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The Association Between Conduct Problems and Maltreatment: Testing Genetic and Environmental Mediation

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Abstract

It is often assumed that childhood maltreatment causes conduct problems via an environmentally mediated process. However, the association may be due alternatively to either a nonpassive gene-environment correlation, in which parents react to children's genetically-influenced conduct problems by maltreating them, or a passive gene-environment correlation, in which parents' tendency to engage in maltreatment and children's conduct problems are both influenced by a hereditary vulnerability to antisocial behavior (i.e. genetic mediation). The present study estimated the contribution of these processes to the association between maltreatment and conduct problems. Bivariate behavior genetic analyses were conducted on approximately 1,650 twin and sibling pairs drawn from a large longitudinal study of adolescent health (Add Health). The correlation between maltreatment and conduct problems was small; much of the association between maltreatment and conduct problems was due to a nonpassive gene-environment correlation. Results were more consistent with the hypothesis that parents respond to children's genetically-influenced conduct problems by maltreating them than the hypothesis that maltreatment causes conduct problems.

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Keywords

Conduct problems; Childhood maltreatment; Mediation; Gene-environment correlation; Genetics; Environment

Introduction

Childhood maltreatment is considered a risk factor for a wide variety of children's negative outcomes and psychopathology, including conduct problems (Cicchetti and Lynch 1995). Experiencing physical abuse, neglect, and sexual abuse have repeatedly been associated with a wide variety of measures of children's externalizing problems, including conduct problems, violence, and substance use (Beeghly and Cicchetti 1994; Widom 1989; Trickett and Kuczynski 1986; Gelles and Strauss 1990; Flisher et al. 1997; Hibbard et al. 1990; Kilpatrick and Saunders 1999; Erickson and Engeland 2002). Researchers have argued that since the questions of *if* and *how much* childhood maltreatment and other risk factors are associated with adverse developmental outcomes are answered, the emphasis should shift to *how* these processes work (Rutter et al. 1997; Schuck and Widom 2003).

Hypothesized relations between maltreatment and conduct problems

Many researchers, clinicians, and laypersons have assumed that childhood maltreatment causes conduct problems in children despite a lack of conclusive evidence (Widom 1989; DiLalla and Gottesman 1991). This hypothesis is consistent with prospective studies demonstrating that maltreatment often precedes children's adverse outcomes (English et al. 2001; Widom 2000; Stouthamer-Loeber et al. 2001). However, at least two possible alternative explanations exist; both implicate genetic influences, not maltreatment, as the cause of conduct problems through either passive or non-passive gene-environment correlation.

Maltreatment may be related to antisocial behavior not because it has a direct environmental influence, but because of the correlation between the genetic influences on antisocial behavior and the "environmental" influence of childhood maltreatment (i.e., gene-environment correlation). Childhood maltreatment may occur more often in families where the parents and children have greater genetic risk for antisocial behavior. These hypotheses are important to consider, as heredity accounts for approximately 40% of population variance in antisocial behavior (Rhee and Waldman 2002).

Gene-environment correlations can be characterized as evocative, active, or passive (Plomin 1990; Neiderhiser et al. 2004). *Evocative* gene-environment correlations occur when genetically influenced characteristics of children, such as disruptive behaviors, evoke responses from the environment, such as harsh parenting. *Active* gene-environment correlations occur when a child selects environments, such as deviant peer groups, that are correlated with his or her genetically influenced characteristics, such as impulsivity. Evocative and active gene-environment correlations both reflect a correlation between children's genes and their environmental experiences and are indistinguishable in a twin study (Neiderhiser et al. 2004); therefore, they are both referred to as *nonpassive* gene-environment correlations in the present study. In contrast, *passive* gene-environment correlations reflect parents' tendency to parent in a certain way and children's vulnerability to behave in a certain way both being influenced by a common heritable vulnerability. For example, a heritable vulnerability to antisocial behavior may both predispose parents to maltreating their children and lead children to develop conduct problems. *Child effects* on a parenting behavior refer to any quality of a child (e.g. behavior, health, age, gender, etc.)

which is associated with that parenting behavior. Genetically-mediated child effects are synonymous with nonpassive gene-environment correlation.

Moffitt (1993) suggested that nonpassive gene-environment correlation plays a role in the etiology of antisocial behavior, noting that "Children's predispositions may evoke exacerbating responses from the environment" (p. 682). Similarly, several theories of the etiology of maltreatment posit that certain qualities of children, such as age, health, disruptive behaviors, prematurity, developmental difficulties, and retardation, influence the probability that caregivers will maltreatment them (Belsky 1993; Steele 1980; Vasta 1982). Empirical research supports this proposition. In a laboratory study, mothers reacted more negatively to unrelated conduct disordered boys than to unrelated non-conduct disordered boys, suggesting that children's conduct problems influence how mothers treat them (Anderson et al. 1986). Similarly, adoption studies showed that compared to adoptees without elevated genetic vulnerability to conduct problems, adoptees with elevated genetic vulnerability (i.e., an antisocial biological parent) evoked more harsh and inconsistent discipline and less nurturing and involved parenting (Ge et al. 1996) and more negative control (i.e. guilt induction, hostility, and withdrawal from relationship; O'Connor et al. 1998).

Moffitt (1993) suggested that passive gene-environment correlations also play a role in the etiology of antisocial behavior. "Vulnerable children are often participant to adverse homes and neighborhoods because their parents are vulnerable to problems too" (pp. 681–682).

