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Estimating the Heritability of Experiencing Child Maltreatment in an Extended Family Design

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Original Article

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Abstract

Child-driven genetic factors can contribute to negative parenting and may increase the risk of being maltreated. Experiencing childhood maltreatment may be partly heritable, but results of twin studies are mixed. In the current study, we used a cross-sectional extended family design to estimate genetic and environmental effects on experiencing child maltreatment. The sample consisted of 395 individuals (225 women; M_{age} = 38.85 years, $range_{age}$ = 7–88 years) from 63 families with two or three participating generations. Participants were oversampled for experienced maltreatment. Self-reported experienced child maltreatment was measured using a questionnaire assessing physical and emotional abuse, and physical and emotional neglect. All maltreatment phenotypes were partly heritable with percentages for h^2 ranging from 30% (SE = 13%) for neglect to 62% (SE = 19%) for severe physical abuse. Common environmental effects (c^2) explained a statistically significant proportion of variance for all phenotypes except for the experience of severe physical abuse (c^2 = 9%, SE = 13%, p = .26). The genetic correlation between abuse and neglect was ρ_g = .73 (p = .02). Common environmental variance increased as socioeconomic status (SES) decreased (p = .05), but additive genetic and unique environmental variances were constant across different levels of SES.

Keywords

child maltreatment, genetics, etiology, families, risk factors, self-report

Each year, approximately 3.4–4.0% of children experience maltreatment in higher income countries (Euser et al., 2013; Sedlak et al., 2010). The etiology of maltreatment is complex, and a single causal pathway to maltreatment does not seem plausible. Rather, multiple risk and protective factors have been identified (Patwardhan, Hurley, Thompson, Mason, & Ringle, 2017). Heritable as well as environmental factors may contribute to maltreatment risk. Risk factors can be present at the level of the parent (e.g., psychopathology), the child (e.g., irritable temperament), or the family (e.g., socioeconomic adversities) and may interact with each other.

Risk factors related to the child have thus far been studied less (Stith et al., 2009) but may play an important role in the etiology of maltreatment. Parenting is a bidirectional process (Klahr et al., 2017), and child-driven factors can contribute to negative parenting (Avinun & Knafo, 2014; Davidov, Knafo-Noam, Serbin, & Moss, 2015). For instance, externalizing behavior (such as conduct problems, antisocial behavior, oppositional defiant disorder, and attention deficit hyperactivity disorder) may increase the risk of maltreatment and other types of victimization (Nobile et al., 2013; Stith et al., 2009).

As these phenotypes are partly heritable (Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Nikolas & Burt, 2010; Porsch et al., 2016), they may genetically mediate the risk of experiencing maltreatment. One children-as-twin study tested this hypothesis and found that corporal punishment but not physical

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abuse was heritable in childhood, suggesting that genetic influences on the phenotype of experienced parenting may be limited to more normative parenting responses (Jaffee, Caspi, Moffitt, Polo-Tomas et al., 2004). However, when the same twin population was studied in adolescence, abuse and neglect were found to be heritable with additive genetic estimates of 71% and 47%, respectively (Fisher et al., 2015). In addition, evidence from two children-as-twin studies (one including adolescents and young adults and one including adults) suggests that approximately one quarter of the variance in experienced maltreatment can be attributed to genetic variation and to a lesser extent to common environment (Schulz-Heik et al., 2009; South, Schafer, & Ferraro, 2015). It should be noted, however, that in both studies, confidence intervals were large and more than half of the variance was explained by unique environmental factors including measurement error.

Importantly, these studies do not suggest that there are no parent effects on maltreatment. Parent effects are part of environmental effects which were consistently present in these studies. Moreover, these models are not deterministic—they do not suggest that children with a specific genetic layout will inevitably be maltreated. Rather, genetic factors (and potentially associated behavior) may increase the risk of being maltreated—especially when combined with environmental risk factors and a vulnerable parent.

Heritability may carry the negative connotation of a trait being fixed, but evidence is accumulating that the influence of heritability may be malleable by environmental factors, that is, gene-by-environment interactions. In psychological research, socioeconomic status (SES), in particular, has been associated with changes in the strength of heritability. The nature of these changes is not clear, however. Research on cognitive abilities has associated low SES with smaller genetic influences (Bates, Lewis, & Weiss, 2013) and greater shared environmental effects (Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011). Similarly, the social push perspective suggests that genetic effects may be suppressed in high-risk environments (Middeldorp et al., 2014; Raine, 2002). In the context of gene-by-environment interaction, it becomes clear that the importance of understanding the heritability of maltreatment goes beyond providing an etiological framework. Namely, some interventions may be effective in reducing environmental risk of child maltreatment but not the genetic risk and vice versa. If research demonstrates that environmental factors such as SES affect the influence of heritability and environment differentially, interventions could be tailored to ensure that both are reduced.

