
Domestic violence is associated with environmental suppression of IQ in young children

KARESTAN C. KOENEN,^a TERRIE E. MOFFITT,^{b,c} AVSHALOM CASPI,^{b,c}
ALAN TAYLOR,^b AND SHAUN PURCELL^b

^aNational Child Traumatic Stress Network, Boston University Medical Center; ^bInstitute of Psychiatry, King's College London; and ^cUniversity of Wisconsin–Madison

Abstract

Research suggests that exposure to extreme stress in childhood, such as domestic violence, affects children's neurocognitive development, leading to lower intelligence. But studies have been unable to account for genetic influences that might confound the association between domestic violence and lower intelligence. This twin study tested whether domestic violence had environmentally mediated effects on young children's intelligence. Children's IQs were assessed for a population sample of 1116 monozygotic and dizygotic 5-year-old twin pairs in England. Mothers reported their experience of domestic violence in the previous 5 years. Ordinary least squares regression showed that domestic violence was uniquely associated with IQ suppression in a dose–response relationship. Children exposed to high levels of domestic violence had IQs that were, on average, 8 points lower than unexposed children. Structural equation models showed that adult domestic violence accounted for 4% of the variation, on average, in child IQ, independent of latent genetic influences. The findings are consistent with animal experiments and human correlational studies documenting the harmful effects of extreme stress on brain development. Programs that successfully reduce domestic violence should also have beneficial effects on children's cognitive development.

More than 10 million children in the United States are exposed to violence between their parents each year (Straus, 1992). Rates of intimate partner violence are highest among women and men in their 20s, which suggests that young children are at high risk of expo-

sure. In fact, over 40% of all households where intimate partner violence occurs contain children under 12 (Rennison & Welchans, 2000). In the present study, we examine the association between domestic violence and children's cognitive development.

Although studies have documented a negative correlation between domestic violence and children's cognitive development in general, the specific relationship between domestic violence and IQ has received little attention (Kolbo, Blakely, & Engleman, 1996). Of the nine studies that have specifically exam-

We are grateful to the study mothers and fathers, the twins, and the twins' teachers for their participation. Our thanks go to Michael Rutter and Robert Plomin for their contributions, to Hallmark Cards for their support, to the anonymous reviewers for their thoughtful suggestions, and to members of the Environmental Risk Team for their dedication, hard work, and insights. Karestan C. Koenen was supported in part by a grant from the Columbia Center for Youth Violence Prevention and a National Research Service Award (NRSA) postdoctoral training grant in psychiatric epidemiology. Terrie E. Moffitt is the recipient of a Royal Society–Wolfson Research Merit Award. The Environmental Risk Longitudinal Twin Study is funded by the Medical Research Council.

Address correspondence and reprint requests to: Karestan C. Koenen, Center for Medical and Refugee Trauma, National Child Traumatic Stress Network, Department of Child & Adolescent Psychiatry, Boston University Medical Center, Dowling One North, One Boston Medical Center Place, Boston, MA 02118.

ined the relationship between domestic violence and children's cognitive development, eight found a negative correlation on a range of outcomes such as school performance, basic skills, and verbal abilities (Huth-Bocks, Levendosky, & Semel, 2001; Kerouac, Taggart, Lescop, & Fortin, 1986; Moore, Galcius, & Pettican, 1981; Pfouts, Schopler, & Henley, 1982; Rousaville & Weissman, 1977–1978; Stagg, Wills, & Howell, 1989; Westra & Martin, 1981; Wolfe, Zak, Wilson, & Jaffe, 1986), although one study found no significant differences on cognitive measures between exposed children and a control group (Christopoulos et al., 1988). Other studies have documented the adverse impact of child maltreatment and exposure to violence (both of which include exposure to domestic violence) on IQ, but these studies did not look specifically at the effects of domestic violence (e.g., Carrey, Butter, Persinger, & Bialik, 1995; Delaney-Black et al., 2002).

Despite the weight of positive findings, it is difficult to draw firm conclusions about the possible harmful effects of domestic violence on children's cognitive development because of five methodological limitations. First, only three of the previous studies contained a well-matched comparison group of unexposed children. Second, few studies used standardized measures of cognitive development. Third, most previous studies relied on clinically identified samples, which can sometimes be biased and unrepresentative because a selective subset of cases comes to clinical attention. Fourth, exposure to domestic violence is statistically associated with increased risk of exposure to child maltreatment (Moffitt & Caspi, 1998); by not accounting for this association, previous research has not established whether domestic violence has unique consequences for child outcomes. Fifth, children exposed to violence often have emotional and behavioral problems (Grych & Fincham, 1990), which could interfere with valid administration of the IQ test and thereby create a spurious association between violence exposure and IQ. Studies that employ a population-based sampling frame, use standard well-characterized measures of domestic violence and cognitive development, and account for

the potential confounding effects of other variables such as child maltreatment and child behavior problems are needed to provide definitive evidence about the negative effects of domestic violence on children's cognitive development. Therefore, the first goal of the present study is to examine whether exposure to domestic violence has a unique, negative association with children's IQ in a population-based sample that has not been clinically ascertained.

A sixth, conceptual, limitation of past research is that the observed association between children's exposure to extreme stressors, such as domestic violence, and low IQ has been *assumed* to be environmentally mediated. However, it is not correct to presume that the correlation between domestic violence and IQ indicates causation, because a third variable may explain the domestic violence–IQ association: children whose parents engage in domestic violence may be at genetic risk for low IQ. Low IQ prospectively predicts both perpetration of domestic violence and victimization by domestic violence (Magdol, Moffitt, Caspi, & Silva, 1998). IQ is moderately heritable (Plomin, DeFries, McClearn, & McGuffin, 2001). Therefore, parents with low IQ may be more likely to become involved in domestic violence and also to genetically transmit lower IQs to their children. This genetic third variable confound is described in detail by DiLalla and Gottesman (1991).

The hypothesis that domestic violence causes low IQ could be tested by experimental studies exposing children to domestic violence (which are unethical) or by longitudinal studies giving IQ tests before and after exposure to domestic violence to assess within-individual change (which are impractical). This causal hypothesis can also be tested via a twin design (Rutter, Pickles, Murray, & Eaves, 2001).

Twin studies offer a natural experiment that can test whether a risk factor, such as domestic violence, has an environmentally mediated effect on child outcomes. The twin method relies on the different level of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twin pairs to estimate the con-

tribution of genetic and environmental factors to individual differences in an outcome of interest. Population variance on a behavioral phenotype (e.g., IQ) may be partitioned into an additive genetic component and two types of environmental components: a shared or familywide environmental effect that serves to make children growing up in the same family similar to each other, and a nonshared or child-specific environmental effect that impinges exclusively on one child and so serves to make children different from their siblings. It is also possible to add measured environmental variables to twin models to test the hypothesis that their influence on a phenotype is environmentally mediated (Caspi, Taylor, Moffitt, & Plomin, 2000; Jaffee, Moffitt, Caspi, Taylor, & Arseneault, 2002; Kendler, Neale, Kessler, Heath, & Eaves, 1992).

