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Nonshared Environment: A Theoretical, Methodological, and Quantitative Review

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When genetic similarity is controlled, siblings often appear no more alike than individuals selected at random from the population. Since R. Plomin and D. Daniels' seminal 1987 review, it has become widely accepted that the source of this dissimilarity is a variance component called nonshared environment. The authors review the conceptual foundations of nonshared environment, with emphasis on distinctions between components of environmental variance and causal properties of environmental events and between the effective and objective aspects of the environment. A statistical model of shared and nonshared environmental variables is developed. A quantitative review shows that measured nonshared environmental variables do not account for a substantial portion of the nonshared variability posited by biometric studies of behavior. Other explanations of the preponderance of nonshared environmental variability are suggested.

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Why Are Children in the Same Family So Different?

In what may have been the most influential article ever written in the field of developmental behavior genetics, Plomin and Daniels (1987) reviewed evidence that a substantial portion of the variability in behavioral outcomes could not be explained by the additive effects of genotype or the environmental influences of families. They suggested that this residual term, which they called the *nonshared environment,* had been neglected by environmentally oriented researchers who assumed that the most important mechanisms of environmental action involved familial variables, like socioeconomic status and parenting styles, that are shared by siblings raised in the same home and serve to make siblings more similar to each other. Indeed, Plomin and Daniels argued, once genetic relatedness has been taken into account, siblings seem to be hardly more similar than children chosen at random from the population.

An important indicator of the influence of Plomin and Daniels' (1987) article is that an entire field of empirical research was generated in an attempt to answer the question posed in its title: Why are children in the same family so different? The content of this research was strongly influenced by Plomin and Daniels, building on earlier theoretical work by Rowe and Plomin (1981), who suggested that the causes of outcome differences among siblings were to be found in differences in the environments they experienced. In a number of related publications, Plomin and colleagues (e.g., Plomin, 1994a, 1994b; Reiss et al., 1994; Rende & Plomin, 1995; hereafter, we attribute the proposal to Plomin) set out a three- or four-step program of empirical research to investigate the origins of nonshared environmental variance. The following is typical:

> Research on nonshared environment can be categorized into (a) analyses of the magnitude of the nonshared environment component of variance, (b) attempts to identify specific nonshared factors that are experienced differently by siblings in a family, and (c) explorations of associations between nonshared factors and behavior. (Rende & Plomin, 1995, p. 308)

It is important to note that the Plomin and Daniels (1987) review consisted of an *observation* of an empirical phenomenon (much of the variability in developmental outcomes is not explained by genotype or shared environment) and a *hypothesis* about the cause of the phenomenon (nongenetic sibling differences are caused by differences in their rearing environments). Once Plomin and Daniels called attention to it, the observation about variance components was uncontroversial. The causal hypothesis required empirical verification, and the purpose of the current article is to review studies that have attempted to provide it.

Nonshared Environment: Objective Versus Effective

When Plomin and colleagues specified the kinds of research that might be conducted under the banner of nonshared environment, they focused exclusively on nonshared events:

> What runs in families is DNA, not experiences shared in the home. However, *environmental factors are very important even though experiences shared by siblings are not* [italics added]. The significant environmental variation lies in experiences *not* [italics in original] shared by siblings. (Plomin & Rende, 1991, p. 180)

> So often we have assumed that the key influences on children's development are shared. . . . *Yet to the extent that these influences are shared by children growing up in the same family, they cannot account for the differences we observed in children's development* [italics added]. . . . The message is not that family experiences are unimportant, but rather that the relevant environmental influences are

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specific to each child, not general to an entire family. (Plomin, 1994a, p. 826)

One direction for research in this area is to identify specific nonshared environmental factors by examining differential experiences of siblings [italics added]. . . . The point of such nonshared environmental analyses is to find those influences that are specific to each child within a family, because such nonshared influences are most important for the development of individual differences for all children, not just for siblings. (Plomin, Manke, & Pike, 1996, pp. 85–86)

Shared factors such as socioeconomic status, child rearing practices, and marital quality were assumed to affect siblings similarly and therefore to have little causal importance:

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* [italics added]. (Plomin & Daniels, 1987, p. 9)

The importance of nonshared environmental factors suggests a reconceptualization of environmental influences that focuses on experiential differences between children in the same family. That is, many environmental factors differ across families; these include socioeconomic status, parental education, and child rearing practices. *However, to the extent that these environmental factors do not differ between children growing up in the same family, they do not influence behavioral development* [italics added]. (Plomin, 1989, p. 109)

It is clear from these quotations that Plomin and his colleagues hypothesize that environmental differences among siblings are the most important cause of nonshared environmental variance in behavioral outcomes. Just as clear, however, is the necessity of distinguishing between objective and effective environments (Goldsmith, 1993). Objective environments refer to environmental events as they might be observed by a researcher, as opposed to how they affect family members. Therefore, the question of whether objective environments are shared or nonshared refers only to whether or not they constitute the environment of more than one sibling in the family, regardless of whether their effects serve to make siblings more alike or more different. Many traditional between-family environmental variables, like socioeconomic status and marital discord, are objectively shared in this sense. Objectively nonshared events are those, like peer relationships and birth order, that constitute the environment of only one sibling, again regardless of whether they work to make siblings alike or different.

Effective environments are defined by the outcomes they produce. The estimate of shared environmental variation that results from biometric studies refers to the effect of environments in creating sibling resemblance, regardless of whether the objective environments were shared or nonshared. Thus, if an objectively shared environmental variable results in nonshared effects, the effective contribution of the objectively shared event is included with the nonshared rather then the shared component of variance.

Consider, for example, parental divorce. Like most other traditional between-family environmental variables, parental divorce is usually an objectively shared event. It is possible, however, that objectively shared events may have different effects on siblings (McCall, 1983; Wachs, 1983). If divorce works to make siblings in the same family different rather than similar, the effective contribution of parental divorce would be nonshared rather than shared (Turkheimer & Gottesman, 1996). However, according to Plomin (1994a), "Assessed in a family-general manner, divorce cannot be a source of differences in siblings' outcomes because it does not differ for two children in the same family" (p. 827).

If the distinction between objective and effective environments is not maintained, Plomin and Daniels' provocative hypothesis that nonshared environmental events are the cause of nonshared environmental variance loses much of its force. If nonshared environment refers to nothing more specific than everything about the environment that ends up making siblings different, Plomin and Daniels' article is reduced to a thorough review of an already well-known phenomenon (Jinks & Fulker, 1970; Loehlin & Nichols, 1976; McCall, 1983; Rowe & Plomin, 1981), that is, a substantial portion of the variability in behavioral outcomes is nonshared. Evidence for Plomin and Daniels' strong empirical hypothesis about nonshared environmental causes of nonshared variance in outcome cannot be found in traditional biometric analyses of twins and families, which demonstrate the importance of nonshared environmental variance but do not establish associations between objectively nonshared events and specific developmental outcomes. To document the importance of objectively nonshared environmental events for nonshared variability in outcome, one must obtain measures of actual environmental variables from multiple siblings (Wachs, 1992). In accordance with Plomin's proposed research program, investigations of environmental influences that make siblings different have focused primarily on events that siblings do not share, including differences in family constellation, differential parenting, and differences in sibling, peer, and teacher relationships. The current article reviews studies of this type.

Methodological Review

Research Designs

Differences models. Several designs have been used to examine the relationship between nonshared environmental events and sibling outcome. One of the earliest and most frequently used is what Plomin et al. (1996) termed a *simple differences model,* in which sibling difference scores are created by subtracting one sibling's score on a measure from the other sibling's on the same measure (Rovine, 1994). Simple differences models involve relating sibling differences on an environmental measure to sibling differences in outcome or to the outcome of a single sibling. For example, associations between sibling differences in maternal treatment and sibling differences in depression or one sibling's depression score might be examined. The relation between difference score correlations and variance accounted for by nonshared environment is actually quite complex, as is demonstrated below.

Difference scores can be computed in several different ways (Rovine, 1994). Relative differences are computed as signed differences between siblings, such as older sibling minus younger sibling. Absolute differences are the absolute value of relative differences and can be computed when no ordering of the siblings is available or desirable. An alternative approach to assessing environmental differences is to ask siblings to rate how different their environments are instead of obtaining environmental scores for each sibling and subtracting. The Sibling Inventory of Differential Experience (SIDE; Daniels & Plomin, 1985) asks siblings to compare their experiences to those of their sibling in the domains

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of parental treatment, sibling interaction, and peer characteristics. Siblings are asked to respond to statements or adjectives describing differences in experience on a 5-point scale indicating both the amount and the direction of difference between themselves and their sibling. As with the simple differences model, relative differences in environment as reported on the SIDE can then be related to a single sibling's outcome or to sibling differences in outcome.

Residualized models. Like simple differences models, residualized models (Plomin et al., 1996) are designed to examine the relation between nonshared environmental events and sibling outcome. Residualized models, however, use linear regression to estimate the relation between an environmental variable describing a child to the same child's outcome, conditional on his or her sibling's score on the environmental variable. In Reiss et al. (1995), for example, regression analyses were used to estimate the relationship between parenting of a child and that child's outcome, controlling for parenting directed toward the child's sibling. Reiss et al. (1995) termed the residualized effect of parenting *specific* in that only variance that is specific to the target child is related to the variance in that child's symptoms. *Cross* effects are regression coefficients that measure the indirect relationship between parenting toward a sibling with outcomes in the target child.

A slightly different approach to residualized scores was adopted by Anderson, Hetherington, Reiss, and Howe (1994). These authors were interested in whether any additional variance is explained when parenting of a sibling is added to a regression model, including parenting of the child in the prediction of the child's outcome. Using a regression model identical to that used by Reiss et al. (1995), Anderson et al. estimated squared semipartial correlations between the environmental measures of the child and the sibling and the outcome measure of the child. The squared semipartial correlations were taken as estimates of the amount of unique variance explained by each predictor: nonshared parenting of the child and parenting shared with the sibling. Once again, relations among standardized regression coefficients, semipartial correlations, and proportions of variance accounted for by shared and nonshared environment turn out to be more complex than has generally been acknowledged. We review statistical approaches to studies of nonshared environment in a subsequent section.

Genetic and longitudinal models. The simple difference design has two major methodological shortcomings. Although the concept of nonshared environment has its origins in developmental behavior genetics, the majority of studies of specific environmental sources of nonshared variance continue to ignore the fact that children are related to their parents through heredity as well as environment. An important reason why children raised in the same family are so different is that, with the exception of monozygotic twins, siblings share no more than half of their genes. Genetic differences between siblings are very easily confounded with environmental differences (Plomin & Bergeman, 1991). The other major shortcoming of the simple difference design involves direction of causal effects. Suppose a study using the simple difference design reports a correlation between differences in maternal negativity directed at siblings and differences between the siblings' acting out behavior. In the absence of other information, it is equally plausible that the negativity is causing the acting out (as hypothesized by the study) or that the acting out is causing the negativity. It is also possible that these influences are bidirectional (Bell & Harper, 1980).

Fortunately, several large studies have used genetically informative, longitudinal designs to examine nonshared environment. The largest of these is the Nonshared Environment and Adolescent Development (NEAD) project (Reiss et al., 1994), a genetically informative, longitudinal study of family and peer influences on the development of competence and psychopathology during adolescence. The NEAD design includes 720 two-parent families with two adolescent siblings no more than 4 years apart in age, ranging from 9 to 18 years at Wave 1 of data collection. The sibling pairs have varying degrees of genetic relatedness—monozygotic (MZ) twins, dizygotic (DZ) twins, and full siblings from nondivorced families, and full, half, and unrelated siblings from "blended" or stepfamilies. Wave 2 data were collected 2 years later.

