Biology-Environment Interaction and Evocative Biology-Environment Correlation: Contributions of Harsh Discipline and Parental Psychopathology to Problem Adolescent Behaviors

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Using an adoption paradigm, the Bioecological Model of development proposed by Bronfenbrenner and Ceci in 1994 was tested by concurrently modeling for biology-environment interaction and evocative biology-environment correlation. A sample of 150 adult adoptees (ages, 18–45 years) provided retrospective reports of harsh adoptive parent discipline, which served as the environmental independent variables. Birth parent psychopathology served as the biological predictor. The dependent variables were retrospective adoptee and adoptive parent reports on adolescent aggressive and conduct-disordered behaviors. Finally, adoptees were classified as experiencing contextual environmental risk using the presence of two or more adverse factors in the adoptive home (e.g., adoptive parent psychopathology) as the cutoff. The contextual environment was found to moderate the biological process of evocative biology-environment correlation, providing empirical support for the Bronfenbrenner and Ceci (1994) Bioecological Model.

KEY WORDS: Biology; environment; discipline; aggression; conduct disorder.

INTRODUCTION

The influence of biological factors on behavior has been increasingly recognized, but the processes involved in the interaction between biology and the environment remain largely unexplored (Rutter and Pickles, 1991; Rutter et al., 1997). One model of how biology and environment combine to affect behavior has been proposed by Bronfenbrenner and Ceci (1994). Based on their model, which they identify as a Bioecological Model, Bronfenbrenner and Ceci (1994) postulate several distinct and specific hypotheses that are testable. In this paper, we interpret the Bioecological Model as combining the processes of biologyenvironment interaction and biology-environment correlation and use an adoption sample consisting of adoptees with or without psychiatric disorder in their

biological background to test our model.5

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⁴ Biological rather than genetic effects will be used in this paper because of the inability to distinguish between intrauterine environmental effects from specific genetic contributions by the birth mother. Given the high-risk nature of our sample, specifically alcohol and illegal substance use, we believe this distinction to be warranted.

⁵ Bronfenbrenner and Ceci (1994) presented the Bioecological Model of development as it pertained to the development of constructive behaviors. In the present paper, the same model is applied to the development of non-constructive or maladaptive behavior. It is assumed that the central presuppositions of the model apply equally to the development of negative behaviors as they do to the development of positive behaviors when the proximal process of interest is reactive to characteristics of the individual. In other words, greater biological influence will be found in the presence of dysfunctional proximal processes.

Propositions of the Bioecological Model

Bronfenbrenner and Ceci (1994) developed three propositions, which we outline briefly below, regarding the nature of the interaction between biological and environmental factors in the development of behavior.

Proposition I: Proximal Processes

The first proposition of the Bioecological Model addresses the complex interaction between the individual and the environment. Precisely defined, proximal processes are "progressively more complex reciprocal interaction[s] between an active, evolving biopsychological human organism and the persons, objects, and symbols in its immediate environment" (Bronfenbrenner and Ceci, 1994, p. 572). These processes are postulated to endure over time, to occur in the immediate environment, and to provide a mechanism for the phenotypic expression of biologically influenced predispositions.

Proposition II: Individual and Contextual Influences on Proximal Processes

The second proposition of the Bioecological Model recognizes the impact of individual characteristics and the broader environmental context on the nature or quality of proximal processes. Specifically, the authors postulate that "the form, power, content and direction of the proximal processes effecting development vary systematically as a joint function of the characteristics of the developing person, of the environment . . . in which the processes are taking place, and of the nature of the developmental outcomes under consideration" (Bronfenbrenner and Ceci, 1994, p. 572). In other words, examining the role of proximal processes in the phenotypic expression of biological factors requires consideration of characteristics of all persons relevant to the specific process of interest, as well as contextual environmental conditions.

Proposition III: Translation of Genotype into Phenotype

The third and final proposition of the Bioecological Model combines the first two propositions as they relate to the expression of biological factors as observed behavior. Specifically, proximal processes serve as mechanisms that influence the degree to which expression of behavior occurs and vary according to the characteristics of the individual and the relevant contextual factors, as well as the developmental outcome of interest (Bronfenbrenner and Ceci, 1994). In other words, the contextual environment, by its moderating

effect upon a proximal process, will magnify or diminish the behavioral expression of biological predispositions.

Hypotheses Suggested by the Theoretical Model

From the above propositions, two general predictions can be made regarding the expression of biological propensities. First, given that the proximal process reinforces the expression of a certain behavior, the presence of that process should increase the strength of the biological influences on the expression of the identified behavior. Second, the increased strength of the biological influence and the reinforcing nature of the proximal process should result in increases in mean levels of behavior when contextual environmental factors promote the establishment and maintenance of the proximal process. It is our opinion that biology-environment correlation and biology-environment interaction can be used to test these predictions.

When identifying a proximal process, Bronfenbrenner and Ceci (1994) indicated that the process should be sensitive to the characteristics of the individuals within the dyad, occur on a frequent basis, and endure over time. It has been recognized for decades that interactions between parents and children are influenced by characteristics of both the parent and the child (Bell, 1968, 1971, 1979; Bell and Chapman, 1986; Maccoby, 1992). However, the degree to which parent-child interactions are mediated biologically has not been fully investigated until recently. By utilizing biologically informative designs, researchers have begun testing for evocative biology-environment correlation (Ge *et al.*, 1996; O'Connor *et al.*, 1998).

Evocative biology-environment correlation refers to the degree to which persons within the environment react to biologically influenced characteristics of an individual (Plomin et al., 1977; Scarr and McCartney, 1983). We classify evocative biology-environment correlation as a proximal process due to the recognition that the immediate environment may react to characteristics of an individual. To evaluate the impact of the contextual environment on the proximal process, we rely on another concept from behavior genetics, biologyenvironment interaction. Our application of biologyenvironment interaction differs somewhat from its traditional use in which the degree to which biological influences vary across environmental conditions is examined (Plomin et al., 1977). We extend the concept by examining differences in the strength of a biologically influenced proximal process, evocative biology-environment correlation, across different environments. Applying biology-environment interaction in this manner represents the combining of evocative biology-environment correlation and biology-environment interaction into a single theoretical model.

Theoretical Model

Identification of Proximal Process

We attempted to identify a proximal process that we believe important in the expression of a biological trait (e.g., a diatheses for externalizing behaviors) as manifest behavior (e.g., adolescent oppositional and conduct-disordered behavior). The proximal process we identified was that of harsh disciplinary practices (Fig. 1). We chose disciplinary practices for several reasons: 1) Researchers have long recognized that child characteristics can influence parental behaviors and that, specifically, negative behavioral child characteristics can contribute to coercive and abrasive interactions between a child and his or her parents (Anderson et al., 1986; Caspi et al., 1987; Dodge, 1990; Lytton, 1982, 1990; Patterson, 1982, 1983; Reid et al., 1982). 2) Punitive disciplinary practices have been found to significantly contribute to adolescent problem behaviors (Burgess and Conger, 1978; Deater-Deckard and Dodge, 1997; Dodge et al., 1995; George and Main, 1979; Meyer et al., 2000; Patterson *et al.*, 1992; Reidy, 1977; Salzinger *et al.*, 1984; Taplin and Reid, 1977). 3) Adolescent problem behaviors have been shown to have some biological basis (Cadoret et al., 1995b; DiLilla and Gottesman, 1991; Lytton, 1990; Rutter, 1997; Rutter et al., 1990). The above findings provide empirical support for the essential requirements of evocation, which will be described in greater detail below.

