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Why are children in the same family so different from one another?

Robert Plomin
Department of Individual and Family Studies, Pennsylvania State University, University Park, Pa. 16802

Denise Daniels
Department of Psychiatry, Stanford University, Stanford, Calif. 94305

Abstract: One of the most important findings that has emerged from human behavioral genetics involves the environment rather than heredity, providing the best available evidence for the importance of environmental influences on personality, psychopathology, and cognition. The research also converges on the remarkable conclusion that these environmental influences make two children in the same family as different from one another as are pairs of children selected randomly from the population.

The theme of the target article is that environmental differences between children in the same family (called "nonshared environment") represent the major source of environmental variance for personality, psychopathology, and cognitive abilities. One example of the evidence that supports this conclusion involves correlations for pairs of adopted children reared in the same family from early in life. Because these children share family environment but not heredity, their correlation directly estimates the importance of shared family environment. For most psychological characteristics, correlations for adoptive "siblings" hover near zero, which implies that the relevant environmental influences are not shared by children in the same family. Although it has been thought that cognitive abilities represent an exception to this rule, recent data suggest that environmental variance that affects IQ is also of the nonshared variety *after adolescence*.

The article has three goals: (1) To describe quantitative genetic methods and research that lead to the conclusion that nonshared environment is responsible for most environmental variation relevant to psychological development, (2) to discuss specific nonshared environmental influences that have been studied to date, and (3) to consider relationships between nonshared environmental influences and behavioral differences between children in the same family. The reason for presenting this article in *BBS* is to draw attention to the far-reaching implications of finding that psychologically relevant environmental influences make children in a family different from, not similar to, one another.

Keywords: behavior genetics; development; environment; heredity; individual differences; intelligence; personality; psychopathology; schizophrenia; twins

The findings of greatest social significance to emerge from human behavioral-genetic research to date involve nurture, not nature. Research in this area, consisting primarily of twin and adoption studies, points to significant genetic influence on individual differences for a wide range of behaviors, including personality, psychopathology, and cognition. When we go beyond the statistical significance of genetic influence to ask about the effect size, it is also apparent that genetic influence is substantial. Nonetheless, the same data provide evidence – indeed, we think the best available evidence – for the importance of environmental variation in each of these domains.

Ten years ago, in order to redress the imbalance of environmentalism, it was necessary to emphasize the possibility that genetic influence could affect behavioral differences that we observe among individuals. Now behavioral geneticists find that they must more often emphasize the importance of environmental variation. Behavioral-genetic research seldom finds evidence that more than half of the variance for complex behavioral traits is due to genetic differences among individuals. Thus, for personality, psychopathology, and cognition, behavioral-genetic research converges on the conclusion that most behavioral variability among individuals is environmental in origin. For example, for schizophrenia, the concordance for first-degree relatives, whose coefficient of genetic relationship is .50, is less than 10%. Identical twins are less than 50% concordant for schizophrenia. Yet schizophrenia is coming to be viewed as a genetic disease. In the rush to find neural causes of schizophrenia, who is now studying the major source of variability – the environment?

Not only does behavioral genetic research document the importance of environmental influence, it also points to a possible treasure of environmental variance hidden in unexplored territory. This research implies that environmental influences that affect psychological development operate in a manner quite different from the way most psychologists thought they worked. Whatever they may be, these environmental influences make children in the same family as different from one another as are children in different families. One purpose of this article is to

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describe the evidence that leads to this conclusion and its implications. Our main goal, however, is to draw attention to this dramatic discovery and to elicit commentary and suggestions from our peers. Despite the far-reaching implications of the evidence that psychologically relevant environmental influences make children in a family different from rather than similar to each other, we are aware of no major criticism of these findings. We expect that *BBS* commentary will rock this boat's smooth sailing and perhaps even alter its course.

1. Quantitative genetics

In order to understand the evidence pointing to the importance of nonshared environment it is necessary to begin with an overview of the theory and methods of quantitative genetics, which, when applied to behavioral phenomena, is referred to as behavioral genetics. After describing the basic twin and adoption designs, we shall examine the implications of twin and adoption data for the separation of shared and nonshared environmental variation in the three domains with the most relevant data: personality, psychopathology, and cognition.

Quantitative genetic theory began in the early part of this century as a solution to the problem of reconciling Mendelian genetics with normal distributions. As anyone who has taken high school biology knows, about a hundred years ago, the monk Gregor Mendel studied dichotomous, either/or, characteristics such as round versus wrinkled seeds in the pea plant. When his work was rediscovered 30 years later it provoked controversy among biometricians who felt that the laws of heredity described by Mendel could not apply to human characteristics because, unlike discontinuous pea plant characteristics, human characteristics nearly always involve a normal, continuous distribution. The resolution to the controversy came when it was understood that a normal distribution would be observed if several genes affected a characteristic. In 1918, when Ronald Fisher put the finishing touches on this theory and spelled out the expectations for familial resemblance based on the theory, quantitative genetics was born.

The theory uses the covariance or correlation among relatives on normally distributed traits to estimate the role of heredity. Although the theory and its methods are usually presented in a sophisticated algebraic manner, the basic idea – which is all that is needed to understand the way in which environmental variation is partitioned in quantitative genetics – is very simple. Details, such as the distinction between additive and nonadditive genetic variance, can be found in textbooks on the topic (e.g., Falconer 1981; Hay 1985; Plomin, DeFries & McClearn 1980). The fundamental tenet of the theory is that individuals in a population differ for both genetic and nongenetic reasons. How can we assess the extent to which phenotypic (observed) variability is due to genetic variation among individuals or to nongenetic differences? In studies of human beings, for whom selection studies or comparisons among inbred strains cannot be conducted, the only way is to study pairs of individuals who differ in genetic resemblance. If heredity is important for a particular characteristic, pairs of individuals who are more similar genetically ought to be more similar for the measured characteristic. For example, third-degree relatives such as cousins will be less similar than second-degree relatives such as half-siblings who, in turn, will be less similar than first-degree relatives such as full siblings. If heredity does not affect the trait, then differences in genetic similarity should not affect the resemblance of these pairs of individuals.

The problem is that environmental resemblance often covaries with genetic relatedness: Cousins, half-siblings, and full siblings, respectively, are likely to share increasingly similar environments. Because relatives share family environment as well as heredity, familial resemblance can be due to environmental influences as well as to hereditary influences. In other words, a portion of environmental influence could be shared by relatives, making them similar to one another. Nonetheless, family studies are useful in estimating limits of genetic and environmental influences. For example, if the correlation for first-degree relatives is zero for a particular trait, then neither shared heredity nor shared family environment affect the trait.

The two major designs of human behavioral genetics – the adoption design and the twin design – were developed to circumvent the problem of conflating genetic and environmental influences in studies of family members who share heredity and family environments. By doing so, these designs partition environmental variance into two components: one shared by members of a family and the other consisting of the remainder of the environmental variance, which is referred to as nonshared environment.

1.1. Adoption design. The basic problem in family studies is that resemblance among relatives could be due to shared heredity or to shared environment. The adoption design powerfully cleaves these two sources of familial resemblance. Genetically related individuals adopted apart and reared in uncorrelated environments will resemble each other only for genetic reasons. Genetically unrelated individuals adopted together in the same family will resemble each other only for reasons of shared environment.

The simplest adoption design to understand is the rare, but dramatic, situation in which identical twins are adopted separately at birth and reared apart in uncorrelated environments. The resemblance of these pairs of twins, expressed as a correlation, is a direct estimate of the proportion of phenotypic variance that is due to genetic variance, a descriptive statistic known as heritability. A correlation of .50 for identical twins reared apart implies that half of the phenotypic variance is genetic in origin.[1]

A technical point that has some bearing on the estimation of nonshared environment concerns the distinction between additive and nonadditive genetic variance. Identical twins share all sources of genetic variance, no matter how complex the interactions among genes. Thus, an estimate of heritability derived from the correlation for identical twins reared apart is referred to as broad heritability – it includes all sources of genetic variance. In contrast, first-degree relatives primarily share only additive genetic variance, genetic effects that add up linearly in their effect on the phenotype; estimates of heritability based on first-degree relatives adopted apart are thus primarily limited to additive genetic variance

and are thus referred to as narrow heritability. This distinction is important to the extent that nonadditive genetic variance is important; if nonadditive genetic variance affects a trait, behavioral genetic designs that assess narrow heritability will misread this genetic variance as nonshared environment. Although most behavioral geneticists discount the importance of nonadditive genetic variance, some recent work suggests that it contributes to certain characteristics (Lykken 1982; Plomin 1986).

Phenotypic variance not explained by genetic variance is ascribed to environmental sources. More properly, this component of variance is nongenetic; that is, it is broader than the usual way psychologists think about the environment in that it includes accidents and illnesses, prenatal influences, cytoplasmic changes, and even DNA changes that are not transmitted hereditarily. Data for relatives adopted apart, as in the case of separately adopted identical twins, cannot by themselves separate shared and nonshared environmental components of nongenetic variance.

Other adoptions designs can assess shared and nonshared environment. Comparisons between relatives adopted apart and relatives reared together permit an indirect assessment. Relatives adopted apart share heredity but not environment, whereas relatives reared together are similar for reasons both of shared heredity and shared environment. If relatives reared together are no more similar than relatives adopted apart we can conclude that growing up in the same family does not add to relatives' resemblance beyond the similarity induced by heredity. In other words, environmental influence operates in a nonshared manner. For example, if, for a particular trait, identical twins reared together are no more similar than identical twins reared in uncorrelated environments, shared environment is unimportant for that trait and all of the environmental variance must be nonshared. On the other hand, if the correlation for identical twins reared together is .75 and the correlation for identical twins reared apart is .50, 25% of the phenotypic variance could be attributed to shared environment and the remaining 25% to nonshared environment.

We have included this concrete example of partitioning only for purposes of clarification. We do not mean to convey that such estimates will be particularly precise. The accuracy of the estimates depends on all of the usual statistical issues such as sample size as well as on the assumptions of behavioral genetic designs. The estimates of nonshared environment described later in our review come from large samples, are replicated in many studies, and are based on quite different designs such as adoption as well as twin studies. Moreover, our estimate of nonshared environment would have to be very substantially wrong before it would seriously affect our conclusion that nonshared environment is responsible for most environmental variation relevant to psychological phenomena.

A direct test of the importance of shared environment comes from the other side of the adoption design in which genetically unrelated individuals are adopted into the same family. These adoptive family members share major features of their environment – the same parents, home, social class, community, schools, and so forth – but they do not share heredity. The correlation for pairs of unrelated children adopted together directly estimates the proportion of phenotypic variance due to shared environment. For example, a correlation of .25 for a trait measured in pairs of adoptees reared in the same adoptive homes suggests that 25% of the phenotypic variation in the trait can be explained by shared environment. A correlation of zero for pairs of adoptees, on the other hand, implies that shared environment contributes nothing to phenotypic variance, which implies that all of the environmental variation is nonshared.

It should be mentioned that the distinction between shared and nonshared environment is not limited to family relationships in which relatives are the same age (such as twins), or relatives who are nearly the same age (such as siblings). We can also consider shared and nonshared environmental factors that affect the resemblance between parents and their offspring. In this case, shared environment refers to environmental influences that increase resemblance between parents and offspring. It does not involve all parental influences on offspring, only those environmental influences that increase phenotypic similarity between parents and their children.

1.2. Twin design. The twin design compares the resemblance of identical twins with that of same-sex fraternal twins. Both types of twins are born at the same time, share the same womb and home, and are of the same sex. One major difference distinguishes the two types: Identical twins are twice as similar genetically (on the average) as fraternal twins. If heredity affects a trait, the twofold greater genetic similarity of identical twins will make them more similar than fraternal twins with respect to a particular trait. The difference between the correlations for identical twins and fraternal twins is an estimate of roughly half of the genetic variance in the population because the coefficient of genetic relationship is 1.0 for identical twins and .50 for fraternal twins. Thus, for a trait completely determined by heredity, the expected correlations are 1.0 for identical twins and .50 for fraternal twins. If the pattern of twin correlations were .75 and .50 for identical and fraternal twins, respectively, heredity would be estimated to explain half of the phenotypic variance for the trait. If heredity does not affect the trait, the twofold greater genetic similarity of identical twins will not make them more similar than fraternal twins for the particular trait.

This discussion has oversimplified the twin method for didactic purposes. For example, assortative mating would raise the fraternal twin correlation and nonadditive genetic variance would lower it. Also, even though twin partners of both types live in the same family, it is possible that identical twins experience more similar family environments than do fraternal twins. If this were the case, some of the greater observed similarity of identical twins might be due to greater similarity of their experience. This possible confounding effect has been examined and, in research to date, does not appear to represent a major problem for the twin design (Plomin, DeFries & McClearn 1980). Finally, genotype–environment interaction and correlation can affect these estimates, as discussed later.

If genetic variance accounts for 50% of the phenotypic variance, the rest of the phenotypic variance is attributed to nongenetic variance, which includes shared and nonshared environment as well as error of measurement. The twin method can be used to partition nongenetic variance

into its shared and nonshared components. Consider two patterns of identical vs. fraternal twin correlations: .75 vs. .50 and .50 vs. .25. Doubling the difference between the twin correlations suggests a heritability of 50% for both patterns of correlations. Thus, for both patterns, the proportion of phenotypic variance due to environmental variance is 50%. In the first case, however, the one with correlations of .75 and .50 for identical and fraternal twins, respectively, half of the environmental variance is shared by the twins, making them resemble each other, and the other half of the environmental variance makes them different. In the case of identical and fraternal twin correlations of .50 and .25, all of the environmental variance contributes to differences within pairs.

The reasoning behind this conclusion is as follows: Differences within pairs of identical twins are due only to nongenetic factors not shared by twins because members of identical twin pairs do not differ genetically. Thus, when identical and fraternal twin correlations are .50 and .25, respectively, .50 of the phenotypic variance is genetic and .50 is nongenetic. Because identical twins are identical genetically and yet their phenotypic correlation is only .50, all of the nongenetic variance (specifically, nonshared environment and error of measurement) leads to differences within pairs. Variance due to error of measurement can be assessed as the difference between the reliability coefficient (e.g., test–retest correlation) and 1.0. For example, if a test–retest correlation is .90, error variance is 10%; the 50% nongenetic variance thus consists of 40% nonshared environmental variance and 10% error variance. When the identical and fraternal twin correlations are .75 and .50, half of the phenotypic variance is again environmental, but in this case only half of the environmental variance (25% of the total phenotypic variance: 1.0 − .75 = .25) is due to nonshared environment and error and the other half is shared. The shared environment component of variance can be estimated as twice the fraternal twin correlation minus the identical twin correlation.

In summary, the twin design provides a direct estimate of nonshared environment – the component of phenotypic variance that is not shared by members of identical twin pairs. In addition, the twin design provides an indirect estimate of shared family environment: It is the component of phenotypic variance that remains after accounting for genetic variance and nonshared environmental variance. The generalizability of twin results concerning shared family environment to the population of nontwin siblings is questionable, however, because it seems likely that twins share family environments to a greater extent than do siblings who are not twins, as will be discussed later.

Thus, adoption and twin studies can separate environmental variance for behavioral traits into two components. One component, called shared environment, includes all environmental influences that make children in a family similar to one another. This component of variance can be estimated in three ways: (1) from the correlation for genetically unrelated children reared together in the same adoptive families, (2) from the difference in correlations for relatives reared together and relatives adopted apart, and (3) from twin studies, as the remainder of phenotypic variance when genetic variance, variance due to nonshared environment, and error are removed. Environmental variance not due to shared environment is called nonshared environment; this portion of environmental variance makes family members different from one another. This variance component is usually estimated as the remainder of phenotypic variance once variance due to heredity, shared environment, and error of measurement is removed. Differences within pairs of identical twins reared together provides a direct estimate of nonshared environment as experienced by identical twins.

Because we are developmentalists, we feel compelled to make the point that all components of variance can change during development. Estimates of genetic and environmental components of variance depend upon the age of the subjects sampled. Genetic change during development is the focus of a new subdiscipline, developmental behavioral genetics (Plomin 1986). Nonshared and shared environmental components can also change during development. Research is needed to trace the developmental course of shared and nonshared environmental variance. For example, there may be a general trend for nonshared environmental variance to increase with age as individuals expand their social and environmental networks beyond the family. On the other hand, as this happens, there may be fewer forces contrasting children in the same family. Research throughout the lifespan – especially research past adolescence – will be needed to resolve such developmental issues. One striking example of developmental change in the relative influence of shared and nonshared environmental variance serves to indicate the potential usefulness of a lifespan perspective: For IQ, the shared environment component of variance diminishes dramatically after childhood, as discussed in the next section.

2. Evidence for the importance of nonshared environmental effects on behavior

This section provides a brief summary of behavioral-genetic research in personality, psychopathology, and cognition that leads to the conclusion that the most important source of environmental variance is nonshared environment. This material is based on a recent review of behavioral-genetic research throughout the lifespan which can be consulted for additional studies and details (Plomin 1986). Although readers might take issue with the precise magnitude of one or another of the estimates, the forest should not be overlooked for the trees. Our point, one that to our knowledge has not been disputed, is that nonshared environment is responsible for most environmental variation relevant to psychological development. Thus, our goal in the following section is not to provide an encyclopedic review of behavioral-genetic studies but rather to summarize the results to the extent needed to understand their message regarding the importance of nonshared environment.

2.1. Personality. The importance of nonshared environment was first highlighted by Loehlin and Nichols (1976) whose twin analyses of personality data led to the following conclusion:

Thus, a consistent – though perplexing – pattern is

> emerging from the data (and it is not purely idiosyncratic to our study). Environment carries substantial weight in determining personality – it appears to account for at least half the variance – but that environment is one for which twin pairs are correlated close to zero. . . . In short, in the personality domain we seem to see environmental effects that operate almost randomly with respect to the sorts of variables that psychologists (and other people) have traditionally deemed important in personality development. (Loehlin & Nichols 1976, p. 92)

Loehlin and Nichols reached this conclusion because identical and fraternal twin correlations were consistently about .50 and .30, respectively, within their large study of high-school-aged twins that used self-report personality questionnaires. This pattern of correlations suggests 40% genetic variance and 60% environmental variance, and that over 80% of the environmental component of variance is due to nonshared environment plus error. (Error accounts for about 20% of the variance.)

These results are not peculiar to Loehlin and Nichols's study of high-school twins. In a review of 10 recent twin studies of personality (Goldsmith 1983), the average twin correlations were .47 for identical twins and .23 for fraternal twins. This pattern of twin correlations suggests that heredity accounts for 50% of the phenotypic variance and that nonshared environment and error of measurement explain the rest.

It might seem odd to report average correlations across a domain as diverse as personality. Nonetheless, the twin results are generally similar across the dozens of traits measured by self-report questionnaires. Consider extraversion and neuroticism, the two "super-factors" in personality, which are associated with Eysenck (e.g., 1967) but also emerge as major second-order factors from other personality questionnaires such as Cattell's Sixteen Personality Factor Questionnaire (Cattell, Eber & Tatsuoka 1970). [See also Zuckerman: "Sensation Seeking" *BBS* 7(3) 1986.] A study of over 12,000 adult twin pairs in Sweden (Floderus-Myrhed, Pedersen & Rasmuson 1980) revealed twin correlations of .51 and .21 for identical and fraternal twins, respectively, for extraversion and correlations of .50 and .23 for neuroticism.

Similar results emerge for less central dimensions of personality as well. For example, Loehlin and Nichols's study used the California Psychological Inventory, which includes diverse scales such as Sense of Well-Being, Tolerance, and Good Impression. The identical and fraternal twin correlations, respectively, for these scales were .50 and .30, .53 and .35, and .48 and .30. Another example involves twin results for a new personality questionnaire, the Differential Personality Questionnaire, which assesses nontraditional dimensions of personality. A twin study of over 200 identical twin pairs and over 100 fraternal twin pairs yielded the following sampling of correlations for identical and fraternal twins, respectively: .50 and .36 for Danger Seeking; .61 and .37 for Authoritarianism; and .58 and .25 for Alienation (Lykken, Tellegen & DeRubeis 1978). The only personality trait that appears to show significant shared environmental influence is masculinity–feminity, which one might argue falls more in the category of attitudes than personality (Loehlin 1982).

These twin studies used self-report questionnaires. Perhaps some artifact exists so that identical twins always rate themselves as 50% similar when asked about their personality. Other assessment procedures, however, yield similar results. For example, in recent years, several twin studies using parental ratings of children's personality have been reported (reviewed by Buss & Plomin 1984). The average identical twin correlation is about .50, again suggesting that about half of the variance is due to nonshared environment. The few twin studies that have used objective observations of personality yield somewhat less ubiquitous evidence for nonshared environmental variance than do paper-and-pencil questionnaires (Plomin & Foch 1980). Nonetheless, estimates of nonshared environmental influence from these studies are still substantial – usually greater than estimates of shared environmental variance, even when error variance is taken into account.

Studies of nontwin siblings and other family relationships confirm the hypothesis that shared family environment accounts for a negligible amount of environmental variance relevant to personality development. For example, one of the earliest studies found an average sibling correlation of .12 (Crook 1937); a recent large family study (Ahern, Johnson, Wilson, McClearn & Vandenberg 1982) yielded an average sibling correlation of .16 for three widely used personality questionnaires. The average parent/offspring correlations in this study were also low: .12 for father/son, .10 for father/daughter, .13 for mother/son, and .14 for mother/daughter.

Four recently reported adoption studies of personality indicate that this modest familial resemblance is not due to shared family environment – the average adoptive sibling correlation is .04 and the average adoptive parent/adopted child correlation is .05 (Loehlin, Horn & Willerman 1981; Loehlin, Willerman & Horn 1985; Scarr, Weber, Weinberg & Wittig 1981; Scarr & Weinberg 1978a). Adoptive sibling correlations are also low in the first report of infant adoptive siblings, involving 61 pairs at 12 months and 50 pairs at 24 months tested as part of the Colorado Adoption Project (Daniels 1985). Parental ratings of temperament yielded average adoptive sibling correlations of .11 at 12 months and .05 at 24 months; tester ratings on the Infant Behavior Record (Bayley 1969) yielded average adoptive sibling correlations of −.14 at 12 months and .05 at 24 months.

2.2. Psychopathology. Behavioral-genetic data on psychopathology are also consistent with the conclusion that environmental variation is preponderantly of the nonshared variety. Research on schizophrenia is difficult to summarize briefly because concordance rates vary widely depending on the following: whether or not age correlations are used, the type of diagnostic criteria used, and the selection and severity of probands. Nonetheless, relying on a recent book-length review (Gottesman & Shields 1982), familial concordance rates for schizophrenia in a dozen studies found about 10% concordance rates for schizophrenia for first-degree relatives. The concordance rate for fraternal twins is also about 10%. Concordance rates for identical twins are substantially higher than those for fraternal twins – indeed, higher

than would be expected on the basis of a simple additive genetic model in which identical twins would be about twice as similar as fraternal twins. For example, Gottesman and Shields review five recent studies that yield an average case-wise concordance of 45% for identical twins. Regardless of the complications this pattern of twin concordance causes for estimates of genetic influence, the results indicate that most schizophrenic identical twins do not have an affected cotwin. Because these are genetically identical pairs of individuals, nonshared environment must be the reason for these striking differences within pairs of identical twins.

This conclusion is confirmed in Gottesman and Shields's review of recent adoption studies in Denmark in which the same concordance of about 10% is found for individuals adopted apart from a first-degree schizophrenic relative. Thus, sharing the same family environment with a schizophrenic relative does not increase familial concordance.

Gottesman and Shields (1982) also review attempts to isolate environmental sources of variance and conclude:

> So far, *no* specific environmental source of liability is known; the most likely environmental contributor, stress, may come from many sources and, apparently, may come during any stage of development. Prenatal or birth complications, early deprivations, broken homes, censuring parents, the death of someone close, failures in school, poor work or social relationships, childbirth, a bad drug trip, as well as all kinds of *good* fortune may have effects on a predisposed individual that are obvious only in retrospect. In prospect, it will be impossible to prophesy the events themselves, let alone their effects. (Gottesman & Shields 1982, pp. 241–42)

We suggest, however, that until more systematic research on nonshared environmental variance sources is conducted it is too early to conclude that the large environmental component of variance in schizophrenia is brought about by idiosyncratic experiences.

Research on manic–depressive psychosis yields results similar to those for schizophrenia (Plomin, DeFries & McClearn 1980). Environmental influences on less severe forms of psychopathology, such as neuroses and alcoholism, also appear to be predominantly nonshared. Sibling concordances are generally less than 20% and when twin and adoption studies have been conducted most of this familial resemblance has been found to be genetic in origin (Fuller & Thompson 1978; Rosenthal 1970). In other words, the most important influences on psychopathology lie in the category of nonshared environment. Much more often than not, affected children in families with more than one child will have unaffected siblings.

2.3. Cognition. Until recently, environmental variance that affects individual differences in IQ was thought to fall primarily in the category of shared environment. In 11 studies, the average IQ correlation for adoptive siblings is .30, suggesting that 30% of the variance in IQ scores is due to shared environmental influences (Bouchard & McGue 1981). Adoptive parent/adopted child IQ correlations are lower, about .20, but still suggest substantial influence of shared environment on parent–offspring resemblance. Twin studies agree: The average IQ correlation in over 30 studies is .85 for identical twins and .58 for fraternal twins (Bouchard & McGue 1981), which suggests again that about 30% of the variance of IQ scores can be accounted for by shared environment.

Although these data appear to converge on the reasonable conclusion that shared environment accounts for a substantial portion of environmental variance relevant to IQ, doubts have begun to arise. For fraternal twins, who share environment to a greater extent than do nontwin siblings, the IQ correlation is about .60, whereas the correlation for nontwin siblings is about .40 – which means that the twin method overestimates the importance of shared environmental in comparison to family studies.

The crucial piece of evidence in support of substantial shared environmental variance is the correlation of .30 for adoptive siblings reared together. These studies have included adoptive siblings still living at home, with two exceptions. The first exception is a study of postadolescent adoptee pairs by Scarr and Weinberg (1978a) which found a correlation of −.03 for IQ. This unsettling finding implies that shared environment is important for IQ during childhood when children are living at home and then fades in importance after adolescence when children have left home.

The hypothesis that shared environmental influences have no lasting impact on IQ is supported by results of a recent study of adoptive and nonadoptive siblings (Kent 1985). The study included 52 pairs of adoptive siblings and 54 pairs of nonadoptive siblings ranging from 9 to 15 years of age, with the average age of 13 years. A battery of cognitive ability measures was developed for administration over the telephone; this battery correlated with face-to-face testing near the reliabilities of the tests. An unrotated first principal component, used as an index of IQ, yielded a reasonable correlation of .38 for nonadoptive siblings; however, the IQ correlation for adoptive siblings was −.16, not significantly different from zero. A similar pattern of results emerged for specific cognitive abilities. The adoptive sibling correlations for verbal, spatial, perceptual speed, and memory abilities were −.06, −.07, −.10, and .16, respectively.

Thus, this study leads to the conclusion that shared environmental influence on IQ and specific cognitive abilities is of negligible importance by the end of early adolescence. Because these estimates of shared environmental influences were obtained directly from adoptive sibling correlations, reasonable confidence can be attached to this conclusion. For example, the sample of 52 pairs of adoptive siblings permits detection of a true correlation of .30 with 70% power; the standard error of the estimates of shared environment were found to be between .10 and .14 when a multiple regression model-fitting approach suggested by DeFries and Fulker (1985) was used.

In summary, nonshared environmental influence is a major component of variance for personality, psychopathology, and IQ (after childhood). We conclude that nonshared environment explains perhaps as much as 40% to 60% of the total variance for these domains. Although one can quibble with the magnitude of our estimates, they would have to be substantially in error before they would affect our argument that most of the environmental variance is nonshared.

3. Shared and nonshared environmental variance

The purpose of this section is to consider some conceptual details of the distinction between shared and nonshared environment before discussing sources of nonshared environment. These details include other labels for shared and nonshared environment, the distinction between environmental components of behavioral variance and the relationship between specific environmental measures and behavior, the impact of nonshared environmental influence on the development of singletons, genotype–environment correlation and interaction, and model-fitting.

3.1. Other labels. Shared and nonshared environmental influences were named by Rowe and Plomin in 1981, although the distinction between environmental influences that contribute to the resemblance between relatives and those that do not has been implicit in quantitative genetics since its inception. Many labels have been used to refer to these two components of environmental variance. Shared environmental influence has been called E2, between-family, and common environmental variance, labels that have been used to refer to nonshared environmental include E1, within-family, individual, unique, and specific environmental variance. Rowe and Plomin suggested that the symbols E1 and E2 (Jinks & Fulker 1970) are probably best in that they carry no connotations, although they have the distinct disadvantage that they provide no mnemonic to remember which is which. Within- and between-family environment are the terms most often used. They are useful for those familiar with the terminology of analysis of variance which considers variance within and between groups. Variance within families refers to differences among family members and variance between families describes resemblance among family members. The term "within-family" environment, however, connotes factors that occur within the confines of the family; whereas nonshared influences are those that cause family members to differ regardless of whether the locus of influence is the family (such as differential treatment by parents) or outside the family (such as different experiences at school or with peers). For these reasons, we suggest that the most descriptive and straightforward terms to use are shared and nonshared.

3.2. Components of variance versus specific measures. It should be noted that this discussion pertains to environmental components of behavioral variance, not to the relationship between specific environmental measures and behavioral measures. In this sense, quantitative genetic analyses describe the "bottom line" of genetic and environmental influence. That is, the total impact of genetic variability on phenotypic variability will be detected regardless of the complexity of the genetic effects – for example, whether the genetic effects arise from variability in structural genes that code for polypeptides or from regulatory genes. Similarly, quantitative genetics estimates the bottom line of environmental influence, regardless of the specific mechanisms by which environmental factors affect behavior. Although this components of variance approach may be unsatisfying for those who would like to know which specific genes and which specific environmental factors are responsible for the components of variance, it seems to be a reasonable first step to ask about components of variance – without this tack, we would not have discovered that nearly all environmental variance is of the nonshared variety. It is a major strength of the approach that it can reveal the presence of genetic and environmental influences even when these are not assessed directly.

Attempts to isolate specific environmental factors will be presented later. A related issue, however, should be mentioned at this time. Traditional environmental research attempts to relate measures of family environment to measures of behavior of one child per family. The yield from such research has been disappointing, especially if one considers the amount of variance explained (Maccoby & Martin 1983). Knowing this research, one might ask why such environmental factors as parental affection should be important *within* families when they account for little variance in behavior *across* families. That is, if it makes little difference that some parents love their children more than other parents love their children, why should parental love make a difference within families if a parent loves one child more than another? The answer is that there is no necessary relationship between the causes of differences *between* families and the causes of differences *within* families. That is, environmental factors that create differences within families can act independently of factors that cause differences between families. For example, a child really knows only his own parents; the child does not know if his parents love him more or less than other parents love their children. A child is likely to be painfully aware, however, that parental affection toward him is less than toward his sibling.

3.3. Singletons. Because over 80% of U.S. families have more than one child, it is important to understand why children in a family are so different from one another. How does nonshared environment relate to singletons? In general, reasons why two children in the same family differ are likely to yield clues as to the environmental source of variance for singletons as well. The easiest example involves nonsystematic events such as accidents and illnesses which are just as likely to befall singletons. However, systematic nonshared influences may also be found to affect singleton variance. For example, if certain characteristics of peer groups differ within pairs of siblings and contribute importantly to behavioral differences within sibling pairs, it is likely that these characteristics also contribute to variance for singletons.

Obviously, singletons do not have siblings with whom they interact; thus, this potential source of nonshared environment cannot contribute variance for singletons. Although it might seem at first that differential parental treatment of two children in the same family is irrelevant to singletons, it is possible that, once identified, such factors might contribute to the variance of singletons. There is evidence that parents with more than one child treat the children similarly if we look at the children at the same age, which suggests that parental treatment is not an important source of nonshared environment (Dunn, Plomin & Nettles 1985). Except for twins, however, siblings are not the same age, and when we examine contemporaneous parental treatment of children of different ages, we find that parents treat the children differ-

ently (Dunn, Plomin & Daniels 1986). Differences in parental behavior during development can also affect singletons in that parents will treat their singleton children differently during the course of development.

Thus, studies of differences within pairs of siblings are likely to illuminate factors responsible for singleton variance as well as sibling variance. The important point in the present context is the obvious one: that the study of singletons cannot isolate factors that make two children in the same family different from one another. Because this is the best clue we have as to the source of environmental variance relevant to psychological development, it makes sense to focus on environmental sources of differences between children in the same family.

3.4. Genotype–environment correlation and interaction. Two complicating factors in the estimation of quantitative genetic parameters are genotype–environment correlation and genotype–environment interaction (Plomin, DeFries & Loehlin 1977). Genotype–environment correlation refers to an increase in phenotypic variance that occurs when children experience environments correlated with their genetic propensities. Phenotypic variance can also be due to genotype–environment interaction when children respond differently to the same environment because of genetic differences among them. What are the effects of genotype–environment correlation and interaction on estimates of shared and nonshared environment? Consider a direct estimate of nonshared family environment: the extent to which the correlation for identical twins reared together is less than 1.0. This estimate will not include either genotype–environment correlation or interaction because identical twins are identical genetically; thus, in terms of genetic propensities, identical twins will correlate and interact with the environment in a similar manner. Similarly, the direct estimate of shared family environment – the correlation for unrelated children reared together – will not include genotype–environment correlation or interaction because these children are genetically uncorrelated; thus, in terms of their genetic propensities, they will correlate and interact with the environment in ways that do not add to their resemblance. However, estimates of nonshared or shared environment derived as the remainder of phenotypic variance after other components of variance are taken into account can be affected by genotype–environment correlation and interaction because of their effects on estimates of genetic variance (Plomin *et al.* 1977).

3.5. Model-fitting. Fitting models to adoption and twin data is a powerful way to estimate quantitative genetic parameters (Loehlin 1978). Although model-fitting techniques differ in their specifics, they all express family resemblance in terms of an underlying model consisting of several unobserved genetic and environmental parameters. The approach is powerful because it makes assumptions explicit, it tests a specific model, and it can incorporate into a single analysis different types of data, such as family and adoption data, rather than analyzing each type of data separately. Model-fitting procedures, however, only find significant parameters when they are implicit in the basic data; for example, in a study of adoptive siblings, a reasonable model-fitting analysis will estimate significant shared family environmental influences only if the correlation for adoptive siblings is significant. For this reason, and because of the relative inaccessibility of most models, we have emphasized the basic correlational data and merely note that model-fitting approaches confirm our conclusions.

4. Categories of nonshared environmental influence

What is happening environmentally to make children in the same family so different from one another? One gloomy prospect is that the salient environment might be unsystematic, idiosyncratic, or serendipitous events such as accidents, illnesses, and other traumas, as biographies often attest. In his autobiography, Darwin noted one example:

> The voyage of the *Beagle* has been by far the most important event in my life, and has determined my whole career; yet it depended on so small a circumstance as my uncle offering to drive me thirty miles to Shrewsbury, which few uncles would have done, and on such a trifle as the shape of my nose. (Darwin 1892, p. 28)

Darwin's comment about his nose refers to the quixotic captain of the *Beagle*, Captain Fitz-Roy, who nearly rejected Darwin for the trip because the shape of his nose indicated to Fitz-Roy that Darwin would not possess sufficient energy and determination for the voyage. (Darwin wrote that, during the voyage, Fitz-Roy became convinced that "my nose had spoken falsely" [p. 27].)

It is possible that nonshared environmental influences could be unsystematic in the sense of stochastic events that, when compounded over time, make children in the same family different in unpredictable ways. Such capricious events, however, are likely to prove a dead end for research. More interesting heuristically are possible systematic sources of differences within families.

Table 1 describes categories of environmental factors that could lead to observed differences between children in the same family. These include such systematic sources of nonshared influence in the family as birth-order and gender differences of siblings, interactions between siblings, differential treatment by parents, and extrafamilial influences such as peers.

In one sense, thinking about environmental influences that create differences between children in the same family represents a dramatic reconceptualization of psychological environments. On the other hand, this reconceptualization need not involve mysterious elements in the environment: Any environmental factor can be viewed in terms of its contribution to nonshared environmental variance. For example, parental affection can be easily construed as a source of differences among children in the same family, because parents may be more affectionate toward one child than another.

In this sense, our conceptualization of nonshared environmental influence is not new and exciting. Although any traditional environmental factor can be viewed in terms of its contribution to nonshared environmental variance, it is important to emphasize the point mentioned earlier: There is no necessary relationship between environmental factors that contribute to differences between families and those that affect differ-

Table 1. *Categories of environmental influences that cause children in the same family to differ*

Categories	Examples
Error of measurement	Test–retest unreliability
Nonshared environment	
Nonsystematic	Accidents, illnesses, trauma
Systematic:	
Family composition	Birth-order; gender differences
Sibling interaction	Differential treatment
Parental treatment	Differential treatment
Extrafamilial networks	Peer groups; teachers; television

Source: Adapted from Rowe and Plomin (1981).

ences between siblings within a family. In some cases, it seems likely that there is no relationship: Socioeconomic status (SES), for example, is an important factor that operates between families, but even though the SES of families changes, it is unlikely that SES is an important source of differences between siblings. Conversely, an environmental factor that makes only a slight difference between families may be critical within families. For reasons such as these, what is needed more than speculation about the most relevant nonshared environmental influences is research identifying relevant factors. This research can at the same time provide insights into theoretical issues such as the relationship between nonshared influences and traditional environmental factors studied across families.

The perspective of nonshared environment does, however, suggest some new ways to study environmental influences. For example, we must focus on measures of experience specific to each child. That is, one implication of our conclusion concerning the importance of nonshared environment is that environmental factors shared by both children in a family are unlikely to be important sources of environmental influence. Environmental measures are needed that capture the major sources of differential experience of siblings. Another strategy for research is exemplified by the emphasis of family therapists on systems theory in which the child is viewed as part of an organized family system, creating and maintaining patterns of behavior (Minuchin 1985). Another strategic suggestion for the study of nonshared environment is to explore environmental sources of developmental differences *within individuals* (McCall 1983): An environmental factor that is responsible for change in a child from early childhood to school age is also likely to make children in the same family different from one another.

Finally, another, even more speculative, methodological lead for research is that subjective, perceived experiences may prove to be important (e.g., Jessor 1981). For example, even if during home observations children in the same family appear to receive the "same" environmental treatment, this does not mean that the children experienced the treatment similarly. We do not mean to suggest that objective assessment of the environment is not also needed – it would be best to use objective and subjective measures in the same study in order to compare their relative effectiveness in predicting sibling differences. A first attempt to assess differences in perceived environments of siblings is discussed in the following section.

5. Attempts to identify nonshared environmental influences

This section explores attempts to assess specific factors within these categories that may be responsible for nonshared environmental variance. Family constellation variables, especially birth order, have been studied extensively. Other categories of possible nonshared environmental influence such as differential parental treatment, differential sibling interaction, and differential extrafamilial experiences have not yet received much attention.

5.1. Birth-order. The only specific source of nonshared family environment to receive considerable attention is birth-order. For example, over 1,000 entries for "birth-order" appear in *Psychological Abstracts*. Birth-order is a prototype of nonshared environmental influence in that it is different for children in the same family and yet cannot originate in genetic differences among siblings. Paradoxically, however, most studies have analyzed its effect across families rather than within families and most of the relationships are weak for IQ (Galbraith 1982) and for personality (Ernst & Angst 1983).

5.2. Other systematic nonshared environmental influences. Although birth-order has received considerable attention, studies of differential parental treatment, sibling interaction, and extrafamilial influences are more promising. In exploring possible nonshared influences, the first step is to ask whether siblings in a family have different experiences. If siblings do not differ in their experience for a particular aspect of the environment then that environmental factor cannot be a source of differences between them. For birth-order, this first step is unnecessary because siblings obviously differ in birth-order. Experiential differences, however, cannot be assumed to affect behavioral differences within pairs of siblings, therefore demonstrating that nonshared experiences are related to differences in sibling behavior is the second step. The third step is to describe the direction of effects when associations are found between differential experience and differences in their behaviors. Do sibling differences in experience affect or merely reflect differences in sibling behavior?