Three genetically informative studies have been reported from the NEAD project. In the first of these studies, an MZ-difference model was used in which differences in parental negativity were related to differences in the adjustment of identical twins (Pike, Reiss, Hetherington, & Plomin, 1996). The MZ-difference model is similar to the simple differences models in analytic strategy (i.e., differences in environment are related to differences in outcome), but confounds with genetic differences are controlled. That is, because MZ twins are identical genetically, differences in environment cannot be caused by genetic differences, and the association between MZ-twin differences on environmental measures and differences in outcome cannot be mediated by genetic effects. A second report of a genetically informative design from the NEAD (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996) used biometric path models to decompose correlations between parental variables and child outcome into genetic, shared environmental, and nonshared environmental components. Finally, Neiderhiser (1994) used multivariate path models to describe genetically informative and longitudinal aspects of the data.

Another source of genetically informative data about nonshared environment is DF analysis, which is based on a regression methodology developed by DeFries and Fulker (1985). In DF analysis, genetically informative samples of siblings and twins are double entered, and the score of each sibling is regressed on the other in a model including a term for degree of genetic relatedness. Rowe and Waldman (1993) and J. M. Rodgers, Rowe, and Li (1994) have shown how extensions of the basic DF model can be used to estimate contributions of specific nonshared variables to sibling differences in outcome.

Causation and Variance in the Nonshared Environment

Like any other area of developmental research, the goal of studies of nonshared environment is to discover causes of variation in human behavioral phenotypes. Because experimental methods for demonstrating causation in natural families are not always feasible, researchers have used quasi-experimental and correlational designs in the hope of converging on specific causes of nonshared environmental variance. In the simple difference design, which we consider at some length, measures of an environmental variable and a child-outcome variable are simultaneously obtained for each of two siblings in a biological family.

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The statistical methods that are used for analysis of the basic design range from simple examination of correlation matrices through a variety of regression models to latent variable path analysis; what they have in common is that the parameters that are estimated and interpreted are standardized, in the form of correlations and standardized regression or path coefficients. Standardized coefficients are variance components, and the emphasis on variance partitioning in the nonshared environment literature is in keeping with its origins in biometric studies of families (Jinks & Fulker, 1970). But relations among causal properties of biological systems, the unstandardized regression coefficients that can be used to estimate them, and the standardized variance components to which standardized parameters can be transformed have always been a source of controversy (Lewontin, 1974; Turkheimer, 1991), and they are especially problematic here. Studies of the nonshared environment, as illustrated by the example below, require particularly careful attention to the methodological and statistical issues that are raised when standardized coefficients are used to describe the causal properties of developmental systems in nonexperimental contexts.

It is useful to begin discussion of this issue with a concrete biological example. Consider a sample of genetically identical rat pups that happen to display a highly structured relation between their food intake and weight:

$$50 + 3.0(\text{cal}) \qquad (1)$$

Weights and food intake for 20 hypothetical pups are given in Table 1. Unrealistically, no error has been included in the model, to eliminate sampling error and force estimates to reproduce population values exactly. Regressing weight on caloric intake reproduces Equation 1.

Now suppose that the same pups are arbitrarily separated into sibling pairs, and each pair is raised by a mother rat. The pups' food intake, however, remains under the control of the experimenter, and indeed each rat is given precisely the same amount of food as in the previous example. The sibling pairings, shown in the column of Table 1 labeled *Mother 1,* were designed so that pups raised by the same mother receive the same amount of food, that is, the intraclass correlation for siblings is 1.0, and all of the variation in food intake is between mothers rather than within them. We can conduct a repeated measures analysis of variance (ANOVA) on these data, expressing feeding and weight as the sum of a family mean (the *between* component) and each sibling's deviation from the family mean (the *within* component, in this case always 0), resulting in the estimated regression equation,

$$50 + 3.0 \text{ cal(between)} + 3.0 \text{ cal(within)} \qquad (2)$$

The unstandardized regression coefficient describing the relation between differences among maternal feeding means and pup weight is still equal to 3.0, as indeed it must be—they're the same rats. Because all of the variability in feeding is between mothers and weight is completely determined by feeding, the *within* component is 0 for all pups and does not contribute to the mean or variance of pup weight.

In the next column of Table 1, labeled *Mother 2,* the same rats have been reassigned so pairs raised by the same mother are no more similar than pairs of rats chosen at random, and the average amount of food provided to each pair is the same across pairs: All of the variability in feeding is now within pairs, and none of it between. A repeated measures ANOVA still results in Equation 2, but now the *between* component has no variance, being equal to 20 for all pups. The variance explained by the model is now entirely within mothers, that is, nonshared by siblings. Finally, in the column of Table 1 labeled *Mother 3,* the pups have been assigned to mothers so that the variability in food intake is partly between mothers and partly within. This pairing still results in Equation 2, but because there is variability both between and within mothers, the explained variation in weight is equally divided into *between* and *within* portions.

The moral of this story is that variability in behavior can be partitioned into shared and nonshared portions, but causes of behavior cannot (Turkheimer, 1991). The equation specifying rat weight as a function of caloric intake is a biological property of the rats and does not depend on how they happen to be divided into families. Although the proportion of weight variance accounted for by between and within components of feeding behavior differed in the three examples, this variation is not the result of changes in the causal pathway between feeding and weight—there is only one causal pathway between feeding and weight in these rats—but is instead a reflection of changes in the feeding behavior of the mothers.

It would be misleading to use Pairing 1 as a basis for concluding that the relation between food intake and weight appears to be mediated by the shared environment, because to do so would confound the causal effect of food intake on weight, which is neither shared nor nonshared, with the partitioning of feeding variance, which is shared in this case but could easily be otherwise, as demonstrated in Pairings 2 and 3. By the same token, a theory of pup weight that accounted for sibling differences in weight by pointing to within-mother differences in feeding would also have to expect between-mother differences if mothers varied in their feeding behavior. Unless some other causal factor is added to the system, it is not possible for the causal relation between feeding

Table 1
Rat Pup Experiment

Pup	Intake (cal.)	Weight (g)	Mother 1	Mother 2	Mother 3
1	15	95	1	1	1
2	15	95	1	2	1
3	16	98	2	3	3
4	16	98	2	4	4
5	17	101	3	5	5
6	17	101	3	6	5
7	18	104	4	7	7
8	18	104	4	8	8
9	19	107	5	9	9
10	19	107	5	10	9
11	21	113	6	10	10
12	21	113	6	9	10
13	22	116	7	8	8
14	22	116	7	7	7
15	23	119	8	6	6
16	23	119	8	5	6
17	24	122	9	3	4
18	24	122	9	4	3
19	25	125	10	2	2
20	25	125	10	1	2

and weight to exist only within families, if there is also detectable variation in feeding between families.

The relevance of this example to the matter at hand is made plain by changing *feeding* to *maternal negativity,* and *weight* to *externalizing behavior.* From a structural point of view, maternal negativity either causes externalizing behavior or it doesn't. If it does, and if such causal forces are brought to bear within mothers to explain sibling differences in externalizing, and if mothers differ in the average level of negativity they display to their children, then it is difficult to understand why the between-mother variation in negativity doesn't cause between-family variation in externalizing.

This analysis has important consequences for studies attempting to identify causal relations between nonshared environmental events and outcomes in children. The search for such causes began with the observation that most important behavioral outcomes vary almost exclusively within families once genetic variation is taken into account. If one takes this finding seriously (and not everyone does, e.g., Stoolmiller, 1998), then it is difficult to see how parental characteristics that vary both within families and between them might cause the phenomenon. If differences in maternal negativity cause externalizing differences in siblings, and if mothers differ in their average level of negativity, as they certainly do, then why isn't there systematic between-family variation in externalizing?

Several alternative explanations are more plausible than the suggestion that parental behavior somehow exerts its effects on children only within families. It might be that the relevant causal factors do vary only within families and not between them, although it is hard to think what such factors might be, other than birth order. Another possibility is that behavior genetic designs, especially adoption studies, may systematically underestimate shared environmental effects because of restriction of environmental variance in adoptive homes (Stoolmiller, 1998). As mentioned above, there might also be additional causal factors operating within families. The usual candidate is a cross-sibling effect, whereby the environment of one sibling has a causal effect on the other (Reiss et al., 1995). In the next section, we develop path models to explore relations among shared and nonshared variance on the one hand and direct and indirect causal effects on the other.

Models for Studies of Nonshared Environment

Basic model. The research design used in studies of nonshared environmental variables is fairly simple and does not vary greatly among studies: Measures of environment and outcome are obtained from pairs of siblings. Genetically informative samples of family members or longitudinal data collection schemes complicate the analysis but do not alter the basic design. The statistical models that have been used in analyzing these studies are quite various, however, and before embarking on a quantitative review of the studies' results, it is necessary to consider the statistical methods in detail so the studies can be meaningfully compared.

We begin with the straightforward regression model used by Reiss et al. (1995), in which the outcome of each child is regressed on his or her own environmental measure and that of his or her sibling. There are only two causal effects in this model, as illustrated in Figure 1: b_s, the specific (Reiss et al., 1995) effect of environment on a child, and b_c, the cross-effect of the environment

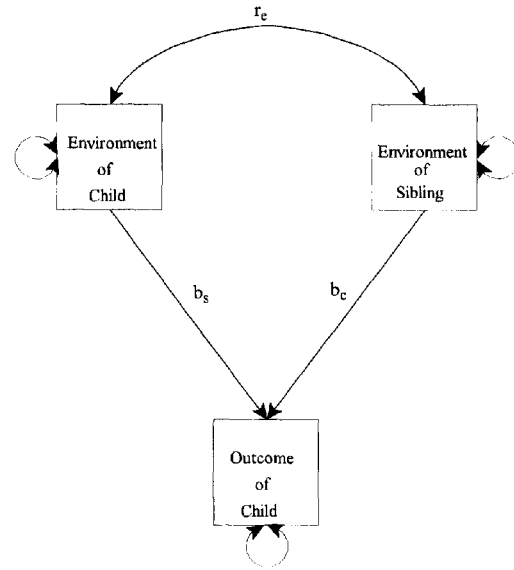

Figure 1. Simple regression model for analysis of sibling differences.

of one sibling on the behavior of the other.[1] Substantive examples of specific and cross-effects can be found in Reiss et al. (1995). The correlation between the environments of the siblings is denoted r_e. Two characteristics of this model are crucial in comparing it to the models that follow: (a) the explicit inclusion of cross-paths, which are parameterized out of some other models, and (b) the structural effects in the model are phenotypic, that is, the causal pathways from environment to outcome originate in the full observed environmental variance, not in the shared or nonshared components of it.