Moderation of Proximal Process

The Bioecological Model also predicts that the context of the environment will moderate the nature of the proximal process (Bronfenbrenner and Ceci, 1994). Again, we relied on previous research to identify those contextual factors that might moderate the process (Greenwald *et al.*, 1997; Patterson, 1983). For example, families experiencing economic stress, as well as additional life stressors, have been found to demonstrate greater hostility and fewer supportive behaviors towards family members (Conger *et al.*, 1992; Conger *et al.*, 1991; Elder and Caspi, 1988; Larzalere and Patterson, 1990; Lempers *et al.*, 1989), and depressed mothers have been shown to behave in a less nurturant manner toward their children than nondepressed mothers (Coyne and Downey, 1991; Downey and Coyne,

1990; Susman *et al.*, 1985; Zahn-Waxler *et al.*, 1990). Therefore, we hypothesized that the presence of psychiatric and legal problems experienced by the adoptive parents might increase stress within the home and increase the reactivity of those parents to the child's irritating behaviors, whereas in families without additional family stress, these same behaviors might be less likely to elicit a punitive response.

In summary, based on our interpretation of the Bioecological Model (Bronfenbrenner and Ceci, 1994), we created a model in which biology-environment interaction and biology-environment correlation were evaluated concurrently. The following associations were tested using a biologically informative adoption design: 1) We examined whether a biological predisposition for antisocial behaviors expressed itself as adolescent problem behaviors, a finding that has been demonstrated with these data (Cadoret et al., 1995a, 1995b).6 2) We tested whether adolescent problem behaviors elicited harsh disciplinary actions from the adoptive parents via the expression of adolescent problem behaviors. 3) We examined the moderating effect of contextual environmental factors on the above process.

METHODS

Sample

Subjects from this study were recruited from four adoption agencies in the state of Iowa: Lutheran Social Services of Iowa; Catholic Charities of the Archdiocese of Dubuque; Hillcrest Family Services; and the Iowa Department of Human Services. Data were collected between the years of 1989 and 1992. One hundred fifty subjects were drawn from the original sample of adult adoptees (n=197). Twenty-five of the potential subjects were excluded due to placement in the adoptive home after six months of age, which makes this a subsample of the original study. Twenty-two of the adoptive parents were not interviewed and these cases were also excluded from analyses leaving an effective sample size of 150. The sample had equal numbers of males (n=71) and females (n=79).

⁶ The sample used for this paper has been presented in previous articles; however, the measures used were not included in these studies. Also, adoptees placed into the adoptive home after 6 months of age were not excluded from previous analyses. Furthermore, this paper extends previous research by examining proximal processes (e.g., disciplinary practices) versus contextual factors (e.g., psychiatric disorder in parents) that are relevant to adolescent problem

Procedure

The data collection procedure was typical of an adoptee study in which the sample is identified via birth parents. For this study, birth parent diagnoses of antisocial personality, criminality, alcoholism, and/or substance use/abuse was the selection criterion and was established by circulating names of birth parents through the following institutions: Iowa Department of Human Services, State of Iowa Mental Health institutions, University of Iowa Hospitals and Clinics, and the Iowa Department of Corrections (see Cadoret et al., 1995a,b). Initially, 11,700 birth parent names were screened. Identification of the correct biologic parent was confirmed by comparing dates of birth of parent, place of parent's birth, names of the biologic parents, names of siblings, and other identifying data found in the institution's record with information in the adoption agency records. Following collection of institutional information, three psychiatrists independently diagnosed the birth parent as antisocial, alcohol or a substance abuser, using criteria from the DSM-III-R. The kappa values for diagnosing anti-social personality and alcoholism were adequate (range: $\kappa =$.67-.90). Differences in diagnoses were resolved through conference, with unresolved differences in diagnosis resulting in the case being discarded. All adult adoptees in the study were separated from their birth mothers at the time of birth. Additional information on other psychiatric conditions (e.g., depression, aggressive behavior) was collected from adoption agency records but not used to classify the adoptee as index or comparison in this study.

Two groups of adult adoptees were created: an index group and a comparison group. The index group consisted of adoptees for whom at least one of the following diagnoses/behaviors were identified in the birth parent: alcohol abuse/dependency, antisocial personality, drug abuse/dependency, and/or criminality, whereas the comparison group consisted of adoptees with birth parents who lacked any evidence of the above psychiatric problems. Additionally, index and comparison adoptees were matched on age and sex of the adoptee and by age of the biological mother at the time of the adoptee's birth. The sample was predominantly Caucasian (94%) and approximately evenly split between female and male adoptees (n = 79 and n = 71). Adoptive mothers and fathers averaged 1 to 2 years of college education (mean = 13-14 years), whereas birth parents averaged 11 years of education. The mean ages of the adoptive and birth parents at the time of placement were as follows: adoptive mothers (mean = $29 \pm$ 4), adoptive fathers (mean = 32 ± 5), birth mother (mean = 21 ± 5), and birth fathers (mean = 23 ± 6). Median adoptive family income was \$30,000–\$39,999 at the time of interview (1989–1994). Selective placement for psychological disorder was examined in these data. The phi coefficient between adoptive parent psychopathology and birth parent psychopathology was non-significant (r = .10, p > .05).

Measures

The adoptive parents completed an interview designed to assess the parent's retrospective view of the adoptee's physical health, temperament, development, school achievement, and social adjustment from infancy to the present. A section of the interview also covered details about emotional or psychiatric problems in the adoptive family. All interviews were conducted in person by interviewers blind to the biological background of the adoptee. Adoptive parents and adoptees were interviewed separately and not necessarily by the same interviewer.

The adoptee interview consisted of the Diagnostic Interview Schedule (DIS; Robins et al., 1989). The DIS allows lay interviewers to assess current and lifetime prevalence of psychiatric illness by DSM-III-R (1994) criteria and Research Diagnostic Criteria, as well as Feighner criteria (Feighner et al., 1972). Prior to data collection, research assistants attended a weeklong DIS instruction course at Washington University (St. Louis, MO). Medical records also were collected to verify treatment and diagnosis of psychiatric conditions or substance abuse. Prior to the interview session, adoptees completed several questionnaires retrospectively assessing the adoptive family atmosphere and the parent-child relationship during the time that the adoptee resided in the adoptive home. As with the adoptive parent interviews, the interviewers were blind to the adoptees' biological background.

Measures of Environmental and Biological Risk

Environmental Risk

As part of an extensive interview, adoptive parents reported the presence or absence of psychopathology and legal or marital problems they had experienced over their life time (Table I). An environmental risk variable was created by counting the number of adverse factors present in the adoptive parents. The count score was dichotomized into two environmental risk groups: low

risk if the score was 0 or 1 (n = 85) and moderate risk if two or more factors were present (n = 65).⁷ As shown in the top portion of Table I, the most common risk factors were depression and anxiety in at least one of the adoptive parents.

Biological Risk

In contrast to earlier studies using this sample of adoptees in which single diagnoses served as indicators of birth parent psychiatric disorder (Cadoret et al., 1995a, 1995b), a count score for birth parent psychopathology was created from four psychiatric conditions: 1) alcoholism, 2) illicit substance abuse, 3) antisocial personality defined as two adolescent conduct disorder symptoms and three adult antisocial symptoms, and 4) criminal behavior defined as a criminal conviction in the absence of ASP criteria. Separate counts were created for each birth parent and summed for a total biological risk variable. We used a cutoff of two birth parent diagnoses to classify adoptees as index cases for several reasons: 1) a similar approach was used by Ge et al., (1996), 2) only 15% of the diagnosed birth parents had single psychiatric diagnoses, 3) only 3% of the index cases had diagnoses in both the biological mother and father, and 4) analyses could not be conducted separately for each biological parent due to low numbers.

