotional expression in the stressful Strange Situation (e.g., Belsky & Rovine, 1987). Although the role of child temperament remains uncertain (e.g. Gunnar, Mangelsdorf, Larson, & Hertsgard, 1989), even if child temperament is unrelated to attachment types other genetically influenced characteristics of children could make a genetic contribution to attachment types.

A recent twin analysis of attachment pooled data for 56 twin pairs from three small studies of twins between 12 and 22 months of age assessed in the Strange Situation (Ricciuti, 1993). Although the total sample size is small, the results are particularly interesting in relation to the hypothesis that temperament is not related to attachment security *per se* but rather to emotional expressiveness. Security of attachment did not show genetic influence, but a variable representing the A1–B2/B3–C2 dichotomy showed significant genetic influence. The author hypothesized that this genetic influence may be due to emotional expression and regulation. Multivariate genetic analyses (described later) could be used to investigate the extent to which genetic influence on the A1–B2/B3–C2 dichotomy is due to genetic influence on temperamental dimensions such as emotional reactivity and regulation.

Empathy

Like attachment, theories of empathy are largely normative theories (e.g., Hoffman, 1975), and much research on the development of empathy consists of charting average empathic function across age (Radke-Yarrow, Zahn-Waxler, & Chapman, 1983). However, research on individual differences is emerging. For example, rank orders in individual differences in empathy have been shown to be relatively unstable during the second year of life (e.g., Cummings, Hollenbeck, Iannotti, Radke-Yarrow, & Zahn-Waxler, 1986).

The first twin study of empathy is part of an ongoing collaborative longitudinal twin study of socioemotional development of nearly 400 pairs of twins known as the MacArthur Longitudinal Twin Study (Emde *et al.*, in press). Children's reactions to simulations of distress in others, videotaped at home and laboratory settings, were assessed for each member of the twin pairs. An unrotated principal component used as a general index of empathic responding showed significant genetic influence at 14 months but not at 20 months (Plomin, Emde, Braungart, Campos, Corley, Fulker, Kagan, Reznick, Robinson, & Zahn-Waxler, 1993). An analysis of specific components of prosocial behavior found especially strong genetic influence for the affective component of emotional concern (Zahn-Waxler, Robinson, & Emde, 1992), which may again reflect temperamental emotionality. Mothers' ratings of their children's prosocial orientation also showed significant genetic influence.

A critical point for developmentalists is that genetic factors can contribute to change from age to age, not just to continuity (Plomin, 1986). Variables such as empathy for which the rank order of individual differences changes substantially from age to age provide an opportunity to investigate genetic contributions to developmental change. However, from 14 to 20 months change scores and more sophisticated model-fitting analyses of change yielded no evidence for genetic influence for a general factor of empathy, although several other socioemotional

variables in the study show significant genetic change (Plomin *et al.*, 1993). It is possible that the low frequency of empathic responding makes the measure unreliable during infancy. The MALTS twins are being studied longitudinally through childhood; it will be interesting to consider the developmental course of genetic influence in this domain.

Social competence

Theories of self-worth and competence tend to be normative theories, although some strands of empirical research in this domain have long considered individual differences (Harter, 1983). Research on the etiology of individual differences in children's self-worth and competence has focused on the family environment (Harter, 1983). It is surprising that the possibility of genetic influence has not been considered previously because it seems likely that genetic influence on personality, psychopathology, and cognitive abilities might lead to genetic influence on perceived competence. Research on the genetic origins of this domain is especially timely given recent criticism that behavioral genetic researchers have not investigated domains such as social competence which are thought to be heavily influenced by nurture rather than nature (Hoffman, 1991).

The first twin and adoption studies in this domain have been reported for teacher, parent, and self ratings in adolescence (McGuire, Neiderhiser, Reiss, Hetherington, & Plomin, in press; Neiderhiser, McGuire, Plomin, Hetherington, & Reiss, 1993) and for teacher and parent ratings in middle childhood (Neiderhiser & McGuire, 1993). These reports come from the Nonshared Environment in Adolescent Development (NEAD) project (Reiss, Plomin, Hetherington, Howe, Rovine, Tryon, & Stanley, 1994) and the Colorado Adoption Project (CAP; DeFries, Plomin, & Fulker, in press). NEAD consists of 720 never divorced and step families with same-sex sibling pairs (identical twins, fraternal twins, full siblings, half siblings and unrelated step siblings) ranging in age from 10 to 18 years. Begun in 1975, CAP is a longitudinal adoption study consisting of 245 adoptive families and 245 matched nonadoptive families in which the birth parents and adoptive parents of the adoptees and the parents of the nonadopted children have been administered a battery of behavioral measures (DeFries, Plomin, & Fulker, in press). The adopted and nonadopted children, their siblings, and their home environments are studied yearly from infancy through adolescence.