The NEAD study has used the model in Figure 1 to estimate b_s and b_c directly (Reiss et al., 1995). This approach has much to recommend it because the causal effects of environment on child behavior are what the studies were designed to estimate, but it should be noted that the magnitude of these coefficients, as was the case in the rat pups, is independent of whether the environment or its effects are shared or nonshared. The effect of environment on child outcome is just that; inclusion of the sibling's environment in the regression model means that b_s estimates the effect of envi-

[1] Throughout the discussion of path models, the siblings are considered to be equivalent, that is, we do not distinguish between older and younger siblings. In this and subsequent path diagrams, we use standard drawing rules: Observed variables are denoted as rectangles and unobserved (latent) variables are circles. Regression paths are shown by single-headed arrows from the independent to the dependent variable. Correlations and covariances are represented by curved two-headed arrows. Variances of variables are indicated by small two-headed arrows from a variable to itself. All observed variables in the diagrams and accompanying equations are standardized, but latent variables are not. For a complete introduction to path diagrams and tracing rules, the reader is referred to Loehlin (1992).

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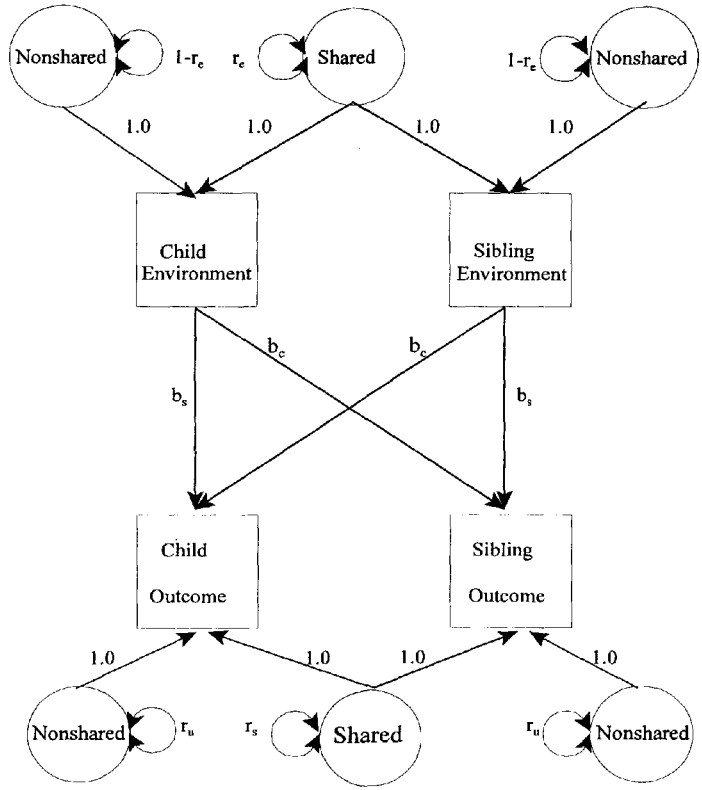

Figure 2. Reparameterized regression model showing contribution of objective shared and nonshared environment to sibling differences.

ronment on a child conditional on the environment of the sibling, but it does not make b_s an estimate of shared or nonshared effects. Specifying the role of shared and nonshared environmental variability in the regression of child outcome on environment requires specific models of the objective and effective environments, as is demonstrated below.

Objective model. In Figure 2, we have reparameterized the model in Figure 1 to clarify the role of objectively shared and nonshared environmental variance in phenotypic causal effects of environment on outcome. The variability of the environmental variables has been partitioned into three parts: a shared portion with variance equal to r_e, and two nonshared portions unique to each sibling, with variances equal to $1 - r_e$. The regression of the outcome of one child on the shared component of environment occurs along two paths, a specific path through the child's phenotype and a cross-path through the sibling's phenotype, for a total regression of $b_s + b_c$. The nonshared components have a specific effect on the sibling to whom they are unique and a cross-effect on the other. The residual variance in outcome has also been partitioned into shared (r_s) and nonshared (r_u) components. Note that the model in Figure 2 is simply a reparameterization of Reiss et al.'s (1995) model in Figure 1, in that it makes precisely the same predictions about the correlations among the siblings' observed environments and outcomes.

The objective environment model in Figure 2 has some counterintuitive properties as a model for nonshared environmental effects. In particular, nonshared variability in the environment of one sibling is allowed to relate to variability in the outcome of the other sibling, which appears to violate the meaning of nonshared environment. The distinction between objective and effective environment clarifies the difficulty. In an objective model, shared and nonshared environment are defined only in terms of variability shared or not shared between the environmental measures, regardless of the effect they may have on outcome. Because the regression paths originate in the observed environmental variability, they apply to objectively nonshared environmental variance just as much as objectively shared variance. So the Reiss et al. (1995) model and our reparameterization of it are models of objective shared and nonshared effects. They answer the question, How much of the variability in outcome is accounted for by objectively nonshared variability in environment?

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The model in Figure 2 makes it clear that cross- and specific-regression coefficients are not the same as the proportions of variance in child outcome attributable to objectively shared and nonshared environment. The proportion of variance in outcome accounted for by shared variance in environment is given by

$$r_e(b_s + b_c)^2, \tag{3}$$

and the proportion of variance in outcome explained by nonshared variation in environment is given by

$$(1 - r_e)(b_s^2 + b_c^2). \tag{4}$$

The latter quantity, which we refer to as *objective* r^2_N, is equal to the proportion of variability in outcome that is explained by objectively nonshared environmental variance.

Effective model. Figure 3 is another parameterization of Figure 1, with shared and nonshared environmental variance represented in terms of the effective environment. Outcome variance has been partitioned into shared and nonshared components. Nonshared variance in outcome is only regressed on nonshared variance in environment, because a path between shared environmental variance and nonshared outcome variance would allow the nonshared outcome components to correlate. Shared variance in outcome can only be regressed on shared environmental variance for the same reason. For convenience, we have expressed the parameters of the model in terms of Figure 1. The effective model answers the question, To what extent does variation in environment contribute to nonshared variability in outcome?

In the effective model, the amount of nonshared outcome variance accounted for by the environment is equal to

$$(1 - r_e)(b_s - b_c)^2, \tag{5}$$

a quantity that we refer to as *effective* r^2_N. Note that objective and effective r^2_N will be equal only when b_c equals zero, that is, when there are no cross-effects between the environment on one sibling and the outcome of the other. When there are cross-effects, it is instructive to consider the relative consequences for objective and effective nonshared environmental effects. Suppose there is a cross-effect of the same magnitude and sign as the specific effect, and substantial objective nonshared environmental variance. Under these conditions, outcomes are influenced by objectively nonshared variance in the environment, but because the nonshared environment of one child has the same effect on the sibling (the cross-effect) as it does on the child (the specific effect), objectively nonshared environment has the effect of making outcomes similar rather than dissimilar.

Cholesky models and difference scores. Several recent studies have used Cholesky decomposition to analyze nonshared environmental effects. Cholesky decomposition is a form of multivariate biometric analysis in which outcome variables in family members are regressed onto biometric variance components in predictors;

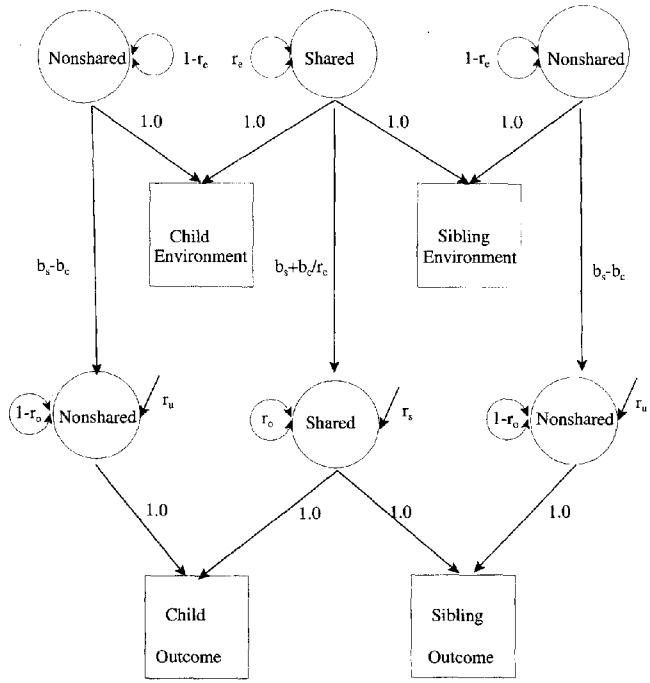

Figure 3. Reparameterized regression model showing contribution of effective shared and nonshared environment to sibling differences.

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residual variance in the outcomes can then be further decomposed (Loehlin, 1996; Neale & Cardon, 1992). Cholesky decompositions are usually used in studies using genetically informative designs, and although the consequences of omitting genetic pathways from such designs is an important topic, we focus on the shared and nonshared environmental components in this article. The effective environmental model in Figure 3 can be rearranged to be equivalent to a Cholesky model (e.g., Pike, McGuire, et al., 1996) by fixing the variances of the shared and nonshared components to unity, rather than fixing the paths to the environmental variance, and collapsing the paths through the shared and nonshared components of outcome. A typical Cholesky parameterization of the model is given in Figure 4, once again with coefficients expressed in terms of Figure 1. Cholesky decompositions are models of the effective environment.

Most studies of specific measures of nonshared environment do not use the regression-based statistical procedures discussed above. Instead, they use difference scores, computing the difference between siblings on an environmental measure and correlating the environmental difference with either the sibling difference on an outcome measure or with the outcome of a single sibling. The relations between correlations among differences and the structural parameters b_s, b_c, and r_e are a little more complex but can also be expressed in terms of Figure 1.

First, consider the correlation r_{DD} between an environmental difference score, $e_1 - e_2$, and an outcome difference score $o_1 - o_2$. When standard formulas for the correlation between difference scores (Tejerina-Allen, Wagner, & Cohen, 1994; derivation in Appendix A) are used, it can be shown that

$$r^2_{DD} = \frac{(1 - r_e)(b_s - b_c)^2}{(1 - r_o)}, \tag{6}$$

in which r_e, b_s, and b_c are defined as in Figure 2 and r_o is the correlation between the children's outcomes. This shows that squared correlations between difference scores estimate a new quantity: the proportion of nonshared variance in outcome that is explained by the environment. Multiplying squared difference correlations by $(1 - r_o)$ will estimate the effective contribution of the environment as described above.

Similarly, the correlation between an environmental difference score and a single sibling's outcome (r^2_{DY}) equals

$$r^2_{DY} = \frac{1 - r_e}{2}(b_s - b_c)^2. \tag{7}$$

This shows that the squared correlation between an environmental difference and a single child's outcome estimates one half of the effective environmental contribution. (It would probably be sensible to orthonormalize the difference by dividing the difference by the square root of two, in which case the squared correlation would estimate the effective contribution directly.)

