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Investigation of Genetically Mediated Child Effects on Maltreatment

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Abstract

Theory and empirical evidence suggest that children's genetically influenced characteristics help to shape the environments they experience, including the parenting they 'receive'. The extent of these genetically-mediated child effects on childhood maltreatment is not well known. The present study estimates the magnitude of genetically mediated child effects on maltreatment in 3297 twins and siblings who were part of a large nationally representative sample of adolescents (ADD Health). Participants in early adulthood retrospectively reported their experiences of physical and sexual maltreatment and neglect. Results are consistent with small genetically-mediated child effects on physical maltreatment and neglect, and none on sexual maltreatment, and all three forms of maltreatment are influenced mainly by idiosyncratic individual circumstances.

In the past, a dominant assumption in the childrearing literature has been that the relationship between children and their environment is unidirectional – that environments shape children. Nonetheless, a minority of developmental theorists has noted the need to consider the possibility that children both shape their environments and are influenced by them (e.g. Bell, 1968; Brofenbrenner 1986). This is true of the parenting that children receive, in particular. Bell (1968) suggested that parents do not have fixed parenting techniques, but rather a repertoire of potential actions. Activation of elements in the repertoire requires both cultural pressures and stimulation from the object of acculturation. Brofenbrenner (1986) similarly emphasized that parenting behavior is influenced by both proximal and distal influences in parents' histories and environments as well as children's evocative behavior.

In particular, several theorists have proposed a role of children's evocative qualities in the etiology of childhood maltreatment (Belsky, 1993; Steele, 1980; Vasta, 1982). They argued that maltreatment is associated with a combination of parent characteristics (such as their personality, psychological resources, their own abuse history, and unreasonable expectations for children), child characteristics (such as their age, health, disruptive behaviors, prematurity, developmental difficulties, and retardation), and community/cultural characteristics (such as shared values, the availability of social support, and stressors such as unemployment and marital instability). The role of these hypothesized child effects on maltreatment has been minimized in the relevant literature despite an absence of evidence suggesting a lack of child effects. This may be due to a fear that evidence of variance in maltreatment associated with child characteristics (i.e. child effects) would erroneously be interpreted as suggesting that maltreatment is children's fault. In reality, however, evidence of child effects would merely document that there are qualities in children that raise the probability that caregivers will maltreat them.

Evidence garnered from a variety of methods converges to demonstrate that children can shape the parenting they receive. An early indication came from observation of a mother who responded differently to two children she adopted simultaneously of the same age and gender (Yarrow, 1963). In a laboratory study, mothers of conduct disordered boys as well as mothers of non-conduct disordered boys reacted more negatively when interacting with unrelated conduct disordered boys than when interacting with unrelated non-conduct disordered boys (Anderson, Lytton, & Romney, 1986). In another laboratory study, the level of dependency girls were manipulated to display positively predicted how interactive and controlling parents behaved toward them (Osofsky & O'Connell, 1972).

Further evidence that children influence the parenting they receive comes from twin and adoption studies of putatively environmental influences. Recently, Kendler and Baker (2007) conducted a review of 55 independent studies examining the magnitude of genetic influences on putatively environmental constructs such as stressful life events, parenting behavior, family environment, and peer interactions and found a mean heritability of 27%. Such evidence of genetic influences on constructs usually thought of as environmental is evidence of gene-environment correlation, or genetic variance among children being correlated with individual differences in their experiences.

There are three types of gene-environment correlations: evocative, active, and passive (Neiderhiser et al., 2004; Plomin, DeFries, & Loehlin, 1977). Evocative gene-environment correlations occur when genetically influenced characteristics of children evoke responses from the environment. For example, genetically influenced disruptive behaviors may provoke caregivers to maltreat them. Active gene-environment correlations occur when a child selects environments that are correlated with his or her genetically influenced characteristics. For example, a child with high genetic proclivity towards reading may choose to spend time in environments in which books are available, such as libraries. Although evocative and active gene environment correlations are fundamentally different processes, both indicate a correlation between children's genes and their environmental experiences and are indistinguishable in a twin study (Neiderhiser et al., 2004); therefore, they are considered together as *nonpassive* gene-environment correlations in the present study. In contrast, passive gene-environment correlations arise because parents' genes are correlated with both the environment they create for their children and their children's genes. For example, parents with a genetic vulnerability to antisocial behavior may maltreat their children and also pass on genetic vulnerability to antisocial behavior to their children; their children's genetic vulnerability to antisocial behavior and their experience of maltreatment could be positively correlated, but without one directly causing the other.

Researchers conduct twin studies to examine the relative magnitude of genetic and environmental influences on phenotypes of interest. Twin studies take advantage of the fact that both monozygotic (MZ) and dizygotic (DZ) twin pairs are born into the same family at the same time, yet they share different proportions of their genes (100% in MZs and 50% on average in DZs). The twin study allows researchers to partition the variance of a phenotype into those explained by three influences: genetic influences (A), which reflect differences in the similarity of MZ and DZ twin pairs, shared environmental influences (C), which reflect familial correlation not due to genetic factors, and nonshared environmental influences (E), which reflect differences between twins not due to genetic factors.

In a study of genetic variance in mothering, Neiderhiser et al. (2004) described how univariate behavior genetic analyses of parenting variables are used to test for the presence of passive or nonpassive gene-environment correlation and how the interpretations of the three factors (i.e., A, C, and E) must be modified to account for the possibility of passive gene-environment correlation. Evidence of genetic influences on child-reported parenting means that MZ twin

pairs, who share 100% genetic similarity, are reporting more similar parenting experiences than DZ twin pairs, who on average share 50% genetic similarity. A, the genetic factor, captures variance in parenting behavior due to genetically influenced characteristics of children (i.e., genetically influenced child effects/nonpassive gene-environment correlation). Importantly, this design does not include a test of which specific child characteristics mediate the relationship between children's genetics and the measured variable. C, the "shared environmental" factor, captures the extent to which parents treat their children similarly regardless of whether they are MZ or DZ twins. Although C includes any environmentally mediated similarities in parenting across siblings, it also includes the effect of any passive gene-environment correlation, or the parents' genetic influences that make them more likely to treat two children similarly regardless of the children's genetic similarity (i.e., whether they are MZ or DZ twins). In studies including both twins and ordinary full siblings, an additional shared environmental factor, T, may be invoked to capture the increased environmental similarity of twins compared to ordinary full siblings; unlike twins, full siblings are different ages, born at different times, and, possibly, in different parental or other family circumstances. Although C and T may include parent effects on maltreatment, they may also reflect child effects; they may reflect stressors that influence both parents and children that are shared by sibling/twin pairs, such as deviant peers, neighborhood influences, and school influences. E, the nonshared environmental factor, includes any environmentally mediated differences in parenting that may be mediated by the child's behavior or reflect idiosyncratic parenting behavior) and measurement error.