Tests of environmental mediation using twin designs

Twin studies can be thought of as "natural experiments" because monozygotic (MZ) and dizygotic (DZ) twin pairs are born into the same family at the same time, but they share different proportions of their genes. A univariate behavior genetic analysis partitions population variance of a phenotype into additive genetic (A), shared environmental (C), and nonshared environmental (E) influences.

In a study of adolescent and mother reports of maternal behavior, Neiderhiser et al. (2004) described how univariate twin studies may be used to test for the presence of passive and nonpassive gene-environment correlation (Table 1) in parenting variables (i.e. maternal control, warmth, positivity, and negativity). In child-based designs (i.e., examining the parenting of MZ and DZ children), the additive genetic factor (A) captures variance in parental behavior associated with genetically influenced characteristics of children (genetically influenced child effects/nonpassive gene-environment correlation). The "shared environment" (C) captures the extent to which parents treat their children similarly regardless of whether they are MZ or DZ twins or full siblings, including true environmental influences. This factor also captures the effects of any passive gene-environment correlation. The nonshared environmental factor (E) includes differences in the way parents treat their children due to environmental influences and measurement error. Though not described by Neiderhiser, twin studies that also include full sibling pairs may also estimate the "twin environment" (T), which captures the extent to which parents treat their twin children similarly over and above the extent to which they treat their nontwin children similarly. Failing to measure T may lead to inflated estimates of C. Like C, T may capture passive gene-environment correlation. For example, a parent's genetic vulnerability to antisocial behavior may not be triggered until the arrival of a major stressor, by which time an older sibling may not be in the period of risk for maltreatment, though two younger twin children are. Neiderhiser demonstrated that the magnitude of passive gene-environment correlation can be estimated in a study using a parent-based design (i.e., examining the parenting of MZ and DZ parents).

In addition, researchers may conduct bivariate quantitative genetic analyses of twin data to partition the *covariation* between two phenotypes into genetic, shared environmental, and nonshared environmental influences. As in the univariate case, interpretations of bivariate analyses including a parenting variable must be modified to account for passive gene-environment correlation (Table 1). Covariation due to common genetic influences evinces a nonpassive gene-environment correlation. Common shared "environmental" influences may include both: (1) true common shared environmental influences between maltreatment and conduct problems, and/or (2) passive gene-environment correlation. Common nonshared environmental influences reflect: (1) differences in levels of maltreatment due to nonshared environmental influences that are correlated with differences in levels of conduct problems due to environmental influences, and/or (2) error in measurement of conduct problems that is correlated with error in measurement of maltreatment.

The effects of gene by shared environment interaction, if any, would be captured in A, and the effects of gene by nonshared environment interaction, if any, would be captured in E (Purcell 2002). The magnitude of parameter estimates is likely to be dependent on the reporter. Compared to parent reports, children tend to report that their parents treat each child more differently (Wade and Kendler 2000; Achenbach et al. 1987; Simonoff et al. 1995), possibly leading to higher estimates of E and lower estimates of C.

We know of three studies that examined genetically mediated child effects on maltreatment. A study of female twins from the Virginia Twin Registry estimated that genetically mediated child effects accounted for 9–21% of parent-reported and 33–40% of child-reported physical discipline (Wade and Kendler 2000). A twin study of parent-reported corporal punishment and physical maltreatment sufficient to injure the child estimated that genetically mediated child effects accounted for 25% of the variance in corporal punishment and 0–7% of the variance in physical maltreatment (Jaffee et al. 2004a, b). A study of retrospectively reported childhood maltreatment in the same sample examined in the present study indicated that approximately 1–6% of the variance in a composite maltreatment variable, 3–28% of variance in physical maltreatment, 3–32% of variance in neglect, and none of the variance in sexual maltreatment is due to genetically mediated child effects (Schulz-Heik et al. 2009).

Tests of environmental mediation of the relation between maltreatment and conduct problems

Jaffee et al. (2002, 2004a, b) have used twin designs to test for genetic and environmental mediation of the relationship between childhood maltreatment and conduct problems. Results of a bivariate genetic model indicated that most of the moderate observed association between corporal punishment and children's antisocial behavior was due to common genetic factors, suggesting that caregivers respond to children's genetically influenced antisocial behavior with corporal punishment (Jaffee et al. 2004a).

Separate analyses on the same sample tested whether physical maltreatment has an environmentally mediated effect on children's antisocial behavior independent of gene-environment correlation (Jaffee et al. 2004b). Expanding a DeFries-Fulker regression analysis of antisocial behavior to include a measure of physical maltreatment, they concluded that slightly less than half of the relationship between physical maltreatment and children's antisocial behavior at age seven was due to environmental factors, which was statistically significant. They concluded that physical maltreatment had an environmentally mediated effect on antisocial behavior.

A third study examined the relationship between witnessing domestic violence and children's externalizing and internalizing problems via an expanded univariate variance

components model of children's problems that included a measured variable of exposure to adult domestic violence (Jaffee et al. 2002). The authors concluded that domestic violence accounted for 2% of the variation in children's internalizing problems and 5% of children's externalizing problems, independent of genetic effects.

