Intergenerational Transmission of Child Abuse and Neglect: A Transdisciplinary Analysis

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Abstract

Parents who experienced maltreatment in childhood may be at an increased risk of presenting abusive or neglectful behavior toward their own children. Research suggests reoccurring patterns of abuse and neglect across generations, but the factors that influence these behavioral patterns are complex and poorly understood. Although abusive or neglectful parenting styles undoubtedly are strongly influenced by social factors (ie, "nurture"), there might be underlying biological components to perpetuating behavioral patterns (ie, "nature"). Importantly, nature and nurture are known to interact in shaping developmental outcomes, and as such should not be considered in isolation. In this review, we examine the evidence regarding the inheritance of abusive and/or neglectful parenting behavior using a transdisciplinary approach. We integrate knowledge of the social and biological science fields on the continuance of abusive and neglectful behavior, as well as the methodological challenges that complicate the interpretation of existing research. Finally, we stress the importance of considering contextual factors of both social and biological research findings concerning the intergenerational inheritance of child abuse and neglect and discuss the potential for early social intervention to disrupt harmful intergenerational patterns.

Keywords

child maltreatment, intergenerational transmission, inheritance, abuse, neglect, biology

Introduction

Child maltreatment can have devastating psychological and neurobiological consequences. Major areas in which damage can occur include behavioral and affect regulation, attachment relationships, development of identity and self-esteem, peer relationships, and academic performance and adaptation.[1] Consequences may extend into adulthood and include internalizing and externalizing behavior problems, post-traumatic stress, alcohol abuse, obesity, chronic pain, and sexually transmitted infections,[2] unemployment, poverty, and social assistance usage,[3] as well as impairments in attention, abstract reasoning,[4] working memory,[5] problem-solving,[6] verbal and nonverbal fluency, verbal and nonverbal inhibition, and directed attention.[7]

To prevent abuse and neglect and the associated consequences, it is critical to determine all the factors that operate in concert to cause maltreatment. The etiology of child abuse and neglect is generally viewed as complex and multiply determined.[8] Estimates of intergenerational transmission of maltreatment vary widely, underscoring the methodological limitations inherent to studying parenting across generations and the lack of clarity in this field of study (see Table 1).

Importantly, existing reviews have only included a focus on either social science research[63] *or* biological research,[64-66] and several have given too much weight to methodologically weak studies (see[63]).

The purpose of this article is to review the evidence on the role of childhood maltreatment in predicting later abusive and neglectful behavior in order to identify possible mechanisms and interactions influencing the continuity and discontinuity of harmful parenting practices across generations. This article

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goes beyond existing reviews by examining a growing body of biological research that focuses on understanding the consequences of abuse and neglect as well as the genetic contributions to parenting behavior, while also addressing the specific social mechanisms that might promote or disrupt harmful parenting practices, with special attention on the role of stress. Importantly, we address the interaction between social factors ("nurture") and biological factors ("nature"), explaining how it is the gene–environment (G×E) interplay that may drive intergenerational patterns of abuse and neglect, rather than either social or biological factors alone. This is the first review to the authors' knowledge that utilizes a critical transdisciplinary approach to understanding the intergenerational transmission of maltreatment.

Methods

Literature searches were conducted in an iterative manner in several major interdisciplinary databases, including JSTOR, EBSCO, Google Scholar, OVID, ProQuest, Project Muse, PubMed, and Scopus. Search terms included "intergenerational or transgenerational," "child abuse or child neglect or child maltreatment," "early life stress," "parenting continuity," "early life stress biology," "epigenetic embedding," and "epigenetic inheritance." Reference lists of seminal articles and existing reviews were examined for additional sources. The review focused specifically on the intergenerational transmission of maltreatment as well as on the general social and biological causes and consequences of maltreatment. We also included sources focused on the general transmission of parenting behavior across generations, or in other words, the ways that the parenting styles of one generation influence the parenting behavior of the next generation.

After screening out irrelevant articles (eg, studies focused very generally on child maltreatment without attention to multiple generations; or studies focused on early-life stresses with no relation to abuse or neglect), 59 original research studies were included for further examination and review. Each study was summarized and critically appraised for its limitations to the present review, and the practical implications of the findings were considered during ongoing collaborative discussions among the authors. The characteristics of the studies were outlined in a table to easily compare and interpret results (see Table 1). Additionally, we reviewed 38 relevant reviews, systematic reviews, and meta-analyses. The findings were synthesized in order to determine whether childhood maltreatment is an important predictor of later abusive and neglectful parenting and to identify mechanisms explaining the transmission of maltreatment across generations.

Can Childhood Maltreatment Predict Parenting Practices Later in Life?

Existing research indicates that childhood maltreatment is an important consideration in determining the potential for child abuse and neglect along with many other factors. Rates of maltreatment in families headed by individuals with childhood histories of abuse and neglect are higher than the general population.[67] One study found that 6.7% of parents with a history of childhood abuse were referred for maltreating their own child.[22] This was compared to under 1% of parents without such histories.[22] Other studies report even higher rates of maltreatment among parents with childhood histories of abuse and neglect, but these higher estimates are usually reflective of the sample and design. For example, studies conducted with samples of parents already identified as abusive or neglectful tend to produce higher estimates of intergenerational transmission, as well as studies employing a retrospective design.[68]

Although researchers have used a wide range of methods to classify maltreatment histories and the perpetration of abuse and neglect, existing research often fails to adequately capture the heterogeneity of maltreatment experiences. Individual experiences of abuse and neglect are diverse in nature (eg, severity, duration, developmental period, relationship to perpetrator) and so are the children who experience the abuse (eg, age, gender, genetic makeup). Understanding the specific characteristics of maltreatment that are most associated with the continuity of harmful parenting is an important yet understudied area. Treating parents with a history of maltreatment in their own childhoods as a homogenous group may mask important differences and sequelae.

Furthermore, experiencing child maltreatment may impact future parenting in nuanced ways. Instead of replicating exact types or forms of maltreatment from their childhoods, it is possible that parents with a history of abuse and neglect may simply have a diminished capacity to cope with the everyday stresses and challenges of raising children (see[69,70]). This is supported by biological studies which show that experiencing abuse or neglect in childhood can have long-lasting effects on the biological systems that allow individuals to cope with stressful situations, altering the behavioral response to stress in adulthood.[71] This may result in these parents engaging in suboptimal caregiving practices that do not necessarily meet the threshold to qualify as maltreatment yet are still detrimental to child development.

Whether the mechanism for intergenerational inheritance for child abuse and neglect is biological, behavioral, and/or mediated by the social environment is difficult to tease apart because these factors are closely correlated and interconnected. Nevertheless, the evidence is strong that these intergenerational patterns exist, and as such it is important to consider all the factors that might drive them to better inform prevention policies. Below we examine the social and biological factors that might contribute to transgenerational patterns of abuse and neglect in an integrative way, organized within the following categories: social support, poverty and structural disadvantage, mental health and emotion regulation, the biology of childhood abuse from an early-life stress perspective, genetic predispositions and individual differences, epigenetic embedding of maltreatment, epigenetic inheritance of maltreatment, and timing of maltreatment. These categories were selected after careful consideration and a synthesis of existing literature to identify

prominent themes. It is important to note that these categories were chosen not because they encompass all the factors contributing to the transmission of child abuse and neglect, but as a means of organizing the many interconnected factors influencing parenting across generations.

Mechanisms to Explain Intergenerational Transmission of Maltreatment

Social Support

The synthesis of the literature suggests that social support and positive relationships are potential mechanisms that influence the discontinuation of maltreatment across generations. Social support refers to the various forms of assistance that individuals receive from family members and others and may include emotional support, instrumental support, and informational support.[72] Social support can enhance resilience to stress and may play a role in optimizing the neurochemical response during and after exposure to a stressor.[73] Individuals who experience maltreatment in childhood may be better able to cope with their traumatic experiences as well as the daily stressors involved in parenting if they have access to social support, such as friends who make them feel cared for, family members who help with childcare and housekeeping, and community services that offer information about positive parenting strategies. Whereas social support appears to act as a buffer against negative outcomes,[23,74] social isolation is associated with an increased risk of perpetrating abuse and neglect among parents with a history of child maltreatment.[8,23] In the absence of social support, individuals with a history of maltreatment may be more likely to engage in abusive and neglectful behaviors when parenting.