Genetically uninformative longitudinal designs have been used to demonstrate interactive, coercive cycles between parents and children in which children's aggressive behavior elicit assertive or coercive parenting, which in turn elicits aggressive behavior in the children, thereby perpetuating a cycle (e.g. Patterson, Bank, & Stoolmiller, 1990). In these studies, the extent to which children's behavior is a precursor to or consequence of parenting is unclear. A strength of the genetically informative study of parenting is that a demonstration of genetically mediated child effects on parenting is clear evidence that children bring their own qualities to the interaction beyond that which is shaped by caregivers. It is important to note, however, that genetic effects are not, as they are often misinterpreted to be, immutable. Rather, genes contribute to a child's vulnerability to or probability of demonstrating a particular behavior, as do environmental influences. A weakness of the behavior genetic study of parenting is that the magnitude of environmentally mediated child effects cannot be directly estimated, given that environmental influences on parenting may be child or parent effects. Therefore, total child effects on parenting may be greater than the genetically mediated child effects.

Twin and adoption studies have been applied to parenting in order to assess the extent to which children's genetics influence the parenting they receive. Of the behavior genetic studies of putatively environmental influences reviewed by Kendler and Baker (2007), 12 examined parenting (e.g., negativity, warmth, control, protectiveness). On average, approximately one quarter of the variance in parenting was attributed to children's genetic influences, but there was variability in the results. The authors noted that measures of positive emotional quality of the parent-child relationship were more heritable than measures of discipline styles; studies of parental warmth showed the greatest heritability (34%–37%), followed by studies of protectiveness (20%–26%), then by studies of control and negativity (12–17%). The authors inferred that positive emotionality in parent-child relationships may be strongly influenced by genetically influenced temperament. In contrast, parents may learn disciplinary styles during their life experiences, and attempt to apply these styles equally to all their children. Therefore, although theories regarding the etiology of physical maltreatment and neglect reviewed above (e.g., Belsky, 1993; Steele, 1980; Vasta, 1982) suggest that the magnitude of child effects is nontrivial, Kendler and Baker's (2007) results suggest that the influence of children's genetics on maltreatment may be small.

We know of only two studies examining genetically mediated child effects on measures closely related to childhood maltreatment. A study of Caucasian female twins drawn from the large, population-based Virginia Twin Registry (Wade & Kendler, 2000) examined genetically mediated child effects on both retrospective child- and parent- reported physical discipline occurring before age 17. Consistent with results of prior research, (Achenbach, McConaughy, & Howell, 1987; Plomin, DeFries, & Loehlin, 1994; Simonoff, Pickles, Hewitt, & Silberg, 1995), parents' reports of parenting were more similar across siblings than children's reports of parenting. For parent report of physical discipline, genetically mediated child effects accounted for 9–21%, and the "shared environment" accounted for 62–72% of the variance. In contrast, for child report of physical discipline, genetically mediated child effects accounted for 33–40%, and the "shared environment" accounted for 21–28% of the variance. The authors speculated that the difference in results across reporters may reflect a combination of three processes. First, the parent report results may reflect a desirability bias, i.e., it may be desirable for parents to report treating their children equally. Second, parents may be reporting general discipline strategies more so than actual behavior. Third, children may be highly sensitive to perceived differences in how they are treated. In addition, any rater bias (or the tendency to overrate or underrate physical discipline for both twins) on the part of parents would inflate estimates of C (Hewitt, Silberg, Neale, & Eaves, 1992). In contrast, measurement error in one child's report is likely to be uncorrelated with measurement error in the other twin's report, increasing E and decreasing A and C.

Jaffee and colleagues (Jaffee, Caspi, Moffitt, & Taylor, 2004) examined the genetic and environmental influences on physical maltreatment sufficient to injure a child and on corporal punishment *via* a maternal-report twin study conducted when the twins were 5 years old. Genetically-mediated child effects/nonpassive gene-environment correlation accounted for 25% of the variance of corporal punishment. In contrast, nonpassive gene-environment correlation accounted for 0–7% of the variance in physical maltreatment and shared environmental influences that may include passive gene-environment correlation accounted for 94% of the variance. The authors concluded that difficult children may provoke corporal punishment, but factors causing physical maltreatment are more likely to be found within the caregiver or family environment. However, Wade and Kendler's results suggest that the magnitude of shared environmental influences may have been overestimated in the Jaffee et al. study. Mothers reported that their children experienced extremely similar levels of physical maltreatment, which may reflect reporting bias that over-estimates the magnitude of the shared environment (Hewitt et al., 1992). Alternatively, the use of a severe threshold in the operational definition of physical maltreatment (i.e., injury) may have captured behaviors that are less likely to be responses to children's behavior.

The present study examined the etiology of three forms of maltreatment (physical maltreatment, neglect, and sexual maltreatment) reported by the child. We know of no research examining genetically-mediated child effects on neglect or sexual maltreatment. Hypotheses and research regarding child effects on maltreatment have focused primarily on physical maltreatment. However, it is possible that the same child characteristics that frustrate parents and lead to parental anger and withdrawal may be expressed as either physical maltreatment or withdrawal of parental attention and care. Also, both physical maltreatment and neglect may reflect negative aspects of the parent-child relationship that may involve parents' learned beliefs about acceptable ways to treat children. We are not aware of hypotheses about child effects on sexual maltreatment, and it is difficult to imagine what genetically influenced child qualities would be associated with sexual maltreatment.

Further, although it is well established that diverse types of maltreatment toed to co-occur (e.g. Dong et al., 2004), we know of no studies testing whether the genetic and environmental influences on one form of maltreatment are shared with the influences on another form. In the

present study, multivariate analyses were conducted to examine the degree to which the covariation among different forms of maltreatment is due to common genetically mediated child effects, common shared environmental influences (which may include passive gene-environment correlation), or common nonshared environmental influences.