Validity of methods examining environmental mediation

Purcell and Koenen (2005) examined the validity of a variety of methods used to test for environmental mediation via methodological arguments and a simulation study. Their simulation results suggested that the method used in the test of the association between corporal punishment and antisocial behavior (Jaffee et al. 2004a)—the bivariate behavior genetic method—is a valid test of genetic and environmental mediation. Therefore the authors' conclusion that the relationship is largely genetically mediated is appropriate.

In contrast, both Purcell and Koenen (2005) and Turkheimer et al. (2005) explained that it is impossible to test for environmental mediation using a twin design when the environmental variable in question is *obligatory-shared*, or necessarily the same for twins reared together (e.g. socioeconomic status). Also, Purcell and Koenen's (2005) simulation results showed that adding a measured 'environmental' variable to a DeFries-Fulker regression model or a univariate variance components model leads to type I errors at an unacceptably high rate when gene-environment correlation is present. Therefore, the conclusions that the associations between physical maltreatment and antisocial behavior (Jaffee et al. 2004b) and domestic violence and children's problems (Jaffee et al. 2002) are environmentally mediated may not be warranted.

The present study

In the present study, we examined the association between maltreatment and children's conduct problems by conducting a bivariate behavior genetic analysis. This extends the literature by using a composite measure of maltreatment rather than corporal punishment only, by assessing maltreatment occurring at a later age than a previous study (Jaffee et al. 2004b), and by using methods that have been demonstrated to be valid. The meaning of each model parameter is summarized in Table 1. Common genetic influences would indicate a nonpassive gene-environment correlation, or parents responding to children's genetically-influenced conduct problems by maltreating them (i.e., genetic mediation). Common shared- and twin-environmental influences could reflect either passive gene-environment correlation or true environmental mediation. Common nonshared environmental influences would reflect common environmental influences that lead siblings to experience dissimilar levels of maltreatment and dissimilar levels of conduct problems (i.e., environmental mediation) or correlated measurement error. Significant common nonshared environmental influences would be consistent with the hypothesis that maltreatment causes conduct problems (although they would not be a proof of this hypothesis).

Method

Participants

Participants were young adults in the sibling sample of the National Longitudinal Study of Adolescent Health. Detailed explanation of the study design and sampling strategy are available elsewhere (Harris et al. 2006; Add-Health 2008). Participants were respondents to an in-school survey with a twin or a sibling (age 12–20 years) from the same biological parents and were randomly selected for inclusion in an in-home interview sample. Within the twin and full-sibling sample, a total of 3,988 individuals participated in this initial in-home interview, with 3,640 participating again 1 year later at wave II, and 3,297 participating again 5 years later at wave III. Mean ages at each of the three interviews were

16.2, 17.1, and 22.5, respectively. Of the 306 monozygotic twin pairs, 447 dizygotic twin pairs, and 1,248 full sibling pairs included in the present study, 47.4% were male. All participants were part of a twin or full sibling pair. The mean age difference between sibling pairs was 2.24 years (SD = 1.03). The ethnic composition, based on self-nomination, was 48.9% Caucasian, 21.1% African–American, 12.9% Hispanic, 6.4% Asian American, and 2.8% Native-American, and 7.8% other.

Measures

Childhood maltreatment—Maltreatment occurring prior to age 12 was assessed retrospectively during wave III using a four-item questionnaire. Two items assessed neglect (How often have your care-givers left you home alone when an adult should have been with you?; How often have your caregivers not taken care of your basic needs, such as keeping you clean or providing food or clothing?). One item assessed physical maltreatment (How often had your parents or other adult caregivers slapped, hit, or kicked you?). One item assessed sexual maltreatment (How often had one of your parents or other adult caregivers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?). The frequency, but not the severity, of each item was assessed via six response options: (1) never, (2) once, (3) twice, (4) three to five times, (5) six to ten times, (6) more than 10 times. While there was a long interval between the age at which maltreatment was assessed and the time period about which it was assessed, retrospective endorsements of child sexual maltreatment (Williams 1992), physical maltreatment (Berger et al. 1988), and other adverse childhood events (Brewin et al. 1993) are typically valid. Also, a review of the heritability of parenting and other ‘environmental’ variables (Kendler and Baker 2007) suggested that self-report and informant-report yielded similar heritability estimates, and the authors concluded that twin studies of environmental variables largely reflect ‘actual behavior’ rather than ‘only perceptions’.

In order to maximize reliability, a composite of physical maltreatment, neglect, and sexual maltreatment was examined, as diverse types of maltreatment tend to co-occur (Dong et al. 2004). Phenotypic correlations among the three forms of maltreatment ranged from 0.36 to 0.48, all of which were statistically significant at the $p < 0.05$ level. These correlations were consistent across male, female, and opposite sex pairs, with no correlation differing by more than 0.08 across these groups. The composite maltreatment variable was computed by first assigning a *z*-score that corresponded to each participant’s location in the distribution for each form of maltreatment. *z*-Scores were averaged across forms of maltreatment, and then this variable was transformed into an ordinal variable with five levels. This composite variable and each of the three individual forms of maltreatment was previously subjected to univariate behavior genetics decomposition (Schulz-Heik et al. 2009). Neglect was the most frequently endorsed form of maltreatment (41% endorsed at least one experience), followed by physical maltreatment (29%) and sexual maltreatment (5%).