Research has shown that additive genetic, shared environmental, and nonshared environmental factors have approximately equal influences on individual differences in IQ during early childhood (Plomin et al., 2001). However, studies have not specified which aspects of the environment (or which genes) are involved. Specifying aspects of the early childhood experience that interfere with normal intellectual development is necessary to inform etiological theories and treatment efforts. Thus, the second goal of the present study is to harness the power of the genetically sensitive twin design to examine whether domestic violence affects children's IQ above and beyond the influences of genetic factors on IQ. Being reared in a household characterized by domestic violence is a familywide experience shared by both twins, and thus if domestic violence has an environmental effect on childhood IQ, we should observe that when a measure of domestic violence is added to the twin model, it should account for some proportion of the variance in IQ attributed to shared environmental factors.

Method

Participants

Participants are members of the Environmental Risk (E-risk) Longitudinal Twin Study,

which investigates how genetic and environmental factors shape children's development. The study follows an epidemiological sample of families with young twins. The E-risk sampling frame consisted of two consecutive birth cohorts (1994 and 1995) in the Twins' Early Development Study, a birth register of twins born in England and Wales (Trouton, Spinath, & Plomin, 2002). The full register is administered by the government's Office of National Statistics, which invited parents of all twins born in 1994–1995 to enroll in the register. Of the 15,906 twin pairs born in these 2 years, 71% joined the register. Our sampling frame excluded opposite-gender twin pairs and began with the 73% of register families who had same-gender twins.

The E-risk Study sought a sample size of 1100 families to allow for attrition in future years of the longitudinal study while retaining statistical power. An initial list of families was drawn from the register to target for home visits, including a 10% oversample to allow for nonparticipation. The probability sample was drawn using a high risk stratification sampling frame. High risk families were those in which the mother had her first birth when she was 20 years of age or younger. We used this sampling (a) to replace high risk families who were selectively lost to the register via nonresponse and (b) to ensure sufficient base rates of problem behaviors, given the low base rates expected for 5-year-old children. Early first child bearing was used as the risk stratification variable because it was present for virtually all families in the register, it is relatively free of measurement error, and it is a known correlate of children's problem outcomes and family risk factors, including domestic violence (Maynard, 1997; Moffitt & the E-risk Study Team, 2002). The sampling strategy resulted in a final sample in which two-thirds of study mothers accurately represent all mothers in the general population (aged 15–48) in England and Wales in 1994–1995 (estimates derived from the General Household Survey; Bennett, Jarvis, Rowlands, Singleton, & Haselden, 1996). The other one-third of study mothers (younger only) constitute a 160% oversample of mothers who were at high risk based on their

young age at first birth (15–20 years). To provide unbiased estimates that can be generalized to the population, all statistics in this article were weighted so the sample represents the proportion of young mothers in the English general population (Bennett et al., 1996).

Of the 1203 eligible families, 1116 (93%) participated in home-visit assessments when the twins were age 5 years, 4% of families refused, and 3% were lost to tracing or could not be reached after many attempts. Zygosity was determined using a standard zygosity questionnaire, which has been shown to have 95% accuracy (Price et al., 2000). DNA testing was used to determine zygosity when the zygosity questionnaire was ambiguous. The sample includes 56% MZ and 44% DZ twin pairs. Gender is evenly distributed within zygosity (49% male).

Data were collected within 120 days of the twins' fifth birthday. Pairs of research workers visited each home for 2.5 to 3 hr. While one interviewed the mother, the other tested the twins in sequence in a different part of the house. Families were given shopping vouchers for their participation, and children were given coloring books and stickers. All research workers had university degrees in behavioral science and experience in psychology, anthropology, or nursing. Each research worker completed a formal 15-day training program on either the mother interview protocol or the child assessment protocol, to attain certification to a rigorous reliability standard.

Measures

Adult domestic violence was assessed by inquiring about 12 acts of physical violence. These included all nine items from the Conflict Tactics Scale (Form R; Straus, 1990) plus an additional three items that describe other physically abusive behaviors (pushed/grabbed/shoved; slapped; shaken; thrown an object; kicked/bit/hit with fist; hit with something; twisted arm; thrown bodily; beat up; choked/strangled; threatened with knife/gun; used knife/gun). We found that 42% of children lived in families where the mothers reported at least one incident of domestic violence. For example, 16% of mothers had been pushed,

grabbed, or shoved by a partner; and 4% had been beaten up with multiple blows. Mothers were asked about their own violence toward any partner and about any partners' violence toward them over the entire 5 years since the twins' birth, responses were *not true* (coded 0) or *true* (coded 2). Another response option (coded 1) was available for women who felt uncertain about their responses, but it was virtually unused. The measure represents the variety of acts of violence mothers experienced as both victims and perpetrators. Scores were summed (range = 0–40; $M = 2.75$, $SD = 5.67$). The internal consistency reliability of the physical abuse scale was .89. Interpartner agreement reliability for this measure is very high (latent correlation = .77; Moffitt et al., 1997). Moreover, this scale is a strong predictor of which couples in the general population experience clinically significant violence, involving injury and intervention by official agencies (Moffitt, Robins, & Caspi, 2001), and high scorers on this scale experience domestic violence that is more chronic (lasts more months, with more incidents per month) than low scorers (Ehrensaft, Moffitt, & Caspi, 2002).

Children's IQ was individually tested at age 5 years using a short form of the Wechsler Preschool and Primary Scale of Intelligence—Revised (WPPSI; Wechsler, 1990). Using two subtests (Vocabulary and Block Design), children's IQs were prorated following procedures described by Sattler (1992, pp. 998–1004). Scores ranged from 52 to 145 (raw $M = 95.79$, $SD = 14.46$; weighted $M = 97.83$, $SD = 14.40$).

Child maltreatment was assessed separately for each twin using the standardized clinical interview protocol from the Multi-Site Child Development Project (Dodge, Bates, & Petit, 1990; Dodge, Petit, Bates & Valente, 1995), which was designed to enhance mothers' comfort with reporting child maltreatment occurring in the first 5 years of life, while making clear researchers' responsibility to secure intervention if maltreatment was current and ongoing. This protocol has interreporter and intercoder agreement of between .63 and .97 in Dodge's study (Dodge, Petit, & Bates, 1994; Dodge et al., 1995) and ours. The protocol included standardized probe

questions such as, “When (name) was a toddler, do you remember any time when s/he was disciplined severely enough that s/he may have been hurt?” and “Did you worry that you or someone else (such as a child minder, relative or neighbor) may have harmed or hurt (name) during those years?” Examples that were reported included children who were sexually abused by family friends, punished by being burned with matches or thrown against doors, had injuries from neglectful or abusive care (such as fractures or dislocations), or were registered with a social services–child protection team. A child was categorized as having been definitely maltreated if the mother reported that her child had been disciplined severely enough to have been hurt, was definitely harmed by a family or nonfamily member, or authorities had been involved with the child because of maltreatment. The prevalence of such definite serious maltreatment as defined in this sample was 1.5% ($N = 34$ children).

Children’s Internalizing and Externalizing Symptoms were assessed with the Child Behavior Checklist (Achenbach, 1991a) and the Teacher Report Form (Achenbach, 1991b). The internalizing syndrome reported in this article is the sum of items in the Withdrawn, Somatic Complaints, and Anxious/Depressed scales; the internal consistency reliabilities of the parent and teacher reports of internalizing problems were both .85. The externalizing syndrome reported in this article is the sum of items in the Delinquency and Aggression scales; the internal consistency reliabilities of the parent and teacher reports were .89 and .94, respectively. Because mothers and teachers provide unique information about children’s behavior and because simple combination rules work as well, if not better than, more complicated ones (Bird, Gould, & Staghezza, 1992; Piacentini, Cohen, & Cohen, 1992), the mother and teacher reports were summed to create composite measures of internalizing and externalizing behavior.

