

Physical Maltreatment Victim to Antisocial Child: Evidence of an Environmentally Mediated Process

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The well-documented finding that child physical maltreatment predicts later antisocial behavior has at least 2 explanations: (a) Physical maltreatment causes antisocial behavior, and (b) genetic factors transmitted from parents to children influence the likelihood that parents will be abusive and that children will engage in antisocial behavior. The authors tested these hypotheses in the representative Environmental-Risk cohort of 1,116 twin pairs and their families, who were assessed when the twins were 5 and 7 years old. Mothers reported on children's experience of physical maltreatment, and mothers and teachers reported on children's antisocial behavior. The findings support the hypothesis that physical maltreatment plays a causal role in the development of children's antisocial behavior and that preventing maltreatment can prevent its violent sequelae.

In recent years, researchers who study how family functioning affects children's outcomes have been faced with the assertion that the rearing environment parents create exerts a relatively weak influence on their children's development. What matters most about parents, say critics, is the genes they transmit to their offspring (Harris, 1998; Rowe, 1994; Scarr, 1992). Behavioral geneticists have demonstrated that measures of the putative family environment are strongly influenced by genetic factors, reflecting the fact that (a) parents' genetically influenced characteristics shape the environment they provide for their children, and (b) children's genetically influenced characteristics lead their parents to treat them the way they do (Bouchard, 1997; Plomin & Bergeman, 1991; Scarr & McCartney, 1983).

Research on child physical maltreatment is at the center of this debate. Well-designed, prospective studies of physical maltreat-

ment and children's outcomes typically find that having been physically maltreated increases an individual's risk of engaging in violent, antisocial behavior in adolescence and adulthood (Cicchetti & Manly, 2001; Lansford et al., 2002; Widom, 1989a, 1989b). This finding has led many researchers to generate hypotheses about the mechanisms by which the cycle of violence is perpetuated across generations (De Bellis, 2001; Dodge, Pettit, Bates, & Valente, 1995; McGee, Wolfe, & Olson, 2001; Straus & Yodanis, 1996; Weiss, Dodge, Bates, & Pettit, 1992).

However, behavioral geneticists have issued a fundamental challenge to the very premise that physical maltreatment is causally associated with children's antisocial behavior. In an influential article, DiLalla and Gottesman (1991) argued that the association between children's experience of physical maltreatment and their antisocial behavior might reflect genetic transmission, wherein parents provide their children's genotype as well as their children's rearing environment. Technically, this is referred to as a *passive gene-environment (G-E) correlation* (Plomin, DeFries, & Loehlin, 1977). Physical maltreatment of a child is one form of antisocial behavior that co-occurs with other forms of adult antisocial behavior (Brown, Cohen, Johnson, & Salzinger, 1998; De Bellis et al., 2001; Dinwiddie & Bucholz, 1993; Moffitt, Caspi, Harrington, & Milne, 2002; Walsh, McMillan, & Jamieson, 2002), and previous research has documented that adults' antisocial behavior is at least moderately heritable (Rhee & Waldman, 2002). Thus, children's experience of physical maltreatment and their subsequent antisocial behavior may be linked, because the same genetic factors transmitted from parents to children influence antisocial behavior in both generations. For example, parents who pass on genes associated with high IQ may also be more likely to read to their children or to expose their children to enriching activities. Thus, the fact that there is an association between being read to as

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a child and IQ does not necessarily mean that the former causes the latter (although it might do so). Rather, the challenge goes, individuals who are read to may have inherited genes associated with high IQ from their parents and would still have a high IQ whether or not they were read to as children. In its most extreme form, the challenge proposes that individuals who are physically maltreated may have inherited genes associated with antisocial behavior from their parents and would still engage in antisocial behavior whether or not they were physically maltreated. Regardless of whether the argument is taken to its extreme, the important point is that it is difficult to disentangle genetic and environmental influences in studies of biologically related parents and children.

To better inform both etiology and practice, it is critical for researchers to establish whether physical maltreatment predicts the development of children's antisocial behavior via an environmental or a genetic route. From a prevention standpoint, if physical maltreatment has causal effects on children's outcomes, then preventing physical maltreatment will break the cycle of violence that arises when physically maltreated children grow up to engage in antisocial behavior (Widom, 1989a). However, if the effect of physical maltreatment is genetically mediated, then children may still be at risk of developing antisocial behavior regardless of whether physical maltreatment is prevented. Of course, preventing physical maltreatment is a worthwhile goal in its own right, but research can inform expectations about prevention outcomes.

From a theoretical and methodological standpoint, the ability to disentangle genetic and environmental influences clarifies efforts to understand G-E interplay (Rutter & Silberg, 2002), particularly Gene \times Environment interactions. Gene \times Environment interactions refer to genetically influenced differences in individuals' susceptibility to environmental experience (Rutter & Silberg, 2002). Many risk factors (including physical maltreatment) are alleged to be environmental, but these may be proxy measures for genetic characteristics of parents (e.g., parental punitiveness may predict child aggression because both are genetically transmitted) or genetic characteristics of children (e.g., children's heritable conduct problems may provoke adult discipline). If the association between an alleged environmental risk factor and an individual's psychopathology is genetically mediated, then a putative Gene \times Environment interaction may actually represent an interaction between one specific gene and other unidentified genes. Thus, researchers who are interested in the degree to which physical maltreatment exacerbates genetic risk for psychopathology must first establish that the experience of physical maltreatment is not genetically mediated.

The goal of this study was to test whether physical maltreatment leads to the development of antisocial behavior via an environmental causal process or via genetic transmission. The standard means of testing causality would have involved an experimental design in which children were randomly assigned to abusive and nonabusive families and followed over time. Clearly, such a design would have been highly unethical. We could, however, combine the strengths of longitudinal-epidemiological strategies with genetically informative designs to shed light on causal questions. Specifically, in the absence of experimental data, we determined six conditions that could provide strong support for the hypothesis that physical maltreatment is an environmental risk factor with a causal role in the development of children's antisocial behavior (Rutter, 2000). First, because causes must precede their effects, the

experience of physical maltreatment measured at one point in time should predict children's antisocial behavior measured at a later point in time. Second, a dose-response relationship should be established between children's experience of physical maltreatment and their antisocial behavior. Third, children's experience of physical maltreatment should predict the emergence of new antisocial behavior.