The question remains of why some maltreatment survivors develop strong social support systems whereas others are unable to establish such relationships. As discussed further below, likely the answer lies at least partially in G×E interactions[74] and individual differences (including sex differences) of susceptibility and resilience to early-life experiences.[10,75]

Poverty and Structural Disadvantage

Child maltreatment is only one risk factor in what is typically a constellation or accumulation of risk in the lives of disadvantaged families.[39,76] A major environmental stressor in the lives of maltreated children is often poverty and marginalization, and all the accompanying hardships such as poor housing and unemployment.[76] Poverty can undermine parenting and heighten the risk of child maltreatment because of the stress caused by living in poor conditions with insufficient resources.[77] The relationship between child maltreatment and poverty and marginalization is generally uncontested. Eckenrode and colleagues[25] examined the relationship between income inequality in US counties and county-level child maltreatment rates. This analysis demonstrated a strong positive linear relationship between county income inequality and maltreatment rates, and this relationship was even stronger in counties with moderate to high levels of child poverty. More broadly, child development is negatively impacted by not only parents' level of socioeconomic disadvantage but also grandparents' level of socioeconomic disadvantage,[43] and there appears to be significant intergenerational continuity of economic hardship.[3,33,54]

It is possible that any intergenerational transmission of parenting is explained in part by the intergenerational transmission of socioeconomic status, with families experiencing economic hardship at greater risk of maltreatment across generations because of their impoverished and stressful living conditions. Children who experience maltreatment and live in the context of socioeconomic *advantage* may be less likely to engage in abusive or neglectful behaviors in adulthood.

Mental Health and Emotion Regulation

Mental health status in adulthood is another factor that may influence the risk of maltreatment perpetration among individuals who were abused or neglected in childhood. Certain experiences of maltreatment can result in persistent changes to mental, emotional, and behavioral functioning, and these changes have consequences that extend to future parenting. Examining continuity in parenting across 2 generations, Neppl and colleagues[44] found that harsh parenting is associated with the development of externalizing behaviors (eg, aggression) in children, and further that these externalizing behaviors continue into adulthood and predict harsh parenting of the next generation. Research also suggests that maternal childhood maltreatment predicts children's exposure to maltreatment only in combination with maternal depression.[49] Children whose mothers were maltreated in childhood but did not suffer from depression late in pregnancy were *not* at increased risk of experiencing abuse or neglect themselves.[49] This again highlights the importance of G×E interactions, as it is likely the interaction between the early-life experience and genetic predispositions that drives the development of mental health disorders in abused children. This explains why only some children who are maltreated develop behavioral disorders in childhood and adolescence which are then carried into parenting practices in adulthood.

Some evidence suggests that the relationship between experiencing and perpetrating childhood abuse is mediated by emotional disorders. In a sample of 83 mothers living in high stress, high-risk environments, with high levels of lifetime trauma exposure, researchers found an association between greater levels of maternal childhood abuse and increased current child abuse potential; and this association was mediated by emotion dysregulation and negative affect.[57] The influence of mental health and emotion regulation on parenting behavior has also been studied from a biological perspective. Biological studies (discussed in detail below) clearly show that early-life stress in the form of abuse or neglect can have life-long consequences for mental health and the ability to deal with everyday stressors. However, it is not fully understood how mental

health and emotions influence the transmission of parenting across generations, as some studies have found that mental health status does not change the relationship between a history of child maltreatment and later maltreatment perpetration.[8,20]

Biology of Childhood Abuse From an Early-Life Stress Perspective

Experiencing abusive environments is stressful, especially for children, and long-term exposure to stress during childhood and adolescence can affect one's ability to deal with stress in adulthood. Stress affects a variety of biological functions and often this is related to changes of activity in the hypothalamic–pituitary–adrenal (HPA) axis,[78] a neuronal and hormonal biological system that regulates the brain's response to stress. Specifically, childhood abuse or neglect has been shown to have profound influences on several components of HPA regulation. Heim and colleagues[32] found that women with a history of childhood abuse exhibited increased adrenocorticotropic hormone (ACTH), an HPA regulator, in response to stress and that this was linked to major depression. These women also demonstrated increased cortisol and increased heart rates in response to psychosocial stress.[32] Rodent and nonhuman primate animal models have provided further evidence that early-life stress results in increased expression of HPA hormones, particularly of the corticotropin-releasing hormone (CRH).[19,50] The main function of CRH is to stimulate the production of ACTH, which at increased levels results in physiological and behavioral changes that closely parallel symptoms of depression and anxiety.[79] Similarly, neglectful mothering results in increased CRH expression and enhanced stress-induced activation of the HPA axis in rats.[11,80] These changes in activity of the HPA's components can have adverse effects on one's emotional stability[16,54,81] and propensity for violent behavior,[82,83] and might present a biological link to neglectful or abusive parenting cycles. In fact, parents who have been maltreated in childhood experience higher levels of stress[58] and a lower capacity for emotional control, factors that reduce the likelihood of sensitive parenting[48] and increase child abuse potential.[27]

The effects of childhood abuse on the HPA might be further exacerbated by socioeconomic disadvantage, one of the main social factors associated with childhood abuse and neglect. Socioeconomic status is strongly correlated with mental health outcomes (eg, negative emotions, depression, anxiety) and this relationship seems to be at least in part mediated through changes in biological stress response systems.[84] Importantly, the responses of the HPA axis to childhood stress vary among individuals and between sexes. Because the HPA axis is a hormonal system, there are inherent hormonal sex differences in its regulation.[85] While some evidence suggest that girls have a naturally stronger HPA axis response to social stress in childhood,[86] other studies found that young men have higher HPA axis responses to psychological stress in adulthood.[87] Nevertheless, systematic reviews show that the presence of sex differences varies largely across studies,[86] likely because of

methodological discrepancies but also because of individual differences within the same sex (see section below).

Genetic Predispositions and Individual Differences

Genetic predispositions can influence the psychological and biological effects of stressful environments.[88] Although we all share the same set of genes in our DNA, genetic variants (single-nucleotide polymorphisms [SNPs]) can differ across individuals and, in some cases, have been shown to affect how an individual will respond to their environment. These G×E interactions have been implicated in a variety of HPA functions. For instance, individuals with an SNP in the serotonin transporter-linked polymorphic region (5-HTTLPR) are at greater risk for severe depression and suicide,[16,29,54,81] when in negative social environments. This is dependent on both the genotype and the environment, as the presence of the SNPs or the social risk factors alone does not result in increased risk. Similarly, SNPs of genes involved in neuronal signaling (primarily in dopamine and serotonin signaling, neurotransmitters that influence mental health and behavior), such as 5-HTTLPR, catechol-O-methyltransferase, monoamine oxidase A, and the dopamine D4 receptor, when combined with adverse environmental factors, have been shown to increase antisocial behavior and impulsive violence.[82] Furthermore, SNPs have been implicated in predispositions to addictions, which are related to other psychiatric diseases by common neurobiological pathways, including those that modulate reward, behavioral control, and anxiety or stress response.[83] Depression, high stress levels, antisocial behavior, and substance abuse are often correlated with abusive or neglectful parenting styles,[12,31,36,89] and G×E interactions influencing these traits are important to consider when studying the biological transmission of maltreating behavior.

Epigenetic Embedding of Maltreatment

It is possible that inability of the nervous system to cope with stress might play a role in abusive parenting, but the question remains about how childhood experiences affect HPA function into adulthood. To explore the possibility of biological factors playing a role in intergenerational transmission of abuse and neglect, it is important to consider the mechanisms that might perpetuate early-life effects. One possible mechanism is epigenetic embedding, a molecular process that can induce stable, long-term alterations in gene function[65] in response to the environment. There are a large variety of epigenetic modifications, which are interconnected in complex pathways that are often not well understood, and the placement and effect of these marks can depend on genetic background.[37,74,90,91] Importantly, epigenetic marks have been shown to be both stable and transient, allowing biological changes to become long term, but reversible.