The prevalence of each condition is presented in the bottom portion of Table I for both environmental risk groups. Chi-square analyses failed to show significant differences across environmental risk for individual diagnoses of birth parents. Independent t-tests also failed to show significant mean differences between the low environmental risk group and the moderate environmental risk group in the number of birth parent psychiatric diagnoses suggesting that adoptees from both environmental risk groups had similar biological liability for these psychiatric disorders (see Table I). Birth parent psychopathology was included in the model as a dichotomous manifest exogenous variable.

Adoptee Behavioral Outcomes

A latent variable was created using two manifest indicators of adolescent problem behaviors: DSM-IV (1994) adolescent conduct disordered behaviors and oppositional behaviors. Adoptive parents retrospectively reported on a 4-point Likert scale (e.g., 0 = never, 1 = rarely, 2 = sometimes, 3 = often) for all items. Items were summed for each indicator of adolescent problem behaviors. Means and standard deviations are presented in Table II for the entire sample and separately by environmental risk groups in Table III.

Adolescent Conduct Disordered Behaviors

Eight items were used to construct the indicator for adoptee conduct disorder: staying out late, bullying, skipping school, running away, early sexual behaviors, fighting, verbally abusing others, and stealing. Internal consistency of the combined score was adequate ($\alpha = .76$).

Adolescent Oppositional Behaviors

Nine items were used to construct adoptee oppositional behaviors: failing to obey rules, defiance, talking back, lying, having trouble with teachers, teasing, quarreling, temper tantrums, and threatening others. Internal consistency was good ($\alpha = .86$).

Adoptive Parent Disciplinary Practices

The adoptee completed the AEIII questionnaire which was developed by Berger, Knutson, Mehm, and Perkins (1988) to obtain retrospective descriptions of disciplinary experiences of adult respondents by soliciting reports of specific parental behaviors and subjective judgments of both harsh and positive treatments the adolescent directly experienced or observed. The AEIII contains 163 true-false items that were assigned to 15 different scales following the rational-statistical approach to questionnaire development (see Loevinger, 1957). The test-retest reliability of the AEIII and internal consistency of the scales has been established in psychometric evaluations of the instrument. Criterion validity of the AEIII was established initially by demonstrating that scales distinguished between abused and non-abused adolescents (Berger et al., 1988).

Two of the 15 scales were used for these analyses: reported harsh physical disciplinary practices directed toward the adoptee (e.g., When I was bad, my parents used to lock me in a closet, I never received any kind of injury from the discipline used by my parents, I was severely beaten by my parents, My parents

⁷ A cutoff of 2 was used for several reasons. Data from the Rochester Longitudinal Study (Sameroff *et al.*, 1997) show socio-emotional development among 4-year-olds begins to decline when at least 2 risk factors are present (see Fig. 22.2, Sameroff *et al.*, 1997). Analysis of adolescent data from the same studies showed a similar decline in functioning when 2 or more risk factors were present. These findings, in conjunction with the small sample size that would arise with a more stringent cutoff, justified the cutoff of 2 risk factors. We believe that this possibly liberal cutoff would, if anything, jeopardize the sensitivity of our design for assessing the effects of adversity on the proximal process.

Table I. Distribution of Adoptive Parent Risk Factors and Biological Parent Risk Factors Across Environmental Risk Groups

	Low environmental risk $(n = 84)$	Moderate environmental risk $(n = 65)$		
Environmental risk factors experienced by adoptive parents				
Alcohol problems	7 (8%)	20 (31%)		
Legal problems	0 (0%)	3 (5%)		
Depression	8 (9%)	47 (72%)		
Anxiety	13 (15%)	44 (68%)		
Drug abuse	0 (0%)	3 (5%)		
Other psychological problems	10 (12%)	31 (48%)		
Parents divorced before	0 (0%)	12 (19%)		
Adoptee age 18				
Biological parent risk				
Alcoholism	24 (28%)	26 (40%)		
Antisocial personality	23 (26%)	15 (23%)		
Drug abuse	25 (29%)	20 (31%)		
Criminality	14 (17%)	15 (23%)		
Total count score				
Mean	.99	1.23		
Standard deviation	1.38	1.51		

Note: Numbers in parentheses are percentages of adoptive parents positive for that behavior.

Table II. Zero-order Correlations Between All Study Variables for the Total Sample (n = 150)

Study variables	BPP	PhysD	PercD	CD	Agg	Gender	Mean	SD
Birth Parent Psychopathology (BPP)							.35	.48
Physical Discipline ^a (PhysD)	.26**						.45	.22
Perception of Discipline ^a (PercD)	.23**	.55***					.39	.30
Adolescent Conduct Behavior Problems ^a (CD)	.24**	.19*	.38***				.35	.37
Adolescent Oppositional Behavior Problems ^a (Agg)	.21*	.25**	.42***	.77***			.35	.44
Adoptee age	23**	12	08	12	06		24.90	6.02
Adoptee gender	.11	.20*	.13	.17*	.17*	02		

Note: Transformed due to non-normality [(log10 (variable + 1)]. Gender was coded as follows: 0 = females, 1 = males. * p < .05. ** p < .01. *** p < .001.

Table III. Zero-order Correlations of Study Variables in the Two Environmental Risk Groups

Study variables	BPP	PhysD	PercD	CD	OD	Age	Gende	er M	SD
1 Birth Parent Psychopathology (BPP)		.28*	.32*	.45***	.44***	20	.23	.39	.49
2 Physical Discipline ^a (PhysD)	.16		.69***	.29*	.35**	.17	.20	$.51^{b}$.23
3 Perception of Discipline ^a (PercD)	.07	.35**		.47***	.44***	.01	.14	.43	.34
4 Adolescent Conduct Behavior Problems ^a (CD)	.01	04	.25*		.76***	.10	.19	.30	.38
5 Adolescent Oppositional Behavior Problems ^a (OD)	.01	.08	.35**	.73***		.05	.22	.45	.45
6 Adoptee age	28*	35**	16	27*	12		.05	24.95	5.69
7 Adoptee gender	.06	.20	.14	.08	.09	11			
Mean (M)	.33	.41 ^b	.36	.31	.42	24.60			
Standard deviation (SD)	.47	.21	.27	.34	.40	6.48			

Note: Correlations for low environmental risk group (LER) are below the diagonal (n = 76). Correlations for moderate environmental risk group (MER) are above the diagonal (n = 57). Gender coded 0 = Females and 1 = Males. Means of the observed variables = intercept + slope * $M_{\text{latent variable}}$ + error.

^a Transformed due to non-normality [(log10 (variable + 1)]. ^bSignificant main effect of environmental risk.

^{*} p < .05. ** p < .01. *** p < .001.

used to hit me with something other than their hands when I did something wrong, My parents used to hit me with their hands, other than spanking) and perceived harshness of discipline directed at the adoptee (e.g., My parents used harsh discipline with me between the ages of 5 and 10, My parents were very strict disciplinarians, My parents' use of discipline was reasonable [negatively scored], My parents were inconsistent in their discipline of me. I never knew whether or not I would be punished for a particular behavior, I was mistreated by my parents). These scales were selected due to the association between hostile parenting and psychopathology in the adoptee's birth parent found by Ge *et al.*, (1996).