The present study examined additive genetic and common and unique environmental effects on experienced abuse and neglect in an extended family design. This design has been applied previously to estimate the heritability of perpetrating maltreatment (Pittner et al., 2019). Extended family designs include family members beyond the nuclear family such as grandparents and cousins and across several generations. Extended family designs work under the assumption that, if a phenotype is heritable, individuals who are more closely related to each other will be more similar in a specific trait. Each family member shares a genetic relatedness with all other family members which generally lies between 6.25% and 50% (Almasy & Blangero, 2010). Lower levels of relatedness are possible if more distant relatives such as fourth cousins are included, and higher levels are possible if monozygotic twins are included.

The primary aim of this study was to compute heritability estimates for experienced maltreatment overall and for abuse and neglect separately. Our hypothesis was that, in line with most previous research (Fisher et al., 2015; Schulz-Heik et al., 2009; South et al., 2015), significant heritability components would be found independent of maltreatment type. Maltreatment was comprised of emotional and physical abuse and emotional and physical neglect—averaged across types. Overall, maltreatment, abuse, and neglect were treated as a continuous measures ranging from "no maltreatment" to "(severe) maltreatment." We also examined whether *severe* physical abuse shows a heritability estimate similar to maltreatment. The aim was to test the idea postulated by Jaffee, Caspi, Moffitt, Polo-Tomas, and colleagues (2004) that child effects do not extend to more severe forms of maltreatment. We focused on physical abuse, rather than other types of maltreatment, in line with Jaffee, Caspi, Moffitt, Polo-Tomas et al.'s (2004) definition of the type of maltreatment that was central to their study. Second, we applied a multivariate approach to test the genetic and environmental overlap of abuse and neglect. Lastly, we explored whether SES moderates heritability of child maltreatment, similar to cognitive abilities displaying smaller genetic influences and larger shared environmental effects in children from low-SES backgrounds (Bates et al., 2013).

Method

Sample

The sample consisted of 395 individuals (225 women; M_{age} = 38.85 years, $range_{age}$ = 7.50–88.42 years) from 63 families with two or three generations participating in the 3G Parenting Study. On average, 6.27 family members per family participated (range: 2–23; see Online Appendix S1, Online Appendix Table S1, and Figure 1). Participants were recruited from three Dutch participant pools: (1) the Netherlands Study of Depression and Anxiety (Penninx et al., 2008), (2) a study on parenting in low-SES families (Joosen, Mesman, Bakermans-Kranenburg, & Van IJzendoorn, 2013), and (3) the Longitudinal Internet Studies for the Social Sciences panel (Scherpenzeel & Toepoel, 2012). From two of these studies, we oversampled participants who reported having experienced maltreatment during childhood, and from the third study, all participants were invited. If the target participant agreed to take part in the study, family members of the target participant and of the target participant's partner were invited to participate (parents [G1], children [G3], siblings [G2], nieces [G3], and nephews [G3]). Family members had to be at least 7.5 years of age to be invited. Families were included if at least two first-degree relatives from two generations agreed to participate (Pittner et al., 2019).