Fourth, physical maltreatment should not be heritable. When a putative measure of the environment (e.g., maltreatment victimization) is assessed separately for each child in a family and the study design is genetically informative (e.g., a study of monozygotic [MZ] and dizygotic [DZ] twins), one can estimate the degree to which variations in maltreatment victimization are accounted for by genetic and environmental factors. If one finds that genetic factors account significantly for variation in maltreatment victimization, this suggests that heritable characteristics of the child influence the child's exposure to physical maltreatment (Ge et al., 1996; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). Thus, if physical maltreatment is an environmental risk factor that is not genetically mediated, maltreatment victimization should not be heritable.

Fifth, the association between children's experience of physical maltreatment and their antisocial behavior must survive after controlling for parental antisocial behavior that might account for the association (i.e., controlling for a passive G-E correlation arising from parents' heritable antisocial behavior). Sixth, because parents' antisocial behavior may not perfectly index children's genetic risk for antisocial behavior, the association between children's experience of physical maltreatment and their antisocial behavior must survive after controlling for all genetic influences that might account for the association.

These questions were tested in a longitudinal-epidemiological study of 1,116 twin pairs. Parents reported on each child's experience of physical maltreatment by the age of 5 years, and each child's antisocial behavior was reported by parents and teachers when he or she was ages 5 and 7 years.

Method

The Environmental Risk (E-Risk) Study Sample

Participants are members of the 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 who were interviewed in the home when the twins were ages 5 and 7 years. The E-Risk sampling frame was 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 and 1995 to enroll. Of the 15,906 twin pairs born in these two years, 71% joined the register. Our sampling frame excluded opposite-sex twin pairs and began with the 73% of register families who had same-sex twins.

The E-Risk Study sought a sample size of 1,100 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, with a 10% oversample to allow for nonparticipation. The probability sample was drawn through the use of 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 childbearing was used as the risk-stratification variable because it was recorded for virtually all families in the register, it is relatively free of measurement error, and it is a known risk factor for children's problem behaviors (Maynard, 1997; Moffitt & E-Risk Study Team, 2002). The sampling strategy resulted in a final sample in which two thirds of study mothers accurately represented all mothers in the general population (ages 15–48 years) in England and Wales in 1994 and 1995 (estimates derived from the General Household Survey; Bennett, Jarvis, Rowlands, Singleton, & Haselden, 1996). The other one third of study mothers (younger only) constituted a 160% oversample of mothers who were at high risk on the basis of their young age at first birth (ages 15–20 years). To provide unbiased statistical estimates from the whole sample that could be generalized to the population of British families with children born in the 1990s, we corrected the data reported in this article with weighting to represent the proportion of maternal ages in that population (Bennett et al., 1996).

Of the 1,203 families from the initial list who were eligible for inclusion, 1,116 (93%) participated in home-visit assessments when the twins were age 5, forming the base sample for the study. Four percent of families refused, and 3% were lost to tracing or could not be reached after many attempts. With parent's permission, questionnaires were mailed to the children's teachers, and teachers returned questionnaires for 94% of cohort children. Written informed consent was obtained from mothers. The E-Risk Study has received ethical approval from the Maudsley Hospital Ethics Committee.

Zygosity was determined with a standard zygosity questionnaire that has been shown to have 95% accuracy (Price et al., 2000). Ambiguous cases were zygosity typed through the use of DNA. The sample included 56% MZ and 44% DZ twin pairs. Sex was evenly distributed within zygosity (49% male).

A follow-up home visit was conducted 18 months after the twins' age 5 assessment, when they were 6.5 years old on average (range = 6.0–7.0 years). Follow-up data were collected for 98% of the 1,116 E-Risk Study families. At this follow-up, teacher questionnaires were obtained for 91% of the 2,232 E-Risk Study children (93% of those taking part in the follow-up). Hereafter, this follow-up is referred to as the age 7 assessment. In both the age 5 and age 7 assessments, 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.

Measures

Child physical maltreatment was assessed separately for each twin by interviewing mothers with the standardized clinical interview protocol from the Multi-Site Child Development Project (Dodge, Bates, & Pettit, 1990; Dodge et al., 1995; Lansford et al., 2002). We interviewed mothers instead of ascertaining cases from Child Protective Service registers for three reasons. First, official record data identify only a small proportion of cases, which may be a biased, unrepresentative subset (Walsh et al., 2002; Widom, 1988). Second, because of time delays in detection, investigation, and legal proceedings against perpetrators, official record data sources tend not to record children as confirmed cases until older ages, and the children in our sample were 5 years old. Third, searching child protection records for this sample would have required parental consent, placing record data at the same potential risk of parental concealment as mothers' reports.

The interview protocol was designed by Dodge and colleagues (Dodge et al., 1990, 1995; Lansford et al., 2002) to enhance mothers' comfort with reporting valid child maltreatment information while also meeting researchers' legal and ethical responsibilities for reporting. Under the United Kingdom's Children Act (Department of Health, 1989), our responsibility was to secure intervention if physical maltreatment was current and ongoing. At the start of the interview about discipline and physical maltreat-

ment, the interviewer explained to the mother that if she reported physical maltreatment that had occurred in the child's first 4 years and was not ongoing, that information could remain confidential. However, if she reported physical maltreatment that occurred in the year prior to the interview and the risk to the child was ongoing, the study would be under legal obligation to assist the family to get help. Thus, when mothers gave informed consent to proceed with the interview, they understood that a report of recent, ongoing physical maltreatment would constitute a request for help (if the physical maltreatment was not already known to authorities). The interview did not ask directly about the timing of incidents, and therefore mothers who wished to report physical maltreatment while avoiding intervention could have opted to describe physical maltreatment as happening in the past. There was a need to intervene on behalf of 15 families. We found that almost all current cases of physical maltreatment were already known to government home health visitors, the family's general practitioner, or child protection teams, although very few of the cases had been officially registered.

The protocol included standardized probe questions such as, "When [name] was a toddler, do you remember any time when he or she was disciplined severely enough that he or she may have been hurt?" and "Did you worry that you or someone else [such as a babysitter, a relative or a neighbor] may have harmed or hurt [name] during those years?" (1% of mothers declined to answer the questions). Questions were carefully worded to avoid implying that the mother was the perpetrator, so mothers might feel more willing to report that a child had been maltreated.