Analyses

Structural equation modeling was used to examine the presence of evocative biology-environment correlation and biology \times environment interaction (Ge *et al.*, 1996). The program, Amos 4.0 (Arbuckle, 1994–1999), was used to fit the models. Due to missing data for 13 of the 150 adoptee reports on parental discipline, full information maximum likelihood (FIML) estimates were computed (Amos, 1994-1999). Prior to conducting the SEM analyses, tests for univariate and bivariate normality were conducted for all study variables. The log transformation was used for both subscales of the AEIII and for both indicators of adolescent problem behaviors (e.g., oppositional and conduct disordered behaviors) due to non-normality. This reduced the skewness and kurtosis of each variable. Finally, because the strength of biological influences have been shown to depend on the type of behavior studied (Eley et al., 1999), the gender of the individual (Eley et al., 1999), and the time period from which the sample was recruited (Jacobson et al., 2000), all of the models tested included adoptee age and gender as exogenous predictors of both adoptee adolescent problem behaviors and parental discipline (Eley et al., 1999; Jacobson et al., 2000).

The first step in the analyses involved testing for evocative biology-environment correlation for the entire sample using the concept of *mediation* (Baron and Kenny, 1986). The conceptual model for testing evocative biology-environment correlation is presented in Figures 1A and 1B. The structural model for testing evocative biology-environment correlation consisted of three direct paths: 1) biological risk to harsh discipline (see path a, Fig. 1), 2) biological risk to adolescent problem behaviors (see path b, Fig. 1), and 3) adolescent problem behaviors to harsh discipline (see path c, Fig. 1). Two models were tested: a Direct Effects Model

(see Fig. 1A) and a Mediational/Evocative Model (see Fig. 1B). The Direct Effects Model estimated only two of the paths from the Mediational/Evocative Model; the path between biological risk and harsh discipline (see path a, Fig. 1A) and the path between biological risk and adolescent problem behaviors (see path b, Fig. 1A). The adolescent behavior-discipline relationship was modeled as a non-causal covariance term in the Direct Effects model.

In the Mediational/Evocative Model, the path between adolescent problem behaviors and harsh discipline was changed from a covariance term to a directional association (see path c, Fig. 1B). If the size of the path coefficient for the relation between biological risk and harsh discipline estimated in the Direct Effects Model was substantially reduced after taking into account the association between adolescent behaviors and harsh discipline in the Mediational/Evocative Model, then the data would be consistent with, at minimum, partial mediation (Baron and Kenny, 1986).

The second step of the analyses involved testing for moderation of the above Mediational/Evocative Model by estimating the model separately for each environmental risk group (see Fig. 1C). We hypothesized that the paths from biological risk to both adoptee adolescent behaviors and adoptive parent harsh discipline would differ significantly between the two environmental risk groups. Specifically, biological effects would be found in the moderate environmental risk group and not the low environmental risk group.

Moderation can be determined two ways. First, moderation is supported if the requirements of mediation are satisfied in one group but not the other. Second, moderation can be shown by testing the difference in both the Total and Indirect Effects of biological risk on harsh discipline. The Total Effect estimate is comprised of the direct path from biological risk to harsh discipline (see path a, Fig. 1B) and the indirect path from biological risk to harsh discipline via adolescent problem behaviors (see paths b, c Fig. 1B). The Indirect Effect estimate is derived solely from the latter path. The estimated indirect effects, variances, and covariances, as fit in Amos, were used for these analyses. Variances for Total and Indirect Effects and their between-group differences, were calculated via the multivariate Delta Method (cf. Bollen, 1989). We then approximated p-values from the subsequent z-scores.

Prior to testing for moderation, we tested for measurement equivalence across the two environmental risk groups to insure that any differences in the structural model could not be attributed to differences in the underlying measurement model (Bollen, 1989). Three

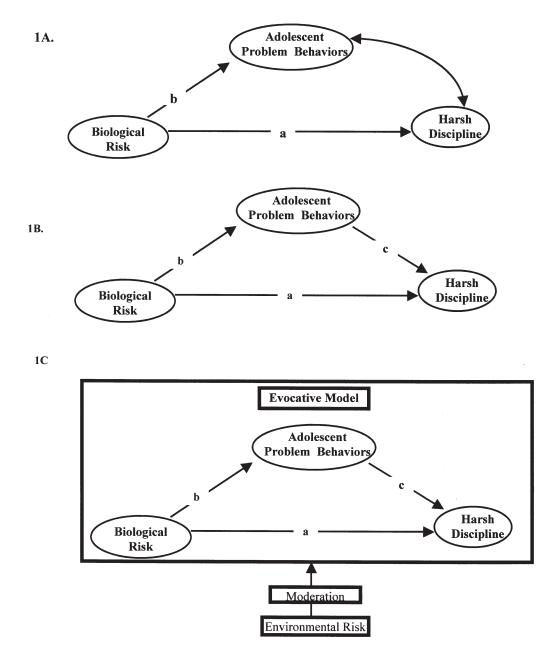


Fig. 1. A, Conceptualization of Direct Effects model. B, Conceptualization of Mediational Effects model. C, Conceptualization of the Bioecological Model.

models were tested and compared using chi-square tests of the difference in lack of fit. The first model estimated all parameters separately for each environmental group. The second model equated the intercepts of the measured variables (i.e., harsh physical discipline, perceived physical discipline, adolescent oppositional behaviors, and adolescent conduct-disordered behaviors) across environmental risk status. For measured

variables, the intercepts refer to the expected level if the associated latent variable has a value of zero and is equal to $x = \text{intercept} + \text{slope*}\ M_{\text{Latent Variable}} + \text{error}$. The third model equated both the slopes (lambdas) and intercepts across environmental risk status. The lambdas represent the expected change in the observed variable per standard deviation change of the associated latent variable.

RESULTS

Testing for Evocative Effect

The entire sample was used to test for the presence of mediation/evocation. Zero-order correlations between all study variables are presented for the total sample in Table II. Preliminary examination of the correlation matrix shows support for two basic requirements of mediation: 1) significant associations between biological risk and both indicators of adolescent problem behaviors and 2) significant associations between biological risk and both indicators of adoptive parent harsh discipline (see Fig. 1A).

Findings from the structural equation modeling analysis showed the following significant paths for the Direct Effects model: 1) adoptee sex significantly predicted adolescent problem behaviors, $\beta = .17$, critical ratio (CR) = 2.02, with males having higher rates of problem behaviors, 2) biological risk significantly predicted adolescent problem behaviors, $\beta = .23$, CR = 2.57, with adoptees having a biological risk for psychopathology showing higher rates of adolescent problem behaviors, and 3) biological risk significantly predicted adoptive parent harsh discipline, $\beta = .25$ CR = 2.35, with harsher discipline directed towards adoptees who had a biological risk for psychopathology.

For the Mediational/Evocative Model (see Fig. 1B), the latent variable, adolescent problem behaviors, was added as a predictor of adoptive parent harsh discipline resulting in a significant path between adolescent problems behaviors and harsh parental discipline, $\beta = .46$, CR = 3.50, and the reduction of the path between biological risk and harsh discipline to nonsignificance, $\beta = .14$, CR = 1.50. The overall model showed a good fit, χ^2 (7) = 8.80, p > .10. These findings are consistent with the presence of mediation/evocation.