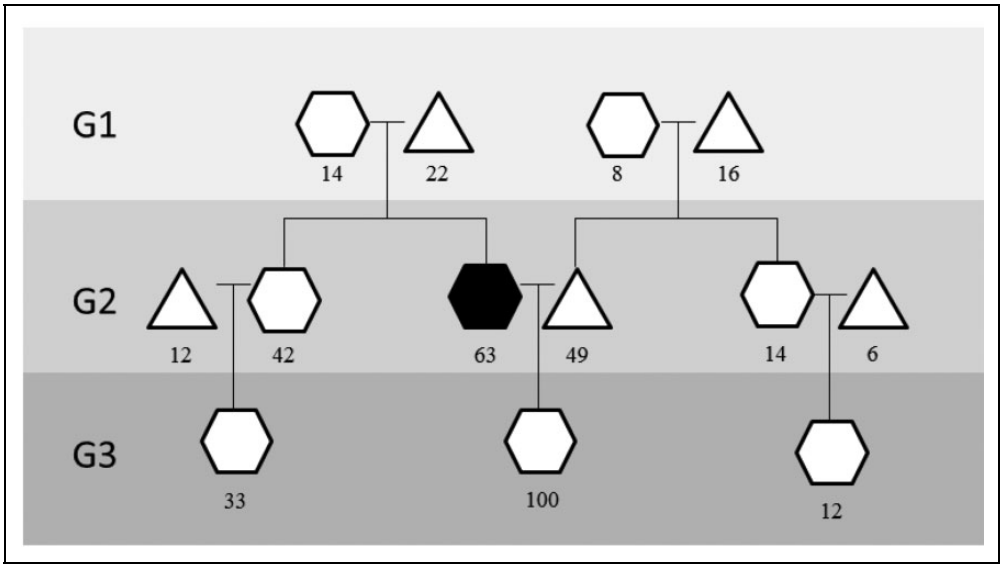

Figure 1. Extended pedigrees from 63 families. Simplified summary pedigree of participants. The black shape indicates the participants who were contacted first (target participant). Shapes without fill denote family members who were recruited around the target participant: partners, parents, children, siblings with partners, nephews/nieces, and family-in-law. Numbers reflect how many participants of each relationship category were included. For instance, 12 of the participants are the partners of the sibling of the target. Note that four participants from the fourth generation were not included in this pedigree, and shapes are not symbolic of sex.

The following distribution for education was found for adult participants (≥18 years, $n = 302$): 6% elementary school, 19% lower vocational school, 40% advanced secondary education, and 28% college or university degree (6% unknown). The sample was economically diverse with the following percentages for yearly household incomes: 6.1% less than €15,000, 11.4% €15,000–€ 24,999, 12.9% €25,000–€ 34,999, 9.1% €35,000–€ 44,999, 8.6% €45,000–€ 54,999, 4.6% €55,000–€ 64,999, and 8.4% more than €65,000. The average household income in the Netherlands in 2014 was €59,600 (Statistics Netherlands, 2017).

Procedure

Nuclear families were invited to attend a 7-hr lab visit at the Leiden University Medical Center. Participants from the second generation came twice—once with their family of origin (parents and siblings) and once with their partner and children. A lab visit involved questionnaires, computer tasks, family interaction tasks, and the collection of saliva and hair samples. Informed consent was obtained from all participants. For participants under 18 years of age, parents cosigned informed consent. After each lab visit, child maltreatment questionnaires were checked for all children under 18 years of age (see Online Appendix S2). Ethical approval was obtained from the Ethics Committee of the Leiden University Medical Centre.

Instruments

Demographic information. Age and gender were included as background variables. Participants of 18 years and older filled out a questionnaire with questions about household income and highest completed education. Yearly household income was measured on a 7-point scale ranging from (1) less than €15,000 to (7) more than €65,000. Due to changes in the Dutch educational system, first- and second-generation participants rated education on a 7-point scale and third-generation participants rated education on a 10-point scale. Both scales were rescaled to a 4-point scale. Based on standardized household income and standardized completed educational level, a composite household SES score was calculated. If data of two partners living in the same household were available, their scores were averaged for the household SES score. Children living with their parents shared their parents' household SES score.

Experienced child maltreatment. Experienced child maltreatment was measured using a combination of the self-reported Parent–Child Conflict Tactics Scales (CTSPC; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998) and the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994; Thombs, Bernstein, Lobbestael, & Arntz, 2009). The following subscales were used: (1) Physical Assault (i.e., physical abuse, 13 items; CTSPC), (2) Psychological Aggression (i.e., emotional abuse, 5 items; CTSPC), (3) Physical Neglect (4 items; CTSPC), and (4) Emotional Neglect (6 items; CTSPC and CTQ). The Physical Abuse Scale consists of three subscales: Minor (5 items), Severe (4 items), and Very Severe (4 items) physical abuse. For consistency in response options, a 5-point scale ranging from 1 (*never*) to 5 (*almost*) always was used for all items.

We assessed maternal and paternal behavior separately. For the separate subtypes, we first calculated averages for maltreatment perpetrated by mother and maltreatment perpetrated by father. Then, per subscale, the higher score of mother or father

was included in the analyses. Internal consistencies of the subscales were as follows: $\alpha_{mother} = .91$, $\alpha_{father} = .91$ for physical abuse, $\alpha_{mother} = .79$, $\alpha_{father} = .74$ for emotional abuse, $\alpha_{mother} = .62$, $\alpha_{father} = .54$ for physical neglect, and $\alpha_{mother} = .92$, $\alpha_{father} = .90$ for emotional neglect.