The differences among the various approaches may be best appreciated by way of a concrete example. Table 2 shows hypothetical scores for 20 pairs of siblings on an environmental measure and an outcome measure. When the data are double entered (to ensure that correlations between environment and outcome will be equivalent in pairs of siblings), the correlation between sibling environments is .51, and the correlation between their outcomes is .39. Regressing the outcome of Sibling 1 on the environments of Sibling 1 and Sibling 2 results in $b_s = .26$, $b_c = .16$. The model accounts for 14% of the variance in outcome, with 5% accounted for by the objectively shared environment and 9% accounted for by the objectively nonshared environment. In contrast, the effective contribution of the nonshared environment is only 0.5% because of the positive cross-effect. The squared correlation between the environmental difference and one child's outcome, r^2_{DY}, is equal to half of the effective nonshared contribution, or 0.25%. The correlation between the environmental difference and the outcome difference, r^2_{DD}, is equal to the proportion of the total

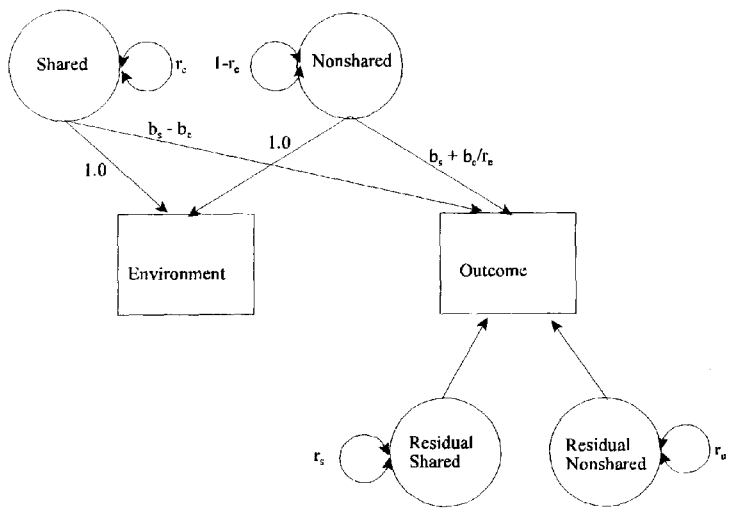

Figure 4. Bivariate Cholesky model parameterized in terms of simple regression model in Figure 1.

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Table 2
Hypothetical Sibling Study

Environment			Outcome		
Sibling 1	Sibling 2	Difference	Sibling 1	Sibling 2	Difference
49	62	−13	38	42	−4
52	61	−9	48	62	−14
51	47	4	27	47	−20
45	42	3	49	44	5
45	41	4	35	48	−13
55	56	−1	30	40	−10
42	42	0	21	44	−23
55	40	15	14	24	−10
30	41	−11	13	25	−12
36	46	−10	48	35	13
59	62	−3	33	39	−6
33	53	−20	15	31	−16
35	53	−18	45	39	6
66	66	0	21	40	−19
41	40	1	20	32	−12
58	49	9	12	30	−18
68	60	8	56	75	−19
67	58	9	47	52	−5
50	56	−6	21	49	−28
44	57	−13	22	47	−25

nonshared outcome variance explained by the environment, or 0.8%. The appropriate conclusion from this study would be that nonshared environment contributes to child outcome, but because of positive cross-paths, the effect of the objectively nonshared environment is to make siblings similar as well as dissimilar. One could also conclude that the measured environmental variable does not appear to be the explanation of nonshared variability in outcome.

Summary

Given the wide variety of statistical procedures that have been used to measure the effects of specific nonshared environmental variables on behavioral outcome, what is the best choice for a standardized measure? Most investigators, we believe, are attempting to estimate effective r^2_N, the nonshared outcome variance accounted for by nonshared variance in the environment. As we have shown, effective r^2_N is a function of the structural parameters b_s and b_c, and the environmental correlation between siblings, r_e. In considering reported values of r^2_N, however, one must bear in mind that they are in large part a function of r_e. More often than not, large values of r^2_N occur for variables with small values for r_e, that is, those for which most of the environmental variability is nonshared.

It is also important to remember that effective and objective r^2_N will not be the same when there are cross-effects. The difference between objective and effective r^2_N will depend on the sign of b_c. Effective r^2_N is a function of $(b_s - b_c)^2$, whereas objective r^2_N is a function of $b_s^2 + b_c^2$. Therefore, when b_c is not equal to zero, objective and effective r^2_N will differ by an amount equal to $-2r_e b_s b_c$. Assuming r_e is positive, when b_s and b_c have the same sign, effective r^2_N will be larger than objective r^2_N; when they have opposite signs, it will be smaller.

Quantitative Review

Literature Search

Criteria for inclusion. A literature search was first conducted to identify all empirical studies examining effects of specific nonshared environmental measures. Reviews of published data were excluded, as were studies that only partitioned genetic and environmental variance without examining specific nonshared variables (e.g., measured differences in maternal negativity), studies providing graphical representation of results only (e.g., Braungart, 1994), studies that included a sibling with a physical disability (e.g., McHale, Crouter, McGuire, & Updegraff, 1995; McHale & Pawletko, 1992), and studies in which sibling relationship quality was used as the only outcome measure (e.g., Brody, Stoneman, & McCoy, 1994; Brody, Stoneman, McCoy, & Forehand, 1992). Results from unpublished dissertations and theses were included.

Search strategy. The literature search entailed two steps. First, a computerized literature search of PsycLIT was conducted with the key words *nonshared, unshared, within-family differences, sibling differences,* and *twin differences.* The time period covered 1967 through November 1997. This search resulted in 271 studies, of which 21 met the criteria for inclusion. The same procedure was conducted for searches using *Dissertation Abstracts,* resulting in nine unpublished dissertations that met inclusion criteria. Second, examination of the reference lists of these articles and theses was completed to identify any references that may have been missed in the computerized literature search, resulting in an additional nine studies. Thus, the total number of studies included in the review is 43. A complete list of studies is provided in Appendix B.

Coding of Studies

For each study, we recorded the total number of sibling pairs, age, gender composition (e.g., same- and opposite-sex dyads), and

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genetic relatedness (e.g., MZ twin, DZ twin, full, half-, and unrelated sibling dyads). Analyses were classified into one of four developmental periods on the basis of the age of the participants: childhood (0–11 years), adolescence (12–18 years), adulthood (>18 years), or some combination. If a study explicitly identified itself as a study of adolescents, it was included as such regardless of the actual age of the participants (e.g., the NEAD study, which included participants from 10–18 years of age). Analyses were also classified as either genetically or not genetically informative on the basis of whether genetic effects were controlled.

The environmental and outcome measures used and the participant providing data (e.g., mothers, fathers, direct observation of siblings) for each measure were also recorded. Environmental measures were categorized into one of six hypothesized nonshared environmental influences (Plomin & Daniels, 1987; Rowe & Plomin, 1981): differential parenting, differential peer relationships, differential sibling interaction, differential teacher relationships, family constellation (e.g., birth order, age, age spacing, gender differences), or some combination of these influences. Outcome measures were categorized into domains reflecting adjustment, personality/temperament, or cognitive ability. Reporters were classified into one of five respondent types: child, parent, teacher, observer, or a combination of reporter types.

The analytic design and statistical method used by each study were recorded. Studies were classified as longitudinal or cross-sectional and genetically informative or nongenetically informative. Statistical methods included simple differences (correlations of sibling environmental differences with either sibling outcome differences or outcome of a single sibling) and residualized (regressions in which the environmental score of one sibling is partialled from the environmental score of the other). Information recorded to code each study is also provided in Appendix B.

Computation of Effect Sizes

We read all studies and extracted statistics describing the relation between specific nonshared variables and sibling outcome. To whatever extent possible, results reported by the studies were transformed to values of effective r^2_N. Computation of effect sizes for each of the statistical methods is described below.

For studies using a simple differences model in which differences on an environmental measure were correlated with the outcome of one sibling only, we used the formula

$$\text{effective } r^2_N = 2r^2_{DY}. \tag{8}$$

For studies using a simple differences model in which sibling differences on an environmental measure are correlated with sibling differences in outcome, we assumed that b_c equaled 0 and inspected the studies to arrive at informed guess of $r_o = .25$, under which conditions

$$\text{effective } r^2_N = .75r^2_{DD}. \tag{9}$$

This equation was used to compute a rough estimate of effective r^2_N.

For the studies using residualized models (models in which an environmental variable for one sibling is related to that sibling's outcome, independent of the other sibling's score on that environmental variable), both specific (b_s) and cross (b_c) effects were recorded and used to compute both objective and effective r^2_N. In Reiss et al. (1995) and O'Connor (1995), b_s, b_c, and r_e were reported directly. In Anderson et al. (1994), b_s and b_c were estimated from reported squared semipartial correlations.

Among studies using a genetically informative design, the simplest (e.g., Pike, Reiss, et al., 1996) correlated MZ twin differences on an environmental measure with MZ twin differences in outcome. All reported correlations were squared to estimate effective r^2_N. Other studies (e.g., Braungart, Fulker, & Plomin, 1992; Pike, McGuire, et al., 1996) reported multivariate genetic analyses, resulting in path models decomposing correlations between environmental and outcome measures into genetic, shared environmental, and nonshared environmental components. For these studies, the reported results were combined with standard path tracing rules to estimate r_e, the correlation between a child's environment and his or her own outcome (r_{11}), and the correlation between a child's environment and his or sibling's outcome (r_{12}). Standard regression formulas were then used to compute b_s and b_c, and Equations 4 and 5 were used to compute objective and effective r^2_N. For DF analyses, regression coefficients corresponding to nonshared effects of specific variables (e.g., J. M. Rodgers, Rowe, & May, 1994) were used as estimates of effective r^2_N.

Meta-Analytic Procedures

We take a descriptive approach to analysis of effect sizes. In particular, we do not include measures of statistical significance in our analyses. We agree with the growing consensus that significance testing is a hindrance to theory development in the behavioral sciences generally (Cohen, 1994; Schmidt, 1996). Our goal in the meta-analysis is to estimate the magnitude of the effect of measured nonshared environmental variables, not to test the uncontroversial null hypothesis that it differs from zero. We have included standard error bars in the graphical presentations of the results so the reader may judge the role played by sampling error in our conclusions.

Most studies included in the review reported more than a single effect size, but in the following tables each study only appears once in a particular category. If a study reported more than one effect size relevant to a particular analysis, the median or mean of all relevant effect sizes from the study is included as a single observation. We conducted analyses by using both unweighted medians and means weighted by the square root of the number of sibling pairs per study.

Results

Effect sizes, r_e and r^2_N, were tabulated according to statistical method, study design, measures of environmental influences and outcome, type of reporter, developmental period, gender composition of the siblings, and publication type (i.e., published chapter or peer-reviewed article vs. unpublished dissertation or thesis). It was also noted whether studies reported results for all measures administered to participants, as opposed to studies only reporting results for measures producing significant results. For the nine studies that only reported significant results, only the reported results were included in our analysis, that is, we did not assign an effect size of zero to the omitted nonsignificant results. We adopted this strategy because it was often difficult to determine

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exactly how many measures had not been reported. As is seen below, a comparison of median effect sizes in studies that report or do not report nonsignificant findings provides an estimate of the magnitude of the consequences of this omission.

The median effect sizes, weighted mean effect sizes, range of effect sizes, and number of studies for which effect sizes were reported or could be estimated are summarized in Tables 3–8. Median effect sizes represent the median across studies of median effect sizes within relevant studies. Mean effect sizes represent the mean across studies (weighted by the square root of the sample size) of mean effect sizes within relevant studies. If a study reported effect sizes relevant to both sides of a contrast (e.g., a study that reported both genetically informative and nongenetically informative data), it was included in both groups, so the total number of studies for some contrasts is greater than 43. Range of effect sizes represents the range of all reported effects.

Regardless of design and method of analysis, publication type, report of nonsignificant results, measures of environmental influences and outcome, reporters on these measures, developmental period, and gender composition of the sibling pairs, estimates of r_N^2 are quite small, with a median value of r_N^2 equal to .016, and a weighted mean equal to .041. The difference between the mean and median effect sizes is an indication of considerable positive skew in the distribution of r_N^2, which has a lower bound of zero (see Figure 5). We now review effect size estimates by the categories described above.