In cases in which mothers reported any physical maltreatment, interviewers probed mothers for details about the incident and recorded notes. Interviewers coded the likelihood that the child had been maltreated on the basis of the mothers' narrative. This classification showed intercoder agreement on 90% of ratings ($\kappa = .56$) in the Dodge et al. study (Dodge, Pettit, & Bates, 1994; Dodge et al., 1995) and in ours. The 10% of codes that disagreed tended to reflect uncertainty about whether physical maltreatment was probable or definite. On the basis of the mother's report of the severity of discipline and the interviewer's rating of the likelihood that the child had been physically maltreated, children were coded as having not been, possibly been, or definitely been physically maltreated. Examples of possible physical maltreatment in our sample ($n = 273$ children) included instances in which the mother reported that she smacked the child harder than she intended to and left a mark or bruise, or cases in which social services were contacted by schools, neighbors, or family members out of concern that the child was being physically maltreated. Examples of definite physical maltreatment included children who were beaten by a teenaged stepsibling, were punished by being burnt with matches or thrown against doors, had injuries (e.g., fractures or dislocations) from neglectful or abusive care, or were formally registered with a social services child protection team. The prevalence of such definite, serious physical maltreatment as defined in this sample was 1.5% ($n = 34$ children). For the purposes of our analyses, the child physical maltreatment variable was recoded into a dichotomous variable representing children who experienced no physical maltreatment (unweighted, the prevalence was 86%; weighted to represent the population, it was 88%) versus a combined group of children who experienced possible or definite physical maltreatment (unweighted, the prevalence was 14%; weighted to represent the population, it was 12%). The prevalence of physical maltreatment was similar among MZ (11%) and DZ (14%) twins. Our combined prevalence of 12% resembles the 15% prevalence estimate reported by Dodge and colleagues (Dodge et al., 1990), whose measurement protocol we used. Our prevalence rate of 1.5% for definite physical maltreatment is consistent with physical maltreatment estimates of 1.5% and 2.3% from population surveys in North America (Bland & Orn, 1986; Egami, Ford, Greenfield, & Crum, 1996) and estimates of 1.2% from nationwide surveys of child protective services in North America (National Child Abuse and Neglect Data System, 2002).

The physical maltreatment interview protocol has (a) good concurrent validity, as evidenced by correlations above .60 with mothers' reports of their child-directed aggression on the Conflict Tactic Scales (Dodge et al., 1990; Straus & Gelles, 1988), (b) good interreporter reliability, as evidenced by a correlation of .60 between mothers' and fathers' reports in 396 couples (Dodge et al., 1995), and (c) good predictive validity, as evidenced by significant 12-year prediction from preschool maltreatment to outcomes in Grade 11, including increased violence, school absenteeism, anxiety and depressive symptoms, and posttraumatic stress disorder symptoms, controlling for a variety of social and family risk factors (Lansford et al., 2002).

The same set of questions about physical maltreatment was asked about each individual twin, and the interviews about each twin were separated by 1.5 hr of questions on other topics. In those families in which at least one twin was physically maltreated, both twins suffered maltreatment in 63% of cases and only one twin suffered maltreatment in 37% of cases.

Children's antisocial behavior was assessed at ages 5 and 7 with the Achenbach family of instruments (Achenbach, 1991a, 1991b). The Aggression and Delinquency scales were supplemented with *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM-IV]; American Psychiatric Association, 1994) items assessing conduct disorder and oppositional defiant disorder. Mothers' reports and teachers' reports of antisocial behavior correlated .29 ($p \leq .001$) and .38 ($p \leq .001$) at ages 5 and 7, respectively, which is typical of interrater agreement about behavioral problems (Achenbach, McConaughy, & Howell, 1987). Mothers' reports and teachers' reports of children's behavior problems were summed (items were scored from 0–2). At age 5, scores ranged from 0 to 130 ($M = 21.17$, $SD = 16.27$), and at age 7, scores ranged from 0 to 132 ($M = 18.48$, $SD = 15.79$). The internal consistency of the combined score was .94 at age 5 and .95 at age 7. In summing mothers' reports and teachers' reports of children's antisocial behavior, items that were common across the two scales were effectively counted twice (72% of the item pool). Prior research has found that the pervasiveness of symptoms across settings is a hallmark of children's behavior problems that are clinically significant, and moreover, research has documented that such pervasive behavior problems have a higher genetic loading than those that are situationally specific (Arseneault et al., 2003).

Father's and mother's prior history of antisocial behavior was reported by the mothers, who were interviewed with the Young Adult Behavior Checklist (Achenbach, 1997), which was modified to obtain lifetime data, and supplemented with questions from the Diagnostic Interview Schedule (Robins, Cottler, Bucholz, & Compton, 1995) that assessed the (lifetime) presence of DSM-IV symptoms of antisocial personality disorder. The internal consistency reliabilities of the maternal and paternal antisocial behavior scales were .90 and .95, respectively. Scores ranged from 0 to 88 ($M = 14.82$, $SD = 16.30$) on the paternal antisocial behavior scale and from 0 to 60 ($M = 11.29$, $SD = 9.72$) on the maternal antisocial behavior scale. A methodological study of mother–father agreement about men's antisocial behavior in a representative subset of this sample showed that the women provided reliable information about their children's father's behavior (Caspi et al., 2001). The correlation between men's and women's reports about men's antisocial behavior was .74 (95% confidence interval [CI] = 0.53, 0.95; Caspi et al., 2001).

Results

Is There an Association Between Children's Experience of Physical Maltreatment and Their Antisocial Behavior?

We first tested whether the well-documented association between children's experience of physical maltreatment and their antisocial behavior would replicate in our sample. Ordinary least squares (OLS) regression models¹ showed that children's experience of physical maltreatment by age 5 predicted their antisocial

behavior at age 5 ($b = 9.11$, $SE = 1.41$, $p \leq .001$) and at age 7 ($b = 7.67$, $SE = 1.31$, $p \leq .001$).

Is There a Dose–Response Association Between Physical Maltreatment and Children's Antisocial Behavior?