Testing for Moderation of Mediational/Evocative Effect

The final analyses tested for moderation of the above Mediational/Evocative effect. In order to test for moderation, we divided the sample into low and moderate environmental risk groups. Zero-order correlations are presented separately for each environmental risk group in Table III. Again, preliminary examination of the correlational tables suggests that mediation/evocation is possible only in the moderate environmental risk group. Biological risk predicts both adolescent problem behaviors and adoptive parent harsh discipline only in the moderate environmental risk group.

The first step of these analyses involved testing for measurement equivalence across the environmental risk groups. 8 The results from the analyses testing for latent model measurement invariance across the two levels of environmental risk status showed nonsignificant differences for the lambdas (regression coefficients representing the association between the observed indicators and the latent variable). The likelihood difference in goodness-of-fit was 2.10, 2 df, p = .35. Invariance was also found when the intercepts were additionally equated across the two environmental risk groups (likelihood difference = 4.33, 2 df, p = .20). The overall test for measurement equivalence (equal lambdas and intercepts) was non-significant (total likelihood change = 5.31, 4 df, p = .26). The final model had an acceptable goodness-of-fit (residual likelihood = 24.67, 19 df, p = .17) supporting that the measurement models did not differ between the environmental risk groups.

The second step looked for both substantive and statistical support for moderation. Substantive support for mediation was found due to the lack of associations between biological risk and both adolescent problem behaviors and adoptive parent harsh discipline in the low environmental risk group (Fig. 2). These paths are significant for the moderate environmental risk group (Fig. 3). With regard to statistical support, a significant difference for the Indirect Effect estimates was found (Table IV). Although the magnitude of the difference for the Total Effect estimates was similar to that found for the Indirect Effect estimates, the difference was not statistically significant (see Table IV). The significant difference between the two environmental risk groups for the Indirect Effect estimates suggests that adolescent problem behaviors mediate the association between biological risk and adoptive parent harsh discipline only when additional environmental stressors are pre-

⁸ For mathematical identifiability, we set the intercepts of the latent variables equal to 0 in the group with low environmental risk. The intercept in the high environmental risk group represents the estimated mean difference of the latent variable due to being in that group after controlling for biological risk, gender, and adoptee age.

The fact that the *intercepts* of the observed variables, specifically physical discipline, were not significantly different across the environmental risk groups in the structural model but the *observed* means tested in Table III were significantly different is not contradictory. The *observed* mean is derived from the following equation: *intercept* + slope (or factor loading) *M_{latent variable} + error. Two of the components of the cited equation are invariant. As a result, the higher levels of physical discipline in the moderate environmental risk group (as indicated in Table III) results from differences in the means of the latent variables and the error terms for the observed variables within each environmental risk group.

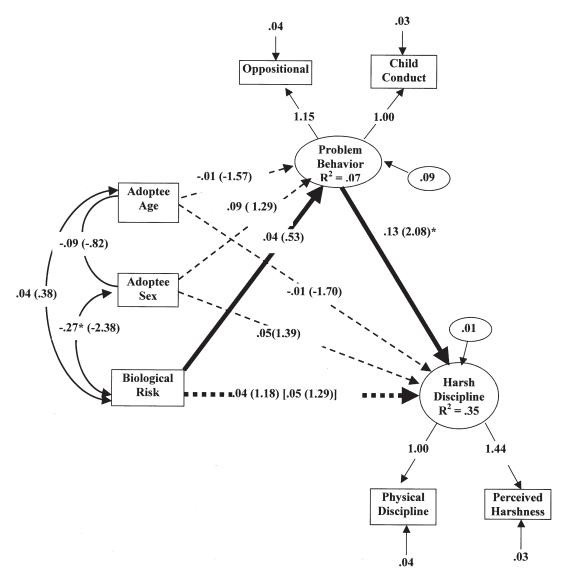


Fig. 2. Unstandardized path coefficients for evocative model for the low environmental risk group. Values in parentheses are the critical ratios for the parameter estimate. Lambdas and intercepts are equated across environmental risk groups. χ^2 (19) 24.67, p = .172, RMSEA = .05. *Critical Ratio (CR) > 1.96. *Note*. Nonsignificant paths are represented by dotted lines. Heavier lines identify theoretically relevant paths. Numbers in brackets represent values from the Direct Model in which the path between adolescent behaviors and harsh discipline was represented as a covariance term.

sent. In other words, the findings suggest that adoptive parents react to the biological predisposition and subsequent manifestation of similar behaviors by the adoptee to a greater degree when under stress.

DISCUSSION

To our knowledge, this study represents one of the first attempts to test concurrently biology-environment correlation and biology-environment interaction using a

biologically informed adoption study. Although evidence is beginning to emerge in support of evocative biology-environment correlation (Ge *et al.*, 1996; O'Connor *et al.*, 1998), the sensitivity of this process to contextual environmental factors has not been addressed by behavior geneticists (Bronfenbrenner and Ceci, 1994; Rutter and Pickles, 1991; Rutter *et al.*, 1997). We conclude that contextual factors do impact the degree to which biological predispositions influence parental behavior. This conclusion is based on the fact that mediation (i.e., evoca-

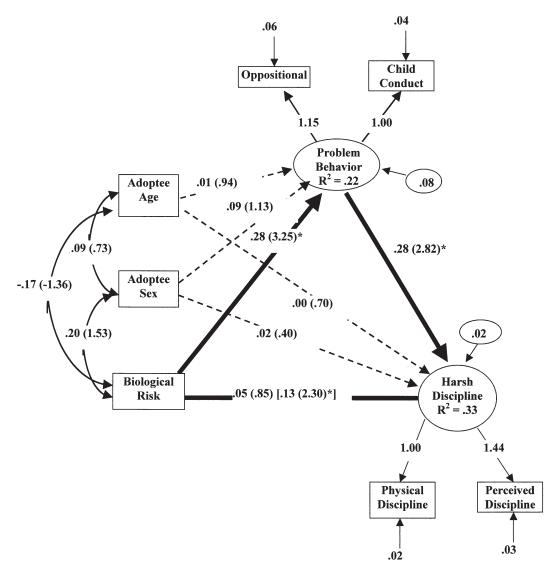


Fig. 3. Unstandardized path coefficients for evocative model for the moderate environmental risk group. Values in parentheses are the critical ratios for the parameter estimate. Lambdas and intercepts are equated across environmental risk groups. χ^2 (19) 24.67, p = .172, RMSEA = .05. *Critical Ratio (CR) > 1.96. *Note*. Nonsignificant paths are represented by dotted lines. Heavier lines identify theoretically relevant paths. Numbers in brackets represent values from the Direct Model in which the path between adolescent behaviors and harsh discipline was represented as a covariance term.

tion) was confirmed only in the moderate risk environmental group and a significant difference was found for the Indirect Effect estimates across environmental risk.