Two striking findings emerged from these studies, both directly contradicting the hypotheses of Hoffman (1991). First, significant genetic influence was found for most dimensions in these domains. Second, shared family environment was largely nonsignificant.

Research in behavioral genetics goes beyond these rudimentary analyses of the magnitude of nature and nurture effects. One example is multivariate genetic analysis which decomposes the covariance between variables into genetic and environmental sources of covariance (Plomin *et al.*, 1990). Three types of multivariate genetic analysis were conducted in these studies of social competence. The first broached the topic of mediators of genetic influence on competence. Multivariate genetic analyses indicated that genetic influence on social competence is completely independent of genetic influence on sociability and that genetic influence on cognitive competence is largely independent of genetic influence on

verbal ability (McGuire *et al.*, in press; Neiderhiser *et al.*, 1993). A second type of multivariate genetic analysis showed that genetic effects on teacher ratings of competence substantially overlap with genetic effects on parental, especially maternal, ratings (Neiderhiser *et al.*, 1993). A third type of multivariate genetic analysis assessed the genetic contribution to longitudinal stability for teacher ratings of competence from first to third grade. These analyses indicated that genetic factors are almost entirely responsible for phenotypic stability of about .40 across the two years (Neiderhiser & McGuire, in press).

It should be noted that although these first studies of attachment, empathy, and social competence suggest genetic influence, it is still very early days in genetic research on social development. Genetic designs do not always find genetic influence. For example, the first twin study of neonatal temperament shows no genetic influence (Riese, 1990). Also, positive low-arousal emotions such as smiling show no genetic influence in several studies in infancy (Plomin, 1987). It remains to be seen whether subsequent studies also find genetic influence in the key domains of social development or in other domains that have not yet been investigated from a behavioral genetic perspective.

Relationships and Interactions

The excitement that emanates from genetic research springs in part from the synergism of looking at old issues in new ways. One example lies in the hyphen in the phrase nature-nurture. Some of the most interesting questions about genetics involves the environment and some of the most interesting questions about the environment involves genetics (Plomin, in press a).

For example, a new area of considerable relevance to social developmentalists involves genetic analysis of measures of psychological environments (Plomin & Bergeman, 1991; Plomin & Neiderhiser, 1992; Plomin, in press a). The relevance of this topic to social developmentalists lies in the fact that most measures of psychological environments involve relationships and interactions. Measures of the family environment are largely measures of family relationships and interactions. Measures outside the family are also usually socially oriented, such as measures of the classroom environment, peer relations, and social support. Even measures of life events and stress used in thousands of studies largely involve social events and social stress.

At first, genetic analyses of environmental measures sound paradoxical because behaviorism has conditioned psychologists to think of the environment as something 'out there' independent of the individual. To the contrary, measures of the psychological environment usually involve behavior. For example, in social development environmental measures often include measures of parental behavior. Differences among parents in their behavior to their children could in part be due to genetically influenced characteristics of the parents, such as their personality. Parental behavior might also reflect genetically influenced characteristics of the parents, such as their personality. In other words, genetic differences among individuals can contribute to their experiences. The hypothesis of genetic influence can be tested by treating the environmental measure as a dependent measure and asking via twin and adoption studies whether genetic factors contribute to the observed variance on the environmental measure.

A score of genetic studies during the past decade consistently implicate a

genetic contribution to such measures of the environment. Each of the types of environmental measures mentioned above has been shown to involve genetic influence. Especially strong genetic influence has been found for characteristics of peer groups. Genetic influence emerges from adoption as well as twin studies, from child-based as well as parent-based genetic designs, from research employing observations as well as self-reports, and from research on extrafamilial environments as well as familial environments (Plomin, in press a). On average environmental measures yield heritability estimates of about .30, suggesting that more than a quarter of the variance of environmental measures can be accounted for by genetic differences (Plomin & Bergeman, 1991). It is interesting that some environmental measures show less genetic influence than others. For example, children's perceptions of parental control show less genetic influence than other dimensions of parenting (Plomin, in press a). Also, on measures of life events, 'controllable' items such as conflict with children show greater genetic influence than 'uncontrollable' items such as illness of children (Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990).