Statistical methods

Our first goal was to examine whether there is an association between exposure to domestic violence and children’s IQ in this sample.

First, we calculated the correlation between domestic violence and child IQ. Next, we used ordinary least squares regression to test whether the effects of domestic violence on IQ increased in a dose–response fashion. Because all children were included in the analyses, these analyses were conducted using the sandwich variance estimator to correct for the nonindependence of data from children in the same family (StataCorp, 2001). We created three dummy variables that categorized families into levels of domestic violence defined as low ($n = 310$ children, range = 1–3), medium ($n = 326$ children, range = 4–9) or high ($n = 302$ children, range = 10–40) for this cohort, with no domestic violence as the reference group ($n = 1280$ children). These dummy variables were entered simultaneously into a regression predicting IQ to determine whether the regression coefficients increased linearly with level of domestic violence. We repeated this regression analysis while controlling for child maltreatment to test whether domestic violence has its own, unique effect on IQ, and we repeated it controlling for internalizing and externalizing symptoms to test whether domestic violence was associated with IQ apart from behavior problems interfering with IQ testing. Our second goal was to test hypotheses about how individual differences in IQ are affected by genetic and environmental factors. Maximum-likelihood estimation techniques were used to fit different structural equations to raw data using Mx (Neale, Boker, Xie, & Maes, 2002; Neale & Cardon, 1992). In the simple ACE twin model for IQ, the variance in IQ is partitioned into the variance due to additive genetic (A), shared environmental (C), and nonshared environmental influences including error (E). The covariance in IQ for MZ twins is $\text{Cov}(\text{MZ}) = A + C$ and for DZ twins, $\text{Cov}(\text{DZ}) = .5A + C$. The square root of the variance component equals the path coefficients a , c , and e , respectively. The ultimate goal of fitting different structural equations to twin data is to account for the observed covariance structure using the most parsimonious number of parameters. We evaluated the fit of the ACE versus CE and AE models using three model-selection statistics. The first was the χ^2 goodness-of-fit statistic. When conducting model fitting to raw data in

Mx, the χ^2 goodness-of-fit statistic is calculated by subtracting the log-likelihood of the fitted model from the log-likelihood of the observed data under a saturated model that equates the expected statistics (means, variances, and covariances) to the corresponding observed statistics. The degrees of freedom for the χ^2 goodness-of-fit statistic equals the degrees of freedom for the fitted model subtracted from the degrees of freedom for the saturated model. When models are nested (i.e., identical with the exception of constraints), the difference in fit between models can be tested by examining the difference in the χ^2 values ($\Delta\chi^2$) using the *df* difference of the two models as the degrees of freedom. If the $\Delta\chi^2$ is not statistically significant, the more parsimonious model is selected, as the test indicates that the constrained model fits equally well to the data. The second model-selection statistic was the root mean square error of approximation (RMSEA), which is an index of the model discrepancy, per degree of freedom, from the observed covariance structure (MacCallum, Browne, & Sugawara, 1996). Values less than .05 indicate a close fit to the data and values less than .08 indicate a fair fit (Browne & Cudeck, 1993). The third model-selection statistic was the Bayesian information criterion (BIC), where increasingly negative values correspond to increasingly better fitting models and, in comparing two models, differences in BIC larger than 10 represent very strong evidence in favor of the model with the smaller value (Raftery, 1995).

After testing the ACE model, we tested the MACE model, which includes the influence on IQ of a continuously measured (*M*) environmental risk variable, domestic violence. As depicted in Figure 1, β_M represents the phenotypic regression of IQ on *M* (domestic violence). A test of whether domestic violence has an environmentally mediated influence on IQ involves constraining β_M to 0. We computed twice the difference in the log-likelihoods of the ACE ($\beta_M = 0$) and the MACE ($\beta_M \neq 0$) models and compared the results to a χ^2 distribution with 1 degree of freedom.

A full Mx script and description of the model are available upon request. In order to ensure that any relationship between domestic

violence and IQ was not confounded by child maltreatment, all model fitting was repeated excluding the maltreated children.

Results

Domestic violence was significantly correlated with IQ in the entire sample ($r = -.18$, $p < .001$) and for both MZ and DZ twins (Table 1). Boys and girls did not differ on IQ, $F(1, 1112) = 0.43$, $p = .51$, or level of exposure to domestic violence, $F(1, 1108) = 0.03$, $p = .86$. A test of the interaction between gender and domestic violence showed the effect of domestic violence on IQ did not differ for males and females, $F(1, 1105) = 2.51$, $p = .11$. All further analyses combined males and females.

Is domestic violence associated with children's low IQ?

Figure 2 displays children's IQs as a function of level of domestic violence. The negative effects of domestic violence on IQ (raw score) increased in a dose-response relationship as follows: low domestic violence ($b = -0.76$, $SE = 1.23$, $p = .54$), medium ($b = -4.78$, $SE = 1.22$, $p < .001$), and high ($b = -8.49$, $SE = 1.21$, $p < .001$). The regression coefficients demonstrate the number of children's IQ points suppressed, on average, with each level of domestic violence as compared with no domestic violence. That is, when compared to children whose mothers reported no domestic violence, low domestic violence is associated with an average suppression of less than 1 IQ point, medium with almost 5 points, and high with greater than 8 points.

As expected, families with domestic violence had elevated risk of child maltreatment (odds ratio = 6.61, 95% confidence intervals = 1.56, 27.98, $p < .01$). The association between child maltreatment and IQ was significant, $F(1, 1111) = 4.59$, $p < .05$. Maltreated children (M IQ = 89.51, $SD = 15.13$) had significantly lower IQs than the other children (M IQ = 97.92, $SD = 14.37$). Controlling for child maltreatment had almost no effect on the dose-response relationship between domestic vio-

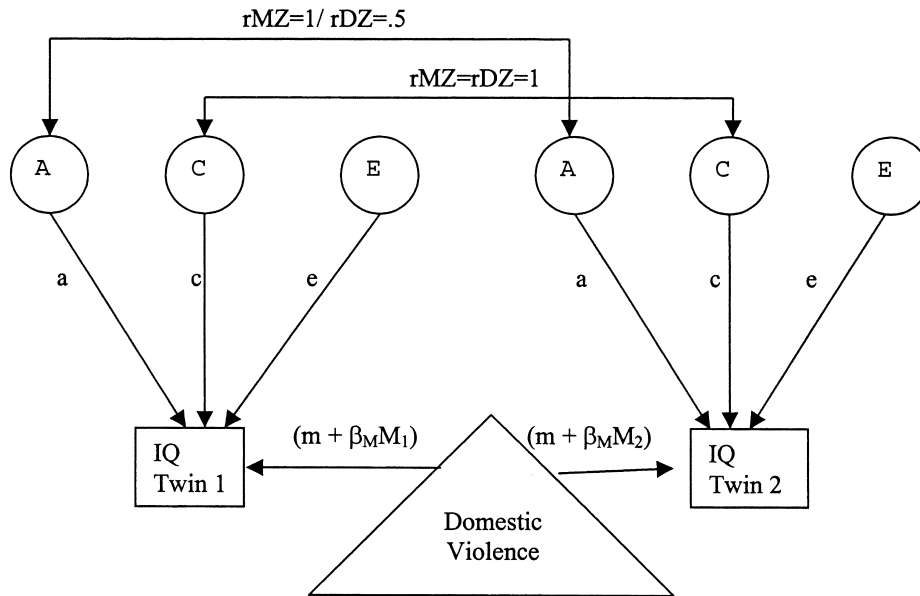


Figure 1. A path diagram showing genetic and environmental influences on IQ (MACE model). Influences on IQ: A, additive genetic; C, shared environmental; and E, nonshared environmental (including error); β_M , the phenotypic regression of IQ on M (domestic violence). A test of whether domestic violence has an environmentally mediated influence on IQ involves constraining β_M to 0.