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 utilized for both the full sample and this genetically informative sub-sample are available elsewhere [Harris, Halpern, Smolen, & Haberstick, 2006; National Longitudinal Study of Adolescent Health, http://cpc.unc.edu/addhealth]. Initially, participants responded to an in-school survey in grades 7–12. Those who reported that they had a twin or a sibling (ages 12–20 years) with the same biological mother and father were identified and subsequently randomly selected for inclusion in an in-home interview sample. Within the twin and full-sibling sample, a total of 3,982 individuals participated in this initial in-home interview, with 3,297 participating 6 years later at wave III. Mean ages and ranges at these interviews were 16.2 (11 – 21), and 22.5 (18 – 27), respectively. The mean age difference between full-sibling pairs was 2.2 years (range 0.7 – 6.8). Self-reported maltreatment was assessed during wave III only. Of the individuals from 271 MZ twin pairs (128 male-male and 143 female-female), 397 DZ twin pairs (119 male-male, 104 female-female, and 174 opposite sex), and 1,143 full sibling pairs (301 male-male, 340 male-female, and 502 opposite sex) who responded to the maltreatment items at wave III, 47.7% were male. 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. Half siblings were excluded from the analyses because they reported more maltreatment and had greater variance in maltreatment than other participants (results are available on request).

Measures

Maltreatment by parents or other adult caregivers before entry into sixth grade (age 12) was assessed by retrospective reports using a four-item questionnaire administered during the wave III of data collection when the participants were young adults. Two items assessed neglect (How often had your parents of adult care-givers left you home alone when an adult should have been with you?; How often had your parents or other adult care-givers 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 care-givers slapped, hit, or kicked you?). One item assessed sexual maltreatment (How often had one of your parents or other adult care-givers 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. These items are not expected to under-estimate maltreatment as much as questions that exclusively rely on respondents to define maltreatment (e.g. ‘Were you sexually maltreated?’). Nonetheless, there are several weaknesses in these items. They may underestimate maltreatment, like all maltreatment items that require participants to define sensitive terms themselves (Carlin, Kemper, Ward, Sowell, Gustafson, & Stevens, 1994; Carlson, 1997; Silvern, Waelde, McClintic, & Kaersvang, 2000). The neglect items require respondents to define when they are able to be left alone, and what constitutes a basic need. The physical maltreatment item may capture corporal punishment and does not query certain severely violent acts that are often included in physical abuse self-reports, such as being beaten repeatedly, choked, assaulted with a weapon, etc. As a result, this item may capture more mild physical maltreatment and corporal punishment. Sexual maltreatment may

be underreported because the item includes the word ‘sexual’, and a child may experience an act as either violent or affectionate, but not sexual.

Statistical Analyses

Data management and computation of descriptive statistics were conducted using SAS 9.1 (SAS Institute, 2002). Distributions of responses to the maltreatment variables were markedly skewed. Therefore, the maltreatment variables were treated as ordinal variables, and then analyzed assuming that 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). Age- and sex-specific thresholds were used. In order to estimate genetic and environmental factors influencing maltreatment in general, a composite maltreatment variable was computed. A z-score was computed for each participant for each form of maltreatment, and then these scores were averaged. This variable was treated as an ordinal variable with five levels.

We used maximum likelihood estimation techniques (Neale & Cardon, 1992) to test univariate and multivariate models examining maltreatment, physical maltreatment, neglect, and sexual maltreatment. These analyses were conducted using Mx (Neale, Boker, Xie, & Maes, 2002). The univariate models and multivariate models decompose the variance in children’s experiences of physical maltreatment, neglect, and sexual maltreatment and the covariance among the three forms of maltreatment, respectively, into that explained by latent additive genetic factors (A), the shared environment (C), the twin environment (T), and nonshared environmental (E) effects.

Sex differences in the magnitude of genetic and environmental influences were examined, as the family processes in maltreatment or the reporting thereof may differ by gender (Bugental & Shennum, 2002; Sunday et al., 2008). First, homogeneity models (figure 1), in which the magnitude of genetic and environmental influences is constrained to be equal across genders, were tested. Second, heterogeneity models, which assume that the same genetic and environmental influences influence both genders but that their effects may differ in magnitude, were tested. Because opposite-sex DZ correlations were not lower than same-sex DZ correlations, models that assume different genetic factors influencing males and females were not considered. To evaluate each of the parameters in the univariate models, reduced models in which each parameter was dropped from the saturated (ACTE) model individually and in conjunction with other parameters were fit.

Results

Descriptive Statistics

Table 1 presents the percentage of respondents in each ordinal category (0 representing no maltreatment and higher numbers representing more frequent maltreatment) for composite maltreatment, physical maltreatment, neglect, and sexual maltreatment for all participants, males, and females. Forty one percent of participants reported being neglected at least once. Physical maltreatment was reported by 29% of respondents, and sexual maltreatment was endorsed by 5% of respondents. Overall, 52% of respondents reported at least one experience of maltreatment. The rates of physical maltreatment and neglect suggest that the infractions captured by these measures may have included relatively mild ones.

Correlations

Table 2 shows cross-sibling within-trait correlations by sibling type and gender. The cross-sibling within-trait correlations could not be calculated separately by gender for sexual maltreatment given the very small number of respondents who reported sexual maltreatment (e.g., among MZ male twin pairs, only 8 individuals endorsed sexual maltreatment). Since MZ twins share 100% genetic similarity whereas DZ twins and full siblings share on average 50% genetic similarity, greater cross-sibling within-trait correlations among MZ twins than among DZ twins and full siblings suggest genetic influence on a trait. Children's genes appear to have a small effect on composite maltreatment, which is driven largely by female pairs. Children's genes appear to have a larger effect on neglect. Interestingly, the pattern of correlations for physical maltreatment across genders suggests an influence of children's genetics. However, no such influence is suggested by the physical maltreatment correlations within gender (note that although the male DZ physical maltreatment is lower than the MZ correlation, the full sibling correlation is not). Children's genetics do not appear to be related to the sexual maltreatment they experience. The MZ correlations are well below one for each maltreatment variable, suggesting nonshared environmental influences and/or measurement error.

Phenotypic correlations among the three forms of maltreatment ranged from .36 to .48, all of which were statistically significant at the $p < .05$ level. Table 3 shows cross-sibling, cross-trait correlations by type of sibling relationship. These correlations could not be calculated separately by gender because of the very small number of children who reported sexual maltreatment. Greater cross-sibling, cross-trait correlations among MZ twins than DZ twins and full siblings would suggest that the two variables are influenced by shared genetic factors. The observed correlations suggest that the associations between different forms of maltreatment generally are not due to common genetic factors.