Figure 1 shows a dose–response relationship between the likelihood of having been physically maltreated and children's antisocial behavior at 5 and 7 years. OLS regression analysis revealed that those who were not maltreated, possibly maltreated, and definitely maltreated had significantly different antisocial behavior scores at age 5, $F(2, 1115) = 23.29$, $p \leq .001$, and 7 years, $F(2, 1088) = 21.77$, $p \leq .001$. Figure 1 shows that the effect sizes for the group differences were moderate to large in magnitude. At both 5 and 7 years, antisocial behavior scores in the possibly maltreated group were approximately half a standard deviation higher than those in the nonmaltreated group, and antisocial behavior scores in the definitely maltreated group were approximately .8 standard deviations higher than those in the nonmaltreated group. Contrast analyses in a regression framework revealed that physically maltreated children had significantly more antisocial behavior problems than nonmaltreated children at age 5 ($b = 7.74$, $SE = 1.40$, $p \leq .001$) and age 7 ($b = 7.15$, $SE = 1.21$, $p \leq .001$). At age 7 years, children who were definitely maltreated had more antisocial behavior problems than children who were possibly maltreated ($b = 3.68$, $SE = 1.74$, $p \leq .05$), although this difference was not significant at age 5 ($b = 3.07$, $SE = 2.05$, $p = .14$).

Does Children's Experience of Physical Maltreatment Predict the Emergence of New Antisocial Behavior Over Time?

OLS regression models were used to test the hypothesis that the experience of physical maltreatment would predict the emergence of antisocial behavior between ages 5 and 7 years. The effect of having been physically maltreated on antisocial behavior at age 7 remained significant even after controlling for age 5 antisocial behavior ($b = 1.77$, $SE = 0.93$, $p = .056$). Thus, compared with nonmaltreated children, physically maltreated children had higher antisocial behavior scores at age 7, even after controlling for the continuity of antisocial behavior from ages 5 to 7.

What Is the Genetic and Environmental Architecture of Physical Maltreatment?

The twin method is a natural experiment that relies on the different levels of genetic relatedness between MZ and DZ twin pairs to estimate the contribution of genetic and environmental factors to individual differences in a phenotype of interest. Phenotypes include any behavior or characteristic that is measured separately for each twin, such as each twin's score on a behavior problem checklist or each twin's experience of physical maltreat-

¹All regression analyses reported in the Results section were based on the sandwich or Huber–White variance estimator (Gould & Sribney, 1999), a method available in Stata 7.0 (StataCorp, 2001). Application of this technique addresses the assumption of the independence of observations. It penalizes estimated standard errors and therefore accounts for dependence in the data due to analyzing sets of twins.

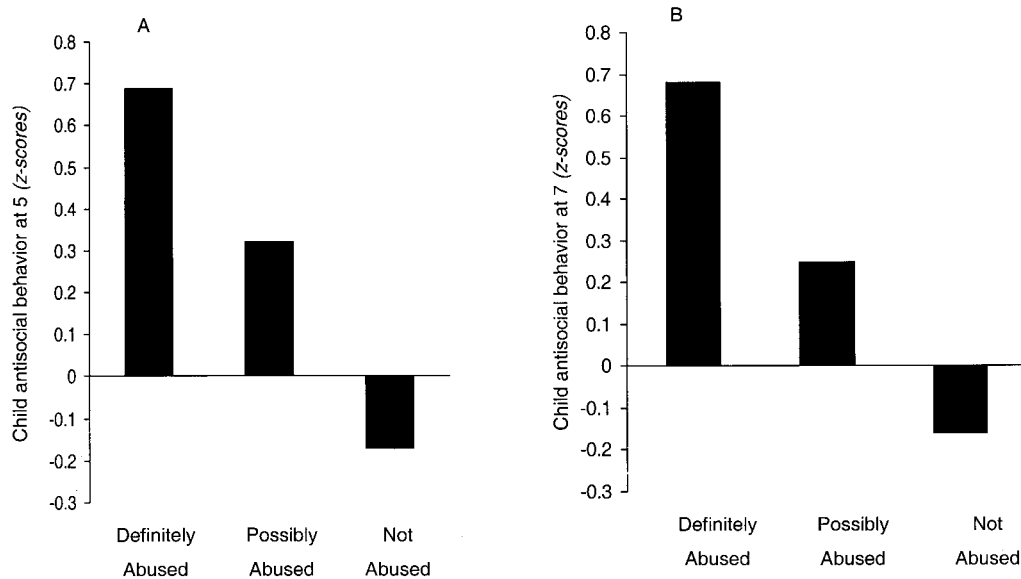


Figure 1. Mean antisocial behavior scores at ages 5 (A) and 7 (B) as a function of physical maltreatment (z scores). Differences between groups can be interpreted in terms of standard deviation units (d), where $d = .3$ is considered a small effect size, $d = .5$ is considered a moderate effect size, and $d = .8$ is considered a large effect size.

ment. Population variation on any phenotype may be partialled into an additive genetic component and two types of environmental components through the use of the following logic. First, MZ twins share all their genes, but DZ twins, like all siblings, share half of their polymorphous genes, on average. Polymorphous genes are genes associated with differences among people. For example, genes influencing eye color are polymorphous, but genes determining that people have eyes are not. As such, a genetic contribution to physical maltreatment exposure is indicated when the similarity of MZ twins is greater than the similarity of DZ twins.

Second, MZ twins' genetic similarity is twice that of DZ twins, and therefore, if nothing more than genes were influencing behavior, then MZ twins' should be at least twice as similar with respect to their experience of physical maltreatment as DZ twins. If not, this indicates that something more than genes has made the twins similar (i.e., environments that the siblings share in common must have enhanced their similarity). Third, twin studies also address the perennial question of why family members differ from one another (Plomin & Daniels, 1987) by using the following logic. If MZ twins, despite sharing all of their genes, are not perfectly identical in their exposure to physical maltreatment, this indicates that nonshared experiences, unique to each family member, reduce their similarity. (For detailed explanations of the statistical methods that are applied to operationalize the logic behind behavior genetic designs, see Plomin, DeFries, McClearn, & McGuffin, 2001.)