The first evidence that maternal behavior produces stable epigenetic alterations was provided by Meaney and

colleagues.[92] This study found that rat mothers vary in how much they lick and groom their pups and offspring who receive less of this maternal care show significantly higher stress levels. Importantly, pups that receive less licking and grooming grow up to provide their offspring with less maternal care, while pups that received more maternal care grow up to provide their pups with higher levels of licking and grooming.[14] Weaver and colleagues[62] found that rat pups that experienced lower maternal care show changes in the epigenetic regulation of the glucocorticoid receptor (*GR*), a gene important in HPA axis regulation. These changes were acquired during the first weeks of life and persisted into adulthood but were absent in pups that were cross-fostered with a high maternal care mother from birth. The work of Weaver and colleagues revealed that the transmission of mothering behavior has a biological component and that the biological effects were dependent on early mother–offspring interactions. Roth and colleagues[53] showed that early adversity of pups—in the form of lower levels of maternal care during infancy—affected epigenetic regulation of genes involved in neuroplasticity. This is important because neuroplasticity allows the adult brain to adapt to new situations, and this is impaired with prolonged stress and depression.[93-95] Other studies show that neglected pups have altered epigenetic regulation of the HPA hormone arginine vasopressin[42] and the transcription factor REST4, resulting in depression-like behaviors following repeated exposure to stress.[60]

Although the research on the epigenetic effects of abuse and neglect is much more extensive in rodent models, there are also human studies showing that early-life stress and abuse can result in epigenetic alteration of HPA axis genes. The nature and ethics of human research create challenges in comprehensively investigating epigenetic changes in the brain, but studies on human blood and saliva have shown that childhood abuse and maternal depression result in epigenetic alteration of HPA axis genes.[41,46,52] Interestingly, Klengel and colleagues[37] found that the susceptibility to epigenetic changes in HPA axis genes due to early-life stress is dependent on genetic predisposition. Humans have 2 genetic variants of the *FKBP5* gene, one that is demethylated in response to childhood trauma and one that is not. The demethylation of the "susceptible" variant of this gene is associated with long-term impairment of stress regulation, while the "protective" variant is not. Another study found that the early care environment shapes genome-wide epigenetic landscapes and that this is driven by the interaction of infant attachment and child genetic variation.[74] The effects of this G×E interaction on epigenetic landscape and behavioral outcomes were more pronounced in females, highlighting the importance of considering sex differences in G×E studies.[74] Interestingly, maternal separation correlates with epigenetic changes that diminish responsiveness to adolescent antidepressant treatment,[38] highlighting the importance of prevention efforts before psychosocial disorders develop.

Although epigenetic mechanisms that perpetuate the effects of early-life stress into adulthood might be conserved across species,[45] none of the studies described so far provide evidence that biological effects associated with the experience of abuse and neglect can be inherited transgenerationally. In fact, as discussed in the next section, the biological barriers to epigenetic transmission, the lack of evidence for true transgenerational epigenetic inheritance, and the cross-fostering experiments in rats mentioned above[62] suggest that the epigenetic effects on HPA regulation are experience-based and acquired anew every generation.

Epigenetic Inheritance of Maltreatment

For epigenetics to drive a perpetuating cycle of abusive and neglectful behavior, these marks would have to be heritable, being transmitted from parents to children. Intergenerational epigenetic inheritance is a controversial topic, since a large body of research shows that to ensure that a single-cell embryo develops into an adult human with many different cell types, epigenetic marks need to be almost completely erased during gamete formation and after fertilization (for review, see[96]). Furthermore, it is hard to rule out the effects of direct exposure, since, in a pregnant mother, 3 generations (ie, the mother, the fetus, and the fetus' germline) are directly exposed to the same environmental conditions. This means that studies must go as far as the fourth generation to distinguish true epigenetic inheritance from direct exposure effects. Since epigenetic changes are transient by nature, they could be reversed by the third or fourth generation. Although some research in humans argues for inheritance of environmentally induced epigenetic marks,[56,61] this research is still unable to address all the caveats mentioned above.

However, several studies show that intergenerational inheritance of epigenetic marks occurs in plants and invertebrates and suggests that it might occur in mammals and humans as well. In fruit flies and mice, epigenetic changes induced by paternal diet are passed on to the next generation.[13,47] Interestingly, the same epigenetic patterns that are inherited in the fly correlate with obesity in humans and mice, suggesting that this intergenerational transmission of epigenetic marks might also occur in mammals (including humans;[47]). Dias and Ressler[21] found that traumatic exposure associated with an odor results in changes in DNA methylation of the odorant receptor *Olfr151* in male mice and that these changes are maintained in sperm and at least 2 generations of offspring. Because father-to-offspring transmission excludes difficult to control oocyte and gestational effects, these studies are an indication of direct transmission of acquired epigenetic marks from one generation to the next. Nevertheless, evidence for epigenetic transgenerational inheritance underlying abusive parenting or even aggressive behavior in general is lacking, and although epigenetic inheritance might present a viable mechanism for biological transmission of behavioral patterns, more research on this subject is needed to establish this link.

Whether the effects of stressful experiences are inherited epigenetically or not, Yehuda and colleagues[61] found significant sex differences in these inheritance patterns. While female and male holocaust survivors both showed the same epigenetic alterations in the HPA axis, the effects on their offspring were

strongly dependent on the sex of the parent. Children whose mothers or both parents survived the holocaust had epigenetic alterations that correlated with higher HPA axis reactivity and higher anxiety, while children whose father was the only parent affected had epigenetic alterations that correlated with decreased HPA axis reactivity and increased emotional detachment.[61]

Timing of Maltreatment

The timing of early-life experiences has recently started to emerge as another important factor that moderates later-life outcomes. The effects of maltreatment depend not only on all the genetic and social factors mentioned above but also on the timing of the traumatic experience. Children's brains are more sensitive to specific experiences during certain windows in development (known as critical periods), meaning that if maltreatment occurs during this window, it might have larger effects.[64,88,97] For instance, Thornberry and Henry[59] suggest that childhood-limited maltreatment is not associated with an increased risk of becoming a perpetrator of maltreatment, whereas adolescent maltreatment and continuous maltreatment through childhood and adolescence are associated with a significant increase in the odds of becoming a perpetrator. Consistent with a dose–response model, maltreatment that occurred in adolescence tended to be more chronic and severe (ie, victims experienced higher levels of abuse for longer periods) compared to childhood-limited maltreatment.[59] Other research also points to the harmful impact of chronic, daily maltreatment, noting that it can cause significantly more damage to developmental health than single dramatic occurrences of abuse.[71] In line with this are findings from the biological literature that show that the timing (ie, when in life, and for how long) of early-life stress strongly influences the later-life biological effects.[42,66] This suggests that more temporary and less severe forms of abuse and neglect—depending on developmental timing—may have a less significant impact on later parenting practices. Importantly, the environment later in life can ameliorate the effects of childhood abuse. Supportive romantic relationships in adulthood have been shown to have a protective influence that might prevent intergenerational transmission of abuse. Conger and colleagues[17] found that warm, positive, and nurturing communication by a partner was associated with a lower risk of harsh parenting among individuals with a history of experiencing harsh parenting in their own childhoods. Similarly, Schofield and colleagues[98] conducted a meta-analysis and found that safe, stable, and nurturing adult relationships moderated the association between parents' history of maltreatment in childhood and their abusive behaviors toward children, such that these relationships acted as a buffer or protective factor.

Methodological Challenges

Studying the intergenerational transmission of maltreatment is challenging and as a result, the body of existing research has significant limitations. Social science studies differ significantly in their methodology and do often not adhere to standards in the field (eg, selecting a representative sample, prospective rather than retrospective data, clear definition of maltreatment).[63] This is important because study quality is negatively associated with the strength of support for the intergenerational transmission of child maltreatment.[67] Similarly, the studies listed in this review vary widely in methods and findings (see Table 1). Some of the main methodological challenges of social science studies include cross-sectional designs that rely on retrospective reports and are subject to recall bias;[18] reliance on child protection records that miss a large population of children for whom maltreatment goes unreported and might not reflect the reality of children's experiences[76]; lack of distinction between substantiated and unsubstantiated cases in child protection records[24]; inconsistent definitions of intergenerational continuity (self-reported vs child protection records; and lack of information on whether children experienced maltreatment from a parent directly).