The initial review of this research (Plomin & Bergeman, 1991) appeared with 30 commentaries and a response to the commentaries. Of the 30 commentaries, 19 explicitly agreed with the major conclusion and 6 implicitly agreed. Only 5 explicitly disagreed, and these denied that it is possible to detect genetic influence on anything, let alone measures of the environment.

The genetic studies described in this review (Plomin & Bergeman, 1991) each included just one or two environmental measures. The NEAD study mentioned earlier focused on the systematic examination of diverse measures of the family environment (Plomin, Reiss, Hetherington, & Howe, in press). In a report of scores of interview and questionnaire measures, three quarters of the measures showed significant genetic effects in model-fitting analyses, and the average heritability estimate was about .25. As in previous studies, measures of parental behavior generally showed genetic effects, whether rated by parents or by children. In addition, the social environment provided by siblings showed genetic effects. In summary, this first systematic genetic exploration of diverse measures of the family environment confirmed the hypothesis of genetic involvement in widely used measures of the environment.

One implication of such findings is that environmental measures cannot be assumed to be environmental just because they are called *environmental.* Research to date suggests that it is safer to assume that ostensible measures of the environment include genetic influence.

Another implication is that, if environmental measures as well as outcome measures show genetic influence, it seems reasonable to consider the possibility that predictions of outcomes from environmental measures may be mediated genetically:

> The blinkered attitude whereby all parent–child correlations are interpreted in environmental terms thus needs to give way to a willingness to consider genetic mediating influences as well, and once that step has been taken, some rather surprising possibilities emerge. (Schaffer, 1992, p. 47)

For example, genetic factors have been implicated in the relationship between home environment and children's later development (Braungart, in press; Braungart, Fulker, & Plomin, 1992). That is, about half of the correlation

between measures of the home environment and children's later cognitive development appears to be mediated by genetic factors.

A key question is how measures of the environment come to show genetic influence. The first attempts to search for mediators of genetic influence have led to the hypothesis that, although some genetic influence on measures of the environment can be explained by genetic influence on traditional traits such as personality, most genetic influence on environmental measures cannot be explained in this way (Plomin, in press a). This suggests that genetic influence on environmental measures may be more than a matter of contamination by genetically influenced traits. Experience itself – how we interact with our environments – may be influenced by genetic factors. Children actively select, modify, and create the environments they experience, at least in part for genetic reasons (Scarr, 1992). Consideration of these active correlations between nature and nurture takes up the challenge of Anne Anastasi in her 1958 APA presidential address to move beyond questions of how much variance is accounted for by genetic and environmental factors to the question of how genetic and environmental variables co-act during development, the developmental duet in which genotypes become phenotypes.

Molecular Genetics

We are at the dawn of a new era in which molecular genetic techniques will revolutionize genetic research on behavior by identifying specific genes that contribute to genetic variance in behavioral dimensions and disorders (Aldhous, 1992; Plomin, 1990b, in press b). It was only ten years ago that the now-standard techniques of the 'new genetics' were first employed to identify genes responsible for single-gene disorders. As described elsewhere (e.g., Plomin *et al.*, 1990), the discovery of restriction enzymes led to recombinant DNA, the ability to sequence DNA, and the discovery of thousands of new DNA markers. DNA markers are genetic differences among individuals that involve DNA itself rather than gene products such as the blood groups and other traditional genetic markers. These new DNA markers have been used to identify chromosomal regions and, eventually, to isolate genes and gene products for single-gene disorders.

Notable early successes in identifying genes and gene products in the human species include cystic fibrosis and Duchenne muscular dystrophy. These are dichotomous traits, like Mendel's smooth vs. wrinkled seeds, in which one gene is necessary and sufficient to explain the observed difference. Several thousand single-gene disorders, most very rare, have been reported, and many affect behavior (McKusick, 1990). However, this by no means implies that behavior is determined by a single gene. A 'mutation' in any one of hundreds of parts of an automobile can impair its functioning, yet none of those parts are in themselves responsible for a fully functioning automobile. For example, children with the recessive genetic disorder phenylketonuria (PKU) are mentally retarded unless a dietary intervention is imposed early in life. Children with a double dose of the recessive allele are unable to metabolize phenylalanine. Without a diet low in phenylalanine, phenylalanine accumulates to levels that are damaging to the developing brain. Although the effect of this single gene on IQ is dramatic, only 1 in 20,000 children is affected. In other words, this gene does not contribute significantly to variability in IQ scores in the population. Although some sledgehammer effects of

major genes may be found for complex behaviors, it seems more likely that many genes nudge development up and down and do not show dramatic effects as in the classical single-gene disorders.