An overall "maltreatment" score was calculated by averaging subscale scores for all subtypes. Both mother and father scores correlated strongly with the overall maltreatment score, mother: $r(393) = .89$, $p < .01$ and father: $r(389) = .85$, $p < .01$, suggesting that the results were not driven by either mother or father. The "abuse" score was the average of physical and emotional abuse, $r(393) = .67$, $p < .01$, and the "neglect" score was the average of physical and emotional neglect, $r(393) = .40$, $p < .01$. The distribution for (very) severe physical abuse was highly skewed to the right (skewness = 4.59, $SE = .12$). Therefore, we distinguished between a group with severe or very severe physical abuse experiences ($n = 155$) and a group without such experiences ($n = 240$) for the additional analyses on abuse severity. The very severe physical abuse scale includes items such as "Grabbed around the neck and chocked" and "Burned or scolded on purpose." For participants under 12 years of age, experienced maltreatment was assessed orally and questions about very severe physical abuse were omitted. For additional details, see Online Supplementary Material (Online Appendix S3). We did not assess sexual abuse in the current study because this maltreatment type has the lowest prevalence rate (Euser et al., 2013). In order to estimate heritability of sexual abuse, a larger sample size would have been necessary.

Analyses

Descriptive and correlational analyses were conducted using SPSS Statistics Version 23.0 (SPSS Inc.). All heritability analyses were performed in the statistical genetic analysis software SOLAR 8.1.1 (Almasy & Blangero, 1998). SOLAR is a comprehensive system for likelihood-based statistical analysis of variance components models. In twin research, high agreement has been found between SOLAR and other methods of estimating heritability (Kochunov et al., 2019). Heritability (h^2) is defined as the proportion of phenotypic variance attributable to additive genetic variance, estimated using a kinship matrix. In the kinship matrix, genetic relatedness for all possible participant pairs is listed. Heritability is estimated under a polygenic model (i.e., multiple gene inheritance), and significance is determined by comparing the log likelihoods of the models when the heritability parameter is estimated and when it is constrained to zero (see Online Appendix S4).

In a first step, preliminary polygenic model analyses were performed for the phenotypes maltreatment, abuse, and neglect with age, age^2, sex, age $\times$ sex, age^2-by-sex, and SES as covariates. The residuals from these three models (one model per trait) were transformed using inverse normalization. In the following step, polygenic heritability analyses were performed for these new phenotypes. To estimate the common environmental variance (c^2), a household component was included in all models. Full- and half-siblings were coded as sharing or having shared the same household if they had grown up in the same household for at least 5 years ($n = 146$). Household was included in the analysis by adding a matrix in the prediction model with value 1 for all pairs of participants who shared a household and value 0 for all other pairs. This means that shared environment in the current study is defined as the environment that is shared by siblings growing up in the same household. It is likely that there is some dependence in environment between the parents' environment and offspring's environment—for instance, if parents grow up in poverty, their children are more likely to grow up in poverty as well. Part of this can be attributed to heritability—which would be estimated by the model. However, this similarity in poverty may partly also be attributed to environmental effects. In the current study, this is only modeled in as far as siblings are similarly affected by growing up in the same or similar environments but not in as far as parents and offspring may grow up in similar environments. This similarity in environments is difficult to model because parents and offspring do not grow up in the same household in the same way as siblings do, and any assumption on the similarity in environment would be speculative unless explicitly assessed. Moreover, this similarity may differ from family to family. In some cases, offspring may, for instance, maintain the social status of their parents, while in others, offspring will have higher or lower SES. Therefore, we assessed SES and controlled for it.

Secondary analyses were conducted for physical and emotional abuse and emotional neglect following the same analysis steps to explore whether the results were specific one or more of these types of maltreatment. Physical neglect was excluded from the analyses as internal consistency was insufficient. Heritability of severe physical abuse was estimated using a liability threshold model for dichotomous phenotypes. The covariates age, age^2, sex, age $\times$ sex, age^2 $\times$ sex, SES and household were included in the analysis. In a sensitivity check, we repeated these analyses using multi-informant scores instead of self-report to measure experienced maltreatment. Multi-informant scores were computed as the averages of self-report and parent report when available (which was the case in 55% of the scores).

A bivariate polygenic model analysis was conducted for abuse and neglect to estimate from the phenotypic covariance their constituent genetic and environmental correlations between the two traits. Further, we tested for potential genotype $\times$ SES interaction effects on overall maltreatment. Genotype $\times$ SES interaction arises if the additive genetic variance underlying the trait of interest changes with the environment (SES in the present case) or if the across-environment genetic correlation is less than 1 or if both conditions are true. The genotype $\times$ SES interaction model is a reparameterized version of the polygenic model in which the additive genetic and residual environmental variances are allowed to change as functions of SES by way of "change" parameters respectively denoted by γ_g, γ_c, and γ_e, and in which the genetic correlation is expressed as an exponential decay function of pairwise differences in SES with parameter λ (Diego, Almasy, Dyer, Soler, &

Table 1. Estimates of Genetic (h^2), and Common (c^2), Unique (e) Environmental Effects on Overall Maltreatment, Neglect, Abuse, and Severe Physical Abuse.