Thus, there are three steps in research on nonshared environmental influences: identifying experiences that are not shared by family members, relating such nonshared environmental factors to differences in sibling behavior, and determining the causal direction of such relationships. Because the topic of nonshared environment is so new, only a few relevant studies have been reported and most of these address the first step.

5.3. Sibling inventory of differential experience. One systematic approach to the topic is the Sibling Inventory of Differential Experience (SIDE; Daniels & Plomin 1985). The 73-item self-report SIDE asks each sibling to compare his experiences to those of a sibling in the domains of

sibling interaction, parental treatment, peer characteristics, and events specific to the individual. For all items, siblings are asked to compare their relative experiences rather than to make absolute judgments about their experience. For example, rather than asking the extent to which "my sibling and I show understanding for each other," the SIDE asks, "Who has shown more understanding for the other?" A 5-point scale is used for the siblings' ratings: 1 = My sibling has been much more this way than I have; 2 = My sibling has been a bit more this way than I have; 3 = My sibling and I have been the same in this way; 4 = I have been a bit more this way than my sibling; and 5 = I have been much more this way than my sibling. This provides relative scores indicating, for example, the extent to which one sibling feels he is understood by the other. Although somewhat unusual, these relative judgments have several advantages. First, they should be easier to make than absolute judgments – for example, on a 5-point scale, how much do you understand your sibling? (compared to what?). Second, relative judgments do not require that a sibling difference be calculated in order to assess nonshared environment. Third, they can be used when data are available from only one member of a sibling pair. The SIDE can also be coded to indicate the absolute rather than relative amount of differential sibling experience by disregarding the direction of the differential experience (i.e., 0 = no difference in sibling experiences; 1 = some difference; 2 = much difference).

The 11 scales of the SIDE (see Table 2) were devised using the results of factor analyses of data on a sample of 396 12- to 28-year-old siblings from the Denver metropolitan area. The word "differential" precedes the label for each scale to emphasize that all items involve relative (differential) ratings. The 2-week, test–retest reliabilities are reasonable, with a mean of .84 and a range from .70 to .94. The scales are virtually independent of siblings' age, birth-order, and gender. Also included in the table are sibling agreement correlations which indicate that siblings agree quite substantially, especially in the areas of differential sibling interaction and peer group characteristics. The sibling agreement correlations are .55, .73, and .60 concerning which sibling's peer group was more college oriented, delinquent, and popular, respectively. Siblings also agree substantially as to which sibling was more jealous (r = .56) and which sibling displayed more caretaking (r = .56). Siblings agree to a lesser extent on differences in parental treatment (r = .26 and .28 for maternal and paternal affection). The median sibling agreement correlation over the 11 SIDE scales is .49, which is above typical interrater agreement on personality and environmental paper-and-pencil measures. The high sibling agreement found for some of the SIDE scales may be due to the fact that siblings are asked to make a relative and specific comparison to their sibling rather than an absolute judgment in comparison to all other children of that age. Because the SIDE intentionally assesses siblings' perceptions of their differential experience, sibling agreement is not an important criterion for the usefulness of the measure as long as the measure is reliable. Other substantive findings from the SIDE are interwoven throughout the following discussion on the major categories of systematic nonshared environment.

5.4. Parental treatment. Environmental research has traditionally focused on parental treatment because parents appear first and foremost in young children's lives. It has not been easy, however, to document parental effects on children's development. A recent review of the relationship between parental treatment and children's development concludes that "in most cases, the relationships that have appeared are not large, if one thinks in terms of the amount of variance accounted for" (Maccoby & Martin 1983, p. 82). Indeed, these findings led the authors to argue for the need to examine intrafamilial variation in the parent–child relationship. It should be reiterated that the importance of nonshared environment does not denigrate the importance of environmental influence. Environmental influence is important but it operates differently from the way we thought it operated. In the case of parental influences, the effect that parents have on their children has little to do with those aspects of parenting that are experienced similarly by two children in their family. Whatever these parental influences might be, they differentiate rather than inte-

Table 2. *Scales of nonshared environmental influence from the Sibling Inventory of Differential Experience (SIDE)*

Category	Scale	Test–retest reliability	Sibling agreement
Sibling interaction	Differential Sibling Antagonism	.83	.39
	Differential Sibling Jealousy	.93	.56
	Differential Sibling Caretaking	.89	.56
	Differential Sibling Closeness	.70	.23
Parental treatment	Differential Maternal Affection	.82	.26
	Differential Maternal Control	.77	.25
	Differential Paternal Affection	.77	.28
	Differential Paternal Control	.85	.49
Peers	Differential Peer College Orientation	.88	.55
	Differential Peer Delinquency	.94	.73
	Differential Peer Popularity	.84	.60

Source: Adapted from Daniels and Plomin (1985).

grate the children. Parenting is likely to be an important source of environmental variance only if parents differentiate their children.

How similarly or differently do parents treat their offspring? The SIDE data indicate that siblings perceive their parents to treat them quite similarly: Only 9% of siblings report "much difference" and 35% report "a bit of difference" in their parents' treatment on the average across parental treatment items. For the four SIDE scales that assess parental treatment, the mean absolute score is .50 (0 refers to no reported difference in sibling experiences, 1 indexes some difference, and 2 indicates much difference). Other categories of nonshared environmental influence show greater differentiation within sibling pairs and are thus more likely to be important sources of nonshared environmental influence. Nonetheless, it is possible that small differences in siblings' perceptions of their parents' treatment lead to large differences in their development.

Another study of adolescent siblings found similar results, not just for adolescents' reports of their parents' treatment, but also for the parents' reports of their treatment of their children (Daniels, Dunn, Furstenberg & Plomin 1985). The 1981 follow-up of the longitudinal National Survey of Children (Furstenberg, Winquist-Nord, Peterson & Zill 1983) included 348 families with two siblings 11 to 17 years of age (mean age = 13.7 years) from a nationally representative sample of 1,077 families. In telephone interviews, each sibling and mother was interviewed individually concerning family cooperation, family stress, parental rule and chore expectations, closeness to mother and father, and child's say in decisions. In contrast to the SIDE study, the ratings of environment in this study are absolute in that parents and siblings were not asked to rate parental treatment as it differed for the two siblings. Sibling intraclass correlations for the measures of parental treatment, as rated by parents and by the siblings themselves, are listed in Table 3. The sibling correlations indicate the extent to which parents and siblings themselves perceive that siblings share similar parental treatment. These data indicate that parents perceive that they treat their two children quite similarly – the sibling correlations range from .38 to .65. In contrast, the siblings do not perceive that their parents' treatment of them is highly similar – the sibling correlations average about .20.

Two twin studies using "absolute" ratings of adolescents' perceptions of parental treatment (Rowe 1981; 1983) have found substantial correlations within twin pairs for parental treatment. Different measures of parental affection and control were used in the two twin studies, and twin correlations of about .45 emerged. Parents appear to treat children less similarly in this study, which used absolute ratings, than in the SIDE research, which used "relative" ratings. It is reasonable, however, that sibling correlations for absolute ratings of parental treatment are lower than those for relative ratings, because the absolute rating procedure asks each sibling to rate his parents' treatment in relation to all other parents; differences between the siblings' responses are used to compute a correlation. The relative approach is more direct for assessing differences in siblings' experiences because it asks them about parental treatment specifically in comparison to their sibling.

Table 3. *Sibling intraclass correlations for environmental measures in the national survey of children sample*

Environmental measure	Parental ratings	Sibling ratings
Family cooperation	—	.17
Family stress	—	.29*
Parental rule expectations	—	.18*
Parental chore expectations	.49*	.21*
Maternal closeness	.38*	.19*
Paternal closeness	.49*	.26*
Child's say in decisions	.65*	.18*

Note: N = 299–348 sibling pairs. * $p < .05$.
Source: Adapted from Daniels, Dunn, Furstenberg, and Plomin (1985).

Two analyses of sibling data in the Colorado Adoption Project include the first "objective" data concerning differential parental behavior towards siblings. Sibling correlations were reported for the interview/observation measure, Home Observation for Measurement of the Environment (HOME; Caldwell & Bradley 1978), for 133 sibling pairs in which both members of each pair were studied at 12 months of age and 103 sibling pairs were studied at 24 months (Daniels 1985). The average sibling spacing was nearly 3 years, nonetheless, the sibling correlations for the HOME were nearly as great as the stability of the HOME measure for all individuals from 12 to 24 months. Using the HOME data when each sibling was 12 months old, the sibling correlation for a general factor of the HOME was .42; at 24 months, it was .43. Sibling correlations at 12 months for the Family Environment Scales (FES; Moos & Moos 1981) also approached the 1-year stability of the measure. The FES is not at all specific to a particular child, however, because it assesses the general social climate of the home; the HOME is only somewhat specific to each child – some items, such as number of books present and visible, are likely to be similar for all children in the family.

The most impressive results suggesting that parents treat their several children similarly comes from a longitudinal study of 50 families in which mothers were videotaped while interacting individually with each of two siblings when each child was 12 months old (Dunn et al. 1985). The children were nearly 3 years apart in age, which means that the observations of maternal behavior toward the two children were separated by nearly three years. Maternal behavior was reliably assessed, and factor analysis yielded three factors: affection, verbal attention, and control. The results indicate that the mothers were remarkably consistent in their behavior toward their two children at the same age: Corrected for unreliability, the average correlation for maternal behavior toward two siblings was .70. These data suggest that differential maternal treatment of their children in infancy does not appear to be a major source of the marked individual differences within pairs of siblings. Other longitudinal studies on this topic agree that mothers are quite consistent in their behavior toward two of their children when the children are studied at the same age (Abra-

movitch, Pepler & Corter 1982; Dunn & Kendrick 1982; Jacobs & Moss 1976).

Nontwin siblings are in fact different in age. Subsequent work by Dunn and her colleagues has indicated that even though mothers treated their two children quite similarly when the children were the same age, longitudinal analyses from 12 to 24 months showed little stability for maternal behavior to the same child. The authors suggest that rank-order of the mothers on these dimensions changes from 12 to 24 months because different mothers respond differently to the new developmental advances of children. Analyses from a study using a very different methodology – extensive and intensive unstructured home observations of a sample of 80 British families – support the same interpretation (Dunn 1977). Individual differences in maternal responsiveness that were highly stable during the first year of life changed markedly with the developments in the children's communicative abilities in the second year. Similarly, in another study, correlations between 12 and 24 months in measures of maternal physical, affectionate, verbal, visual, and responsive behavior were very low: −.05, .05, .05, .04, and .17, respectively (Clarke-Stewart & Hevey 1981).

The implication of these results is that in a cross-sectional slice of time siblings differ in age and are treated quite differently. Thus, the possible effect of differential parental treatment on siblings of different age needs further exploration.

In summary, sibling reports, parental self-reports, and observational studies yield no clear conclusion concerning differential parental treatment. To the extent that parents treat their children similarly, we would not expect parental treatment to be a major source of nonshared environmental influence, although, as mentioned earlier, it is possible that small differences in parental treatment lead to large differences in development.

5.5. Sibling interaction. The possiblity that siblings' interactions with each other are a source of nonshared environmental influences has not been studied nearly as much as parental treatment. It is noteworthy, however, that the results of intensive observational studies of mother–sibling–sibling triads emphasize the importance of sibling–sibling interactions (Dunn 1983; Dunn & Kendrick 1982). Twin data on sibling interaction have been reported for 88 pairs of high-school twins (Rowe & Plomin 1981). For a Liking scale, the correlation for all 88 pairs was .61, indicating that twins' liking and disliking of each other is mutual. Twins generally like each other though (the average response was 4.4 on a 5-point scale), which means that this result involves only a small amount of variance. Two other scales, Respect and Understanding, yielded more variance than the Liking scale, and twin correlations of .35 and .30, respectively, indicated considerable differences within pairs of twins. These twin correlations for twins' respect for and understanding of each other are lower than those found in Rowe's studies of twins' perceptions of their parents' treatment, thus suggesting that siblings might provide more nonshared environment than do parents.

The SIDE explores sibling interaction with scales that assess differential sibling antagonism, caretaking, jealousy, and closeness. As indicated earlier, only 9% of 396 siblings reported "much difference" in their parents' treatment on the average and the mean absolute score is .50. In contrast, 19% of the siblings report "much difference" and 40% report "a bit of difference" in their siblings' treatment of them; the mean absolute score is .80 (Daniels & Plomin 1985).

In summary, although a few relevant studies have been reported, some data suggest that each member of a sibling pair may provide a substantially different environment for the other member of the pair, especially when the data are based on adolescents' self-reported perceptions. In terms of components of variance, one might predict that, to the extent that siblings affect one another, the variance of individuals who are siblings should exceed the variance of individuals who are singletons. We are not aware of any tests of this prediction. However, there may be other factors diluting this variance difference between siblings and singletons – for example, it is not implausible to suggest that parents of siblings have less of an effect on each of their children than do parents of singletons.

5.6. Peer characteristics. Even less is known about extrafamilial sources of nonshared environment such as peers. The only report of peers as a possible source of differential experience for siblings is based on the SIDE (Daniels & Plomin 1985). For the 26 peer characteristic items, 20% of the siblings report "much difference" and 42% report "a bit of difference" in their peer groups' characteristics. The mean absolute score is .83 for the three peer scales of the SIDE, which suggests that siblings experience peer differences as great as the differences they experience in their interaction with each other.

6. Relationships between nonshared factors and sibling differences in behavior

The first question in studies of nonshared environmental influences is whether such factors exist. The answer is clearly affirmative: Siblings in the same family experience different environments, perhaps with respect to parental treatment, and probably in their interaction with each other and in characteristics of their peer groups. The next question is whether these differences in experience are related to differences in behavioral development.

The study of adolescent siblings from the National Survey of Children (Daniels et al. 1985) related differential parental treatment to differences in sibling adjustment. As in all studies of personality and psychopathology, the siblings were only moderately similar for adjustment, with correlations of about .20, which means that the great majority of reliable variance is not shared by siblings. Table 4 lists multiple regression coefficients when sibling differences in adjustment measures are regressed on several of the sibling differences in environment listed in Table 3.

Most of the multiple regressions are significant, and adjusted R^2 values of about 10% on the average indicate that nonshared environmental influences are systematically related to differences in the siblings' adjustment. For example, the last row of Table 4 shows associations between nonshared environment and an aggregate measure of disobedience based on parent, sibling, and teacher ratings. The significant regressions indicate that for both parental and sibling ratings of sibling experience,

Table 4. *Multiple regressions of differences in sibling adjustment on differences in sibling environments*

Adjustment measure[a]	Multiple R's	
	Parental ratings of sibling differences in experience	Sibling ratings of sibling differences in experience
Parental report of emotional distress	.38*	.25
Parental report of delinquency	.37*	.25
Parental report of disobedience	.37*	.26
Self report of emotional distress	.12	.28*
Self-report of delinquency	.29*	.37*
Self-report of dissatisfaction	—[b]	.35*
Teacher report of disobedience	—[b]	.35*
Parent-sibling-teacher aggregate score of disobedience	.40*	.34*

[a]The multiple regressions involve sibling difference scores for each adjustment measure.
[b]Not available.
*$p < .05$.
Source: Adapted from Daniels, Dunn, Furstenberg, and Plomin (1985).

differential experiences of siblings are related to differences in disobedience. It is noteworthy that some significant relationships emerge when different individuals rate the siblings' adjustment and the siblings' environment. For example, parental perceptions of sibling differences in environment are related to differences in the siblings' own perception of delinquency, and sibling perceptions of environmental differences are related to teacher ratings of disobedience. With regard to the specific environmental differences that relate to sibling differences in the adjustment measures, both the parent and sibling reports of the environment converge on the finding that the sibling who experiences more maternal closeness, more sibling friendliness, more say in family decision making, and more parental chore expectations, as compared to the other sibling, is better adjusted psychologically.

Other studies that relate differential sibling experience to differences in siblings' behavior have been reported; however, these studies have used twin and adoption designs to test the possibility that such relationships are mediated genetically. For this reason, these studies are described in the following section.

7. Direction of effects

Once relationships are identified between any environmental factor and behavior, one can address the issue of direction of effects: Does the environmental factor affect or merely reflect differences among individuals (Bell 1968)? The direction-of-effects issue is just as relevant to the study of nonshared environmental influences as it is in traditional studies. For example, differential parental affection might be related to differences in siblings' sociability because preexisting differences in the siblings' sociability elicit differences in their parents' affection toward them.

Behavioral-genetic designs can be profitably applied to this issue because one possible explanation for a child-to-environment direction of effects is genetic differences between the siblings. That is, siblings might report differences in treatment that occur as a result of genetic differences between them. Finding genetic influence on a nonshared environmental measure suggests that genetic differences between the siblings underlies, at least in part, their experiential differences. There are two subsidiary issues: Do measures of nonshared environment show genetic influence? Are relationships between measures of nonshared environment and measures of behavior mediated genetically?

It should be noted that failure to find genetic influence does not prove that the measured nonshared environmental influence causes behavioral differences within pairs. It is possible, for example, that behavioral differences within pairs of siblings originate from prior experiences with which the contemporaneous measure of nonshared environment is correlated.

7.1. Do measures of nonshared environment show genetic influence? One study (Daniels & Plomin 1985) exists that explored the origins of differential sibling experi-

ence. SIDE data from 222 adoptive siblings were compared to data from 174 biological siblings. If the SIDE reflects genetic differences, mean SIDE differences should be greater for adoptive than for biological pairs because adoptive siblings are uncorrelated genetically in the absence of selective placement, whereas biological siblings correlate .50 genetically. Samples of this size have 80% power to detect mean differences in experience that account for as little as 2% of the variance. In general, the SIDE measures of differential experience were similar for adoptive and nonadoptive siblings: Average correlations were .76 for adoptive siblings and .69 for biological siblings. Thus, the SIDE scales on average suggest negligible genetic influence, which implies that the origins of perceived differential experience are indeed environmental. However, 4 of the 11 SIDE scales yielded significantly greater differences for adoptive siblings than for biological siblings that accounted for 4%–12% of the variance, thus suggesting slight genetic influence for some of the SIDE scales.

Finding little genetic influence on the SIDE measure is both surprising and interesting because behavioral genetic studies of most behavioral traits and of *shared* environmental measures do show considerable genetic influence (reviewed by Plomin 1986). Although replication of this finding is necessary, it may be that differential experiences of siblings are in fact insensitive to genetic differences between the siblings. Because siblings are asked to make relative comparisons to their other sibling on the SIDE, this micro-analysis may go beyond the genetic make-up of family members. Regardless of the explanation, it is noteworthy that this first study of the etiology of nonshared environment as assessed by the SIDE shows little evidence of genetic influence.

7.2. Are relationships between nonshared environment and behavior mediated genetically? Thus, one set of data has implied that nonshared environmental influences may be virtually uncontaminated by hereditary influences. If a measure of nonshared environment is not influenced by heredity, its relationship to behavioral differences is unlikely to be mediated genetically. Nonetheless, because so little work has been done in this area, it is important to ask the next question: whether genetic differences on measures of nonshared environment (assessed directly by the SIDE or indirectly through sibling difference scores on shared environment measures) are translated into behavioral differences between siblings. The possibility of genetic mediation of relationships between environment and behavior has recently been discussed (Plomin, Loehlin & DeFries 1985), although not in the context of nonshared environmental influences.

One way to study nonshared environment free of genetic bias is to relate experiential differences within pairs of identical twins to behavioral differences within the twin pairs. Because identical twins share exactly the same heredity, environmental and behavioral differences within pairs cannot be explained by genetic differences. Twin studies can also assess possible genetic influences by comparing the relationship between experiential and behavioral differences within identical twin pairs to the relationship within fraternal twin pairs. If heredity is influential, the correlations will be greater for fraternal twins than for identical twins because differences within pairs of fraternal twins are due to genetic differences as well as nonshared environmental influences. Although this approach has not been used systematically, a study by Rowe and Plomin (1981) examined the relationship between differences in interpersonal treatment of the twins and differences in self-reported personality. The authors noted that the relationships between twin differences in the measures of nonshared environment and twin differences in the measures of personality were generally weak for both identical and fraternal twins. The fact that the fraternal twin correlations were no greater than the identical twin correlations suggests that what little relationship exists between nonshared environment (as measured in this study in terms of the twins' perceptions of their interpersonal relationship) and personality does not appear to be mediated by heredity.

As mentioned earlier, twins probably share more environmental influences than do nontwin siblings. For this reason, the twin method is not a powerful approach to the study of relationships between nonshared environmental influences and behavioral differences. That is, twins may experience more similar environments and be more similar behaviorally than nontwin siblings. Another method that is less direct but might prove to be more generalizable to the nontwin situation is to compare correlations between nonshared environmental differences and behavioral differences for pairs of adoptive and nonadoptive siblings. Behavioral differences within pairs of nonadoptive siblings could be either genetic or environmental in origin because first-degree relatives are 50% similar genetically. In the absence of selective placement, however, adoptive sibling pairs do not resemble each other genetically. Thus, if the relationship between nonshared environmental measures and differences in sibling behaviors reflects genetic differences within pairs of siblings, we would expect correlations for sibling differences in environment and in behavior to be greater for adoptive siblings than for nonadoptive siblings.

A recent study of adoptive and nonadoptive infant siblings in the Colorado Adoption Project explored this issue (Daniels 1985). Although no measure designed specifically to assess differential sibling experiences was used, the HOME and FES were included. As mentioned earlier, the results suggested little differential experience for two siblings when each sibling's environment was assessed separately at the time the child was 12 months of age. However, even these slight differential experiences of the siblings as assessed by the HOME and FES showed some association (r's = .2 – .3) with behavioral differences between the infant siblings. For hundreds of comparisons between sibling differences on the HOME and FES and various sibling behavioral differences, over 13% were significant. For example, at 12 months, differences in the extent to which mothers consciously encouraged developmental advance (as measured by the HOME) correlated .31 with differences in the siblings' activity level (as assessed by the tester using the Infant Behavior Record). More to the point, this study showed only environmental mediation in that no differences were found between correlations for adoptive and nonadoptive sibling pairs.

In the only other study examining nonshared environment–behavior relationships, the SIDE scales were related to adolescent sibling personality differences

(Daniels, in press). In this study of adoptive and nonadoptive siblings, no genetic influence was detected, even though the SIDE accounted for 6%–26% of the variance of sibling personality difference scores. For example, the sibling who experienced more sibling closeness and peer popularity also reported more sociability as compared to his sibling; the sibling who reported more sibling jealousy and peer delinquency also reported more emotionality as compared to his sibling. Although longitudinal work is necessary to address the direction of effects in these relationships, it can at least be said that genetically influenced personality differences between the siblings do not lead to differences in their interactions with siblings and peers.

In summary, the results of these two studies suggest that, at least in infancy, heredity does not importantly mediate relationships between siblings' experiential differences and differences in their behavior. A reasonable priority for research would be to identify relationships between nonshared environment and sibling differences in behavior using nontwin siblings in nonadoptive families and to worry about the direction of effects only after such relationships are found.

8. Implications and conclusions

In this target article we have presented evidence that converges on the conclusion that children in the same family experience practically no shared environmental influence that makes them similar for behavioral traits. In other words, the effective environments of siblings are hardly any more similar than are the environments of strangers who grow up in different families. This conclusion has been put particularly forcefully by Scarr and Grajek (1982, p. 361):

> Lest the reader slip over these results, let us make explicit the implications of these findings: Upper middle-class brothers who attend the same school and whose parents take them to the same plays, sporting events, music lessons, and therapists, and use similar child rearing practices on them are little more similar in personality measures than they are to working class or farm boys, whose lives are totally different. Now, perhaps this is an exaggeration of the known facts, but not by much. Given the low correlations of biological siblings and the near zero correlations of adopted siblings, it is evident that most of the variance in personality arises in the environmental differences among siblings, *not* in the differences among families.

This unsettling fact is rich in implications for research, theory, and application. In terms of research implications, studies of the family environment and socialization can take advantage of the key of nonshared environment by studying more than one child per family in order to identify environmental factors that make children in a family so different from one another. Recent studies presented in this review indicate that this is a promising area for research.

The importance of nonshared environment also suggests the need for a theoretical reconceptualization of environmental influences in development. Most important, the child rather than the family must be considered the unit of socialization. The search for nonshared environmental influences will be aided by theories of the processes by which nonshared environment can lead to developmental differences between siblings. Nearly every psychological theory – including learning, psychoanalytic, Piagetian, ethological, biopsychological, family-system, and social-psychological theories – has something to offer when viewed from the perspective of nonshared environment. To mention but a few examples, learning theory offers sibling conditioning and modeling as processes by which nonshared environment may leave its mark; sibling deidentification and split-parent identification have emerged from psychoanalytic theory (Schachter 1982); social psychology could offer contrast effects and attribution differences as possible mediators of nonshared experience. Developing a coherent theory of the processes by which nonshared experience lead to differences between children in the same family is a high priority for the area.

Our new knowledge concerning the importance of nonshared environment may have its deepest implications for intervention. The data are descriptive, not proscriptive. That is, they indicate that, of the variability that exists in children's environments, the portion of the environmental variability that affects children's psychological development is nearly exclusively of the nonshared variety. It does not mean that shared environmental factors cannot or should not affect the development of children. Nonetheless, it is critical for interventionists to know, for example, that what parents do that is experienced similarly by their children does not have an impact on their behavioral development. If the effects of parents on their children lie in the unique environments they provide for each child, childrearing books need to be rewritten, and early childhood education and interventions aimed at the prevention of psychopathology need to be rethought. The importance of nonshared environments, as it works both systematically and stochastically, implies that the environmental impact on children works through the power of differentiation within the family. The possibly subtle differences experienced or perceived by children in the same family are the environmental factors that drive behavioral development.

In conclusion, although it was less than a decade ago that the importance of nonshared environmental influences was brought to the attention of behavioral scientists, the results of research in this area have led to the following conclusions:

1. Behavioral-genetic studies consistently point to nonshared environment as the most important source of environmental variance for personality, psychopathology, and IQ after childhood.

2. When more than one child is studied per family, it is apparent that siblings in the same family experience considerably different environments, in terms of their treatment of each other, in their peer interactions, and perhaps in terms of parental treatment.

3. Family composition variables such as birth-order and gender differences account for only a small (1%–5%) portion of the variance of sibling differences in development.

4. Differences in siblings' experiences relate significantly to siblings' differences in behavior, implying that nonshared environmental influences are at least in part systematic.

5. Measures of nonshared environment do not primarily reflect genetic differences between children in the same family.

The first conclusion is the strongest: Nonshared environment is a major part of the answer to the question posed in the title of this article. The other conclusions are better viewed as initial hypotheses for future research. Despite this attempt to impose some order on the results of the few extant studies of nonshared environment, questions are certainly more obvious than answers. A crucial question is whether most nonshared environmental variance is systematic. Other questions emerge from the recognition that nonshared environmental influences and their effects on behavioral development are likely to be specific: Which specific nonshared environmental factors account for most variance? Which sibling differences in behavior are most strongly related to specific nonshared influences? Is there a general theory predicting these relationships? What are the developmental provenances and processes of specific nonshared environmental factors and their relationship to behavioral differences between children in the same family?

A few faltering first steps have been taken toward exploring nonshared environmental influence. A long road lies ahead but, because most of the environmental variance that affects behavioral development is of the nonshared variety, this is surely an important road to travel.

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NOTE

1. The correlation is not squared because the issue is not whether we can predict one twin's score from the other twin's score. Rather, the issue is the extent to which observed variance is due to shared variance – that is, covariance – among the pairs. The correlation itself rather than its square expresses the proportion of total variance that is shared within pairs (Fisher 1918; Ozer 1985).

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Perceptions are nonshared environments

Irwin S. Bernstein

Department of Psychology, University of Georgia, Athens, Ga. 30602

There is a certain admirable elegance in the logic of behavior genetics. The relative contributions of heredity and environment, to the variance in the population are carefully teased apart using studies of fraternal and identical twins reared together and apart. Adding additional data on other relatives, siblings, adopted children, and other unrelated individuals reared in other families allows for remarkable precision in assessing the contributions of shared and nonshared environments to the variance. Of course, any student of statistics knows that the variance is extremely important, but not the whole story. In behavior genetics, however, without variation in the population we can make no estimate at all of the relative importance of heredity and environment in the development of a trait. This approach will never indicate how much of a trait is due to nature and how much is due to nurture, but it will be able to indicate how much of the variance in the trait is accounted for by each. We cannot really state that "adaptive sibling pairs do not resemble each other genetically." Of course they do. I don't know how many alleles are shared by members of the same taxon, but certainly if one adopted sibling were a chimpanzee we might conclude that most of the variance was genetic. The variance in the number of fingers people have is primarily due to accidents (nonshared environment), but this is not to say that there is no genetic input to the number of fingers on the human hand.

Of course, identical twins have identical genotypes, and this may predispose them to perceive things similarly, but inasmuch as no two objects can occupy the same place at the same time, the twins will have different world experiences. Genetic similarity is not unimportant, but variance due to nonshared environments will gradually increase. Plomin & Daniels (P&D) emphasize the developmentalist approach and their data support the gradual increase in individual variance during development. In their important studies demonstrating that mothers treat successive children very similarly at 12 months of age, they also note that as the child develops and differentiates, mothers respond to the behavior of the child and treat successive children differently at later ages. A mother's behavior is not simply a function of her internal motivations. The more different her children become, the more differently a mother treats them. The more differently the children are treated, the greater will be their nonshared environments and the differences due to nonshared environment.

Perhaps the most important contribution is P&D's position that the child is the unit of socialization. If we agree that the child constructs its own reality, then it clearly follows that it is the child's perception of the experience, rather than the objectively described experience, that is crucial. Identical twins with identical genetic predispositions will have nonidentical objectively described experiences which will then build different realities. These differences will then mean different perceptions of future experience such that what we may see as a common experience becomes part of their nonshared environment. The further along they get in development the more idiosyncratic become their experiences.

P&D state that "subjective perceived experiences may prove to be important." Furthermore, they state that "effective environments of siblings are hardly any more similar than are the environments of strangers." It is hard, however, to see how one could measure shared environments to support the idea that these "are unlikely to be important sources of environmental influence." If we use rating scale responses to indicate that children experienced what their parents did similarly and that this had no impact, our data are certainly indirect. "Possibly subtle differences experienced or perceived by chil-

dren . . . drive behavioral development." Stress "perceived" and you can drop "subtle."

All of this makes the study of developmental processes very difficult indeed. Objective data on behavior and interactions are not necessarily going to reveal what the "experience" is, nor which "experience" led to what effect. Asking a subject to introspect and self-report, on the telephone or directly, is not guaranteed to reveal what a subject is actually perceiving as reality. Rating scales, like the Sibling Inventory of Differential Experience (SIDE) (Sec. 7.1, para. 1), may be standard methods, and perhaps the best available, but terribly crude. If two siblings both agree that their mother was fair, then we have interobserver reliability, but no test of validity. Children may both agree that parents and mothers are to be portrayed in the best light when asked questions by others, whereas they can express sibling rivalry, or individual independence by exaggerating the differences in their friendships. Who can really tell why a subject (or two subjects) gives a particular answer? Humans are complex. It is this complexity and the lability of behavior in humans that allows them to fine tune their behavior as a function of experience.

Humans are the best example of the first law of animal behavior: "Individuals vary." Human variation may indeed be due to cognitive capacities, the ability to modify behavior as a function of experience and the ability to construct diverse perceptions and realities from the same objective reality. Studying this variance will continue to absorb many of us into the indefinite future. We are only beginning to shake off "introspectionism" in developing ways of studying awareness and perception.

Contributions of the biometrical approach to individual differences in personality measures

R. Darrell Bock and Michele F. Zimowski

Department of Behavioral Sciences, University of Chicago, Chicago, Ill. 60637

Plomin & Daniels (P&D) are to be congratulated for their effort to bring the methods of quantitative behavior-genetics to a wider audience. They describe these methods in the context of a specific source of variation in personality measures, namely, variation between siblings that cannot be accounted for by biological inheritance. The discussion is carried out almost exclusively in verbal terms without the usual variance component formulas and the partitioning of total variance into heritable, environmental, and error sources. This approach has both advantages and disadvantages: The advantages are that it does not put off the nonquantitative reader and that it allows fragmentary results to be brought into the discussion in qualitative terms; its disadvantage is that it does not place before the reader the scheme, unique to the biometrical approach, that yields a systematic identification and estimation of those sources of variation that account for the total variance in the population. Rather than lose the clarity and specificity that this formalization conveys, P&D might have made some attempt to assemble such information in an appendix. Those of us who are of the quantitative bent, who find our sense of direction faltering in the twists and turns of a long verbal argument, would benefit from the fixed landmarks of the quantitative formulation.

One thing that the variance source table would reveal is that the component attributable to P&D's nonshared environment is estimated as a remainder – a balance category that makes up the difference between the total and the other sources of variation. This expedient is necessary because the nonshared component is not directly measurable in their approach. In consequence, any source of variation in the table that is underestimated or missing will inflate the estimate of the variation from the source that is a remainder. In appraising the evidence presented by P&D we must ask whether they have underestimated any of the other sources or neglected one or more. There is reason to believe that both of these are real possibilities here.

The source of variation that may be seriously underestimated, especially in studies that rely on personality inventories and self-reports, is measurement error. We know from the studies of method effects, as revealed in the analysis of multimethod–multitrait matrices, that the test–retest reliabilities used by P&D often grossly overestimate the dependability of the trait measurement. Test–retest reliability confounds trait variance with the many method and response-set artifacts that inflate correlations between scores obtained by the same or similar methods. The multimethod–multitrait studies of personality measures show that reports on questionnaires, in particular, are especially subject to these artifactual effects. Research on personality variables needs to include multiple instruments, as different as possible in format and procedure, for measuring the trait in question. A quantitative measure of the reliable trait variation and its structural relation to other variables can then be estimated from these multiple measurements. The problem is that components-of-variance analysis is, in effect, an estimation of variation in differences. The relative contribution of error to these differences is much greater than to the original scores and must be accurately estimated if the partition of variance is to be taken at face value.

More accurate assessments of the error variance components in the present study would undoubtedly lead to a more conservative estimate of the amount of between-sibling variation within family that cannot be accounted for by biological inheritance. The result would not necessarily alter P&D's conclusion that there is an important source of nonshared variation between siblings, but it might bring the figures for personality measures more in line with the generally more dependable cognitive measures, such as IQ. Data such as those of Bouchard and McGue (1981) would suggest that the nonshared component in IQ measures is a rather small fraction of the total variation.

A source of variation that P&D seem to omit entirely is the influence of the prenatal environment. Many recent studies have suggested that there is much more variation in the fetal environment, often deleterious and long-lasting, due to the contingencies of the hormonal status of the mother during pregnancy, histo-incompatibilities between mother and fetus, placental insufficiency, birth trauma, and so forth, than is commonly appreciated or acknowledged. Some of these effects are especially severe in twins, both identical and fraternal, and are believed to account for much of the variation between identical twins that P&D use as a measure of the nonshared environment. There is considerable evidence that the discordance of identical twins for psychopathology, for example, can arise from this source. Moreover, recent studies provide evidence that there are clear personality effects of brain damage, especially that affecting the right frontal lobe, which suggests the importance of unfavorable pre- and perinatal events on lifelong behavioral patterns. This source of variation should be on the agenda of future studies of unshared environmental influences both of personality and cognitive measures.

Nevertheless, we tend to agree with P&D that the decisive role in children's behavioral development often assigned to within-family influences has never been supported by objective data. It is easy to forget that as children grow older, they spend more of their waking hours outside the home than in. In pressing this point in the final section of the paper, however, P&D seem to go beyond the more guarded discussion of earlier sections. They extend their argument to cover not just personality and psychopathology, but also IQ after childhood. The

reader might be led to believe that the argument would apply to cognitive proficiencies, as indicated by the quote from Scarr and Grajek (1982) to the effect that upper-middle-class siblings are as similar to working-class children in personality measures as they are to each other. We know from the transclass adoption studies of Schiff, Duyme, Dumaret and Tomkiewicz (1982) and the transracial adoption studies of Scarr and Weinberg (1976) and Moore (1980), that there is a nearly one sigma increase in IQ associated with the moving of children at a young age from a culturally deprived to a middle-class environment. (See also Bock & Moore 1986.) It is therefore quite clear that the advantages children share in the more favorable environment do have an important impact on cognitive performance.

Admittedly, there is no evidence that these gains are specific effects of different practices of child rearing within the family. They are just as likely, or perhaps more likely, to be the result of the total impact of the peer environment, the community, *and* the family. It is perhaps significant in this connection that in families in which the parents speak a language different from the larger community, or have a variant accent, children invariably grow up speaking the language and accent of the community and not that of the home. This again suggests the extent to which we tend to overestimate the importance of parental influence on the cognitive development of children.

If the conclusions of the transclass and transracial adoption studies concerning IQ do not apply to personality measures, the reason may be merely that the latter instruments are not measuring any definite trait, or at least not measuring it with sufficient accuracy to be valid for the small differences that must be assessed in quantitative genetic studies. Another, and rather unsettling, possiblity is that the attributes we call "personality" are precisely those aspects of behavior that are *not* subject to shaping by example, percept, instruction, or education in the shared environment. Perhaps that is why we do not attempt to shape personality by formal education. If the latter explanation is correct, the variables among which we should look for shared environmental effects are not personality measures, but those that measure learned manners, school achievement, manual skills, and so forth. It is here that the contribution of family environment to resemblance between siblings should be more clear, and it should extend to adoptive siblings as well as to natural siblings. If different classes of behavioral variables respond differently to genetic and to shared and nonshared environmental influences, then the biometric approach needs to be broadly multivariate. It should provide comparisons and contrasts between behavioral variables as well as between the sources of variation.

The unmapped methodological territory between one gene and many comprises some intriguing environments

Charles E. Boklage

Genetics Program, School of Medicine, East Carolina University, Greenville, N.C. 27834

Plomin & Daniels (P&D) must be congratulated for pulling a useful if potentially disturbing heuristic structure out of some difficult material into much better light.

One problem with appreciating their work, a problem that affects the work remaining to be done, is that "environment" is still the error term in quantitative genetic analysis. Even supposing that most of genetics is more concrete than most of psychology, it should be clear that mechanisms of genetic influence are also much more complex than is understood. Genetic variance not effectively estimated by the prevailing paradigm and confounded with "environment" may be substantial.

What is "genetic" under quantitative genetic analysis models is limited to *measured* effects of those influences of which the average pair of first-degree relatives share 50%. The variation not correlated with such measures is left for attribution to causes outside (in the most precise version I have seen) the zygote nucleus or (more usually) the individual after birth.

Under the simplifying assumptions of equality and additivity among the independently assorted and expressed Mendelian members of a postulated set of polygenes, quantitative genetic models consider contributions from gene–gene interaction (epistatic and dominance variance components) to be zero, and they usually do the same with gene–environment interaction. Unless estimated heritability exceeds 100%, or is significantly greater from sib–pair than from parent–child estimates, little thought is given to dominance. Epistasis has been so intractable that even the most cogent arguments against its likelihood of significant influence sound very much like whistling in the dark.

The lack-of-fit, defined by the model as "nongenetic," thus "environmental," includes not only every life experience, but also every gene, every gene–gene interaction and every gene–environment interaction which is not specifically represented in the model.

Suppose the word "environment" could be removed from every such writing as this, and replaced with "that which is not explained by the kinds of genetic influence of which first-degree relatives have 50% in common."

The variance not modeled as genetic and confounded with environment would include all of extranuclear inheritance. Mitochondrial inheritance might appear as "maternal effects." Mitochondrial-nuclear interactions must be expected from the known cell physiology, but considerations of their transmission are primitive. Stable messenger RNAs in oocytes probably direct most of the first two weeks of development, but any variation there will not show a Mendelian distribution of phenotypes. It is not clear that monozygotic twins would be, or that dizygotics would not be, more alike than siblings for traits influenced by any such mechanism. Twins still seem potentially useful, but more for the differences between their development and that of the other 98+% of the human population than for anything they may contribute to fractionating behavioral variance in the general population.