Genes that affect behavioral traits are transmitted hereditarily according to Mendel's laws in the same way as genes that affect any other phenotype. However, the genetics of behavior is special in three ways. First, unlike Mendel's smooth vs. wrinkled seeds, most behavioral dimensions and disorders are not distributed in simple either/or dichotomies, although in psychopathology we often pretend that a line exists that sharply separates the normal from the abnormal. Second, behavioral traits are substantially influenced by nongenetic factors: heritabilities rarely exceed 50 per cent. Third, behavioral dimensions and disorders are likely to be influenced by many genes, each producing small effects.

The challenge is to use DNA markers to find genes in these complex systems of behavior that involve multiple-gene effects as well as multiple nongenetic factors. Such multiple genes of varying effect size that contribute to quantitative traits are called quantitative trait loci (QTL; Gelderman, 1975). Given the breathtaking pace of technological advances in molecular genetics, the safest bet is that at the turn of the century we will be investigating multiple-gene influences for complex dimensions and disorders using completely different techniques from those in use today. Nonetheless, we can begin the quest for QTL using DNA markers for quasi-candidate genes relevant to the nervous system. As explained elsewhere (Plomin, 1990b), linkage, the standard method for attempting to identify genes for single-gene disorders in the human species, is not likely to be successful in identifying QTL for quantitative dimensions of behavior because such traits are not dichotomous and because multiple genes as well as nongenetic factors are involved. The emerging view is that major genes will not be found for human behavior (Plomin, 1990b) or for common medical diseases (King, Rotter, & Motulsky, 1992). Rather, for each individual, many genes make small contributions to variability and vulnerability. In this view, the genetic quest is to find, not *the* gene for a behavioral trait, but the multiple genes that affect the trait in a probabilistic rather than predetermined manner.

New strategies are needed to identify genes that affect behavioral traits, even when the genes account for only a small amount of variance, when nongenetic factors are important, and when the traits are quantitatively distributed. In short, we need to use molecular genetic techniques in a quantitative genetic framework. One possibility is to apply a strategy called QTL allelic association (Edwards, 1991). Linkage refers to loci rather than alleles. That is, linked traits such as hemophilia and color-blindness do not occur together in the population. In contrast, allelic association occurs when a DNA marker is so close to a trait-relevant gene (or is part of the gene) that its alleles are correlated with the trait in unrelated individuals in the population. For example, allelic associations have been found between disease states and markers in the HLA histocompatibility complex (Tiwari & Terasaki, 1985). That is, particular HLA alleles increase risk for certain diseases. For normal variation, the best example of allelic association is serum cholesterol levels for which about a quarter of the variance can be explained by four apolipoprotein gene markers (Sing & Boerwinkle, 1987). In psychiatry, a marker in the D2 dopamine receptor has been reported in several studies to be associated with alcoholism. The frequency of an allele of this marker appears to be greater in severe alcoholics than in controls, although failures to replicate have

been reported (Cloninger, 1991). A major advantage of allelic association analysis is that it can employ samples of unrelated individuals, whereas linkage requires pedigrees of related individuals. By increasing the sample size, allelic association analysis can be made sufficiently powerful to detect small genetic effects. In addition, allelic association is just as applicable to quantitative dimensions as to disorders.

The goal is to identify some, certainly not all, genes that contribute to the ubiquitous genetic variance found for behavioral traits. This allelic association approach is currently being used in an attempt to identify QTL associated with cognitive ability (Plomin & Thompson, 1993). The breathtaking pace of development in molecular genetics leads me to predict that by the turn of the century behavioral scientists will use DNA markers as a tool in their research to identify relevant genetic differences among individuals. These DNA markers can be assessed inexpensively by DNA factories from just a drop of saliva or blood, thanks to a revolutionary technique of DNA amplication called polymerase chain reaction (Saiki, Gelfand, Stoffel, Sharf, Higuchi, Horn, Mullis, & Erlich, 1988). If a set of DNA markers were identified that together accounted for a reasonable portion of the genetic variance for a particular phenotype in social development, they could be used as a probabilistic index of genetic factors. Such genetic indices will facilitate much more powerful analyses of the developmental interface between nature and nurture.