Table 1. Correlations and descriptive statistics by zygosity (raw scores)

	Domestic Violence	Twin 1 IQ	Twin 2 IQ
MZ twins ($n = 610$)			
Domestic violence	1.00		
Twin 1 IQ	-.19***	1.00	
Twin 2 IQ	-.11*	.70***	1.00
M (SD)	2.85 (5.74)	96.83 (14.24)	97.33 (13.78)
DZ twins ($n = 485$)			
Domestic violence	1.00		
Twin 1 IQ	-.22***	1.00	
Twin 2 IQ	-.22***	.53***	1.00
M (SD)	2.64 (5.33)	98.69 (14.65)	98.72 (14.98)

Note: n varies from 1095 to 1109 because 7 families were missing data on domestic violence and 19 families were missing data on IQ.
* $p < .05$. ** $p < .01$. *** $p < .001$.

lence and IQ (low domestic violence, $b = -0.76$, $SE = 1.23$, $p = .54$; medium, $b = -4.70$, $SE = 1.22$, $p < .001$; high, $b = -8.27$, $SE = 1.24$, $p < .001$). Controlling for internalizing symptoms (low domestic violence, $b = -0.54$, $SE = 1.22$, $p = .66$; medium, $b = -4.43$, $SE = 1.21$, $p < .001$; high, $b = -7.76$, $SE = 1.21$, $p < .001$) and externalizing symptoms (low

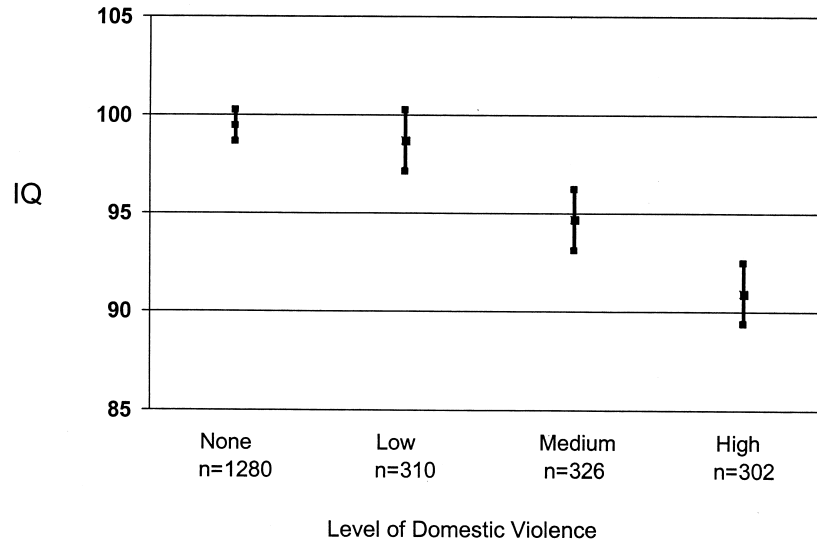


Figure 2. Children's IQ (mean with 95% confidence intervals) by the level of their mother's experience of domestic violence.

domestic violence, $b = -0.17$, $SE = 1.22$, $p = .89$; medium, $b = -3.84$, $SE = 1.23$, $p < .01$; high $b = -7.25$, $SE = 1.21$, $p < .001$) also had almost no effect on the association between domestic violence and IQ.

Is domestic violence associated with children's low IQ after accounting for genetic factors?

The MZ and DZ cross-twin correlations presented in Table 1 provide rough estimates of the extent to which genetic, shared environmental, and nonshared environmental factors account for individual differences in children's IQ. Heritability is estimated by $2(r_{MZ} - r_{DZ})$ and the shared-environment effect is estimated by $2r_{DZ} - r_{MZ}$, where r = phenotypic correlation (Plomin et al., 2001). The DZ correlations were more than half the size of the MZ correlations for IQ, suggesting substantial additive genetic and shared environmental effects on IQ.

Table 2 shows the model-fitting results. The ACE model adequately fit the data, $\Delta \chi^2(6) = 13.96$, $p > .25$; RMSEA = 0.08, BIC = -28.15. Two reduced models were tested to establish the most parsimonious model for these data. The fit statistics in Table 2 show that the models without a genetic parameter

(Model 2, CE; RMSEA = 0.15, BIC = -17.32) or a shared environmental parameter (Model 3, AE; RMSEA = 0.13, BIC = -22.60) provided a significantly worse fit. The failure of both these two-parameter models against the full ACE model indicates that additive genetic, familywide, and child-specific environmental factors were *all* necessary to explain individual differences in young children's IQ. The variance in IQ that is accounted for by each parameter in the model (Figure 3a) can be derived by squaring the standardized parameter estimate. Thus, the proportion of variance in IQ accounted for by additive genetic effects was 35% (95% CI: 23–49%), by familywide environment was 36% (95% CI: 23–49%), and by child-specific environment was 29% (95% CI: 26–33%).

Whereas the first three models in Table 2 involve latent factors that are not directly measured, Model 4 (MACE, where M refers to measured environment) includes our specific measure of the shared environment, domestic violence, to test the hypothesis that it influences children's IQ independent of familial genetic influence on IQ. Table 2 shows that MACE was a better fitting model than ACE. Figure 3 shows the path models with standardized parameter estimates for the ACE (Figure 3a) and MACE (Figure 3b) models.

Table 2. Does domestic violence influence children's IQ after accounting for genetic effects? Standardized parameter estimates of biometrical modeling

Model	Main Effects Model				<i>df</i>	-2 Log-Likelihood	Model Comparison	<i>df</i> _{diff}	$\Delta\chi^2$
	A	C	E	DV					
1. ACE	0.59	0.60	0.54	—	2,192	5,902.13			
2. CE	—	0.84	0.61	—	2,193	5,933.93	vs. ACE	1.00	31.80***
3. AE	0.84	—	0.53	—	2,193	5,928.65	vs. ACE	1.00	26.52***
4. MACE	0.60	0.57	0.54	-0.19	2,191	5,854.66	vs. ACE	1.00	47.47***

Note: The bold row is the best-fitting model. A, additive genetic effects; C, familywide or shared environment effect; E, child-specific or nonshared environment effect; DV, domestic violence.