Significant genetic effects on individual differences in children's experience of physical maltreatment would imply that heritable characteristics of the child influence the child's exposure to physical maltreatment. Significant shared environmental effects would imply that characteristics that differ between families (e.g., low socioeconomic status) account for individual differences in physical maltreatment. Significant nonshared environmental ef-

fects would imply that factors that vary among children in the same family (e.g., the extent to which a parent scapegoats one child more than another) account for individual differences in physical maltreatment.

We used maximum-likelihood estimation techniques to test univariate models of children's experience of physical maltreatment (Neale & Cardon, 1992). These models decompose the variance in children's experience of physical maltreatment into that which can be accounted for by latent additive genetic (A), shared environmental (C), and nonshared environmental (E) factors. Because the latent variables are unmeasured, they do not have a natural scale and must be assigned a variance (i.e., the variance is fixed at 1.0). The goal of fitting different structural equations to twin data is to account for the observed covariance structure with the most parsimonious number of parameters. To compare the fit of different models, we used two model-selection statistics. The first was the chi-square goodness-of-fit statistic. Large values indicate poor model fit to the observed covariance structure. When two models are nested (i.e., identical with the exception of constraints placed on the submodel), the difference in fit between them can be evaluated with the chi-square difference, using as its degrees of freedom the degrees of freedom difference from the two models. When the chi-square difference is not statistically significant, the more parsimonious model is selected, as the test indicates that the constrained model fits equally well with the data. The second model-selection statistic was the root-mean-square error of approximation, 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 close fit and values less than .08 indicate fair fit to the data (Browne & Cudeck, 1993).

Table 1 displays tetrachoric correlations indexing the similarity of MZ twins ($r = .77$) and DZ twins ($r = .71$) on physical

Table 1
Tetrachoric Correlations for Monozygotic and Dizygotic Twins and Estimates of Genetic and Environmental Influences on Maltreatment Victimization

Variable	MZ	DZ	A	C	E	χ^2	df	RMSEA
Physical maltreatment	.77*	.71*	.07 (.00-.21)	.88 (.75-.96)	.05 (.02-.09)	2.88	3	.006
Physical maltreatment (A = 0)			—	.94 (.91-.96)	.06 (.04-.09)	4.60	4	.011

Note. Values in parentheses represent 95% confidence intervals. Dash indicates that the effect was not estimated in that model. For chi-square values, $N = 1,115$. MZ = monozygotic twins; DZ = dizygotic twins; A = additive genetic effects; C = shared environmental effects; E = error or nonshared environmental effects; RMSEA = root-mean-square error of approximation.
* $p \leq .001$.

maltreatment exposure. There was little indication that MZ twins' greater genetic similarity made them more concordant for exposure to physical maltreatment. The pairwise concordance was used to calculate the proportion of pairs in which both twins were physically maltreated, which has the formula $C/(C + D)$, where C is the number of concordant pairs and D is the number of discordant pairs (i.e., pairs in which only one twin was physically maltreated). The pairwise concordance for MZ twins was 66%, and the pairwise concordance for DZ twins was 60%. Maximum-likelihood threshold models estimated in Mx (Neale, Boker, Xie, & Maes, 2002) with contingency table data showed that most of the variation in children's experience of physical maltreatment was accounted for by shared environmental influences (see Table 1). To test the hypothesis that children's physical maltreatment victimization is not heritable, we set the genetic path to 0. The reduced model in which genetic influences were hypothesized to have no effect on physical maltreatment victimization did not fit significantly worse than the full model, $\chi^2_{diff}(1, N = 1,115) = 1.72, ns$. Thus, genetic factors did not account for significant variation in children's experience of physical maltreatment in this representative sample. This finding eliminates the possibility that any heritable characteristic of the child (i.e., antisocial behavior or otherwise) provoked physical maltreatment.

Passive G-E Correlations: I. Is the Association Between Children's Experience of Physical Maltreatment and Their Antisocial Behavior Accounted for by Their Parents' Prior History of Antisocial Behavior?

Table 2 presents correlations among child antisocial behavior, parent antisocial behavior, and children's experience of physical maltreatment. Parents who engaged in high levels of antisocial behavior were more likely to maltreat their children. For example,

Table 2
Correlations Among Child and Parent Antisocial Behavior and Physical Maltreatment

Variable	1	2	3	4
1. Child antisocial behavior (at 7 years)	—			
2. Mother antisocial behavior	.34*	—		
3. Father antisocial behavior	.27*	.53*	—	
4. Physical maltreatment	.16*	.24*	.20*	—

* $p \leq .001$.

high maternal antisocial behavior (defined as the top quartile of the maternal antisocial behavior distribution) increased the odds of physical maltreatment by more than three times (odds ratio [OR] = 3.20, 95% CI = 2.24, 4.58, $p \leq .001$). The results for fathers' antisocial behavior were similar (OR = 3.05, 95% CI = 2.12, 4.36, $p \leq .001$). We conducted an OLS regression to test whether the parents' history of antisocial behavior accounted for the association between children's experience of physical maltreatment and their antisocial behavior. Mothers' ($b = 0.42, SE = 0.07, p \leq .001$) and fathers' ($b = 0.12, SE = 0.04, p \leq .01$) prior antisocial behavior predicted children's antisocial behavior at age 7 years. The parents' history of antisocial behavior accounted for nearly 50% of the effect of physical maltreatment on children's antisocial behavior at age 7 (reducing the coefficient for physical maltreatment from $b = 7.67, SE = 1.31, p \leq .001$, to $b = 3.48, SE = 1.19, p \leq .01$). However, the effect of physical maltreatment remained significant. Thus, the association between physical maltreatment victimization and children's antisocial behavior was not entirely accounted for by the fact that parents who have a prior history of antisocial behavior are more likely to maltreat their children.

Passive G-E Correlations: II. Does Physical Maltreatment Predict Children's Antisocial Behavior Controlling for All Other Genetic Influences?