Gene–environment interactions are getting better attention, as seen in P&D. However difficult, gene–gene interactions also need and deserve much more sophisticated attention. Even catastrophic disorders transmitted in "simple" Mendelian fashion vary in expression among their victims. Among families, such variation may well represent multiple mutant alleles, but differences between sibs urge the conclusion that few if any human genes act alone. Given the usual definition of the terms of genetic models, the effects of such interactions might do much to explain sib–sib differences in "that which (etc.)."

The methodological repertory of genetic analysis contains little or nothing useful for dealing with the prospect that the first and perhaps most pervasive elements (of "that which [etc.]") are the products of other genes. One sees only occasional two-gene models, having little hope of support without joint measurements of two or more phenotypes.

It is impossible for me to believe that the human genetic protocol consists only of independent effects of single loci, or that their only interactions are additive. Another limitation on effective extrapolation from one gene to many is the tendency to think in terms of two (kinds of) alleles at each locus, differing greatly in functionality, one of which is rare. The math is quite different when there are many, especially if differences in frequency or functionality are not dramatic. The intervening methodological territory, over which we travel in presumptive strides of unknown length, is unknown in size or shape.

The usual focus is on the fact that I share half my genes with each sib or parent. But if just one gene heterozygous in either parent also happens to be involved in even one significant two-

way interaction with any other gene that happens to be heterozygous in either parent, our chance of exactly matching in effective genotype is halved. The differences are genetic, but not represented as such in the additive model, making them part of "that which (etc.)"; and they are at least not fully shared. The utility of refining the fractionation of "environmental" variance is therefore limited by the extent to which it might well not be environmental at all.

Given the same parental genotypes and the same potential distribution of the elements thereof, the a priori probability of one sibling having a given genotype will be the same as that for another of the same sex, regardless of the transmission mechanism. For all but the simplest modes of transmission, that number is small and its square very small.

Very early in our genetics course for medical students we attempt to focus their attention on the mechanisms by which genetic diversity among humans is generated and distributed, and to convey in our lectures some appreciation of its potential range, by some variation of the following:

> Human gametogenesis selects one from each of 23 pairs of chromosomes in each parent. Any given child represents one of 2^{46} (about 70 trillion) possible choices of a normal set of chromosomes from those of the parents, assuming each chromosome comes to the child exactly as it passed from a grandparent to one of the parents. They rarely do that. Individual genes mutate, and members of chromosome pairs exchange parts with each other before being sorted into future gametes. This recombination averages in excess of one exchange per chromosome arm, the longer arms having more.
>
> Recombination of genes between chromosome homologues increases potential genetic variation. Each point on the "average chromosome" at which recombination might take place doubles the *base* of the exponentiation. Given even one point on each chromosome arm (two per chromosome) at which recombination might occur, the number of available structures for each chromosome increases from two to eight, and potential genotypes from 2^{46} to 8^{46}. This is more than the total of human gametes ever fertilized, and still a drastic underestimate. The children of unrelated parents have more differences from which to choose.

It seems at least as reasonable to consider the observed range of sibling differences anomalously narrow as to believe it oddly broad. Perhaps many of the possible combinations are phenotypically equivalent. The extent of euploid prenatal mortality, plus postnatal lethals, indicates that perhaps as many as half are forbidden. The potential variety, even between siblings, remains astronomical. I am not satisfied that current linear genetic models account for anywhere near all of it.

Absence or underestimation of shared environment?

Dorret I. Boomsma

Department of Experimental Psychology, Free University, Amsterdam, The Netherlands

How different children from the same family can be is well illustrated by the famous Mitford family. The six sisters from this upper-class English family were brought up in the twenties and thirties under very similar conditions: Not allowed to go to school or to have friends outside the family, they were all educated at home. Two sisters became convinced fascists. Unity lived in Germany as a member of Hitler's inner circle until the war broke out and Diana married the head of the British Union of Fascists. Of the other sisters, two did not seem to have any interest in politics, Nancy flirted a little with socialism, and Jessica became a devoted member of the American Communist Party. In her own account of these events Jessica Mitford (1977, p. 24) asks: "What propelled us in these different directions?" Her answer is "the Zeitgeist of the thirties." And in "Unity Mitford: A Quest," David Pryce-Jones (1976, p. 4) writes: "Out of this childhood . . . the one evolved as a fascist and the other as a communist: one experience, but two outcomes."

Plomin & Daniels's (P&D's) target article is an important contribution towards understanding such differences among children of the same family. They have performed a useful service in demonstrating that behavior-genetic methods can be used, not only to estimate genetic variance, but also to partition environmental variance into a part shared by family members and a part unique to each individual. Their discussion of research on psychological characteristics can be supplemented with other examples: The importance of nonshared environmental factors is also evident in physiological measures such as lipid and lipoprotein levels (e.g., Namboodiri, Kaplan, Heuch, Elston, Green, Rao, Laskarzewski, Glueck, & Rifkind 1985) and blood pressure (e.g., Iselius, Morton & Rao 1983).

When various measures seem to show such a consistent picture of large unique influences and absence of shared family environment then either the phenomenon is real or there is a chance that our methods somehow underestimate the importance of shared environmental factors. One crucial assumption in behavior-genetics research is that all participating groups represent a random sample of all genotypes and environments present in the population. If nonparticipation is spread along the entire range of family environments, no problem arises. If certain groups are less likely to participate, however, then the effects of shared environment will be underestimated. In general, because children are adopted by parents of above average IQ and socioeconomic status, there is a fair chance that adoption studies are biased against detecting the influence of shared family environment. Alternatively, the absence of shared environmental effects in the American population may be caused by the large degree of uniformity of the environment produced by the schools and other public agencies (Woodworth 1941, in Scarr & Carter-Saltzman 1983, p. 219–20). In his discussion of a French adoption study by Schiff, Duyme, Dumaret, Stewart, Tomkiewicz, and Feingold (1978), Willerman (1979) also suggested that the standard deviation of environments sampled in American adoption studies may be too narrow to provide a test of social class environmental effects. Schiff et al. compared adopted children reared in high socioeconomic environments with their own full or half siblings who were brought up by their true mother in a lower social class. Adopted children had higher IQ (110.6 versus 94.7) and less failure in school (13% versus 55%). This contrast is close to that in the general population between children of upper-middle-class parents and unskilled workers. Schiff et al. emphasized that the adopted children and their own siblings are biologically equivalent so that the contrast between them is mainly of environmental origin.

Even if nonshared environmental factors are important in explaining sibling differences, nonshared environment is not the only part of the answer to the question posed in the title of P&D's article. Only for differences between identical twins are nonshared environmental factors the sole explanation. Jinks and Fulker (1970) showed how genetic variance may also be partitioned into genetic effects between and within families. If the behavior we study is influenced by genetic factors then the other part of the answer is genetic factors that are not shared by family members.

Finally, P&D's question of whether most nonshared variance is systematic or specific may be answered by multivariate behavior-genetics methods such as those developed by Martin and Eaves (1977) and Fulker (1979). Martin and Eaves found, in their analysis of monozygotic (MZ) and dizygotic (DZ) twins on five of Thurstone's Primary Mental Abilities, that most nonshared environmental variance was specific. That is, the environmental factors not shared by family members were also specific to different mental abilities. In contrast, the influence of shared environmental (and genetic) factors was more systematic in that they had a general influence on all abilities. Fulker (1979)

provides a multivariate analysis of specific cognitive skills. According to his analysis of Lochlin and Nicols's large twin study of the National Merit Scholarship Qualifying Test, social environment appears to be very important in cognitive development. His analysis also suggests that shared family environmental effects are of a similar nature, whatever cognitive skills are involved, and that nonshared family influences exert little general effect. In the same article Fulker also carried out a multivariate genetic analysis of Taubman's twin data on schooling, occupation, and earnings. In this example, the general nature of shared family environment was even clearer. Moreover, this example showed that genetic and shared environmental factors that influence schooling subsequently influence adult occupational status and income. Nonshared environmental factors had little later influence. However, large independent genetic and nonshared environmental effects played the major role in explaining later differences in occupation and income.

Evolutionary hypotheses and behavioral genetic methods: Hopes for a union of two disparate disciplines

David M. Buss

Department of Psychology, University of Michigan, Ann Arbor, Mich. 48109

Plomin & Daniels (P&D) present one of the most startling and important findings to emerge from behavioral-genetic research in the past decade – the pervasiveness of nonshared environmental influences on personality, psychopathology, and cognition. The finding is startling because nearly all current theories of environmental influence emphasize, implicitly or explicitly, environmental factors shared by children within the same family, such as socioeconomic status or common parental child-rearing attitudes, and these factors appear now to be of little causal import. The finding is important because it remains one of the few clues we currently have about how environments influence dispositions.

Because of its recency, little theoretical guidance yet exists to facilitate the empirical search for systematic sources of nonshared environmental influences. Recent work in evolutionary biology, however, points directly to specific hypotheses that can be tested with behavioral-genetic methods. This commentary will address a few of these. The comments are meant to suggest the possibility of a fruitful union between two biologically oriented disciplines that have developed in apparent isolation from one another: *behavioral genetics*, which has developed powerful methods that can be applied to nearly any domain and are therefore largely content- and theory-free, and *evolutionary biology*, which has proven a fertile source of hypotheses but, at least with humans, has not developed powerful methods to test these hypotheses.

The following list provides some evolutionarily based hypotheses about systematic sources of nonshared environmental influences that could be tested with behavioral-genetic methods:

Hypothesis 1: *Parents will expend more effort and confer greater resources on those children that are best able to translate parental investment into reproduction.* Based on Trivers's (1972) theory of differential parental investment, strategies that ensure distribution of parental investment to offspring that can best convert such favoritism into gene copies will be selected (see also Alexander 1979). [See also Vining: "Social Versus Reproductive Success" *BBS* 9(1) 1986.]

Hypothesis 2: *Because optimal reproductive strategies typically differ for human males and females, parents will socialize their male and female offspring differently.* Specifically, males will be socialized to be more aggressive, hasty, and wanton in sexual conduct. Females will be socialized to be more circumspect, cautious, and coy in sexual conduct. Furthermore, males will be socialized to embody characteristics that females value highly in mate selection (e.g., ambition, industry, good financial status); females will be socialized to embody characteristics that males highly value in mate selection (e.g., frugality, physical attractiveness) (Buss 1985; in press; Buss & Barnes 1986; Symons 1979; Trivers 1972). [See also multiple book review of Symons's *Evolution of human sexuality*, *BBS* 3(2) 1980.]

Hypothesis 3: *Parental favoritism toward male or female children will interact with the socioeconomic status of the parents.* Specifically, males will be more favored by their parents in higher socioeconomic groups, whereas females will be more favored by their parents in lower socioeconomic groups (Alexander 1974; Dickmann 1979; Trivers & Willard 1973).

Hypothesis 4: *Characteristics that covary with paternity confidence (e.g., physical or behavioral similarity to the father; effectiveness of mate-guarding tactics) will partly drive differential parental investment* (cf. Daly & Wilson 1982). The more similar a child is to the father, the greater will be the father's investment in that child. The evolutionary rationales stemming from theories of paternity confidence (Alexander 1979; Hartung 1985) and of genetic similarity (Rushton 1984) yield this prediction.

Hypothesis 5: *Birth-order will sometimes determine parental favoritism.* Older children are sometimes better able to translate parental investment into gene copies and so will be favored under certain conditions (Alexander 1979). Major exceptions will occur with the last-born as parental investment need not be saved for additional or future children (Alexander 1979).

These hypotheses, drawn from the existing literature, represent a small sampling of differential environments implied by evolutionary concept. They remain to be operationalized. Their potential effects on differential behavioral development (e.g., self-esteem, ambition, industry, coyness, aggression, impulsivity, dominance) also remain to be examined. Additional evolutionary hypotheses will undoubtedly be generated, and many will prove to be misguided or false upon empirical scrutiny. But evolutionary theory does provide a rich theoretical perspective that can guide the search for systematic sources of nonshared experiences and their behavioral consequences. Evolutionary hypotheses can be tested with behavioral genetic methods. Drawing on each other's strengths in this way provides a step toward unifying these powerful scientific disciplines. [See also multiple book review of Kitcher's *Vaulting Ambition*, *BBS* 9(4) 1986.]

Genes and environmental factors in the determination of behavioral characters

Ernst W. Caspari

Department of Biology, University of Rochester, Rochester, N.Y. 14627

It is well established that the expression of all phenotypic characters, quantitative as well as Mendelian, depends to varying degrees on both genetic and environmental influences. In the last few years our information about the nature and functioning of individual genes has become much more concrete. Environmental influences can be controlled in certain types of experiments, such as studies of the growth of microorganisms and plants, and even studies of some developmental processes in animals such as the fly *Drosophila*. In some more general characters, however, the environmental and genetic factors involved in the expression of particular phenotypes are ill defined and difficult to analyze. These characters include fitness, life cycle characters, and behavioral traits. In the case of behavior, this is partly because the definition of environment is frequently negative. The twin design and the sibling adoption

design provide evidence primarily for genetic influences; environmental factors are often defined as the remainder of the variance which cannot be accounted for by genes.

Because in the last few years much progress has been made in the study of the function of genes, this topic will be briefly discussed here. For some time, genes have been divided into structural and regulatory genes. This division has been defined more clearly by Paigen (1971; Lusis & Paigen 1975) who distinguished between structural, regulatory, architectural, and temporal genes. It has turned out that many of the "regulatory" genes are themselves "structural" genes in so far as they determine the amino acid composition of other proteins, suppressor and enhancer proteins or receptor proteins. An example is the so-called P_1-450 protein in the mouse, a protein that can be induced by treatment with certain toxic substances. The ability to react to these toxic substances with the production or elevation of P_1-450 protein is dependent on a gene *Ah*. This gene determines the structure of a receptor protein which is necessary to transport the toxic substance into the cell, that is, from the point of view of the P_1-450 protein gene *Ah* is a regulatory gene (Nebert, Felton & Robinson 1975; Nebert, Negishi, Lang, Hjelmeland & Eisen 1982).

A gene can be defined as a stretch of DNA which can be transcribed into RNA. For every protein there is a gene which is responsible for its structure. Regulation of gene action involves several different aspects: Particular genes may be active continually, or they may be turned on only in specific cells and at specific developmental times. This aspect may be regulated by different proteins, such as the *Ah* receptor protein mentioned above. In addition there are, in the neighborhood of structural genes, usually in flanking regions, specific DNA sequences which determine the cells in which a gene becomes active, and the time in development when it becomes active. The amounts of a particular protein are determined by DNA sequences close to a gene, the allelic state of the gene itself, and by other genes (Benyajati 1984).

DNA, besides its ability to be transcribed, also has the ability to react specifically with certain proteins. The best examples are the restriction enzymes which cut DNA at certain sequences, as well as the enzymes involved in DNA replication and transcription. These controlling DNA sequences differ from "structural" DNA by the fact that they do not have to be transcribed and translated to exert their function. They thus affect only the structural gene located on the same chromosome, acting in *cis* position. They may be relatively short but are able to combine with certain proteins and thus give rise to a reaction.

It is useful to distinguish between phenotypic characters which are "close" to a gene or "remote" from a gene. The character closest to a structural gene is the amino acid sequence of its protein, the primary structure. One step removed are characters which depend directly on the activity of the protein, that is, the amount of product and precursor of an enzymatic reaction. Characters remote from the gene involve many proteins and therefore many genes which cannot be identified individually; they are the subject of quantitative genetics. The relation of quantitative genetics to Mendelian genetics has been discussed by Caspari (1977). For present purposes it is sufficient to state that quantitative genetic systems are in principle identical to Mendelian genetic systems with all the complications of simple genetic systems indicated earlier. The methods of analysis, however, are quite different. Because the vertebrate brain contains a large number of proteins and polypeptides that are determined by genes and whose cellular function may depend on specific receptor proteins, it is not surprising that behavioral characters belong to the "remote" category which has to be analyzed by the methods of quantitative genetics.

These methods consist primarily in partitioning the variance for a character. The primary partition is the partition into genetic and environmental influences. Even this is not so easy because gene–environment interaction and covariance between genes and environment may occur. Most of the further partitioning affects the genetic component. Heritability can be divided into "broad" and "narrow" heritability (Lush 1949). The genetic variance can be separated into additive and nonadditive components, the latter including dominance, epistasis, and maternal effects. On the other hand, a further subdivision of the environmental variance has only recently been started. This may be due in part to the fact mentioned above: that environmental variance was originally defined negatively, the part of the variance not dependent on genes.

Human behavior is strongly affected by environmental influences, and there has actually been much interest and a great amount of work concerned with this component. It is to the great merit of the target article by Plomin & Daniels that a first subdivision of environmental factors into shared and nonshared components has been proposed and methods for evaluating them have been described. The result is that, at least for the phenotypes studied, the nonshared component is predominant. This result is completely unexpected and contradicts assumptions which had previously been made about the environmental component. Thus, many of the studies carried out previously may be less important and pertinent than had been assumed.

Let us consider the roles of temperament and of fortuitous events

Stella Chess

Department of Psychiatry, New York University Medical Center, New York, N.Y. 10016

It would appear reasonable to expect that brothers and sisters will show greater psychological similarity to each other compared to children from other families. After all, sibs do share some genetic inheritance and are exposed to many similar, if not identical, experiences when raised by the same parents in the same family structure. We would not expect them to be carbon copies of each other, but at least to show some degree of similarity to each other when compared to pairs of children from other families.

Yet the facts belie this reasonable idea, as they so often do. The marked differences in the personality characteristics of sibs has been noted by clinicians, including myself. Over the years working with families that had children with behavior problems it has become clear to me that parents often treat their children differently and that even when they treat them alike, the effect on sibs might be quite different. Fortuitous and unexpected talents, opportunities, or misfortunes could profoundly influence the development of any individual child's personality. We have been struck by this same observation of the marked differences among brothers and sisters as we have followed the developmental courses of the subjects in our New York Longitudinal Study from infancy to adult life.

A number of developmental psychologists have also recently pointed out this issue of the striking differences among children raised in the same family (Kagan 1984; Maccoby & Martin 1983; McCall 1983; Scarr & Grojek 1982). A typical formulation from this literature is McCall's statement that the "environmental variation that occurs within families but is not shared by siblings – nonshared environmental variation – is a major influence on general mental performance and has largely been ignored" (1983, p. 408).

The target article by Plomin & Daniels (P&D), now provides us with a wealth of data on this subject of the differences between sibs. They have analyzed genetic and environmental influences from the vantage point of behavioral genetics, using both adoptive and twin designs. They have considered the issue of shared and nonshared family environment in identical twins, fraternal twins, sibs, adoptees, and singletons. Their findings

are definitive and justify their categorical conclusion that "environmental influences make two children in the same family as different from one another as are pairs of children selected randomly from the population" (Abstract). Though their paper will undoubtedly represent the authoritative study of this issue, I feel that P&D could have given greater recognition to the similar observations made earlier by other investigators.

P&D discuss briefly some directions for future research on the causes of these marked differences among children in the same family. Surprisingly enough, the influence of temperamental differences among children in the same family is not mentioned, except for a casual reference on (Sec. 2.1, para. 7), even though Plomin himself has been an important investigator in the field of temperament. A number of researchers and a large series of publications, including those from our New York Longitudinal Study, which we began in 1956, have demonstrated the functional significance of temperamental characteristics for the child's psychologic development (Ciba Foundation Symposium 89, 1982; Plomin & Dunn 1986; Thomas & Chess 1977; Thomas, Chess & Birch 1968).

Parents may make the same demands on two children with different temperaments, and the effects on the children will be different. Thus, for example, parents may expect their child to adjust quickly and easily to beginning school, but this may only be possible for the child who responds to new situations positively and adapts quickly to change. Successful adjustment will meet with parental approval and praise, making the parent–child interaction a positive one. If the child instead responds to new situations uneasily, tends to try to withdraw from them, and adapts slowly to change, the child will find the initial adjustment to school difficult. If the parents do not recognize this response as normal for the child, given the child's temperament, they may criticize and demand a quickness of adjustment of which the child is not capable. The parent–child interaction will be negative, in contrast to the positive interchange with the first case. In effect, the two children are experiencing the same parental attitudes and expectations, yet the effects are different, and the two children are experiencing a radically different nonshared environment.

Furthermore, the child's temperament may have an important influence on the parental attitudes and behavior. A child may have the temperamental pattern we have called the "difficult child" (biologic irregularity, withdrawal from the new, slow adaptability, and relatively frequent intense mood expressions), which make his care and rearing difficult. This may create all kinds of adverse reactions in the parents – confusion, guilt, anger, vacillation. These attitudes may then affect the parents' behavior to the child in many ways and in many different situations. If, on the other hand, the youngster is temperamentally an easy child (the characteristics opposite to those of the difficult child), the parents will respond differently – with pleasure, a sense of ease, and approval of the child. These attitudes will in turn affect the parent–child relationship in many different ways. Again, the difficult versus the easy child often experiences different nonshared environments.

Many other examples can be given. A child with a high activity level who gets restless if confined to one spot for several hours may experience the same long automobile trip very differently from his low activity level sib who can sit easily through the same trip. The girl with a low sensory threshold who is uncomfortable with any rough or tight clothing may evoke parental reactions of bewilderment, annoyance, or victimization each morning while being dressed for school; reactions that will not occur toward the sib with a high sensory threshold.

It might be argued that, since several studies have shown a genetic factor in temperament (Torgersen & Kringlen 1978) – Plomin has even considered such a genetic factor as an essential component to his definition of temperament (Buss & Plomin 1975) – and that this would tend to make sibs more alike than nonsibs. Being more similar, they could then stimulate some degree of similarity in their parents' reactions and behavior. However, the issue is not that simple. Two sibs may be similar in temperament and yet may evoke different parental responses because of age differences or other characteristics. As an example, in our longitudinal study, two daughters both had similar patterns of difficult temperament. The older girl, the first to come along, became the family scapegoat and suffered severe criticism and punishment, especially from the father. The father was so intent on making this older child conform to his impossible expectations, that he ignored the same issues in the younger girl. As a result, the two girls, though similar in temperament, had very different parent–child relationships, which was reflected in marked differences in their psychologic characteristics.

We are by no means saying that differences in temperament will cause all or even most of the nonshared environment of brothers and sisters. These differences may not even be influential, if the parents understand their child and respond appropriately with what we have called a "goodness of fit" between parent and child. Where the parents lack this understanding, as in the examples given above, there could be a "poorness of fit" between parental expectations and the child's characteristics, and the extent of nonshared environment between sibs with different temperaments will be accentuated (Chess & Thomas 1984).

Goodness or poorness of fit between parent and child can affect the extent of the children's nonshared environment in other ways. A child who meets the parents' expectations for intellectual functioning, athletic skills, or even physical appearance will experience a different environment than the child who fails to meet the parental standards.

Finally, I have one specific difference with P&D's judgments. They tend to minimize the effect of accidental, unpredictable events, and state that "such capricious events, however, are likely to prove a dead end for research." It is my distinct impression from our New York Longitudinal Study (Chess & Thomas 1984) however, that such unpredictable factors may play a significant role in a child's development. An unexpected talent may emerge which may alter dramatically the parents' attitudes and behavior toward that child. The death of a parent or parental divorce may have different effects on sibs, depending on age and various idiosyncratic factors. A severe illness or accident may alter the child's place and experiences in the family. Considering such accidental factors may make research into the nonshared environment difficult but it cannot be written off as "a dead end for research." To ignore this issue runs the risk of overlooking highly significant influences in individual children and families.

On the need for longitudinal evidence and multiple measures in behavioral-genetic studies of adult personality

Paul T. Costa, Jr. and Robert R. McCrae

Gerontology Research Center, National Institute on Aging, NIH, Baltimore, Md. 21224

If a poll were taken today of clinical, developmental, and personality psychologists, a majority would probably assert that personality is largely the product of experience, and that parental child-rearing styles are the single most important determinant. The studies cited by Plomin & Daniels (P&D) give double offense, first in insisting on the importance of genetic effects on personality, and second in denying the importance of the environmental influences most commonly assumed to be crucial. The opposition between the received wisdom of most psychol-

ogists and the nearly unanimous findings of behavioral-genetic studies is an enigma that can only partially be explained by American psychologists' ideological preference for environmental explanations. If P&D are right, why hasn't the sheer weight of evidence forced developmentalists and clinicians to a new point of view?

Part of the answer, we think, lies in the failure of psychologists to use longitudinal methods to trace the course of development over the lifespan. Clinical psychologists who work with adults often find it useful to attempt to understand current problems in the context of childhood experience, but they do so only post hoc. With a little ingenuity, it is not difficult to construct an interpretation of parental behavior that is consistent with the patient's adult personality and psychopathology. These idiographic explanations are rarely falsifiable, so the clinician has little reason to abandon the belief that early environmental influences are crucial in personality development. [See multiple book review of Grunbaum's *The Foundations of Psychoanalysis*, *BBS* 9(2) 1986.]

Developmental psychologists, by contrast, study personality in the making, and directly observe the influence of parental behavior on the adjustment and achievement of children, often using longitudinal studies that follow the same subjects from the age of 12 to 24 months, or 1 to 3 years (Parke & Asher 1983). [See also Lamb et al.: "Security of Infantile Attachment as Assessed in the 'Strange Situation'" *BBS* 7(1) 1984.] But very few studies trace development from childhood into adulthood, and those that do find very limited evidence of enduring influences (Kagan & Moss 1962). P&D note that environmental effects on intelligence seen in childhood may not be sustained into adulthood.

Only in the last decade have a number of longitudinal studies appeared that cover extended periods of time into and within the adult years (McCrae & Costa 1984). These studies consistently show remarkable stability of personality over intervals of up to 30 years, despite biological aging, the acquisition and loss of social roles, and the occurrence of major life events. The stability of personality in adulthood does not, of course, speak directly to its origins, but these data do call into question the prevalent idea that environmental forces are all-important in shaping personality, and they are entirely consistent with the findings reviewed by P&D.

As an alternative to traditional environmental determinants, P&D have emphasized the contribution of substantive nonshared environmental influences, and have developed some ingenious approaches to their study. We hope that these new approaches will be examined in the context of long-term consequences: Like shared environmental influences, nonshared influences may also be of only temporary significance in the individual's development. Systematic retrospective studies of nonshared environmental influences on adult personality would seem to be a useful preliminary to longitudinal studies of children.

It might also be wise to attempt first to estimate more precisely the magnitude of measurement error. Because it is neither genetic nor shared, measurement error is classified as a nonshared environmental effect. Theoretically, however, error is quite different from substantive influences such as accidents or role models. Instead of partitioning variance into genetic, shared environmental, and nonshared environmental sources, it might make better sense to separate true score variance from error, and then to seek the determinants of the former.

P&D cite retest unreliability as an example of error of measurement, and this is the only type of error normally taken into account in behavioral genetics studies. However, many other forms of error also exist, and retest reliability systematically underestimates some of these. Of two equally extraverted twins, the one who tends to use more extreme response categories will score higher on measures of extraversion; because styles of responding may be very stable, this source of error will not appear as retest unreliability. Similarly, two subjects may consistently understand a word or question differently. Correcting only for retest unreliability may underestimate the true association.

Correlations between self-reports and ratings of the same individual rarely exceed .60 (e.g., Jackson 1976; McCrae 1982), even when the rater knows the ratee intimately or when ratings are aggregated. This might be seen as evidence that only 60% of personality test variance is attributable to the underlying true score: from this perspective, the typical correlations of .50 seen between identical twins appears to represent the great bulk of the variance, and substantive nonshared environmental influences become far less interesting.

One way to circumvent the problem of systematic error variance is through the simultaneous use of self-reports and ratings in behavioral-genetic studies (McCrae & Costa 1986). Consider, for example, a study of identical twins reared apart and their spouses. The correlation between first twins' self-reports and their spouses' ratings of them would probably be around .50; that correlation is limited by error in both self-reports and ratings. The correlation of the first twins' spouse ratings with the *second* twins' self-reports would be equally attenuated by measurement error, but would be smaller than .50 to the extent that the twins differed because of environmental effects on personality development. In fact, the ratio of the second-twin/first-spouse correlation to the first-twin/first-spouse correlation would estimate heritability of the true scores; the rest of true-score variance would presumably be due to substantive nonshared environmental influences. These estimates would give a better idea of how fruitful explorations of nonshared influences are likely to be.

The myth of the shared environment

Hans J. Eysenck

Department of Psychology, Institute of Psychiatry, University of London, London SE5 6AF, England

Plomin & Daniels (P&D) bring out very clearly both the methodology that led to the conclusion that environmental factors are very important consequences for theories of intelligence and personality which follow from this discovery. What they say is not always historically accurate, however. Thus, they say that: "The importance of nonshared environment was first highlighted by Loehlin and Nichols (1976)," but a similar conclusion was reported by Eaves and Eysenck (1975) when they wrote that:

> between 30% and 40% of the variation in components of extraversion may be due to environmental factors that cannot be attributed to the inconsistency of the test. All of the detectable environmental variation is specific to individuals rather than common to families. This suggests that attempts to relate extraversion to aspects of the individuals "family background" are unlikely to be productive unless the family background has a direct genetical association with extraversion. (p. 111)

This finding has indeed been a consistent feature of our work in this field with neuroticism and psychoticism no less than with extraversion (Eaves & Eysenck 1975; 1976a; 1976b; 1977; Eysenck 1983; Eysenck & Fulker 1983). This large series of studies, and the importance of nonshared environments, come out very clearly in Fulker's (1981) account. Clearly the effect is observable in cultures other than the North American.

P&D bring out very clearly the problems created by the fact that nonshared environmental factors are largely responsible for nongenetic determinants of personality, and they suggest appropriate strategies for the theorist. The great majority if not all of the major theories in the field interpret empirical facts in

terms of environmental rather than genetic factors, and usually as arising from shared rather than nonshared environments. For example, the correlation between parents' cruelly beating their children, and subsequent cruelty and aggressiveness appearing as the children grow up has been typically interpreted in direct causal terms as an effect of the parent's behaviour on the child's development. This type of interpretation, although almost universal, was never defensible; yet alternative hypotheses were sternly neglected, and would usually not even be mentioned. Thus the possibility of genetic determinants of the relationship was usually not even discussed as a viable hypothesis, nor was the very real likelihood that the child's behaviour as a youngster would both attract punitive behaviour on the part of the parents, and be predictive of the child's future behaviour as an adult.

The effects summarised by P&D certainly constitute a revolution in our view of personality, although P&D do their best to make this revolution as painless as possible. At first sight, we seem to be faced with chaos. All our traditional theories invoking differences between families as the causes of differences in the behaviour of children have been abolished at one stroke, and what takes the place of these plausible but incorrect theories would seem to be the accidental factors in individual lives which can neither be controlled nor measured across samples.

Yet on second thought it would seem that the data fit certain models rather well, such as those of neurosis and criminality (Eysenck 1977; Eysenck & Rachman 1964). These models invoke the importance of traumatic and subtraumatic conditioning experiences in the life of the individual, and these will inevitably tend to be parts of a nonshared rather than a shared environment. Clearly such events can be documented, if only or mainly in retrospect, and can be made the cornerstone of a within-family environmental theory. This is particularly true when we consider the experimental possibilities opened up by treatment intervention.

There is only one point on which I would disagree rather strongly with P&D. They argue, with respect to intelligence, "that most of the environmental variance is unshared." This is not the conclusion to which Fulker and Eysenck (1979) came after a thorough review of the evidence. Their estimate of genetic variation for intelligence was 69%, leaving only 31% attributable to the environment.

> What we found was that the 31% environmental variation could be subdivided into 17% common environmental variance, V (CE), and 13% specific environmental variance, V (SE). In fact, since the reliability of IQ test is at most 0.95, V (SE) can account for no more than 8% of reliable IQ variation, compared with 18% for V (CE). With these percentages in mind, both of them small, it is apparent not only that social influences in the environment are likely to outweigh other environmental influences by a factor of two, but also that many environmental factors might individually contribute no more than one or two per cent to total variation. In terms of mean effects this will be less than four IQ points. Four or five independent influences of this order of magnitude could, for example, completely account for V (SE).

The one or two new studies cited by Plomin & Daniels are interesting, but they do not seriously challenge the weight of evidence from previous work.

Evaluation of gene–environment interaction requires more precise description of both environment and behavior

Lawrence V. Harper

Department of Applied Behavioral Sciences, University of California at Davis, Davis, Calif. 95616

Plomin & Daniels (P&D) address an important issue for students of human behavioral development. They have highlighted a glaring deficiency in psychology: Our very profound ignorance of what constitutes *effective* environments (for the differential expression of hereditary potentials). To the extent that this target article stimulates advances in the conceptualization and measurement of environmental influences, P&D will have made a substantial contribution.

Unfortunately, P&D's use of the "standard" shorthand terminology of behavior genetics leads to inconsistent emphasis on the fact that they are talking about contributors to *differences* among individuals with the (unintended) possibility of sustaining nonspecialists' misconceptions of gene–environment relations (cf. Oyama 1985). For example, P&D state (sec. 1.2, para. 2) "*If* heredity affects a trait," and so forth (my emphasis). A literal reading of many passages would lead to the impression that "traits" could exist without any "genetic contribution." Although it may be "an empty truism that there can be no behavior without both genes and environment" (Plomin 1981a, p. 260); and wording so imprecise as to imply otherwise weakens the force of the argument.

The impact of P&D's message appears to be further weakened by several inconsistencies: They point out that the causes of differences within families need *not* be the same as the causes of differences between families and then they minimize birth-order effects on the basis of low *between*-family relationships. A plausible case could be made, however, for the prediction that within-family differences should be greater than between-family variation. If hunting/gathering was indeed the context in which *Homo sapiens* evolved, then we might expect that, when siblings are spaced apart in age by only 2–3 years, having a sibling could exert profound effects on behavior. According to Konner (1982), a woman simply could not sustain herself and more than one child under the age of about 4 years. If so, the appearance of a younger sibling could be a significant environmental "signal" causing the older child to display a range of behaviors designed to ensure that care would be forthcoming.

Similarly, although P&D acknowledge the existence of gene–environment interactions, they make several statements implying that these effects are unimportant. For example, to assert that shared environmental factors (including parenting) or similar (self-reported) subjective experiences are unlikely to be important sources of nonshared environmental influence implies that different genotypes will not respond differently either to the same settings or to similar subjective assessments of conditions. It is a bit paradoxical to point out on the one hand how inadequate our traditional views of environmental influence really are and then, on the other hand, to use the same traditionalist logic to dismiss the possibility that two people who assess a situation similarly could respond to it differently. Moreover, there is direct, longitudinal evidence for sex differences response to the same input (Martin 1981; Werner & Smith 1982) and different parental treatment in response to child gender (e.g., Jacobs & Moss 1976). Therefore, direct observation, including assessment of direction of effects, might be a first rather than a last step in identifying nonshared environmental influences, particularly since Plomin and Foch's (1980) findings suggest that objective assessment yields more conservative estimates of nonshared variance.

In other sections, P&D's reasoning seems suspect. They essentially rule out the possibility that differential parental treatment of children might serve to increase behavioral similarities. Parents, who have firm expectations concerning standards, however, may try to act in just such ways if their children differ substantially in temperament or activity level, for example. Indeed, this possibility is explicit in the concept of reactive gene/environment correlations (cf. Plomin, DeFries & Loehlin 1977).

In their discussion of parental treatment, P&D's conclusions simply go beyond their data. Although they cite one study that does document real consistency in parental "style," more than half the variance is still unaccounted for. Consistency in style is

not the same as identity of treatment. Furthermore, they also overlook the fact that Jacobs and Moss's (1976) major finding was in fact the existence of systematic birth-order *differences* in parenting that were related to sex of the *firstborn* child. Thus there is no reason to believe that first- and second-born are treated *the same*.

Similarly, they conclude that sex differences do not account for much nonshared variance, yet they provide no solid evidence in support of that contention. Standardized psychometric tests tend to be deliberately designed to minimize sex differences. In contrast, more direct observation of behavior reveals substantial gender-related variation (Harper & Sanders 1975; Martin 1981; Werner & Smith 1982).

Despite these flaws in their presentation, Plomin & Daniels have pointed to an important issue; what is needed now is more careful description and measurement of environmental and behavioral variation.

On nonheritable genetic differences

John Hartung

Department of Anesthesiology, SUNY Health Science Center at Brooklyn, Brooklyn, N.Y. 11203

Phenotypic variance among poker players is continuous and normally distributed – a few players win big, a few lose big, and most crowd around breaking even. This variance can be partitioned into two broad determinants – how good the individuals' cards are, and how well those cards are played. For many behavioral traits, especially those of personality and psychopathology, differences in genes are analogous to differences in cards – in the sense that differences in genes can account for differences between people without those differences breeding true. That is, just as one good or bad poker hand does not beget another, a combination of genes that contribute to being an extrovert, or to being schizophrenic, is likely to be broken up in the genetic shuffle of sexual reproduction. Accordingly, we have traits for which variance is substantially genetic (is due to differences in genes), but which have low heritability. For such traits we should expect low concordance between sibs (fraternal twins or not) and relatively high concordance between identical twins.

For example, consider just two genes (two loci) at which each of two parents are heterozygous (have two alleles). If two of the four possible combinations present in these particular parents would result in an offspring who will have a .67 probability of becoming schizophrenic, the probability that two siblings from these parents will be schizophrenic is $(.5 \times .67)^2$ – or about .10. At the same time, the probability that two identical twins who are dealt the cards in question will be schizophrenic is $.67^2$ – or about .45.

Though this phenomenon (a trait that is substantially genetic without having substantial heritability) is often gussied up by calling it nonadditive genetic variance, it is neither uncommon, nor complicated, nor mysterious. Indeed, Plomin & Daniels (P&D) understand it well. The mystery is: Why have they dealt with it so mysteriously? They admit straight-out that nonadditive genetic variance will be misread as nonshared environment by the analyses on which they base the bulk of their case, then they say that "most behavioural geneticists discount the importance of nonadditive genetic variance," adding the caveat that "some recent work suggests that it contributes to certain characteristics (Lykken 1982; Plomin 1986)" (sec. 1.1, para. 3). In reality, nonadditive genetic variance cannot be *discounted*, because it has not been *counted*. It simply fails to be a topic of much discussion in behavioral genetics, not because it affects only "certain characteristics," but because it is, at least to date, an intractable variable. That is, for predictive purposes, it is idiosyncratic and thereby of no use to behavioral geneticists (though it can open doors for neurobiologists – see below).

The problem here is that if you subtract misread nonadditive genetic variance from nonshared environmental variance, you will often have very little left – especially after you also subtract variance due to somatic nuclear genetic differences, mitochondrial genetic differences, other cytoplasmic genetic differences, genetically influenced nonshared or shared environmental differences (e.g., two sibs are allergic to ragweed and one goes to summer camp in a ragweed area [nonshared], or one sib is allergic and they both live in a ragweed area [shared]), and measurement error.

With regard to what *is* left, I hope that many commentators will balk at P&D's implausible arguments that nonshared environment is systematic. The driving force behind these arguments is their necessarily prejudical premise that "unsystematic, idiosyncratic, or serendipitous" nonshare environment is "one gloomy prospect" that is "likely to prove a dead end for research" while "systematic" nonshared environment is "more interesting heuristically." Unfortunately, wishing does not make it so – and giving noise a name does not make it music. On logical grounds, and as evidenced by most of the references cited by P&D in this connection, the nonshared component of environmental variance is likely to be as idiosyncratic as the nonheritable component of genetic variance. I do not have space to go into this, but it should suffice to indicate two points:

(1) The finding that "differences in siblings' experiences relate significantly to siblings' differences in behavior" *does not imply* "that nonshared environmental influences are at least in part systematic" (see conclusion #4, target article).

(2) For traits with low heritability and little idiosyncratic genetic determination of variance, if nonshared environmental influences were systematic, the correlation between individuals not reared in the same family should be *greater* than the correlation for individuals reared in the same family (related or not). That is, a factor that *systematically* causes differences between children reared together in one family should cause similar differences between children reared together in other families, such that traits of individuals similarly affected but from different families will correlate. This, however, is not what is found. Instead, we find either no difference in correlation, or a correlation within families that is greater than the correlation between families.