Conduct problems—An 11-item scale was created to assess conduct problems by matching ADDHEALTH questions to DSM IV-TR (American Psychiatric Association 1994) Conduct Disorder criteria, replicating a measure used previously (Miles et al. 2002). Certain items included yes/no responses, others included a four-point Likert scale ranging from “never” to “5 or more times,” and some open-ended questions required participants to report the number of times they performed an act. Due to the small sample sizes for some responses, a dichotomous measure was constructed for each item to ensure that items were weighted equally.

The following seven symptoms were coded positively if the participant endorsed performing the act in the prior 12 months: (1) stealing, (2) breaking into someone else’s house, building,

or car, (3) destroying others' property, (4) forced sexual activity (assessed only in males), (5) use of a weapon, (6) stealing with confrontation of a victim, and (7) physically harming others. Four symptoms were coded positively if the participant met a specific threshold: (1) running away from home—more than twice; (2) lying to guardians—five or more times; (3) being truant from school—10 or more times; and (4) fighting physically—three or more times. A total composite conduct problems symptom score was calculated by summing the 11 dichotomous items. The Cronbach's alpha of the 11-item scale was 0.73. If four or more items were missing, the conduct problems sum was coded missing. Although a participant could omit four items and still indicate substantial conduct problems, the estimate of the participant's level of conduct problems would be unreliable.

To utilize all available and appropriate data, a criterion was counted as met if a positive response was given at either wave I or wave II. Conduct problem data from wave III were not incorporated because several items were judged to not be applicable to adults (e.g. truancy from school) and omitted at wave III. For participants who completed wave I and wave II, this yielded a score that was, on average, 36% greater than their wave I scores. Therefore, a multiplier of 1.36 was applied to the wave I scores of the 9.4% of participants who did not complete wave II. Those who did not complete Wave II reported slightly more conduct problems at Wave I than those who did (1.12 vs. 0.92, $p < 0.05$, Cohen's $D = 0.12$), and those who did not complete wave III had a slightly higher conduct problems symptom sum than those who did (1.40 vs. 1.23, $p < 0.05$, Cohen's $D = 0.08$),

Conduct problems were assessed at waves I and II regarding the prior year. Given that participants were asked questions regarding maltreatment occurring before age 12, the conduct problems assessed in the present study occurred after the maltreatment assessed in the present study.

Statistical analyses

Data management and descriptive statistic computation were conducted using SAS 9.1. Both the maltreatment variable and the conduct problems variable were positively skewed. Therefore, the data were analyzed assuming normal continuous liability distributions underlie the ordinal variables. This method retains the statistical advantages conferred by the normality assumptions for the underlying liability, retains an explicit mapping between the underlying liability and observed behavior, and correctly recovers the underlying correlations and parameter estimates (Stallings et al. 2001). Derks et al. (2004) also found that analyses of categorical data results in correct parameter estimates being recovered, although statistical power is decreased.

Frequencies of item endorsement for maltreatment differed significantly by sex for each form of maltreatment, and also by age for neglect. Also, the mean number of conduct problems differed significantly by sex. Therefore, sex-specific thresholds for physical maltreatment, sexual maltreatment, and conduct problems and age- and sex-specific thresholds for neglect were implemented via definition variable statements in Mx (Neale et al. 2003).

Mx uses the full information maximum likelihood method, which uses observed data to impute missing data (Carter 2006). Due to the different completion rates of waves I, II, and III, analyses included data on conduct problems for 99.6% of participants and on maltreatment for 80.6% of participants.

Maximum likelihood estimation techniques (Neale and Cardon 1992) were used in Mx (Neale et al. 2003) to calculate within-trait and cross-trait correlations and conduct univariate and bivariate (Fig. 1) behavior genetic models examining maltreatment and

conduct problems. Raw ordinal data were analyzed, and age- and sex-specific thresholds were included as definition variables. In analyzing ordinal data, Mx uses the multinormal threshold maximum likelihood fit function, which is described in detail in Neale et al. (2003).

The inclusion of full sibling pairs in the present study allowed us to estimate (co)variation due to the environment shared only by twins. Therefore, these models decompose (co)variance into that which can be accounted for by latent additive genetic factors (A), the shared environment (C), the twin environment (T), and the nonshared environment (E). A homogeneity model which constrains parameter estimates to be equal across genders was compared to a heterogeneity model in which estimates are free to vary across genders, as the family processes in maltreatment and the reporting thereof may differ by gender (Bugental and Shennum 2002; Sunday et al. 2008). Exploratory analyses were also conducted to test whether the pattern of results were similar for the covariation between specific forms of maltreatment (i.e., physical maltreatment and neglect) and conduct problems. We did not examine the covariation between sexual maltreatment and conduct problems because the prevalence of sexual maltreatment was low (i.e., 5.1%).

Results

Descriptive statistics

Table 2 presents the percentage of participants in each ordinal category of maltreatment and conduct problems. For maltreatment, “0” indicates no maltreatment, and higher numbers represent more severe maltreatment. For conduct problems, “0” indicates no conduct problem symptoms, “1” indicates one or two symptoms, “2” indicates three or four symptoms, “3” indicates five or six symptoms, and “4” indicates seven or more symptoms. Fifty-three percent of participants reported at least one experience of maltreatment, and 53% endorsed at least one conduct problem symptom.

