Child Maltreatment and Risk for Psychopathology in Childhood and Adulthood

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Abstract

Although rates of child maltreatment are declining, more than 600,000 children in the United States are substantiated victims of abuse or neglect. The focus of this review is on the relationship between maltreatment and mental health problems in childhood and adulthood. Children and adults who are exposed to abuse or neglect in childhood are at risk for a range of poor mental health outcomes, including internalizing and externalizing psychopathology, posttraumatic stress disorder, psychotic symptoms, and personality disorders. I review three potential mechanisms by which maltreatment may increase risk for various forms of psychopathology, (*a*) hypervigilance to threat, (*b*) deficits in emotion recognition and understanding, and (*c*) low responsivity to reward. I also review genetic and psychosocial factors that moderate the relationship between maltreatment and risk for psychopathology. Finally, I discuss methodological limitations of the literature on maltreatment, with an emphasis on the challenges associated with establishing a causal role for maltreatment (and moderators or mediators of maltreatment) in the development of mental health problems and the reliance of many studies on retrospective self-reports.

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EPIDEMIOLOGY OF ABUSE AND NEGLECT

In 2014, the most recent year for which figures are available, approximately 3.9 million children were investigated for allegations of abuse or neglect. Twenty percent of these investigations were substantiated (or indicated), representing 702,000 children nationally, or 9.4 per 1,000 children in the population (US Dep. Health Hum. Serv. 2016). The focus of this review is the relationship between childhood maltreatment and mental health in childhood and adulthood and the biological and psychosocial factors that mediate and moderate this association.

Maltreatment comprises neglect and emotional, sexual, and physical abuse. In 2014, 75% of substantiated victims of maltreatment experienced neglect, defined as the failure to meet children's basic physical needs with respect to clothing, hygiene, food, and safety. Physical abuse is defined as harm by a caregiver or someone who has responsibility for the child resulting in nonaccidental physical injury (from minor bruises to severe fractures or death) (Leeb et al. 2008). In 2013, 17% of substantiated victims experienced physical abuse. Eight percent of children were victims of sexual abuse, and 6% were victims of psychological maltreatment, the core feature of which is a pattern of behavior that impairs a child's emotional development or sense of self-worth (Leeb et al. 2008). Finally, 6.8% of children were victims of other forms of abuse which included, for example, "threatened abuse" or a parent's drug or alcohol abuse (US Dep. Health Hum. Serv. 2016).

According to 2014 statistics, children under the age of three were victimized at higher rates than older children, and children under the age of one year were victimized at the highest rates (24.4 per 1,000 children) (US Dep. Health Hum. Serv. 2016). Boys and girls were equally likely to be victims of abuse or neglect, but 15.3 per 1,000 African-American children were victimized as opposed to 8.4 per 1,000 white or 8.8 per 1,000 Hispanic children (US Dep. Health Hum. Serv. 2016). Other sociodemographic predictors of maltreatment include family poverty, young motherhood (Mersky et al. 2009, Sedlak et al. 2010, Thornberry et al. 2014), parental history of antisocial behavior (Jaffee et al. 2004, Thornberry et al. 2014), and a perpetrator's history of maltreatment (Schofield et al. 2013, Widom et al. 2015).

MEASURING MALTREATMENT

In studies of adults, childhood maltreatment is typically measured by retrospective self-report. Retrospective reports have been shown to be biased by current mental state (Colman et al. 2016) and subject to omission (Widom & Morris 1997). For example, when adults were asked during two separate interviews whether they had been maltreated as children, 51% of those with documented records of sexual abuse did not report their abuse at one or both interviews (Widom & Czaja 2012). In contrast, 20% of those without documented records of sexual abuse reported at one or both interviews that they had been sexually abused (Widom & Czaja 2012), which could mean that either their sexual abuse was not detected by authorities when these individuals were children or their reports of sexual abuse were fabricated. Retrospective self-reports of physical abuse were also inconsistent with documented records (Widom & Czaja 2012). Moreover, experiences in adulthood also biased retrospective reports. For example, 75% of adults who had a lifetime diagnosis of drug abuse reported that they had been victimized by abuse or neglect in childhood (Widom et al. 1999). Viewed prospectively, however, only 35% of those with documented cases of abuse or neglect were subsequently diagnosed with drug abuse (Widom et al. 1999) and rates of drug abuse in adulthood were equally high for those without a history of childhood abuse, indicating that a childhood history of maltreatment is actually a poor predictor of who will abuse drugs as an adult (Widom et al. 1999). Given the problems associated with retrospective self-report, we highlight prospective, longitudinal studies in which maltreatment was measured in childhood from Child Protective Services records or other informant reports and participants were followed into adulthood. However, in the section titled Mediators of Maltreatment Effects, the majority of studies described involve retrospective self-reports.