Future research should use multiple sources of information to assess maltreatment experiences. In fact, one of the few studies to use multiple sources of information to measure intergenerational maltreatment found differences in the results depending on the source of information, with researchers noting that relying on only one source of information may lead to incorrect conclusions.[99]

Methodological inconsistencies in defining maltreatment also have trickle-down effects that hamper genetics research. The search for genetic or epigenetic variants that might influence maltreatment relies on consistent measures of maltreatment, and the inheritance of such factors relies on the biological parent being the perpetrator. In addition, the fact that some genetic variants are more sensitive than others to abuse adds another layer of complication (for further reading on the differential susceptibility hypothesis, see[75]). This means that some children who experience abuse might not show any biological effects, resulting in more heterogeneous study cohorts that are more difficult to analyze. If the characterization of behavioral phenotypes or abusive experiences is inconsistent, the correlations obtained in these studies might be erroneous.

Another limitation is that few social science studies on intergenerational maltreatment include a representative sample; most focus on clinical samples and/or relatively homogenous populations. Interestingly, one of the few studies to use a population-based sample reported not only a low prevalence of parental history of maltreatment but also a low rate of intergenerational transmission of abuse.[23]

It is also important to note several broader issues with studying parenting practices. Holden and Miller[100] point out that it has long been assumed by researchers that parenting is unchanging and stable across time and children. However, parents may interact with children in the family in various ways and may also structure children's physical and social worlds quite differently, depending on factors such as age, sex, birth order, and temperament. Parents may also change their behavior depending on the context, even the time of day. Methodologically, it is

Table 1. Summary of Methods and Findings of Original Research Papers Included in This Review.

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Augusti, 2013[5]	8- to 12-year-olds of European, middle eastern, African and Asian descent	44	Trauma assessed with TSCYC; spatial working memory tested with the Cambridge Neuropsychological Test Automated Battery; Executive function tested with Delis-Kaplan Exec Function System Color-Word Interference Test	Maltreated children performed significantly poorer on Spatial Working Memory tasks, but not on executive functions tests.	Small, heterogeneous sample.
Bakermans-Kranenburg, 2006[9]	10-month-olds with twin siblings sampled through the Netherlands Twin Register	47	Maternal sensitivity rated from 1.5 hours of videotaped observations with Ainsworth's 9-point rating scale. Child behavior assessed with the Child Behavior Check List (CBCL). DRD4 gene allele assessed by PCR.	Maternal insensitivity was associated with externalizing behaviors, but only in the presence of the DRD4 7-repeat allele. Children were differentially susceptible to insensitive parenting based on which 7-repeat DRD4 allele they had.	Small sample size.
Beers, 2002[4]	8- to 14-year-olds with and without PTSD of white and African American descent.	29	Trauma assessed by interview with psychiatrist. PTSD diagnosis based on DSM-IV. Neuropsychological function measured with language, attention, abstract reasoning/ executive function, learning and memory, visual-spatial processing, and psychomotor function tests.	Children with PTSD performed more poorly on measures of attention and abstract reasoning/executive function	Small sample size. Lack of a comparison group of maltreated children without PTSD.
Belsky, 2005[10]	DMHDS Cohort (Dunedin, New Zealand), European ancestry, Maori, and Polynesian.	228	Measurement of parenting (videos of parent–child interactions); longitudinal measures of the parent's childrearing history 3-15 years of age; measures of the quality of the parent's partner or spouse relationship.	Childrearing history significantly affected mothers' but not father's parenting. Romantic relationship quality didn't affect mothering or fathering behavior.	Study measured levels of warm-sensitive-stimulating rather than harsh parenting behavior and did not include cases of maltreatment. Parent's childhood history was self-reported (long time gap).
Berlin, 2011[8]	12- to 41-year-old mothers with infants from the United States. Wide range of ethnicity and income level.	499	Mothers' history of maltreatment was assessed with PC-CTS. Measures of mental health (CIDI-SF) and social information processing were self-reported. Records of offspring maltreatment.	Mothers who experienced physical abuse were 20% more likely to maltreat their children, independent of ethnicity, age, education, family income, and childhood neglect.	Parent's history of abuse was self-reported (long time gap).
Caldji, 1998[11]	Model organism (rats)	3-5/exp. condition	Measures of mothering behavior in dams; fear/anxiety behavior of the offspring; and molecular markers for fearfulness in the brain of offspring.	Offspring of lower interaction mothers was more fearful, and more sensitive to stress. The effects were mediated by molecular changes in the brain.	Unknown if findings translate to humans. Mothering behavior not measured in offspring. Only 1 generation tested.
Campbell, 2004[12]	Mothers and 36-month-old infants from the NICHD Study of Early Child Care; 85% white, 15% ethnic minority.	1077	Maternal depression (CES-D) and maternal sensitivity (observation) assessed at 1, 6, 15, 24, and 36 months. Attachment security measured (videos) 36 months.	Insecure attachment correlated with maternal depression in a dose-dependent manner. Course and timing of maternal depressive symptoms interacted with maternal sensitivity to predict insecurity.	Maternal depression was self-reported, and few of the subjects were likely clinically depressed.

Table I. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Carone, 2010[13]	Model organism (mice)	4-16/exp. condition	Manipulation of paternal diet and epigenetic profiling of offspring	Diet-induced epigenetic changes on metabolic genes are passed on to the offspring.	Unknown if findings translate to humans. Only 1 generation tested.
Champagne, 2003[14]	Model organism (rats)	40	Maternal behavior of each dam was observed for 5, 72-minute observation periods daily for the first 6-8 days postpartum.	Rat dams exhibit individual differences in licking/grooming behavior independent of litter size, or gender ratio of the litter. Maternal licking/grooming is transmitted to female offspring.	Does not address the mechanism or transmission of mothering behavior.
Chen, 2008[15]	Cohort of parents who attended seventh grade in Houston Independent School District in 1971	1560	Questionnaires administered to prospective parents at 3 developmental stages—early adolescence, young adulthood, and middle adulthood. Assessment of parenting style and depressive symptoms by questionnaire.	Constructive parenting during adolescence was positively related to marital satisfaction, educational attainment, and constructive parenting of offspring. Marital satisfaction and educational attainment mediated the intergenerational transmission of constructive parenting.	Based on questionnaires, measured constructive parenting rather than harsh parenting. Analysis restricted to parents in a marital relationship.
Cicchetti, 2010[16]	6- to 13-year-old children from low-income households, 60.6% African American, 21.4% white, 18% Other.	850	Maltreated history based on reports to authorities. Child depressive and suicidal symptoms were self-reported, 5-HTTLPR genotypes were determined by fragment analyses.	Genotypic variation of the serotonin transporter gene-linked promoter region (5-HTTLPR) moderates the effect of maltreatment on suicidal ideation in school-aged children	Only a single item was utilized to assess suicidal ideation. Might have missed unreported maltreatment cases. Only 1 generation assessed.
Conger, 2003[17]	3 generations of parents and offspring from the Family Transitions Project. Lower middle- or middle-class families.	75	G1 and G2 angry, aggressive parenting assessed by observation of family interactions and the puzzle task, respectively; G2 and G3 angry, aggressive behavior assessed by observation of family interactions and during the clean-up task, respectively.	G1 aggressive parenting correlated directly with G2 aggressive parenting 5-7 years later. G2 aggressive behavior as an adolescent and G3 aggressive behavior as a child were related to parenting behavior but not directly to one another.	Small sample size. Interaction tasks involved different procedures, different participants, and participants of different ages. Families of rural background and singular ethnicity.
Conger, 2013[18]	Generation R Study cohort in the Netherlands. 75% Western origin, 25% non-Western origin.	4438	Parents self-reported on psychological distress and harsh discipline when the child was 3 years. Children's internalizing and externalizing problems assessed by parental reports (CBCL/1.5-5 and BPI) and child interview at age 6.	Maternal maltreatment history was associated to offspring's externalizing problems, association was explained by maternal hostility and harsh discipline and, at least partially, also by paternal hostility and harsh discipline.	Single generation, short time span. Parenting behavior was self-reported.
Coplan, 1996[19]	Model organism (bonnet macaques)	30	Mothers were exposed to stressful early rearing conditions (unpredictability of food supply to the mother), and cerebrospinal fluid corticotropin-releasing factor (CRF) concentrations were measured in the offspring.	Infant monkeys raised by mothers foraging under unpredictable conditions exhibited persistently elevated cerebrospinal fluid concentrations of CRF.	Unknown if findings translate to humans. Mothering behavior not measured in offspring. Only 1 generation tested.