The Role of Gender in Univariate Models

Table 4 shows the fit of the homogeneity and heterogeneity univariate models for composite maltreatment, physical maltreatment, and neglect. For each variable, the homogeneity model exhibited the best combination of fit and parsimony, as exhibited by Akaike's Information Criterion (AIC), though the difference was small for physical maltreatment. The magnitude of genetic and environmental influences can be constrained to be equal for males and females without a statistically significant decrease in fit. However, this may be due in part to low statistical power. Therefore, parameter estimates from both homogeneity and heterogeneity models were examined.

What is the Extent of Genetic and Environmental Influences on maltreatment?

Table 5 shows the results of a series of univariate models tested to determine whether dropping parameters from the heterogeneity saturated model (i.e., the ACTE model) leads to a significant decrease in fit. For all variables, each individual parameter (A, C, and T) could be dropped from the model without a statistically significant decrease in fit. However, for composite maltreatment, physical maltreatment, and neglect, A and C could not both be dropped without a significant decrease in fit. Therefore, parameter estimates from the saturated model are presented.

Table 6 presents parameter estimates from the homogeneity and heterogeneity saturated models. Results of both models for composite maltreatment suggest that genetically driven child effects are small, and almost thirty percent of the variance is due to shared and twin environmental influences. Two-thirds of the variance is due to nonshared environmental influences and measurement error.

The results of the homogeneity model for physical maltreatment suggest that approximately one quarter of the variance is due to nonpassive gene-environment correlation, about one fifth is due to the shared and twin environmental influences, and the remainder is due to the nonshared environment. In contrast, the heterogeneity model suggests that genetically mediated child effects on physical maltreatment are small for both males and females. Shared environmental influences accounted for approximately one quarter of the variance for both genders, whereas twin environmental factors appear to not influence males but accounted for another quarter of females' variance.

An examination of the cross-sibling within-trait correlations in Table 2 sheds light on this pattern of results. In males, the MZ correlation is substantially higher than the DZ correlation (.33 vs. .03), but the MZ and full sibling correlations are the same (.33 and .33). Therefore, this result indicates a lack of genetic influences on physical maltreatment in males. In females, the MZ and DZ correlations are very similar (.55 and .56), and are higher than the full sibling correlation (.30), also indicating a lack of genetic influences on physical maltreatment. However, if data from males and females are combined, the overall MZ correlation is higher than the DZ and full sibling correlations (.46 for MZs, .32 for DZs, and .30 for full siblings). Therefore, the conclusions from the homogeneity and heterogeneity models are conflicting.

The homogeneity model results for neglect suggest that about one quarter of the variance is due to genetically driven child effects and the shared and twin environments each exert a small effect. The remaining two-thirds of the variance are due to nonshared environmental factors and error. The heterogeneity model results suggest that there are moderate genetic influences on neglect for females, but very little for males. The shared and twin environments combined explain 28% of the variance in males' experience of neglect.

Due to low endorsement rates of sexual maltreatment, and therefore small cell sizes when analyzing genders separately, only the results of the homogeneity model of sexual maltreatment are presented. The results suggest that almost one quarter of the variance in sexual maltreatment is due to the shared environment and three quarters is due to the nonshared environment. There is no evidence of genetically-driven child effects.

Are Genetic and Environmental Influences on Different Forms of Maltreatment Shared or Unique?

A multivariate Cholesky model was tested to examine the magnitude of additive genetic, shared environmental, twin environmental, and nonshared environmental influences on the covariation among the forms of maltreatment. The results from this model were used to derive the correlations among physical maltreatment, neglect, and sexual maltreatment that is attributable to A, C, T, and E. Table 7 shows that the associations among different forms of maltreatment are not due to common genetic influences but mostly due to common nonshared environmental influences.

Discussion

The goal of the current study was to quantify genetic and environmental influences on children's experience of maltreatment and to assess the extent to which these influences are common or specific to different forms of maltreatment. The results of these analyses must be interpreted in light of the fact that maltreatment, unlike many other variables studied with these methods, often reflects an interaction between two genetically related people - parents and children. Therefore, both the children's and caregivers' genes may influence maltreatment. Additive genetic influences (A) on maltreatment in this child-based design suggest evidence for the effect of children's genes on maltreatment. Throughout this paper, this effect has been referred to interchangeably as genetically-driven child effects or non-passive gene-

environment correlation. Shared environmental influences (C) or twin environmental (T) influences include the effects of passive gene-environment correlation and/or true environmental effects. As noted in the introduction, it is not possible to distinguish between these two influences in a twin study. Therefore, tests of nonshared environmental influences (E) on maltreatment provide the only true tests of environmental influences on childhood maltreatment, though even this parameter is confounded by measurement error and possible G X E interaction. The effects of children's genes on maltreatment are clearly child effects, as siblings' genetic similarity is related to the similarity in their maltreatment experiences. Shared environmental, twin environmental, and nonshared environmental influences may or may not represent child effects. That is, it is possible that these factors capture caregiver behavior that may or may not be in direct response to children's behavior.

The homogeneity models, which constrain the parameter estimates to be equal across the sexes, did not fit significantly worse than the heterogeneity models, which allow the parameter estimates to vary across the sexes. However, the patterns of results from the homogeneity and heterogeneity models were substantively different for physical maltreatment and neglect, indicating the possibility there was insufficient power to differentiate between the homogeneity and heterogeneity models. Therefore, results from both the homogeneity and heterogeneity models were interpreted for these variables.

The results from the saturated homogeneity model of composite maltreatment indicated that genetically-mediated child effects were small (6%). The shared- and twin-environmental influences combined to account for approximately 30% of the variance in maltreatment. The homogeneity and heterogeneity models of physical maltreatment exhibited similar ratios of fit to parsimony as measured by AIC despite yielding substantially different parameter estimates. Whereas results from the homogeneity model indicated modest to moderate genetically-mediated child effects and small shared- and twin- environmental effects, the results from the heterogeneity model suggested small genetically-mediated child effects and shared- and twin-environmental effects that were moderate for males and large for females. Models examining neglect suggest that about one quarter of the variance was due to genetically driven child effects. Shared- and twin-specific environmental influences combined to account for approximately 13% of the variance. The results from the heterogeneity model suggest that the magnitude of genetic influences is greater in females than in males. Most of the variance in sexual maltreatment was due to nonshared environmental influences. In contrast to physical maltreatment or neglect, there was no evidence for genetically-driven child effects on sexual maltreatment. The majority of variance in each form of maltreatment was due to nonshared environmental influences.