In the previous analysis we tested whether passive G-E correlations accounted for the association between maltreatment victimization and children's antisocial behavior by controlling for parent antisocial behavior, which was assumed to index genetic risk for antisocial behavior that parents transmit to children. However, a more direct measure of genetic risk is the estimate of the heritability of children's antisocial behavior. Thus, DeFries-Fulker (D-F) regression analyses were conducted to further test whether passive G-E correlations accounted for the association between children's maltreatment victimization and their antisocial behavior. D-F analysis uses kinship-pair data (e.g., data from twins or adoptive siblings) to separate heredity and shared environmental influences in a regression framework (DeFries & Fulker, 1985; for examples of this approach in studies of child and family development, see Jaffee, Moffitt, Caspi, & Taylor, 2003, and Rodgers and colleagues [Rodgers, Kohler, Kyvik, & Christensen, 2001; Rodgers, Rowe, & Li, 1994]). The sandwich variance estimator was used to correct for the nonindependence of twin observations, as recommended by Kohler and Rodgers (2001). The equation for the basic D-F regression model is as follows:

Table 3
Results of Regression Analysis Testing Whether Physical Maltreatment Predicts Children's Antisocial Behavior After Controlling for Genetic Risk for Antisocial Behavior

Regression terms	Not controlling for genetic risk		Controlling for genetic risk	
	<i>b</i>	95% CI	<i>b</i>	95% CI
Constant	17.52	16.64, 18.40	16.55	12.31, 20.79
Physical maltreatment	7.67*	5.10, 10.23	3.37*	1.90, 4.85
<i>R</i>			-11.95*	-16.57, -7.33
ABS _{twin2} (estimate of shared environment)			0.06	-0.17, 0.29
<i>R</i> × ASB (<i>h</i> ²)			0.67*	0.42, 0.93

Note. *R* = coefficient of genetic relatedness; ASB = antisocial behavior; *h*² = heritability; CI = confidence interval.

* *p* ≤ .001.

$$ASB_{twin1} = \beta_1 + \beta_2(R) + \beta_3(ASB_{twin2}) + \beta_4(R \times ASB_{twin2}) + e.$$

In this equation, ASB_{twin1} represents Twin 1's antisocial behavior score at age 7; β_1 represents the constant term; The coefficient β_2 is not usually interpreted in D-F models. *R* represents the coefficient of genetic relatedness (1.0 for MZ twins and .5 for DZ twins); ASB_{twin2} represents Twin 2's antisocial behavior score at age 7; β_4 represents the population heritability estimate *h*² because, when statistically significant, it demonstrates that Twin 1 and Twin 2's resemblance for antisocial behavior is conditioned on their degree of genetic relatedness; and β_3 estimates shared environmental variation because it represents the twins' resemblance for antisocial behavior independent of genetic resemblance (Rodgers et al., 1994).

The hypothesis that genetic transmission accounts for the association between having been physically maltreated and children's antisocial behavior predicts that the effect of physical maltreatment on children's antisocial behavior will no longer be significant once genetic influences on children's antisocial behavior are controlled. The basic D-F model can be expanded to test this prediction. Thus, the effect of physical maltreatment β_5 (MALTREAT) was estimated in an augmented model:

$$ASB_{twin1} = \beta_1 + \beta_2(R) + \beta_3(ASB_{twin2}) + \beta_4(R \times ASB_{twin2}) + \beta_5(MALTREAT) + e.$$

The results of the D-F regression analysis are presented in Table 3. Two findings are noteworthy. First, approximately two thirds of the variation in children's antisocial behavior at age 7 was accounted for by genetic factors (*h*² = .67, 95% CI = .42, .93). Second, physical maltreatment victimization significantly predicted elevated antisocial behavior scores even after controlling for genetic effects on children's antisocial behavior. However, genetic factors accounted for 56% of the effect of physical maltreatment on children's antisocial behavior (reducing the coefficient from *b* = 7.67, *SE* = 1.31, *p* ≤ .001, to *b* = 3.37, *SE* = 0.75, *p* ≤ .001). This finding provides partial support for the genetic transmission hypothesis but also documents that physical maltreatment has environmentally mediated effects on children's antisocial behavior beyond genetic transmission.²

Discussion

The results from this study satisfy six conditions that together support the hypothesis that physical maltreatment is an environmental risk variable that is causally linked to children's antisocial behavior. We found that (a) physical maltreatment prospectively predicted antisocial outcome, (b) physical maltreatment bore a dose-response relation to antisocial outcome, (c) physical maltreatment was followed by the emergence of new antisocial behavior, (d) children's maltreatment victimization was not influenced by genetic factors, (e) the effects of physical maltreatment remained significant after controlling for parents' history of antisocial behavior, and (f) the effect of physical maltreatment was significant after controlling for any genetic transmission of antisocial behavior, although genetic factors accounted for approximately half of the association between physical maltreatment and children's antisocial behavior.

In their 1991 article, DiLalla and Gottesman concluded that whereas the experience of having been maltreated might perpetuate the cycle of violence, the relative contributions of environmental and genetic factors to the cycle of violence phenomenon were unknown. They noted that by assuming a causal relation between the experience of physical maltreatment and victims' antisocial behavior without testing the alternative genetic transmission hypothesis, psychologists might overestimate the potential of physical maltreatment prevention programs to reduce the intergenerational transmission of antisocial behavior. Our analyses help to disentangle these genetic and environmental contributions and provide evidence that approximately half of the intergenerational transmission of antisocial behavior is environmentally mediated. To the degree that physical maltreatment is genetically mediated, it appears that heritable characteristics of the parent that are transmitted to children are correlated with parents' perpetration of physical maltreatment (i.e., a passive G-E correlation). Parents

²The results of the passive G-E correlation analyses (Parts I and II) were unchanged when the age 5 measure of antisocial behavior served as the dependent variable instead of the age 7 measure. The effect of physical maltreatment remained statistically significant in both analyses, and the reduction in the effect of maltreatment after controlling for parent antisocial behavior and any other genetic risk for antisocial behavior was comparable. These analyses are available from Sara R. Jaffee on request.

who had a prior history of antisocial behavior were more likely to maltreat their children, and these parents also had children who engaged in high levels of antisocial behavior. Nevertheless, the effect of physical maltreatment on children's antisocial behavior was significant, even after controlling for parents' history of antisocial behavior.