MALTREATMENT AND RISK FOR PSYCHOPATHOLOGY

This section describes studies that have tested whether maltreated children and adults who were maltreated in childhood are at elevated risk for psychopathology. The review is focused on the most methodologically rigorous studies that include the following features: (*a*) a prospective research design in which maltreatment predated the onset of psychopathology; (*b*) a demographically matched control sample or statistical adjustments for variables that could confound the association between maltreatment and risk for psychopathology; and (*c*) psychometrically valid measures of psychopathology, including (but not limited to) diagnostic measures. In the majority of these studies, information about maltreatment came from Child Protective Services records, although in some studies maltreatment was reported by caregivers. Because official records are likely to underestimate the true prevalence of maltreatment, studies that combine official records with informant reports are likely to provide the most valid information (Cohen et al. 2001, Widom et al. 2015).

Maltreatment and Risk for Externalizing and Internalizing Psychopathology

Victims of maltreatment are at elevated risk for a range of externalizing problems in childhood and adolescence, including attention deficit/hyperactivity disorder (ADHD), conduct disorder (CD), oppositional defiant disorder (ODD; Cohen et al. 2001, Famularo et al. 1992), delinquency (Lansford et al. 2007, Stouthamer-Loeber et al. 2001, Widom 1989b,Williams et al. 2010), and antisocial behavior (Jaffee et al. 2004, Jonson-Reid et al. 2010, Lansford et al. 2002, Manly et al. 2001, Moylan et al. 2010). Some studies also identify elevated symptoms of substance use in maltreated versus nonmaltreated youth (Kaufman et al. 2007, Lansford et al. 2010, Rogosch et al. 2010), but others have not observed this pattern (Cohen et al. 2001).

Risk for externalizing problems extends into adulthood, when victims have significantly elevated rates of antisocial personality disorder (Johnson et al. 1999, Luntz & Widom 1994), self-reported crime (Thornberry et al. 2010), and criminal arrests (Maxfield & Widom 1996). Findings with respect to drug and alcohol use have been mixed, with some studies identifying elevated rates of drug and alcohol use among young adults with a history of maltreatment versus those without such a history (Cohen et al. 2001, Noll et al. 2009, Scott et al. 2010, Thornberry et al. 2010), and others finding that the relationship between these problems and child maltreatment is stronger in women than men and is detectable in middle age, but not in young adulthood (Widom et al. 1995, Widom et al. 2006).

Victims of maltreatment are also at risk for a range of internalizing problems in childhood, including major depressive disorder (Brown et al. 1999), anxiety disorders (Cohen et al. 2001), posttraumatic stress disorder (PTSD) and symptoms of trauma (Crusto et al. 2010, Famularo et al. 1992, Milot et al. 2010, Putnam et al. 1995), and internalizing symptoms (Bolger & Patterson 2001, Lansford et al. 2002, Manly et al. 2001, Moylan et al. 2010). Risk for internalizing disorders extends into adulthood. Victims have significantly elevated rates of major depressive disorder (Brown et al. 1999, Noll et al. 2009, Scott et al. 2010, Widom et al. 2007), depressive symptoms (Thornberry et al. 2010), and anxiety disorders (Cohen et al. 2001, Scott et al. 2010) compared to adults without a history of child maltreatment. In a meta-analysis of eight cohort studies from the United States, New Zealand, and Australia, all but one of which relied on official records to prospectively ascertain maltreatment, a documented history of childhood maltreatment was associated with an increased odds in adulthood of major depressive disorder [odds ratio (*OR*) = 2.03, 95% confidence interval = 1.37 to 3.01] and any anxiety disorder (*OR* = 2.70, 95% confidence interval = 2.10 to 3.47) (Li et al. 2016). The authors estimated that 59% of depression and anxiety cases worldwide are attributable to childhood maltreatment and that a 10% reduction in child maltreatment could potentially prevent 31.36 million cases of depression and anxiety (Li et al. 2016).