Study Design

When genetic relatedness is controlled, estimates of r_N^2 and r_e decrease substantially (see Table 3). Estimates of r_N^2 for those studies using genetically informative designs (*Mdn* = .010, *M* = .019, *n* = 11) are less than half the size of estimates from studies not controlling for genetic differences between siblings (*Mdn* = .025, *M* = .048, *n* = 33). Box and whisker plots of median effect sizes for genetic and nongenetic studies are provided in Figure 5. When genetic relatedness is taken into account, correlations between siblings' environments are also smaller (*Mdn* = .354, *M* = .340, *n* = 5) than environmental correlations reported for studies not controlling for genetic relatedness (*Mdn* = .523, *M* = .510, *n* = 12).

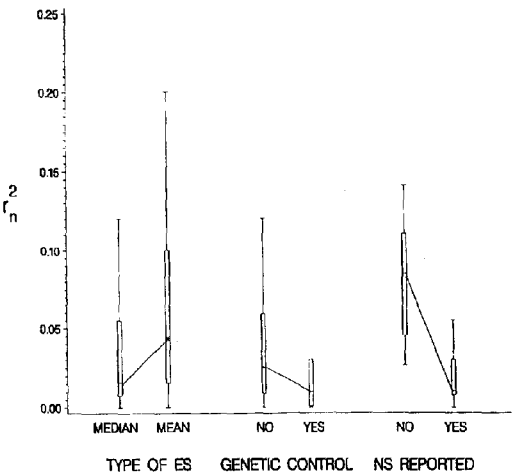

Figure 5. Box and whisker plot of weighted mean and median effect size (ES) for all studies and median effect size for studies employing genetically informative designs (genetic control) and reporting nonsignificant results (NS reported). The boxes extend from the 25th to the 75th percentiles of the distributions; the whiskers extend from the 5th to the 95th percentiles. Lines in boxes are at the median except for the box explicitly labeled *Mean*.

Estimates of r_N^2 for studies performing cross-sectional (*Mdn* = .015, *M* = .039, *n* = 42) analyses are smaller than those from studies performing longitudinal analyses (*Mdn* = .044, *M* = .058, *n* = 8). For studies performing both longitudinal and cross-sectional analyses (*n* = 7), however, estimates of r_N^2 are larger for cross-sectional (*Mdn* = .052, *M* = .051) than longitudinal analyses (*Mdn* = .032, *M* = .047). Also, several of the longitudinal analyses were based on correlations between environmental differences measured at Time 1 and outcome differences at Time 2 without controlling for differences in environment at Time 2. Estimates of r_N^2 for studies that did control for environmental differences at Time 2 (*Mdn* = .024, *M* = .032, *n* = 4) were less than half the size of estimates from studies that did not (*Mdn* = .076, *M* = .070, *n* = 5).

Statistical Methods

Table 4 presents effect sizes broken down by the statistical method used. Separate estimates of objective and effective r_N^2 are presented for studies using residualized statistical methods so both could be estimated. Estimates of effective and objective r_N^2 are roughly similar as would be expected given that b_c tends to be quite small and of negative sign (b_c ranges from −.69 to .33, *Mdn* = −.005, *M* = −.024). Estimates of r_N^2 are smaller for studies using residualized methods than for studies using simple differences models. Environmental correlations are also smaller for residualized (*Mdn* = .380, *M* = .429, *n* = 7) versus simple differences models (*Mdn* = .518, *M* = .496, *n* = 10).

Publication Type and Report of Nonsignificant Results

Estimates of r_N^2 are smaller for published papers, including edited chapters and peer-reviewed articles (*Mdn* = .015, *M* = .039, *n* = 33), than estimates from unpublished dissertations and theses (*Mdn* = .022, *M* = .048, *n* = 10) (see Table 5). Differences in estimates of r_N^2 also appear between studies reporting and not reporting nonsignificant results. Median estimates of r_N^2 for studies reporting significant results only (*Mdn* = .084, *n* = 9) are over eight times as large as estimates from studies including both significant and nonsignificant results (*Mdn* = .010, *n* = 34). Mean estimates of r_N^2 for studies reporting significant results only (*M* = .080) are over twice as large as estimates from studies including both significant and nonsignificant results (*M* = .035). Median effect sizes for studies reporting and not reporting nonsignificant results are presented graphically in Figure 5.

Measures of Environmental Influences and Outcome

Estimates of r_N^2 also vary depending on the measure of environmental influence and outcome examined (see Table 6). Estimates

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Table 3
Effect Sizes by Study Design

Study design	Effective r_N^2				r_e			
	Mdn	Weighted *M*	Range	*n*	*Mdn*	Weighted *M*	Range	*n*
Genetic vs. nongenetic								
Genetic	.010	.019	0.0–.605	11	.354	.340	.05–.83	5
Nongenetic	.025	.048	0.0–1.00	33	.523	.510	−.08–.96	12
Longitudinal vs. cross-sectional								
Longitudinal	.044	.058	0.0–1.00	8	.655	.440	.05–.78	3
Cross-sectional	.015	.039	0.0–1.00	42	.430	.460	−.08–.96	17

of r_N^2 are largest for studies examining multiple measures of differential environment (*Mdn* = .133, *M* = .109, *n* = 2) and for studies examining differential peer and teacher interaction (*Mdn* = .053, *M* = .091, *n* = 8). For the two studies examining multiple measures of differential environment, however, both perform multiple regression analyses in which only statistically significant predictors are included. Estimates of r_N^2 are smallest for studies examining family constellation variables such as differences in birth order and age spacing (*Mdn* = .011, *M* = .010, *n* = 4). Figure 6 provides box and whisker plots of median effect sizes for each type of environmental measure.

For outcome measures, median effect-size estimates of r_N^2 for adjustment (*Mdn* = .016, *M* = .046, *n* = 30), personality and temperament (*Mdn* = .015, *M* = .049, *n* = 13), and cognitive ability (*Mdn* = .015, *M* = .016, *n* = 5) are roughly equal. Weighted mean estimates of r_N^2 for studies examining cognitive ability are, however, much smaller than estimates from studies examining either adjustment or personality or temperament.

Reporters

For environmental measures (see Table 7 and Figure 7), estimates of r_N^2 are largest for observational (*Mdn* = .034, *M* = .055, *n* = 10) and child reports (*Mdn* = .026, *M* = .049, *n* = 19) and smallest for father report (*Mdn* = .012, *M* = .032, *n* = 16). Environmental correlations are also much smaller for child reports of differential environment (*Mdn* = .245, *M* = .343, *n* = 4) compared with other reporter types (median r_e ranges from .380 to .630, and weighted mean r_e ranges from .419 to .656). For outcome measures, estimates of r_N^2 are largest for mother (*Mdn* = .038, *M* = .079, *n* = 11) and parent reports (*Mdn* = .031, *M* = .066, *n* = 13) and smallest for child report (*Mdn* = .013, *M* = .027, *n* = 23).

Developmental Period and Gender Composition

For developmental period (see Table 8), estimates of r_N^2 are greater when measured during childhood (*Mdn* = .031, *M* = .052, *n* = 15) and adulthood (*Mdn* = .035, *M* = .063, *n* = 6) than during adolescence (*Mdn* = .011, *M* = .030, *n* = 19) or when a combination of development periods or ages is examined (*Mdn* = .009, *M* = .066, *n* = 3). Environmental correlations are much smaller when measured during adulthood (*Mdn* = .280, *M* = .250, *n* = 4) than during other periods. Median estimates of r_N^2 do not appear to vary much by whether studies examined same- or mixed-sex sibling pairs. Mean estimates of r_N^2 are smallest for mixed-sex (*M* = .032, *n* = 21) as opposed to same-sex pairs (*M* = .048, *n* = 19).

Conclusion: The Gloomy Prospect?

This article has provided a theoretical, quantitative, and meta-analytic review of studies of specific sources of nonshared environmental influences on child development. We begin our discussion with a review of our major conclusions.

1. It is important to maintain a distinction between the objective and effective aspects of nonshared environment. Biometric family studies, as reviewed by Plomin and Daniels (1987), have shown that a substantial portion of the variability in child outcomes can be

Table 4
Effect Sizes by Statistical Method

Statistical method	Effective r_N^2			Objective r_N^2[a]		
	Mdn	Weighted *M*	Range	*Mdn*	Weighted *M*	Range
Residualized (*n* = 7)						
r_N^2	.015	.049	−.021–.315	.015	.036	.0–.276
r_e	.380	.429	−.08–.96	.380	.429	−.08–.96
b_s	.185	.198	−.40–.91	.185	.198	−.40–.91
b_c	−.005	−.024	−.69–.33	−.005	−.024	−.69–.33
Simple differences (*n* = 36)						
r_N^2	.025	.042	0.0–1.00	—	—	—
r_e	.518	.496	.03–.78	—	—	—

[a] Values for r_e, b_s, and b_c are the same for effective and objective r_N^2 because they are computed from the same effect sizes.

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Table 5
Effect Sizes by Publication Type and Report of Nonsignificant Results

Publication type and report of nonsignificant results	Effective r_N^2				r_e			
	Mdn	Weighted *M*	Range	*n*	*Mdn*	Weighted *M*	Range	*n*
Publication type								
Published papers	.015	.039	0.0–1.00	33	.518	.507	.03–.96	14
Unpublished dissertations and theses	.022	.048	0.0–.605	10	.375	.245	−.08–.67	3
Nonsignificant results								
Reported	.010	.035	0.0–1.00	34	.430	.458	−.08–.96	15
Not reported	.084	.080	0.0–.627	9	.488	.477	.19–.72	2

attributed to a component called nonshared environment. In terms of the effective environment, this conclusion is uncontroversial: something about the environment must be causing differences among genetically related siblings reared together. In terms of the objective environment, however, the outcome is not so clear: Plomin and Daniels (1987) conjectured that nonshared environmental variance in child outcome was caused by objectively nonshared environmental events. Empirical tests of this conjecture were the subject of our quantitative review.

2. Another important theoretical distinction needs to be maintained between environmental causes of behavior, which are neither shared nor nonshared, and environmental variance in behavior, which can be partitioned into shared and nonshared components. For a particular environmental event to be a substantial cause of nonshared variation in outcome, three conditions must be met: The environmental event must be a significant cause of behavioral outcomes, and variability for the environmental event must be substantially nonshared among siblings. These two considerations are fundamentally independent of each other. In addition, there must not be cross-effects of equal sign and magnitude to the specific effects because under these circumstances the specific effects and cross-effects will negate each other.

3. Methods of statistical analysis for studies of nonshared environment can be parameterized in terms of three quantities: the cross- and specific-effects of environment on behavior, and the magnitude of the correlation between siblings' environments. The objective and effective environmental contributions to outcome can be computed from these quantities and will only be the same when there are no cross-effects between the environment of one child and the outcome of the other. The major statistical procedure that has emerged for the analysis of measured environmental sources of nonshared variability in outcomes, involving correlations between environmental and child outcome difference scores, provides a rescaled estimate of the effective contribution of the environmental measure.

4. The commonplace practice of ignoring genetic effects in studies of nonshared environment cannot be justified. When genetic effects are included, as in the NEAD project, they are usually the most important terms in the model by a significant margin.