These findings also suggest that etiologically different processes may be at work when considering environments of varying contextual risk. For example, it is possible that in the low environmental risk group, depending on their biological risk, the *adoptee* may have reacted differentially to *parental* treatment. In

contrast, the *adoptive* parents may have reacted differentially to *adoptee* behaviors in the moderate environmental risk group. Support for these disparate processes is suggested by the significant difference for the Indirect Effect estimates but the nonsignificant difference for the Total Effect estimates. The former test confirmed that adolescent behavior elicited harsher discipline from the adoptive parents in the moderate environmental risk group. The latter test, which is equivalent

Effect	Nonstandardized effect	Std Error	Z-score	Approx. <i>P</i> -value
Low environmental risk				
Total effect	.0496	.0384	1.29	.20
Indirect effect	.0054	.0105	.52	.61
Moderate environmental risk				
Total effect	.1272	.0552	2.30	.02
Indirect effect	.0786	.0361	2.18	.03
Group difference (moderate-low)				
Total effect	.0780	.0660	1.17	.24
Indirect effect	.0731	.0375	1.95	.05*

Table IV. Direct and Indirect Effect Estimates for Moderated Mediation Model

Note: The Total Effect is equivalent to testing the significance of the direct path between biological risk and adoptive parent harsh discipline in the nonmediational model. The Direct Effect was not tested since it is nonsignificant by definition in the mediational model. Finally, the effects of adoptee age and gender do not contribute to the direct and indirect effects of biological risk on harsh discipline since they are exogenous variables and are merely correlated with biological risk.

to testing the direct association between biological risk and adoptive parent harsh discipline, suggests that some biological association between biological risk and discipline exists regardless of environmental risk. We suggest that although the association between biological risk and adoptive parent harsh discipline did not differ statistically across environmental risk, the quality of the association may be different. In the low environmental risk group, biological risk did not predict adolescent problem behaviors. However, adolescent behaviors and harsh parental discipline were significantly associated. It is possible that adoptees with a biological risk for psychopathology reacted to harsh parental discipline with increased problem behaviors. In contrast, harsh discipline may have reduced problem behaviors among adoptees without any biological risk for psychopathology. The differential reaction of the adoptee could have resulted in an association between the latter and parental discipline in the low environmental risk group.

In the moderate environmental risk group, the association between biological risk and parental harsh discipline was accounted for by the adoptees' expression of problem behaviors or, in other words, evocation of parental behavior. The fact that prior research has shown that parents under stress tend to react more harshly to their children's behavior supports these conclusions (Bates *et al.*, 1999; Coyne and Downey, 1991; Downey and Coyne, 1990; Greenwald *et al.*, 1997; Susman *et al.*, 1985; Zahn-Waxler *et al.*, 1990). These conclusions also suggest that the processes hypothesized are most likely reciprocal in nature with child behavior influencing parental behavior and vice versa. It

is the origination of the mutually reinforcing process (parent versus child) that seems to differ between the two environmental groups.

Concordance of Findings with Previous Studies

The findings from this study differ from previous studies in which biological contributions to parental warmth and support, but not parental control, have been found (Elkins *et al.*, 1997; O'Connor *et al.*, 1998; Rowe, 1981, 1983). Our results, however, are consistent with those found by Ge *et al.* (1996) in which significant evocative effects were found for irritable and hostile parent-child interactions, which is notable given that our study was retrospective and the Ge *et al.* (1996) study used observational measures to assess parent-child interaction.

One factor that could account for differences between the findings of Cadoret et al. (1995a, 1995b) and Ge et al. (1996) and those of Elkins et al. (1997) and O'Connor et al. (1998) is the sampling scheme. The recruitment scheme for the former set of studies was as follows: no evidence of psychiatric disorder was found for the birth parents of the control adoptees, whereas institutional records showed evidence of either substance abuse or antisocial personality for birth parents of the proband adoptees. The stringent selection process of Cadoret et al. (1995a, 1995b) and Ge et al. (1996) might have created distributions that increased the likelihood of detecting significant interactions, including biology by environment interactions (McClelland and Judd, 1993), while findings from other researchers have been mixed with respect to problem behaviors (DeFries

^{*} p < .05. ** p < .01.

et al., 1994). Another possibility is that the procedure for selecting adoptive families could have resulted in a restriction of environmental pathology thereby inflating the relative importance of biological estimates (Stoolmiller, 1999). We have not attempted any quantitative correction for this possibility, but do recognize it as a caveat in generalizing our findings to non-adoption populations.

Implications for Behavioral Genetic Research

Several implications for behavioral genetic research arise from these findings. First, the presence of a biology-environment interaction can lead to erroneous conclusions regarding the importance of biological influences in development. For example, the zero-order correlations between biological risk and indicators of adolescent problem behaviors for the total sample were small to moderate and explained around 6 percent of the variance leading to the conclusion that biology played a small role in problem behavior. Estimating these same correlations separately for the low risk and moderate-risk groups, however, showed that biological risk explained almost 20% of the variance in adolescent problem behaviors for the moderate environmental risk group versus less than 1% for the low environmental risk group; a substantial improvement in variance explained.

A second implication deals with the role of process and the manifestation of biological propensities as observable behavior. Although researchers recognize the necessity of moving beyond partitioning of variance, few have attempted to examine the processes that are involved in translating biological diatheses into observed behavior using biologically informed data (Bronfenbrenner and Ceci, 1994; Rutter and Pickles, 1991; Rutter et al., 1997). The fact that we found a biological association between biological risk for psychopathology and adolescent problem behaviors in the moderate environmental risk group could be due to an ongoing reciprocating evocative process in which the adolescent acted out and the parent reacted with harsh discipline. This may, in turn, result in further acting out by the adolescent. The reason that a biological component in adolescent problem behaviors was not found in the low environmental risk group could be due to the fact that this mutual process does not occur frequently enough to reinforce the biological predispositions of the adoptee to the same degree.

Finally, the moderation of evocative biologyenvironment correlations and the possibility that the processes relevant to particular behaviors depend on contextual environmental factors introduces yet another challenge to behavioral genetics. Our model suggests that biology-environment correlation is, in fact, moderated by contextual environmental factors in this case, psychopathology in an adoptive parent. In contrast to the standard model, which assumes that biology-environment correlation does not vary across different environments (Scarr and McCartney, 1983), 10 this phenomenon adds yet another dimension to the developmental model. We suggest that this moderation is due to different processes occurring in different environments. In other words, the contextual environment influences the degree to which biological influences play a significant role in shaping behavior representing another type of biology-environment interaction. Further research should explore other mechanisms that might mediate even the impact of parental psychopathology on parentchild interactions (e.g., parental warmth).

Future Research

This study represents another intermediate step in understanding biology-environment interaction and correlation and their roles in the development of psychopathology. The next step would be to conduct a prospective, longitudinal study that would address many of the limitations of this study. For example, multiple measurement points would allow the determination of the age when the processes discussed are established, when the processes begin to influence behavior, the directional nature of the process (e.g., reciprocal), as well as the degree of exposure necessary for the processes to affect behavior. Because our data were cross-sectional and retrospective, we could not identify the onset age of both parental behaviors and adoptee behaviors. For a subset of subjects (n = 41) in the moderate environmental risk group, we were able to determine whether parental psychopathology was present while the adoptee was under the age of 18. If we limited the correlational analyses to those subjects, then the associations between biological parent psychopathology and adoptive parent discipline increased in strength (physical

¹⁰ The distinction between "good enough" and "not good enough" environments is not being referred to here. In fact, the findings here suggest that a "good enough" environment, as indicated by an absence of multiple environmental risk factors, does not allow biology to express itself to a greater degree. Perhaps, the concept of "good enough" environment and impact of biological expression should be tempered by the nature of the outcome examined (e.g., psychopathology versus "normative" behavior).

discipline = .47, p < .01, Standard Error of Estimate [SEE] = .18, 95% CI = .19, .68 and harsh discipline = .42, p < .01, SEE = .31, 95% CI = .13, .65). The increase in the strength of the correlations when controlling for whether the adoptee was in the home at the time of adoptive parent problems further reinforces the potential moderating impact of additional parental stress on interactions with their children and the need for prospective longitudinal data.