Maltreatment Type	h^2 (*SE*), %	CI (%)	*p*	c^2 (*SE*), %	CI (%)	*p*	e (%)
Overall maltreatment	38 (19)	[1, 75]	.003	29 (9)	[11, 47]	.001	33
Neglect	30 (13)	[5, 55]	.01	28 (10)	[8, 48]	.002	42
Abuse	41 (13)	[16, 66]	.001	29 (10)	[9, 49]	.002	30
Severe physical abuse	62 (19)	[25, 99]	.001	9 (13)	[−16, 34]	.26	25

Note. *SE* = standard error; CI = 95% confidence interval.

Blangero, 2003). We first compared the full genotype × SES interaction model to the polygenic model by a likelihood ratio test. If warranted by the results of this general comparison, we then compared the full genotype × SES interaction model to either of its constrained versions in which the additive genetic variance was constrained to be constant ($\gamma_g = 0$) or in which the genetic correlation was constrained to be 1 ($\lambda = 0$). Next, common and unique environmental variances were constrained to be constant in turn ($\gamma_c = 0$, $\gamma_e = 0$).

Results

The number of participants who experienced the various types of maltreatment never, once, or more than once is displayed in Online Appendix Table S2, and the distributions are shown in Figure S1. Descriptive statistics and correlations between all variables are reported in Online Appendix Table S3. Abuse and neglect were correlated, phenotypic covariance, $r(393) = .56$, $p < .01$. Men and women did not differ on experienced maltreatment, abuse, neglect, age, or SES ($ps > .09$). Older participants reported more maltreatment and lower SES ($ps < .01$). Participants with lower SES tended to report more neglect, $r(393) = .13$, $p = .01$, but not abuse, abuse: $r(393) = -.04$, $p = .41$, severe physical abuse: $r(393) = -.06$, $p = .20$.

Heritability Analyses

All maltreatment phenotypes were partly heritable (Table 1 and Online Appendix Table S4) with estimates ranging from .30 ($SE = .13$) for neglect to .62 ($SE = .19$) for severe physical abuse indicating that a statistically significant proportion of the phenotypic variance was explained by kinship. Overlapping 95% confidence intervals (CIs) indicate that heritability estimates were similar for all maltreatment phenotypes. Common environmental effects explained a statistically significant proportion of variance for all phenotypes except for severe physical abuse ($c^2 = 0.09$, $SE = .13$, $p = .26$). CIs overlapped for all maltreatment phenotypes. In a secondary analysis, we confirmed that these results were not specific to the physical or emotional dimension of maltreatment (Online Appendix Table S5). In addition to self-reports, parent reports were available for 218 participants. In a sensitivity check, we showed that CIs for self-report and multi-informant report overlapped (Online Appendix Table S6). Justifications and results of power analysis are reported in Online Appendix S3. Power to detect heritability and common environment was adequate to excellent (.77–.93) with the exception of common environmental effects on severe physical abuse for which power was only .17. Power was also calculated for the same traits under constrained heritability models where the heritability was constrained to .25, .48, and .71. Only a large heritability estimate leads to sufficient power for most types of maltreatment except for severe physical abuse that occurred rather infrequently in our sample. Replication is needed in larger (at risk) samples with higher prevalence of the various maltreatment types, in particular severe physical abuse.

Bivariate Heritability Analysis

The bivariate analysis showed that the genetic correlation between abuse and neglect differed significantly from 0 ($p = .02$) as well as from 1 ($p = .02$) indicating partial pleiotropy (Figure 2). Pleiotropy occurs when the same genes contribute to different phenotypes. The genetic correlation was $\rho_g = .73$, indicating 53% overlap. This means that shared as well as different genetic factors contribute to abuse and neglect. The common environmental correlation of $\rho_c^2 = .74$ differed significantly from 0 ($p < .01$) but did not differ significantly from 1 ($p = .08$) indicating that there was substantial overlap in shared environmental factors accounting for variance in abuse and neglect. The unique environmental correlation was not significantly different from 0 ($\rho_e = .20$, $p = .40$) showing that the unique environmental factors associated with abuse and with neglect are not the same.

Genotype × SES

The genotype × SES model fitted the data significantly better than the main effects model ($p < .001$; Online Appendix Table S7 and Figure 3). Next, we compared the full genotype × SES model to its constrained alternatives ($\gamma_g = 0$ or $\lambda = 0$, $\gamma_c = 0$, and $\gamma_e = 0$). Model fit decreased significantly after constraining γ_c to zero ($p = .048$), indicating that the common environmental variance changed with SES. Specifically, common environmental variance increased as SES decreased ($\gamma_c = -1.11$, $SE = .40$). Constraining γ_g, λ, and γ_e to zero did not lead to significant changes in model fit, indicating that additive genetic and unique environmental variances were constant across different levels of SES.