****p* < .001.

The variance in IQ that is accounted for by each parameter in the model (Figure 3b) can be derived by squaring the standardized parameter estimates. Thus, domestic violence accounted for 3.8% (95% CI: 1.8–5.7%) of the variance in children's IQ. The variance in IQ accounted for by other shared environmental influences (C) dropped from 36 to 32% when domestic violence was included in the model, indicating that 10.6% of the variance in IQ accounted for by the latent shared environment in the ACE model was accounted for by domestic violence in the MACE model.

Model fitting was repeated excluding the 34 cases of definite child harm from the sample and the results were identical, indicating the influence of domestic violence on IQ was not due to confounding by child harm.

Discussion

In recent years, a growing body of research has attempted to document harmful effects of exposure to extreme stress, such as domestic violence, on children's neurocognitive development (De Bellis, 2001). To our knowledge, the present study provides the strongest evidence thus far that domestic violence in the home is associated with children's delayed intellectual development, by demonstrating a dose-response association using standardized measures in a population-based sample not selected for exposure. Furthermore, the size of the association is noteworthy. Children exposed to high levels of domestic violence had IQs that were on average 8 points lower than children who were not exposed.

Research showing associations between extreme rearing stress in childhood and negative child outcomes has been criticized for failure to take into account the genetic influences that may link stressful family circumstances with poor child outcomes (DiLalla & Gottesman, 1991). This is the first study to show that domestic violence is linked to an environmental effect on young children's IQ that is independent of possible confounding genetic effects on IQ. Moreover, we were able to show that the environmental effect is likely to be specifiable as domestic violence, because the negative effect of domestic violence persisted even after we controlled for the other, rarer, major source of extreme childhood stress, maltreatment. The relationship between domestic violence and children's low IQ is also not merely due to children's emotional or behavioral problems, as the relationship was largely unaffected by controlling for these symptoms. Finally, this study demonstrated how including measured environmental variables in genetically sensitive designs is a method for identifying modifiable environmental influences on the development of young children.

Traditional twin models have consistently shown that a shared environment component accounts for about 25–30% of the variance in young children's general cognitive ability (Plomin et al., 2001), and our study replicates these findings. However, this previous work, while important in demonstrating environmental effects on cognitive ability, is limited in that it treats shared environment as a latent "black box" component of the variance in IQ

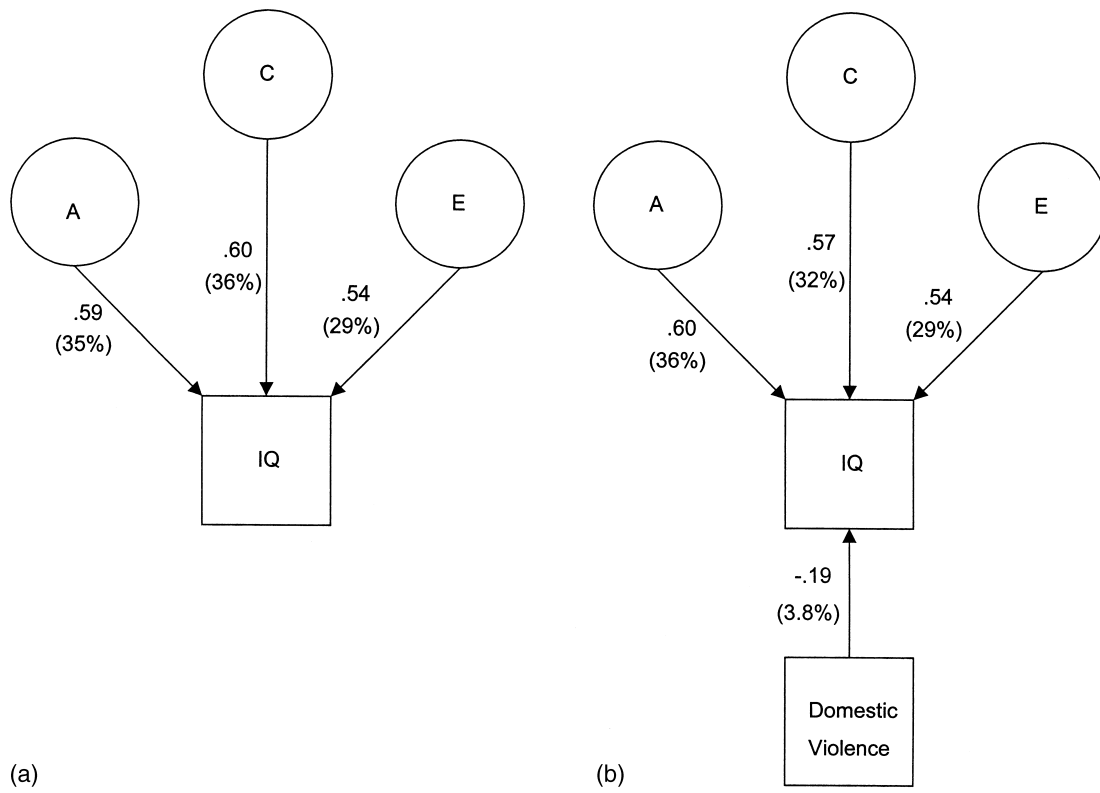


Figure 3. The results of biometrical modeling of children's IQ data. Influences on IQ: A, additive genetic; C, shared environmental; and E, nonshared environmental (including error) influences on IQ. (a) The standardized parameter estimates of the ACE model for IQ. The model is displayed for twin 1 only; the model for twin 2 would look identical. The variances of the latent variables are fixed at 1. The parameter estimates are squared to determine how much of the variance in IQ is accounted for by the latent factors. (b) The standardized parameter estimates of the MACE model for IQ. In the present study, a measured index of domestic violence was added. The path from domestic violence to IQ represents the main effect of domestic violence on individual differences in IQ. The model is displayed for twin 1 only; the model for twin 2 would look identical. The variances of the latent variables are fixed at 1, and the variance of the measured variable (domestic violence) is freely estimated. The parameter estimates are squared to determine how much of the variance in IQ is accounted for by the latent factors.

and has not specified which features of the environment affect children's cognitive ability. Identifying specific environmental risk factors is necessary to inform intervention planners aiming to promote children's cognitive development. This study suggests that domestic violence is one feature of the shared environment that adversely affects children's IQ.

How might exposure to domestic violence result in lower IQ? Violence between parents is very threatening and, therefore, extremely stressful for children (Emery, 1989; Fincham, Grych, & Osborne, 1994; Grych & Fincham,

1990, 2001; Margolin & Gordis, 2000). Domestic violence is also correlated with impairments in the parent-child relationship, such as harsh and unpredictable interactions, which, in turn, increase children's stress response (Grych & Fincham, 1990). Animal experiments and correlational human studies suggest that extreme stress can harm brain development and lead to lower IQ (Cicchetti & Walker, 2001; De Bellis, 2001; Perry, 1994; Sanchez, Ladd, & Plotsky, 2001; Sapolsky, Uno, Rebert, & Finch, 1990). The stress response is characterized by elevations in levels

of catecholamines and cortisol which are thought to adversely influence brain development (see Bremner, 1999; De Bellis, 2001, for reviews). Chronically elevated cortisol has been associated with poorer performance on a variety of neuropsychological tasks including those used to measure IQ in our study (Starkman, Giordano, Berent, Schork, & Schteingart, 2001).