Table 1. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Cort, 2011[20]	10- to 12-year-olds and their mothers recruited through the Department of Human Services Registry to confirm maltreatment (maltreated), and the Temporary Assistance to Needy Families program (nonmaltreated).	104	Measures of maternal childhood maltreatment (Childhood Trauma Questionnaire [CTQ]); Maternal romantic attachment (Adult Attachment Scale); Maternal intimate partner violence victimization (Conflict Tactics Scales–Form N); Maternal psychological distress (Beck Depression Inventory–II); and Child multitype maltreatment (Maltreatment Classification System)	Mothers' childhood multitype maltreatment directly predicted their children's multitype maltreatment, instead of having indirect effects through maternal romantic attachment, intimate partner violence, and psychological distress.	Small sample size. Mothers of singular ethnicity, limited range of socioeconomic status (SES), and educational backgrounds (single African American mothers). Might have missed unreported maltreatment cases.
Dias, 2014[21]	Model organism (mice)	4-12/exp. condition	Fathers were fear conditioned to an odor, and sensitivity to the odor was measured in offspring. Epigenetic modification at the odor receptor gene were measured in G1 and G2 sperm.	Father's fear-response to odors was inherited through 2 generations. IVF, cross-fostering experiments were indicative of biological inheritance. Epigenetic signatures in sperm of fear conditioned males were changed for 2 generations, providing a possible mechanism for biological inheritance.	Unknown if findings translate to humans. Does not address maltreatment, or parenting behavior.
Dixon, 2005[22]	English cohort with children born in 1995-1998, >95% white.	4351	All information collected by community nurses, trained by expert psychologists, during home visits.	Within 13 months after birth, 6.7% of parents with history of abuse were referred for maltreating their own child. Main mediating factors were parenting under 21 years, history of mental illness or depression, and residing with a violent adult.	Small sample of parents with history of abuse (135; 3.1%).
Dixon, 2009[23]	English cohort with children born in 1995-1998, >95% white.	4351	All information collected by community nurses, trained by expert psychologists, during home visits.	6.7% of parents with history of abused their own children (intergenerational transmission); 93.4% of parents with history of abuse did not abuse their own children (cycle breakers); 0.4% of parents without history of abuse went on to abuse their own children (initiators).	Child maltreatment only assessed within the first year of an infant's life, maternal history of abuse was self-reported. Small sample of parents with history of abuse (135; 3.1%).
Drake, 2009[24]	Subsample of the Survey of Child and Adolescent Well-Being (NSCAW).	1,820	Assessment of recidivism in the form of any rereport, substantiated rereport, and foster care placement, in families investigated for child maltreatment.	There was no difference in recidivism between substantiated and unsubstantiated cases of child maltreatment.	Rereports were underestimated, and records were incomplete for some children. No records on services that might have mitigated differences between groups
Eckenrode, 2014[25]	3142 US counties	3142	Counts of substantiated reports of child abuse and neglect (National Child Abuse and Neglect Data System), and assessment of County-level data on income inequality (Gini coefficient) and children in poverty.	Higher income inequality and child poverty rate across US counties was significantly associated with higher county-level rates of child maltreatment.	Only substantiated reports of maltreatment included in the analysis. Analysis did not differentiate specific subtypes of maltreatment.

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Table I. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
English, 2015[26]	Subset of maltreated children from the LONGSCAN longitudinal study cohort.	203	Child maltreatment data collected from Child Protective Service's case records. Child outcomes assessed with in person interviews with mothers and children.	Effects of maltreatment differed depending on maltreatment type, severity, chronicity, and age at first report.	Study design would have missed unreported maltreatment cases. Only a small subsample of the LONGSCAN cohort was used.
Finzi-Dottan, 2014[27]	Jewish and Arab parents recruited from families registered and treated at the Jaffa Welfare Department in Tel-Aviv. Diverse SES and education levels.	213	Parents completed 6 questionnaires assessing child abuse potential, childhood history of abuse/neglect, attachment, emotional control, stress, and cognitive appraisal of parenthood.	Parents who experienced childhood abuse and neglect scored significantly higher in child abuse potential. The increased child abuse potential was mediated emotional control deficits.	Measured potential for child abuse, rather than actual child abuse.
Folger, 2013[28]	Undergraduate volunteers enrolled in psychology courses at a Midwestern university. 91.9% Caucasian.	344	Measures of specific types of maltreatment experiences (Lifetime Experiences Questionnaire [LEQ]), traumatic impact (TSC-40), dating abuse (Index of Dating Abuse [IDA]), aggression, and perceived social support (PSS-Fa and PSS-Fr).	Social support had a strong positive impact on later affective consequences (depression/anxiety and anger/hostility) in general but acted as buffer against negative outcomes for people with lower, but not higher, levels of childhood abuse.	Ethnically and socioeconomically homogeneous sample. Self-reported experiences may not have been as severe as clinical populations or other community populations (students were functioning adequately in college).
Gibb, 2006[29]	Participants recruited from a general psychiatric inpatient unit at Butler Hospital in Providence, Rhode Island, between May and August 2003.	30	Maltreated history assessed with CTQ. Patient history of suicide attempts assessed in clinical interview. 5-HTTLPR genotypes were determined by fragment analyses.	5-HTTLPR genotype moderated the link between childhood physical and sexual, but not emotional, abuse and adult's histories of suicide attempts.	Small sample size. Childhood abuse was self-reported (long time gap). No distinction between types of abuse, or number of suicide attempts.
Hammen, 2015[30]	Subset of 22- to 25-year-olds from the Mater University Study of Pregnancy (MUSP) birth cohort study. 92.5% Caucasian, largely working/lower middle class.	385	Youth Borderline Personality Disorder (BPD) symptomatology was assessed in clinical interview (SCID-II). Youth depressive symptoms were self-reported (BDI-II). Oxytocin Receptor allele assessed by PCR.	Oxytocin receptor genotype significantly moderated the link between early family quality and later BPD. Individuals showed differential susceptibility to developing BPD depending on genotype.	Unknown if the risk genotype and development of BPD link to subjects own parenting behavior.
Hans, 2000[31]	African American families from a 10-year longitudinal study Cohort in Chicago. Low to very low SES	69	Maternal substance abused assessed by questionnaire and urine test. Maternal psychopathology assessed by interview (DSM-III, SADS-L, and CAPPS). Parenting behavior assessed at 4, 12, and 24 months of age with laboratory mother–child activities. Child's perception of parent assessed by interview (PBI).	Maternal drug dependence was related to whether mothers were able to remain primary caregivers. Maternal psychopathology was related to unresponsive and negative parenting behavior, and to children's feelings of rejection.	Small sample size. Incidence of depression might have been underestimated due to the dichotomous measure of depression used. Maternal diagnoses were made only once, during pregnancy. Child's behavior was not assessed.
Heim, 2000[32]	Healthy women recruited from May 1997 to July 1999 at the General Clinical Research Center of Emory University Hospital, Atlanta, Georgia.	49	Major depression symptoms diagnosed by physician (DSM-IV). Childhood abuse was assessed with the Early Trauma Inventory (ETI). Adrenocorticotropic hormone (ACTH) and cortisol levels and heart rate responses were measured in response to standardized psychosocial laboratory stressors.	Women with a history of childhood abuse exhibited increased pituitary–adrenal and autonomic responses to stress compared with controls. This effect was particularly robust in women with current symptoms of depression and anxiety.	Small sample size. Study suggests that increased stress response is a persistent consequence of childhood abuse that may contribute adulthood psychopathology, but study does not directly relate to parenting behavior.