P&D have complicated some simple relevant concepts while delving into some genuinely complex concepts that are not relevant in support of their conclusions. Moreover, unlike behavioral-genetic partitioners of variance, those investigators who are "in the rush to find neural causes of schizophrenia" are quite likely to find them (e.g., Wasserman 1986). The identical twin concordance for schizophrenia makes the existence of such systematic neural substrates almost certain. In comparison to ferreting out the evernebulous, if not quixotic, elements of nonshared environment, this pursuit is infinitely promising.

In short, I do not share enthusiasm for nonshared environment and would rather see effort (and funding) directed toward neurobiological research.

A cumulative model of within-family differences

David A. Hay

Department of Genetics and Human Variation, LaTrobe University, Bundoora, Victoria 3083, Australia

Plomin & Daniels (P&D) have drawn the attention of a larger audience to an issue which has been of interest to many behaviour geneticists in the last few years. However, I am not convinced that with the methods they use it will ever be possible

to determine the extent to which such within-family effects are environmental or genetic. Based on our own experience with a very large study of twin children, the LaTrobe Twin Study (Hay & O'Brien 1981; 1984), I propose that both parents and children may seek an optimum level of differentiation and that this would confound any attempt at genetic analysis. It would also explain P&D's "surprise and interest" in finding little genetic influence on the Sibling Inventory of Differential Experience (SIDE) questionnaire.

I admit that my view is speculative, but I think it represents something worth considering when trying to identify the nature of within-family differences. My concern arises from two observations. First, when twin children were asked to compare their twin and themselves on 20 items (Hay & O'Brien 1981), they were unlikely to do so on any item concerned with being liked by family members. They would only admit differences on such neutral items as "who read more" or "who was more interested in drawing." This may coincide with P&D's report of siblings agreeing to a lesser extent on differences in parental affection. Second, Hay and O'Brien (1984) provide examples where parents base a differentiation of their newborn twins on such systematic factors as birth-order. Such a distinction is particularly common in identical twins, less common in same-sex fraternal twins and rare in opposite-sex fraternal twins. We argue that such systematic differentiation is therefore something parents are likely to do when there are few other differences on which to base distinctions.

Spillman (1984, unpublished thesis) has presented a similar model based on what are often very small birth-weight differences between twins, and her observation is consistent with the data on twins discordant for schizophrenia. Gottesman and Shields (1982) indicate how the lighter twin in such studies was more often the schizophrenic, even when the birth-weight difference was only .5 oz. This would seem to rule out birth trauma, but is consistent with an explanation based on parental distinctions between the twins. Hay and O'Brien (1984) do indicate that such differences can persist, increase, and are clearly evident in primary school-age twins. Hay and O'Brien (submitted) present data on twins who after the birth were released from hospital at different times; the teachers of such twins 8–10 years later still rated the one who came home second as having many more problems on the Bristol Social Adjustment Guide. All such results confirm Dibble and Cohen's (1980) observation of how early events may "lead to enduring patterns of interaction emphasizing children's difficulties or sensitivities" (p. 101).

Such differentiation between the children has two specific implications for the data P&D offer. P&D state their reluctance to consider model-fitting approaches to genetic analysis where models specifying a variety of parameters are fitted to data from many relationships. One major advantage of model-fitting over basic correlational data on twin or family or adoption data is that features specific to that particular relationship can be isolated (Hay 1985). The problems of twin data are indicated by the examples above where the differentiation may be based on features specific to twins and not relevant to the general population. One could argue the same about adoptive families with the unusual nature of any bonding and the potentially more nurturant caregiving. In focusing on specific relationships the approach of P&D may confound such effects.

The second point concerns the nature of the data, for both the questionnaire and the observational methods have their problems. Plomin (1981b) himself has raised the issue of questionnaire subjectivity with his distinction of "molar" and "molecular" questions; parents are much less subjective in their responses to the specific or "molecular" questions. This may imply that one can never really get a true idea from questionnaires of differential attitudes toward the twins on such vital but imprecise attributes as "affection." The use of relative rather than absolute questionnaires probably accentuates this problem by focusing the attention of parents or siblings on how they differ. Recently our own approach has gone much more towards the Bene Anthony Family Relations Test (Hensley 1984) and similar absolute measures of distinctions within the family. Observational measures may fare no better. With a video camera filming her behaviour towards the children, it may not be surprising that mothers treated their children similarly. Such behaviour may be reliable in this artificial context, but does it extrapolate to the home setting?

With such caveats, consider P&D's discussion of the two crucial observations that adoptive and biological siblings do not differ on the SIDE and that an equivalent degree of differentiation is seen in identical and fraternal twins. P&D go for the obvious explanation that such homogeneity across genetically distinct relationships implies no genetic determination. Our data on the way in which parents of identical twins seek out systematic factors on which they can distinguish their children argues more for parents mediating any genetic effect. If the children were unrelated and adopted, there are probably already enough differences, if they are siblings or fraternal twins, some differences can be accentuated: and if they are identical twins, differences may have to be created. I consider that parents and children may seek some degree of differentiation between children. If it is not there, they will create it, and a cumulative pattern of distinctions will emerge.

It is unclear why this is done and what its implications are. The only way to resolve this is by longitudinal studies, following children from soon after birth and seeing whether small initial differences become accentuated in a systematic way. The Colorado Adoption Project offers one approach and the La Trobe Twin Study data are currently being analysed from this viewpoint.

Unconfounding genetic and nonshared environmental effects

Arthur R. Jensen

School of Education, University of California, Berkeley, Calif. 94720

The most general and important lesson driven home by Plomin & Daniels (P&D) is one that is at last gaining recognition by a greatly increasing number, if not yet a majority, of behavioral scientists: Apart from a true environment-manipulating experiment, environmental influences on the development of a trait cannot be properly studied, indeed cannot really be studied at all, without a biometrical analysis of the genetic variance in the trait. It was only through this route that P&D have been able to demonstrate, fairly convincingly, I believe, the predominance of nonshared over shared environmental influences on a number of important human traits. This is a finding that has been turning up quite consistently in recent biometrical analyses, and it will undoubtedly be the subject of further investigations with respect to various behavioral traits.

It therefore seems worth mentioning a few of the questions and concerns I would anticipate in research on the relative effects of shared and nonshared environments. These two main sources of environmental variance are perhaps better known technically as *between*-family and *within*-family environments, although the terms *shared* and *nonshared* environment suggest an appropriately broader meaning. For consistency's sake, I will adopt the authors' terminology and refer to shared environmental variance (V_S) and nonshared environmental variance (V_{NS}). I also refer to the total genetic variance (V_G) and the total phenotypic variance (V_P), which includes measurement error.

A primary concern is selection of the kinds of data for which analysis can yield unconfounded estimates of V_S and V_{NS}. This limits us to monozygotic twins reared together (MZT) or apart (MZA) and genetically unrelated children reared together (UT).

Only the phenotypic correlations between MZ twins, who have a genetic correlation of 1, and between genetically unrelated individuals reared together, who have a genetic correlation of 0, are completely unambiguous with respect to the possible effects of genetic dominance, epistasis, assortative mating, genotype–environment interaction, and genotype–environment covariance. Any one or combination of these sources of variance may be confounded to some unknown degree with the effects attributable to nonshared environment in a biometrical analysis. Thus, using kinships that have some genetic correlation other than 1 or 0 runs the risk of spuriously inflating estimates of V_{NS} with some part of the genetic components listed above. P&D have not addressed this problem in sufficient detail. Until methods of biometrical analysis that can completely overcome this confounding problem are fully described, I believe that convincing evidence for the magnitude of V_{NS} must rest entirely on studies of MZ twins and unrelated adoptees reared together as siblings.

If we know the reliability (r_{xx}) of the trait measurement, and can demonstrate *uncorrelated environments* for MZ twins reared apart and *uncorrelated genotypes* for adopted "siblings," we can estimate the following components of variance without risk of inflating our estimates of the nonshared environment (i.e., V_{NS}) through confounding it with genetic effects.

$$\begin{aligned} r_{MZA}V_P &= V_G \\ (r_{xx} - r_{MZA})V_P &= V_S + V_{NS} \\ (r_{xx} - r_{MZT})V_P &= V_{NS} \\ (r_{MZT} - r_{MZA})V_P &= V_S \\ r_{CT}V_P &= V_S \\ (r_{xx} - r_{MZA} - r_{CT})V_P &= V_{NS} \end{aligned}$$

The different methods of obtaining either V_S or V_{NS} should, of course, result in the same values (within the limits of sampling error) if we are to have confidence in comparisons of the relative magnitudes of V_{NS} and V_S.

The proportion of V_{NS} appears to be larger in personality traits than in mental abilities. But one can wonder how much this difference may be confounded with method variance. There seems to be an important difference in the method of measurement, and probably also in the construct validity, of personality traits and cognitive traits. Personality is usually measured by self-report (or parent-report, teacher-report, etc.) whereas mental ability is measured directly and objectively by the person's performance on cognitive tasks. Based on my clinical experience of interviewing persons concerning their own estimates of their intelligence and comparing these estimates with their IQs on nationally normed tests, it has been my impression that the variance of self-estimates is considerably less than the variance of actual ability test scores. I suspect that the main shrinkage of variance in self-reports, relative to objective test scores, is largely in the shared or between-families (or between other reference groups) component of the variance. Such shrinkage of V_S would favor the appearance of relatively large V_{NS}. Would self-report assessments of abilities and of various physical traits show different proportions of V_G, V_S, and V_{NS} than objective measurements? If so, comparisons of personality traits and cognitive abilities along these lines would seem suspect due to the noncomparability of the methods of measurement in the two domains.

The increase in heritability ($h^2 = V_G/V_P$) from early childhood to maturity (at least for IQ) and the corresponding decrease in V_S, raises the question of whether this trend is due to a widening sphere of extrafamilial environmental influences as the child grows up or is due merely to the gradual averaging out of an even increasing number of more or less random or unsystematic favorable and unfavorable microenvironmental influences on the development of intelligence. Distinguishing between systematic macroenvironmental effects and random microenvironmental effects contributing to V_{NS} would be a methodological tour de force. But it is the next logical step in the research on the nature of the nonshared environment. One possible test of the hypothesis that random microenvironmental factors greatly outweigh systematic factors would be to look at the distribution of the squared differences between MZ twins reared together (MZT). These twin differences reflect only V_{NS} and variance due to measurement error (i.e., $1 - r_{xx}$). If the distribution of these twin differences were significantly different from the theoretical chi square distribution (for 1 degree of freedom), we could infer the existence of nonrandom effects in the nonshared environmental variance (V_{NS}). Failure to reject the null hypothesis, of course, would leave the hypothesis in doubt, since systematic effects contributing to the nonshared environment could also be randomly distributed in the population.

The statement of P&D that "nonshared environment explains perhaps as much as 40% to 60% of the total variance" in IQ will be welcomed by environmentalists, but it must certainly be a slip in wording (60% of the total *environmental* variance is probably what was meant), because the statement as it stands is clearly refuted by all of the evidence on MZT (with an average correlation of about .85) and MZA (with an average correlation of at least .70), and neither of these correlations (which estimate the proportions of $V_G + V_S$ and V_G, respectively) when subtracted from the proportion of total nonerror variance (i.e., r_{xx}) could leave a residual larger than about .25 attributable to any nongenetic effects.

Finally, it should be emphasized that what might be called the sociological view of the source of individual differences in psychological traits is largely refuted by the findings reported by P&D. This view puts all of its stock in the shared, or between-family, environmental differences, particularly the kinds of variables reflecting differences in socioeconomic status (SES) and cultural background, effects which might be altered by economic changes and better schooling. The finding that the predominant source of environmental variance is *within* families greatly diminishes the purported importance of SES and cultural background, which, by definition, contribute only to the shared or *between*-family environmental variance. Given the large nonshared environmental component in addition to the even larger genetic component in intelligence, the component of variance attributable to the shared or between-family environment affords little leverage indeed for materially reducing differences in intelligence by manipulating the environmental conditions commonly associated with the between-family component of variance.

Secular change in the relative influence of G, E1, and E2 on cognitive abilities

Ronald C. Johnson[a] and Craig T. Nagoshi[b]

[a]*Behavioral Biology Lab, University of Hawaii, Honolulu, Hawaii 96822 and* [b]*Institute for Behavioral Genetics, University of Colorado, Boulder, Colo. 80309*

Plomin & Daniels (P&D) are to be commended for pointing out the importance of nonshared environmental influences (E1) on the development of cognitive abilities, personality, and psychopathology and for developing a measure, the Sibling Inventory of Differential Experience (SIDE), to get at some of these nonshared influences. That much of the variance in these important areas of behavior is not accounted for by genetic factors (G) or shared family environment (E2) puts the findings of behavioral genetics (with its emphasis on heritabilities) in perspective. The need to understand E1 also provides an opportunity to integrate behavioral-genetic techniques with mainstream developmental psychology.

As P&D describe, nonshared environmental influences are

the variance "left-over" after genetic and shared environmental influences on a behavior are accounted for in behavioral-genetic paradigms. P&D are, of course, well aware that some of this left-over variance is due to the unreliability of the measures used and the related problem of transient environmental influences and fluctuations in the physiological states of subjects affecting task performance on a particular day. Of the more enduring nonshared environmental influences, the important ones may turn out to be mostly idiosyncratic (e.g., the anecdote given by the authors on how the direction of Charles Darwin's life was affected by the shape of his nose). An obvious task for developmental psychologists and behavioral geneticists is to determine the amount of variance of a given behavior that can be accounted for by *systematic* nonshared environmental influences, such as the differences in sibling perceptions of their childhood environments as measured by the SIDE.

P&D rightly point out that the relative influence of G, E1, and E2 on a behavior can change during the lifespan of a sample of individuals, and they provide a plausibly explained example of how the variance accounted for by shared environment on intelligence has been found to decrease dramatically after childhood. We would like to point out that, at least for the large body of accumulated data on cognitive abilities and their correlates (e.g., academic performance, educational, and occupational attainment), there is also compelling evidence for a *historical* change in the relative influence of G, E1, and E2. This secular change is not only interesting for the understanding of the factors influencing the development of intelligence, but it also has important social implications.

Socioeconomic status (SES) has been the most commonly used measure of shared environment, as it is easily measured and typically moderately to highly correlated with aspects of the family environment such as number of books in the home, quality of parent–child interactions, parental achievement motivation, and so forth. White's (1982) meta-analyses of studies done since the 1920s indicated that not only does parental SES have only a moderate (mean $r = 0.22$) influence on their children's academic achievement, but that this influence appears to have declined in recent years. This decline is perhaps due to the influence of television and other entertainment media, greater physical and social mobility, and compensatory education programs, in reducing social class differences. White did find that specific measures of the home environment were more highly correlated with academic achievement, but all of these studies failed to take into account that parental SES and the home environment are also highly correlated with parental intelligence and its genetic bases. Several studies (Gottfried & Gottfried 1984; Johnson & Nagoshi 1985; Longstreth, David, Carter, Flint, Owen, Rickert & Taylor 1981; Ramey, Yeates & Short 1984) have demonstrated that once parental intelligence is partialled out, SES and measures of the home environment have either no influence or are statistically significant but often trivial influences on offspring cognitive abilities.

Other evidence than that cited by P&D was thus available to support their contention that shared environment has a minimal influence on cognitive abilities. This evidence also suggests a historical decline, at least in the U.S., in the influence of shared environment. On the other hand, reviews of the literature by Henderson (1982) and Plomin and DeFries (1980) indicate that there may have also been a historical decline over the past fifty years in the amount of variance in intelligence accounted for by genetic factors, with recent studies reporting substantially lower heritabilities than studies done in the 1920s and 1930s.

There are several plausible hypotheses to account for this decline in heritabilities, for example, the use of different measures and samples, but this decline may also reflect the reduction of social class differences in the U.S. during this century. Such a historical reduction in social class differences certainly accounts for the significant ethnic group-by-generation interactions for cognitive abilities in the Hawaii Family Study of Cognition (HFSC) obtained by DeFries, Corley, Johnson, Vandenberg, and Wilson (1982) and by Nagoshi and Johnson (1985), where it was found that children of Japanese ancestry living in Hawaii had substantially increased in their cognitive test scores over those of their parents, whereas no such increase was found for Caucasian children. Johnson, Nagoshi, Ahern, Wilson, DeFries, McClearn, and Vandenberg (1983) describe the historical changes in Hawaii that resulted in a considerable increase in social, economic, and political status for Japanese in Hawaii during the years that the parents in the HFSC were growing up. As might be expected when such major historical changes affect nearly everyone in a generation (although constant mean changes across generations should theoretically leave the familial correlations unaffected), HFSC parent-offspring resemblances for cognitive abilities were generally higher for Caucasian than for Japanese families, but the reverse was the case for sibling resemblances (DeFries, Johnson, Kuse, McClearn, Polovina, Vandenberg & Wilson 1979).

Such a reduction of social class differences would also account for the historical decline in degree of assortative mating for intelligence and education reported by Ahern, Johnson, and Cole (1983), Johnson, Ahern, and Cole (1980), and Johnson, Nagoshi, and Ahern (in press). This decline in degree of assortative mating would by itself reduce parent-offspring and sibling resemblances in cognitive abilities. Whereas it may be expected that a reduction of social class influence would have the effect of allowing greater expression of genetic factors, it is just as reasonable to believe that the opposite would occur, with individuals now being more affected by idiosyncratic events and/or systematic nonshared environmental influences. All of the above suggests that not only are Plomin & Daniels right in asserting the need to study nonshared environmental influences, but it would appear that these nonshared influences have increased in importance in recent years.

Quantitative genetics and developmental psychology: Shall the twain ever meet?

Joseph K. Kovach

Research Department, The Menninger Foundation, Topeka, Kans. 66601

Behavioral geneticists have been discussing for some time the lack of vertical environmental transmission of behavioral traits. We have long known, for example, that the morbidity risk of schizophrenia is elevated among first degree relatives of schizophrenics, regardless of whether they are raised by biological or adoptive parents; and that the rearing of a biologically unrelated child by an adoptive parent who is schizophrenic does not increase the child's initial minimal chances that he too will become schizophrenic (Heston 1966; Higgins 1976). These observations support Plomin & Daniels's (P&D's) thesis that the environmental component of the variance of schizophrenia is entirely of the nonshared type. Neither these observations, however, nor the type of quantitative genetic data discussed by P&D can throw much light on the developmental causes of the nonshared environmental variance.

The nonshared environmental variance of a behavioral trait identified by the procedures of quantitative genetics is a mixed bag of overt variations originating in systematic and stochastic environmental effects, systematic and stochastic influences in the developmental organization of the nervous system, nonadditive gene effects, and measurement error. P&D are quite right in searching for systematic environmental factors in this hodgepodge of potential sources, but they are wrong in neglect-

ing the other sources. I particularly disagree with their view that "capricious events, however, are likely to prove a dead end for research." Gene expression and encounters with decisive environmental events are both probabilistic, and stochastic processes may account for a large portion of nonshared environmental variance (see Fuller & Thompson 1967; Kovach 1986). The following example of accidental variation in the development of human-handedness (the environmental variance of which also appears to be entirely of the nonshared type, see Annett 1981; Carter-Saltzman 1980) illustrates the point. [See also Corballis & Morgan: "On the Biological Basis of Human Laterality" *BBS* 1(2) 1978.]

The mean frequency of right-handedness in the general population is about 85%, which is generally believed to originate in a strong genetic bias for left-hemisphere dominance, which is in turn believed to control dextrality. Collins (1977), examining the concordance of right- (R) and left- (L) handedness in twins, found the proportions of RR, RL, and LL pairs of both monozygotic (MZ) and dizygotic (DZ) twins to conform to the expectations of a binomial of about P = .85. He argued that the similarity of MZ and DZ twins and the binomial fit (and thus randomness) of the data counterindicate genetic determination.

In terms of quantitative genetics, for which the primary problem is the genetic origin of dextral bias, the variation examined by Collins would be automatically labeled as "nonshared environmental variation." But in developmental terms, the data simply mean random variation about a very high mean for dextrality. Whether this variation is associated with one or more or all of (1) probabilistic events associated with gene expression, (2) stochastic processes intrinsic to development, and (3) accidental environmental influences in the development of handedness, is anybody's guess. However, some data suggest the involvement of random events intrinsic to neural development.

Gur and colleagues (1984) observed an irreversible shift from left- to right-handedness following callosotomy in a left-handed and left-hemisphere dominant person. Efforts to retrain the preoperative left-handed skills of this person were ineffective. Apparently, callosotomy interrupted some interhemispheric connections that prior to surgery overrode the control of handedness by the dominant hemisphere. Such data suggest that the stochastic processes discussed by Collins may be intrinsic to the developmental organization of the nervous system. One cannot dismiss the possiblity that some aberrant neural connections in schizophrenia (see Taylor 1975; Schneider 1979) and the partial noncondordance of MZ twins (see Kendler 1983) may also be due to such processes.

Another issue relevant in the search for the developmental causes of trait variance is the human propensity for selecting and creating particular experiences from whatever the available environment. Farber (1981) cites some reasonably convincing data that MZ twins reared apart have more similar personalities than MZ twins reared together. These data suggest that the shared or nonshared components of environmental variance need not originate in actually shared or nonshared environments. Relegating this problem to the subjective realm (as done by P&D in their Sibling Inventory of Differential Experience study) only complicates matters that call for direct developmental examination.

The procedures and data of quantitative genetics simply cannot deal with the nuances of developmental self-regulation and they do not permit P&D's conclusion that "whatever parents do that is experienced similarly by their children does not affect the children's behavioral development."

The variance components of a trait partitioned by the procedures of quantitative genetics are narrowly specific to the trait, to the population of subjects that exhibit the trait, and to the environment in which the trait is expressed. They are also specific to the genetic context of their identification, which ideally requires independent estimates of additive genetic effects, nonadditive genetic effects, error in development, error in episodic expression, and measurement error. Relating a variance component to developmental considerations also requires examining the nature of environmental effects identifiable by family resemblance studies, twin and adoption studies, intraclass correlations, and the like. By contrast, the developmental causes of a trait are always interactive. They cannot be separated into categories of independent genetic and environmental determination. The following example from schizophrenia research illustrates this point.

In a large-scale prospective adoption study, Tienari, Sorri, Lahti, Naarala, Wahlberg, Ronkko, Pohjola and Moring (1985) found that the morbidity risk of schizophrenia among separately adopted biological children of schizophrenic parents is seven times as high as among separately adopted children of nonschizophrenic parents. This factor is very close to the one (6 : 1) as the one identified by Kety (1983) and colleagues. Because the factor is about the same when the children are reared by their biological parents, Tienari's results agree well with P&D's thesis that the environmental component of the variance of schizophrenia is nonshared. Tienari and colleagues, however, also examined the contribution of family environment to the occurrence of schizophrenia, using measures other than familial resemblance and intraclass correlation. They found manifest psychosis only among those adoptees who were raised in families diagnosed as "disturbed." They interpreted their data in terms of a diathesis model: Family health may prevent and family disturbance may evoke the occurrence of schizophrenia in *genetically predisposed* individuals.

Unfortunately, Tienari's interpretation is not unequivocal. The data could be explained equally well by families becoming "disturbed" when their adopted children are at actual genetic risk (see Beichtman 1985, for childhood schizophrenia) and remaining "healthy" when the children are only at nominal risk. The study also confounds development and overt expression. (Did family environment influence the development or merely the overt expression of schizophrenia?) But even if considered only as a hypothetical instance of family environment effecting trait expression, this study illustrates well the conceptual discontinuity between quantitative genetics and developmental psychology. In quantitative-genetic terms, the Tienari study confirms the nonshared environmental component and vertical nontransmission of schizophrenia. Yet, simultaneously, the study rejects the notion that shared experiences do not influence the individual expression of schizophrenia.

The concepts of quantitative behavioral genetics and developmental psychobiology seem to me as distinct as are the concepts of functional and causal explanation in evolutionary biology, or the particulate and wave explanations of quantum mechanics. Partitioning trait variances into genetic and environmental components cannot tell us much about the factors responsible for the development of a trait in a particular individual. The name of the game of jointly considering trait variation in a population and trait development in individuals is complementarity, and it has been badly neglected by P&D.

The major task ahead, it seems to me, is to identify the complementary objectives and procedures of behavioral genetics and developmental psychology. In trying to come to grips with this task, I regard constitutional vulnerabilities and predispositions as the traits of choice for quantitative genetic analysis. By definition, such traits should include vertical environmental nontransmission among their identifiers, along with sensitive biological and behavioral markers. Developmental examinations could then focus on those systematic and stochastic processes of neurobiological and environmental determination, and genotype–environment (or vulnerability/ experience) interaction that are responsible for variable phenotypic expression of the related behaviors.

Niche picking by siblings and scientists

Michael E. Lamb

Departments of Psychology, Psychiatry, and Pediatrics, University of Utah, Salt Lake City, Utah 84112

One of the ironies of our overspecialized age is that the assumptions of one discipline or subdiscipline are the focus of exciting discoveries of another. Thus Plomin & Daniels (P&D) write that "thinking about environmental influences that create differences between children in the same family represents a dramatic reconceptualization of psychological environments," whereas I suspect that the conclusion will not be surprising for many other psychologists, especially those with a developmental bent. On the other hand, the reconceptualization may be profound for behavior geneticists, whose perspective is nicely encapsulated in the words of Loehlin and Nichols (1976, p. 92) cited approvingly by P&D: "environmental effects . . . operate almost randomly with respect to the range of variables that psychologists (and other people) have traditionally deemed important in personality development."

Many behavior geneticists were raised in an era in which psychologists were hostile to the mere suggestion of biogenetic influences, and doubtless their recent appreciation of the importance of within-family environmental variation stems from their long-term preoccupation with demonstrating that aspects of human personality and intellect were indeed significantly heritable. Having made their case persuasively, they have apparently raised their regard from the image in the microscope before them, only to discover a world beyond it. That new discovery is the awareness of nonshared environmental influences.

As P&D acknowledge, precise estimation of the extent of such influences does not guarantee awareness of the factors involved. With respect to parental influences, which may well turn out to be among the most overrated formative factors in child development, researchers are hampered by powerful social desirability tendencies that shape the parents' behavior, perhaps obscuring many of the individual differences of greatest interest to researchers. Then there is the related problem of child gender – perhaps one of the more important elements in the nonshared environment puzzle, yet one whose exploration is hampered by the existence of tendencies to respond in a fashion thought appropriate in the context (observation/interview by a behavioral scientist). The gender of the child also has major implications for factors other than parental influence: Not only may there be inherent gender differences in patterns of response to environmentally determined experiences, but there surely are also differences between the treatment of boys and girls. Furthermore, as Kohlberg (1969) (among others) has argued, the awareness of one's own gender sets in motion self-socializing tendencies that lead children to attend differentially to gender-appropriate models. Indeed, the whole issue of gender is one that receives surprisingly little attention in P&D's target article, perhaps because the confound of environmental and biogenetic influences is of unknown magnitude.

There are a number of interacting factors – including parental behavior, sibling interaction, and peer group experiences, all themselves confounded with gender – that may help explain why there are such small similarities between siblings on the various aspects of personality that have been studied by behavior geneticists.

This does not justify the stated conclusion that "the effective environments of siblings are hardly any more similar than are the environments of strangers who grow up in different families." Surely, the degree to which this is true depends on just how "strange" the strangers are, and this too speaks to the need for interdisciplinary perspectives. Even if it were true that the upper-middle-class and rural boys (described by Scarr & Grajek 1982) are as similar to one another as to brothers, would we also expect upper-middle-class city-raised American brothers to be as similar to one another as to brothers (or sisters) raised in the Ituri forest? Indeed, the statements by P&D and by Scarr and Grajek appear to contradict P&D's earlier (and probably correct) assertion that there is no necessary relationship between the causes of differences between families and the causes of differences *within* families (Sec. 3.2, para. 2).

As social scientists studying Americans who are scientifically sophisticated and evaluation conscious, it behooves us to be aware of the biases built into the data we gather by observation or interview – biases that may conspire against our ability to identify the individual differences in behavior that are evident when evaluation apprehension is not excited. We also need to appreciate the limited reliability and validity coefficients of many of our measurement instruments, and to be appropriately circumspect in interpreting the failure of measures based on brief selected slices of life to quantify individual variations among individuals or families sensitively. Stated differently, our failure to identify the factors responsible for within-family variations may be at least due in part to the poverty of our attempts to measure them, not to the fact that parental, sibling, or peer behavior is not influential.

Twin studies, environment differences, age changes

John C. Loehlin

Department of Psychology, University of Texas at Austin, Austin, Tex. 78712

First, in case anyone has any lingering suspicions that Plomin & Daniels (P&D) have been looking selectively at the evidence on shared environments and personality, let me note that the data from two recently reported twin studies continue to support their position that the effect of the former on the latter is virtually undetectable. One study is of a sample of Indiana University twin pairs tested twice at ages 20 and 25 (Pogue-Geile & Rose 1985). The other is of a large sample of adult twin pairs from Australia (Martin & Jardine 1986). A nice feature of both studies was the inclusion of an attitude measure – a religiosity scale in the first and a conservatism scale in the second – which, unlike the personality scales, *did* show a substantial effect of shared family environment.

Second, I would like to call attention to another way of exploring within-family variation, namely, by relating personality differences between the two members of monozygotic (MZ) twin pairs to experiential differences between them. Any associations found must be environmental, since the twins do not differ genetically. Robert Nichols and I did a certain amount of this sort of exploring with the data of the National Merit twin study (Loehlin & Nichols 1976). For example, we selected from the 514 MZ twin pairs in the total sample the 50-odd pairs in which – according to a parent questionnaire – one twin had had a serious illness in infancy but the other twin had not. We divided this subsample in two (for cross-validation purposes) and examined a selection of some 18 personality variables to see whether there were any differences in adolescent personality characteristics between the twin who had had the illness and the twin who had not. There were none that held up across the two half-samples. The traits examined included six California Psychological Inventory scales (Dominance, Sociability, Self-Acceptance, Responsibility, Socialization, and Achievement via Independence), six self-ratings of interpersonal problems (with boys and girls own age, mother, father, teachers, and other adults), and six self-ratings on trait scales (calm, energetic, giving in easily, carefree, politically conservative, and outgoing).

The same analysis was carried out for MZ pairs in which, according to parental report, one twin (compared to the other)

had more minor illnesses as a child, got more attention from the mother, was spanked more often as a child, or was rocked and held more often as a child. In each such case, most of the pairs in the sample did not differ; some did, however, and we looked at those. In no cases was there a consistent relationship to the adolescent personality measures.

In another related analysis we asked – across the whole sample – whether MZ twin pairs who had been subjected to more similar experiences were more alike in personality. These experiences included such variables as whether they had been dressed alike, how much they played together, whether they slept in the same or different rooms, whether they usually had the same or different teachers in school, and so on. None of these variables was substantially related to personality differences between the twins.

MZ twins are, of course, something of a special case, and I would not want to discourage further research with other groups along the lines that P&D suggest. However, I did want to point out that at least one previous foray into this territory has been made, one that ought to have come up with some relationships had they abounded, but it failed to find anything.

Finally, a word or two on P&D's conjecture that shared environmentally effects on intellectual abilities may diminish with age. There is support for this hypothesis for the very early years – up to age 6 – from the Louisville twin study (Wilson 1978). But in the Texas adoption study we found only slight evidence along these lines for ages above 5 years. For example, using a path model we estimated genetic and shared-environmental parameters on performance IQ measures for children aged 5 to 7 versus children aged 8 or older (Horn, Loehlin & Willerman 1982). There was a small difference in the shared-environment parameter – c^2 was about .15 for the younger children and .11 for the older; in the right direction for P&D, but almost certainly not statistically significant.

This was derived from cross-sectional data. A sharper test is to look at the same children longitudinally over time. My colleagues and I have just completed a 10-year follow up of our original sample of adoptive families. For approximately 250 adopted children measured in both studies, the correlations with adoptive mothers and fathers were .15 and .17 at the time of the first study (average age about 8), and .08 and .09 at the time of the second (average age about 18).

Thus we have some, although not overwhelming, evidence in support of Plomin & Daniels's notion that the shared effects of familial environment on intellectual traits might decline with age.

An alternative explanation for low or zero sib correlations

David T. Lykken

Department of Psychiatry, University of Minnesota, Minneapolis, Minn. 55455

Most of the environmental factors that have a lasting effect on cognitive skills and personality are influences and experiences that we do not share with our siblings. Many of the environmental influences that developmental psychologists study and theorize about – parents' education and socioeconomic status, their personalities and child-rearing practices, the neighborhood in which they bring up their children – turn out, on analysis, to be factors that are shared by siblings and hence not generally important. The domain of significant experiences, those which are unique to each child in a family, remains largely unexplored. This is the main message of Plomin & Daniels's (P&D's) target article and I believe that it is both true and important.

These generalizations, based though they are on remarkably well-replicated findings, should not be taken to mean that parenting behavior is inevitably impotent or inconsistent in its effects. One need only think of examples, like the family of Joseph and Rose Kennedy, to realize that at least some parents can and do manage to impose a considerable shaping influence on each of their offspring, an influence limited in its extent and consistency only or largely by the genetic differences between the offspring. The evidence cited by P&D indicates that such charismatic or otherwise-effective parents are too few in numbers to have much effect on group statistics. In addition to calling for study of nonshared environmental influences in development, therefore, I think these same data suggest the importance of studying families that are exceptions to the rule, families that manage to provide a shared environment that does influence each of the siblings in similar ways.

I have one additional quibble. In the first paragraph of P&D's Abstract it is asserted that "these environmental influences make two children in the same family as different from one another as are pairs of children selected randomly." This eye-catching statement is simply wrong, I believe. The average intraclass correlation for sibs on cognitive or personality traits is greater than zero; sibs are not in general as different as random pairs of unrelated children. Moreover, there is an alternative explanation for near-zero sib correlations when they do occur, namely, that the genetic mechanism that helps determine the trait in question is nonadditive, epistatic, or "emergenic" (Lykken 1982.)

Impulsiveness, for example (the Control scale of Tellegen's Multidimensional Personality Questionnaire), correlates .41 among 236 pairs of monozygotic (MZ) twins and only −.01 among 116 pairs of dizygotic (DZ) twins (Tellegen, Lykken, Bouchard, Wilcox, Segal & Rich, in press). Among 35 pairs of MZ twins reared apart (MZA twins), the correlation is .51 whereas in a small sample of 15 pairs of DZA twins, it is −.29 (not significantly different from zero.) The most reasonable explanation for such findings is that impulsiveness is substantially determined by a configuration of polygenes. The essence of configural determination is that even a small change – that is, one different gene in the pattern, like one different digit in a telephone number – can make a large or even qualitative change in the result. MZ twins, sharing all their genes, will share all gene configurations, but sibs and other first-degree relatives are unlikely to share all the polymorphic genes, so their similarlity on the "emergenic" trait is likely to be much lower than their genetic correlation. It is not true that all instances of low or zero sib (or DZ) similarity must be attributed to unshared environmental factors.

Some models where independent ≠ different

J. J. McArdle and I. I. Gottesman

Department of Psychology, University of Virginia, Charlottesville, Va. 22903

The target article by Plomin & Daniels (P&D) makes the claims that (1) a large portion of variation in behavioral measures is due to environmental sources, and (2) the largest source of this environmental variation is not shared by members within the same family. Following Rowe and Plomin (1981), P&D support these claims with empirical research of their own and by others. They also point out the importance of quantitative model-building, but they suggest that "model-fitting procedures, however, only find significant parameters when they are implicit in the basic data" and "for this reason, and because of the relative inaccessibility of most models, we have emphasized the basic correlational data and merely note that model-fitting approaches confirm our conclusions" (Sec. 3.5, para. 1).

From a model-building perspective, however, the correlation-based conclusions described by P&D are ambiguous at best. P&D do in fact rely on models that are accessible, but

these models are (1) not specified in explicit detail, (2) have parameters that are not restricted in any testable way, and (3) promote hypotheses that are not formally tested against alternatives. This simplified approach does not reflect contemporary scientific research in behavioral genetics (cf. Cloninger, Rice & Reich 1979; DeFries & Fulker 1986; Eaves, Last, Young & Martin 1978; Loehlin 1979; McGue, Gottesman & Rao 1983). We now briefly address model-building issues which raise potential challenges to the validity of P&D conclusions.

1. Independence due to unreliability. In the biometric designs discussed by P&D, the statistical assumptions of errors of measurement (E_m) are identical to the statistical assumptions for independent environments (E_1) so the terms are strictly confounded. This means that the model parameters for both $V\{E_m\}$ and $V\{E_1\}$ are not jointly identifiable within an explicit model. Thus, unless the modeling strategy includes some accounting for unreliability of the measure, variance due to errors of measurement ($V\{E_m\}$) will lead to an *inflated* estimate of the variance of the independent environment ($V\{E_m\}$).

P&D point to this serious problem of measurement error and make some attempts to introduce corrections for attenuation. They also attempt to identify manifest indicators for the relevant source variables. Unfortunately, these ad hoc model-building strategies minimize the potential importance of this confound. The classical treatment of Cattell (1953) originally suggested several ways to correct for unreliability (and see Loehlin 1979). The more recent availability of computer programs for multiple-measure structural equation-estimation (i.e., LISREL-V, COSAN-II) allows for the psychometric estimation of common and specific sources within the context of any biometric model (see McArdle 1986). In broad terms, errors of measurement $V\{E_m\}$ need not be confounded with specific environmental variance $V\{E_1\}$.

Without more details about models of unreliability, children in the same family may appear independent simply because we have underestimated errors of measurement.

2. Independence due to missing alternatives. P&D are explicit about their use of simple "correlational" models, but they do not emphasize that simplistic models can bias their conclusions about the relative importance of independent environments. The main biometric model implicitly used herein proposes orthogonal effects due to additive genetic source (D_r) and specific environments (E_1). In some models the effects of common environments (E_2) was also added, but exactly how such model pieces were put together is unclear and this can be important.

The basic estimation equations (and program output) show that the orthogonal latent scores D_r and E_2 yield parameter estimates $V\{D_r\}$ and $V\{E_2\}$ which are *not orthogonal* (e.g., within the twin design $V\{D_r\}$ and $V\{E_2\}$ are negatively correlated; see Eaves et al. 1979). As in any multiple regression analyses, the appropriate interpretation of any variance component is limited by other effects in a model. Substantive examples of this problem are numerous (e.g., McArdle 1986), and orthogonalized effects remain ambiguous. But still, models that reject alternatives should allow these alternatives to stand on equally solid ground.

Without details on the direct comparison of alternative models the major source of environmental variance may appear to be independent, for example, simply because we have underestimated the contribution of the common environments.

3. Independence instead of differences. P&D are clear about their statistical definition of environmental "difference" – The model estimation of $V\{E_1\}$ references a source of variance where individuals within a pair are stochastically independent, or linearly unpredictable, from one another. Unfortunately, the use of the colloquial phrase "so different" in the title, and the continual use of "difference" interpretations, can easily mislead the reader.

One common misinterpretation of this "difference" model is that individuals have "opposite" scores on these latent variables. Indeed, if there are limited resources within a family and if one family member gets some of these resources, it follows that the other members cannot get these resources. But in this model of opposing forces, the covariance within pairs can become negative and orthogonal latent variable models can attribute these differences to the common environmental component ($V\{E_2\}$). Still more confounding comes when the pairs are slightly opposite (yielding some negative correlation) and misspecified models orthogonalize variation in unexpected ways.

P&D further describe some measurement techniques for creating manifestations of different environments. For example, the Sibling Inventory of Differential Experience (SIDE) allows an easy rating of another family member on a similar versus different scale. This follows the basic logic of individual differences multidimensional scaling (see De Soete & Carroll 1983) and makes it easy to conceptualize a model where INDSCAL techniques are mixed within biometric models, but, instead, P&D advocate this measurement technique as a way to provide a direct estimate of the unobserved biometric construct. The latter approach to understanding differences would be informative if it were at all feasible.

Without more details on these models and methods for differences, children in the same family may seem less different than they really are.