A childhood history of maltreatment is also associated with increased risk of PTSD (Breslau et al. 2014, Scott et al. 2010) and PTSD symptoms (Widom 1999). Moreover, childhood maltreatment and mental disorders measured in childhood are independently and prospectively predictive of the emergence of PTSD in adulthood in response to a trauma (Breslau et al. 2014). Thus, maltreatment increases risk for PTSD in adulthood via at least two pathways. First, maltreated youth are at heightened risk for mental disorders in childhood and adolescence, and a history of mental health problems increases the risk that an adult who experiences a trauma will develop PTSD. Second, independent of juvenile disorder, maltreatment sensitizes the neurobiological response to subsequent trauma, increasing the risk that an adult who experiences trauma will develop PTSD (Breslau et al. 2014).

Maltreatment and Risk for Personality Disorders, Psychotic Symptoms, and Suicide

As adults, victims of child maltreatment are at risk for borderline personality disorder (Johnson et al. 1999, Widom et al. 2009), with one study also showing risk for Cluster B (dramatic, emotional, erratic) and Cluster C (anxious, fearful) personality disorders more broadly (Johnson et al. 1999). In addition, victims of child maltreatment are at elevated risk for suicide in adolescence and adulthood (Brown et al. 1999, Thornberry et al. 2010) and engage in elevated rates of self-injury (Yates et al. 2008). Moreover, at least one study demonstrates that child victims of maltreatment experience elevated rates of psychotic symptoms in early adolescence compared with nonmaltreated youth (Arseneault et al. 2011). Results from a meta-analysis of 41 prospective cohort, population cross-sectional, and case-control studies indicated that individuals who experienced childhood

trauma (defined as sexual, emotional, or physical abuse; physical or emotional neglect; or parental death) were at elevated risk for psychotic disorder, schizoaffective disorder, or schizophrenia or for psychotic symptoms, with an *OR* of 2.78 (Varese et al. 2012). The magnitude of this effect did not vary as a function of study design, but the association between parental death and psychotic outcomes was smaller than the association between all other forms of trauma and psychotic outcomes (Varese et al. 2012).

CAUSAL STATUS OF EFFECTS

Being neglected or abused as a child could cause victims to develop mental disorders that emerge in childhood or adulthood. Alternatively, the risk for psychopathology associated with a childhood history of maltreatment could be accounted for by genetic or nongenetic factors associated with maltreatment. Unfortunately, the research designs that are best suited to choose between genetic and nongenetic explanations are particularly ill-suited to the study of maltreatment. In an adoption design, for example, researchers can eliminate the possibility that maltreatment is a marker for genetic risk for psychopathology that parents transmit to children. The parents and children in question are not biologically related, so any elevation in child psychopathology associated with maltreatment from an adoptive parent is likely caused by the maltreatment. With good reason, however, social workers screen out potential adoptive parents who are at high risk of perpetrating abuse or neglect, and rates of maltreatment in adoptive families are correspondingly low. In the cotwin control design, causal effects of maltreatment are inferred if rates of psychopathology are higher in the twin who has been maltreated than the twin who has not. Twins who grow up in the same family are exposed to many of the same risk factors for psychopathology (e.g., family poverty, a parent's mental illness) and are genetically similar (or identical, in the case of monozygotic twins), so any within-pair differences in risk for psychopathology are attributed to differences in the twins' experiences. It is rare, however, for one twin but not the other to have been maltreated (Jaffee et al. 2004), and very large, high-risk samples would be required to identify discordant cases.