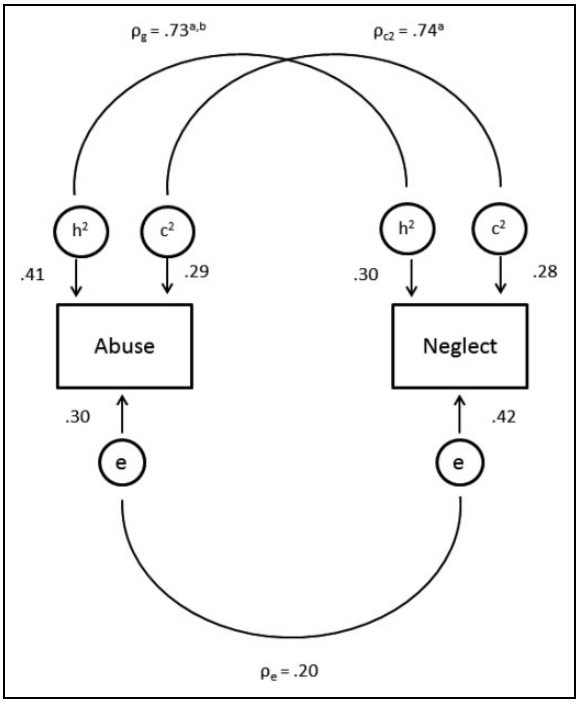

Figure 2. Bivariate relationship between abuse and neglect. [a]Significantly different from 0. [b]Significantly different from 1.

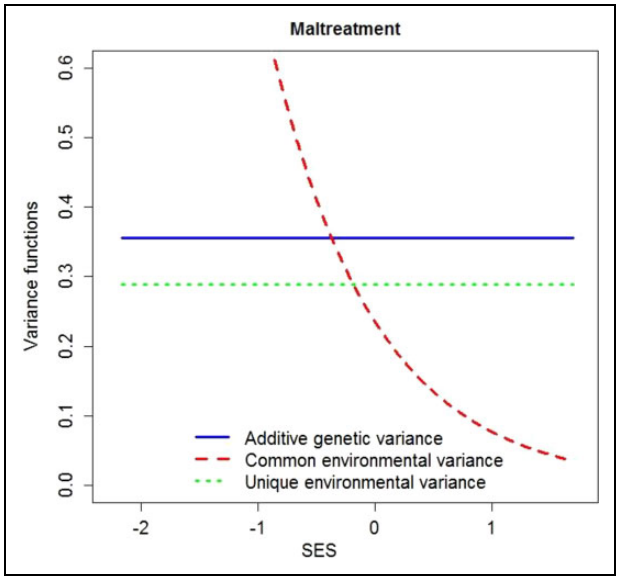

Figure 3. Genotype-by-socioeconomic status (SES) for overall maltreatment. Additive genetic and unique environmental variances were constant across different levels of SES. Common environmental variance decreased with higher SES. SES is an aggregate of standardized education and income per household (i.e., units approximately correspond to standard deviations).

Discussion

This extended family study demonstrates that experiencing maltreatment during childhood is partly heritable. Heritability was not restricted to a specific type of maltreatment, and shared genetic factors contributed to abuse and neglect. Common and unique environmental factors explained a considerable proportion of phenotypic variance, and common environment had a greater effect on maltreatment in low-SES families.

Heritability estimates ranged from 30% for neglect to 62% for severe physical abuse. These findings suggest that child maltreatment is in part genetically mediated by child effects.

The finding that child factors contribute to maltreatment does not imply, however, that the responsibility for maltreatment perpetrated by parents lies with the child. It is the role of parents to respond appropriately to challenging child behavior, and they might need support to fulfill this role adequately in case of challenging child behaviors. Interventions may benefit from incorporating parent training that supports more effective strategies of responding to potentially challenging behavior. Results from earlier genetically informed studies (i.e., adoption and twin designs) provide support for an evocative role of externalizing problems in negative parenting and maltreatment (Marceau et al., 2013; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998; Schulz-Heik et al., 2010). The association between externalizing behavior and maltreatment may be bidirectional as maltreatment increases antisocial behavior over time, even when taking into account genetic effects (Jaffee, Caspi, Moffitt, & Taylor, 2004).