We were also unable to control for potential response bias on adoptees' ratings of parental behavior (Prescott *et al.*, 2000). A prospective study would allow more accurate and sensitive evaluation of the identified processes over time by including multiple reporters via the use of observational data, as well as allow for separation of the potential impact of biologically influenced traits on evaluations of the environment.

Furthermore, additional information on the specific source of biological or genetic influence, including prenatal and perinatal factors, could also be collected with a prospective study. Research has shown sex-specific biological effects, with biological influences on behavior dependent on the parent from which the biological material originated (Durcan and Goldman, 1993). Finally, sex differences in behaviors of both children and parents could be examined. Differences in parental treatment of males and females could be compared, as well as sex differences in other important environmental influences during childhood (e.g., school, peers) (Maccoby, 1998; Maccoby and Martin, 1983).

Conclusions

The above list is not exhaustive, but rather, demonstrates the complex issues that could be addressed with a prospective adoption study. Despite the limitations of our study, we have demonstrated that the context in which processes involved in the manifestation of biological predispositions occur should be taken into consideration. Specifically, as predicted by Bronfenbrenner and Ceci's (1994) Bioecological model, the manifestation of problem behaviors was greatest when the parentchild interactions of interest reinforced the biological tendency for problem behavior and the additional risk factors were present in the environmental context in which these interactions occurred. This is a step beyond the traditional conceptualization of biology-environment interaction in which behaviors rather than processes are predicted (Cadoret and Cain, 1981; Cadoret et al., 1983; 1995b; Hershberger, 1994; Plomin et al., 1997). A greater understanding of these processes should contribute greatly to our ability to intervene and prevent the development of problematic child behaviors.

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REFERENCES

- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders*, Fourth Edition. Washington, D.C.: American Psychiatric Association.
- Anderson, K. E., Lytton, H., and Romney, D. (1986). Mothers' interactions with normal and conduct-disordered boys: Who affects whom? *Dev. Psychol.* 22:604–609.
- Arbuckle, J. L. (1994–1999). AMOS 4.0. Chicago: Smallwaters Corporation.
- Bates, J., Pettit, G. S., Dodge, K. A., and Ridge, B. (1999). Interaction of temperamental resistance to control and restrictive parenting in the development of externalizing behavior. *Dev. Psychol.* 34:982–995.
- Baron, R. M., and Kenny, D. A. (1986). The moderator-mediator distinction in social psychological research: Conceptual, strategic, and statistical considerations. J. Pers. Soc. Psychol. 51:1173–1182.
- Bell, R. Q. (1968). A reinterpretation of the direction of effects in studies of socialization. *Psychol. Rev.* **75**:81–95.
- Bell, R. Q. (1971). Stimulus control of parent or caretaker behavior by offspring. *Dev. Psychol.* **4**:63–72.
- Bell, R. Q. (1979). Parent, child, and reciprocal influences. Am. Psychol. 34:821–826.
- Bell, R. Q., and Chapman, M. (1986). Child effects in studies using experimental or brief longitudinal approaches to socialization. *Dev. Psychol.* 22:595–603.
- Berger, A., Knutson, J., Mehm, J., and Perkins, K. (1988). The self-report of punitive childhood experiences of young adults and adolescents. *Child Abuse & Negl*, **12**:251–262.
- Bollen, K. A. (1989). Structural equations with latent variables. New York: John Wiley & Sons.

- Bronfenbrenner, U., and Ceci, S. (1994). Nature-nurture re-conceptualized in developmental perspective: A bio-ecological model. *Psychol. Rev.* **101**:568–586.
- Burgess, R. L., and Conger, R. D. (1978). Family interaction in abusive, neglectful, and normal families. *Child Dev.* **49**:1163–1173.
- Cadoret, R. J., Yates, W., Troughton, E., Woodworth, G., and Stewart, M. (1995a). Adoption study demonstrating two genetic pathways to drug abuse. *Arch. Gen. Psychiatry* 52:42–52.
- Cadoret, R. J., Yates, W., Troughton, E., Woodworth, G., and Stewart, M. (1995b). Genetic-environmental interaction in the genesis of aggressivity and conduct disorders. *Arch. Gen. Psychiatry* 52:916–924.
- Cadoret, R. J., and Cain, C. A. (1981). Genetic-environmental interaction in adoption studies of antisocial behavior. In C. Perris, G. Struwe, and B. Jansson (eds.), *Biological psychiatry*. Elsevier, North Holland: Biomedical Press.
- Cadoret, R. J., Cain, C. A., and Crowe, R. R. (1983). Evidence for gene-environment interaction in the development of adolescent antisocial behavior. *Behav. Genet.* 13:301–310.
- Caspi, A., Elder, G. H., and Bem, D. J. (1987). Moving against the world: Life course patterns of explosive children. *Dev. Psychol.* 23:308–313.
- Conger, R. D., Conger, K. J., Elder, G. H., Jr., Lorenz, F. O., Simons, R. L., and Whitbeck, L. B. (1992). A family process model of economic hardship and adjustment of early adolescent boys. *Child Dev.* 63:526–541.
- Conger, R. D., Lorenz, F. O., Elder, G. H., Jr., Melby, J., N., Simons, R. L., and Conger, K. J. (1991). A process model of family economic pressure and early adolescent alcohol use. *J. Early Adolesc*. 11:430–449.
- Coyne, J. C., and Downey, G. (1991). Social factors and psychopathology: Stress, social support, and coping processes. In M. R. Rosenzweig and L. W. Porter (eds.), *Annual review of psychology* (Vol. 42, pp. 401–425). Palo Alto, CA: Annual Reviews.
- Deater-Deckard, K., and Dodge, K. (1997). Externalizing behavior problems and discipline revisited: Nonlinear effects and variation by culture, context, and gender. *Psychol. Inquiry* **8**:161–175.
- DiLalla, L., and Gottesman, I. (1991). Biological and genetic contributors to violence-Widom's untold tale. *Psychol. Bull.* **109**:125–129.
- Dodge, K. A. (1990). Nature versus nurture in childhood conduct disorder: It is time to ask a different question. *Dev. Psychol.* 26:698-701.
- Dodge, K. A., Pettit, G. S., Bates, J. E., and Valente, E. (1995). Social information-processing patterns partially mediate the effect of early physical abuse on later conduct problems. *J. Abnormal Psychol* 104:632–643.
- Downey, G., and Coyne, J. C. (1990). Children of depressed parents: An integrative review. *Psychol. Bull.* **108**:50–76.
- Durcan, M. J., and Goldman, D. (1993). Genomic imprinting: Implications for behavioral genetics. *Behav. Genet.* **23**:137–143.
- Elder, G., and Caspi, A. (1988). Economic stress in lives: Developmental perspectives. J. Soc. Issues 44:25–45.
- Eley, T. C., Lichtenstein, P., and Stevenson, J. (1999). Sex differences in the etiology of aggressive and nonaggressive antisocial behavior: Results from two twin studies. *Child Dev.* **70**:155–168.
- Elkins, I. J., McGue, M., and Iacano, W. G. (1997). Genetic and environmental influence on parent-son relationships: Evidence for increasing genetic influence during adolescence. *Dev. Psychol.* 33:351–363.
- Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Jr., Winokur, G., and Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. Arch. Gen. Psychiatry 35:837–844.
- Ge, X., Conger, R. D., Cadoret, R. J., Neiderhiser, J. M., Yates, W., Troughton, E., and Stewart, M. A. (1994). The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Dev. Psychol.* 31:574–589.

