

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 non-genetically 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

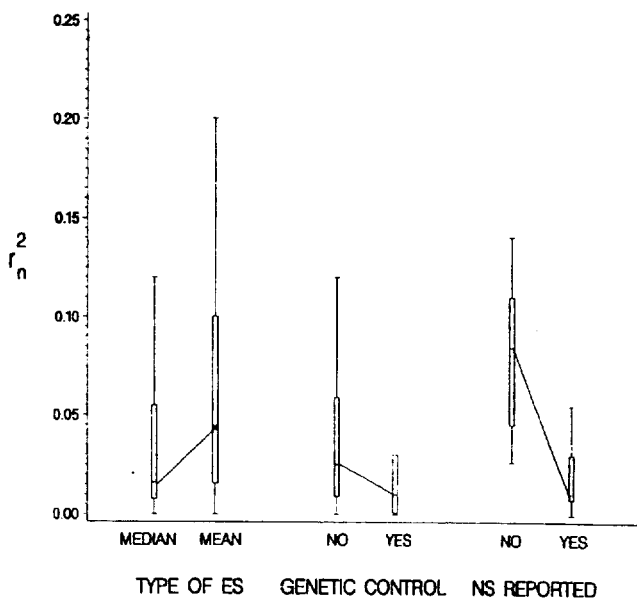


Figure 5. Box and whisker plot of weighted mean and median effect size (ES) for all studies and median effect size for studies employing genetically informative designs (genetic control) and reporting nonsignificant results (NS reported). The boxes extend from the 25th to the 75th percentiles of the 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.

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 ($Mdn = .523$, $M = .510$, $n = 12$).

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

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.

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

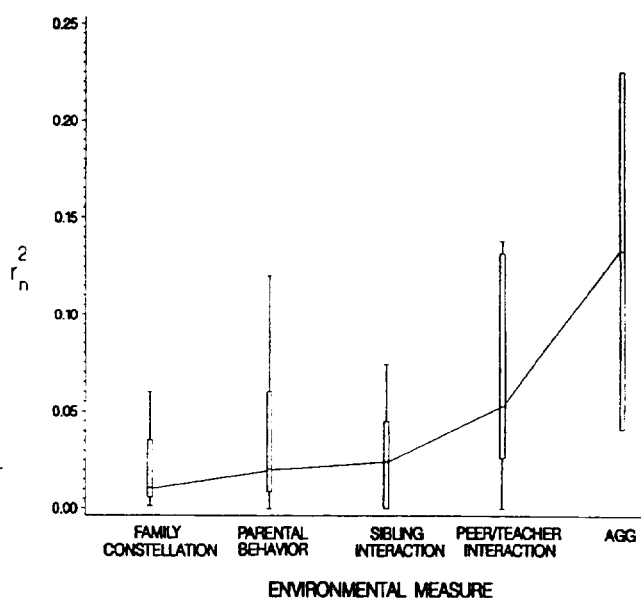


Figure 6. Median effect size by environmental measure. AGG indicates aggregates of more than one measure. (See Figure 5 caption for explanation of box and whisker plots.)

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 expla-

nation, 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 ($G \times E$) 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 $G \times E$ 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 $G \times E$, we have argued that they may be more accurately characterized as phenotype by environment ($P \times E$) interactions (Turkheimer, 1999). The idea of $P \times E$ 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

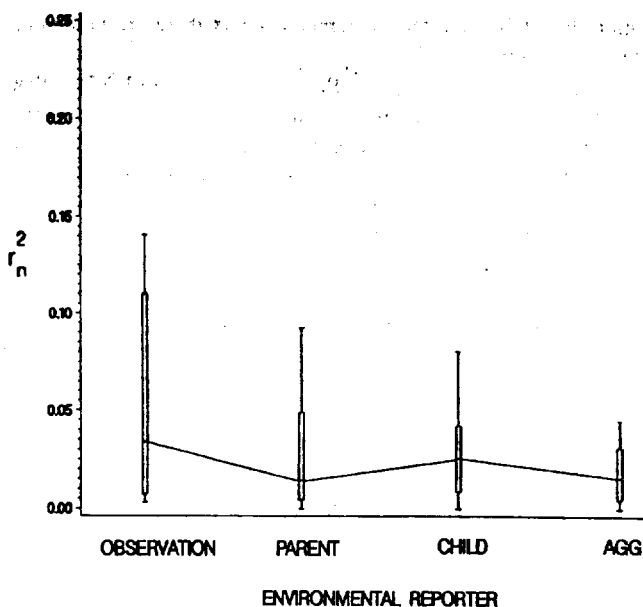


Figure 7. Median effect size by environmental reporter. AGG indicates aggregate of more than one reporter. (See Figure 5 caption for explanation of box and whisker plots.)

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 $G \times E$ interaction (Scarr, 1992; Scarr & McCartney, 1983). Models with $P \times E$ interaction contain complex reciprocal effects between phenotype and environment: Phenotype affects the organism's choice of environments, environments cause changes in pheno-

types, 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 *emergensis* (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. Kurnit, Layton, and Matthyse (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

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 (Strohmman, 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)

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

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

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

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

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

(Appendixes continue)

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