Moreover, our findings ruled out a second route by which genetic transmission could theoretically account for the association between physical maltreatment victimization and children's antisocial behavior. Because physical maltreatment victimization was not heritable, it is not possible that heritable characteristics of the child evoke physical maltreatment. Although this finding appears to contradict prior studies reporting genetically mediated child effects on adoptive parents' disciplinary styles (Ge et al., 1996; O'Connor et al., 1998), those studies did not examine physical maltreatment specifically. In summary, our findings suggest that DiLalla and Gottesman (1991) were correct to remind psychologists that the link between physical maltreatment and victim antisocial behavior may be genetically mediated because it is, in part. However, physical maltreatment plays a causal role in the development of children's antisocial behavior beyond this genetically mediated effect.

Implications for Research

Our finding that physical maltreatment victimization was not genetically mediated by evocative G-E correlations and only partially genetically mediated by passive G-E correlations has implications for researchers who study why some children who are maltreated do not grow up to engage in violent, antisocial behavior (Widom, 1989b). Two recent studies reported an interaction between environmental risk and genetic risk, showing that the effect of maltreatment on antisocial behavior in childhood and adulthood depended on the individual's genetic makeup. Using data from the E-risk study of 2,200 5-year-old twins, Jaffee and colleagues (Jaffee, Caspi, et al., 2004) found that conduct problems were elevated among children who were at high genetic risk for conduct disorder and who had experienced physical maltreatment. However, conduct problems were not as elevated among children who were at low genetic risk for conduct disorder, even though they too experienced physical maltreatment. Similarly, using data from a prospective, longitudinal study of 500 adult males, Caspi and colleagues (Caspi et al., 2002) found that antisocial behavior was elevated among men who had the low-activity monoamine oxidase A (MAOA) genotype and who experienced childhood maltreatment. However, antisocial behavior was not as elevated among men who had the more common high-activity MAOA genotype even though they too experienced maltreatment.

If maltreatment is genetically mediated, then what researchers have called Gene \times Environment interactions (e.g., MAOA \times Maltreatment) may actually reflect Gene \times Gene interactions (Rowe & Harris, 2002). Responding to these criticisms, we believe our finding that physical maltreatment victimization was not strongly genetically mediated demonstrates that researchers are justified in conceptualizing physical maltreatment as a measure of environmental risk that interacts with an individual's genetic makeup to predict antisocial behavior. It is important here to clarify the distinction between genetic mediation and genetic moderation. *Genetic mediation* refers to the degree to which passive or evocative G-E correlations account for the association between a

putative measure of the environment and a child's outcome (e.g., a child's antisocial behavior). However, a risk factor that is not genetically mediated may be moderated by genetic risk, as demonstrated by the findings of the Gene \times Environment interaction described above. *Genetic moderation* refers to variation in sensitivity to environmental experiences (e.g., physical maltreatment) as a function of one's genetic makeup. As elaborated below, by identifying subgroups for whom the effect of physical maltreatment is particularly detrimental, findings of genetic moderation have important implications for prevention efforts.

Implications for Methodology

Researchers who study biologically related parents and children must contend with the complexities of G-E interplay in their attempts to understand how parents' behavior affects children's outcomes. One genetically sensitive design that has been useful in helping researchers understand G-E interplay is the adoption study. The cardinal advantage to the adoption design is that it rules out the possibility that passive G-E correlations account for the association between a putative environmental risk factor and a child's outcome. That is, the parents who shape the child's environment (i.e., the adoptive parents) are not the same parents who provided the child's genotype (i.e., the biological parents). Given that passive G-E correlations are likely to be pervasive in families (indeed, they were present in our data), the ability of the adoption design to test the effects of environmental risk and protective factors on children's outcomes, while eliminating the possibility that passive G-E correlations account for these associations, is a significant advantage of the design.

However, two other features of the adoption design limit its ability to help researchers understand G-E interplay. First, the adoption design does not automatically rule out the possibility that evocative G-E correlations account for the association between the environmental risk and the child's outcome. That is, as adoption studies themselves have shown, heritable characteristics of the child may provoke a particular response from the environment regardless of the child's genetic relatedness to the parent (Ge et al., 1996; O'Connor et al., 1998). Thus, the adoption design does not necessarily ensure that genes and environments are independent. Second, adoption studies are not well suited to explore the effects of severe environmental risk factors, such as physical maltreatment or extreme poverty, on child outcomes because adoptive homes are carefully screened by social service agencies who seek to place adoptees in healthy environments (Stoolmiller, 1999).

In contrast to adoption studies, twin studies in which families are representative of the population, can yield a sufficient range of data on severe environmental risk experiences. However, given the biological relatedness of twin children and their parents, researchers must assume that passive or evocative G-E correlations might account for the association between the environmental risk experience and the children's outcomes. As we have shown, however, twin designs can be used to test such assumptions and, consequently, better inform efforts to understand G-E interplay. Given their complementary strengths, the most powerful efforts to understand G-E interplay are those in which the findings from adoption studies corroborate findings from twin studies and vice versa. However, to our knowledge there is no adoption study in which investigators have collected data on physical maltreatment.

Implications for Clinical Practice

Our finding that physical maltreatment is an environmentally mediated risk factor for children's antisocial behavior, combined with previous research showing that the effect of maltreatment is moderated by genetic risk (Caspi et al., 2002; Jaffee, Caspi, et al., 2004), provides the clearest support possible within the limits of ethical human research for the hypothesis that preventing physical maltreatment should break the link between maltreatment victimization and antisocial behavior, particularly for vulnerable subgroups of children (i.e., those who are at high genetic risk for antisocial behavior). Thus, identifying programs that are successful in preventing maltreatment is crucial. Early interventions targeting high-risk families have been shown to be effective. For example, an evaluation of a nurse home-visitation program targeting high-risk families found that fewer children in the nurse-visited group were maltreated relative to comparison children (Olds et al., 1997). Moreover, those children in the nurse-visited group who were maltreated did not have more behavior problems compared with their nonmaltreated nurse-visited peers because the intervention prevented maltreatment from becoming persistent (Eckenrode et al., 2001). It bears noting that this experimental demonstration of the effect of maltreatment prevention on children's antisocial behavior adds a seventh piece of evidence to the six presented above that the link between maltreatment victimization and children's antisocial behavior is environmentally mediated. Demonstrating that antisocial behavior is reduced when maltreatment is prevented is not by itself sufficient to prove that maltreatment causes antisocial behavior, for the same reason that reducing fever by taking aspirin does not prove that aspirin deficiency causes fevers. Nonetheless, the nurse home-visit study, when combined with the six findings we report in this article, provides strong evidence that physical maltreatment plays a causal role in the etiology of antisocial behavior. Our finding that physical maltreatment is causally implicated in the cycle of violence lends support to the value of implementing these prevention efforts.