Within- and cross-trait correlations

The phenotypic correlation between maltreatment and conduct problems was small but statistically significant ($r = 0.15$, $p < 0.05$). Table 3 shows cross-sibling within-trait and cross-trait correlations by gender and sibling type. Since MZ twins are more similar genetically than DZ twins and full siblings, greater cross-sibling correlations between MZ twins than between DZ twins and full siblings suggest genetic influence. Therefore, Table 3 suggests that children’s genes exert only a small influence on the maltreatment they report. Shared environmental influences are also suggested, as the DZ correlation is greater than half the MZ correlation. The MZ and DZ correlations are greater than the full sibling (FS) correlations, which is not surprising given that twins are the same age but FS are not, suggesting a role of the twin-specific environment. Most of the variance in maltreatment appears to be due to nonshared environmental influences, as the correlations for MZ twins, who share 100% of their genes and the shared environment, are well below one. Detailed behavior genetic decompositions of each form of maltreatment (i.e. physical maltreatment, neglect, and sexual maltreatment) are presented elsewhere (Schulz-Heik et al. 2009).

In contrast, a significant portion of the variance in conduct problems appears to be due to genetic influences, as conduct problem correlations between MZ twins are significantly higher than between DZ twins and full siblings. The DZ correlations are greater than FS correlations, indicating a role of the twin-specific environment. A large portion of conduct problem variance appears to be influenced by nonshared environmental factors, as MZ correlations are approximately 0.6.

Table 3 also shows that in general, the cross-trait cross-twin correlation between maltreatment and conduct problems in MZ twins is greater than that in DZ twins and full siblings. Therefore, although only a small portion of the variance in maltreatment appears to be associated with children's genetic vulnerabilities, a portion of the covariance between maltreatment and conduct problems is due to common genetic factors. The MZ cross-trait correlation is similar in magnitude to the phenotypic correlation between maltreatment and conduct problems, suggesting a lack of common nonshared environmental influences between maltreatment and conduct problems.

To what extent do genetic and environmental factors mediate the maltreatment-conduct problems relationship?

Model fitting results for the univariate and bivariate analyses are shown in Table 4. Individual parameters could be dropped from the univariate model examining maltreatment without a statistically significant decrease in fit, though the A and C parameters could not be dropped simultaneously. In contrast, only C could be dropped from the models of conduct problems. Due to the relatively low power to discriminate the effects of A *vs.* C in the maltreatment model, the saturated bivariate (ACTE) model was selected for interpretation. Each covariance path in the bivariate model could be dropped individually, but common genetic and shared environmental influences could not be dropped simultaneously.

The results of the univariate and bivariate behavior genetic analyses are shown in Table 5. A small portion of the variance in maltreatment appears to be associated with additive genetic influences (A), a moderate amount with shared- (C) and twin-environmental (T) influences, and a large amount with nonshared environmental influences and measurement error (E). A moderate portion of the variance in conduct problems is due to A, a negligible portion to C, a small to moderate portion to T, and a moderate portion to E.

The path estimates from the bivariate model displayed in Fig. 2 were used to derive the percentage of covariance and the phenotypic correlation due to each factor, which are shown in the bottom half of Table 5. The total expected phenotypic correlation of 0.14 results from the aggregate contributions of +0.10 (0.18 × 0.56) from common genetic factors, +0.07 ([0.42 × 0.08] + [0.35 × 0.11]) from the combination of common shared- and twin-environmental factors, and −0.03 (0.82 × −0.03) from common nonshared environmental factors. Therefore, much of the covariation between maltreatment and conduct problems is due to shared genetic factors (70%), a considerable portion to the combination of common shared-and twin-environmental factors (50%), and a negative contribution stems from nonshared environmental factors (−18%). The small negative value for nonshared environmental factors suggests that these factors alone would lead to a small negative correlation between maltreatment and conduct problems.

Similar patterns were found when results were examined separately for males and females, and a model in which parameter estimates were free to differ across genders did not fit better than the model in which parameter estimates were constrained across genders ($\Delta\chi^2_{(10)} = 8.72$, $p = 0.56$). Exploratory analyses examining the association between the individual forms of maltreatment (i.e., physical maltreatment and neglect) and conduct problems also indicated similar results: moderate to large portions of the covariance due to common genetic factors, small to moderate portions to common shared- and twin-environmental factors, and at most small portions to common nonshared environmental factors.

Discussion

The present study examined the genetic and environmental mediation of the well-established relationship between childhood maltreatment and children's conduct problems. It is one of

few studies to address this question using the bivariate behavior genetic method, which has been shown to provide a valid test of environmental mediation (Purcell and Koenen 2005). It examined a genetically informative subsample of the National Longitudinal Study of Adolescent Health, a large, longitudinal study of adolescent health and behavior.

Although the effects of children's genetic vulnerabilities on the experience of maltreatment in this sample appear to be small (Schulz-Heik et al. 2009), the present study suggests that a relatively large portion of the small but statistically significant phenotypic correlation between maltreatment and conduct problems may be due to non-passive gene environment correlation. This suggests that a significant portion of the correlation between maltreatment and conduct problems may not be due to maltreatment causing conduct problems, but rather to parents responding to children's genetically-influenced behaviors related to conduct problems by maltreating them.

In contrast, there appears to be only a very low or even negative correlation between the nonshared environmental influences on maltreatment and conduct problems. This suggests that although this parameter explains a large portion of the variance in maltreatment and conduct problems, it does not contribute to their covariance. This parameter reflects environmental influences and does not capture genetic influences. Therefore, this parameter estimate is not consistent with the environmental mediation hypothesis.