In the present study, we used a continuous variable of maltreatment ranging from "no maltreatment," to "harsh parenting," and to "maltreatment." Most participants reported experiences of maltreatment at the lower end of the spectrum. Consequently, our findings may be restricted to more typical harsh parenting rather than maltreatment. However, we found that severe physical abuse seemed highly heritable, in contrast with an earlier twin study showing that harsh parenting, but not maltreatment, was heritable (Jaffee, Caspi, Moffitt, Polo-Tomas et al., 2004). Whereas Jaffee, Caspi, Moffitt, Polo-Tomas et al. (2004) assessed maltreatment up to the age of 5 years, the present study covered maltreatment to the end of adolescence, when individuals gain agency to shape their environment (Bergen, Gardner, & Kendler, 2007; Elkins, McGue, & Iacono, 1997), which may increase child-based genetic influences. Another explanation for the discrepancy might be that the present study used self-report, while in their twin study Jaffee, Caspi, Moffitt, Polo-Tomas et al. (2004) used mother report about the twins' maltreatment histories, which may have led to an overestimation of shared environment because parents tend to perceive the environment of their children as more similar than the children themselves do (Wade & Kendler, 2000). Other studies using self-report measures also found experienced maltreatment to be partly heritable (Fisher et al., 2015; Schulz-Heik et al., 2009; South et al., 2015). Arguably, children may have a tendency to emphasize the difference between the way they themselves were treated and how their siblings were treated. However, since siblings completed the questionnaires independent of each other, it is unlikely that in our study, using self-report has strongly increased the similarity between siblings. Moreover, using a multi-informant approach that included parent reports when available, we see the same

pattern of results with slightly higher estimates for heritability and common environment. This likely stems from a reduction of measurement error evident in the lower unique environment estimates.

Common and Unique Environment

The present findings suggest that similarity between siblings in terms of maltreatment experiences should not be attributed to genetic effects only but also to common environment. This points to the role of the family environment and is in line with studies showing that parental psychopathology, parenting stress, lack of social support, and larger family size are important risk factors for maltreatment. Low SES has repeatedly been shown to be associated with maltreatment (Euser et al., 2013; Sedlak et al., 2010; Slack, Holl, Mcdaniel, Yoo, & Bolger, 2004; Stith et al., 2009). In our study, SES was related to neglect. While some of these factors, such as family size, are difficult to change, addressing factors such as parenting stress and social support may have a particularly high payoff as they would benefit all children in the family.

For the etiology of maltreatment, it is important to not only understand what makes children growing up in the same family similar but also what makes them different in the experience of maltreatment—the unique environment (Plomin, 2011). We found an estimated influence of unique environment (including measurement error) of 29–42%, which concurs with previous studies (Fisher et al., 2015; Schulz-Heik et al., 2009). The importance of the unique environment points to the need for an individual child approach in addition to a family-centered approach when estimating risk. That is, it is important to improve the specific parent–child relationship. Nonetheless, specific unique environmental risk factors have remained elusive after taking measurement error into account (Deater-Deckard et al., 2001; Mullineauxa, Deater-Deckard, Petrillb, & Thompson, 2009). One suggested factor is that parents might perceive siblings as differently attractive or difficult, and thus trigger differential parenting (Burt, McGue, Iacono, & Krueger, 2006; Deater-Deckard, Smith, Ivy, & Petril, 2005; Feinberg & Hetherington, 2001; Reiss et al., 1995), which constitutes a potential target of intervention.

Genetic Correlation Between Abuse and Neglect

Our bivariate analysis indicates that the same common environmental factors are related to abuse and neglect. Approximately 50% of the genetic factors were overlapping, and the other 50% were uniquely related to abuse or neglect. This may suggest that some child factors put a child at risk of experiencing abuse but not of neglect and vice versa. Our findings illustrate why abuse and neglect often co-occur (Euser et al., 2013; Vachon, Krueger, Rogosch, & Cicchetti, 2015) notwithstanding etiological differences. Conversely, interventions may need to address abuse and neglect individually, even when they co-occur, since the heritable and unique environmental risk factors do not (fully) overlap.

Genotype × SES

Moreover, a genotype × SES interaction analysis demonstrated that in low-SES families, common environment explained more variance in experienced maltreatment than in high-SES families. Overall, low-SES families showed greater variance in experienced maltreatment, and our findings suggest that this can be attributed to common environment. Lower SES may add a range of common environmental factors negatively affecting child development. For instance, children from low-SES families experience more instability, more crowding at home, more pollution, and more danger in the neighborhood (Chen & Miller, 2013; Evans, 2004; Miller et al., 2009). Together, these factors may increase the risk of developing externalizing problems. On a population level, this suggests that fighting child poverty may have far-reaching preventive consequences.