Model-building summary. In the simplest models the equality of the specific variance $V\{E_1\}$ and common variance $V\{E_2\}$ can be proposed as an explicit model. If P&D's interpretations are correct this equality model will be rejected in favor of a model where $V\{E_1\} > V\{E_2\}$. The standardization of variance estimates into proportions of variance is a natural modeling byproduct but confidence boundaries (which are not proportional) are required for all model interpretations. The basic hypothesis of $V\{E_1\} = 0$ is also easy to define but difficult to test statistically without multivariate data. Similar technical modeling details also apply to various statements about aging changes over the life span, the validity of childhood personality assessments, the effects of assortative mating and epistasis, and the use of more intricate relative groupings.

In broad terms these model-building issues may or may not pose serious threats to the validity of the conclusions reached by P&D. The authors have implicitly ruled out a wide variety of potential threats by using some simple biometric models; they do point out some important confounds apparent with "nonadditive genetic sources" as well as genetic/environmental "covariation and interaction"; and they have provided a few hypotheses that can now be examined as explicit models. Also, the direct measurement of biometric latent sources is no doubt an important and difficult task. Still, it is disappointing that P&D ignore many technical modeling features because these formalisms can help clarify their own ideas. From this perspective, then, the results of P&D are provocative but inconclusive.

In summary, we agree that the isolation of environmental variation is "one of the most important findings that has emerged from human behavioral genetics." But we also think that more explicit model-building is required to demonstrate and use this "remarkable conclusion."

The problem of documenting systematic nonshared environmental effects directly

Kathleen McCartney

Department of Psychology, Harvard University, Cambridge, Mass. 02138

I suspect that few will take great exception to Plomin & Daniels's (P&D's) main point concerning the importance of the nonshared environment for development. Some will quibble with the hyperbole that children in the same family are as

different from one another as children in different families. For IQ, at least, the average difference between siblings is about 12 points, whereas the average difference between randomly paired people is 18 points (Jensen 1980). For personality, siblings appear at least somewhat more similar than random people, albeit not much more so. Others will want to point out rightly that the shared environment is only unimportant when one considers good, middle-class environments that are functionally equivalent in some way (Scarr & Weinberg 1978b). These environments, and not deprived environments, are the kinds that developmentalists tend to study. Thus, the implications of P&D's argument for intervention may not be as radical as is implied. Certainly, however, their main point is important and well-argued: Behavior-genetic studies suggest that the nonshared environment accounts for the majority of environmental variance.

And why should it be otherwise? Why have developmentalists expected that, the shared environment is of critical importance, as evidenced by the vast number of studies on the effects of the home environment? There are at least two reasons. The first is pragmatic. Developmentalists know how to study the shared environment. We have been able to specify what it consists of and we have been able to construct both observational and questionnaire measurement tools. Equally important, it is easier to think about childhood intervention, which is integral to a theory of environment, within the context of the shared environment: by definition the shared environment is more systematic than the nonshared environment. Second, developmentalists have probably been biased by viewing the family as a cultural unit. Families share similar values and attitudes, which make them appear similar among themselves, to psychologists and nonpsychologists alike. Families develop and pass down rules about everything from vacation strategies to helping behavior. This view is consistent with P&D's interpretation of Loehlin's (1982) data concerning the significant shared environmental influence on masculinity-femininity. P&D argue, correctly I think, that sex role is more in the category of attitude than personality.

Although I strongly suspect that P&D are right about the importance of the nonshared environment for the development of both intelligence and personality, there is a problem with the argument as it now stands. Given our current data base in developmental psychology, it is not possible to document the importance of any one form of systematic nonshared environmental effect *directly*. P&D base their argument on estimates derived indirectly from behavior-genetic models.

Consider the conclusions that P&D draw for us on the specific forms of systematic nonshared environmental effects that form their taxonomy (see P&D's Table 1). First, there is not enough research on the importance of either sibling interaction or extrafamilial networks (e.g., peer groups) to make any meaningful statement. Second, most developmentalists agree that family constellation variables, such as birth-order, have small predictive power. Third, reports from parents and siblings and observational studies show similar rather than dissimilar parental treatment. Here I suspect the data do not warrant any conclusion about differential parental treatment for two reasons: (1) There are too few studies and (2) it does not make sense that parents would not respond to individual differences in their offspring. Perhaps family members are not able to perceive or report differential treatment of potential import. Is the just noticeable difference that family members are able to detect about differential treatment too insensitive? For example, do egocentric young siblings of different ages know that their mother is tailoring her language to each child's level of understanding (Shatz & Gelman 1973)? Do mothers even realize this? Do school-aged siblings know that their father is more likely to become impatient with the more active child (Buss 1981)?

In these examples lies the implication that differential parental experience is correlated with the genetic predispositions of siblings. P&D suggest otherwise when they argue that the origins of perceived differential experience are environmental and not genetic. Here again, and for the same reason, I suspect the data do not warrant any conclusion. Daniels's (1985) research shows that correlations between adopted siblings and biological siblings on a differential experience questionnaire do not differ. Although it is impressive that these judgments are related to self-reported differences on personality inventories, the ability of siblings to detect important differences must be questioned, especially in a culture where uniform treatment of offspring is valued. The possibility of a halo effect between Daniels's two measures also exists.

If the source of nonshared environmental experience is not genetic, as P&D suggest, then how can this experience be systematic? There would be little reason to assume that there would be continuity in the nonshared environment without input from the genotype or phenotype. Uncorrelated environments imposed on children (e.g., school teachers) change. The lack of long-lasting effects of intervention programs suggests the importance of the continuing environment. What kinds of experiences could be part of the continuing environment that are not strongly related to predispositions? Only family constellation variables, including sibling de-identification, appear to be plausible candidates, but research here is not encouraging. We need to know to what extent children and adults make their own environments through active niche-building and evocative processes (Scarr & McCartney 1983). This is probably the place to start in the search for direct evidence for nonshared environmental effects.

This direct evidence may be long in coming, depending upon the partition between systematic and unsystematic effects. Plomin & Daniels are understandably conservative about attributing a great deal of importance to the unsystematic nonshared environment, which consists of "stochastic events," because of the depressing implications for research. This state of affairs would leave environmentalists with only the case study as a method. And yet, Darwin's story of how his *Beagle* voyage was nearly denied because of the shape of his nose is compelling. I suspect that all of us could share similar stories of seemingly random, but critical, events in our lives.

Shared environment and cultural inheritance

Newton E. Morton

Department of Epidemiology and Biostatistics, Memorial Sloan-Kettering Cancer Center, New York, N.Y. 10021

The large variance component due to nonshared environment merits emphasis. Evidence is strong for normal variation, including not only personality traits and cognition, but also physiological and anthropometric traits. For psychopathology the evidence is not so straightforward, partly because concordance is not a simple function of covariance (e.g., Rao, Morton, Gottesman & Lew 1981). On the concordance scale, identical twins are expected to be more than twice as similar as fraternal twins when genes are additive on an underlying liability scale that gives affection by truncation. The weakest part of the evidence concerns dissipation of covariance due to shared environment after adolescence, which depends entirely on the cited study by Scarr and Weinberg (1978) that did not include preadolescent pairs. The average age was only 13 in the unpublished dissertation of Kent (1985). Adoption studies may involve restriction of variance, placement after infancy, and modified parent–child interaction. Is covariance due to shared environment lost after adolescence for adopted children, and if so, is this a general phenomenon? If it were, we would expect that parent–offspring and sibling correlations would decrease with age for both biological and adoptive pairs and that at

maturity they would be less than in childhood by the amount of the covariance due to shared environment. I am not aware of convincing evidence that this is the case. Path analysts are currently preoccupied with temporal trends, but critical data are scarce.

It should perhaps be pointed out that variance components are model-dependent. For example, on simple assumptions the sibling correlation is $G + E_2$ where G and E_2 are the correlations due to genes and shared environment respectively. One convention replaces G by $h^2/2$, where h^2 is genetic heritability. Another convention replaces E_2 by $C^2(2f^2 + b^2)$, where c^2 is cultural heritability, f is the transmissibility of environmental factors from parent to child, and b is the transmissibility of nonrandom sibship environment. Often $2f^2 + b^2 < 1$, and then cultural heritability exceeds shared environment, just as genetic heritability exceeds the correlation due to genes. The residual variance due to unshared environment is $1 - h^2 - c^2(2f^2 + b^2)$ whereas the residual variance not due to genetic or cultural heritability is $1 - h^2 - c^2$. Most recent effort has gone to discriminating genetic from cultural heritability, which appear equally important for IQ (Rao, Morton, Lalouel & Lew 1982).

Individual differences or different individuals? That is the question

Helmuth Nyborg

International Research Center for Psychoneuroendocrinology, University of Aarhus, Risskov, Denmark

I am unhappy about the approach used by Plomin & Daniels (P&D) to explain why single children in the same family are so different from one another. They use the traditional approach of behavioral genetics (BG). That approach fails to account adequately for particular differences in psychological make-up (Nyborg 1983; 1986). A better approach would be to use a person-specific analysis. Let me briefly illustrate how P&D happen to sit between two chairs and suggest a person-specific, double-chair solution to their problem.

P&D get the wrong answer to the right question for the following reasons. BG originated in quantitative and population genetics and abounds with valuable information from family, twin, and adoption studies about how much of the total phenotypic variance of a given trait can be ascribed to genetic and to environmental variance, respectively. BG methods are, however, completely unable to cope with individualized data. BG methods provide a heritability estimate, but that measure is neither informative – nor even a first step – with respect to pinning down causal agents in a particular child's ontogenesis. The dilemma of whether to calculate population averages or to study particular persons, having individual development in mind, is most certainly not new to psychology. From time to time it appears in the literature as the nomethetic-idiographic controversy or as the problem of clinical case studies versus experimental psychology. No one has yet succeeded in finding a solution to the painful problem of dealing simultaneously with two methodologically incompatible psychologies, and P&D try to have it both ways with their nomothetic approach.

Another problem with applying the classical BG approach in the present context is that people differ in susceptibility to various environmental influences, but the BG approach chosen by P&D averages out such differences. Some people go through life rather unaffected by environmental pressures. Other people have sensitive periods, and still others are abnormally sensitive all the time. P&D clearly miss this point of individuality by generalizing that nonshared environment is the most important source of environmental variance for personality, psychopathology, and IQ.

A third problem with P&D's BG approach is that it cannot cope with covariant development or "positive manifold" expression of clusters of cognitive and personality traits in individuals. Usually, cognition and personality are considered separately in BG analyses. But even if considered together, their relationship is dealt with statistically rather than in individualized, biological terms, or the connection is left unexplained by a reference to unknown genes interacting in unspecified ways. The lack of person-specificity in P&D's approach may be acceptable to some as a first step as long as they are interested in anonymous, average trends in a population. But even so, it can go wrong. P&D note, for example, that estimates of "gender differences account for only a small (1%–5%) portion of the variance of sibling differences in development." The conclusion that gender is unimportant for psychological differentiation is clearly challenged by simple everyday observations of profound gender-related differences in prepubertal play pattern and temperament, of an astonishingly large and persistent bias in the educational and occupational choice repertoire, of the tremendous gender-related differences in the social power structure, and so forth. Knowing the gender is by far the best basis for predicting membership in engineering or helping professions. It is, by the way, saddening to see that psychological tests do not clearly reflect the easily observable gender bias in society.

A final problem with the approach used by P&D is that of the "fallacy of negative proof" (Fischer 1970). Their logic is as follows. Shared environmental factors fail to account for similarities between siblings (low correlations for fraternal twins, etc.). Therefore, differences between siblings are caused by nonshared environmental factors. Nobody has illustrated this fallacy better than Lewis Carroll (1869): "I see nobody on the road," said Alice. "I only wish I had such eyes," the King remarked in a fretful tone. "Able to see Nobody! And at that distance too!"

In order to obtain the right answer to P&D's important question, we have to perform a person-specific analysis. Such an analysis starts with the single individual, not with an average, and is the only way to safely establish a genuinely general trend (Nybord 1974). We also need a more flexible and dynamic kind of analysis than BG allows in order to keep up with the complexity of the question. I suggest that we start by acknowledging our tremendous biochemical individuality, and then try to probe deep down under the skin of single individuals rather than satisfying ourselves with an outside look. To find the singular we must study the dynamics of how *their* genes and *their* environment influence *their* person – specific internal milieu and condition their unique differentiation. If we find enough people with similar dynamic characteristics we might have stumbled upon something general.

Let me give an example of how such an individualized analysis can run. Experiments show that exposure of an organism to gonadal steroids has profound effects on its body, its brain (McEwen 1983; Toran-Allerand 1984), and on the phenotypic expression of cognitive and personality traits (Nyborg & Nielsen 1981; Nyborg 1983; 1986). What happens is that the steroids selectively modulate sets of genes by influencing their protein production and thus "masculinize" or "feminize" the brain. Fetal secretion of, and differential receptor sensitivity to, gonadal hormones are partly under genetic control by the fetus itself, partly influenced by placental and maternal secretions, the latter again being under environmental influence in an intricate dynamic interplay that changes radically over time. Add to this that the outcome of the steroidal impact also depends on "sensitive" periods in neural development that are probably controlled by fetal genes other than those controlling the steroid output in addition to being partly under environmental control. The early, predominantly organizational effects of steroid hormones on the fetus later combine at puberty with activational

effects of surges in plasma steroid values. All these effects contribute to individual variations and to person-specific covariation of bodily, cognitive, and personality traits (Nyborg 1984). The mechanisms *are* intricate, but it should be possible to study them at different levels of complexity.

The sex hormonal history of a given person has been found to be a better predictor variable for later psychological differentiation than is the karyotype, the genital type, or any particular environmental agent (Nyborg 1983; 1984; 1986). If this finding can be confirmed in future studies, it seems likely that the power of person-specific analyses of biochemical individuality to explain why people become different surpasses that of the statistically anonymizing, dichotomous gene-environment analyses.

Perhaps the time has come for a change of paradigm in the developmental study of cognition, personality, and psychopathology. Averaging individuals will no longer do. The last ten years of research in psychoneuroendocrinology suggest that different individuals and not individual differences are what matter most when we ask questions about how a particular child developed differently from another child. Moreover, similarities across different individuals, not average differences between them, are the stuff that general trends are made of.

Shared experience and similarity of personality: Positive data from Finnish and American twins

Richard J. Rose[a] and Jaakko Kaprio[b]

[a]Department of Psychology, Indiana University, Bloomington, Ind. 47401 and [b]Department of Public Health, University of Helsinki, Helsinki, Finland SF00140

Twenty years ago, the relevant environment in genetic/environmental models of schizophrenia was assumed to lie in the patterns of parenting then believed to be unique to "schizophrenogenic" households. But Heston (1966) then demonstrated that a child at elevated risk for schizophrenia (by virtue of being born to a schizophrenic mother) was at no less risk when separated from that mother at birth and reared by nonrelatives. These and other data led clinicians away from their preoccupation with parenting practices and other sources of shared family experience to more idiosyncratic events in individual lives. Asking why siblings who grow up together turn out differently, Plomin & Daniels (P&D) argue that what is true for schizophrenic outcome is true for all behaviors – that family environment shared by siblings is of negligible importance and only idiosyncratic experience influences personality and cognitive development.

We disagree. Data not cited by P&D challenge their position on psychopathology and cognition. We present in this commentary new twin data that are inconsistent with P&D's denial of shared environmental effects on personality.

For major psychopathologies, the familial environment widely hypothesized to be etiologically relevant is still exposure to a deviant parental model. That hypothesis can be tested by contrasting at-risk adoptees, separated from an affected biological parent, who are reared in adoptive homes with and without deviance in the adoptive parent(s) – a human parallel of cross-fostering designs of animal genetics. For schizophrenia, there is no increased risk to children who are reared by an adoptive parent with schizophrenia. But for both criminality (Hutchings & Mednick 1974) and alcohol abuse (Cadoret, O'Gorman, Troughton & Heywood 1985; Cloninger, Bohman & Sigvardsson 1981), there are sons at genetic risk (criminality or alcoholism in biological father) who are significantly more likely to be deviant if reared by a deviant adoptive parent. For at least some psychopathologies in which social modeling by parents is developmentally salient, experiences shared within a household play a significant role.

For the development of cognitive abilities, we note that the only data suggesting an absence of common environmental influence are two studies (Kent 1984; Scarr & Weinberg 1978a) cited by P&D (Sec. 2.3) of small samples of adoptive siblings. The most likely sources of relevant between-family environmental variation (making siblings alike) are differences in social and economic class and in ethnic and subcultural values and practices. A limited sampling of agency-screened and self-selected adoptive parents is very unlikely to reflect adequately the range of between-family variation in the population, and one must be conservative in inferring null effects from such samples. The claim (Sec. 3.5, para. 1) that modeling family data reveals no shared environmental effect on IQ is not correct (e.g., Vogler & Rao 1986); and suggesting that "shared environmental influence on IQ . . . is of negligible importance by the end of adolescence" (Sec. 2.3, para. 5) implies that familial covariance for IQ declines thereafter, as adult siblings leave home. It does not. Nor do appraisals of cognitive abilities in elderly twins reveal significant changes in the composition of environmental variance from adolescence to senescence.

What, then, of personality? Familial aggregation for normal personality traits depends on (1) the traits measured and (2) the strategy of test development by which the trait are assessed, and (3) the magnitude of genetic and environmental variation between the families under study. Familial resemblance will be significantly greater for some traits than others on personality scales derived by rational rather than empirical methods, and in representative data sets rather than samples in which trait-relevant demographic variation is restricted by selection biases.

Standard scales of the MMPI (Minnesota Multiphasic Personality Inventory), empirically derived and heterogenous in their item-content, *do* exhibit the pattern of twin correlations that P&D describe. For example: Median correlations for a sample ($N > 400$ pairs) of adolescent/young adult twins tested in Indiana (Rose submitted) yield no evidence of shared experience (median r = .48 for the MZs [monozygotic twins] and .22 for DZs [dizygotic twins]). But scales composed of items that are homogenous in meaning show quite a different pattern: for the nine components identified (Costa, Zonderman, McCrae & Williams 1985) in a factor analysis of all MMPI items, median MZ/DZ correlations of 0.56 and 0.41 estimate effects of shared experience = .26. P&D correctly state that attitude scales assessing stereotypic interest patterns (e.g., femininity or religiosity) provide evidence for shared environmental influences: Correlations for the Costa et al. factor of religious orthodoxy indicate that 61% of its variance is due to shared experience! But P&D incorrectly imply that these are the *only* scales for which shared experience has significant influence. For the Costa et al. extraversion factor, MZ/DZ correlations are 0.60 and 0.42, suggesting that 25% of the variance in extraversion is attributable to experiential effects shared by age- and gender-matched siblings.

Twin data permit more direct tests of shared experience. P&D suggest testing for *nonshared* environmental effects "free of genetic bias" by relating experiential differences *within* pairs of MZ twins to the behavioral differences observed within those parts. Agreed: Monozygotic twins discordant for smoking, for use of oral contraceptives, or for exposure to industrial solvents (examples of twin control methods) provide instructive test cases. By the same logic, an incisive test of *shared* environmental effects, again free of genetic bias, relates experiential differences *between* pairs of identical twins to the behavioral differences observed between those pairs. We illustrate with direct evidence that identical twins who differ in shared experience differ significantly in their behavioral similarity.

Table 1 (Rose and Kaprio). *Intraclass correlations for EPI neuroticism (N) scores in MZ twins categorized by the frequency of their social contact*

		Frequency of social contact by cotwins				
		Living together	Daily	Weekly	Monthly	Rarely
1,293	Female MZs	.709	.439	.399	.359	.256
1,027	Male MZs	.501	.410	.280	.278	.171

The evidence, from analyses of personality data within the Finnish Twin Cohort, (Koskenvuo, Langinvainio, Sarna, Kaprio & Rose 1986) is from 4,640 monozygotic twins, age 24–49, representing nearly all living identical twin pairs of that age in Finland. These completed a medical/psychosocial questionnaire containing a short form of the Eysenck Personality Inventory (EPI) used in the Swedish twin study (Floderus-Myrhed, Pedersen & Rasmuson 1980) cited by P&D as evidence for absence of shared experiential influences on adult personality. The twins also reported the frequency of their social contact with one another.

MZ pairs were in more frequent contact than were DZ pairs (twice as many MZs reported daily social interaction with their twin) but the frequency of social interaction varied widely within both MZ and DZ twins. The frequency of twins' social interaction is proportional to the magnitude of their intrapair differences in EPI scores. DZ pairs who rarely contact one another may of course share fewer genes; it is only on average that DZ twins share half their genes identically by descent, and those pairs who are genetically less similar may, for that reason, seek one another's company less often *and* may be less similar in personality. But no genetic differences exist within MZ pairs. Evidence that identical twins in less frequent social interaction are less alike therefore suggests that shared social contact, and common experience derived from it, directly influences intrapair resemblance in personality.

The effect of social contact on MZ twin correlations for the EPI neuroticism (N) scale is illustrated in Table 1. The effect is large, linear, and present in both brothers and sisters. The effect is not an artifact of age: Younger pairs tend to be in more frequent contact, but age does not correlate with intrapair differences in N scores across the 2300+ MZ pairs. To confirm that effects of social contact on intrapair resemblance are not mediated by age (or by gender) we performed stepwise multiple regression on a double-entry matrix of the MZ twin data, predicting each twin's N score from that of the twin, that pair's age, gender, and social contact, and all interactions. The order in which predictive terms entered the equation was controlled, so that the effects of age and gender were removed before any effect of social contact was evaluated. A significant increment in predictive variance remains attributable to the interaction of the twin's score with pairwise frequency of social contact. We have found similar effects on drinking habits in more than 5,600 Finnish twin brothers; those in more frequent contact are much more alike in their consumption of alcohol (Kaprio, Koskenvuo, Langinvainio, Romanov, Sarna & Rose, in press).

The conclusion seems inescapable: Experiences shared by adult identical twins significantly enhance their behavioral similarities.

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Toward a relevant application of nonshared environment

Pierre Roubertoux and Marika Nosten

Génétique et comportement, Unité d'Enseignement et de Recherche biomédicale de Paris V, Paris 75006, France

"Nonshared environment," like the solution to Aesop's enigma, can be both the best and the worst of notions. It is the best when it takes into account a set of sources of variation that are often mistakenly pooled as error variance. Plomin & Daniels's (P&D's) attempt to identify nonshared environment and outline the influences of the mechanisms involved in fact lead the reader to more radical conclusions:

1. Some sources of nonshared environment are systematic. In inbred mice, pup care behavior varies as a function of the mother's strain (shared environment) (Carlier, Roubertoux & Cohen-Salmon 1982), but also as a function of the pup's strain, as cross-fostering studies have shown (Carlier, Roubertoux & Cohen-Salmon 1983; Cohen-Salmon, Carlier, Roubertoux, Jouhaneau, Semal & Paillette 1985).

2. Some sources of nonshared environment are stable but their effects are different depending on the behavioral sequence. This is clearly indicated in studies on sensory motor development in preweaned mice. Maternal effects interact with pup genotype, but these can either accelerate or retard the rate of development of certain traits (Carlier et al. 1983; Carlier & Roubertoux 1985).

3. Certain sources of nonshared environment may initially be distributed randomly across individuals but produce systematic long-term effects. For example, both the position of the fetus in the uterus and the nipple a rat pup is suckled on are initially a matter of chance. It has been shown, however, that behavioral characteristics of adult female mice are correlated with uterine androgenization, which is dependent upon the female's proximity to male fetuses (Vom Saal & Bronson 1980). Similarly, the first random contact of a pup with a nipple leads to what can be termed an "assignment" of this pup to the nipple (in Rosenblatt & Siegel 1981). It has been demonstrated in rodents that all nipples are not equivalent for quantity or for quality of milk and this has an impact on pup development, in particular on weight (Rageneau, in press).

It is clear that a detailed analysis of the processes involved in nonshared environment in human species would be difficult. Two directions appear, however, to hold promise. The first is related to the nonshared uterine environment. The fetus perceives sensations and can hear (Granier-Deferre, Lecanuet, Cohen & Busnell 1985). Does auditory stimulation, in particular from the mother, have differential effects on the fetus, thus producing nonshared environments in siblings? The second is related to an ethological approach to parent–offspring relationships. Here again a behavioral analysis of the mechanisms involved is more informative than a global approach consisting of partitioning the variance.

The nonshared environment can be the worst of notions when

it is used for data adjustment to a genetic model. I will restrict myself to examples from psychiatry. If we look at the data over the last 30 years on schizophrenia, affective disorder, and autism, the paradox is striking: In every study, the concordance rate is higher for monozygotes (MZs) than for dizygotes (DZs), which is a strong argument in favor of the presence of genetic factors in the etiology of these psychoses. However the concordance rate in MZs is less than the 100% necessary to support an exclusively genetic hypothesis. One explanation is that these genes have different effects in different environments and that MZs of the same pair have only a partially shared environment (Zazzo 1965). A second set of data adds to this paradox. When models are put forward to account for familial resemblance, certain cases deviate from expected values. The incidence of bipolar illness in daughters whose fathers are afflicted is below the risk predicted for monogenic dominent X-linked transmission (Mendlewicz 1978). Here again the hypothesis of variable gene expression and its corollary, nonshared environment, should be taken into consideration. This can lead to a form of circular reasoning unless these notions are used to explain variable gene expression in *identified* genes.

An alternative is an approach founded upon etiological heterogeneity which is more operational and has ample empirical support. The fact that there are MZs discordant for one psychiatric trait, and that in general MZs exhibit greater similarity than DZs demonstrates that this trait can be genetically transmitted in some families and nongenetically in others. Folstein and Rutter (1977) have shown that a perinatal trauma in one MZs of a pair can result in autistic disorders. Thus, the departure from 100% in MZs of whom one is afflicted with an affective disorder might result from phenocopies. If so, this would be a case of nonshared environment. In those cases where the disorder is related to genetic factors, a given trait may be correlated with different genes. Take affective disorders (see Roubertoux 1981 for an overview), for example. It has been shown that unipolar and bipolar illnesses have different modes of transmission. The risk of bipolar illness in relatives of probants afflicted with unipolar illness does not differ from the risk observed in the general population, and there is no link with X-chromosome markers. In contrast, for certain families having bipolar illness, a correlation has been found for X-linked markers and markers on the long arm of the X chromosome (G6PD, abnormal color vision). This linkage, however, is not present in all families having this disorder. On the other hand, a linkage disequilibrium with the PC1A Duarte autosomic locus has been reported for bipolar illness (Commings 1979). As concerns infantile autism, the high incidence of this disorder in boys has led (among other possibilities) to the hypothesis that certain cases of autism are linked to a fragil X. Similarly, the hypothesis of independent autosomal correlates with the disorder have been shown in other families (Spence, Ritvo, Marazita, Funderbunk, Sparkes & Freeman 1985). [See also Gualtieri & Hicks: "An Immunoreactive Theory of Selective Male Affliction" *BBS* 8(3) 1985.]

The fact that a given trait has different genetic and environmental correlates in different families is consistent with research on infrahuman species. One of the best examples is the study by Michard and Carlier (1985) on intermale aggression, which shows that genetic or environmental mechanisms vary as a function of the strains observed.

An analysis of effects associated with a nonshared environment should help avoid the pitfalls of the partitioning of the variance and should pave the way towards an analysis of the processes involved, thus avoiding the trap Hirsch and McGuire (1982) have already cautioned against: "A very common error in experiments is to treat all animals as if they are identical by averaging over their differences and assuming that the mean response of the population is characteristic of all individuals" (p. 12).

The puzzle of nonshared environmental influences

David C. Rowe

Department of Psychology, University of Oklahoma, Norman, Okla, 73019

If solving nature's puzzles is partly the attraction of science, then behavioral scientists have the excitement of a new one. Twin, adoption, and other studies send a clear message: Environmental influences that siblings share lack impact; rather, the developmentally effective environmental influences are ones that differentiate siblings. In an earlier article with Robert Plomin, we made this point (Rowe & Plomin 1981). Plomin & Daniels (P&D) have elaborated on it further with the addition of new evidence and insights in the target article. Thousands of studies have been conducted on divorce, parenting styles, father-absence, social class, and other factors siblings largely share with one another. The absence of shared influences violates both this research tradition and intuition, yet the conclusion would seem unassailable. What, then, are the environmental influences?

P&D offer a list of new environmental factors that have disparate effects on siblings; yet some of the influences in this list sound like the failed shared-type of influence. For example, several nonshared factors, including differential parental treatments, different teachers, and different peer groups, also exist as influences across families. Consider parental affection; if this fails to make one child more psychologically adjusted than another in a different family (using a research design where heredity has been controlled), then why should it make one sibling happier than a brother or sister? Furthermore, the differences across families are probably greater as viewed by an observer than those within the family. How can we reconcile the greater effects of environments within families with the larger range of environmental differences between them?

One answer to these dilemmas is in various kinds of environment/genotype interactions. If genetically different siblings react in a dissimilar way to the same environments, then we can understand why broad differences among families are so small, whereas sibling differences are large. P&D summarize studies showing that siblings are treated similarly by their parents; yet siblings' perceptions of parental treatments seemed more disparate than the treatments themselves. This observation meshes with the notion of different genotypes reacting in different ways to environments that seem alike to an observer – hence, the possible genetic regulation of siblings' perceptions of parental love and affection (Rowe 1983).

Siblings not only react differently to the same environment; they also actively choose different environments. One area of nonshared environment where data do suggest genetic differences is in the selection of friends (Daniels & Plomin 1985; Rowe & Osgood 1984). Genetically more similar pairs (identical twins versus fraternals; biological siblings versus adoptive) describe themselves as having friends with more similar characteristics.

P&D, however, downplay the importance of these kinds of interactions. Their data suggests that in other areas, sibling differences are unassociated with the genetic differences of the sibling pairs. It seems intuitively plausible, though, that the different personalities of siblings are bound to interact with environmental contingencies. Finding effects of the same kind across different personalities is unlikely. Work on extraversion–introversion, for instance, documents a wide range of differences in reactivity to environmental contingencies in people differing on this heritable trait dimension (Wilson 1978).

Does this mean that environmental effects are idiosyncratic and unpredictable, as the difficulty of finding uniform environmental influences on schizophrenia might suggest? Probably not. It means that to predict environmental effects, individuals must first be grouped according to their genetic similarity.

Because this is an impossible task at present, the more modest method of looking for interactions between personality scores and environmental measures would seem to be the most fruitful approach: Although the record to date of identifying such interactions is not satisfactory, I still believe that this is one place to search. Genetic influences stand out more than environmental ones precisely because they show up against a range of environmental backgrounds. I do not believe that the opposite will hold.

Another explanation for the prominence of nonshared influences focuses on psychological factors instead of genotype/environment interactions. The shared influences – existing across all families in a population – express the full range of environmental differences. The nonshared influences – the differences which siblings experience or perceive – are only a part of this range. Yet, to a child, these nonshared influences may seem larger. What provokes sibling rivalry is how much less love one sibling has received than another. The amount of love received by the children in a family down the street hardly matters. Schachter's (1982) deidentification theory and some attribution-theoretic notions in social psychology posit personality differences arising from such relative comparisons. These ideas deserve more elaboration. Do relative comparisons affect behavior more than the development of enduring traits? If they do affect enduring traits, are they relatively independent of genotype? After all, not every child perceives a 10% inequality in parental affection as cause for disliking a sibling. Do younger siblings rely more on relative comparisons, and older ones, with a greater store of knowledge and experience, more on absolute ones?

In closing, I emphasize my basic agreement with Plomin & Daniels. They urge research on nonshared environmental influences using designs that sample more than one child per family. This is essential, and it is the one practical message of behavioral genetics that must be heard by developmental psychologists, sociologists, and other scientists. They try to identify factors that explain sibling differences; I agree with their list of factors (and would add others, such as rapid cultural change, for instance, from the marijuana culture to the cocaine culture); but we emphasize somewhat different mechanisms of action. I expect we will find few nonshared environmental effects that are "main effects" (meaning consistent for all people). If such effects are found, they may revolve around siblings' perceptions of relative differences in their families, a subtle kind of environmental effect that may nevertheless be powerful.

Distinctive environments depend on genotypes

Sandra Scarr

Department of Psychology, University of Virginia, Charlottesville, Va. 22901

Plomin & Daniels (P&D) describe the most significant body of research on the sources of individual differences in behavior. Their summary of results collides with contemporary psychological theory. In brief, psychology has no adequate theories to account for individual variation in behavior because our theories address the wrong sources of variation (Scarr 1986a; 1986b). Among the families studied in adoption and twin research, individual differences in personality, interests, and cognitive and intellectual abilities arise from genetic differences and from environmental (or nongenetic) differences *within* families, not from environmental differences *between* families. Psychological theories chiefly address differences between families, and all of the correlated phenomena of neighborhood, community, schooling, and so forth. These theories do not address how children in the same family come to be so different from each other.

Lest we conclude that environmental opportunities do not matter, let us remember that samples in all of the cited studies include only families that are working class or above. There are no lower-class, unemployed, abusive, or neglectful families in the studies. Thus, inferences about sources of variation between families must be qualified by considerations about what kinds of families were in the samples studied.

As I have stressed in recent papers, contemporary psychological theories no longer account for observations from family studies that consider both genetic transmission and environmental effects (Scarr 1985). New observations have rendered socialization and social class theories weak and even invalid within the range of families studied. These findings will in the long run spell the death of those theories because they attempt to account for developmental processes for the majority of children. It is clear that current socialization theories cannot account for even normative patterns of development, let alone individual differences. There is no way to emphasize enough the inescapable conclusion from many family studies, but the psychological community seems utterly unprepared to deal with it.

P&D are right in their review of the literature, and they are right in their assessment of previous research. I think, however, that they are too pessimistic about establishing how environments contribute to consistent sources of individual differences in experience. They claim that differences in siblings' perceived treatment, correlated with differences in their personalities, can be a test of the effect of genetic differences on sibling differences in environment. Not even P&D believe that the test has the power (the correlation of differences with differences) to test the possible effect of genetic differences between siblings on the differences they experience in interaction with teachers, parents, boy scout leaders, peers, and so forth. The test they propose is exceedingly weak in statistical power (see Cohen & Cohen 1983; Judd & Kenny 1981).

Rather, the more powerful research they surprisingly do not cite is that of Lytton (1980), who studied identical and fraternal twins and their parents in detailed interactional sequences. This unique research design speaks most directly to the question of how differences in siblings' environments arise by describing the behavioral initiations and responses of children and their parents in everyday situations. Lytton details the sequences of interaction between parents and children. He found that the initiations of behavioral sequences were due more to the children than to their parents. Thus, the greater similarity in treatment of identical than fraternal twins arises from the evocation of more similar treatment by identical twins than by fraternal twins.

Translated into ordinary family interactional terms, this means that parents are very likely to interact with their children according to the children's personal characteristics. (If you are a parent of more than one child, you will understand this statement.) Richard Q. Bell (1968; 1979) cited the neglected direction of effect: child to parent. In the last 10 years, evidence has accumulated for reciprocal and child-directed socialization effects (Shaw & Bell, submitted). Hence the environments children receive are at least in part the result of their own personal characteristics, such as interests, talents, and personality (Scarr & Weinberg 1978a).

Developmentally, children are treated largely in age-appropriate fashion (Dunn, Plomin & Nettles 1985). It is not surprising that 12-month-olds are treated more similarly to each other than they are to 24-month-olds, regardless of family ties. Can you imagine speaking to a 1-year-old as you would to a 2-year-old, even if you are the most insensitive parent in the world? Can you imagine telling a 15-year-old that he is not allowed to cross the street? Yet one would surely enforce this prohibition strongly, even forcibly, with a 24-month-old. Age differences in parents' responses to the same child and to several children are understandable differential strategies in sensible child rearing.

But age-appropriate treatment will not endure the preschool

years, and certainly not the school years, without consideration for individual differences. Again, any parent of two or more children knows this. Children who present greater challenges to parental authority (control issues) are treated differently from those who present lesser challenges. Children who are warm and affectionate are treated differently from those who are aloof or provocative (love issues). Parental treatment of older children and adolescents depends on the characteristics of the child (Scarr & McCartney 1983).

P&D argue that the evidence for genetic differences affecting environmental differences is shaky. But their argument is flawed. The inconclusiveness of P&D's argument rests on correlations of differences in siblings' perceptions of within-family differences with measured differences among the siblings' personality, interests, and so forth. Many hundreds of sibling pairs would be required to test such a hypothesis with appropriate statistical power (Cronbach & Furby 1970; Judd & Kenney 1981). Null results, based on samples of less than 600 pairs, are uninformative.

P&D suppose, as I do (Scarr & McCartney 1983), that the influences of the family and its correlated opportunities decrease over a child's development such that older children, adolescents, and young adults are less influenced by differences among families than are infants and preschool children.

Shared variances, as defined by P&D, include all of those environments that come in a package of family influences – school, neighborhood, community, nation, and historical period. According to their review, including our own results (Scarr & Weinberg 1978b), late adolescents are apparently not much influenced by differences in opportunities their families afford them. But recall that children in the United States have access to public schooling, public libraries, and community groups that sponsor everything from baseball to drama and geology. Most children can find a niche in contemporary society. Differences among children's outcomes therefore depend more on their own characteristics than on differences in their opportunities to match themselves to their environments.

Why does human twin research not produce results consistent with those from nonhuman animals?

J. P. Scott

Center for Research on Social Behavior, Bowling Green State University, Bowling Green, Ohio 43403

Reading Plomin & Daniels's (P&D's) target article stimulated in me a veritable flood of ideas, of which I shall list those that arise directly from my own parallel work with nonhuman animals. The primary result from such studies (Scott & Fuller 1965) is that behavior *is not inherited as such but develops*. Furthermore, genetically different animals are more similar in early development, and only differentiate later when exposed to similar experience. We shall never understand how genetics affects behavior until we study its development. Among humans, progress has been made with respect to the development of cognitive abilities (although seldom in consideration of genetic variation) but, so far as I know, no studies have been concentrated on the development of personality traits.

Second, our studies with the development of differential problem-solving abilities in dogs indicated that there were very few purely cognitive characteristics involved, but that motivational and emotional characteristics were extremely important. It follows that differential motivation, leading to differential development of abilities through practice, and so forth, may be the essential variable leading to differences in human intelligence test scores. The possibility should be explored.

Third, in highly social species such as dogs or humans, almost all behavior is expressed within social relationships. In such a relationship, the behavior of the individuals involved tends to differentiate as they organize it relative to each other. That is, the behavior expressed by an individual depends not only on his own genotype, but also on that of one or more others. In the case of individuals with similar genotypes, the relationship will still differentiate, but less so than in the case where the genotypes are divergent, as illustrated in the expression of agonistic (aggressive) behavior in dogs (Scott & Fuller 1965).

Applied to the problem of twin-based human research, this would be a possible explanation of the low correlations between relatives on personality tests. These presumably measure the organization of an individual's behavior with respect to other people, but since the tests only measure an individual's generalized responses, without any measures of the genotypes of the people with whom the organization was developed, it would be almost impossible to determine the effect of genetic variation. The animal research shows that the expression of aggressive behavior (for example) is overwhelmingly determined by the nature of the social relationship within which it is expressed.

Fourth, human twin research is limited by the fact that it is necessarily nonexperimental: The researcher has to study cases as they occur by chance. Such a limitation does not apply to animal research. My colleagues and I did preliminary studies that demonstrated the feasibility of experimentally manipulating a twin rearing technique. Puppies of different breeds (and hence different genotypes) were reared in like or unlike pairs (corresponding to monozygotic or dizygotic twins, although with some intrabreed genetic variation) under precisely controlled conditions, humans acting as surrogate parents. The results indicated, among other findings, that a portion of the behavioral differences developed by the pure breeds was due to being reared with puppies of similar genotypes (Scott 1977; Scott, Bronson & Trattner 1968).

Finally, the genetic theory commonly used in analyzing behavior-genetic research may be inadequate. As P&D point out, the theories developed for the analysis of quantitative characteristics such as growth depend on the assumption of equal and additive effects of genes, plus the random distribution of positive and negative effects of genes, so that a normal curve is produced.