Table 1. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Jeon, 2016[33]	Families from the Family Transitions Project (FTP) with 3 generations of participants. Caucasian and lower middle- or middle-class from rural Iowa.	559	Longitudinal data collection on economic hardship, parental positivity, positive parenting, and G3 child positive behavior from G2 adolescence to G3 childhood (3- to 5-years-old). Assessments based on home interviews and interaction tasks.	In both G1 and G2, economic hardship negatively influenced parental positivity and positive parenting. G1 positivity and positive parenting was related to G2 positive parenting. G2 positive parenting was associated with G3 positive behavior to G2.	Low-risk study cohort (Caucasian, middle class). Protective effects of positivity might not hold up in high-risk cohorts.
Jokela, 2007a[34]	Young adults from the population-based "Cardiovascular Risk in Young Finns" study.	1224	Urban/rural residency was determined by subjective report and population density of the residential area. Depressive symptoms were measured in 2 test settings 4 years apart. HTR2A gene allele assessed by PCR.	The HTR2A gene allele was associated to the development of depression depending on rural or urban living environment. Carriers of one allele were more likely to develop depression in a rural environment, whereas carriers of the alternative allele were more likely to develop depression in an urban environment.	Study links a gene–environment interaction between HTR2A allele and living environment to development of depressive symptoms, but it does not address if this interaction influences parenting behavior.
Jokela, 2007b[35]	Young adults from the population-based "Cardiovascular Risk in Young Finns" study.	341	Social support was assessed on the Perceived Social Support Scale–Revised (PSSS-R) and depressive symptoms on a modified version of the Beck's Depression Inventory (BDI). HTR2A gene allele assessed by PCR.	Low social support predicted depressive symptoms more strongly in individuals carrying A alleles of the TPH1 than in others.	Study links a gene–environment interaction between TPH1 and social environment depressive symptoms, but does not address if this interaction influences parenting behavior.
Kelley, 1992[36]	Mothers with 1- to 33-month-olds recruited from an urban teaching hospital for children in the Northeast United States. 83.3% African American, low SES.	48	The Parenting Stress Index (PSI) was used to assess parenting stress. Background and drug use data were obtained by interview with a healthcare professional. Child maltreatment data were obtained from child protective services (CPS).	A strong association was found between maternal use of drugs and child maltreatment serious enough to necessitate removal of the children by CPS. Maternal drug use was associated with higher maternal and child-related stress.	Small nonrandom sample. Biased toward mothers that sought preventive health care for their children. Missing data on abuse for children in foster care at the time of the study.
Kirke-Smith, 2014[7]	11- to 18-year-olds recruited from specialist schools for youth with emotional and behavioral difficulties (maltreated group) and mainstream secondary schools (not maltreated)	80	Executive functioning was assessed by testing executive loaded working memory, verbal and nonverbal fluency, and verbal and nonverbal switching. Anxiety and depression were assessed with the Beck Youth Inventories for Anxiety and Depression (BYI-A and BYI-D).	Maltreated adolescents had significantly lower performance than nonmaltreated adolescents on executive functioning tasks. 50%-75% of the maltreated sample demonstrated weaknesses in executive functioning.	Maltreated and control groups differed in diagnostic status and education. Unknown history of abuse in the control group. Effects might be overestimated because the maltreated group consisted only of individuals who developed behavioral disorders.
Klengel, 2013[37]	Primary cohort of African American, highly traumatized, urban population of low SES.	1963	PTSD symptomatology was assessed by mPSS and CAPS. BDI was used to assess depressed mood. The CTQ was used as a continuous measure of childhood abuse and neglect. Molecular measures included FKBP5 genotyping, epigenetic profiling, expression analysis, and in vitro functional experiments.	The interaction between childhood trauma and FKBP5 genotype affects the risk of developing stress-related psychiatric disorders in adulthood. The mechanism underlying this interaction is FKBP5 allele-specific epigenetic modification in response to childhood trauma.	Study provides a molecular mechanism for differential susceptibility to childhood trauma, but does not address intergenerational transmission or parenting behavior specifically.

Table 1. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Levine, 2012[38]	Model organism (mice)	4-10/exp. condition	Mice of 2 strains were subjected to infant maternal separation and tested for fearful behavior in adulthood. Expression of epigenetically modified proteins was tested by Western blotting. Drugs were administered in drinking water.	Mice that differed in their resilience to infant maternal separation showed different epigenetic signatures in response to maternal separation in the adult brain. Experiments suggested that the changes in response to early-life stress (ELS) were of adaptive nature.	Unknown if findings translate to humans. Mothering behavior not measured in offspring. Only I generation tested.
MacKenzie, 2011[39]	Subset of mothers from the Stress, Social Support and Abuse & Neglect in High Risk Infants Study (SSS). 80% of infants recruited had at least I risk factor that qualified them for North Carolina's High Priority Infant Program (HPIP).	242	The State Central Registry of Maltreatment was then reviewed over each child's first 4 years of life to assess for early maltreatment. Following the neonatal interviews, mothers completed the CBCL when their children were 4, 6, 8, 10, and 12 years old.	Early maltreatment appeared to be important to early child functioning, but the cumulative level of risk predicted long-term clinical behavioral difficulty more strongly. High-risk children who were not reported for maltreatment by age 4 had greater behavioral problem trajectories than low-risk children with a maltreatment report.	Incidence of maltreatment might have been underestimated, as only reported cases of maltreatment were included in the analysis. Measures of parenting behavior were not included in the study.
Maguire-Jack, 2016[40]	Participants recruited in 6 Women, Infants, and Children (WIC) clinics in Franklin County, Ohio.	1053	Service use and availability was self-reported by parents. Child maltreatment was estimated using the child neglect and physical child abuse subscales of the Conflict Tactics Scale (CTS-PC). Parent stress was estimated using the Parent Stress Index (PSI-SF).	Social service availability had a potential protective effect against physical abuse and neglect and decreased the relationship between parent stress and abuse and neglect. Service receipt was positively correlated with maltreatment and increased the relationship between parenting stress and neglect.	Results hard to interpret as service availability and service receipt had opposite effects.
McGowan, 2009[41]	Postmortem brain tissue from 12 suicide victims with a history of childhood abuse, 12 suicide victims with a negative history of childhood abuse (matched for psychiatric diagnoses), and 12 controls.	36	Psychiatric diagnoses and history of childhood abuse or neglect were obtained using Structured Clinical Interviews for DSM-III-R I and Childhood Experience of Care and Abuse questionnaire adapted for psychological autopsies. The NR3C1 gene was tested for genotype, expression, and epigenetic modification.	Suicide victims with a history of childhood abuse had altered epigenetic profiles and decreased expression of the glucocorticoid receptor (GR; NR3C1), an HPA axis regulator. This study translated previous results from rat to humans, suggesting a common effect of parental care on epigenetic regulation of the HPA.	Small sample size. Study provides a molecular mechanism for the effects of childhood abuse and neglect but does not address intergenerational transmission or parenting behavior specifically.
Murgatroyd, 2009[42]	Model organism (mice)	8-16/exp. condition	Pups were subjected to maternal separation stress, and stress response behavior was tested in adulthood. Molecular measures included hormone assays, epigenetic profiling, expression analysis, and in vitro functional experiments.	ELS in mice caused enduring increase of corticosterone and alterations in passive stress coping and memory. This phenotype was related to epigenetic modifications and expression the HPA regulator arginine vasopressin (AVP).	Unknown if findings translate to humans. Mothering behavior not measured in offspring. Only I generation tested.

(continued)