However, almost half of the covariance is due to common shared- and twin-"environmental" factors. These factors may reflect passive gene-environment correlation, or children's genetic factors being correlated with the environments they experience due to the fact that both are related to parents' genetic factors. Alternatively, these factors may reflect true environmental effects. These processes cannot be distinguished in a child-based twin study. Therefore, while these results do not support environmental mediation, we cannot reject the hypothesis that the relationship between maltreatment and conduct problems is in part environmentally-mediated.

Results of the present study are consistent with prior research suggesting that most of the moderate association between corporal punishment and conduct problems in a British sample is due to common genetic factors (Jaffee et al. 2004a). In contrast, no evidence was found for an effect of children's genetic vulnerability on physical maltreatment in the British study. The fact that our results are closer to Jaffee et al.'s results regarding corporal punishment than physical maltreatment are not surprising; Jaffee et al. used a more severe operational definition of physical maltreatment (i.e., enough to cause injury), whereas the present study's physical maltreatment measure included relatively mild maltreatment and corporal punishment. Also, Jaffee et al. used parent report, which tends to produce lower estimates of A and E and higher estimates of C compared to the child report method used in the present study (Achenbach et al. 1987; Hewitt et al. 1992; Wade and Kendler 2000).

Differences in results also reflect the fact that Jaffee et al. (2004a, b) assessed corporal punishment and physical maltreatment when the child was 5-year-old. In contrast, the present study retrospectively assessed maltreatment occurring prior to age 12 when participants were an average age of 22 years old. Thus, measurement of influences on maltreatment may be obfuscated by influences on its perception and retrospective recall in the present study.

The correlation between maltreatment and conduct problems was small, and the bivariate analyses lacked power to reliably distinguish common genetic factors from common shared environmental factors mediating this correlation. Therefore, the present results must be interpreted cautiously. Several factors may contribute to this lack of power. Participants reported at different times about maltreatment and conduct problems they experienced at

different periods of their life. Maltreatment may have been assessed unreliably because it was reported retrospectively, only four items were used, and respondents were required to define whether they were able to be left alone, what constitutes a basic need, and whether an experience was sexual. The physical maltreatment item captured relatively mild maltreatment and corporal punishment, which may minimize its relationship with conduct problems. Although this study included approximately 4,000 participants, only 81% of those who completed items about conduct problems (Waves I & II) also completed maltreatment items (Wave III), and those who did not complete wave III had slightly more conduct problem symptoms than those who did, as incarcerated individuals were not assessed at wave III (Haberstick et al. 2005). Further, many participants did not endorse any maltreatment or conduct problems.

Given these limitations, additional studies of this important relationship are needed. Future research should test whether these findings replicate in other samples. Selected samples with higher rates and greater severity of maltreatment and conduct problems may be particularly informative, although recruiting highly selected twin samples would be difficult. Studies of data collected from multiple reporters would decrease measurement error and reduce any inflation in correlation due to common rater bias across measures. Future research should also assess the extent to which common shared- and twin-environmental influences on the relationship between maltreatment and conduct problems reflect passive gene-environment correlation and true environmental mediation. This could be done by studying twin pairs who are parents, which would test the effect of parents' genes on the provision of parenting rather than the effect of children's genes on the elicitation of parenting.

Also, theory and empirical evidence suggest that children's phenotypes other than conduct problems, including health, disruptive behaviors, and developmental difficulties, may evoke maltreatment (Belsky 1993; Steele 1980; Vasta 1982; Needell and Barth 1998; Sidebotham et al. 2003; Spencer et al. 2006). Assessing the direction of causality of the relationship between these other phenotypes and maltreatment may further elucidate the etiology of maltreatment.

In conclusion, the results of the present study are consistent with the hypothesis that a large part of the association between maltreatment and conduct problems is the result of a nonpassive gene-environment correlation in which caregivers respond to children's conduct problems by maltreating them. The remaining part of the association may reflect either environmental mediation or the passive gene-environment correlation resulting from common genetic influences on parents' potential to maltreat and children's potential for conduct problems. Preventing maltreatment is important in its own right, but this preliminary study suggests that it may be less important as a means to prevent development of conduct problems than is often assumed. Rather, this study's results suggest that interventions with parents intended to eliminate maltreatment should address how to respond to children's conduct problems. Such interventions may both reduce maltreatment by giving caregivers more appropriate strategies and reduce children's conduct problems. This is consistent with the finding that Parent Training, which provides parents with appropriate strategies for responding to children's conduct problems, both is an efficacious treatment for conduct disorder and provides a variety of benefits to caregivers (Kazdin 2005; Adams 2001).