Additive genetic variance, in absolute terms, remained stable across different levels of SES. Since overall variance decreased as SES increased, relative contribution of genetic variance component increased. Given the substantial effects of heritability this and previous studies have indicated, a more comprehensive exploration of environmental effects on heritability may uncover new intervention targets. A better understanding of the child traits mediating the heritable risk might offer insight into which environmental manipulations would be most effective in lowering heritable risk.

In order to interpret any variance component across a changing environment, it is important to consider changes in the other variance components. In a genotype-by-sex interaction study of physical activity behavior by Diego et al. (2015), the issue of the indeterminacy of environment-specific heritability was broached. The authors found that the heritability could be constant across an environmental contrast if the constituent variance components changed in the same direction and at the same rate. They also noted that it was theoretically possible that a nonconstant heritability across an environmental contrast could arise from a changing residual environment component in the face of a constant additive genetic variance. This concept is relevant to properly contextualizing our results with existing reports on the heritability of maltreatment. In particular, Schulz-Heik et al. (2009) and South, Schafer, and Ferraro (2015), respectively, reported a higher and lower proportion of the total phenotypic variance attributed to the shared environment relative to the heritability. Regarding our study, we can actually claim both scenarios because the shared environment variance component declined relative to a constant additive genetic variance from the low end of the SES spectrum to the high end.

Extended Family Design

For the current study, we decided to use an extended family design to add to the existing twin research. Extended family designs have more variability in genetic relatedness and common environment than twin designs. In addition, twins create a

unique family constellation and parenting demands may be atypical when caring for two same-aged children (Olivennes, Golombok, Ramogida, & Rust, 2005). Consequently, results from twin studies may not be generalizable to typical family constellations.

Moreover, the extended family design decreases the confounding between genetic relatedness and shared environment compared to nuclear families (Almasy & Blangero, 2010; Diego, Kent, & Blangero, 2015). By including horizontal relationships (e.g., cousins, half-siblings), in addition to vertical relationships (e.g., grandparent–grandchild), a systematic correlation between genetic distance and age difference is eliminated. For instance, half-siblings and grandparent–grandchild pairs have the same genetic distance, but half-siblings tend to be similar in age whereas grandparent–grandchild pairs are not.

Limitations

A limitation of this study is the retrospective assessment of maltreatment; no conclusions about causality can therefore be drawn. For ethical reasons, research on maltreatment is generally incompatible with experimental designs except for intervention studies that combined with a prospective design can be highly informative. The present study assessed maltreatment retrospectively, and time between potential maltreatment and assessment varied. Moreover, estimates of unique environment should be interpreted with caution as it is impossible to disentangle unique environmental effects from measurement. It is interesting to note, however, that estimates of unique environment decreased when including parent reports for a multi-informant approach. This could point to a reduction in measurement error. Future research should strive to replicate these findings in a larger, representative sample and in other populations (e.g., non-Western). Estimates from quantitative genetic research are population-specific. Even if genetic variation is stable across populations (which we do not know), environmental variability will affect estimates for both heritability and environment because these estimates represent relative contributions (Plomin, 2018; Velden, 1997). Lastly, the present sample may have been too small to estimate moderator effects reliably (Glahn et al., 2010)—especially because the effect of SES on common environment was small and the moderation analysis was exploratory. Future studies should replicate the genotype $\times$ SES effect in larger samples as these findings suggest that environmental interventions can be particularly useful.

Implications

Ideally, interventions are based on empirically supported, theoretical frameworks of etiology. The current study suggests that such frameworks should incorporate the heritability of experiencing maltreatment and that interventions should address both heritable and environmental risk factors. More research is needed to determine how to best reduce those risk factors. Moreover, it would be useful to explore other environmental factors than SES and how they moderate heritability—preferably factors that can be the focus of interventions.

Conclusion

The present study shows that both genetic and environmental factors are critically involved in experiencing maltreatment. Findings further suggest that abuse and neglect share common pathways that are important because experiencing more than one type of maltreatment is related to worse outcomes (Vachon et al., 2015). Meta-analytic evidence suggests that in general, current interventions are insufficient in preventing maltreatment (Euser, Alink, Stoltenborgh, Bakermans-Kranenburg, & Van IJzendoorn, 2015). A better understanding of the transactional relationship between child and parent risk factors may be crucial in developing more targeted prevention measures. For instance, interventions can use video feedback to train parents in strategies to respond to specifically challenging problem behavior in children (Klein Velderman et al., 2006). The importance of intervention cannot be underestimated: Not only because the current study shows considerable associations with environmental factors but also because heritability does in no way preclude or limit the influence of environmental change.

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Supplemental Material

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