Conclusion

In this article just a few examples were mentioned of the advances in behavioral genetics that are relevant to social developmentalists. Other examples include methodological advances such as analysis of the etiological relationship between the normal and abnormal (DeFries & Fulker, 1985, 1988) and model-fitting (Loehlin, 1987; Neale & Cardon, 1992; Plomin *et al.*, 1990), and substantive findings such as the importance of nonshared environment (Dunn & Plomin, 1990; Hetherington, Reiss, & Plomin, 1994; Plomin & Daniels, 1987) and findings concerning developmental change and continuity (Plomin, 1986; Plomin, Emde, Braungart, Campos, Corley, Fulker, Kegan, Reznick, Robinson, Zahn-Waxler, & DeFries, 1993).

The momentum of these findings and methods will propel the field of behavioral genetics far into the next century. Another reason for optimism about the future of the field is that it is successfully being given away. Some leading developmental researchers are beginning to incorporate genetic strategies in their research. For example, Jerome Kagan's twin study of behavioral inhibition (Robinson, Kagan, Reznick, & Corley, in press) is part of a collaboration between developmental psychologists and behavioral geneticists known as the MacArthur Longitudinal Twin Study (MALTS). As mentioned earlier, other MALTS collaborators include the project's leader, Robert N. Emde (Emde *et al.*, 1992), Carolyn Zahn-Waxler who has reported the first twin analysis of empathy (Zahn-Waxler *et al.*, 1992), and Joseph Campos who was also involved in an earlier twin study on temperament (Goldsmith & Campos, 1986). The NEAD longitudinal twin/stepfamily study mentioned earlier is a collaborative project with David Reiss and E. Mavis Hetherington (Reiss, Hetherington, Plomin, Howe, Rovine, Tryon, & Stanley, 1994). Judy Dunn has brought her expertise in siblings

to bear on issues of nonshared environment using genetic designs (Dunn & Plomin, 1990). In developmental psychopathology, Michael Rutter's work has shifted substantially to genetic research (e.g., Rutter, 1991; Rutter, Macdonald, Le Couteur, Harrington, Bolton, & Bailey, 1990). Another example in developmental psychopathology is Craig Edelbrock, who has begun a mid-career shift to incorporate genetic strategies in his research on dimensional and diagnostic issues (e.g., Edelbrock, Rende, Plomin, & Thompson, in press).

Other developmentalists may be considering the possibility of incorporating behavioral genetic strategies in their research but do not know how to begin. Most of the developmentalists mentioned above began to use genetic strategies as part of collaborative 'big science' projects. But this is not the only route to genetics. My suggestion is to begin by adding siblings in one's research. More than 80 per cent of families have more than one child and it is relatively easy to recruit a sibling of a subject. After analyzing the topic of interest (getting two children for the price of one), the data can be examined from a new perspective that considers sibling similarities and differences. How similar are siblings in the same family for this phenomenon? For most traits siblings are not very similar, which leads to the question of nonshared environment – why are siblings in the same family so different? Multivariate questions can also be asked: Do the familial or nonfamilial influences on one aspect of the phenomenon overlap with effects on another aspect? Developmental questions can be asked about age differences and age changes in sibling resemblance.

Although sibling analyses are familial rather than genetic, such analyses represent an important first step in understanding the etiology of individual differences. A next step is twins. Twins are not nearly as difficult to find as one might think – about one per cent of all births are twins. Moreover, twins and their parents are particularly willing to participate in research because twins are so obviously special. Even adoption designs are not impossible. During the 1960s and early 1970s, about one per cent of all births involved nonfamilial adoptions and about a third of adoptive parents adopted a second child (Mech, 1973). However, with contraception and abortion, the numbers of such adoptions declined dramatically during the 1970s. However, little use has been made in behavioral genetics of the large numbers of half-siblings and unrelated siblings that can be found in step-families (Plomin, Manke, & Pike, in press; Reiss, in press).

The reason for trying to give behavioral genetics away is that I believe that the best behavioral genetic research in social development will be done by social developmentalists who are not primarily behavioral geneticists. Experts in social development will ask theory-driven questions and interpret their research findings in a way that will make most sense for the field.

Acknowledgements

Some of the research described in this paper was supported by grants from the National Institute of Child Health and Human Development (HD-10333 and HD-18426), National Institute of Mental Health (MH-43373, MH-43899), National Science Foundation (BNS-91-88744), and the John D. and Catherine T. MacArthur Foundation. This paper was written on sabbatical leave supported by a James McKeen Cattell Sabbatical Award and a Fogarty Senior International Fellowship.

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