The preliminary finding that genetically-mediated child effects on maltreatment appear to be small relative to other parenting and home environment variables suggests that maltreatment may be less a response to children's behaviors and more associated with characteristics and experiences of caregivers. That is, although much of caregivers' more normative parenting and disciplining behavior may be a response to children's behavior, parents' violating social norms and failing to meet children's basic needs may be more associated with their own limitations. A small to moderate portion of the variance in maltreatment variables is due to the shared and twin environments, which may reflect environmental processes and/or passive gene-environment correlation. Most of the variance in maltreatment is due to nonshared environmental influences, which may include either idiosyncratic child effects (e.g., an illness that affects only one of the siblings) or parent effects (e.g., a stressor experienced by the parent when only one of the siblings is present).

Consistent with prior research (e.g. Dong et al., 2004), the three forms of maltreatment were moderately correlated with each other. The results of the multivariate Cholesky model

assessing the overlap of factors influencing the three forms of maltreatment must be interpreted cautiously due to the low power to analyze covariation among individual forms of maltreatment. There is suggestive evidence that although children's genes may affect both physical maltreatment and neglect, the genetic factors that were associated with physical maltreatment did not overlap with those associated with neglect. If replicated, this would suggest that the child behaviors that are evocative of neglect are different from those that are evocative of physical maltreatment. The correlations among the three forms of maltreatment were largely due to common nonshared environmental influences. However, common nonshared environmental influences also might have reflected common measurement errors across different forms of maltreatment.

The present study's preliminary finding of small genetically-influenced child effects on maltreatment is consistent with results suggesting that children's genetics influence their environment in general (e.g. Plomin, 1995) and the parenting they 'receive' (e.g. Kendler & Baker, 2007) in particular. The results are inconsistent with those from a study of child-reported and parent-reported physical discipline of girls in the Virginia Twin Registry (Wade & Kendler, 2000), which found children's genetics had a moderate effect. The suggestive evidence for small genetically-mediated child effects on physical maltreatment is more consistent with results from a parent-report study of British children of small to moderate effects for corporal punishment and none on physical maltreatment (Jaffee et al., 2004). However, the present study's moderate C and T on maltreatment were smaller than the British study's substantial C for both corporal punishment and physical maltreatment. These differing results may reflect differences in assessment method (i.e., self reports in the present study and maternal report in the British study), and are consistent with the literature suggesting that correlations for parenting of children in the same family are more similar in parent reports than in child reports (Achenbach et al., 1987; Hewitt et al., 1992; Wade & Kendler, 2000). These differences may also reflect the fact that Jaffee et al. (2004) assessed corporal punishment and physical maltreatment occurring prior to age 5 whereas the present study queried maltreatment occurring prior to age 12.

Several strengths of the present study are noteworthy. It includes the first tests for genetically influenced child effects on neglect and sexual maltreatment known to the authors. The analyses benefited from a genetically informative subsample of the largest nationally representative study of adolescent health ever conducted in the United States. Both twin and full sibling pairs were included, allowing us to estimate the magnitude of twin environmental influences. Also, the use of child report of maltreatment ensures that C and T are not overestimated due to rater biases.

Several limitations of the present study should be considered when interpreting the results. First, the assessment of maltreatment was not ideal. Neglect was assessed via only two items and physical and sexual maltreatment were assessed via only one item each. Each item was assessed on an ordinal scale. Also, maltreatment experiences that occurred prior to the age of 12 were assessed retrospectively at an average age of 21. Thus, results may not only reflect influences on maltreatment but also on its retrospective recall. Therefore, the large estimates of the nonshared environment may actually reflect large measurement error. However, research suggests that retrospective reports affirming the occurrence 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, Kendler and Baker's (2006) review of the heritability of parenting and other 'environmental' variables suggested that self-report and informant-report yielded similar heritability estimates and that observer-reports yielded lower estimates. They argued that this is likely due to unreliability of observer reports, which were based on ten minutes or less of observation. Therefore, they concluded that twin studies of environmental variables largely reflect 'actual behavior' rather than 'only perceptions'. In a

separate study, maternal reports of family environment were modestly to moderately correlated over a twenty-five year time span (Finkel & McGue, 1993). Second, despite the large sample size, power was an issue, as evidenced by the fact that the homogeneity and heterogeneity models fit similarly despite substantially different parameter estimates across the sexes and the lack of power to distinguish A and C in the univariate models. Considered individually, none of the estimates of A, C, or T was significantly greater than zero. Almost half of the participants reported no experiences of maltreatment, and the low prevalence of sexual maltreatment precluded consideration of sex differences in the magnitude of the parameter estimates.

The present study represents a preliminary step in the investigation of the role of genetics in childhood maltreatment, and the first such analysis known to us of neglect and sexual maltreatment in particular. The results in the literature and the present study suggest several directions for future studies examining the etiology of childhood maltreatment. First, the present study's findings that small genetically-driven child effects may exist for physical maltreatment and neglect, but not for sexual maltreatment, need to be replicated. Second, the discrepancy between the results of Jaffee et al.'s study (2004), which used parent report, and the results of the present study, which used self report, suggests that a study examining reports of maltreatment from multiple raters and evaluating the degree of rater bias (e.g., Hewitt et al., 1992; Simonoff et al., 1995) would be useful. Third, the specific child traits or behaviors increasing the risk for maltreatment should be identified. Fourth, studies using designs that can distinguish between passive gene-environment correlations and shared environmental influences, such as the children of twins design, would also be helpful. Fifth, studies evaluating gene-environment interaction in maltreatment should be conducted.

In conclusion, the present study's results suggest that there may be small genetically-mediated child effects on physical maltreatment and neglect, and none on sexual maltreatment. The effects of "shared environment" on maltreatment, which may include passive gene-environment correlation, were moderate. Most of the variance of maltreatment was due to nonshared environmental influences, suggesting that maltreatment occurs largely as a function of idiosyncratic individual circumstances. The covariances among different forms of maltreatment does not appear to be due to common genetic influences being associated with behaviors that evoke various forms of maltreatment, but rather to correlated nonshared environmental influences, or common measurement error.

Although the present study's results suggest that a small magnitude of variance in maltreatment may be associated with children's genetic factors, this does not mean that children cause or are responsible for the maltreatment they experience. That is, difficult child behavior is not a justification for maltreatment. Rather, the results provide further evidence that interventions with adults intended to prevent maltreatment should include efforts to address how to respond to difficult child behavior. The results are also consistent with research documenting that parent training not only reduces problematic child behavior but also provides a variety of benefits to parents (Kazdin, 2005; Adams, 2001).