The model does fit growth pretty well (any factor can only add or subtract in a dimension of size). Also, intelligence tests were designed to fit the model. Each question or problem can only have two results, correct or incorrect: The questions are selected so that approximately 50% of a given age group will pass. The result of each question is then added to others to produce a total score that, not unexpectedly, falls into a normal distribution. I am suggesting that the tests have been constructed to fit the model, not the behavior, which may explain the relatively low predictive value of IQ scores.

Now consider the behavior. Studies of the development of learning and problem-solving indicate that it does not proceed by slow accretion of individual items of information but by leaps and jumps. If one studies the development of problem-solving, the animal usually does better over time, but in a very irregular fashion, sometimes with wide swings in performance, sometimes with performance staying in a plateau for several days and then taking a sudden upward swing. And when one looks at the data from such experiments. it does not fall into normal curves. In order to use normal curve statistics and the associated correlational techniques, we have had to force the data into normal distributions (Scott & Fuller 1965).

In addition, there is strong evidence from studies of gene interaction that genetic effects are not only unequal but nonadditive, and that each gene affects many characteristics (pleiotropy). It follows that the important variables in behavior genetics are not the individual genes but gene combinations, and we need new theoretical models for their analysis. The distribution of gene combinations does not follow the conven-

tional model of genetic correlation between relatives, and thus casts doubt on the use of the usual correlation techniques. Such considerations may account for some of the apparent anomalies noted by P&D.

In summary, the results of twin research on human cognitive abilities and personality traits do not agree with those of parallel studies on nonhuman animals. One possible explanation is differences in methodology; data on humans cannot be derived experimentally in the same sense that it is in animal breeding. But the twin method can be adapted to experimental manipulation in dogs; the parallel technique might illuminate some of the mysteries of the human findings. Another possibility lies in the nature of behavior itself: Behavioral adaptation is not often additive and necessarily involves large amounts of nongenetic variation. A third possibility is that the genetic model commonly used is inadequate, being based on an over-simple set of assumptions about the nature of gene action and interaction.

Behavior genetics moves beyond percentages – at last

Robert J. Sternberg

Department of Psychology, Yale University, New Haven, Conn. 06520

It has become the most oft-told tale – the story of arguments among behavior geneticists regarding the percentages of this or that attribute of cognition or personality that are attributable to heredity and environment. The tale has become boring to at least some investigators outside the field, because the results were pretty predictable on the basis of who collected or compiled the data. One scarcely needed the data to predict the outcome, given knowledge of the investigator. Moreover, it was never clear what the percentages meant, anyway. What do we really learn about intelligence, for example, or its mechanisms of transmission, when we are told that the heritability of intelligence is, say, 50%? Given the susceptibility of the h^2 (heritability) statistic to population and to the circumstances under which the population is measured, as well as to the tests by which it is assessed, the amount of incremental information gained by successive behavior-genetic studies seemed to have reached the point of diminishing returns, with a few exceptions. Behavior genetics seemed to be making increasingly less contact with other fields within the social and natural sciences, because it seemed to have so little to say either about what the phenomena under study are (should we really be measuring intelligence, for example, by IQ test scores?) or about how the phenomena are transmitted (what genes or environmental variables really make a difference?).

The recent work of Plomin (e.g., Plomin 1986), Scarr (e.g., Scarr & Grajek 1982), Bouchard (e.g., Bouchard & McGue 1981), and others seems at last to be taking us beyond stale percentages and toward an understanding of what really matters – psychologically and sociologically – in the transmission of cognitive and personality attributes. The present review is an excellent example of work in this tradition. Although Plomin & Daniels (P&D) have relatively little to offer us by way of answers, at least they are asking the right questions. In particular, they are attempting to understand what it is about environments that is responsible for the environmental portion of various psychological attributes. I wish they had more answers, but at least they chart a useful path for future research.

At the same time, I could visualize researchers of their ilk repeating some of the mistakes of the personality theorists. Having finally recognized the importance of situations, personality theorists of a decade ago tried to study situations in the same way they studied traits, with relatively little success. Greater success was achieved later through the study of person–situation interactions.

I doubt that the usual run of variables – birth-order, number of siblings, education of parents, and the like – is going to yield many answers. P&D seem to agree. Rather, I think we need to understand the psychosocial dynamics of the family and the school, bringing behavior-genetic research more squarely into the club of which other psychologists and sociologists have been members for some time.

My own preference as to how problems of psychosocial transmission could well be studied is through an understanding of how patterns of environmental adaptation, shaping, and selection differ within families. Consider, for example, parental reward and punishment of children, something that is likely to have a substantial effect on the development of various psychological attributes in children. We could study the standard run of variables, such as gender, birth-order, and the like. But I believe we would do better to study how children react adaptively or maladaptively to punishment, thereby shaping their parents' environments, and ultimately and reciprocally, their own environments. Children largely create their own environments, and their creations reflect upon their parents' creations, which in turn reflect upon the children's environments. In the present target article, environments are viewed in rather more static terms than in some of Plomin's other writings, where environments are viewed as more dynamic and as resulting from the mutual creations of parents, children, and the children's siblings. The environments are not just there, but rather constructed through the mutual interactions of multiple parties. Such interactions are not well characterized by static sets of environmental variables that do not do justice to the often idiosyncratic outcomes within a given family.

The view of the importance of environmental adaptation, selection, and shaping in development would explain why it is *non*shared aspects of the environment that matter so much in the behavior-genetic studies. These functions depend on the interaction of person with environment, not simply on the environment per se. Moreover, they concern as much the style of *utilization* of cognitive and personality attributes as the attributes themselves. Thus, two individuals may have the same basic level of intelligence or introversion (or whatever) but may exploit it in different ways. As a result, measures of levels of personal or environmental variables simply do not reflect the ways in which these variables interact and the styles in which they are exploited.

To conclude, the target article by Plomin & Daniels represents an important step in a constructive direction for behavior genetics that will enhance its links with other areas of the study of psychosocial functioning.

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How do vulnerability effects relate to the nonshared environment?

Jim Stevenson

Department of Psychology, University of Surrey, Guildford Surrey GU2 5XH, England

Plomin & Daniels (P&D) have formulated a distinctive and important approach to theory about environmental effects on child development. The behavioral-genetic origins of their approach, however, results in an unresolved ambiguity. This stems from the initial need to identify nonshared environmental influences by their consequences rather than by directly considering the processes underlying them. With behavioral-genetic

methods, the nonshared environment is defined as those influences acting to make members of the same family unlike each other. This is a definition by effect, regardless of whether other evidence indicates that an event or circumstance is in fact commonly shared, as determined by direct measurement of life experiences by subjective report.

For example, housing conditions are experienced by all members of a sibship. It is known that adverse experiences do not have a uniformly detrimental effect on all children (Rutter 1983a). With the behavioral-genetic approach, a pattern of differential vulnerability to poor housing in a sibship will lead to an increase in variance attributed to the nonshared environment.

In this sense, a portion of nonshared environmental variance is attributable to differential vulnerability to shared environmental influences. It has been argued that temperament is one of the main influences on vulnerability (Rutter 1983). The nonshared environment may then subsume vulnerability effects which may themselves be subject to some genetic influences. P&D argue that their data show little evidence for genetic mediation of nonshared effects. This conclusion, however, is based upon an analysis of direct genetic influences on differential sibling experience and not upon differential sibling responses to the same experience.

These vulnerability effects are tangentially referred to in the target article as genotype–environment interactions. Although P&D admit that these effects cannot be identified in the twin and adoption studies that are the major source of their evidence, P&D do not go further to give any estimate of their potential contribution to phenotypic variance.

The first difficulty with P&D's model concerns the uncertain status of these vulnerability effects. A second difficulty concerns gender differences. In studies of monozygotic twins, estimates of nonshared effects necessarily leave out gender effects. This limits very much the generalisability of twin results to the general population. P&D refer repeatedly to such limits on twin data but not specifically to the absence of gender effects with the twin design. Indeed gender effects are given a strangely equivocal status throughout the target article. They are introduced in Table 1 and are commented upon again in the conclusions. The gender effects are said to account for a small portion of variance. I can find no evidence, however, reported in the paper to support the conclusion that along with birth-order effects they account for only 1%–5% of the variance of sibling differences in development. This is an important conclusion that relegates to a minor role an environmental influence that would appear to be a prime candidate as a source of nonshared effects; it requires justification.

The relevance of the concept of nonshared environment to the study of environmental influences: A paradigmatic shift or just some gears slipping?

Theodore D. Wachs

Department of Psychological Sciences, Purdue University, West Lafayette, Ind. 47907

In the midst of extolling the virtues of the concept of nonshared environment, Plomin & Daniels (P&D) note: "Our conceptualization of nonshared environmental influence is not new and exciting." Given the problems inherent in this concept, such caution is more than warranted.

Is there evidence to support the concept of nonshared environment? One argument made by P&D in support of a reconceptualization of the nature of environmental influences is that the results of traditional environmental studies have been "disappointing." This sweeping conclusion is based on a single reference (Maccoby & Martin 1983). A more comprehensive review of available evidence on environment-development relations would have resulted in quite the opposite conclusion. (See reviews by Gottfried 1984; Hunt 1979; Parkinson, Wallis, Prince & Harvey 1982; Rutter 1983; Seginer 1983; Wachs & Gruen 1982.) Given this data base, the validity of P&D's conclusion seems akin to my dismissing all of behavior-genetic research as suspect, based on evidence showing that Cyril Burt had fabricated his family and twin data (Dorfman 1978).

A second source of data used to support the concept of nonshared environment comes from research on the development of personality and psychopathology in twins. However, even genetic data suggest cause for concern over the generalizability to nontwins of results obtained with twins (Henderson 1982). Furthermore, in the overwhelming majority of twin and adoptive studies cited by P&D, environment is rarely measured directly. In an earlier paper I documented the impossibility of drawing valid conclusions about the nature of environmental action when indirect or residual measures of the environment are used (Wachs 1983). Not surprisingly, in the few twin studies where environment was directly measured, the results are quite discrepant from what is typically reported when indirect or residual measures of environment are used (Bronfenbrenner 1985; Lytton 1980). Thus, while I cannot doubt the relevance of twin studies for genetic research, the above factors make it extremely risky to draw conclusions about environmental influences from these types of studies.

The third argument used by P&D revolves around the low personality correlations for biological siblings living in the same home. This argument is valid only if we accept two assumptions: first, that biological sibs are actually encountering similar environments and, second, that there is no organism/environment interaction occurring. In terms of studies supposedly supporting the first assumption, although Dunn, Plomin and Nettles (1985), conclude that the stability of maternal behaviors toward same-age sibs approaches that of the stability of the observations, the modest overall mean correlation of maternal behavior toward sibs (.33) is well below the overall mean reliability of the observations (.58) (Dunn et al.'s Table 3). In the remaining studies cited as evidence which were available to me, Jacobs and Moss (1976) report more differences than similarities in treatment of same-age sibs, although Abramovitch, Pepler, and Corter (1982) report "complex and uninterpretable" results. Hence, the studies cited by P&D offer little support for the assumption of similar treatment of same age siblings. In contrast, the data they cite on differential treatment of different age sibs, plus other evidence on the indirect (Wachs 1985) and direct (Abramovitch et al. 1982; Norman-Jackson 1982) environmental impact of older sibs, indicate that biological sibs, though reared under the same roof, may be living in radically different environments.

Concerning the second assumption of no interaction, P&D argue that environmental factors which are commonly experienced cannot be a source of differences. A number of studies, however, have reported that differential impact of the environment occurs as a function of the sex of the child (Block, Block & Morrison 1981; Pedersen, Rubenstein & Yarrow 1979; Wachs 1979; 1984) and child temperament (Wachs & Gandour 1983), in spite of the fact that there were no differences in the environments provided to males versus females or easy versus difficult temperament children. These results not only make it difficult to assume no interaction in sib studies, but also directly contradict one of the major predictions drawn by P&D in their discussion of nonshared environmental influences.

The fourth line of evidence cited by P&D involves the low phenotypic correlations reported between adopted children and adopted parents, as well as the decline in existing correlations when adopted children leave home. Again, as with twins, adoption studies rarely measure environment, but only infer

environmental influences. In addition, conclusions drawn from adoption studies depend on what statistics are used to measure the effects of adoption (Weizmann 1971). For example, clearcut cognitive gains occur for disadvantaged children adopted into more optimal environments (Clarke & Hanisee 1982; Scarr & Weinberg 1976; Schiff, Duyne, Dumaret, Stewart, Tomkiewicz & Feingold 1978), in spite of the fact that the correlations between the child's IQ and that of the adoptive parent are typically zero order. Furthermore, given evidence that later changes in environment can offset the effects of prior experience (Clarke 1984), it is not at all surprising that correlations between adopted sibs decline after they leave home; if anything, these results show that when shared environment (such as it is) diminishes, relations between adopted sibs are attentuated.

Do we need the concept of nonshared environmental influences? Although the empirical basis underlying the concept of nonshared environmental influences is, at best, highly questionable, P&D could argue that this concept is needed to answer the question: "Why are children in the same families so different from one another." To the extent that P&D view environmental influences only in terms of producing similarities among individuals, the need for such a conceptual revision would be obvious. Surprisingly, no environmental researchers are cited by P&D supporting this viewpoint. This is because no major environmental researcher or theoretician has made the claim that environment acts primarily to produce similarities among individuals. Rather, like behavior geneticists, environmentalists are primarily interested in identifying what aspects of the environment relate to variability in behavioral development (Wachs 1983).

In addition, in basing their concept of environment and their arguments for nonshared variance primarily on behavior genetics research, P&D ignore current models of environmental action, which can easily answer the question without recourse to a concept of nonshared environment. As an example, I will use two principles developed from my own environmental action model (Wachs 1985; Wachs & Gruen 1982).

1. *Environmental specificity:* Different aspects of the environment predict different aspects of development.
2. *Organismic specificity:* Individual differences will mediate the influence of the environment.

To the extent that sibs encounter different environmental conditions, a situation which we have documented earlier as quite plausible, environmental specificity would predict different outcomes for sibs. To the extent that sibs differ on various organismic parameters such as temperament, organismic specificity would predict differential reactivity to the environment, leading to differential outcomes. Data I have cited earlier clearly support this conclusion as well.

Wither the concept of nonshared influences. If P&D are willing to accept more modest goals, rather than insisting on a paradigm shift, some testable questions emerge from their target article. For example, the development of instruments like the Sibling Inventory of Differential Experience (SIDE) may allow us to increase our understanding of environmental specificity by determining which aspects of development are most sensitive to directly observed (objective) aspects of the environment compared to those which are more sensitive to subjective aspects (the child's perception).

P&D have also illustrated one important point, though not the one they intended. Presenting the concept of nonshared environmental influences without reference to methods and concepts used by environmental researchers illustrates the importance of cross-disciplinary collaboration, rather than simply assuming that we understand the methods and concepts used by other disciplines (Wachs 1983). Assuming rather than collaborating virtually guarantees that attempts to bridge disciplines will primarily be of the nonshared variety.

All parents are environmentalists until they have their second child

Marvin Zuckerman

Department of Psychology, University of Delaware, Newark, Del. 19716

What is it that happens to parents after they have their second child that shakes their belief in the *tabula rasa*? With an *n* of one, parents can assume that all of their child's positive traits are due to their enlightened methods of child rearing (negative traits being assigned to a genetic influence from other relatives). Then they use the same methods with the second child, who turns out to be so different from the first that they have to question their premises. This is essentially what Plomin & Daniels's (P&D's) target article is about. The behavioral-genetic data refute the popular notion among laymen, and most social psychologists, that individual differences in personality, intelligence, and psychopathology are a result of familial (shared) environmental influences. However, P&D may be attributing too much importance to nonshared social environmental influences and minimizing other influences and the genotype–environment interaction. Their studies of nonshared sources of variance in siblings do not establish whether these are simple environmental influences or reactions of the social environment to phenomenal expressions of the genotype.

Until recently, twin comparison has been the primary biometric method for estimating the influences of heredity and environment on a particular trait. Studies of separated identical twins have been of particular value because, barring selective placement, they provide a direct estimate of heritability in the correlation between the twins. Table 1 shows results from four such studies (Lykken, Bouchard, Tellegen, Wilcox, Segal & Rich 1985; Newman, Freeman & Holzinger 1937; Pederson, Friberg, Florderus-Myrhed, McClearn & Plomin 1984; Shields 1962) dealing with personality traits along with some of the larger studies of twins reared together in the same family. Since slight variations in twin correlations can produce marked differences in heritability estimates, it is important that large numbers of twins be used in any study of heritability.

Rather than speaking of the results for personality in general, as most reviewers have done, I believe it is important to look separately at three broad traits which have been found to provide the most reliable description of personality across cultures, genders, and age (Eysenck & Eysenck 1985): Extraversion (or Sociability); Neuroticism (or Emotionality); and a factor that Eysenck and Eysenck (1976) have called "Psychoticism," but which can better be described as "Psychopathy" versus "Socialization," or "Impulsive Unsocialized Sensation Seeking" (Zuckerman, Kuhlman & Camac in press). On the basis of a reanalysis of some of the Loehlin and Nichols (1976) data, Loehlin (1982) reported that there is evidence for differential heritability of different personality traits, a conclusion that had been questioned in the earlier work. If there is differential heritability we must look separately at different traits of personality.

The median correlation values in Table 1 suggest several immediate conclusions. The lack of difference between the correlations of monozygotic twins raised apart (MZa) and those reared in the same family (MZt) suggest: (1) Shared environment is unimportant in the observed similarities in identical twins; (2) the broad heritability of the three major personality traits, estimated directly from the MZa correlations, is about .5 for all three traits; (3) whereas the MZ correlations are fairly consistent among the three traits, the correlations between dizygotic twins raised together (DZt) vary considerably – the median correlations for E, N, and P are .12, .24, and .30 respectively. If we were to calculate the Falconer index ($2 \times$ MZt − DZt r's) of heritability (h2) from these correlations, the values would be .78 for E, .56 for N, and .46 for P. However, these

Table 1 (Zuckerman). *Twin studies of personality: Correlations of monozygotic twins raised together (MZt), monozygotic twins raised apart (MZa), and dizygotic twins raised together (DZt)*

Studies	Extraversion (E)			Neuroticism (N)			Psychopathy (P) type scales		
	MZa	MZt	DZt	MZa	MZt	DZt	MZa	MZt	DZt
Newman et al. (1937)[a]	—	—	—	.58	.56	.37	—	—	—
Shields (1962)[b]	.61	.42	−.17	.53	.38	.11	—	—	—
Lykken et al. (1985)[c]	.44	.60	.01	.66	.67	.43	.52	.52	.33
Pederson et al. (1984)[d]	.54	.44	.26	.18	.37	.18	—	—	—
Floderus et al. (1980)[e]	—	.51	.21	—	.50	.23	—	—	—
Loehlin & Nichols (1976)[f]	—	.60	.24	—	.53	.25	—	.54	.32
McClearn & DeFries (1973)[g]	—	.45	.12	—	.44	.14	—	.48	.27
Buss & Plomin (1984)[h]	—	.53	−.03	—	.63	.12	—	—	—
Fulker et al. (1980)[i]	—	—	—	—	—	—	—	.60	.21
median correlations	.54	.51	.12	.56	.52	.24	.52	.53	.30

[a]Based on 50 MZt, 50 DZt, 19 MZa, mean ages: MZt, DZt = 13–14, MZa = 26. Test of neuroticism was the Woodworth-Matthews (number of neurotic traits). See S. I. Franz.

[b]Based on 44 MZt, 44 MZa, 32 DZt, most twins in 30's or 40's, tests of E and N in a questionnaire devised by Eysenck.

[c]Based on 174 MZt, 44 MZa, 110 DZt, mean ages: MZt = 20.6, MZa = 36.7, DZt = 21.4; test used was Tellegen's Multidimensional Personality Inventory (Tellegen 1985); results for 3 factors: Positive Emotionality (like E), Negative Emotionality (like N), Constraint (like P−).

[d]Data reported in Plomin (1986); 36–59 MZa, matched sample of MZt, 121–159 DZt, E and N scores from a short form of the Eysenck Personality Inventory (EPI) (Eysenck & Eysenck 1964).

[e]Based on 5025 MZt, 7873 DZt, ages 18–50. E and N scores from short form of EPI.

[f]Based on 514 MZt, 336 DZt, high school seniors who took National Merit Scholarship exam; E and N scores based on scores derived from factor-related scales of Gough's California Psychological Inventory (Gough 1957); the data on the Socialization scale are used to represent the P factor.

[g]Based on 120 MZt, 132 DZt, combined data from 3 separate studies; Scales from the Minnesota Multiphasic Personality Inventory (MMPI) used to represent the three factors: Social Introversion for E, Depression for N, and Psychopathic Deviate for P.

[h]Based on 228 MZt, 172 DZt, mean age 61 months, from 4 studies; Parents rated children on scales from the Buss and Plomin Emotionality Activity Sociability Test (EAS) (Buss & Plomin 1975).

[i]Based on 466 MZt, 276 DZt, mean ages MZt = 32.9, DZt = 30.4, P factor based on Zuckerman's Sensation Seeking Scale (Zuckerman, Eysenck & Eysenck 1978), Total score form V (SSSV).

variations in h2 are produced by the variations in the DZ, not the MZ median correlations. The frequently low DZ correlations for extraversion may indicate the influence of nonadditive genetic variance or simply chance fluctuations around the "true" correlation. The largest studies, by Floderus-Myrhed, Pederson and Rasmuson (1980) and Loehlin and Nichols (1976), yielded DZ correlations (.21 and .24) that are only a little less than half of the MZ ones (the proportion that would be expected from an additive genetic model). Estimates of heritability using the model-fitting method of Jinks and Fulker (1970) have produced estimates of heritability of all three traits that are close to the .50 estimate (Fulker 1981).

As P&D say, the issue about the magnitude of the genetic contributions does not affect their point that the environmental part of the variance is almost entirely of the nonshared environment type. The importance of these nonshared environmental factors, however, does depend on how we estimate the variance. The treatment of the error factor in the trait measurements is important, and it is not accurate to add it into the environmental part of the equation through simple subtraction of the genetic component. Although some personality measures have a reliability of .9, reliabilities of .8 and lower are more common. This means that 20% of the variance is due to error of measurement. It could be argued from the evidence that genetic variance accounts for 63% of the *reliable* variance (.5/.8) while nonshared environment accounts for 37% of this variance (.3/.8) for the three broad personality traits.

Putting the influence of nonshared environment in better quantitative perspective, what are the possible sources of this variance? Test reliability has already been discussed although it is possible that it may vary from age to age, making comparisons between siblings or between parents and children problematic. The unsystematic or serendipitous events may be a "gloomy prospect" for investigators, but in particular cases they may be quite crucial. Pregnancy and birth complications, which may affect one twin or sibling more than the other, increase the risk factor in schizophrenia for those who are genetically vulnerable (Mednick 1966).

Because there does not seem to be a great deal of assortative mating for the three major traits (Fulker 1981), chance factors in the selection of spouses or close friends can certainly influence personality, at least in its behavioral manifestations. Differential treatment of siblings and sibling interactions have been emphasized by P&D, but Loehlin and Nichols (1976) could find no relationship between differential parental treatment of either identical or fraternal twins or the time the twins played or spent together or had the same teachers, and the resemblance in their personality traits. P&D's data (in their Table 2) on the Sibling Inventory of Differential Experience show a very low agreement between siblings on perceptions of differential parental

treatment and not much better agreement on sibling interaction measures. They argue that the sibling's perceptions are more important than the "real" interactions. But a person's social perceptions, particularly the idiosyncratic ones, are a function of their personality, which is to a large degree a function of genetic influences. P&D dismiss a genetic influence on these experience measures because the relationships of differences in experience and differences in personality are not higher for fraternal than for identical twins or for adoptive than for nonadoptive siblings. But P&D admit that the relationships were weak for both types of twins and adoptees. Perhaps these differences in experience are simply not important for either type of relationship or the measures are not valid enough to reflect important differences in treatment.

There is simply no way to disentangle cause and effect using such concurrent data. What are we to make of the fact that differences in mothers' encouragement of development correlate with differences in siblings' activity levels, or that differences in peer popularity of siblings correlate with differences in self-reported sociability? It is just as plausible to assume that mothers react to the differences in their children's innate activity levels and that peers or siblings react to the sociability of the individual as to suppose that these environmental differences produce the differences in personality. Lytton (1977), in a rather elegant behavioral analysis, found that parents responded more similarly to identical twin behavior than fraternal twin behavior as a function of the differences in the twins' behavior, rather than that the parental behavior produced the twin differences.

Apart from differences in personality traits, differences in age (of siblings) could influence differences in personality. Not all personality traits appear in the same way at different ages but few sibling studies have reported the influence of age differences. Gender differences have been found for two of the three major dimensions of personality (Zuckerman et al. in press) and it would be surprising if opposite sex siblings did not elicit different treatment as a function of their gender. Where are the data comparing opposite sex and same sex siblings? Siblings of the same sex may differ in physical attractiveness, coordination, athletic ability, and intelligence, all traits which may strongly affect the reactions of others toward of them.

What I am suggesting is that a great part of the nonshared environment of fraternal twins or siblings is due to genotype–environment interaction rather than representing a pure environmental source of variance. This would augment the similarity of identical twins but could reduce the similarity of fraternal twins or siblings. The simple additive model discussed in this paper cannot disentangle this kind of interaction. Scarr, Webber, Weinberg, and Wittig (1981) have described an interactive hypothesis in the following terms:

> If as the twin studies suggest, the heritability of personality is .5, then the resemblance of genetic relatives should be far greater than we found it to be. It may be however, that individual genotypes evoke and select different responses from their environments, thereby creating genotype-environment correlations of great importance. . . . Thus it may be that the unique genotypes of individual children determine much of the environment they experience and explain much of the variance we are able to measure in studies of personality. (p. 897)

This kind of model would explain the astounding similarity of many identical twins reared apart, in their specific interests, mannerisms, and habits as well as broader personality traits (Lykken 1982). Given a genotype of a certain kind, there is a tendency to select the type of environment that best fits the needs stemming from the genotype. Sociable people seek other sociable people who reinforce their social habits. Antisocial adolescents associate with other delinquents who provide models and reinforce their own antisocial behavior. A more conforming sibling may join the boy scouts who model and reinforce prosocial behavior. The public tends to take extreme positions like "bad seed" or "bad company" as the explanation for antisocial behavior. Our models must allow for the complexities of interaction.

Editorial commentary

How is one to differentiate *independent* nonshared environmental effects on (nontwin) siblings from those that depend systematically on gene-environment correlations, with the latter perhaps more accurately interpreted as the environmental potentiation or amplification of genetic differences? Certainly any case of differential *self-selection* of an environment on the basis of a genetic disposition or inclination would fall into this category (e.g., the tone-deaf, athletically gifted sibling who consequently spends all his time working out in the gym, compared to the uncoordinated but musically talented one who accordingly prefers to spend that time practicing singing). So would any clearcut disposition-induced differential *social reaction pattern* (the gregarious sibling versus the loner; the self-willed one versus the compliant one; the chipper, ambitious optimist versus the gloomy, passive pessimist, etc.). Such initial constitutional differences may be overlain and modulated by a world of subsequent environmental difference, but those environmental differences would hardly seem to represent *primary* causes. And neither systematic differential birth-order effects nor unsystematic differential environmental perturbations look to be strong enough to bear this primary causal burden. (Identical-twin effects may just be a nonrepresentative special case of birth-order effects in which deliberate efforts to promote distinctness, including environmental distinctness, have played an artifactual role.)

Authors' Response

Children in the same family are very different, but why?

Robert Plomin[a] and Denise Daniels[b]

[a]Department of Individual and Family Studies, Pennsylvania State University, University Park, Pa. 16802 and [b]Department of Psychiatry, Stanford University, Stanford, Calif. 94305

Our main goal in writing the target article on nonshared environmental influences was to stimulate our peers to think about a provocative finding from the field of behavioral genetics – that children in the same family are very different from one another – and to ask "why?" The response, seen in the accompanying commentaries from distinguished scientists from many disciplines, may prove to be a watershed for research on this topic. We are grateful to these luminaries for casting their light in this direction.

The most important point to note from the commentaries is that no one disagrees with our conclusion that children in the same family are very different. This is significant because environmental influences have usually been assumed to be shared by children in the same

family. In contrast, the major implication of our target article is that the important environmental influences are those that make children in the same family different from one another.

Although the commentators agree that children in the same family are very different from one another, there is some disagreement as to the importance of nonshared environmental influences in explaining these differences. After discussing this, we turn to commentators' suggestions about possible environmental sources of differences between children in the same family and new methods that might assist us in the search for nonshared environmental influences. A few other general issues that emerged in several commentaries are discussed next, and the remaining specific issues raised by the commentators are addressed last.

Explanations of differences between children in the same family

Several interesting ideas emerged in the commentaries as to the source of differences between children in the same family. Most of these ideas involve nonshared environment. However, two arguments should be mentioned first because they suggest that differences between children in a family are not due to nonshared environment. First, several of the geneticists suggested that the differences are really genetic. Second, several commentators argued that error of measurement is responsible for the differences.

Is it all genetic? Several of the commentators (**Boklage, Boomsma, Hartung, Hay, Jensen, Lykken, Scott, Zuckerman**) suggest that children in the same family are different from one another, not for reasons of nonshared environment, but rather for genetic reasons. Although Boklage speculates that extranuclear inheritance might account for some of the differences between children in the same family, the possible importance of nonadditive genetic variance is the major objection raised in these commentaries.

Because these commentaries focus on nonadditive genetic variance as an alternative explanation for why children in the same family are so different, the reader with less than eidetic memory might derive the impression that we have not addressed this issue. So, we begin by referring the reader to our passage about nonadditive genetic variance in the target article (Sec. 1.1, para. 3).

As indicated in that passage, **Lykken** has highlighted the possible importance of nonadditive genetic variance – one of the most interesting genetic developments that we have seen in the past decade in the field of quantitative genetics. In his commentary, Lykken describes some personality data that fit his hypothesis – high identical twin correlations and low fraternal twin correlations. The main point of his theory bears repetition: "The essence of configural determination is that even a small change – that is, one different gene in the pattern, like one different digit in a telephone number – can make a large or even qualitative change in the result."

We agree that nonadditive genetic variance is an important possibility for some traits. However, to the nongeneticist this hypothesis smacks of trying to say that all variation is really genetic in origin. Furthermore, the hypothesis cannot explain two facts. First, identical twin correlations are generally substantially lower than the reliability of the measures. Nothing can explain such differences within pairs of identical twins other than nonshared environment. Second, the few studies that have investigated measured aspects of nonshared experience have found some systematic environmental variance of this type, as discussed in our article.

Recent developments in molecular genetics suggest that it is highly unlikely that all phenotypic variance is genetic. **Caspari** explains some of the complexities that have been found in recent work in eukaryotic genetics. As a more general example, consider *Caenorhabditis elegans*, a 959-celled roundworm with about two thousand genes, which has the distinction of being the first organism with a complete wiring diagram of its nervous system, a complete developmental fate map of each cell, and extensive information about the genetic expression of its genes. Despite all of this knowledge, molecular geneticists still have little idea of the molecular mechanisms of development. There is, however, a growing consensus that there is no simple chemical device as there is in the case of the genetic code (Wilkins 1986). Simple regulation metaphors such as the operon model do not generally apply to eukaryotic development. The only certainty is that genetic influences on development are complex. The message for us is that it would be amazing if this complexity did not admit environmental perturbations as part of the provenance of variability in development.

Scarr is not concerned about nonadditive genetic variance. Nonetheless, her commentary implies that all phenotypic variance is ultimately genetic, which is part of her theory (Scarr & McCartney 1983, p. 424) of genotype→environment processes: "The environments that children receive are . . . at least . . . in part the result of their own personal characteristics, such as interests, talents and personality." She disagrees with our conclusion that genetic factors seem to be less involved in environmental differences perceived by siblings than they are in traditional environmental measures; her theory, although not phrased in terms of nonshared environment, would predict that environmental differences are correlated with genetic differences.

We agree that genotype–environment correlation is important and that much more work is needed on this interesting issue (see also the **editorial commentary** and the commentary by **Rowe**). Genotype–environment correlation, however, is not an environmental effect – it refers to variance due to environmental exposure correlated with genetic dispositions. To say that genotype–environment correlation underlies nonshared environment is really to say that there is no "main effect" of nonshared environment, that experiential differences between siblings really reflect genetic differences between them. Although genotype–environment correlation might be shown to underlie some of the differences in behavior that we have identified as nonshared environment, it runs into trouble in trying to explain results for identical twins. Members of identical twin pairs do not experience differences in their environments because of genetic differences between them: they are genetically identical. Yet we find that identical twins are by no means identical phenotypically. What accounts for the dif-

ferences within pairs of identical twins? The answer is nonshared environment, not genotype–environment correlation. (The only way out is to argue, as does the **editorial commentary**, that identical twin data may not be generalizable; that argument, however, seems post hoc and unparsimonious.)

Finally, it is noteworthy that commentaries from other geneticists do not try to argue that genetic variance can explain away nonshared environmental influence: **Bock & Zimowski, Caspari, Johnson & Nagoshi, Loehlin, McArdle & Gottesman, Morton, Rose & Kaprio,** and **Rowe.**

Error of measurement. Several commentators (most notably, **Bock & Zimowski, Costa & McCrae, Kovach, Zuckerman**) indicate that error of measurement will make children in the same family appear to be different from one another and must therefore be disentangled from nonshared environmental sources of differences. We said this in several places (see especially sec. 1.2, para. 4), but it apparently needs greater emphasis. However, it takes some contortions to suggest that error of measurement can account for all of the variance we identify as nonshared.

Costa & McCrae also suggest that reliabilities of personality questionnaires may be as low as .60 if one were to depend on correlations between self-reports and ratings by others, rather than the .80 reliabilities typically suggested by test–retest reliability. If the reliability of self-report personality questionnaires were really as low as .60 we would have to conclude that most of the nongenetic variance was due to error of measurement. However, unless one is trying to explain away nonshared environmental variance, reliability based on ratings by others is not a sensible estimate of reliability for the self-report data used in most twin and adoption studies. Test–retest reliability is far more appropriate as a gauge of the reliable variance in correlations between pairs of individuals such as twins than the correlation between self-report and ratings by others. Moreover, one could argue in the opposite direction that estimates of reliability based on internal consistency are more appropriate; internal consistency estimates are usually higher than test–retest estimates and would thus ascribe an even greater role to nonshared environment.

We agree with the additional point made by **Bock & Zimowski, Costa & McCrae,** and **Jensen** that psychometric issues other than reliability need greater attention. Bock & Zimowski and MacArdle & Gottesman recommend the use of multitrait–multimethod approaches and structural equation modeling to attack some of these problems. These methods, however, are not the panacea that these commentators seem to suggest. The idea of modeling causal relationships among "error-free" constructs is appealing, but its success will depend on the marker variables used to construct the constructs, which raises long-standing problems in multivariate research concerning domain specification and representative sampling from domains (Nesselroade & McArdle 1986). We do not disagree with Bock & Zimowski, however, that more thought and research need to be devoted to the effect of error of measurement in quantitative genetic analyses.

In summary, we maintain that the differences between children in the same family are not all due to genetic factors, nor are they simply due to error of measurement. Because we believe that the differences are due to nonshared environment, we were particulary interested to see what ideas the commentators had as to where we might look to isolate specific sources of nonshared environment. As **McCartney** points out in the title of her commentary, the problem is now to document systematic nonshared environmental effects.

Evolutionary hypotheses. One of the most novel commentaries is that of **Buss,** who tackles head-on the most important issue of where we should look for systematic sources of nonshared environment. Rather than a hodgepodge list of possibilities, he provides a series of hypotheses about differential parental treatment that emerge from an evolutionary perspective. He suggests, for example, that differential parental favoritism might occur as a result of differences in parental investment or differences in paternity confidence (as indexed by physical or behavioral similarity to the father).

Prenatal environments. It is suggested by **Bock & Zimowski** and by **Roubertoux & Nosten** that prenatal environments are an important source of differences between children in the same family. We would not wish to discourage research on any possible source of nonshared environment. It needs to be noted, however, that Bock & Zimowski include no references for their statement that prenatal effects are important and long-lasting. In contrast to the abundant literature on prenatal causes of biological abnormality, there is a sparse literature on prenatal causes of personality differences among normal children (Kopp & Parmelee 1979). So far as we know, the only trait for which prenatal factors relate significantly to behavioral differences in the normal range is IQ. Twins and babies born prematurely are known to have lower IQ than the average of the population. However, data from the Collaborative Perinatal Project, which included over 26,000 children, suggest that the combination of prenatal and perinatal factors explains less than 4% of the variance of IQ by the age of 4 years (Broman, Nichols, & Kennedy 1975).

Bock & Zimowski state that prenatal effects are especially severe in twins and "are believed to account for much of the variation between identical twins that P&D use as a measure of the nonshared environment." We disagree for two reasons. First, although twins tend to be born premature and with low birth weights, twin decrements in verbal performance disappear by school age (Wilson 1978). The current view is that prenatal and perinatal factors have few long-term effects unless the child's environment continues to be adverse (Sameroff & Chandler 1975). Second, if prenatal effects are severe for both identical and fraternal twins, as suggested by Bock & Zimowski, these effects would be read as *shared* environmental variance. This must be nearer the mark because twin research in infancy, when prenatal effects are likely to be strongest indicates that both identical and fraternal twin correlations are high for IQ – about .70 (Wilson 1983).

Family constellation variables. Several commentators (**Harper, Lamb, Nyborg, Stevenson**) think that family constellation variables such as birth-order and gender are better bets than we suggest. Let us be clear: We have

nothing against birth-order or gender – we are just trying to suggest other directions for research that we think will be more profitable.

Consider birth-order. **Harper** suggests that the impact of our message is weakened by "several inconsistencies." One of these is that we say that causes of differences within families need not be the same as causes of differences between families "and then they minimize birth-order effects on the basis of low between-family relationships." By way of reply, the reader is referred to our original passage (sec. 5.1, para. 1). In addition, it is noteworthy that **McCartney** in her commentary states that "most developmentalists agree that family constellation variables, such as birth-order, have small predictive power."

Stevenson asks for references in relation to our conclusion that gender differences explain little variance. The authors of the best-known book on gender differences (Maccoby & Jacklin 1974) now tend to emphasize the similarity of boys and girls: "The most important point is that there is very little to explain. Recent publications concentrate not on whether a sex difference exists but on how large a difference really exists" (Jacklin & Maccoby 1983, p. 183). For example, one of the best documented gender differences in the cognitive realm is the superiority of females on tests of verbal ability. This average difference in verbal ability between the genders, however, accounts for only about 1% of the variance (Plomin & Foch 1981). It is our reading of the literature that this conclusion has become widely accepted during the past five years. Nonetheless, one could argue, as **Lamb** does, that gender has most often been studied across families rather than within families. This does not seem to be an important source of variance either, however, because behavioral genetic studies yield similar resemblances for same-sex and for opposite-sex sibling pairs.

Nonetheless, we reiterate that even if gender explains only a minuscule portion of variance, we do not wish to inhibit researchers from studying gender-related issues as a possible source of nonshared environmental influence. We simply suggest that more dynamic family variables are better bets.

Interactions. Temperamental differences among children, suggest **Chess** and **Wachs,** might alter the effects of experiences and such interactions could lead to behavioral differences among children. Chess is particularly interested in interactions of the type described in the Chess and Thomas model of goodness-of-fit. Wachs emphasizes what he refers to as "organismic specificity."

Temperament-mediated interactions represent an important direction in the exploration of nonshared environmental influences. So far, however, it has proven to be surprisingly difficult to find such interactions (Plomin & Daniels 1984). Perhaps the search for nonshared environmental influences will sharpen our measurement of the environment and reveal interactions that have been elusive in research using traditional measures of the environment.