Evidence indicates that chronic activation of the stress response results in neuronal death in specific brain regions (Sapolsky 2000a, 2000b). The adverse impact of stress on brain development may be especially profound during early childhood, when rapid neuronal growth and immense neuroplasticity make the brain especially sensitive to environmental input (Cicchetti & Tucker, 1994; Nelson & Carver, 1998). During early childhood, there is a normal increase in cortical grey matter (Giedd, Blumenthal, Jeffries, Castellanos, et al., 1999) and region-specific increases (midsagittal and splenium) in the corpus callosum, the brain region connecting the left and right hemispheres (Giedd, Blumenthal, Jeffries, Rajapakse, et al., 1999). Limbic system (e.g., hippocampus) and subcortical structures also increase in volume during this period (Giedd, Blumenthal, Jeffries, Castellanos, et al., 1999). Grey matter volume, as well as intracranial, cerebral, temporal lobe, hippocampal, and cerebellar volume, are significantly correlated with IQ in normal individuals (Andreasen et al., 1993). If elevated levels of catecholamines and cortisol resulting from chronic exposure to extreme stress cause neuronal death or interfere with neuronal growth in the developing brain, then children exposed to chronic stress would be expected to exhibit lower IQs.

Evidence for biological mechanisms, such as cortisol-induced neuronal loss, underlying the association between family violence and IQ was reported in both of De Bellis and colleagues 1999 studies. They showed that maltreated children with posttraumatic stress disorder had relatively smaller intracranial and cerebral volumes than nonmaltreated matched controls. The midsagittal area of the corpus callosum was also smaller in the maltreated children (De Bellis, Keshavan, et al., 1999). Moreover, IQ showed a significant positive

correlation with intracranial volume and a significant negative correlation with duration of maltreatment (De Bellis, Keshavan, et al., 1999). Although these studies did not specifically examine the effects of domestic violence on children, the authors' definition of maltreatment included domestic violence as well as other stressors such as child harm (De Bellis, Baum, et al., 1999). Thus, although the research with human children reported by De Bellis was unable to control as we did for the possibility of genetic effects on brain development (see Posthuma et al., 2002, for the heritability of brain volume), the De Bellis findings fit with our own. An important caveat is that, although the acute stress response is associated with elevated levels of cortisol, chronic hypercortisolism is not ubiquitous among traumatized children. The specific pattern of neuroendocrine dysregulation differs by type of trauma and symptom patterns experienced (Cicchetti & Rogosch, 2001a, 2001b). Thus, other or multiple neurobiological mechanisms might explain the relationship between domestic violence and low IQ for particular children.

Mechanisms other than neuronal damage could potentially mediate the relationship between domestic violence and children's low IQ. The variance in IQ explained by the shared environment drops from about 30% in childhood to 0% in adulthood (Plomin et al., 2001; Rose, 1995) and some researchers interpret this finding as suggesting that shared environmental effects on childhood IQ are transient. If this is the case, our findings may literally reflect short-term suppression of IQ due to children's current family environment. For example, children were tested in their homes and if children experience homes with domestic violence as unsafe, this might impair their performance on the IQ test. Because we do not have direct measures of the threat experienced by these children or the results from IQ tests given outside the home, we cannot test this hypothesis. However, the possibility that a threatening home environment completely explains the relationship between domestic violence and low IQ is made less plausible by our findings that this relationship was unchanged after excluding maltreated chil-

dren. Such children would be the most likely to experience their environment as threatening.

Children exposed to domestic violence are also more likely to be depressed, anxious, disruptive, or aggressive (Jaffee et al., 2002), symptoms that could potentially impair performance on the IQ test. However, the correlation between domestic violence and IQ did not change when we controlled for the children's internalizing or externalizing problems. This finding suggests that the relationship between domestic violence and children's low IQ is not merely due to the children's current distress impairing their test performance.

Limitations

The present study has several limitations. The first issue concerns whether findings from twins can generalize to singletons in terms of (a) mean IQ, (b) children's risk of exposure to domestic violence, and (c) the effect of domestic violence on children's IQ. The mean and standard deviation for IQ in our sample of twins ($M = 98$, $SD = 14.4$) is similar to that of singletons ($M = 100$, $SD = 15$; Wechsler, 1990). The percentage of women reporting domestic violence over a 5-year period was similar in our sample of mothers aged 19–48 years (42%) and a representative cohort of women aged 21–26 years (51%; Moffitt et al., 2001). The correlation between domestic violence and IQ in our twin sample ($r = -.18$) is similar to that from a recent study of exposure to violence and IQ in singletons ($\beta = -.20$, Delaney–Black et al., 2002).

A second limitation is that we did not administer full IQ tests and our results rely on prorated scores based on a subset of the full battery. Third, we did not examine whether the study twins were present during their mother's violent incidents. However, the practical constraints of rearing very young twins suggest that twins were seldom far from their mother and would have been exposed to most of her experiences. Fourth, our measure of domestic violence did not establish that domestic violence was chronic, which would best fit theories specifying how chronic stress affects neural development. However, in other

samples, high scores on the domestic violence scale that we used are associated with longer duration of domestic violence (in months) and greater frequency of domestic violence incidents per month (Ehrensaft et al., 2002).

Fifth, we did not collect information on stress hormones or conduct neuroimaging exams to establish empirically that any biological mechanisms mediate the pathway from violence exposure to lower IQ in this cohort. Sixth, although we accounted for maltreatment, we did not examine the role of harsh, unpredictable parent–child interactions. Such interactions are associated with domestic violence and may mediate the relationship between domestic violence and children's stress response. Seventh, our study is a crosssection, and it does not address the very important question of whether the effect of domestic violence on IQ at age 5 is temporary or will persist over time. Future research should extend the present study by using more in-depth measures of domestic violence and IQ, measures of stress hormones and neuroanatomy, targeted neuropsychological assessments of specific brain regions, and longitudinal follow-up to ascertain whether the effect of domestic violence on IQ is limited to childhood or longer lasting.

Implications

Our findings have implications for future research and clinical practice. With respect to future research, the present study provides further evidence that twin studies offer a practical way of testing causal relationships between environmental risks and child outcomes, particularly in situations where causality cannot be established through experimental methods. In order to test for environmentally mediated causal effects, twin studies need to do three things: (a) collect psychometrically sound measures of environmental risk factors; (b) ensure that their samples represent the full range of risk in the population by using registers instead of volunteers and by combating participant attrition; and (c) include a large enough sample size so they have adequate power to detect small to moderate environmental effects. Sample sizes of well over

1000 twin pairs are needed for statistical power to detect a latent shared environmental effect that accounts for 10–15% of the variance in a moderately heritable trait such as IQ (Martin, Eaves, Kearsley, & Davies, 1978). By including a standard measure of domestic violence and including high risk families in the English population in our sample, we were able to demonstrate that domestic violence accounts for approximately 4% of the variance in children's IQ.