Although individuals' genotypes moderate the effect of maltreatment on risk for antisocial behavior, we advocate maltreatment prevention efforts over gene therapies for antisocial behavior for two reasons. First, antisocial behavior is a complex disorder that is likely to involve multiple genes of small effect interacting not only with the environment but also with other genes. Given this complexity, the field is many years away from identifying genes that may be targeted in pharmacological or gene therapy interventions for antisocial behavior. Second, genes whose variants are common in the population are likely to have many functions, some desirable and some not so. For example, the gene that increases risk for sickle-cell anemia in black Africans also confers protection against malaria. The fact that a given gene may have many functions beyond increasing risk for disorder, combined with the fact that genes interact in complex systems, suggests that removing the effects of one gene via gene therapy may not be effective and may even prove dangerous (Nuffield Council on Bioethics, 2002). Third, many genes are likely to have effects on behavior only in combination with relevant environmental experiences (Hamer, 2002). For example, as reported by Caspi and colleagues (Caspi et al., 2002), MAOA activity had no effect on adult men's antisocial behavior except in combination with a history of maltreatment. Although gene therapies that, for example, replace the risky low-activity MAOA allele with the protective high-activity allele may

sever the association between childhood maltreatment and adult antisocial behavior, gene therapies will not affect the likelihood that children will fall victim to maltreatment in the first place. Clinicians and child welfare professionals would almost certainly agree that regardless of its consequences for antisocial behavior, becoming a victim of maltreatment is not a desirable end, and gene therapies alone will not protect children from this eventuality.

Limitations

There are several limitations to our findings. First, because our group of physically maltreated children was small in number, it was not possible to compare subtypes of maltreatment or to compare groups according to severity, chronicity, or precise developmental period of maltreatment (although all cases were necessarily confined to the infancy-toddlerhood and preschool years; Barnett, Manly, & Cicchetti, 1993). Whether some subtypes of maltreatment are more heritable than others is an empirical question that we could not address, although there is little theoretical rationale to suggest differential heritability.

Second, although our measure of physical maltreatment has shown good interreporter agreement in other samples, reports of physical maltreatment in our sample came from a single source (the mother). Ideally our results should be replicated in studies that use independent ratings of child physical maltreatment. On the one hand, the possibility that mothers concealed instances of physical maltreatment would have exerted a conservative effect on our findings by causing us to misclassify maltreated children as non-maltreated. On the other hand, it is possible that mothers' concealment of physical maltreatment may have artifactually inflated the estimate of shared environmental influence on physical maltreatment (because there were relatively few families in which twins were discordant for physical maltreatment). That said, strenuous efforts were made to enhance mothers' comfort in reporting instances of physical maltreatment, the physical maltreatment protocol we used has been validated in other samples, and the prevalence rates of physical maltreatment in our sample match those in other epidemiological samples, including ones in which a different physical maltreatment measure was used. Thus, it is unlikely that mothers concealed physical maltreatment for a great many cases.

Third, mothers were not asked to report who had maltreated their children. Our test of genetic transmission assumed that heritable characteristics of parents are correlated with children's experience of physical maltreatment, but in cases in which children are maltreated by someone outside the family, this assumption is less likely to be true.

Fourth, our data were collected in the United Kingdom. Although base rates of child maltreatment (Sedlak & Broadhurt, 1996; United Kingdom Department of Health, 2000) and antisocial behavior (Lahey et al., 2000; Melzer, Gatward, Goodman, & Ford, 2000) are similar in the United States and in the United Kingdom, more research is necessary to determine whether our findings will replicate in other populations.

Fifth, we followed the physically maltreated children in our cohort only as far as age 7 and so measured only childhood antisocial behaviors. However, the cycle of violence connects childhood physical maltreatment with violent crime in adulthood. There is strong continuity from childhood antisocial behavior emerging at ages 5 and 7 to adult violence (Moffitt et al., 2002), and this justifies our focus on childhood behavior. However,

research applying the design features of our study to adult twins is needed to test whether the causal effects of physical maltreatment we found will extend over many years of development.

Sixth, our findings do not elucidate the more proximal processes by which physical maltreatment leads to antisocial behavior. A range of processes may explain why children who are maltreated are subsequently at risk for antisocial behavior, including the modeling of adults' aggression (Bandura, 1973; Straus & Yodanis, 1996), the development of an aggression-prone social information processing style (Dodge et al., 1995), attributions of blame (McGee et al., 2001), maltreatment-induced traumatic brain injury (Miller, 1999), stress-induced neuroregulatory dysfunction (Cicchetti & Rogosch, 2001; De Bellis, 2001; Glaser, 2000), and potentially, difficulties in emotion recognition (Pollak, Cicchetti, Hornung, & Reed, 2000; Pollak & Sinha, 2002). By ruling out strict genetic accounts of the cycle of violence and establishing that physical maltreatment plays a causal role in the development of children's antisocial behavior, our findings undergird these efforts to identify more proximal mechanisms.

Conclusion

Despite claims to the contrary (Turkheimer, 2000), the nature–nurture debate is far from over, as evidenced by the recent publication and media scrutiny of books such as *The Nurture Assumption* (Harris, 1998) and *The Blank Slate: The Modern Denial of Human Nature* (Pinker, 2002), which question the degree to which parents influence their children's development beyond providing their children's genetic makeup. Conventional wisdom dictates that preventing physical maltreatment is a public health priority if people as a society care about breaking the cycle of violence and protecting children's well-being. However, if one cannot dismiss the possibility that children's antisocial behavior is primarily explained by the genes they inherit from their parents, one cannot say with confidence that preventing physical maltreatment would break the cycle (Rowe & Harris, 2002). Responding to these criticisms, our findings provide evidence that preventing physical maltreatment should be a public health priority because doing so is likely to reduce rates of antisocial behavior in the future. Given that 879,000 children per year are maltreated in the United States (National Child Abuse and Neglect Data System, 2002), physical maltreatment and its effects on children deserve our closest attention.

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