- George, C., and Main, M. (1979). Social interactions of young abused children: Approach-avoidance and aggression. *Child Dev.* 50:306–318.
- Greenwald, R. L., Bank, L., Reid, J. B., and Knutson, J. F. (1997).
 A discipline-mediated model of excessively punitive parenting.
 Aggress. Behav. 23:259–280.
- Hershberger, S. L. (1994). Genotype-environment interaction and correlation. In J. C. DeFries, R. Plomin, and D. W. Fulker (eds.). Nature and nurture during middle childhood (pp. 281–294). Oxford, UK: Blackwell.
- Jacobson, K. C., Prescott, C. A., Neale, M. C., and Kendler, K. S. (2000). Cohort differences in genetic and environmental influences on retrospective reports of conduct disorder among adult male twins. *Psychol. Med.* 30:775–787.
- Larzalere, R. E., and Patterson, G. R. (1990). Parental management: Mediator of the effect of socioeconomic status on early delinquency. *Criminology* 28:301–323.
- Lempers, J., Clark-Lempers, D., and Simons, R. L. (1989). Economic hardship, parenting, and distress in adolescence. *Child Dev.* 60:25–39.
- Lytton, H. (1982). Two-way influence processes between parents and child—when, where, how? Can. J. Behav. Sci. 14:259–275.
- Lytton, H. (1990). Child and parent effects in boys' conduct disorder: A reinterpretation. Dev. Psychol. 26:683–697.
- Maccoby, E. E. (1992). The role of parents in the socialization of children: An historical overview. *Dev. Psychol.* 28:1006– 1017.
- Maccoby, E. E. (1998). The two sexes: Growing up apart, coming together. Cambridge, MA: The Belknap Press of Harvard University Press.
- Maccoby, E. E., and Martin, J. M. (1983). Socialization in the context of the family: Parent-child interaction. In E. M. Hetherington (ed.), Socialization, personality, and social development (Vol. IV, Chap. 1). In Handbook of Child Development series (G. H. Mussen, ed.). New York: Wiley.
- McClelland, G. H., and Judd, C. M. (1993). Statistical difficulties of detecting interactions and moderate effects. *Psychol. Bull.* 114:376–390.
- Meyer, J. M., Rutter, M., Silberg, J. L., Maes, H. H., Simonoff, E., Shilladyy, L. L., Pickles, A., Hewitt, J., and Eaves, L. J. (2000). Familial aggregation for conduct disorder symptomatology: the role of genes, marital discord, and family adaptability. *Psychol. Med.* 30:759–774.
- O'Connor, T. G., Deater-Deckard, K., Fulker, D., Rutter, M., and Plomin, R. (1998). Genotype-environment correlations in late childhood and early adolescence: Antisocial behavioral problems and coercive parenting. *Dev. Psychol.* 34:970–981.
- Patterson, G. R. (1982). A social learning approach, Vol. 3: Coercive family process. Eugene, OR: Castalia Publishing Company.
- Patterson, G. R. (1983). Stress: A change agent for family process. In N. Garmezy and M. Rutter (eds.), Stress, coping and development in children (pp. 235–264). New York: McGraw-Hill.
- Patterson, G., Reid, J., and Dishion, T. (1992). Antisocial boys. Eugene, OR: Castalia Publishing Company.
- Plomin, R., DeFries, J. C., and Loehlin, J. C. (1977). Genotypeenvironment interaction and correlation in the analysis of human behavior. *Psychol. Bull.* 84:309–322.
- Plomin, R., DeFries, J., and Fulker, D. (1994). *Nature and nurture during middle childhood*. Blackwell: Oxford, England.
- Prescott, A., Bank L., Reid, J. B., Knutson, J. F., Burraston, B. O., and Eddy, J. M. (2000). The veridicality of punitive childhood experiences reported by adolescents and young adults. *Child Abuse Neglect* 24(3):411–423.
- Reid, J. B., Patterson, G. R., and Loeber, R. (1982). The abused child: Victim, instigator, or innocent bystander. In D. Berstein (ed.), Response structure and organization. Lincoln, NE: University of Nebraska Press.

- Reidy, T. J. (1977). The aggressive characteristics of abused and neglected children. *J. Clin. Psychol.* **33**:1140–1145.
- Robins, L., Helzer, J., Cottler, L., and Goldring, E. (1989). NIMH diagnostic interview schedule, version 3, revised (DIS-III-R).St. Louis, MO: Washington University School of Medicine.
- Rowe, D. (1981). Environmental and genetic influences on dimensions of perceived parenting: A twin study. *Dev. Psychol.* 17:203–208.
- Rowe, D. (1983). A biometrical analysis of perceptions of family environment: A study of twin and singleton sibling kinships. *Child Dev.* 54:416–423.
- Rutter, M. (1997). Implications of genetic research for child psychiatry. *Can. J. Psychiatry* **42**:569–576.
- Rutter, M., and Pickles, A. (1991). Person-environment interactions:
 Concepts, mechanisms, and implications for data analysis. In
 T. D. Wachs and R. Plomin (eds.), Conceptualization and measurement of organism-environment interaction. Washington,
 D.C.: American Psychological Association.
- Rutter, M., Dunn, J., Plomin, R., Simonoff, E., Pickles, A., Maughan, B., Ormel, J., Meyer, J., and Eaves, L. (1997). Integrating nature and nurture: Implications of person-environment correlations and interactions for developmental psychopathology. *Dev. Psychol.* 9:335–364.
- Rutter, M., Macdonald, H., Couteur, A. L., Harrington, R., Bolton, P., and Bailey, A. (1990). Genetic factors in child psychiatric disorders II. Empirical findings. J. Child Psychol. Psychiatry 31:39–83.

- Salzinger, S., Kaplan, S., Pelcovitz, D., Samit, C., and Krieger, R. (1984). Parent and teacher assessment of children's behavior in maltreating families. J. Am. Acad. Child Psychiatry 23:458–464.
- Sameroff, A. J., Seifer, R., and Bartko, W. T. (1997). Environmental perspectives on adaptation during childhood and adolescence. In S. Luthar, J. A. Burack, D. Cicchetti, and J. R. Weiszeds.), Developmental psychopathology: Perspectives on adjustment, risk, and disorder. Cambridge, UK: Cambridge United Press.
- Scarr, S., and McCartney, K. (1983). How people make their own environments: A theory of genotype → environment effects. *Child Dev.* **54**:424–435.
- Stoolmiller, M. (1999). Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. *Psychol. Bull.* 125:392–409.
- Susman, E., Trickett, P., Iannotti, R., Hollenbeck, B., and Zahn-Waxler, C. (1985). Child-rearing patterns in depressed, abusive, and normal mothers. *Am. J. Orthop.* **55**:237–251.
- Taplin, P. S., and Reid, J. B. (1977). Changes in parent consequences as a function of family intervention. J. Consult. Clinical Psychol. 45:973–981.
- Zahn-Waxler, C., Ianotti, R. J., Cummings, E. M., and Denham, S. (1990). Antecedents of problem behaviors in children of depressed mothers. *Dev. Psychopathol.* 2:271–291.

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