Kovach, Nyborg, and **Zuckerman** suggest that genotype–environment interaction is likely to be important. This issue was discussed in our target article; we also indicated how it bears on the estimation of shared and nonshared components of environmental variation. Kovach describes a new study by Tienari, Sorri, Lahti, Naarala, Wahlberg, Ronkko, Pohjola, and Moring (1985) that adds to the weight of our conclusions concerning schizophrenia: Heredity plays some role and nonshared environment accounts for the rest. Using specific measures of the environment, however, Tenari et al. found some evidence of an interaction in that schizophrenia occurred primarily among adoptees reared in "disturbed" families. Interpreting this finding is not easy, as Kovach mentions.

Just as we are not opposed to studying family constellation variables, we are not against interactions as a possible source of nonshared environmental influence. In fact, we have developed methods that permit us to identify such interactions (e.g., Plomin & Daniels 1984; Plomin, DeFries & Loehlin 1977), but so far they have been difficult to find.

For example, although the diathesis-stress model of schizophrenia has been around for a long time, empirical support for this reasonable interaction hypothesis has not been forthcoming. [See also Anisman & Zacharko: "Depression: The Predisposing Influence of Stress" *BBS* 5(1) 1982.] In an excellent review of schizophrenia, Gottesman and Shields (1982) conclude:

> This line of reasoning from the diathesis-stressor framework makes it clear that although the genes may be necessary but not sufficient for causing schizophrenia, the environmental contributors may also be necessary but not sufficient, and not yet specifiable other than on a case-by-case basis. (Gottesman & Shields 1982, p. 181)

Accidents and illnesses. The possible importance of accidental and unpredictable events is stressed by **Chess, Lovach,** and **McCartney** who feel that we minimized the effects of such events. When we said that "such capricious events are likely to prove a dead end for research," we did not mean to minimize the possible importance of such events as sources of nonshared environment. Our point was that it makes sense to start the search by looking for systematic sources of variance.

Perceptions and attributions. Emphasis is placed by **Bernstein** on the possible importance of perceptions of the environment, "the ability to construct diverse perceptions and realities from the same objective reality." We agree with his emphasis. Indeed, the recognition of this possibility led us to develop the Sibling Inventory of Differential Experience (SIDE) (Daniels & Plomin 1985). We also agree with Bernstein's point that it will not be easy to study perceived experiences because of problems with self-report data, a point that **Harper** also makes.

However, rather than saying, as Bernstein does, that the SIDE is "terribly crude," it would be more helpful to think constructively about alternatives. Our recent attempts to observe mother–infant interactions were in part motivated by the thought that the boundary between the subjective and the objective is less sharply defined for very young children.

Rowe raises the general issue of attribution and calls for more research in this vein. It should be noted, however, that if nonshared environmental variance primarily involves perceptions and attributions, we will still need to ask why two children in the same family develop such

different views of their home and community environments.

Motivation. In his extensive research with problem-solving abilities in dogs **Scott** has found that differential motivation was a prime factor. He suggests that differential motivation leads to differential development of abilities through practice. It may be time for us to dust off the old construct of motivation.

Social relationships. It is also suggested by **Scott** that the study of social relationships might provide a key for understanding nonshared environments. He notes that in highly social species such as dogs or humans, almost all behavior is expressed within social relationships and that the behavior of individuals differentiates as they organize their behavior relative to others. In a similar vein, **Sternberg** suggests that "we need to understand the psychosocial dynamics of the family and the school."

Traumatic conditioning. His model of neurosis and criminality, **Eysenck** notes, could contribute to a theory of nonshared environment. Specifically, Eysenck's model invokes the importance of traumatic and subtraumatic conditioning experiences, which he suggests will inevitably be of the nonshared variety. We hope that Eysenck will further explicate this idea, for we could certainly use a theory of nonshared environment.

Learning processes. It is cogently argued by **Sternberg** that patterns of environmental adaptation, shaping, and selection should be studied as a source of nonshared environmental influence. Specifically, he suggests that research should consider "how children react adaptively or maladaptively to punishment, thereby shaping their parents' environments, and ultimately and reciprocally, their own environments."

Niche-picking. It is proposed by both **McCartney** and **Scarr** that their theory could serve as a starting point in the search for nonshared environment. Their theory of "genotype→environment effects" attempts to describe processes transacting at the developmental interface between nature and nurture (Scarr & McCartney 1983). The theory emphasizes the possibility that children evoke certain responses as a function of their genotypes and that the children select and even synthesize their own environments. As mentioned earlier, Scarr & McCartney' theory is related to genotype–environment correlation, which refers to an increase in phenotypic variance that occurs when children experience environments correlated with their genetic propensities. Although genotype–environment correlation cannot explain differences within pairs of identical twins, it could, as both Scarr and McCartney have pointed out, explain increasing differences within pairs of biological or adoptive siblings. Leaving aside the components of variance of quantitative genetics, their theory points to important processes by which children in the same family might diverge during development.

Extremes. In addition to these suggestions concerning specific sources of nonshared environment, commentators made three methodological suggestions that might assist us in the search. **Lykken** notes that some parents – he uses the Kennedy family as an example – appear to have a considerable impact on their children. He suggests that research could focus on "families that are exceptions to the rule, families that manage to provide a shared environment that does influence each of the siblings in similar ways." We have become interested in the possibilities of a related tack: Study siblings or twins who are very different in order to sharpen the search for nonshared environmental causes of their differences.

General versus specific nonshared factors. The useful point is made by **Boomsma** that multivariate behavioral genetic methods can be used to explore the extent to which nonshared environmental variance is general or specific in its effects on behavior. She mentions two multivariate analyses that suggest that, in the domain of cognitive abilities, genetic and shared environmental effects are quite general in their effect, whereas nonshared environmental effects appear to be specific.

It should be pointed out that the most straightforward approach to this issue uses differences within pairs of identical twins. As indicated earlier, reliable differences within pairs of identical twins occur only as the result of nonshared environment. Thus, if identical twin differences on trait X correlate with identical twin differences on trait Y, we can conclude that nonshared environmental influences that affect trait X also affect trait Y. A matrix of such correlations can be factor analyzed to address the issue of dimensionality.

The first attempt along these lines was reported by Loehlin and Vandenberg (Loehlin & Vandenberg 1968; Vandenberg 1965). They factor analyzed a correlational matrix of identical twin differences for cognitive abilities. The factor analytic results were quite similar to those for the phenotypic correlations among traits for all individuals as well as to those for pair sums which involve shared sources of variance. Loehlin and Nichols (1976) found similar results in analyses of scholastic abilities, personality, and life goals. For all three domains, the pattern of intercorrelations and factor analyses of matrices of pair differences were similar to those for pair sums and for phenotypic correlations. Finally, using observational data for social responding in a small sample of infant twins, Rowe and Plomin (1979) found similar results in factor analyses of twin differences.

If no covariation existed in matrices of identical twin differences, this would suggest that there are no general nonshared environmental effects. Such results would not necessarily imply that there are no systematic nonshared effects – the effects could be systematic but specific in their impact. If a single factor emerged in factor analyses of the differences within pairs of identical twins, this would suggest that a general, monolithic environmental influence might underlie nonshared environment. The results so far are in between these two extremes, consistent with the notion that there are some systematic nonshared environmental influences that are neither very general nor very specific in their effect on behavioral differences between children in the same family. This issue of the dimensionality of nonshared environmental influences deserves further attention.

In a similar vein, **Jensen** suggests that the distribution of squared differences within pairs of identical twins could be examined to explore the extent to which random

microenvironmental effects contribute to nonshared environment. If the distribution of differences were significantly nonnormal, nonrandom effects could be inferred. Loehlin and Nichols (1976) conducted analyses of this type and found no obvious departures from normality. However, as Jensen points out, systematic effects contributing to nonshared environment could also be randomly distributed in the population.

Longitudinal analysis. It would be helpful to add a longitudinal dimension in research on nonshared environmental influences, **Costa & McCrae** note. We fully agree with their specific suggestion that systematic retrospective studies of nonshared environmental influences on adult personality would be a useful first step in a program of research on nonshared environment. There could be no better resource for this work than Costa & McCrae's Baltimore Longitudinal Study of Aging (e.g., McCrae & Costa 1984). Of course, prospective longitudinal studies would also be welcome. **Hay,** for example, suggests that we need to study children soon after birth to see "whether small initial differences become accentuated in a systematic way."

Other comments

The highlight of the commentaries from our perspective was their rich store of ideas about where to look for specific sources of nonshared environment and about the processes that might be involved – over and above the few ideas we mentioned in our target article, which included examining family systems theory, exploring environmental sources of developmental differences *within* individuals, and, most important, studying more than one child per family. Two general issues are mentioned by several commentators – IQ and "credit" – but other comments are for the most part, unique to each commentator, so we address them in alphabetical order. (The fact that the comments are so disparate and nonoverlapping suggests to us that the discussion of nonshared environment has not begun to reach the point of diminishing returns.)

Neurobiology and quantitative genetics. On the first page of our target article, we mention our view that behavioral genetic research is as important for understanding environmental influence as it is for understanding genetic influence. We use schizophrenia research as an example and note that the concordance for first-degree relatives, whose coefficient of genetic relationship is .50, is less than 10%. Identical twins are less than 50% concordant for schizophrenia. These data are compatible with a hypothesis of some genetic influence, but they also indicate that schizophrenia is influenced by nonshared environmental factors. Then we say that, despite this, schizophrenia is coming to be viewed as a genetic disease, which is followed by the sentence: "In the rush to find neural causes of schizophrenia, who is now studying the major source of variability – the environment?" **Hartung** disagrees, suggesting that neurobiologists *are* likely to find the causes of schizophrenia. The major point of our target article, however, is that an important part of the answer to why one individual is diagnosed as schizophrenic and another is not may lie with environmental variation, not biological variation. We believe that interdisciplinary efforts involving behavioral as well as biological researchers are needed to understand problems as complex as schizophrenia.

Quantitative genetics is just as relevant for the brain sciences as for the behavioral sciences. Neurotransmitters and neuromodulators are transcribed directly from DNA; however, the synthesis, storage, receptor sensitivity, and re-uptake that contribute to their levels add complexities that take them far beyond the simple transcription of DNA. We bet that when the story is told, there will be a lot of environmental variation for any complex phenotype such as the level of a neurotransmitter *and* that most of this environmental variation will be of the nonshared variety (Plomin & Deitrich 1982). **Boomsma** also makes the point that nonshared environmental factors are important for physiological as well as for behavioral measures.

Hartung assumes that all phenotypic variation is genetic variation. As discussed earlier, however, nonadditive genetic variance cannot explain all the phenotypic variation if identical twins are reliably different from one another.

It may be easier to assume that all phenotypic variance is genetic than to conduct the appropriate research, but that is not the way of science. **Hartung** uses allergies in one of his examples, assuming again that all the variance is really genetic, but quantitative genetic studies of allergies do not support this assumption. In a study of allergy in 7,000 twin pairs (Edforst-Lubs 1971), the concordance for identical twins for hay fever was only 21%! For fraternal twins, the concordance was 14%. Thus, these twin results suggest only slight genetic influence; moreover, most of the environmental variance must be of the nonshared variety.

An even more decisive example concerns cancer. The geneticist Bodmer has recently concluded that "cancer is essentially a genetic disease at the cellular level, but not a disease with a major inherited component" (Bodmer 1982, p. 1). In other words, cancer involves DNA but genetic (hereditary) influences are slight. For example, a recent twin study found only 6% concordance for identical twins for breast cancer, only 2% concordance for colon cancer, and 2% concordance for rectal cancer (Holm, Hauge & Jensen 1982). The corresponding fraternal twin concordances were 5%, 3%, and 0%.

Our point is that complex phenotypes, including neural as well as behavioral phenotypes, cannot be assumed to be due entirely to genetics.

Traditional environmentalism. Our off-hand remark that the results of traditional environmental studies have been disappointing seems to have caused considerable concern to **Wachs.** The major thrust of our article, after all, is to argue for the importance of environmental influence. Moreover, the purpose of the remark was only to bring out the distinction between environmental factors that operate within families and those that operate between families.

It is not important to our argument that specific measures of the environment bear little relationship to individual differences in development in traditional environmental research. As we indicated in the target article, there is no necessary relationship between the causes of

differences within and between families. In addition, environment–behavior relationships found between families may not be causal: The environmental measure might reflect rather than affect the child, or the environment–behavior relationship might be mediated genetically (Plomin, Loehlin & DeFries 1985).

The rest of **Wachs**'s arguments seem mostly polemical. For example, he indicates that we say siblings in the same family are treated the same and that there is no organism–environment interaction. On the contrary, what we are looking for is any explanation of why children in the same family are so different from one another. The possibilities listed in our article included differential treatment of siblings. Thanks to Wachs (and several other commentators), we now think that interactions of various types should be considered more seriously as possible sources of nonshared environmental influence.

Wachs also states that our conclusion is based on studies of twins which have limited generalizability. As emphasized in the target article, however, the adoption design provides the most impressive evidence for the absence of shared environmental influence (see Sec. 1.1, para. 7). Our review of studies of adoptive siblings indicates little resemblance for personality, psychopathology, and IQ (after childhood).

Wachs has previously written about the need for behavioral geneticists and environmentalists to work together (Wachs 1983), yet in this commentary he suggests that existing models of environmental action can easily encompass predictions derived from the concept of nonshared environmental variance. But the point is not whether environmental theories can be twisted post hoc to accommodate the evidence of the importance of nonshared environment. Where are the references in which environmentalists have written about the concept of nonshared environment? Where is the research that includes more than one child per family?

IQ. Several commentators (**Bock & Zimowski, Eysenck, Jensen, Morton, Rose & Kaprio**) do not like the idea that nonshared environmental influences increase in importance during adolescence for IQ. Bock & Zimowski state that "the nonshared component in IQ measures is a rather small fraction of the total variation." That is the accepted view concerning IQ, a view that we presented fairly in our article. Jensen suggests that it may have been a slip in wording when we said that nonshared environment explains perhaps as much as 40% to 60% of the total variance in IQ. This statement, he says, "is clearly refuted by all of the evidence." The difference is that our statement did not just say "IQ" but rather "IQ (after childhood)."

What is exciting to us is that no one noticed that studies of twins reared together have exaggerated the shared environmental component for IQ and that IQ studies of adoptive siblings have been based on children still living at home. We mentioned Scarr and Weinberg's (1978b) study of postadolescent adoptee pairs, which found a correlation of −.03 for IQ, and we noted another recent study that likewise found no resemblance between adoptive siblings with the average age of 13 years (Kent 1985). These new results suggest that shared environment may be important for IQ during childhood and then fades in importance during adolescence.

We have recently learned of another relevant study of adult adoptive siblings (Teasdale & Owen 1984). Although the sample size is small (24 pairs), the study can be used as an additional test of the hypothesis that shared environment is unimportant for IQ after childhood. This study of adult adoptive siblings yielded an IQ correlation of .03! Clearly, this will not be the last word on the topic; however, it is remarkable that all three recent studies point to the same conclusion. Furthermore, **Loehlin** provides additional support from cross-sectional and longitudinal parent–offspring analyses of the Texas Adoption Project. This issue has been recently reviewed (Plomin, in press).

Sharing the credit for nonshared environment. One indication that nonshared environment must be an important topic is that several commentators seem concerned about who receives credit for it. **Chess** lists four references from 1982 to 1984 and suggests that we should have given greater recognition to "the similar observations made earlier by other investigators." However, we cited the three of these four references that were relevant; moreover, all three of these articles cited an earlier paper (Rowe & Plomin 1981) which was apparently the first article focused on the topic. (The title of the article, published in *Developmental Psychology*, was "The importance of nonshared (E1) environmental influences in behavioral development.")

In our target article, however, we gave credit to Loehlin and Nichols (1976) for first highlighting the importance of nonshared environment in their book, which summarized diverse data for 850 pairs of high-school twins. **Eysenck** notes that he and Eaves published an article in 1975 that noted a similar finding in a twin study on extraversion and neuroticism. Most twin research on personality has found results compatible with the conclusion that environmental variation consists of the nonshared variety, and many include a sentence or two remarking on this. However, to our knowledge, Loehlin and Nichols were the first to emphasize the importance of this finding in other than a sentence or two.

Lamb suggests that psychologists, especially those of a developmental bent, already knew all this. (Who was it that said that the last stage in the acceptance of a new finding occurs when people say "that's obvious" or "we always knew that anyway"? For this reason, Arnold Buss (RP's mentor) used to say that he would like as his epitaph: "He discovered the obvious – first.") Lamb cites no references. If developmental psychologists have known about the importance of nonshared environment, why have their environmental studies included only one child per family?

We issue a direct challenge: Show us some references where developmental psychologists other than behavioral geneticists have addressed the issue of the importance of nonshared environmental influences. It should be noted that other developmentalists do not agree with **Lamb** on this point. **McCartney**, for example, asks "why have developmentalists expected that the shared environment is of critical importance, as evidenced by the vast number of studies on the effects of the home environment?"

We don't mind if credit for the finding is given to people other than Loehlin and Nichols – or to all of

developmental psychology. We would be willing to place a substantial bet that a nineteenth-century scientific reference could be found describing great differences among children in the same family. Biographical descriptions of differences are bound to go back much farther still. Is it possible that Aristotle had nothing to say about this topic?

Specific comments. In this section we attempt to address the remaining specific comments and criticisms.

Bernstein and **Harper** emphasize that behavioral genetics focuses on variation in a population rather than on universals. We agree, and this is why we always try to use phrases like "variability" and "individual differences." If readers are not aware of this distinction, its importance cannot be exaggerated (e.g., Plomin, submitted).

Bock & Zimowski did not like our attempt to avoid the presentation of quantitative genetic theory in algebraic terms. Readers who might prefer an algebraic presentation are urged to consult any quantitative genetics textbook. We wanted to do it differently, and it was our hope that a verbal exposition would appeal to a wider audience. Our rationale for doing this bears rereading (see target article, Sec. 1, para. 3).

Bock & Zimowski (and **Boomsma**) also indicate that the outcomes of studies of group differences (such as moving lower-class children to middle-class environments via adoption) suggest environmental effects. However, such average group differences permit various interpretations, including genetic ones (Jensen 1973), and they bear no necessary relation to the etiology of individual differences (Plomin, submitted).

Boklage says that the problem with our work "is that *environment* is still the error term in quantitative genetic analysis." We thought we had made this point in the target article (Sec. 1.1., para. 4). **Caspari** makes a similar point: The environmental component of variance in quantitative genetic analysis is basically a "remainder" term. **Nyborg** puts it more strongly, calling it "fallacy of negative proof." This is not quite right: For example, the correlation for genetically unrelated children adopted together provides a direct estimate of the total effect of being reared together in the same family. This correlation is as powerful for detecting the influence of shared environment as is the correlation for identical twins adopted apart for detecting genetic influence. One of our main points is that correlations for adoptive siblings consistently point to negligible influence that comes from growing up in the same home with another, genetically unrelated, individual. Moreover, differences within pairs of identical twins directly assess nonshared environmental influence.

Costa & McCrae recommend the simultaneous use of self-reports and ratings in behavioral genetic studies. A similar approach has been used previously (Plomin 1974), with parents rating their twin children. Mothers rated one twin and fathers rated the other; twin cross-correlations for identical and fraternal twins yielded results similar to the usual twin correlations, although, of course, cross-correlations were lower because mothers and fathers only agree about .50 in their ratings of the temperament of their children.

Harper seems to have misread parts of our target article. The misunderstanding about birth-order was mentioned earlier. Among the "inconsistencies," "flaws," and "suspect reasoning" he attributes to us are the following two examples: (1) **Harper** says that we dismiss the possibility that two people who assess a situation similarly could respond to it differently. We have difficulty making sense of this criticism, because all we did say is that two people can perceive the same situation differently. This does not appear to dismiss any possibilities. (2) Harper also says that we rule out the possibility that differential parental treatment might increase behavioral similarities. What we did say was that if parents treat their children similarly then parental treatment is not likely to be a source of differences between the children.

Hay conjectures that both parents and children seek an optimum level of differentiation; in one study in which twins were asked to compare themselves directly to one another, they reported few differences in the extent to which they were liked by family members. It is not clear to us, however, why Hay takes this to be a sign of bias or of support for a theory of "optimum level of differentiation." It is likely that children are contrasted and that labeling occurs – as suggested by Hay's examples of birth-order and birth-weight differences. We do agree in general with the proposition that the study of attributional processes such as those involved in familial contrasts could prove to be a fruitful direction for research on nonshared environmental influences.

Johnson & Nagoshi add an interesting historical perspective to the discussion of nonshared environment. They suggest that nonshared environmental influence may have increased in importance during the past fifty years; they mention research which indicates that the relationship between parental socioeconomic status and children's achievement has declined. There has also apparently been a decline in the heritability of IQ during the past fifty years. Johnson & Nagoshi suggest that both declines may be due to the reduction of social class differences in the U.S. during this century.

Kovach and **Scott** emphasize that quantitative genetics is not sufficiently developmental. We agree, but believe we have tried as hard as anyone to merge behavioral genetics and developmental psychology. We do disagree, however, with Kovach's argument that quantitative genetics and development cannot be brought together. One important interface lies in the genetic analysis of age-to-age change as well as continuity in development, which requires longitudinal data. The concepts of genotype–environment interaction and correlation also attest to the usefulness of bringing the two fields together. These and other points of contact are discussed in a recent book (Plomin 1986).

Loehlin adds new data in support of our major conclusions. We had indicated that one way to study nonshared environment free of genetic bias is to relate experiential differences within pairs of identical twins to behavioral differences within the twin pairs. We mentioned one study (Rowe & Plomin 1981) that found only weak relationships for identical twins between differences in environment and differences in personality. **Loehlin** describes his earlier analyses of this type which yielded similar results (Loehlin & Nichols 1976). One problem with the twin approach is that twins share environmental influences to a greater extent than do nontwin siblings.

For this reason, we decided to try a different approach – using differences within pairs of adoptive siblings. Although their differences could be due to genetic differences as well as to nonshared environment, comparisons with the results for nonadoptive siblings can explore the issue of possible genetic mediation of relationships between sibling differences in experience and their differences in behavior. Our guess was that it would be best to maximize the chances of finding relationships between sibling differences and environmental differences and then to worry about the extent to which such relationships are mediated genetically.

Lykken disagrees with a sentence in our abstract: "These environmental influences make two children in the same family as different from one another as are pairs of children selected randomly." He says that the correlation for siblings is not zero. We agree that siblings resemble each other; our point is that they do so for genetic reasons. The emphasis in the sentence should be on *these* environmental influences. The same confusion occurs in **McCartney**'s opening paragraph.

McArdle & Gottesman argue that until we demonstrate that model-fitting techniques confirm our conclusions, they will be "provocative but inconclusive" and that "the correlation-based conclusions described by P&D are ambiguous at best." They disagree with our statement that "model-fitting procedures only find significant parameters when they are implicit in the basic data."

Although this is an issue that will appear somewhat esoteric to readers outside the field of behavioral genetics, it should be noted that model-fitting techniques represent the state of the art in quantitative genetic analyses. **McArdle & Gottesman** make the pertinent passage (Sec. 3.5, para. 1, target article) sound as if we were against model-fitting. In the interest of making our point to the widest possible audience, we chose to emphasize the basic data rather than more comprehensive models. We are not opposed to model-fitting; in fact, we have published such analyses, especially with our colleagues John DeFries and David Fulker, who have developed several of the most advanced approaches to model-fitting in quantitive genetic analysis (e.g., Fulker & DeFries 1983). **McArdle & Gottesman**'s commentary, however, seems to suggest that theirs is the only way to conduct quantitative genetic analyses. Our view is that behavioral genetic designs are like simple natural experiments and that it is sometimes appropriate to analyze simple data in a simple way. Moreover, data that can be suitably summarized in a simple way are often the strongest data. For example, as we have noted several times, the correlation for adoptive siblings is a direct estimate of the importance of shared environment; if that correlation is near zero – and we show that this seems to be the case in several domains – then shared family environment is not important. To the extent that identical twins differ reliably, nonshared environmental influences must be important. The point was made by Aristotle (see 1959), who noted that it is the mark of the educated man to look for precision in each class of things just so far as the nature of the subject admits.

McCartney suspects that the data about differential parental treatment do not warrant any conclusions because there are too few studies and because it does not make sense that parents would not respond to indiviudal differences in their offspring. All we concluded was that sibling reports, parental self-reports, and observational studies yield no clear conclusion concerning differential parental treatment. We agree that further research on differential parental treatment is called for. McCartney also is concerned that differential experience reflects genetic differences between children in a family. Once relationships are found between experiential differences and behavioral differences in siblings, we will have the luxury of worrying about the direction of effects in interpreting such relationships. If it turns out that the experiential differences merely reflect genetically conditioned behavioral differences between the siblings, then this is not a nonshared environmental influence. At the point that one wishes to assess the influence of genetic differences, behavioral genetic designs are needed. If, for example, the relationship holds up for identical twins, genetics cannot be a factor. In our work with the SIDE using nonadoptive and adoptive siblings, we found little evidence of genetic influence, a point which stands in its favor as a potential measure of nonshared environment.

Morton notes that estimates of components of variance in quantitative genetic models are model-dependent. He and his colleagues have developed sophisticated path analytic approaches that attempt to model several types of shared environmental influence. Perhaps their ideas can be translated to the analysis of nonshared environment.

Nyborg is unhappy about our use of traditional quantitative genetics; he wants to understand individuals rather than individual differences in a population. It is not that, as he suggests, we "get the wrong answer to the right question," but that he wants to ask a different question. It is true that behavioral genetic methods are unable to cope with data for a single individual; quantitative genetic methods are limited to differences among individuals in a population. Nyborg says in several places that quantitative genetics is the study of averages, which is incorrect – it is the study of individuals, whose scores are expressed as deviations from a population average. Finally, it should be noted that Nyborg is hardly offering an approach to understanding a single individual in saying: "I suggest that we start by acknowledging our tremendous biochemical individuality, and then try to probe deep down under the skin of single individuals." We do agree, however, that case studies may be helpful heuristically in the search for nonshared environmental influences.

Rose & Kaprio assert that "data not cited by P&D challenge their position on psychopathology and cognition." Although they do not dispute our conclusion concerning schizophrenia, they suggest that criminality and alcohol abuse show evidence of the influence of family environment. What those data imply, however, is the possibility of some genotype–environment interaction, *not* a direct effect of shared family environment. The point is that the rearing environment made some difference only for sons who were at genetic risk in the sense that their biological father had a criminal record or abused alcohol. Even if Rose & Kaprio had been able to show some direct effect of shared family environment, however, this would not negate the conclusion that most of the environmental variance is of the nonshared variety. Rose & Kaprio add that "the claim that modeling family data

reveals no shared environmental effect on IQ is not correct" but they seem to have misread the sentence in question (Sec. 3.5, para. 1), for we said no such thing. Furthermore, on the basis of a single study of extraversion that gave some evidence for shared environment, they try to deny our conclusions about the importance of nonshared environment for personality when dozens of other studies show no evidence of shared environment for extraversion.

Rose & Kaprio propose that "an incisive test of *shared* environmental effects, again free of genetic bias, relates experiential differences *between* pairs of identical twins to the behavioral differences observed between those pairs." The only data they mention involve an unpublished finding that differences *between* pairs of identical twins in the frequency of their contact with one another correlated with differences *within* pairs in extraversion. In other words, differences within pairs of identical twins were greater when twins spent less time together.

This is not an analysis of shared environmental influences at all, it is an analysis of nonshared environment. **Rose & Kaprio** are asking what causes behavioral differences within pairs of identical twins. Their confusion seems to come about for two reasons. First, small differences within pairs of identical twins can be thought of as similarity or resemblance, but the data are still just differences within pairs which are produced by nonshared (not shared) environment. Second, the environmental variable, the time twins spend together with each other, is difficult to interpret. We think of it as a dyadic measure that summarizes many experiential differences within the twin pairs. Thus, greater environmental differences lead to greater extraversion differences within pairs of identical twins. Rose & Kaprio apparently confuse themselves by turning this around to say that greater environmental similarity leads to greater behavioral resemblance (smaller differences within pairs).

Roubertoux & Nosten make the useful point that heterogeneity needs to be kept in mind: A given trait can have different genetic and environmental influences in different families. They also list some examples from the mouse literature of systematic sources of nonshared environment.

Scarr makes the important point that the environments that behavioral genetic research considers are those in the normal range, not, for example, abusive parents. She also argues that correlating behavioral differences within pairs of siblings with the experiential differences between the siblings may not be a powerful approach for exploring nonshared environment because difference scores are unreliable. Difference scores are unreliable only to the extent that two variables are correlated and, in the case of siblings, we know that the correlations are low. Nonetheless, in order to avoid difference scores and absolute judgments of the environment, another, more successful approach with twins is the use of an instrument like the SIDE to determine whether relative comparisons of twin experiences can explain their behavioral differences, an approach which sidesteps the use of difference scores, at least for the environmental measure.

Sternberg appreciates the importance of nonshared environment in moving beyond components of variance. However, we cannot resist the temptation to respond to his remarks about the "boring" results of behavioral genetics. He asks "what do we really learn about intelligence, for example, or its mechanisms of transmission, when we are told that the heritability of intelligence, is, say, 50%?" In our view, there is no more powerful finding in the behavioral sciences; in contrast, if we bring together everything else that we know about cognitive abilities, we doubt that we would explain 10% of the variance. The result is also important because if heredity had accounted for 100% of the variance, it would mean that extant environmental variation is of no importance in the development of individual differences in cognitive abilities. We cannot, of course, accept his surprising judgment that behavioral genetic results are "pretty predictable on the basis of who collected or compiled the data."

We disagree with **Stevenson** when he says that "a portion of nonshared environmental variance is attributable to differential vulnerability to shared environmental influences." His point is that what might appear to be a shared environmental factor – his example is housing conditions – could be experienced differently by two children in the same family and could thus have different effects on the children. There is a paradox here only if we limit the definition of the environment to objective phenomena, excluding subjective experiences. For example, in our view, if some measurable aspect of housing conditions shows differences between children within a family (as assessed by children's perceptions) then this is a perfectly good measure of nonshared environment. If we cannot measure the salient qualities that differ in the environments of children in the same family then we are not able to identify environmental sources of behavioral differences between children. Nonetheless, we agree with Stevenson's suggestion that the relationship between differential vulnerabilities and nonshared environment should be explored.

Zuckerman does not think much of the suggestions that we made for possible sources of nonshared environmental influence and he notes the difficulties that will be encountered in trying to disentangle cause and effect even if relationships are found between nonshared environment and behavioral differences within a family. He pins his hopes on analyses of what he calls "genotype–environment interaction" but what is really genotype–environment correlation. These topics are discussed earlier in our response. However, we remind readers that if researchers are able to find environmental factors that explain behavioral differences within pairs of identical twins, such relationships cannot be attributed to genotype–environment interaction or correlation, nonadditive genetic variance, error of measurement, or anything else: They are nonshared environmental influences.

Conclusion

As indicated earlier, we were pleased with the outpouring of ideas about possible sources and processes of nonshared environment. There is consensus that children in the same family are very different from one

another. There is also general agreement that most of the environmental variance is of the nonshared variety. So, the question now is, "Why?" Most of the criticisms came from people who did not like our suggestions as to likely sources and processes of nonshared environment. For example, several commentators were bothered by our suggestion that family constellation variables such as gender and birth-order do not appear to be good bets so far. Others weighted our suggested sources of nonshared environmental influence differently; proponents of interaction put the greatest feeling into their disagreement. As we have said several times in our response to the commentaries, we do not wish to squelch interest in any possible source of nonshared environment.

Most exciting were the novel ideas about potentially important nonshared factors and methods of finding them, although no general theory of nonshared environmental influences was proposed. We have, however, learned of an approach to nonshared environment specific to parental behavior that appears to be a step in the right direction. Judy Dunn (1986) suggests that the parental behaviors most likely to be sources of lasting nonshared environmental influence are those that meet three criteria: They are (1) stable over time, (2) differential to siblings observed when the siblings are the same age, and (3) differential to siblings observed at the same time when the siblings are of different ages. Dunn has found that certain parental responses to children (such as irritability) meet only the first criterion: these behaviors tend to correlate with parental characteristics such as personality and demographic background, but not with characteristics of the children. Other parental behaviors such as cuddling are strongly influenced by the age of the child and for this reason meet only the third criterion. Parental behaviors that meet all three criteria are, fortunately, those that relate more to characteristics of children than to parental attributes; Dunn's research suggests that these parental behaviors include affection and conflict. We need such theories to guide research through the labyrinth of possibilities.

We believe that the commentaries will stimulate research that asks why children in the same family are so different from one another. The question is broad; it is likely to lead to different answers for different domains of behavior, for different phases of the life span, and for different individuals. However, the answers that emerge are not only answers to the question, Why are children in the same family so different from one another? Their importance is far more general: understanding the environmental origins of individual differences in human development.

References

Abramovitch, R., Pepler, D. & Corter, C. (1982) Patterns of sibling interaction among preschool-age children. In: *Sibling relationships: Their nature and significance across the lifespan*, ed. M. Lamb & B. Sutton-Smith. Erlbaum. [aRP, TOW]

Ahern, F. M., Johnson, R. C. & Cole, R. E. (1983) Generational differences in spouse similarity in educational attainment. *Behavior Genetics* 13:95–8. [RCJ]

Ahern, F. M., Johnson, R. C., Wilson, J. R., McClearn, G. E. & Vandenberg, S. G. (1982) Family resemblances in personality. *Behavior Genetics* 12:261–80. [aRP]

Alexander, R. A. (1974) The evolution of social behavior. *Annual Review of Ecology and Systematics* 5:325–83. [DMB]

(1979) *Darwinism and human affairs*. University of Washington Press. [DMB]

Annett, M. (1981) The genetics of handedness. *Trends in NeuroSciences* 10:256–58. [JKK]

Aristotle (1959) *Nichomaehean ethics, Book 1*. Springer-Verlag. [rRP]

Bayley, N. (1969) *Manual for the Bayley Scales of infant development*. Psychological Corporation. [aRP]

Beichtman, J. H. (1985) Childhood schizophrenia. A review and comparison with adult-onset schizophrenia. *Psychiatric Clinics of North America* 8:793–814. [JKK]

Bell, R. Q. (1968) A reinterpretation of the direction of effects in socialization. *Psychological Review* 75:81–95. [aRP, SS]

(1979) Parent child and reciprocal influences. *American Psychologist* 34:821–6. [SS]

Benyajati, C. (1984) DNA sequences that are required for the alcohol dehydrogenase gene expression in *Drosophila melanogaster*. In: *Progress in cancer research and therapy*, ed. M. L. Pearson & N. L. Sternberg. Raven Press. [EWC]

Block, J., Block, J. & Morrison, A. (1981) Parental agreement-disagreement on child rearing orientations and gender related personality correlates in children. *Child Development* 52:965–74. [TDW]

Bock, R. D. & Moore, E. J. G. (1986) *Advantage and disadvantage: A profile of American youth*. Erlbaum [RDB]

Bodmer, W. F. (1982) Cancer genetics. *Cancer Surveys* 1:1–15. [rRP]

Bouchard, T. J., Jr. & McGue, M. (1981) Familial studies of intelligence: A review. *Science* 212:1055–59. [aRP, RDB, RJS]

Broman, S. H., Nichols, P. L. & Kennedy, W. A. (1975) *Preschool IQ: Prenatal and early development correlates*. Erlbaum. [rRP]

Bronfenbrenner, U. (1985) *Organism environment interaction from an ecological perspective*. Paper presented at meeting of Society for Research in Child Development, Toronto. [TDW]

Buss, A. H. & Plomin, R. (1975) *A temperament theory of personality development*. Wiley. [SC, MZ]

(1984) *Temperament: Early developing personality traits*. Erlbaum. [aRP, MZ]

Buss, D. M. (1981) Predicting parent–child interactions from children's activity level. *Developmental Psychology* 17:59–65. [KMc]

(1985) Human mate selection. *American Scientist* 73:47–51. [DMB]

(in press) Sex differences in human mate selection criteria: An evolutionary perspective. In: *Sociobiology and psychology: Issues, ideas, and findings*, ed. C. Crawford, D. Krebs & M. Smith. Erlbaum. [DMB]

Buss, D. M. & Barnes, M. (1986) Preferences in human mate selection. *Journal of Personality and Social Psychology* 50:559–70. [DMB]

Cadoret, R. J., O'Gorman, T. W., Troughton, E. & Heywood, E. (1985) Alcoholism and antisocial personality: Interrelationships, genetic and environmental factors. *Archives of General Psychiatry* 42:161–67. [RJR]

Caldwell, B. M. & Bradley, R. H. (1978) *Home observation for measurement of the environment*. University of Arkansas. [aRP]

Carlier, M. & Roubertoux, P. (1985) Motor development of newborn mice: Genetic analysis. In: *Developmental psychology*, ed. C. J. Brainer & V. F. Reyna. North-Holland. [PR]

Carlier, M., Roubertoux, P. & Cohen-Salmon, Ch. (1982) Differences in pup care in *Mus musculus domesticus*: I. Comparison between eleven inbred strains. *Behavioral and Neural Biology* 35:205–10. [PR]

(1983) Early development in mice: I. Genetic and postnatal maternal effects. *Physiology and Behavior* 30:837–44. [PR]

Carroll, L. (1869) *Alice's adventures in wonderland*. Macmillan. [HN]

Carter-Saltzman, L. (1980) Biological and sociocultural effects on handedness: Comparison between biological and adoptive families. *Science* 209:1263–65. [JKK]

Caspari, E. W. (1977) Genetic mechanisms and behavior. In: *Genetics, environment and intelligence*, ed. A. Oliverio. Elsevier/North-Holland. [EWC]

Cattell, R. B. (1953) Research design in psychological genetics, with special reference to the multiple variance method. *American Journal of Human Genetics* 5:76–93. [JMc]

Cattell, R. B., Eber, H. & Tatsuoka, M. M. (1970) *Handbook for the Sixteen Personality Factor questionnaire*. Institute for Personality and Ability Testing. [aRP]

Chess, S. & Thomas, A. (1984) *Origins and evolution of behavior disorders*. Brunner/Mazel. [SC]

Ciba Foundation Symposium 89 (1982) *Temperamental differences in infants and young children*. Pitman. [SC]

Clarke, A. (1984) Early experience and cognitive development. *Review of Research in Education* 11:125–57. [TDW]

Clarke, E. & Hanisse, J. (1982) Intellectual and adaptive performance of Asian

children in adoptive American settings. *Developmental Psychology* 18:595–99. [TDW]

Clarke-Stewart, K. A. & Hevey, C. M. (1981) Longitudinal relations in repeated observations of mother-child interaction from 1 to 2 1/2 years. *Developmental Psychology* 17:127–45. [aRP]

Cloninger, C. R., Bohman, M. & Sigvardsson, S. (1981) Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. *Archives of General Psychiatry* 38:861–68. [RJR]

Cloninger, C. R., Rice, J. & Reich, T. (1979) Multifactorial inheritance with cultural transmission and assortative mating III. Family structure and the analysis of separation experiments. *American Journal of Human Genetics* 31:366–88. [JMc]

Cohen, J. & Cohen, P. (1983) *Applied multiple regression: Correlational analysis for the behavioral sciences.* 2nd ed. Erlbaum. [SS]

Cohen-Salmon, Ch., Carlier, M., Roubertoux, P., Jouhaneau, J., Semal, C. & Paillette, M. (1985) Differences of pup care in mice. V. Pup ultrasonic emissions and pup care behavior. *Physiology and Behavior* 35:167–74. [PR]

Collins, R. L. (1977) *Origins of the sense of asymmetry:* Mendelian and non-Mendelian models of inheritance. *Annals of the New York Academy of Sciences* 299:283–305. [JKK]

Commings, D. F. (1979). Pc1 Duarte, a common polymorphism of a human brain protein and its relationship to depressive disease and multiple sclerosis. *Nature* 277:28–32. [PR]

Costa, P. T., Zonderman, A. B., McCrae, R. R. & Williams, R. B. (1985) Content and comprehensiveness in the MMPI: An item factor analysis in a normal adult sample. *Journal of Personality and Social Psychology* 48:925–33. [RJR]

Cronbach, L. J. & Furby, L. (1970) How we should measure "change" – or should we? *Psychological Bulletin* 74:68–80. [SS]

Crook, M. N. (1937) Intra-family relationships in personality test performance. *Psychological Record* 1:479–502. [aRP]