5. Quantitative analysis of studies of specific nonshared environmental events shows that effect sizes measuring the effects of such variables on child outcomes are generally very small. Effect sizes are largest when confounds with genetic variability and outcome-to-environment causal effects are not controlled. When such confounds are controlled, as in the most recent reports from the NEAD project, effect sizes become smaller still. The largest effect sizes are found when researchers rely on direct observation of environment rather than indirect reports from others (Wachs, 1983). Measures of nonfamilial sources of nonshared environment (e.g., peers and teachers) produce larger effect sizes than sources

Table 6
Effect Sizes by Environmental and Outcome Measures

Measure	Effective r_N^2				r_e			
	Mdn	Weighted *M*	Range	*n*	*Mdn*	Weighted *M*	Range	*n*
Environmental measure								
Family constellation	.011	.010	0.0–.106	4	—	—	—	—
Differential parental behavior	.023	.045	0.0–1.00	41	.430	.442	−.08–.96	17
Maternal	.023	.051	0.0–1.00	32	.465	.414	−.04–.86	14
Paternal	.016	.041	0.0–.406	17	.510	.471	−.08–.96	7
Differential sibling interaction	.024	.043	0.0–.300	9	.810	.810	.79–.83	1
Differential peer and/or teacher interaction	.053	.091	0.0–.605	8	—	—	—	—
Aggregate of environmental measures	.133	.109	0.0–.248	2	—	—	—	—
Outcome measure								
Adjustment	.016	.046	0.0–1.00	30	.520	.479	−.08–.96	13
Personality/temperament	.015	.049	0.0–.620	13	.378	.402	.18–.71	4
Cognitive ability	.015	.016	0.0–.605	5	.375	.375	.31–.44	1

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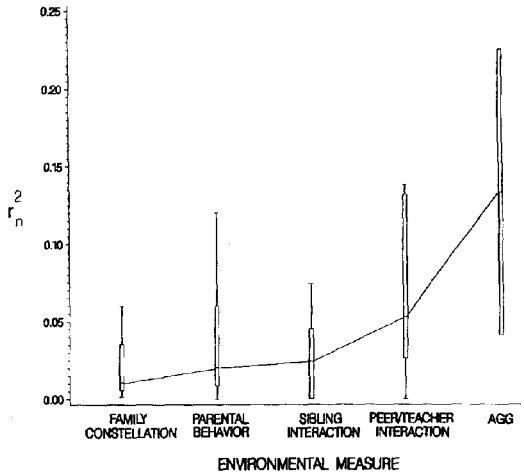

Figure 6. Median effect size by environmental measure. AGG indicates aggregates of more than one measure. (See Figure 5 caption for explanation of box and whisker plots.)

of nonshared environment originating in the family (Harris, 1995). Not surprisingly, studies reporting nonsignificant results had much smaller median effect sizes than studies that listed only significant findings. More realistic estimates of the magnitude of nonshared environmental effects will be obtained if future studies report all effect sizes that are estimated.

We emphasize that these findings should not lead the reader to conclude that the nonshared environment is not as important as had been thought. Rather, we believe that the appropriate conclusion is that the causal mechanisms underlying nonshared environmental variability in outcome remain unknown. The first candidate to receive serious consideration—objectively nonshared environmental events—does not appear likely to provide such an explanation, but it is important to remember that there are numerous other possibilities.

One possibility discussed by Plomin and Daniels (1987) is that objectively nonshared environmental events are indeed the source of nonshared variability in outcome, but the causal impact of any single environmental event is very small and unsystematic; it is only the cumulative effect of a multitude of small environmental differences that cause detectable outcome differences among siblings. Plomin and Daniels (1987) dismiss this "gloomy prospect" (p. 8) because its methodological consequences appear so dismal, but as several commentators pointed out at the time (Chess, 1987; Hartung, 1987; Kovach, 1987; McCartney, 1987), the fact that a conclusion makes life more difficult for social scientists does not make it untrue.

If the effects of environmental events depend on the genotype of the affected individual, even objectively shared environmental events will have differential effects on genetically nonidentical siblings (McCall, 1983; Wachs, 1983). Like multiplicative genetic effects, on theoretical grounds gene by environment (G × E) interactions would appear to be an important source of developmental differences between siblings, but methodological complexities render them very difficult to detect in actual data. The classic treatment of these difficulties, focusing on shortages of statistical power for the detection of interaction in linear models, is Wahlsten (1990). Even more troubling is Molenaar, Boomsma, and Dolan's (1997) article in which they demonstrated that if G × E interactive processes are misspecified as genetic and environmental main effects, the misspecification is only detectable in the fourth moments of the resulting distributions, and even that effect averages out as effects are accumulated over the course of development.

Although such interactions have always been conceptualized as G × E, we have argued that they may be more accurately characterized as phenotype by environment (P × E) interactions (Turkheimer, 1999). The idea of P × E interactions does not make sense in strictly cross-sectional models because it would involve an interaction between a dependent (P) and an independent (E) variable, but in developmental models it makes perfect sense to postulate that the effect of an environmental event depends on the

Table 7
Effect Sizes by Reporters on Measures

Measure	Effective r_N^2				r_e			
	Mdn	Weighted *M*	Range	*n*	*Mdn*	Weighted *M*	Range	*n*
Environmental measure								
Observation	.034	.055	0.0–.627	10	.534	.545	.19–.78	4
Parent	.017	.034	0.0–1.00	20	.585	.541	.03–.75	6
Mother	.020	.036	0.0–1.00	19	.615	.582	.03–.75	6
Father	.012	.032	0.0–.406	6	.630	.656	.29–.75	2
Child	.026	.044	0.0–.620	19	.245	.343	.17–.75	4
Aggregate of reporters	.016	.035	0.0–.480	12	.380	.419	−.08–.96	8
Outcome measure								
Observation	.015	.049	0.0–.627	7	.378	.408	.23–.72	4
Parent	.031	.066	0.0–.480	13	.563	.506	.03–.72	4
Mother	.038	.079	0.0–.480	11	.605	.398	.03–.72	3
Father	.021	.048	0.0–.219	2	—	—	—	—
Child	.013	.027	0.0–.620	23	.430	.475	.17–.78	5
Teacher	.025	.054	0.0–1.00	4	.605	.586	.32–.71	1
Aggregate of reporters	.018	.047	0.0–.440	9	.367	.421	−.08–.96	6

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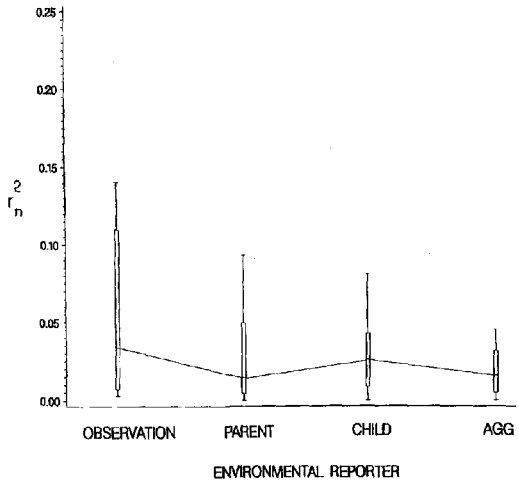

Figure 7. Median effect size by environmental reporter. AGG indicates aggregate of more than one reporter. (See Figure 5 caption for explanation of box and whisker plots.)

phenotype of the organism at the time the event occurs; indeed, this model appears much more plausible than the idea that environmental effects are somehow mediated directly by the genotype. If intelligent children evoke more complex linguistic interactions with their caregivers, it is observable phenotypic aspects of their behavior, not their genotype, that is having an effect on surrounding adults. This phenotype is in turn the cumulative result of developmental interactions between the child's genotype and previous environmental events.

Turkheimer and Gottesman (1996) used computer simulations to show that models of this kind can avoid the exclusively genetic conclusions that are sometimes reached by theorists of G × E interaction (Scarr, 1992; Scarr & McCartney, 1983). Models with P × E interaction contain complex reciprocal effects between phenotype and environment: Phenotype affects the organism's choice of environments, environments cause changes in phenotypes, and the nature of environmental effects on future phenotypes are moderated by the current phenotype. Interestingly, the simulations suggest that in models with these kinds of interactions, linear effects of genotype are much easier to detect than linear effects of environment: The environment is all interaction and little main effect.

Other sources of sibling differences originate in the genome. Multiplicative effects among several genes contributing to variability in a trait, called epistasis in classical genetics and more recently termed *emergenesis* (Lykken, 1982; Lykken, McGue, Tellegen, & Bouchard, 1992), will make only identical twins more similar. Siblings, who will not in general share the entire complex of relevant genes, will be made dissimilar. Although nonadditive genetic effects are notoriously difficult to detect for complex human phenotypes for which errors of measurement at the phenotypic level are typically larger than the differences between additive and multiplicative models (Eaves, 1983), the commonplace finding that MZ twins are more than twice as similar as DZ twins provides general evidence that additive models of genetic effects are not sufficient.

Stochastic developmental processes at the cellular level produce differences even between identical twins. Kumit, Layton, and Matthysse (1987) used computer simulations to show that small amounts of randomness in early developmental processes can lead to formally unpredictable nonlinear processes. Molenaar, Boomsma, and Dolan (1993) have proposed that the cumulative effects of nonlinear developmental processes constitute a "third source" of developmental differences and offered preliminary suggestions about how epigenetic process could be included in more traditional biometric designs. Molenaar et al. (1993) emphasized that variability arising in epigenetic processes will be confounded with nonshared environmental variance if it is not specifically taken into account, concluding:

> In our opinion, an important reason why the sources of [nonshared environmental] influences are still unknown is because a significant part of nonshared environmental influences may not be due to environmental differences at all, but result from intrinsic variability in the output of deterministic, self-organizing developmental processes. (p. 523)

An interesting concept in developmental genetics that deserves greater consideration in the realm of behavior is developmental

Table 8
Effect Sizes by Developmental Period of Siblings Studied and Gender Composition of Sibling Pairs

Variable	Effective r_N^2				r_e			
	Mdn	Weighted *M*	Range	*n*	*Mdn*	Weighted *M*	Range	*n*
Developmental period								
Childhood	.031	.052	0.0–1.00	15	.520	.463	.03–.72	7
Adolescence	.011	.030	0.0–.605	19	.430	.465	−.08–.96	9
Adulthood	.035	.063	0.0–.620	6	.280	.250	.18–.29	1
Combination of developmental periods	.009	.066	0.0–.248	3	—	—	—	—
Gender composition of sibling pairs								
Same sex	.017	.048	0.0–.627	19	.448	.456	−.08–.96	8
Mixed sex	.015	.032	0.0–1.00	21	.520	.511	.03–.78	7
Gender composition not reported	.011	.057	0.0–.620	3	.315	.315	.17–.64	3

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instability, which refers to failures in the normal buffering of the genome against environmental perturbations that threaten the progression of developmental processes (Markow, 1994). There appear to be measurable and reliable individual differences among humans in developmental instability, marked by differences in the degree of morphological and functional asymmetry and by the occurrence of minor physical anomalies, all of which are thought to be consequences of disrupted developmental sequences. Developmental instability has been shown to be associated with differences in handedness (Yeo & Gangestad, 1993) and sexual attractiveness (Thornhill & Gangestad, 1996) and has been proposed as a factor in the etiology of major psychopathology (Markow, 1992). Our laboratory is currently investigating applications of the developmental instability to sibling phenotypic differences.