Table 1. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Najman, 2004[43]	MUSP prospective prebirth cohort	4600	Child's SES was measured using maternal age, family income, and marital status and the grandfathers' occupational status. Measures of child's cognitive development (PPVT-R) and emotional health (CBCL) obtained at 5 and 14 years of age.	Family income was related to all measures of child cognitive development, emotional health and, and smoking, independently of all other SES indicators. Grandfathers' occupational status was independently related to child cognitive development.	Changes to the child's circumstances during the duration of the study were not considered. Study does not address parenting behavior.
Neppl, 2009[44]	Families from the FTP with 3 generations of participants. Caucasian and lower middle- or middle-class from rural Iowa.	187	Longitudinal data collection on G1 and G2 harsh and positive parenting, G2 adolescent and adult externalizing behavior and academic achievement, and G3 child harsh and positive behavior. Assessments based on self-reports and observation of interaction tasks.	G1 harsh or positive parenting was directly associated to G2 harsh or positive parenting. G2 externalizing behavior mediated the relationship between G1 and G2 harsh parenting, while G2 academic attainment mediated the relationship between G1 and G2 positive parenting.	The direct effect of G1 parenting on G2 parenting was modest. Low-risk study cohort (Caucasian, middle class). Parenting was measured at different developmental periods for the G1 and G2 parents.
Nieratschker, 2014[45]	Cross-species approach involving human cord blood (cohort from the Rhine-Neckar Region of Germany), blood from newborn and adolescent nonhuman primates (Macaca mulatta), and adult rat brain tissue.	180 human/ 14-30 monkeys/ 7-9 rats	Structured interview and questionnaires were used for ELS assessment in the human sample. Monkeys and rats were exposed to ELS paradigms. DNA was extracted from all blood and brain samples and tested for genome-wide promoter DNA methylation.	ELS was related to epigenetic changes at the MORC1 gene in immune and stem cells derived from the blood of human and monkey neonates, as well as immune cells derived from the blood of adolescent monkeys and in the prefrontal cortex of adult rats. The MORC1 was associated with major depressive disorder (GWAS data from a previous study).	Study provides the first identified epigenetic marker of ELS to be present in blood cells at birth and in the brain in adulthood, but the function of the MORC1 gene is unknown. Study does not address parenting behavior directly.
Nikulina, 2013[6]	Prospective Midwestern metropolitan cohort of court-substantiated cases of childhood abuse and neglect (ages 0–11) and matched controls. 59% white, 34% black, 4% Hispanic, low SES.	792	Executive functioning was assessed with the Trail Making B test and nonverbal reasoning with Matrix Reasoning. PTSD (DSM-III-R lifetime diagnosis) was assessed at age 29.	Childhood maltreatment overall and childhood neglect predicted poorer executive functioning and nonverbal reasoning at age 41, whereas physical and sexual abuse did not. History of PTSD did not mediate or moderate these relations.	Findings might not be generalizable to unreported or unsubstantiated cases of child abuse and neglect or to higher SES groups.
Oberlander, 2008[46]	Cohort of depressed and nondepressed mothers, recruited in their early second trimester as part of a study of the impact of prenatal psychotropic medication exposure on neonatal health.	82	Epigenetic modifications at the NR3C1 gene were assessed in infant cord blood cells of depressed mothers (treated or not treated with antidepressants) and nondepressed mothers. HPA function (salivary cortisol) was assessed at 3 months in response to a stressor.	Prenatal exposure to increased third-trimester maternal depressed/anxious mood was associated with epigenetic modification of the NR3C1 gene and increased salivary cortisol stress responses at 3 months.	Study only measured infant stress response at 3 months and does not address if early changes relate to parenting behavior later in life.

(continued)

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Table 1. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Ost, 2014[47]	Model organism (*Drosophila*)	15–40/exp. condition	Diet-induced epigenetic changes and gene expression were measured in G1 males, G1 sperm, and offspring. Data from human and mice microarrays were mined to test for conserved patterns.	Paternal diet-induced epigenetic changes are passed on to offspring through the germline. This molecular pathway might be conserved in mice and humans.	Presents a mechanism for the inheritance of experience-based epigenetic marks but does not address parenting behavior.
Pereira, 2012[48]	Mothers recruited from urban and suburban areas. Low-risk sample, upper-middle and upper class, large range of ethnicities (67.2% Caucasian).	291	Maternal history of maltreatment and parenting stress were assessed via self-report inventory (CTQ and PSI-SF). Maternal sensitivity toward the infant was assessed with 2 hours of direct behavioral observation (MBQS).	Mothers who reported more maltreatment in childhood and mothers who reported more current parenting stress were less sensitive with their infants. The relationship of maternal maltreatment history and parental behavior was mediated by parenting stress.	Small effect (model explained only 3.3% of the variance in maternal sensitivity), but potential moderators were not assessed, and only a single aspect of parenting was assessed.
Plant, 2013[49]	Subsample of pregnant mothers from a prospective longitudinal South London Child Development Study. Mostly working class and 72% white British.	125	Maternal history of maltreatment and antisocial behavior was self-reported, maternal depression was diagnosed using the Clinical Interview Schedule. Offspring childhood maltreatment, adolescent antisocial behavior, and depression were obtained from parents and offspring through clinical interview.	History of maltreatment was associated with greater levels of maternal depression, and the combination of maternal childhood maltreatment and depression was associated with significantly greater levels of offspring maltreatment and antisocial behavior.	Maternal depression was retrospectively self-reported. Small low-risk sample with few mothers and offspring with childhood maltreatment and psychopathology.
Plotsky, 2005[50]	Model organism (rats)	5–10/exp. condition	CRF, an HPA axis regulator, was measured in the brain and cerebrospinal fluid of adult male rats that were reared with several levels of maternal separation.	Male rats that were separated from their mothers for 180 minutes daily have significant higher HPA axis reactivity, measured as higher stress hormones in the cerebrospinal (CORT and ACTH) fluid, and higher transcription of CRF in the brain.	Results are hard to interpret because handling the pups without maternal separation increased stress reactivity, as did 180 min of separation/d, but 15 min separation/d had no effect.
Renner, 2006[51]	Participants of the Illinois Families Study (IFS), families receiving Temporary Assistance for Needy Families benefits in the United States. 81% African American.	1005	Participants experiences with intimate partner violence and child maltreatment in childhood and adulthood were assessed by interview, retrospectively.	Moderate association between child maltreatment and adult victimization. Weak association between experiencing and perpetrating child maltreatment.	All measures of experiencing and perpetrating abuse were retrospectively self-reported. Weak associations might be due to insufficient or inaccurate data.
Romens, 2014[52]	Participants recruited through the Dane County (Wisconsin) Department of Human Services (substantiated cases of child maltreatment), and flyers (nonmaltreated). 66% white, 30% black.	56	Child maltreatment assessed by accessing CPS records of all families (38 participants had no records and 18 participants had reports of physical maltreatment). Epigenetic changes in the *GR* gene, an HPA regulator, were examined in whole blood.	Children exposed to physical maltreatment had greater methylation in the NR3C1 promoter region of the GR gene compared to nonmaltreated children.	Very small (and uneven between groups) sample size. Only substantiated reports of maltreatment included in the analysis. Analysis did not differentiate specific subtypes of maltreatment.

Table 1. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Roth, 2009[53]	Model organism (rats)	4-9/exp. condition	Infant rats were exposed to stressed caretakers that predominately displayed abusive behaviors. Epigenetic patterns and gene expression of the *BDNF* gene in the brain were assessed throughout the life span in the next generation of infants.	Early maltreatment produced persisting epigenetic changes in the *BDNF* gene that caused altered *BDNF* gene expression in the adult brain. Offspring of previously maltreated females also showed the same epigenetic changes, and cross-fostering did not rescue the effect.	Unknown if findings translate to humans. Mothering behavior not measured in offspring.
Roy, 2007[54]	Clinical population of substance-dependent patients at high risk of suicide, childhood trauma, and other stress. All African American.	438	Suicide attempt history was assessed by interview and childhood trauma was assessed with the 34-item CTQ. 5-HTTLPR genotype was assessed by fragment analyses.	Patients with low expression 5-HTTLPR genotypes and high CTQ scores were more likely to attempt suicide. Higher CTQ scores correlated with increasing risk of suicide attempt and this relationship was stronger in the low expression 5-HTTLPR genotype.	Study links a gene–environment interaction between 5-HTTLPR allele and childhood abuse to suicide risk, but it does not address if this interaction influences parenting behavior.
Scaramella, 2008[55]	Families from the FTP with 3 generations of participants. Caucasian and lower middle- or middle-class from rural Iowa.	154	G1 poverty status was assessed with detailed reports of economic circumstances. G2 harsh parenting assessed by observation of family interactions during the puzzle task; G3 externalizing behavior assessed in interview with the CBCL.	Adolescent poverty predicted earlier age of parenthood in the second generation (G2). Younger G2 parents were harsher with their own 2-year-old child (G3) and harsh parenting predicted increased G3 externalizing problems from age 2 to age 3.	Selective nature of the sample may limit the generalizability of the findings. Low-risk study cohort.
Sen, 2016[56]	Mothers recruited from Health Fairs in 3 Detroit communities with high prevalence (8%–11%) of high blood lead levels (Rosa Parks, Chene, and Kettering-Butzel).	35	Maternal and neonatal lead blood levels were measured. Genome-wide epigenetic profiling (DNA methylation) was performed on neonatal and maternal dried blood spots. Effects of led exposure on germ cells was assayed in vitro (cell culture).	Infants of mothers with high neonatal blood lead levels had significant changes in their neonatal and current epigenetic profile. Germ cells exposed to lead had the same epigenetic changes.	Study presents a possible mechanism for the inheritance of environmentally induced epigenetic marks but does not address childhood abuse or neglect or parenting behavior.
Smith, 2014[57]	Low-income mothers of 6- to 13-year-olds, recruited from clinics at the Grady Health Systems in Atlanta, Georgia. 96% African American.	83	Questionnaires were used to assess mother's childhood trauma (CTQ), traumatic events (TEI), emotional dysregulation (EDS), negative affect (PANAS), and child abuse potential (CAPI).	Mother's childhood abuse predicted later risk for abusive parenting. Maternal negative affect and EDSs mediated the relationship between childhood maltreatment and the risk of perpetuating child abuse.	All variables were self-reported. Small, homogeneous sample. Could benefit from better measures of parenting behavior. Only looked at a single generation.
Steele, 2016[58]	Mothers recruited from a Group Attachment Based Intervention for the prevention of child maltreatment (low SES, n = 33), and from the community (high SES, n = 85). Mixed ethnicities.	118	Questionnaires were used to assess mother's history of abuse and neglect (ACE, CTS, CTQ), household dysfunction (CTS), and parenting stress (PSI-SF).	Adverse childhood experiences and parenting stress were significantly higher in mothers with low SES. Controlling for SES and mother's adverse childhood experiences increased the probability of parenting stress.	All variables were self-reported. While high SES mothers were mostly white, low SES mothers were mostly African American and Hispanic. Different sample sizes for low and high SES groups.