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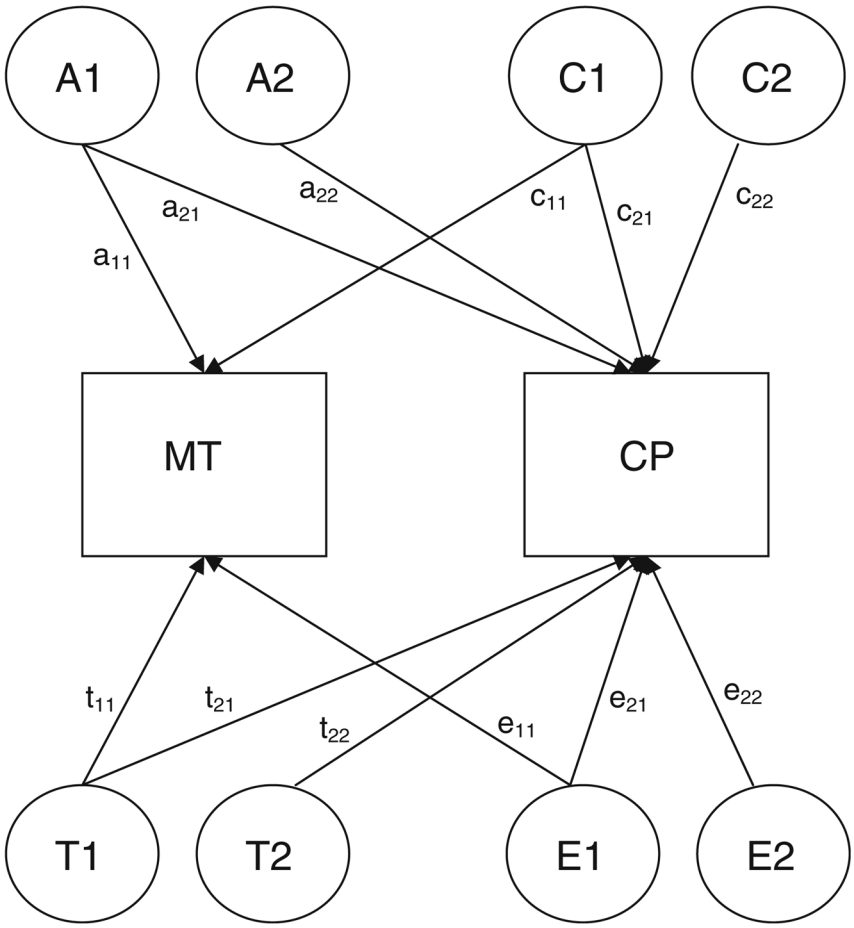

Fig. 1.
Bivariate model. A, additive genetic influences; C, shared environmental influences; T, twin environmental influences; E, nonshared environmental influences; MT, maltreatment; CP, conduct problems

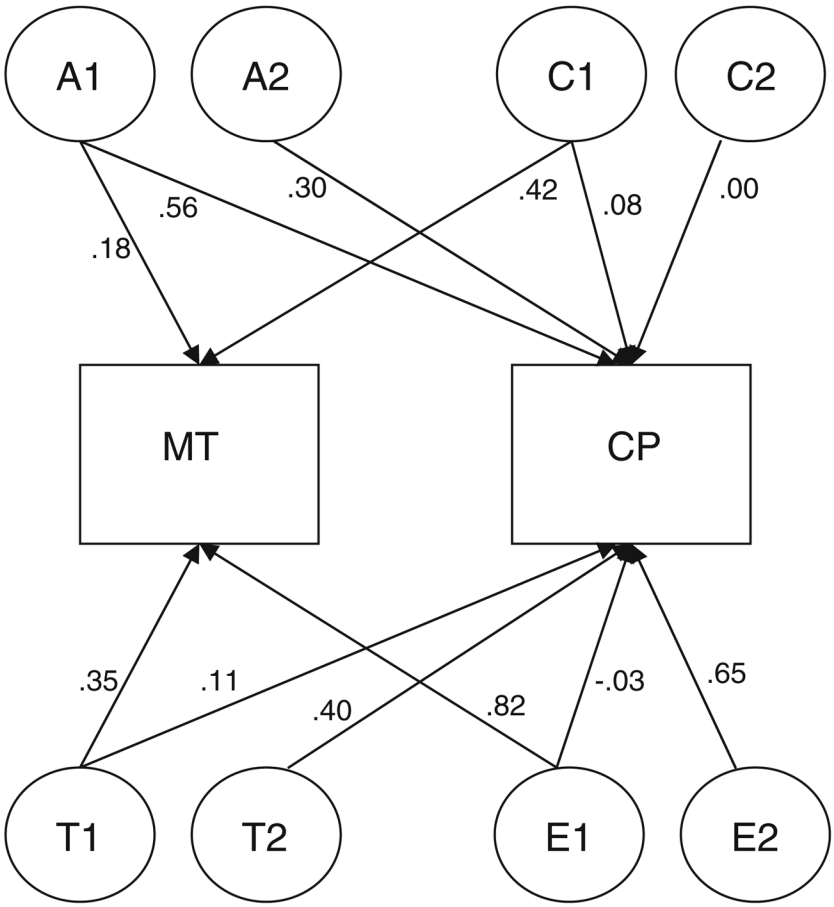

Fig. 2.