Research designs that match maltreated children with sociodemographically similar, nonmaltreated youth may be, therefore, the most feasible way of estimating the unique effects of maltreatment on risk for psychopathology, and these designs do a good job of ruling out the possibility that nongenetic factors (e.g., family or neighborhood poverty) confound observed associations between maltreatment and child or adult psychopathology. For example, using propensity score matching methods, Thornberry et al. (2010) showed that maltreated youth had significantly more depressive symptoms and suicidal thoughts, substance use problems, and criminal behaviors in young adulthood compared with nonmaltreated youth who were matched on pre-existing individual and family characteristics. Although studies that have used sociodemographically matched controls have identified unique effects of maltreatment on risk for psychopathology, they have also demonstrated that socioeconomic disadvantage itself is associated with high rates of emotional and behavioral health problems, poor life course outcomes, and risk for maltreatment perpetration (Nikulina et al. 2011, Widom et al. 2015).

INTERGENERATIONAL TRANSMISSION OF MALTREATMENT

The cycle of violence hypothesis proposes that adults who experienced abuse or neglect in childhood are at elevated risk of maltreating their own children. This hypothesis has been difficult to evaluate adequately due to the preponderance of small samples and the lack of valid data on abuse and neglect across generations (Widom 1989a). In an effort to overcome these limitations, four different research teams conducted parallel analyses to determine whether parents' history of

maltreatment was predictive of children's exposure to maltreatment. Although studies differed with respect to whether parents' history of maltreatment was measured retrospectively or prospectively and whether maltreatment was self-reported or assessed via official records, results were consistent, with z-transformed r values ranging from 0.12 to 0.55 and a median effect size of $r = 0.31$ (Conger et al. 2013, Herrenkohl et al. 2013, Jaffee et al. 2013, Schofield et al. 2013, Thornberry et al. 2013).

The best-designed studies of the cycle of maltreatment are ones in which official records of maltreatment are available in both generations and are supplemented with self-reports of maltreatment. In the only such study in the literature, the odds of an individual being reported to Child Protective Services because their child was maltreated were twice as great for parents who had documented childhood histories of victimization versus nonmaltreated, sociodemographically matched controls (21% versus 12%) (Widom et al. 2015). On the basis of self-reports, however, rates of child maltreatment were the same for parents with and those without documented histories of childhood victimization. Indeed, the overrepresentation of parents with histories of childhood victimization in the child welfare system could reflect detection bias: Among parents who reported engaging in abuse or neglect, 31% of those who were themselves maltreated in childhood were investigated by Child Protective Services, whereas only 15% of those who did not have childhood histories of victimization were investigated by Child Protective Services (Widom et al. 2015).

Together, these findings suggest two things. First, the cycle of violence is not inevitable. The majority of adults who were victimized as children do not themselves perpetrate abuse or neglect. In some cases, this may be because—as adults—they are buffered by safe and nurturing relationships with partners and other adults (Schofield et al. 2013). For example, being in a nonabusive adult relationship and having a warm and supportive relationship with a partner distinguished women who broke the cycle of maltreatment (i.e., they had histories of childhood abuse but their children did not) from women who maintained the cycle of maltreatment (i.e., they and their children had histories of maltreatment) (Jaffee et al. 2013). Second, Child Protective Services has a bias to investigate and substantiate cases of abuse or neglect when they know the parent has a history of maltreatment. Thus, detection bias may inflate estimates of the intergenerational transmission of maltreatment.

MEDIATORS OF MALTREATMENT EFFECTS

Researchers have proposed multiple potential mechanisms by which maltreatment increases risk for psychopathology, from epigenetic processes and gene expression to neuroendocrine, immune, and neurotransmitter systems, to brain structure and function, and to social cognition. To present state-of-the-science findings, we highlight studies that attempt to link multiple levels of functioning. We focus on three phenomena that have been robustly associated with a history of maltreatment, (*a*) hypervigilance to threat, (*b*) deficits in emotion recognition, and (*c*) insensitivity to reward. We review the evidence for these potential mechanisms by which a history of maltreatment increases risk for psychopathology and—where relevant—the neural circuitry underlying these phenomena. The effects of maltreatment on specific biological systems has been reviewed elsewhere (Danese & McEwen 2012, Jaffee 2017).