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References

Achenbach TM, McConaughy SH, Howell CT. Child/adolescent behavioral and emotional problems: implications for cross-informant correlations for situational specificity. Psychological Bulletin 1987;101(2):213–232. [PubMed: 3562706]

Adams JF. Impact of parent training on family functioning. Child & Family Behavior Therapy 2001;23 (1):29–42.

Anderson KE, Lytton H, Romney DM. Mothers' interactions with normal and conduct disordered boys: Who affects whom? Developmental Psychology 1986;22:604–609.

Bell RQ. A reinterpretation of the direction of effects in studies of socialization. Psychological Review 1968;75(2):81–95. [PubMed: 4870552]

Belsky J. The etiology of child maltreatment: A development-ecological analysis. Psychological Bulletin 1993;114(3):413–434. [PubMed: 8272464]

Berger AM, Knutson JF, Mehm JG, Perkins KA. The self-report of punitive childhood experiences of young adults and adolescents. Child Abuse and Neglect 1988;12:251–262. [PubMed: 3395899]

Brewin CR, Andrews B, Gotlib IH. Psychopathology and early experience: A reappraisal of retrospective reports. Psychological Bulletin 1993;113(1):82–98. [PubMed: 8426875]

Bronfenbrenner U. Ecology of the family as a context for human development: Research perspectives. Developmental Psychology 1986;22(6):723–742.

Bugental DB, Shennum W. Gender, power, and violence in the family. Child Maltreatment 2002;7(1): 56–64. [PubMed: 11838515]

Carlin AS, Kemper K, Ward NG, Sowell H, Gustafson B, Stevens N. The effect of differences in objective versus subjective definitions of childhood physical abuse on estimates of its incidence and relationship to psychopathology. Child Abuse & Neglect 1994;18(5):393–399. [PubMed: 8032969]

Carlson, EB. Trauma Assessment: A Clinician's Guides. New York: Guilford; 1997.

Dong M, Anda RF, Felitti VJ, Dube SR, Williamson DF, Thompson TJ, et al. The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. Child Abuse & Neglect 2004;28(7):771–784. [PubMed: 15261471]

Finkel D, McGue M. Twenty-five year follow-up of child-rearing practices: Reliability of retrospective data. Journal of Personality and Individual Differences 1993;15:147–154.

Harris KM, Halpern CT, Smolen A, Haberstick BC. The national longitudinal study of adolescent health (Add Health) Twin Data. Twin Research and Human Genetics 2006;9(6):998–997. [PubMed: 17254443]

Hewitt JK, Silberg JL, Neale MC, Eaves LJ. The analysis of parental ratings of children's behavior using LISREL. Behavior Genetics 1992;22(3):293–317. [PubMed: 1616461]

Jaffee SR, Caspi A, Moffitt TE, Polo-Tomas M, Price TS, Taylor A. The limits of child effects: Evidence for genetically mediated child effects on corporal punishment but not on physical maltreatment. Developmental Psychology 2004;40(6):1047–1058. [PubMed: 15535755]

Kazdin, AE. Parent management training: Treatment for oppositional, aggressive, and antisocial behavior in children and adolescents. New York, NY, US: Oxford University Press; 2005.

Kendler KS, Baker JH. Genetic influences on measures of the environment: a systematic review. Psychological Medicine 2007;37:615–626. [PubMed: 17176502]

Neale, MC.; Cardon, LR. Methodology for genetic studies of twins and families. Dordrecht, the Netherlands: Kluwer; 1992.

Neale, MC.; Boker, SM.; Xie, G.; Maes, HH. Mx: Statistical modeling. Vol. 6. Richmond, VA: Virginia Commonwealth University, Department of Psychiatry; 2003.

Neiderhiser JM, Reiss D, Pederson NL, Lichtenstein P, Spotts EL, Hansson K, Cederblad M, Elthammer O. Genetic and environmental influences on mothering of adolescents: A comparison of two samples. Developmental Psychology 2004;40(3):335–351. [PubMed: 15122961]

Osofsky JD, O'Connell EJ. Parent-child interaction: Daughters effects upon mothers' and fathers' behaviors. Developmental Psychology 1972;7(2):157–168.

Patterson, GR.; Bank, L.; Stoolmiller, M. The preadolescent's contributions to disrupted family process. In: Montemayor, R.; Adams, GR.; Gullotta, TP., editors. From Childhood to Adolescence. Newbury Park, CA: Sage; 1990. p. 107-133.

Plomin R. Genetics and children's experiences in the family. Journal of Child Psychology and Psychiatry 1995;36(1):33–68. [PubMed: 7714029]

Plomin R, DeFries JC, Loehlin JC. Genotype-environment interaction and correlation in the analysis of human behavior. Psychological Bulletin 1977;84(2):309–322. [PubMed: 557211]

Plomin R, Reiss E, Hetherington M, Howe GW. Nature and nurture: genetic contributions to the family environment. Developmental Psychology 1994;30(1):32–43.

SAS Institute, Cary, NC, (2002).

Silvern L, Waelde L, McClintic B, Kaersvang L. Two methods of eliciting retrospective reports of child abuse. Child maltreatment 2000;5(3):236–250. [PubMed: 11232270]

Simonoff E, Pickles A, Hewitt J, Silberg J. Multiple raters of disruptive child behavior: Using a genetic strategy to examine shared views and bias. Behavior Genetics 1995;25(4):311–326. [PubMed: 7575360]

Stallings MC, Hewitt JK, Lessem JM, Young SE, Corley RP, Mikulich SK, Crowley TJ. Modeling the familial transmission of alcohol dependence symptom counts in clinical and control family pedigrees [Abstract]. Behavior Genetics 2001;31:470.

Steele, B. Psychodynamic factors in child abuse. In: Kempe, CH.; Helfer, RE., editors. The Battered Child. Vol. 2. University of Chicago Press; 1980. p. 52-56.

Sunday S, Labruna V, Kaplan S, Pelcovitz D, Newman J, Salzinger S. Physical abuse during adolescence: Gender differences in the adolescents' perceptions of family functioning and parenting. Child Abuse & Neglect 2008;32(1):5–18. [PubMed: 18082259]

Vasta R. Physical child abuse: A dual-component analysis. Developmental Review 1982;2:125–149.