Daly, M. & Wilson, M. (1982) Whom are newborn babies said to resemble? *Ethology and Sociobiology* 3:69–78. [DMB]

Daniels, D. (1985) Understanding the family environment: A study of adoptive and nonadoptive infant siblings. Unpublished doctoral dissertation, University of Colorado. [aRP, KMC]

(in press) Sibling personality differences and differential experience of siblings in the same family. *Journal of Personality and Social Psychology.* [aRP]

Daniels, D., Dunn, J., Furstenberg, F. F., Jr. & Plomin, R. (1985) Environmental differences within the family and adjustment differences within pairs of adolescent siblings. *Child Development* 56:764–74. [aRP]

Daniels, D. & Plomin, R. (1985) Differential experience of siblings in the same family. *Developmental Psychology* 21:747–60. [arRP, DCR]

Darwin, C. (1892) In: *The autobiography of Charles Darwin and selected letters,* ed. F. Darwin. Dover. [aRP]

DeFries, J. C., Corley, R. P., Johnson, R. C., Vandenberg, S. G. & Wilson, J. R. (1982) Sex-by-generation and ethnic group-by-generation interactions in the Hawaii Family Study of Cognition. *Behavior Genetics* 12:223–30. [RCJ]

DeFries, J. C. & Fulker, D. W. (1985) Multiple regression of twin data. *Behavior Genetics* 15:467–73. [aRP]

(1986) Multivariate behavioral genetics and development: An overview. *Behavior Genetics* 16:1–10. [JMc]

DeFries, J. C., Johnson, R. C., Kuse, A. R., McClearn, G. E., Polovina, J., Vandenberg, S. G. & Wilson, J. R. (1979) Familial resemblance for specific cognitive abilities. *Behavior Genetics* 9:23–43. [RCJ]

De Soete, G. & Carroll, J. D. (1983) A maximum-likelihood method for fitting the wandering vector model. *Psychometrika* 48:553–66. [JMc]

Dibble, E. O. & Cohen, D. J. (1980) The interplay of biological endowment, early experience, and psychosocial influence during the first year of life: An epidemiological twin study. In: *The child in his family,* ed. E. J. Anthony & C. Chiland, vol. 6. Wiley. [DaH]

Dickmann, M. (1979) The reproductive structure of stratified human societies: A preliminary model. In: *Evolutionary biology and human social organization: An anthropological perspective,* ed. N. A. Chagnon & W. G. Irons. Duxbury Press. [DMB]

Dorfman, D. (1978) The Cyril Burt question: New findings. *Science* 201:1177–86. [TDW]

Dunn, J. (1977) Patterns of early interaction: Continuities and consequences. In: *Studies in mother–infant interaction,* ed. H. R. Schaffer. Academic Press. [aRP]

(1983) Sibling relationships in early childhood. *Child Development* 54:787–811. [aRP]

(1986) *Parents and siblings: Developmental implications of changes in relationships within the family.* Developmental Psychobiology Research Group Conference on Social Factors in Early Developmental Psychopathology, Colorado. [rRP]

Dunn, J. F. & Kendrick, C. (1982) *Siblings: Love, envy, and understanding.* Grant McIntyre. [aRP]

Dunn, J. F., Plomin, R. & Daniels, D. (1986) Consistency and change in mothers' behavior towards young siblings. *Child Development* 57:348–56. [aRP]

Dunn, J. F., Plomin, R. & Nettles, M. (1985) Consistency of mothers' behavior towards infant siblings. *Developmental Psychology* 21:1188–95. [aRP, SS, TDW]

Eaves, L. & Eysenck, H. J. (1975) The nature of extraversion: A genetical analysis. *Journal of Personality and Social Psychology* 5:219–21. [HJE]

(1976a) Genetic and environmental components of inconsistency and unrepeatability in twins' responses to a neuroticism questionnaire. *Behavior Genetics* 6:145–60. [HJE]

(1976b) Genotype × age interaction for neuroticism. *Behavior Genetics* 6:359–62. [HJE]

(1977) A genotype-environmental model for psychoticism. *Advances in Behaviour Research and Therapy* 1:5–26. [HJE]

Eaves, L. J., Last, K. A., Young, P. A. & Martin, N. G. (1978) Model-fitting approaches to the analysis of human behavior. *Heredity* 41:249–320. [JMc]

Edforst-Lubs, M-L. (1971) Allergy in 7000 twin pairs. *Acta Allergologica* 26:249–85. [rRP]

Ernst, C. & Angst, J. (1983) *Birth order: Its influence on personality.* Springer-Verlag [aRP]

Eysenck, H. J. (1967) *The biological basis of personality.* Charles C. Thomas. [aRP]

Eysenck, H. J. (1977) *Crime and personality.* Paladin.[HJE]

(1983) A biometrical-genetical analysis of impulsive and sensation seeking behaviour. In: Biological bases and sensation seeking, impulsivity and anxiety, ed. M. Zuckerman. Erlbaum. [HJE]

Eysenck, H. J. & Eysenck, M. W. (1985) *Personality and individual differences: A natural science approach.* Plenum. [MZ]

Eysenck, H. J. & Eysenck, S. B. G. (1964) *Manual of the Eysenck Personality Inventory.* University of London Press & Educational and Industrial Testing Service. [MZ]

Eysenck, H. J. & Eysenck, S. B. G. (1976) *Psychoticism as a dimension of personality.* Hodder & Stoughton. [MZ]

Eysenck, H. J. & Fulker, D. W. (1983) The components of type A behaviour and its genetic determinants. *Personality and Individual Differences* 4:499–505. [HJE]

Eysenck, H. J. & Rachman, S. (1964) *Causes and cures of neurosis.* Routledge and Kegan Paul. [HJE]

Falconer, D. S. (1981) *Introduction to quantitative genetics.* Longman. [aRP]

Farber, S. L. (1981) *Identical twins reared apart: A reanalysis.* Basic Books. [JKK]

Fischer, D. H. (1970) *Historians' fallacies.* Routledge and Kegan Paul. [HN]

Fisher, R. A. (1918) The correlation between relatives on the supposition of Mendelian inheritance. *Transactions of the Royal Society of Edinburgh* 52:399–433. [aRP]

Floderus-Myrhed, B., Pederson, N. & Rasmuson, S. (1980) Assessment of heritability for personality based on a short form of the Eysenck Personality Inventory. *Behavior Genetics* 10:153–62. [aRP, RJR, MZ]

Folstein, S. & Rutter, M. (1977) Genetic influences and infantile autism. *Nature* 265:726–28. [PR]

Franz, S. I. *A handbook of mental examination methods.* Macmillan. [MZ]

Fulker, D. W. (1979) Some implications of biometrical genetical analysis for psychological research. In: *Theoretical advances in behavior genetics,* ed. J. R. Royce & L. P. Mos. Netherlands: Sijthoff & Noordhoff. [DIB]

Fulker, D. W. (1981) The genetic and environmental architecture of psychoticism, extraversion and neuroticism. In: *A model for personality,* ed. H. J. Eysenck. Springer. [HJE, MZ]

Fulker, D. W. & DeFries, J. C. (1983) Genetic and environmental transmission in the Colorado Adoption Project: Path analysis. *British Journal of Mathematics and Statistical Psychology* 36:175–88. [rRP]

Fulker, D. W. & Eysenck, H. J. (1979) Nature and nurture: Environment. In: The structure and measurement of intelligence, ed. H. J. Eysenck. Springer. [HJE]

Fulker, D. W., Eysenck, S. B. G. & Zuckerman, M. (1980) The genetics of sensation seeking. *Journal of Personality Research* 14:261–81. [MZ]

Fuller, J. L. & Thompson, W. R. (1967) *Behavior genetics.* Wiley. [JKK]

(1978) *Foundations of behavior genetics.* Mosby. [aRP]

Furstenberg, F. F., Jr., Winquist-Nord, C., Peterson, J. L. & Zill, N. (1983) The life course of children of divorce. *American Sociological Review* 48:656–68. [aRP]

Galbraith, R. C. (1982) Sibling spacing and intellectual development: A closer

look at the confluence models. *Developmental Psychology* 18:151–73. [aRP]
Goldsmith, H. H. (1983) Genetic influences on personality from infancy to adulthood. *Child Development* 54:331–55. [aRP]
Gottesman, I. I. & Shields, J. (1982) *Schizophrenia: The epigenetic puzzle.* Cambridge University Press. [arRP, DAH]
Gottfried, A. (1984) *Home environment and early cognitive development.* Academic Press. [TDW]
Gottfried, A. E. & Gottfried, A. W. (1984) Home environment and mental development in middle-class children in the first three years. In: *Home environment and early cognitive development: Longitudinal research*, ed. A. E. Gottfried. Academic Press. [RCJ]
Gough, H. G. (1957) *Manual for the California Psychological Inventory.* Consulting Psychologists Press. [MZ]
Granier-Deferre, C., Lecanuet, J. P., Cohen, M. & Busnel, M. C. (1985) Feasability of prenatal hearing test. *Acta-Ato-Laryngology* 421:93–101. [PR]
Gur, R. E., Gur, R. C., Sussman, N. M., O'Connor, M. J. & Vey, M. M. (1984) Hemispheric control of the writing hand: The effect of callosotomy in a left-hander. *Neurology* 34:904–8. [JKK]
Harper, L. V. & Sanders, K. M. (1975) Preschool children's use of space: Sex differences in outdoor play. *Developmental Psychology* 11:119. [LVH]
Hartung, J. (1985) Matrilineal inheritance: New theory and analysis. *Behavioral and Brain Sciences* 8:661–88. [DMB]
Hay, D. A. (1985) *Essentials of behaviour genetics.* Blackwells. [aRP, DAH]
Hay, D. A. & O'Brien, P. J. (1981) The interaction of cognitive abilities in the LaTrobe Twin Study of Behavioural and Biological Development. In: *Twin research 3*, ed. L. Gedda, P. Parisi & W. E. Nance. A. R. Liss. [DAH]
(1984) The role of parental attitudes in the development of temperament in twins at home, school, and in test situations. *Acta Geneticae Medicae et Gemellologiae* 33:191–204. [DAH]
Hay, D. A. & O'Brien, P. J. (submitted) Early influences on the school social adjustment of twins. *Acta Geneticae Medicae et Gemellologiae/Twin Research.* [DAH]
Henderson, N. D. (1982) Human behavior genetics. *Annual Review of Psychology* 33:403–40. [RCJ, TDW]
Hensley, R. (1984) Australian studies with the Family Relations Test – Children's Version I. A revision and validation. *Australian Council for Educational Research Bulletin for Psychologists* 36:16–18. [DAH]
Heston, L. L. (1966) Psychiatric disorders in foster home reared children of schizophrenic mothers. *British Journal of Psychiatry* 112:819–25. [JKK, RJR]
Higgins, J. (1976) Effects of child rearing by schizophrenic mothers: A follow-up. *Journal of Psychiatric Research* 13:1–9. [JKK]
Hirsch, J. & McGuire, T. (1982) *Behavior-genetic analysis.* Hutchinson Ross. [PR]
Holm, N. V., Hauge, M. & Jensen, O. M. (1982) Studies of cancer aetiology in a complete twin population: Breast cancer, colorectal cancer and leukaemia. *Cancer Surveys* 1:17–32. [rRP]
Horn, J. M., Loehlin, J. C., & Willerman, L. (1982) Aspects of the inheritance of intellectual abilities. *Behavior Genetics* 12:479–516. [JCL]
Hunt, J. McV. (1979) Psychological development: Early experience. *Annual Review of Psychology* 30:103–44. [TDW]
Hutchings, B. & Mednick, S. A. (1974) Registered criminality in the adoptive and biological parents of registered male adoptees. In: *Genetics, environment and psychopathology*, ed. S. A. Mednick. F. Schulsinger, J. Higgins & B. Bell. North-Holland. [RJR]
Iselius, L., Morton, N. E. & Rao, D. C. (1983) Family resemblance for blood pressure. *Human Heredity* 33:277–86. [DIB]
Jacklin, C. N. & Maccoby, E. E. (1983) Issues of gender differentiation. In: *Developmental behavioral pediatrics*, ed. M. D. Levine, W. B. Carey, A. C. Crocker & R. T. Gross. Saunders. [rRP]
Jacobs, B. S. & Moss, H. A. (1976) Birth order and sex of sibling as determinants of mother–infant interaction. *Child Development* 47:315–22. [aRP, LVH, TDW]
Jackson, D. N. (1976) *Jackson Personality Inventory manual.* Research Psychologists Press. [PTC]
Jensen, A. R. (1973) Let's understand Skodak and Skeels, finally. *Educational Psychologist* 10:30–35. [rRP]
Jensen, A. R. (1980) *Bias in mental testing.* Basic Books. [KMc]
Jessor, R. (1981) The perceived environment in psychological theory and research. In: *Toward a psychology of situations: An interactional perspective*, ed. D. Magnusson. Erlbaum. [aRP]
Jinks, J. L. & Fulker, D. W. (1970) Comparison of the biometrical genetical, MAVA and classical approaches to the analysis of human behavior. *Psychological Bulletin* 73:311–49. [aRP, DB, MZ]
Johnson, R. C., Ahern, F. M. & Cole, R. E. (1980) Secular change in degree of assortative mating for ability? *Behavior Genetics* 10:1–8. [RCJ]
Johnson, R. C. & Nagoshi, C. T. (1985) Parental ability, education and occupation as influences on offspring cognition in Hawaii and Korea. *Personality and Individual Differences* 6:413–23. [RCJ]
Johnson, R. C., Nagoshi, C. T. & Ahern, F. M. (in press) A reply to Heath et al. on assortative mating for educational level. *Behavior Genetics.* [RCJ]
Johnson, R. C., Nagoshi, C. T., Ahern, F. M., Wilson, J. R., DeFries, J. C., McClearn, G. E. & Vandenberg, S. G. (1983) Family background, cognitive ability, and personality as predictors of educational and occupational attainment. *Social Biology* 30:86–100. [RCJ]
Judd, C. M. & Kenny, D. A. (1981) *Estimating the effects of social interventions.* Cambridge University Press. [SS]
Kagan, J. (1984) *The nature of the child.* Basic Books. [SC]
Kagan, J. & Moss, H. A. (1962) *From birth to maturity.* Wiley. [PTC]
Kaprio, J., Koskenvuo, M., Langinvainio, H., Romanov, K., Sarna, S. & Rose, R. J. (in press). Genetic and social influences on use and abuse of alcohol: A study of Finnish twin brothers. *Proceedings Third Congress of International Society For Biological Research on Alcoholism.* Pergamon Press. [RJR]
Kendler, K. S. (1983) Overview: A current perspective on twin studies of schizophrenia. *American Journal of Psychiatry* 140:1413–25. [JKK]
Kent, J. (1985) Genetic and environmental contributions to cognitive abilities as assessed by a telephone test battery. Unpublished doctoral dissertation, University of Colorado. [arRP, NEM, RR]
Kety, S. S. (1983) Mental illness in the biological and adoptive relatives of schizophrenic adoptees: Findings relevant to genetic and environmental factors in etiology. *American Journal of Psychiatry*, 140:720–727. [JKK]
Kohlberg, L. (1969) Stage and sequences: The cognitive developmental approach to socialization. In: *Handbook of socialization theory and research*, ed. D. A. Goslin. Rand McNally. [MEL]
Konner, M. (1982) *The tangled wing: Biological constraints on the human spirit.* Holt, Rinehart & Winston. [LVH]
Kopp, C. B. & Parmelee, A. H. (1979) Prenatal and perinatal influence on infant behavior. In: *Handbook of infant development*, ed. J. D. Osofsky. Wiley. [rRP]
Koskenvuo, M., Langinvainio, H., Sarna, S., Kaprio, J. & Rose, R. J. (1986) A developmental-genetic analysis of adult personality: Data from the Finnish Twin Cohort. Paper presented at International Congress of Twin Studies, Amsterdam. [RJR]
Kovach, J. K. (1986) Toward the genetics of an engram: The role of heredity in visual preferences and perceptual imprinting. In: *Perspectives in Behavior Genetics*, ed. J. L. Fuller & E. C. Simmel. Erlbaum. [JKK]
Loehlin, J. C. (1978) Heredity-environment analysis of Jencks's IQ correlations. *Behavior Genetics* 8:415–36. [aRP]
Loehlin, J. C. (1979) Combining data from different groups in human behavior genetics. In: *Theoretical advances in behavior genetics*, ed. J. R. Royce & L. P. Mos. Netherlands: Sijthoff & Noordhoff. [JMc]
Loehlin, J. C. (1982) Are personality traits differentially heritable? *Behavior Genetics* 12:417–28. [aRP, KMC, MZ]
Loehlin, J. C., Horn, J. M. & Willerman, L. (1981) Personality resemblance in adoptive families. *Behavior Genetics* 11:309–30. [aRP]
Loehlin, J. C. & Nichols, R. C. (1976) *Heredity, environment, and personality.* University of Texas Press. [arRP, HJE, JCL, MEL, MZ]
Loehlin, J. C. & Vandenberg, S. G. (1968) Genetic and environmental components in the covariation of cognitive abilities: An additive model. In: *Progress in human behavior genetics*, ed. S. G. Vandenberg. Johns Hopkins Press. [rRP]
Loehlin, J. C., Willerman, L. & Horn, J. M. (1985) Personality resemblance in adoptive families when the children are late adolescents and adults. *Journal of Personality and Social Psychology* 48:376–92. [aRP]
Longstreth, L. E., David, B., Carter, L., Flint, D., Owen, J., Rickert, M. & Taylor, E. (1981) Separation of home intellectual environment and maternal IQ as determinants of child IQ. *Developmental Psychology* 7:532–41. [RCJ]
Lush, J. L. (1949) Heritability of quantitative characters in farm animals. *Hereditas, Supplementary Volume*:356–75. [EWC]
Lusis, A. J. & Paigen, K. (1975) Genetic determination of the α-galactosidase development program in mice. *Cell* 6:371–78. [EWC]
Lykken, D. T. (1982) Research with twins: The concept of emergensis. *Psychophysiology* 19:361–73. [aRP, DTL, MZ]
Lykken, D. T., Bouchard, T. J., Jr., Tellegen, A., Wilcox, K., Segal, N. & Rich, S. (1985) Personality similarity in twins reared apart and together. Paper presented at the Second International Conference of the Society for the Study of Individual Differences, St. Feliu, Spain. [MZ]
Lykken, D. T., Tellegen, A. & DeRubeis, R. (1978) Volunteer bias in twin research: The rule of two-thirds. *Social Biology* 25:1–9. [aRP]

Lytton, H. (1977) Do parents create, or respond to, differences in twins? *Developmental Psychology* 13:456–59. [MZ]
(1980) *Parent-child interaction: The socialization process observed in twin and singleton families.* Plenum. [SS, TDW]
Maccoby, E. E. & Jacklin, C. N. (1974) *The psychology of sex differences.* Stanford University Press. [rRP]
Maccoby, E. E. & Martin, J. A. (1983) Socialization in the context of the family: Parent-child interaction. In: *Handbook of child psychology (4th ed.). Socialization personality, and social development*, ed. P. H. Mussen. Wiley. [aRP, SCTDW]
Martin, B. (1981) A longitudinal study of the consequences of early mother-infant interaction: A microanalytic study. *Monographs of the Society for Research in Child Development* 46(3):Serial no. 190. [LVH]
Martin, N. G. & Eaves, L. J. (1977) The genetical analysis of covariance structure. *Heredity* 38:79–95. [DIB]
Martin, N. G. & Jardine, R. (1986) Eysenck's contributions to behaviour genetics. In: *Hans Eysenck: Consensus and controversy*, ed. S. Modgil & C. Modgil. Falmer Press. *[JCL]*
McArdle, J. J. (1986) Latent variable growth within behavior genetic models. Behavior Genetics 16: 163–200. [JMc]
McCall, R. B. (1983) Environmental effects on intelligence: The forgotten realm of discontinuous nonshared within-family factors. *Child Development* 54:408–15. [aRP, SC]
McClearn, G. E. & DeFries, J. C. (1973) *Introduction to behavior genetics.* Freeman. [MZ]
McCrae, R. R. (1982) Consensual validation of personality traits: Evidence from self-reports and ratings. *Journal of Personality and Social Psychology* 43:293–303. [PTC]
McCrae, R. R. & Costa, P. T., Jr. (1984) *Emerging lives, enduring dispositions: Personality in adulthood.* Little, Brown. [rRP, PTC]
(1986) *The use of self-reports and ratings in behavioral genetics.* Unpublished manuscript. [PTC]
McEwen, B. S. (1983) Gonadal steroid influences on brain development and sexual differentiation. In: *International review of psychology, vol. 27*, ed. R. O. Greep. University Park Press. [HN]
McGue, M., Gottesman, I. I. & Rao, D. C. (1983) The transmission of schizophrenia under a multifactorial threshold model. *American Journal of Human Genetics* 36:1161–78. [JMc]
Mednick, S. A. (1966) A longitudinal study of children with a high risk for schizophrenia. *Mental Hygiene* 50:522–35. [MZ]
Mendlewicz, J. (1978) *l'Etiologie génétique des psychoses dysthymiques.* Paris: Masson. [PR]
Michard, C. & Carlier, M. (1985) Les conduites d'agression intraspecifiques chez la souris domestique: Différences individuelles et analyse génétique. *Biology of Behavior* 10:123–46. [PR]
Minuchin, P. (1985) Families and individual development: Provocations from the field of family therapy. *Child Development*, 56:289–302. [aRP]
Mitford, J. (1977) *A fine old conflict.* Michael Joseph. [DIB]
Moore, E. J. G. (1980) *The effects of cultural style on black children's intelligence test achievement.* Doctoral dissertation, University of Chicago. [RDB]
Moos, R. H. & Moos, B. S. (1981) *Family Environment Scale manual.* Consulting Psychologists Press. [aRP]
Nagoshi, C. T. & Johnson, R. C. (1985) Ethnic group-by-generation interactions in WAIS scores in the Hawaii Family Study of Cognition. *Intelligence* 9:259–64. [RCJ]
Namboodiri, K. K., Kaplan, E. B., Heuch, I., Elston, R. C., Green, P. P., Rao, D. C., Laskarzewski, P., Glueck, C. J. & Rifkind, B. M. (1985) The collaborative lipid research clinics family study: Biological and cultural determinants of familial resemblance for plasma lipids and lipoproteins. *Genetic Epidemiology* 2:227–54. [DIB]
Nebert, D. W., Felton, J. S. & Robinson, J. R. (1975) Developmental and genetic aspects of drug and environmental toxicity. *Proceedings of the European Society for Toxicology* 16:82–95. Elsevier. [EWC]
Nebert, D. W., Negishi, M., Lang, M. A., Hjelmeland, L. M. & Eisen, H. J. (1982) The *Ah* locus, a multigene family necessary for survival in a chemically adverse environment: Comparison with the immune system. In: *Advances in genetics*, ed. E. W. Caspari & J. G. Scandalios, vol. 21. Academic Press. [EWC]
Nesselroade, J. R. & McArdle, J. J. (1986) Multivariate causal modeling in alcohol use research. *Social Biology* 32:272–96. [rRP]
Newman, H. H., Freeman, F. N. & Holzinger, K. J. (1937) *Twins: A study of heredity and environment.* University of Chicago Press. [MZ]
Norman-Jackson, J. (1982) Family interaction, language development and primary reading achievement of Black children in families of low income. *Child Development* 53:349–58. [TDW]
Nyborg, H. (1974) *The rod-and-frame test and the field dependent dimension: Some methodological, conceptual, and developmental considerations.* Dansk Psykologisk Forlag. [HN]
(1983) Spatial ability in men and women: Review and new theory. *Advances in human research and therapy, vol. 5.* Pergamon Press. [HN]
Nyborg, H. (1984) Performance and intelligence in hormonally different groups. In: *Sex differences in the brain: The relation between structure and function. Progress in brain research*, ed. G. J. De Vries, J. P. C. DeBruin, H. B. M. Uylings & M. A. Corner, vol. 61. Elsevier. [HN]
(1986) *Sex chromosomes, sex hormones, and developmental disturbances: In search of a model.* Paper presented at Annual Meeting of the American Association for the Advancement of Science, Philadelphia. [HN]
Nyborg, H. & Nielsen, J. (1981) Sex hormone treatment and spatial ability in women with Turner's syndrome. In: *Human behavior and genetics*, ed. W. Schmid & J. Nielsen. Elsevier/North-Holland. [HN]
Oyama, S. (1985) *The ontogeny of information.* Cambridge University Press. [LVH]
Ozer, D. J. (1985) Correlation and the coefficient of determination. *Psychological Bulletin* 97:307–15. [aRP]
Piagen, K. (1971) The genetics of enzyme realization. In: *Enzyme synthesis and degradation in mammalian systems*, ed. M. Recheigl. Karger. [EWC]
Parke, R. D. & Asher, S. R. (1983) Social and personality development. *Annual Review of Psychology* 34:465–509. [PTC]
Parkinson, C., Wallis, S., Prince, J. & Harvey, D. (1982) Rating the home environment of school age children: A comparison with general cognitive index and school progress. *Journal of Child Psychology and Psychiatry* 23:329–33. [TDW]
Pedersen, F., Rubenstein, J. & Yarrow, L. (1979) Infant development in father absent families. *Journal of Genetic Psychology* 135:51–61. [TDW]
Pederson, N. L., Friberg, L., Florderus-Myrhed, B., McClearn, G. E. & Plomin, R. (1984) Swedish early separated twins: Identification and characterization. *Acta Geneticae Medicae et Gemellologiae* 33:243–50. [MZ]
Plomin, R. (1974) *A temperament theory of personality development: Parent-child interactions.* Unpublished doctoral dissertation, University of Texas. [rRP]
(1981a) Ethological behavioral genetics and development. In: *Behavioral development*, ed. K. Immelmann, G. W. Barlow, L. Petrinovich & M. Main. Cambridge University Press. [LVH]
(1981b) Hereditary and temperament: A comparison of twin data for self-report questionnaires, peer ratings, and objectively assessed behaviour. In: *Twin research 3*, ed: L. Gedda, P. Parisi & W. E. Nance. A. R. Liss. [DAH]
(1986) *Development, genetics, and psychology.* Erlbaum. [arRP, RJS, MZ]
(Submitted) *Various man: Group differences and individual differences.* [rRP]
(In press) The nature and nurture of cognitive abilities. In: *Advances in the psychology of human intelligence*, ed. R. Sternberg, vol. 4. Erlbaum. [rRP].
Plomin, R. & Daniels, D. (1984) The interaction between temperament and environment: Methodological considerations. *Merrill-Palmer Quarterly* 30:149–62. [rRP]
Plomin, R. & DeFries, J. C. (1980) Genetics and intelligence: Recent data. *Intelligence* 4:15–24. [RCJ]
Plomin, R., DeFries, J. C. & Loehlin, J. C. (1977) Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin* 84:309–22. [arRP, LVH]
Plomin, R., DeFries, J. C. & McClearn, G. E. (1980) *Behavioral genetics: A primer.* Freeman. [aRP]
Plomin, R. & Deitrich, R. A. (1982) Neuropharmacogenetics and behavioral genetics. *Behavior Genetics* 12:111–21. [rRP]
Plomin, R. & Dunn, J., eds. (1986) *The study of temperament: Changes, continuities and challanges.* Erlbaum. [SC]
Plomin, R. & Foch, T. T. (1980) A twin study of objectively assessed personality in childhood. *Journal of Personality and Social Psychology* 39:680–88. [aRP, LVH]
(1981) Sex differences and individual differences. *Child Development* 52:383–5. [rRP]
Plomin, R., Loehlin, J. C. & DeFries, J. C. (1985) Genetic and environmental components of "environmental" influences. *Developmental Psychology* 21:391–402. [arRP]
Pogue-Geile, M. F. & Rose, R. J. (1985) Developmental genetic studies of adult personality. *Developmental Psychology* 21:547–57. [JCL]
Pryce-Jones, D. (1976) *Unity Mitford: A guest.* Weidenfeld & Nicolson. [DIB]
Rageneau, S. (in press) A simple device to milk mice. *Physiology and Behavior* [PR]
Ramey, C. T., Yeates, K. O. & Short, E. J. (1984) The plasticity of

intellectual development: Insights from preventive intervention. *Child Development* 55:1913–25. [RCJ]

Rao, D. C., Morton, N. E., Gottesman, I. I. & Lew, R. (1981) Path analysis of qualitative data on pairs of relatives: Application to schizophrenia. *Human Heredity* 31:325–33. [NEM]

Rao, D. C., Morton, N. E., Lalouel, J. M. & Lew, R. (1982) Path analysis under generalized assortative mating. II. American IQ. *Genetical Research* 39:187–98. [NEM]

Rose, R. J. (submitted) Genetic and environmental variance in content dimensions of the MMPI. [RJR]

Rosenblatt, J. & Siegel, H. (1981) Factors governing the onset and maintenance of maternal behavior among nonprimate mammals. In: *Parental care in mammals*, ed. D. Gubernick & P. Klopfer. Plenum Press. [PR]

Rosenthal, D. (1970) *Genetic theory and abnormal behavior*. McGraw-Hill. [aRP]

Roubertoux, P. (1981) Apport de l'analyse génétique à l'étiologie des depressions. *Encephale* 49:126–38. [PR]

Rowe, D. C. (1981) Environmental and genetic influences on dimensions of perceived parenting: A twin study. *Developmental Psychology* 17:203–8. [aRP]

(1983) A biometrical analysis of perceptions of family environment: A study of twin and singleton sibling kinships. *Child Development* 54:416–23. [aRP, DCR]

Rowe, D. C. & Osgood, W. (1984) Heredity and sociological theories of delinquency: A reconsideration. *American Sociological Review* 49:526–40. [DCR]

Rowe, D. C. & Plomin, R. (1979) A multivariate twin analysis of within-family environmental influences in infants' social responsiveness. *Behavior Genetics* 9:519–25. [rRP]

(1981) The importance of nonshared (E1) environmental influences in behavioral development. *Developmental Psychology* 17:517–31. [arRP, JMc, DCR]

Rushton, J. P. (1984) Genetic similarity theory. *Behavior Genetics* 14:179–93. [DMB]

Rutter, M. (1983) School effects on pupil progress: Research findings and policy implications. *Child Development* 54:1–29. [TDW]

Sameroff, A. & Chandler, M. (1975) Reproductive risk and the continuum of caretaking casualty. In: *Review of child development research* vol. 4, ed. F. Horowitz. University of Chicago Press. [rRP]

Scarr, S. (1985) Constructing psychology: Making facts and fables for our times. *American Psychologist* 40:499–512. [SS]

(1986a) "A theory of individual encounters with the world." To be published in a conference volume from the Claremont Conference on Applied Social Psychology, February 15, 1986. [SS]

(1986b) "How genotypes and environments combine: Development and individual differences." To be published in a volume based on SRCD workshop on interacting systems in human development, ed: U. Bronfenbrenner, Cambridge University Press. [SS]

Scarr, S. & Carter-Saltzman, L. (1983) Genetics and intelligence. In: *Behavior genetics, principles and applications*, ed. J. L. Fuller & E. C. Simmel. Erlbaum. [DIB]

Scarr, S. & Grajek, S. (1982) Similarities and differences among siblings. In: *Sibling relationships: Their nature and significance across the lifespan*, ed. M. E. Lamb & B. Sutton-Smith. Erlbaum. [aRP, RDB, SC, MEL, RJS]

Scarr, S. & McCartney, K. (1983) How people make their own environments: A theory of genotype → environment effects. *Child Development* 54:424–35. [rRP, KMc, SS]

Scarr, S., Webber, P. I., Weinberg, R. A. & Wittig, M. A. (1981) Personality resemblance among adolescents and their parents in biologically related and adoptive families. *Journal of Personality and Social Psychology* 40:885–98. [aRP, MZ]

Scarr, S. & Weinberg, R. A. (1976) I.Q. test performance of Black children adopted by White families. *American Psychologist* 31:726–39. [RDB, TDW]

(1978a) Attitudes, interests, and IQ. *Human Nature* April:29–36. [aRP, RR, SS]

(1978b) The influence of "family background" on intellectual attainment. *American Sociological Review* 43:674–92. [arRP, KMc, SS]

Schachter, F. F. (1982) Sibling deidentification and split-parent identification: A family tetrad. In: *Sibling relationships: Their nature and significance across the lifespan*, ed. M. E. Lamb & B. Sutton-Smith. Erlbaum. [aRP, DCR]

Schiff, M., Duyme, M., Dumaret, A., Stewart, J., Tomkiewicz, S. & Feingold, J. (1978) Intellectual status of working-class children adopted early into upper-middle-class families. *Science* 200:1503–4. [DIB, TDW]

Schiff, M., Duyme, M., Dumaret, A. & Tomkiewicz, S. (1982) How much would we boost scholastic achievement and I.Q. scores?: A direct answer from a French adoption study. *Cognition* 12:165–96. [RDB]

Schneider, G. E. (1979) Is it really better to have your brain lesion early? A revision of the "Kennard Principle." *Neuropsychologia* 17:557–83. [JKK]

Scott, J. P. (1977) Social genetics. *Behavior Genetics* 1:327–46. [JPS]

Scott, J. P., Bronson, F. & Trattner, A. (1968) Differential human handling and the development of agonistic behavior in basenjis and Shetland sheepdogs. *Developmental Psychobiology* 1:133–40. [JPS]

Scott, J. P. & Fuller, J. L. (1965) *Genetics and the social behavior of the dog*. University of Chicago Press. [JPS]

Seginar, R. (1983) Parent's educational expectations and children's academic achievement: A literature review. *Merrill-Palmer Quarterly* 29:1–23. [TDW]

Shatz, M. & Gelman, R. (1973) The development of communication skills: Modifications in the speech of young children as a function of listener. *Monographs of the Society for Research in Child Development* 38(Serial No. 152). [KMc]

Shaw, D. S. & Bell, R. Q. (submitted) Bidirectional effects in the development of social competence in children. [SS]

Shields, J. (1962) *Monozygotic twins brought up apart and brought up together*. Oxford University Press. [MZ]

Spence, A., Ritvo, E. R., Marazita, M. L., Funderbunk, J., Sparkes, R. S. & Freeman, B. J. (1985) Gene mapping studies with the syndrome of autism. *Behavior Genetics* 15:1–14. [PR]

Spillman, J. R. (1984) The role of birthweight in mother-twin relationships. Unpublished thesis, Cranfield Institute of Technology. [DAH]

Symonds, P. M. (1931) *Diagnosing personality and conduct*. Appleton-Century. [MZ]

Symons, D. (1979) *The evolution of human sexuality*. Oxford University Press. [DMB]

Taylor, D. C. (1975) Factors influencing the occurrence of schizophrenia-like psychosis in patients with temporal lobe epilepsy. *Psychological Medicine* 5:249–54. [JKK]

Teasdale, T. W. & Owen, D. R. (1984) Heredity and familial environment in intelligence and educational level – a sibling study. *Nature* 309:620–22. [rRP]

Tellegen, A. (1985) Structure of mood and personality and their relevance to assessing anxiety, with an emphasis on self-report. In: *Anxiety and the anxiety disorders*, ed. A. H. Tuma, & J. D. Maser. Lawrence Erlbaum Associates. [MZ]

Tellegen, A., Lykken, D. T., Bouchard, T. J., Wilcox, K., Segal, N. & Rich, S. (in press) Personality similarity in twins reared apart and together. Journal of Personality and Social Psychology. [DTL]

Thomas, A. & Chess, S. (1977) *Temperament and development*. Brunner/Mazel. [SC]

Thomas, A., Chess, S. & Birch, H. (1968) *Temperament and behavior disorders in children*. New York University Press. [SC]

Tienari, P., Sorri, A., Lahti, I. Naarala, M., Wahlberg, K.-E., Ronkko, T., Pohjola, J. & Moring, J. (1985) The Finnish adoptive family study of schizophrenia. *The Yale Journal of Biology and Medicine* 58:227–37. [rRP, JKK]

Toran-Allerand, C. D. (1984) On the genesis of sexual differentiation of the central nervous system: Morphogenetic consequences of steroidal exposure and possible role of α-feto protein. In: *Sex differences in the brain: The relation between structure and function. Progress in brain research. vol. 61*, ed: G. J. De Vries, J. P. C. DeBruin, H. B. M. Uylings & M. A. Corner. Elsevier [HN]

Torgersen, A. M. & Kringlen, E. (1978) Genetic aspects of temperamental differences in infants. *Journal of the American Academy of Child Psychiatry* 17:434–44. [SC]

Trivers, R. L. (1972) Parental investment and sexual selection. In: *Sexual selection and the descent of man: 1871–1971*, ed. B. Campbell. Aldine. [DMB]

Trivers, R. L. & Willard, D. (1973) Natural selection of parental ability to vary the sex ratio of offspring. *Science* 179:90–2. [DMB]

Vandenberg, S. G. (1965) Multivariate analysis of twin differences. In: *Methods and goals in human behavior genetics*, ed. S. G. Vandenberg. Academic Press. [rRP]

Vogler, G. P. & Rao, D. C. (1986) Using genetic models to explore environmental sources of familial resemblance for intelligence. Paper presented at Annual Meeting of Behavior Genetics Association, Honolulu. [RJR]

Vom Saal, F. S. & Bronson, H. H. (1980) Sexual characteristics of adult female mice are correlated with their blood testosterone level during prenatal development. *Science* 208:547–99. [PR]

Wachs, T. D. (1979) Proximal experience and early cognitive-intellectual development: The physical environment. *Merrill-Palmer Quarterly* 25:3–41. [TDW]

(1983) The use and abuse of environment in behavior genetic research. *Child Development* 54:396–407. [rRP, TDW]

(1984) Proximal experience and early cognitive intellectual development: The social environment. In: *Home environment in early development*, ed. A. Gottfried. Academic Press. [TDW]

(1985) Models of physical and environmental action. In: *Play interactions: The contribution of play materials and parental involvement to children's development*, ed. A. Gottfried & C. Brown. Lexington Books. [TDW]

Wachs, T. D. & Gandour, M. J. (1983) Temperament, environment and six-month cognitive intellectual development. *International Journal of Behavior Development* 6:135–52. [TDW]

Wachs, T. D. & Gruen, G. (1982) *Early experience and human development*. Plenum. [TDW]

Wassermann, G. D. (1986) Note on an abnormality of the operator for the structural gene of the dopamine D_1 receptor as a possible partial cause of schizophrenia. *Journal of Theoretical Biology* 120:277–83. [JH]

Weizmann, F. (1971) Correlational statistics and the nature-nurture problem. *Science* 171:589. [TDW]

Werner, E. E. & Smith, R. (1982) *Vulnerable but invincible*. McGraw-Hill. [LVH]

White, K. R. (1982) The relation between socioeconomic status and academic achievement. *Psychological Bulletin* 91:461–81. [RCJ]

Wilkins, A. S. (1986) *Genetic analysis of animal development*. Wiley-Interscience. [rRP]

Willerman, L. (1979) Effects of families on intellectual development. *American Psychologist* 34:923–9. [DIB]

Wilson, G. (1978) Introversion/Extroversion. In: *Dimensions of personality*, ed. H. London & J. E. Exner, Jr. Wiley. [DCR]

Wilson, R. S. (1978) Synchronies in mental development: An epigenetic perspective. *Science* 202:939–48. [rRP, JCL]

(1983) The Louisville Twin Study: Developmental synchronies in behavior. *Child Development* 54:298–316.

Woodworth, R. S. (1941) *Heredity and Environment*. Social Science Research Council. [DIB]

Zazzo, R. (1965) *Les jumeaux, le couple, la personne*. Paris: Presses Universitaires de France [PR]

Zuckerman, M., Eysenck, S. B. G. & Eysenck, H. J. (1978) Sensation seeking in England and America: Cross-cultural, age, and sex comparisons. *Journal of Consulting and Clinical Psychology* 46:139–49. [MZ]

Zuckerman, M., Kuhlman, D. M. & Camac, (in press) What lies beyond E and N? Factor analyses of scales believed to measure basic dimensions of personality. *Journal of Personality and Social Psychology*. [MZ]

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