Plomin and Daniels (1987) were correct that none of these alternatives to systematic linear associations between specific environmental events and specific developmental outcomes offer a clear methodological pathway for the developmental social science of the future. New methodological paradigms will no doubt evolve (Strohmann, 1997), but some aspects of the development of complex human behavior may remain outside the domain of systematic scientific investigation for a very long time. Although developmentalists may be disappointed that a substantial portion of human development remains too complex, too interactive, and too resistant to controlled investigation and straightforward statistical methods to yield to systematic scientific analysis as we currently understand it, it must be remembered that the alternative—a world in which human behavior could be understood all the way down in terms of correlations between difference scores—would present its own gloomy prospects in the ethical evaluation of human agency. The limitations of our existing social scientific methodologies ought not provoke us to wish that human behavior were simpler than we know it to be; instead they should provoke us to search for methodologies that are adequate to the task of understanding the exquisite complexity of human development.

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Appendix A

Derivation of Equations 6 and 7

If one assumes that the direct and indirect correlations between environment and outcome are equal in two siblings, the correlation between two difference scores is equal to (Tejerina-Allen, Wagner, & Cohen, 1994)

$$r_{DD} = \frac{r_{11} - r_{12}}{\sqrt{(1 - r_e)(1 - r_e)}}, \tag{A1}$$

where r_{11} is the correlation between a child's own environment and outcome, r_{12} is the cross-correlation between the sibling's environment and the child's outcome, and r_e is the correlation between the siblings' environments. As shown in Figure 1, the direct correlation between a child's environment and his or her environment equals $b_s + r_e b_c$; the cross-correlation between the environment of one sibling and the outcome of the other equals $b_c + r_e b_s$. Substituting in the above and simplifying, one obtains

$$r^2_{DD} = \frac{1 - r_e}{1 - r_o}(b_s - b_c)^2. \tag{A2}$$

Similarly, the correlation between an environmental difference score and a single sibling's outcome (r_{DY}) equals

$$r_{DY} = \frac{r_{11} - r_{12}}{\sqrt{2(1 - r_e)}}. \tag{A3}$$

Substituting as above and simplifying, one obtains

$$r^2_{DY} = \frac{1 - r_e}{2}(b_s - b_c)^2. \tag{A4}$$

(*Appendixes continue*)

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Appendix B

Characteristics of Studies

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Anderson, Hetherington, Reiss, & Howe (1994)	NEAD	708	10–18	Same sex	MZ, DZ, full, half- & step-sibs	1. Maternal warmth/ support (aggregate of observation, parent, child) 2. Paternal warmth/ support (aggregate of observation, parent, child) 3. Maternal conflict/ negativity (aggregate of observation, parent, child) 4. Paternal conflict/ negativity (aggregate of observation, parent, child) 5. Maternal monitoring/control (aggregate of observation, parent, child) 6. Paternal monitoring/ control (aggregate of observation, parent, child)	1. Social responsibility (aggregate of mother, father, child) 2. Cognitive agency (aggregate of mother, father, child) 3. Sociability (aggregate of mother, father, child) 4. Autonomy (aggregate of mother, father, child) 5. Self-worth (child)	Nongenetic	Cross-sectional	Reported	b_s, b_c
Baker & Daniels (1990)	Southern CA Twin Project	161	18–75	Same sex	MZ twins	1. Twin interaction (child) 2. Parental treatment (child) 3. Peer group characteristics (child)	1. Personality scales from EPQ (child) 2. Affect balance (child) 3. Depression (child)	Genetic	Cross-sectional	Not reported	r^2_{DD}
Bouchard & McGue (1990)	MSTRA	45	19–68	Same sex	MZ twins	1. Family cohesion vs. conflict from FES (child) 2. Family positive constraint from FES (child) 3. Family encouragement of individual growth from FES (child)	1. Personality scales/factors from CPI (child)	Genetic	Cross-sectional	Reported	r^2_{DD}

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Braungart, Fulker, & Plomin (1992)	CAP	190	1–2	Mixed sex	Adoptive & nonadoptive sibs	1. Home environment quality (aggregate of observation, mother)	1. Mental development (observation)	Genetic	Cross-sectional	Reported	b_s, b_c
Brody, Stoneman, & Burke (1987)	—	40	4–9	Same sex	Full sibs	1. Maternal positive behavior (observation) 2. Maternal verbalization (observation) 3. Maternal management (observation)	1. Prosocial behavior during sib interaction (observation) 2. Agonistic behavior during sib interaction (observation) 3. Verbalization during sib interaction (observation)	Nongenetic	Cross-sectional	Reported	r^2_{DY}
Brody, Stoneman, & McCoy (1992)	—	109	4–11	Same sex	Full sibs	1. Maternal & paternal control (observation) 2. Maternal & paternal responsiveness (observation) 3. Maternal & paternal negative behavior (observation)	1. Negative behavior during sib interaction (observation) 2. Positive behavior during sib interaction (observation)	Nongenetic	Longitudinal & cross-sectional	Not reported	r^2_{DY}
Carpey (1989)	—	52–59	3–6	Same sex	MZ and DZ twins	1. Maternal discipline (parent) 2. Maternal affect (parent) 3. Maternal warmth (observation) 4. Maternal control (observation)	1. Temperament (observation, mother) 2. Mood (observation) 3. Affect (observation)	Nongenetic	Cross-sectional	Reported	r^2_{DD}
Conger & Conger (1994)	Iowa Youth and Family Project	359	9–21	Mixed sex	Full sibs	1. Maternal & paternal hostility (observation)	1. Delinquency (child)	Nongenetic	Longitudinal & cross-sectional	Reported	r^2_{DD}

(*Appendixes continue*)

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Daniels (1985)	CAP	133	1–2	Mixed sex	Biological & adoptive sibs	1. Maternal encouragement of developmental advance (aggregate of observation, mother) 2. Maternal restriction/ punishment (aggregate of observation, mother) 3. Maternal and paternal general behavior (aggregate of observation, mother) 4. Personal growth (aggregate of mother, father)	1. Attention span (aggregate of mother, father) 2. Activity level (observation) 3. Behavior problems/difficult temperament (aggregate of mother, father)	Nongenetic	Cross-sectional	Not reported	r^2_{DD}
Daniels (1986)	—	148	12–28	Mixed sex	Biological & adoptive sibs	1. Multiple measures (e.g., parental treatment, sib interaction, peer characteristics [child])	1. Temperament (child) 2. Expected education (child) 3. Expected occupation (child)	Nongenetic	Cross-sectional	Reported	r^2_{DD}
Daniels, Dunn, Furstenberg, & Plomin (1985)	NSC	348	11–17	Mixed	—	1. Multiple measures (e.g., family cooperation, family stress, parental rule expectations, maternal closeness, paternal closeness, say in decisions, sib friendliness, peer friendliness [mother, child])	1. Emotional distress (mother, child) 2. Delinquency (mother, child) 3. Disobedience (mother, child, teacher)	Nongenetic	Cross-sectional	Not reported	r^2_{DD}

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Deater-Deckard (1996)	—	112	1–11	Mixed sex	Full sibs	1. Maternal negative affect (parent) 2. Paternal negative affect (parent) 3. Maternal physical control (parent) 4. Paternal physical control (parent) 5. Maternal verbal control (parent) 6. Paternal verbal control (parent)	1. Internalizing (parent) 2. Externalizing (parent)	Nongenetic	Cross-sectional	Reported	r^2_{DD}
Dudley (1994)	—	28	17–35	Mixed sex	—	1. Parental affection (child) 2. Parental control (child) 3. Sib interaction (child) 4. Peer characteristics (child)	1. Symptom count (child)	Nongenetic	Cross-sectional	Reported	r^2_{DD} and r^2_{DY}
Dunn, Stocker, & Plomin (1990)	CSS (subsample of CAP)	67	Range not provided (all children age 7)	Mixed sex	Full sibs	1. Maternal affection (observation, mother) 2. Maternal control (observation, mother) 3. Sib positivity (mother) 4. Sib negativity (mother) 5. Sib cooperation (observation) 6. Sib conflict (observation) 7. Multiple measures of maternal behavior (observation, mother) 8. Multiple measures of sib behavior (observation, mother)	1. Internalizing (mother) 2. Externalizing (mother)	Nongenetic	Cross-sectional	Reported	r^2_{DY}

(*Appendixes continue*)

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Henderson, Hetherington, Mekos, & Reiss (1996)	NEAD	516	10–18	Same sex	Full and half-sibs	1. Maternal warmth/support (aggregate of observation, parent, child) 2. Paternal warmth/support (aggregate of observation, parent, child) 3. Maternal conflict/negativity (aggregate of observation, parent, child) 4. Paternal conflict/negativity (aggregate of observation, parent, child) 5. Maternal monitoring/control (aggregate of observation, parent, child) 6. Paternal monitoring/control (aggregate of observation, parent, child)	1. Emotionality (aggregate of mother, father)	Nongenetic	Cross-sectional	Reported	r^2_{DD}
Honbo (1991)	Hawaii Family Study	418	Range not provided (M age = 30)	—	—	1. Parental closeness (child) 2. Parental affection (child) 3. Sib antagonism (child) 4. Sib closeness (child) 5. Peer characteristics (e.g., delinquency, popularity, college orientation [child])	1. Personality characteristics (e.g., extroversion, neuroticism, psychoticism [child]) 2. Educational attainment (child) 3. Occupational attainment (child)	Nongenetic	Cross-sectional	Not reported	r^2_{DY}

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Jang (1993)	Western Ontario Twin Project	114	16–45	Same sex	DZ & full sibs	1. Maternal affection (child) 2. Maternal control (child) 3. Teacher control (child) 4. Peer delinquency (child) 5. Multiple measures (e.g., paternal control, achievement orientation [child])	1. Schizophrenia (child) 2. Social introversion (child) 3. Succorance (child) 4. Play(fulness) (child) 5. Psychasthenia (child) 6. Addiction (child)	Nongenetic	Cross-sectional	Not reported	r^2_{DD}
Jodyl (1997)	NEAD	56	10–18	Same sex	Full sibs	1. Maternal and paternal autonomy/ relatedness (observation)	1. Internalizing (aggregate of observation, parent, child) 2. Externalizing (aggregate of observation, parent, child)	Nongenetic	Longitudinal & cross-sectional	Reported	r^2_{DD} & r^2_{DY}
Lewis (1992)	—	31–35	11–17	Mixed and same sex	MZ, DZ, & full sibs	1. Parental treatment (mother, child) 2. Parental affection (mother, child) 3. Parental control (mother, child) 4. Sib interaction (mother, child) 5. Peer characteristics (mother, child) 6. Specific events (mother, child)	1. Reading, math, language, & total achievement (scores of child)	Genetic & nongenetic	Longitudinal & cross-sectional	Not reported	r^2_{DD}
McGonigle, Smith, Benjamin, & Turner (1993)	—	25	27–63	Same sex	MZ twins	1. Maternal belittling/ blaming (child) 2. Parental belittling/ blaming (child) 3. Paternal attacking/ rejecting (child)	1. Hostility (child)	Genetic	Cross-sectional	Not reported	r^2_{DD}
McGuire & Dunn (1994)	CAP	54–100	7–10	Mixed sex	Full & adoptive sibs	1. Maternal acceptance (mother) 2. Maternal negative control (mother) 3. Maternal consistency (mother)	1. Internalizing (mother) 2. Externalizing (mother) 3. Temperament (mother, teacher)	Nongenetic	Longitudinal & cross-sectional	Reported	r^2_{DY}

(*Appendixes continue*)