Wade TD, Kendler KS. The genetic epidemiology of parental discipline. Psychological Medicine 2000;30:1303–1313. [PubMed: 11097071]

Williams, LM. Adult memories of child abuse: Preliminary findings from a longitudinal study. Paper presented at the 1992 National Symposium on Child Abuse and Neglect; Washington, D.C. 1992.

Yarrow LJ. Research in dimensions of early maternal care. Merrill-Palmer Quarterly 1963;9(2):101–114.

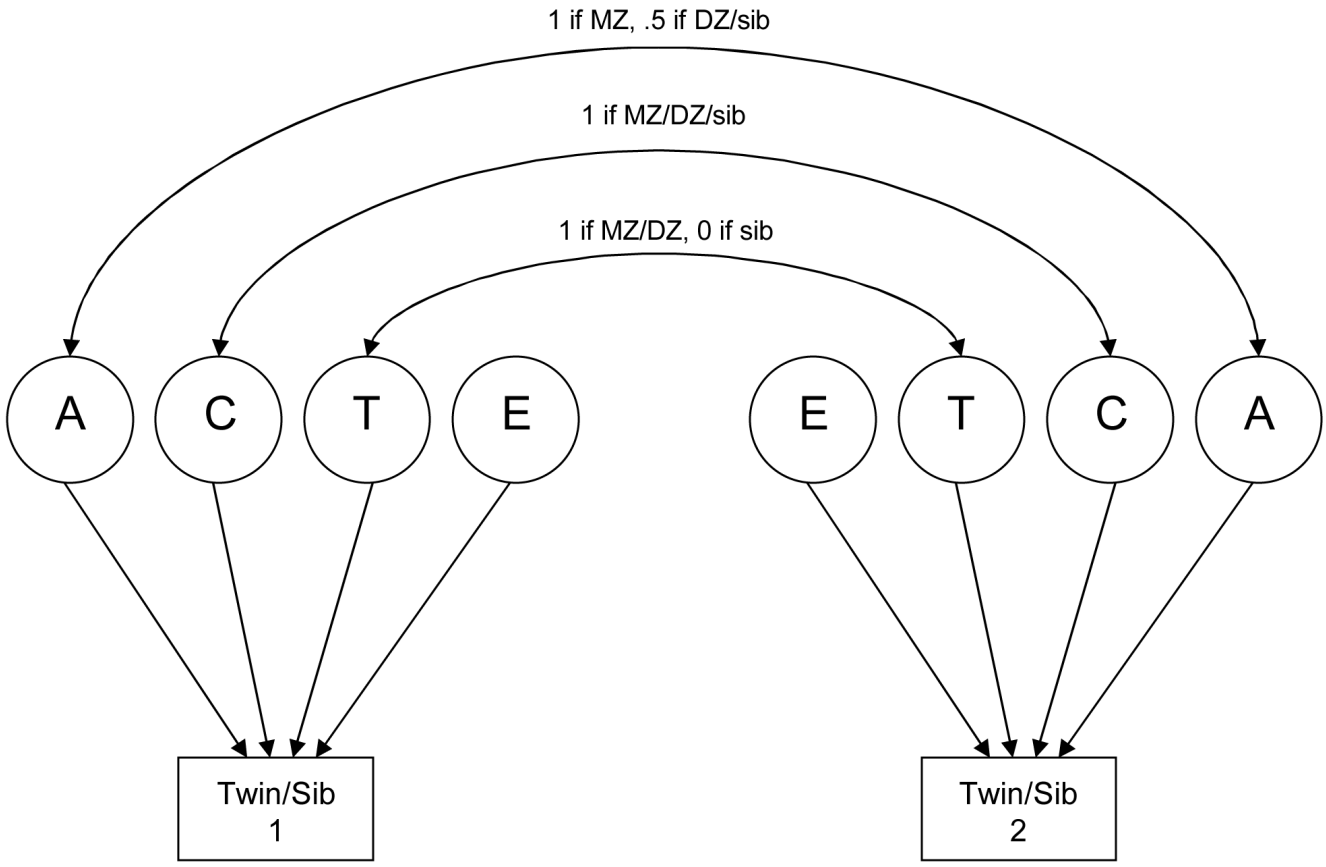

Figure 1.
Univariate Homogeneity Model. A = additive genetic influences; C = shared environmental influences; T = twin environmental influences; E = nonshared environmental influences; MZ = monozygotic; DZ = dizygotic; sib = nontwin full sibling.

Table 1

Percentage of Participants in Each Ordinal Category of Maltreatment

	All				Male				Female			
Level	**MT**	**PMT**	**Neglect**	**SMT**	**MT**	**PMT**	**Neglect**	**SMT**	**MT**	**PMT**	**Neglect**	**SMT**
0	46.9	71.3	58.8	94.9	44.6	69.8	56.3	95.3	48.9	72.6	61.1	94.6
1	25.5	13.7	18.4	5.1	26.6	14.0	18.8	4.7	24.4	13.4	18.1	5.4
2	10.6	9.3	10.7		12.0	9.4	11.9		9.4	9.1	9.7	
3	10.5	5.7	12.0		10.1	6.7	13.0		10.9	4.8	11.1	
4	6.6				6.7				6.4			

Note. MT = composite maltreatment; PMT = physical maltreatment; SMT = sexual maltreatment.

Table 2

Cross-Sibling Within-Trait Polychoric Correlations by Sibling Type and Gender

	MT	PMT	Neglect	SMT
All	MZ .34 (.21–.46)	.46 (.27–.61)	.36 (.21–.50)	.13 (−.38–.58)
	DZ .31 (.20–.42)	.32 (.15–.48)	.25 (.25–.10)	.34 (−.03–.64)
	FS .19 (.12–.27)	.30 (.21–.40)	.18 (.09–.27)	.24 (−.05–.50)
Male	MZ .22 (−.01–.42)	.33 (−.01–.59)	.31 (.08–.52)	
	DZ .27 (.06–.46)	.03 (−.28–.35)	.28 (.02–.50)	
	FS .17 (.03–.32)	.33 (.14–.50)	.18 (.00–.36)	
Female	MZ .41 (.25–.55)	.55 (.32–.72)	.41 (.19–.58)	
	DZ .36 (.11–.55)	.56 (.27–.75)	.20 (−.10–.46)	
	FS .21 (.07–.34)	.30 (.12–.46)	.24 (.07–.40)	
Opposite Sex	DZ .32 (.14–.47)	.35 (.08–.57)	.25 (.01–.46)	
	FS .20 (.09–.30)	.29 (.14–.43)	.14 (.00–.27)	

Note. SMT correlations were not computed by gender due to small cell sizes. MT = composite maltreatment; PMT = physical maltreatment; SMT = sexual maltreatment. MZ = monozygotic; DZ = dizygotic; FS = nontwin full sibling.