Bivariate model with path estimates. A, additive genetic influences; C, shared environmental influences; T, twin environmental influences; E, nonshared environmental influences; MT, maltreatment; CP, conduct problems. Mx was not able to estimate reliable confidence intervals

Table 1

Parameters in decomposition of parenting variables

Abbreviation	A	C	T	E
Name	Genetic	Shared “environment”	Twin “environment”	Nonshared environment
Meaning	Nonpassive gene environment correlation/ genetically mediated child effects	Shared environment and/or passive gene environment correlation	Twin environment and/or passive gene environment correlation	Nonshared environment and/ or measurement error
Twin/sib correlation	1 if MZ, 0.5 if DZ or sib	1	1 if MZ or DZ, 0 if sib	0
Mediation	Genetic	Indeterminate	Indeterminate	Environmental

MZ, monozygotic; DZ, dizygotic; sib, nontwin full sibling

Table 2

Percentage of participants in each ordinal category of maltreatment and conduct problems

Level	Maltreatment	Conduct problems
0	46.9	47.2
1	25.5	34.3
2	10.6	12.0
3	10.5	4.0
4	6.6	2.4

Conduct problem of 0 indicates no symptoms, 1 indicates 1–2 symptoms, 2 indicates 3–4 symptoms, 3 indicates 5–6 symptoms, 4 indicates 7+. Maltreatment of 0 indicates no maltreatment, and higher values indicate increasingly frequent and/or diverse maltreatment

Table 3

Cross-sibling within-trait and between-trait correlations by gender and sibling type

		MZ	DZ	FS
Within-trait correlations				
MT	All	0.34 (0.21–0.46)	0.31 (0.20–0.42)	0.19 (0.12–0.27)
	Male	0.22 (−0.01–0.42)	0.27 (0.06–0.46)	0.17 (0.03–0.32)
	Female	0.41 (0.25–0.55)	0.36 (0.11–0.55)	0.21 (0.07–0.34)
	OS	–	0.32 (0.14–0.47)	0.20 (0.09–0.30)
CP	All	0.61 (0.50–0.69)	0.36 (0.25–0.46)	0.20 (0.15–0.26)
	Male	0.56 (0.42–0.67)	0.37 (0.15–0.54)	0.24 (0.12–0.35)
	Female	0.66 (0.51–0.77)	0.42 (0.21–0.59)	0.15 (0.02–0.27)
	OS	–	0.31 (0.14–0.46)	0.20 (0.09–0.29)
Cross-trait correlations				
MT–CP	All	0.17 (0.08–0.25)	0.13 (0.05–0.20)	0.08 (0.03–0.14)
	Male	0.21 (0.08–0.33)	0.06 (−0.08–0.20)	0.12 (0.02–0.21)
	Female	0.13 (0.02–0.24)	0.05 (−0.10–0.20)	0.06 (−0.04–0.16)
	OS	–	0.21 (0.09–0.32)	0.08 (0.08–0.15)

95% confidence intervals are included in parentheses

MT maltreatment, *CP* conduct problems, *MZ* monozygotic twins, *DZ* dizygotic twins, *FS* full sibs

Table 4

Model fitting results

	−2LL	*df*	AIC	$\Delta\chi^2$	Δ*df*	*p*
Maltreatment univariate model						
ACTE	8948.38	3,224	2500.38			
ACE	8951.24	3,225	2501.24	2.86	1	0.09
ATE	8951.59	3,225	2501.59	3.21	1	0.07
CTE	8948.49	3,225	2498.49	0.11	1	0.75
AE	8952.91	3,226	2500.91	4.53	2	0.10
CE	8953.60	3,226	2501.60	5.22	2	0.07
TE	8974.51	3,226	2522.51	26.13	2	<0.01
E	9024.62	3,227	2570.62	76.24	3	<0.01
Conduct problems univariate model						
ACTE	9256.36	3,985	1286.36			
ACE	9265.01	3,986	1293.01	8.65	1	<0.01
ATE	9256.36	3,986	1284.36	0.00	1	1.00
CTE	9267.30	3,986	1295.30	10.94	1	<0.01
AE	9265.01	3,987	1291.01	8.65	2	0.01
CE	9292.96	3,987	1318.96	36.60	2	<0.01
TE	9299.65	3,987	1325.65	43.29	2	<0.01
E	9405.53	3,988	1429.53	149.17	3	<0.01
Maltreatment—conduct problem bivariate model						
Saturated	18150.28	7,205	3740.28			
Drop A_{21}	18151.43	7,206	3739.43	1.15	1	0.28
Drop C_{21}	18150.66	7,206	3738.66	0.37	1	0.54
Drop T_{21}	18151.01	7,206	3739.01	0.73	1	0.39
Drop E_{21}	18150.77	7,206	3738.77	0.49	1	0.49
Drop A_{21} & C_{21}	18161.10	7,207	3747.10	10.82	2	<0.01

−2LL, −2 log likelihood; *df*, degrees of freedom; AIC, Akaike's information criterion; $\Delta\chi^2$, difference in chi square from ACTE model; *p*, probability; A, additive genetic influences; C, shared environmental influences; T, twin environmental influences; E, nonshared environmental influences

Table 5

Magnitude of genetic, shared environmental, twin environmental, and nonshared environmental influences

	A	C	T	E
Variance in maltreatment				
All	0.06	0.17	0.12	0.66
Male	0.01	0.17	0.07	0.75
Female	0.05	0.19	0.17	0.59
Variance in conduct problems				
All	0.41	0.00	0.17	0.42
Male	0.45	0.03	0.08	0.44
Female	0.34	0.00	0.29	0.37
Correlation between maltreatment and conduct problems				
All	0.10	0.03	0.04	−0.03
Male	0.12	0.04	0.05	−0.04
Female	0.08	0.03	0.02	0.00
% of Covariance between maltreatment and conduct problems				
All	70	22	27	−18
Male	74	23	30	−27
Female	62	19	15	3

Variance estimates are derived from univariate models. Covariance and correlation estimates are derived from the bivariate model

A genetic influences, *C* shared environment, *T* twin environment, *E* nonshared environment