In terms of clinical implications, the size of the effect of domestic violence on children's IQ is substantial, with decrements of 5 IQ points for medium and 8 IQ points for high domestic violence. In comparison, exceeding the toxic threshold for lead exposure is associated with a loss of 2–3 points in children's IQ (Stein, Schettler, Wallinga, & Valenti, 2002). Thus, the magnitude of the effect is clinically significant. Moreover, our study of 5-year-

olds shows that this suppression of IQ comes at the same time children are beginning formal schooling. Whether the influence of domestic violence on IQ reflects long-lasting structural alterations in brain development or a transient effect on cognitive abilities, starting school with an IQ disadvantage has been shown to have long-term damaging implications. Poor skills of the type assessed by the WPPSI-R vocabulary and block design tasks are known to predict poor academic performance (e.g., reading) throughout the school years and poor adjustment (e.g., behavior problems) across the life course (Gottfredson, 1997; Moffitt, 1993). Our results indicate that interventions that reduce rates of domestic violence should have added benefits for children's cognitive development and may potentially prevent some of the long-term adverse consequences of IQ disadvantage.

References

- Achenbach, T. M. (1991a). *Manual for the child behavior checklist/4–18 and 1991 profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Achenbach, T. M. (1991b). *Manual for the teacher's report form and 1991 profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Andreasen, M. D., Flaum, M., Swazey, V., O'Leary, D. S., Alliger, R., Cohen, G., Ehrhardt, J., & Yuy, W. T. C. (1993). Intelligence and brain structure in normal individuals. *American Journal of Psychiatry*, *150*, 130–134.
- Bennett, N., Jarvis, L., Rowlands, O., Singleton, N., & Haselden, L. (1996). *Living in Britain: Results from the General Household Survey*. London: HMSO.
- Bird, H. R., Gould, M. S., & Staghezza, B. (1992). Aggregating data from multiple informants in child psychiatry epidemiological research. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 78–85.
- Bremner, J. D. (1999). Does stress damage the brain? *Biological Psychiatry*, *45*, 797–805.
- Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.
- Carrey, N. J., Butter, H. J., Persinger, M. A., & Bialik, R. J. (1995). Physiological and cognitive correlates of child abuse. *Journal of the American Academy of Child & Adolescent Psychiatry*, *34*, 1067–1075.
- Caspi, A., Taylor, A., Moffitt, T. E., & Plomin, R. (2000). Neighborhood deprivation affects children's mental health: Environmental risks identified in a genetic design. *Psychological Science*, *11*, 338–342.
- Christopoulos, C., Cohn, D., Shaw, D., Joyce, S., Sullivan-Hanson, J., Kraft, S., & Emery, R. (1988). Children of abused women: I. Adjustment at time of shelter residence. *Journal of Marriage and the Family*, *49*, 611–619.
- Cicchetti, D., & Rogosch, F. A. (2001a). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology*, *13*, 677–693.
- Cicchetti, D., & Rogosch, F. A. (2001b). The impact of child maltreatment and psychopathology on neuroendocrine functioning. *Development and Psychopathology*, *13*, 783–804.
- Cicchetti, D., & Tucker, D. (1994). Development and self regulatory structures of the mind. *Development and Psychopathology*, *6*, 533–550.
- Cicchetti, D., & Walker, E. F. (2001). Editorial: Stress and development: Biological and psychological consequences. *Development and Psychopathology*, *13*, 413–418.
- De Bellis, M. D. (2001). Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy. *Development and Psychopathology*, *13*, 539–564.
- De Bellis, M. D., Baum, A. S., Birmaher, B., Keshavan, M. S., Eccard, C. H., Boring, A. M., Jenkins, F. J., & Ryan, N. D. (1999). Developmental traumatology part I: Biological stress systems. *Biological Psychiatry*, *45*, 1237–1258.
- De Bellis, M. D., Keshavan, M. S., Clark, D. B., Casey, B. J., Giedd, J. N., Boring, A. M., Frustaci, K., & Ryan, N. D. (1999). Developmental traumatology part II: Brain development. *Biological Psychiatry*, *45*, 1271–1284.
- Delaney-Black, V., Covington, C., Ondersma, S. J., Nordstrom-Klee, B., Templin, T., Ager, J., Janisse, J., & Sokol, R. (2002). Violence exposure, trauma,

- and IQ and/or reading deficits among urban children. *Archives of Paediatric and Adolescent Medicine*, *156*, 280–295.
- DiLalla, L. F., & Gottesman, I. I. (1991). Biological and genetic contributions to violence—Widom's untold tale. *Psychological Bulletin*, *109*, 125–129.
- Dodge, K. A., Petit, G. S., & Bates, J. E. (1994). Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development*, *65*, 649–665.
- Dodge, K. A., Petit, G. S., Bates, J. E., & Valente, E. (1995). Social information processing patterns partially mediate the effect of early physical abuse on later conduct problems. *Journal of Abnormal Psychology*, *104*, 632–643.
- Dodge, K. A., Bates, J. E., & Petit, G. S. (1990). Mechanisms in the cycle of violence. *Science*, *250*, 1678–1683.
- Ehrensaft, M., Moffitt, T. E., & Caspi, A. (2002). *Prevalence, correlates and developmental risk profiles for clinically abusive relationships in an unselected birth cohort*. Manuscript submitted for publication.
- Emery, R. E. (1989). Family violence. *American Psychologist*, *44*, 321–328.
- Fincham, F. D., Grych, J. H., & Osborne, L. N. (1994). Does marital conflict cause child maladjustment? Directions and challenges for longitudinal research. *Journal of Family Psychology*, *8*, 128–140.
- Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., Paus, T., Evans, A. C., & Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*, *2*, 861–863.
- Giedd, J. N., Blumenthal, J., Jeffries, N. O., Rajapakse, J. C., Vaituzis, C., Liu, H., Tobin, M., Nelson, J., & Castellanos, F. X. (1999). Development of the human corpus callosum during childhood and adolescence: A longitudinal MRI study. *Progress in Neuropsychopharmacology*, *23*, 571–588.
- Gottfredson, L. S. (1997). Why g matters: The complexity of everyday life. *Intelligence*, *24*, 79–132.
- Grych, J. H., & Fincham, F. D. (1990). Marital conflict and children's adjustment: A cognitive-contextual framework. *Psychological Bulletin*, *108*, 267–290.
- Grych, J. H., & Fincham, F. D. (Eds.). (2001). *Interparental conflict and child development: Theory, research, and applications*. Cambridge: Cambridge University Press.
- Huth-Bocks, A. C., Levendosky, A. A., & Semel, M. A. (2001). The direct and indirect effects of domestic violence on young children's intellectual functioning. *Journal of Family Violence*, *16*, 269–290.
- Jaffee, S. R., Moffitt, T. E., Caspi, A., Taylor, A., & Arseneault, L. (2002). The influence of adult domestic violence on children's internalizing and externalizing problems: An environmentally-informative twin study. *Journal of the American Academy of Child & Adolescent Psychiatry*, *41*, 1095–1103.
- Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). Childhood parental loss and adult psychopathology in twin women. *Archives of General Psychiatry*, *49*, 109–116.
- Kerouac, S., Taggart, M., Lescop, J., & Fortin, M. (1986). Dimensions of health in violent families. *Health Care for Women International*, *7*, 413–426.
- Kolbo, J. R., Blakely, E. H., & Engleman, D. (1996). Children who witness domestic violence: A review of empirical literature. *Journal of Interpersonal Violence*, *11*, 281–293.
- MacCallum, R. C., Browne, M. W., & Sugawara, H. M. (1996). Power analysis and determination of sample size for covariance structure modelling. *Psychological Methods*, *1*, 130–149.
- Magdol, L., Moffitt, T. E., Caspi, A., & Silva, P. A. (1998). Developmental antecedents of partner abuse: A prospective-longitudinal study. *Journal of Abnormal Psychology*, *107*, 375–389.
- Margolin, G., & Gordis, E. B. (2000). The effects of family and community violence on children. *Annual Review of Psychology*, *51*, 445–479.
- Martin, N. G., Eaves, L. J., Kearsley, M. J., & Davies, P. (1978). The power of the classical twin study. *Heredity*, *40*, 97–116.
- Maynard, R. A. (Ed.). (1997). *Kids having kids: Economic costs and social consequences of teen pregnancy*. Washington, DC: Urban Institute Press.
- Moffitt, T. E. (1993). The neuropsychology of conduct disorder. *Development and Psychopathology*, *5*, 135–151.
- Moffitt, T. E., & Caspi, A. (1998). Annotation: Implications of violence between intimate partners for child psychologists and psychiatrists. *Journal of Child Psychology & Psychiatry*, *39*, 137–144.
- Moffitt, T. E., Caspi, A., Krueger, R. F., Magdol, L., Margolin, G., Silva, P. A., & Sydney, R. (1997). Do partners agree about abuse in their relationship? A psychometric evaluation of interpartner agreement. *Psychological Assessment*, *9*, 47–56.
- Moffitt, T. E., & the E-risk Study Team. (2002). Teenaged mothers in contemporary Britain. *Journal of Child Psychology & Psychiatry*, *43*, 1–16.
- Moffitt, T. E., Robins, R. W., & Caspi, A. (2001). A couples analysis of partner abuse with implications for abuse prevention. *Criminology and Public Policy*, *1*, 5–36.
- Moore, J. G., Galcius, A., & Pettican, K. (1981). Emotional risk to children caught in violent marital conflict—The basildon treatment. *Child Abuse and Neglect*, *5*, 147–152.
- Neale, M. C., Boker, S. M., Xie, G., & Maes, H. H. (2002). *Mx: Statistical modelling*. VCU, Richmond: VA, Department of Psychiatry.
- Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer.
- Nelson, C. A., & Carver, L. J. (1998). The effects of stress and trauma on brain and memory: A view from developmental cognitive neuroscience. *Development and Psychopathology*, *10*, 793–809.
- Perry, B. D. (1994). Neurobiological sequelae of childhood trauma: PTSD in children. In M. Murberg (Ed.), *Catecholamine function in post-traumatic stress disorder: Emerging concepts* (pp. 233–255). Washington, DC: American Psychiatric Press.
- Pfouts, J. H., Schopler, J. H., & Henley, H. C., Jr. (1982). Forgotten victims of family violence. *Social Work*, *27*, 367–368.
- Piacentini, J. C., Cohen, P., & Cohen, J. (1992). Combining discrepant diagnostic information from multiple sources: Are complex algorithms better than simple ones? *Journal of Abnormal Child Psychology*, *20*, 51–63.
- Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2001). *Behavioral genetics* (4th ed.). New York: Worth.