(continued)

Table 1. (continued)

First Author, Year	Population (Cohort)	Sample Size	Methods	Results	Limitations
Thornberry, 2013[59]	Participants of the Rochester Youth Development Study (RYDS), targeted toward youth at high risk for serious delinquency and drug use.	816	History of child maltreatment (from birth to age 17) and later maltreatment perpetration was assessed using records of substantiated from CPS.	Maltreatment in adolescence (age 12-17) or from childhood through adolescence increased odds of perpetuating maltreatment, but childhood maltreatment (age 0-12) did not. Adolescent maltreatment was often more serious, which could be a cause of maltreatment perpetration.	Potentially underestimated abuse. Potentially influenced by other risk factors.
Uchida, 2010[60]	Model organism (rats)	6-9/exp. Condition	Infant rats were exposed to maternal separation and stress response was assessed in adulthood. Molecular measures included hormone assays, gene expression analysis, in vitro functional experiments, and in vivo gene expression manipulations.	Maternally separated rats had increased HPA stress responses and depression-like behaviors. Increased stress levels in maternally separated rats correlated with increased expression of the HPA axis regulator REST and related genes in the brain.	Unknown if findings translate to humans. Only I generation tested. Does not address parenting behavior directly.
Yehuda, 2015[61]	Holocaust survivors and their adult offspring, and demographically matched controls.	8-32	Psychiatric diagnoses (DSM-V), childhood abuse (CTQ), and PTSD symptoms were determined using Structured Clinical Interviews. Methylation differences in blood cells were evaluated for the HPA axis regulator FKBP5.	Holocaust survivors and their children had epigenetic modifications at the FKBP5 gene. Methylation was higher in the holocaust survivors but lower in their parents. Childhood abuse was associated to different epigenetic modifications at the same gene.	Small sample size. Results are hard to interpret because although both parents and offspring had epigenetic changes, they went in opposite directions.
Weaver, 2004[62]	Model organism (rats)	4-5/exp. Condition	Rat pups from dams with high or low pup licking and grooming (LG) and arched-back nursing (ABN) were tested for epigenetic marks (DNA methylation, histone acetylation), GR gene expression, and hormonal response to stress.	Pups that experienced low or high maternal care differed in several epigenetic marks at the GR gene promoter, and in the HPA axis response to stress. Group differences could be removed by cross-fostering and pharmacological intervention.	Unknown if findings translate to humans. Only I generation tested.

Abbreviations: BPI, Behavior Problems Index; CIDI-SF, Composite International Diagnostic Interview–Short Form; DSM-IV, *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition; HPA, hypothalamic–pituitary–adrenal; IVF, in vitro fertilization; NICHD, National Institute of Child Health and Human Development; PC-CTS, Parent Child Conflict Tactics Scale; PCR, polymerase chain reaction; PTSD, post-traumatic stress; TSCYC, Trauma Symptom Checklist for Young Children.

very difficult to assess the variability of parenting behavior because it is dependent on so many factors.[100]

Furthermore, from a biological perspective, it is extremely challenging to study biological changes in the human brain because brain tissue is rarely available. Biological changes in blood or saliva might not reflect changes in the brain, since these tissues are composed by completely different cell types. It is also hard to differentiate biological changes that are intergenerationally inherited from biological changes that are acquired anew every generation due to parenting behavior or other external factors. Furthermore, to rule out the effects of direct exposure to the stressful experience (of the parent, embryo or reproductive cells), studies must follow progeny for at least 3 to 4 generations, to study maternal or paternal inheritance, respectively. Lastly, due to the ethics associated with human research, experimental evidence relies on model organisms and it is difficult to know how well these findings translate to humans.

Discussion

This article presents an examination of the literature on the intergenerational transmission of maltreatment, focusing specifically on (1) understanding whether it is an important factor to consider when determining risk of future maltreatment and (2) understanding the social and biological mechanisms that might explain continuity and discontinuity of abusive and neglectful parenting.

Although the social and biological literature reviewed here seems to lend strong support for a history of child abuse predicting abusive or neglectful parenting practices, there is a large amount of individual variation in this relationship. Maltreatment experiences are diverse in nature, as are the children who endure abuse and neglect. Types of maltreatment can cooccur concurrently or consecutively,[26] and experiences vary in terms of age at onset, the number of development periods in which the incidents occur, the impact on developmental milestones,[1] and the severity of the incidents.[101] Given the vast variability in child maltreatment experiences, dichotomous measures reflecting the presence or absence of maltreatment in childhood are insufficient.[8] Children also have unique biological compositions that influence the way they interact with the world, while the environment influences their biology.[88] Biological consequences of abuse and neglect are dependent on developmental timing[42] and genetic predispositions.[9,30,34,35] From an ecological perspective, human development occurs through this complex process of a biopsychological human interacting with the environment.[78,102] The literature clearly demonstrates that the developmental processes that ultimately influence parenting style in adulthood are complex; single experiences or characteristics cannot explain harmful parenting practices in isolation. Recognizing the diversity of individual experiences will enrich efforts to respond to the unique needs and deficits of children who are maltreated and their caregivers. It is imperative for research to move forward in a way that examines and acknowledges these variations in order to determine which specific dimensions of maltreatment and what specific individual and contextual factors increase the propensity for harmful parenting toward the next generation.

If cycles of maltreatment and abuse have a biological underpinning, the regulation of stress response systems might constitute a plausible mechanism. Experiencing maltreatment in childhood is stressful, and it appears to have short- and long-term effects on how individuals respond to future stressful situations. It is possible for abuse and neglect to diminish the biological capacity of an individual to cope with daily stressors, such as school difficulties in childhood, romantic relationship formation in adolescence, and parenting in adulthood. Parents who cannot cope with the daily stressors involved in childrearing may find it challenging to manage feelings of frustration or anger and meet the basic physical and emotional needs of their developing children.

It is important to acknowledge that while we know maltreatment can change children's biology, we also know that the effects of abuse and neglect on stress responses are reversible in the context of nurturing environments. Intervening early to prevent recurrent maltreatment and promote nurturing caregiving may help children to cope with stress in adaptive ways that will continue into adulthood. It is also critical that the consequences of maltreatment be addressed, as these consequences may mediate the relationship between a history of abuse and neglect and later perpetration. Maltreated children and youth need help early in life and as they grow older and transition to adulthood, in order to succeed academically, secure employment, delay childbearing, and enter supportive romantic relationships and friendships. These are all protective factors that may help prevent the continuation of harmful and maltreating parenting practices.[15,22,40]

For future studies on intergenerational maltreatment, it will be important to use complex measures of abuse and neglect and examine maltreatment prospectively over several generations. Incorporating biological information into these studies would complement the analysis of what factors are involved in transmission of maltreatment, and perhaps help explain some of the large variance in findings.

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