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
McGuire, Dunn, & Plomin (1995)	CSS (subsample of CAP)	82	3–13	Mixed sex	Full & adoptive sibs	1. Maternal ease of discipline (mother) 2. Maternal frequency of discipline (mother) 3. Maternal affection (mother) 4. Maternal attention (mother)	1. Internalizing (mother, teacher) 2. Externalizing (mother, teacher)	Nongenetic	Longitudinal & cross-sectional	Reported	r^2_{DV}
Mekos, Hetherington, & Reiss (1996)	NEAD	516	10–18	Same sex	Full, half-, & step-sibs	1. Maternal warmth/support (observation, parent, child) 2. Paternal warmth/support (observation, parent, child) 3. Maternal conflict/negativity (observation, parent, child) 4. Paternal conflict/negativity (observation, parent, child) 5. Maternal monitoring/control (observation, parent, child) 6. Paternal monitoring/control (observation, parent, child) 7. Exposure to discord (mother, father, child)	1. Alcohol use (child) 2. Marijuana use (child) 3. Deviance (child)	Nongenetic	Cross-sectional	Reported	r^2_{DD}

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Monahan, Buchanan, Maccoby, & Dornbusch (1993)	Stanford Child Custody Study	133	10–18	Mixed sex	—	1. Parental involvement (parent) 2. Household organization (child) 3. Parental monitoring (child) 4. Closeness to residential parent (child) 5. Closeness to nonresidential parent (child) 6. Parent–child conflict (child) 7. Feelings of being caught between parents (child)	1. Depression (child) 2. School deviance (child) 3. Substance use (child) 4. Antisocial behavior (child) 5. Grades (child) 6. School effort (child) 7. "Worst of three outcomes" (child)	Nongenetic	Cross-sectional	Reported	r^2_{DD}
Neiderhiser (1994)	NEAD	395	10–18	Same sex	MZ, DZ, full, half-, & unrelated sibs	1. Maternal negativity (aggregate of observation, parent, child) 2. Paternal negativity (aggregate of observation, parent, child)	1. Antisocial behavior (aggregate of observation, mother, father, child) 2. Depression (aggregate of mother, father, child)	Genetic	Longitudinal & cross-sectional	Reported	b_g, b_c
O'Connor (1995)	NEAD	405–516	10–18	Same sex	Full, half-, & unrelated sibs	1. Maternal positivity (aggregate of observation, parent, child) 2. Paternal positivity (aggregate of observation, parent, child) 3. Maternal negativity (aggregate of observation, parent, child) 4. Paternal negativity (aggregate of observation, parent, child)	1. Antisocial behavior (aggregate of observation, mother, father, child) 2. Depression (aggregate of observation, mother, father, child)	Nongenetic	Cross-sectional	Reported	b_g, b_c

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Pike, McGuire, Hetherington, & Plomin (1996)	NEAD	719	10–18	Same sex	MZ, DZ, full, half-, & unrelated sibs	1. Maternal positivity (aggregate of observation, parent, child) 2. Paternal positivity (aggregate of observation, parent, child) 3. Sib positivity (aggregate of observation, parent, child) 4. Maternal negativity (aggregate of observation, parent, child) 5. Paternal negativity (aggregate of observation, parent, child) 6. Sib negativity (aggregate of observation, parent, child)	1. Antisocial behavior (aggregate of observation, mother, father, child) 2. Depression (aggregate of observation, mother, father, child)	Genetic	Cross-sectional	Reported	b_s, b_c
Pike, Reiss, Hetherington, & Plomin (1996)	NEAD	93	10–18	Same sex	MZ twins	1. Maternal negativity (observation, parent, child, & aggregate of reporter types) 2. Paternal negativity (observation, parent, child, & aggregate of reporter types)	1. Antisocial behavior (observation, mother, father, child, & aggregate of reporter types) 2. Depression (observation, mother, father, child, & aggregate of reporter types)	Genetic	Cross-sectional	Reported	r^2_{DD}

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Plomin, Manke, & Pike (1996)	NEAD	93–181	10–18	Same sex	Full sibs	1. Maternal positive behavior (parent, child) 2. Paternal positive behavior (parent, child) 3. Maternal negative behavior (parent, child) 4. Paternal negative behavior (parent, child) 5. Maternal control (parent, child) 6. Paternal control (parent, child)	1. Self-worth (child)	Nongenetic	Cross-sectional	Reported	r^2_{DD}
Reiss et al. (1994)	NEAD	214	10–18	Same sex	Full, half-, & unrelated sibs	1. Maternal aggression (parent, child) 2. Paternal aggression (parent, child)	1. Internalizing (mother, father, child) 2. Externalizing (mother, father, child)	Nongenetic	Cross-sectional	Reported	r^2_{DY}
Reiss et al. (1995)	NEAD	708	10–18	Same sex	MZ, DZ, full, half-, & unrelated sibs	1. Maternal warmth/support (aggregate of observation, parent, child) 2. Paternal warmth/support (aggregate of observation, parent, child) 3. Maternal conflict/negativity (aggregate of observation, parent, child) 4. Paternal conflict/negativity (aggregate of observation, parent, child) 5. Maternal monitoring/control (aggregate of observation, parent, child) 6. Paternal monitoring/control (aggregate of observation, parent, child)	1. Antisocial behavior (aggregate of observation, mother, father, child) 2. Depression (aggregate of mother, father, child)	Nongenetic	Cross-sectional	Reported	b_s, b_c

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
J. L. Rodgers & Rowe (1985)	—	173	7–12	Mixed sex	—	1. Family structure (e.g., birth order, spacing, no. of intervening sibs, & sex similarity [mother])	1. IQ	Nongenetic		Reported	r^2_{DD}
J. M. Rodgers, Rowe, & May (1994)	NLSY	1,044	5–12	Mixed sex	Twins, full & half-sibs, and cousins	1. Maternal reading (aggregate of mother, child) 2. Maternal spanking (aggregate of mother, child) 3. Number of books (aggregate of mother, child) 4. Visits to museum (aggregate of mother, child) 5. Visits to theater (aggregate of mother, child) 6. Quality of home (aggregate of mother, child)	1. Math (child's score) 2. Reading recognition (child's score) 3. Reading comprehension (child's score) 4. Picture vocabulary (child's score) 5. Wechsler Digit Span (child's score) 6. General cognitive ability (child's score)	Genetic	Cross-sectional	Reported	r^2_{DD}
Rowe, Rodgers, & Meseck-Bushey (1992)	NLSY	4,117	15–22	Mixed sex	—	1. Family structure (e.g., birth order, spacing, no. of intervening sibs, & sex similarity (child)	1. Delinquency (child)	Nongenetic		Reported	r^2_{DD}
Saudino & Plomin (1997)	CAP		12–24 mos.	Mixed sex	Adoptive & nonadoptive sibs	1. Home environment (aggregate of observation & parent)	1. Mental development (observation) 2. Task orientation (observation)	Genetic	Cross-sectional	Reported	b_a, b_c
Shoenwald (1993)	—	75	Range not provided (*M* age of target sib = 10; *M* age of sib = 13)	Mixed	Full sibs	1. Maternal control (mother, child) 2. Maternal affection (mother, child) 3. Sib interaction (mother, child)	1. Aggression (parent, child, teacher)	Nongenetic	Cross-sectional	Reported	r^2_{DY}

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Appendix B (*continued*)

Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Stocker (1993)	CAP	77	Range not provided (older sib *M* age = 7 & younger sib *M* age = 4 at T1; assessed again 3 years later)	Mixed	Full & adoptive sibs	1. Maternal control when sibs at same age (mother) 2. Maternal control when sibs are different ages (mother) 3. Maternal positive behavior when sibs at same age (mother) 4. Maternal affection when sibs are different ages (mother)	1. Internalizing (mother) 2. Externalizing (mother)	Nongenetic	Longitudinal & cross-sectional	Reported	r^2_{DD} and r^2_{DY}
Stocker (1995)	—	63	Range not provided (*M* age of target sib = 8)	Mixed	—	1. Maternal closeness (parent) 2. Paternal closeness (parent) 3. Maternal conflict (parent) 4. Paternal conflict (parent) 5. Maternal favoritism (parent) 6. Paternal favoritism (parent) 7. Mother–child relationship quality (parent) 8. Father–child relationship quality (parent)	1. Sociability (mother) 2. Emotionality (mother) 3. Activity (mother) 4. Shyness (mother) 5. Internalizing (mother) 6. Externalizing (mother)	Nongenetic	Cross-sectional	Reported	r^2_{DD} & r^2_{DY}
Tarullo, DeMulder, Ronsaville, Brown, & Radke-Yarrow (1995)	—	77	Range not provided (older sib *M* age = 6.4 & younger sib *M* age = 2.6 at T1; assessed again 3 & 6 years later)	Mixed	—	1. Maternal treatment (observation)	1. Symptom count (mother, child)	Nongenetic	Longitudinal	Reported	r^2_{DY}

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Study	Data set	*N* sib pairs	Age range	Gender composition of sib pairs	Genetic relatedness of sibs	Environmental measure (reporter)	Outcome measure (reporter)	Genetic vs. nongenetic	Cross-sectional vs. longitudinal	*NS* results reported	Effect type
Tejerina-Allen, Wagner, & Cohen (1994)	Children in the Community	178	11–21	—	—	1. Maternal bond (mother, child, & aggregate of reporter types) 2. Maternal punishment (mother, child, & aggregate of reporter types)	1. Depression (aggregate of mother, child) 2. Suicidal ideation (child) 3. Opposition (aggregate of mother, child)	Nongenetic	Cross-sectional	Reported	r^2_{DD}
Vernon, Jang, Harris, & McCarthy (1997)	Western Ontario Twin Project	106	16–45	Same sex	MZ, DZ, & full sibs	1. Parental affection and control (child) 2. Interpersonal relationships (child) 3. Acceptance–rejection (child) 4. Multiple measures of classroom environment (child) 5. Multiple measures of family environment (child)	1. Autonomy (child) 2. Conscientiousness (child) 3. Neuroticism (child)	Genetic & nongenetic	Cross-sectional	Not reported	r^2_{DD}
Wagner & Cohen (1994)	Children in the community	178	11–21	—	—	1. Maternal warmth (mother, child) 2. Maternal discipline (mother, child)	1. Suicidal ideation (child)	Nongenetic	Cross-sectional	Reported	r^2_{DD}
Wonderlich, Ukestad, & Perzacki (1994)	—	57	18–46	—	—	1. Sib interaction (child) 2. Parental treatment (child) 3. Peer group characteristics (child)	1. Histrionic (child) 2. Borderline (child) 3. Avoidant (child) 4. Dependent (child) 5. Compulsive (child) 6. Depressive (child)	Nongenetic	Cross-sectional	Reported	r^2_{DY}

Note. CA = California; CAP = Colorado Adoption Project; CPI = California Psychological Inventory; CSS = Colorado Sibling Study; DZ = dizygotic; EPQ = Eysenck Personality Questionnaire; FES = Family Environment Scale; MSTRA = Minnesota Study of Twins Reared Apart; MZ = monozygotic; NEAD = Nonshared Environment and Adolescent Development; NLSY = National Longitudinal Survey of Youth; NSC = National Survey of Children; sib = sibling; T1 = Time 1. r_{DD} is the correlation between an environmental difference score and an outcome difference score; r_{DY} is the correlation between an environmental difference score and a single sibling's outcome. Dashes indicate that the information could not be determined.

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