- Posthuma, D., De Geus, E. J., Baare, W. F., Hulshoff Pol, H. E., Kahn, R. S., Boomsma, D. I. (2002). The association between brain volume and intelligence is of genetic origin. *Nature Neuroscience*, *5*, 83–84.
- Price, T. S., Freeman, B., Craig, I., Petrill, S. A., Ebersole, L., & Plomin, R. (2000). Infant zygosity can be assigned by parental report questionnaire data. *Twin Research*, *3*, 129–133.
- Raftery, A. E. (1995). Bayesian model selection in social research. *Sociological Methodology*, *25*, 111–163.
- Rennison, C. M., & Welchans, S. (2000, May). *Intimate partner violence: Bureau of Justice Statistics special report*. Washington, DC: U.S. Department of Justice.
- Rose, R. J. (1995). Genes and environment. *Annual Review of Psychology*, *46*, 625–654.
- Rousaville, B., & Weissman, M. M. (1977–1978). Battered women: A medical problem requiring detection. *International Journal of Psychiatry in Medicine*, *8*, 191–202.
- Rutter, M., Pickles, A., Murray, R., & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behavior. *Psychological Bulletin*, *127*, 291–324.
- Sanchez, M. M., Ladd, C. O., & Plotsky, P. M. (2001). Early adverse experience as a developmental risk factor for later psychopathology: Evidence from rodent and primate models. *Development and Psychopathology*, *13*, 419–449.
- Sapolsky, R. M. (2000a). Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. *Archives of General Psychiatry*, *57*, 925–935.
- Sapolsky, R. M. (2000b). The possibility of neurotoxicity in the hippocampus in major depression: A primer on neuron death. *Biological Psychiatry*, *48*, 755–765.
- Sapolsky, R. M., Uno, H., Rebert, C. S., & Finch, C. E. (1990). Hippocampal damage associated with prolonged glucocorticoid exposure in primates. *Journal of Neuroscience*, *10*, 2897–2902.
- Sattler, J. (1992). *Assessment of children: WISC-III and WPPSI-R Supplement*. San Diego, CA: Jerome M. Sattler.
- Stagg, V., Wills, G. D., & Howell, M. (1989). Psychopathology in early childhood witnesses of family violence. *Topics in Early Childhood Special Education*, *9*, 73–87.
- Starkman, M. N., Giordani, B., Schork, A., & Scheingart, D. E. (2001). Elevated cortisol levels in Cushing's disease are associated with cognitive decrements. *Psychosomatic Medicine*, *63*, 985–993.
- StataCorp. (2001). *Stata statistical software: Release 7.0*. College Station, TX: Stata Corporation.
- Stein, J., Schettler, T., Wallinga, D., & Valenti, M. (2002). In harm's way: Toxic threats to child development. *Development and Behavioral Pediatrics*, *23*, S13–S22.
- Straus, M. A. (1990). Measuring intrafamily conflict and violence: The Conflict Tactics (CT) Scales. In M.A. Straus & R.J. Gelles (Eds.), *Physical violence in American families: Risk factors and adaptations to violence in 8,145 families* (pp. 403–424). New Brunswick, NJ: Transaction.
- Straus, M. A. (1992). Children as witnesses to marital violence: A risk factor of lifelong problems among a nationally representative sample of American men and women. In D. F. Schwartz (Ed.), *Children and violence: Report of the twenty-third Ross Roundtable on critical approaches to common pediatric problems* (pp. 98–109). Columbus, OH: Ross Lab.
- Trouton, A., Spinath, F. M., & Plomin, R. (2002). Twins Early Development Study (TEDS): A multivariate, longitudinal genetic investigation of language, cognition and behaviour problems in childhood. *Twin Research*, *5*, 444–448.
- Wechsler, D. (1990). *Wechsler Preschool and Primary Scale of Intelligence—Revised*. London: The Psychological Corporation, Harcourt Brace and Company.
- Westra, B., & Martin, H. P. (1981). Children of battered women. *Maternal Child Nursing Journal*, *10*, 41–55.
- Wolfe, D. A., Zak, L., Wilson, S., & Jaffe, P. (1986). Child witnesses to violence between parents: Critical issues in behavioral and social adjustment. *Journal of Abnormal Child Psychology*, *14*, 95–104.