Table 3
Cross-Sibling Cross-Trait Correlations by Sibling Type

	PMT	Neglect	SMT
MZ Twins			
PMT	1.00		
Neglect	.17	1.00	
SMT	.04	.21	1.00
DZ Twins			
PMT	1.00		
Neglect	.21	1.00	
SMT	.11	.17	1.00
Full Sibs			
PMT	1.00		
Neglect	.08	1.00	
SMT	.11	.05	1.00

Note. PMT = physical maltreatment; SMT = sexual maltreatment; MZ = monozygotic; DZ = dizygotic.

Table 4

Homogeneity and Heterogeneity Model Fits

		Homogeneity	Heterogeneity	Comparison Statistics	
MT	−2LL	8,948.38	8946.03	$\Delta\chi^2$	2.35
	df	3224	3221	Δdf	3
	AIC	2500.38	2504.03	p	.50
PMT	−2LL	5537.28	5532.52	$\Delta\chi^2$	4.76
	df	3123	3120	Δdf	3
	AIC	−708.72	−707.48	p	.19
Neglect	−2LL	7066.70	7065.58	$\Delta\chi^2$	1.12
	df	3192	3189	Δdf	3
	AIC	682.70	687.58	p	.77

Note. The heterogentiy model was not tested for SMT due to small cell sizes. −2LL = −2 log likelihood; df = degrees of freedom; AIC = Akaike's information criterion; $\Delta\chi^2$ = difference in chi square from homogeneity model; p = probability. MT = composite maltreatment; PMT = physical maltreatment.

Table 5

Univariate Results

	−2LL	df	AIC	$\Delta\chi^2$	Δdf	p
MT						
ACTE	8946.03	3221	2504.03			
ACE	8949.09	3223	2503.09	3.06	2	.22
ATE	8950.07	3223	2504.07	4.04	2	.13
CTE	8946.09	3223	2500.09	.06	2	.97
AE	8951.40	3225	2501.40	5.37	4	.25
CE	8951.78	3225	2501.78	5.75	4	.22
TE	8972.20	3225	2522.20	26.17	4	< .01
E	9024.62	3227	2570.62	78.59	6	< .01
PMT						
ACTE	5532.52	3120	−707.48			
ACE	5534.91	3122	−709.09	2.39	2	.30
ATE	5536.75	3122	−707.25	4.23	2	.12
CTE	5532.58	3122	−711.42	.06	2	.97
AE	5537.11	3124	−710.90	4.59	4	.33
CE	5537.13	3124	−710.87	4.61	4	.33
TE	5566.82	3124	−681.18	34.30	4	< .01
E	5605.82	3126	−646.18	73.30	6	< .01
Neglect						
ACTE	7065.58	3189	687.58			
ACE	7066.39	3191	684.39	.81	2	.67
ATE	7066.40	3191	684.40	.82	2	.66
CTE	7067.47	3191	685.47	1.89	2	.39
AE	7067.00	3193	681.00	1.42	4	.84
CE	7070.48	3193	684.48	4.90	4	.30
TE	7081.91	3193	695.91	16.33	4	< .01
E	7110.06	3195	720.06	44.48	6	< .01
SMT						
ACTE	1259.47	3154	−5084.54			
ACE	1259.48	3155	−5050.52	.01	1	.92

	−2LL	df	AIC	$\Delta\chi^2$	Δdf	p
ATE	1260.79	3155	−5049.21	1.32	1	.25
CTE	1259.47	3155	−5050.54	.00	1	1.00
AE	1260.82	3156	−5051.18	1.35	2	.51
CE	1259.48	3156	−5052.50	.01	2	.51
TE	1262.22	3156	−5049.78	2.75	2	.25
E	1265.27	3157	−5048.74	5.80	3	.12

Note. Heterogeneity models are shown for MT, PMT, and Neglect. The homogeneity model is shown for SMT. −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. MT = composite maltreatment; PMT = physical maltreatment; SMT = sexual maltreatment. A = additive genetic influences; C = shared environmental influences; T = twin environmental influences; E = nonshared environmental influences.

Table 6
Parameter Estimates from Homogeneity and Heterogeneity Models

	A	C	T	E
MT				
All	.06 (.00–.39)	.17 (.00–.26)	.12 (.00–.24)	.66 (.54–.76)
Males	.01 (.00–.34)	.17 (.00–.29)	.07 (.00–.25)	.75 (.58–.86)
Females	.05 (.00–.44)	.19 (.00–.33)	.17 (.00–.35)	.59 (.46–.73)
PMT				
All	.28 (.00–.61)	.17 (.00–.38)	.02 (.00–.20)	.54 (.39–.72)
Males	.07 (.00–.52)	.23 (.00–.41)	.00 (.00–.15)	.69 (.44–.85)
Females	.03 (.00–.60)	.29 (.01–.46)	.24 (.00–.45)	.44 (.28–.62)
Neglect				
All	.24 (.00–.47)	.06 (.00–.25)	.07 (.00–.23)	.64 (.51–.79)
Males	.03 (.00–.49)	.16 (.00–.34)	.12 (.00–.35)	.69 (.49–.85)
Females	.32 (.00–.56)	.06 (.00–.36)	.03 (.00–.28)	.59 (.43–.80)
SMT				
All	.00 (.00–.59)	.24 (.00–.44)	.02 (.00–0.42)	.73 (.40–.95)

Note. MT = composite maltreatment; PMT = physical maltreatment; SMT = sexual maltreatment. A = additive genetic influences; C = shared environmental influences; T = twin environmental influences; E = nonshared environmental influences.

Table 7
Correlation Between Forms of Maltreatment Due to A, C, T, and E

	PMT	Neglect
A		
Neglect	.01	
SMT	−.07	.09
C		
Neglect	.08	
SMT	.12	.02
T		
Neglect	.08	
SMT	.04	.07
E		
Neglect	.19	
SMT	.38	.21

Note. PMT = physical maltreatment; SMT = sexual maltreatment. A = additive genetic influences; C = shared environmental influences; T = twin environmental influences; E = nonshared environmental influences.