Hypervigilance to Threat

Attention bias refers to the tendency to devote disproportionate attention to relatively mild threats. Attention bias is a robust correlate of anxiety in adults (Bar-Haim et al. 2007); however, there is relatively little research on the etiology of negative attention bias in adults or children. Pollak

(2003) proposed that children could become sensitized to threatening stimuli because of their early exposure to anger and other negative emotions in families characterized by nonnormative parenting (e.g., physical abuse). This sensitization to threat could then increase children's risk for anxiety. Support for this theory comes from a series of studies showing that physically abused children are selectively attentive to anger cues (Pollak & Tolley-Schell 2003), have difficulty disengaging from anger cues (Cicchetti & Curtis 2005, Curtis & Cicchetti 2011, Pollak et al. 2001, Pollak & Tolley-Schell 2003, Shackman et al. 2007), are more likely to identify ambiguous facial expressions as angry (Gibb et al. 2009, Pollak & Kistler 2002) or fearful (Masten et al. 2008), and need less perceptual information than nonabused children to accurately identify facial expressions of anger (Pollak et al. 2009, Pollak & Sinha 2002). Moreover, excessive attention to anger explains why physically abused youth have more symptoms of anxiety than nonabused youth (Shackman et al. 2007).

A commonly used method for assessing attention bias in children is to show them pairs of faces posed by the same model that appear on either side of a monitor. One face displays an angry (or happy or sad) expression, and the other face displays a neutral expression. Following presentation of the faces, a dot appears on either side of the screen and children are instructed to press a button with their left or right index finger to indicate the side of the screen where the dot appeared. Scores for attention bias to anger are higher when children take longer to identify the dot's location when it appeared on the same side of the screen as the neutral face than when it appeared on the same side as the angry face. That is, if a child is fixated on an angry face, she will notice the dot more quickly if it replaces the angry face (where she has been looking) than if it replaces the neutral face (where she has not been looking).

A number of studies have shown that children and adults with histories of maltreatment exhibit biased attention to angry faces, but not to sad or happy faces (Gibb et al. 2009). In a study of 4- to 7-year-olds, youth who were exposed to probable abuse showed a greater bias to anger relative to youth who experienced moderate or low levels of harsh parenting (Briggs-Gowan et al. 2015). Moreover, youth who exhibited a bias toward angry faces had significantly higher levels of observed anxiety than children who did not exhibit such a bias. Although the authors did not test whether attention bias to anger mediated the association between probable abuse and anxiety, they did show that probable abuse was more strongly associated with observed anxiety (and that exposure to intimate partner violence was associated with generalized anxiety disorder symptoms) when children displayed higher levels of attention bias toward angry faces (Briggs-Gowan et al. 2015).

The attention bias to angry faces has also been shown to characterize children exposed to more normative forms of harsh parenting. In a study of 9- to 15-year-olds, youth whose parents engaged in higher levels of authoritarian parenting, were more critical, or who displayed higher levels of negative affect during a social interaction task with their children had a greater attention bias for angry faces in a modified version of the dot probe task than youth whose parents were less authoritarian, critical, or negative in their parenting style (Gulley et al. 2014). Moreover, their attention bias to threat explained why these youth also had elevated symptoms of social anxiety (Gulley et al. 2014). Similarly, in a randomized control study of a foster care intervention with youth in Romania who were abandoned at birth, children who received care as usual (the majority of whom were being raised in government-run institutions or foster care) showed a significantly greater attention bias to angry faces compared with intervention youth (who were removed from institutions to receive high-quality foster care) (Troller-Renfree et al. 2015). In contrast, youth who were randomized at birth to receive the foster care intervention showed a significantly larger positive bias (i.e., to happy faces) compared with the care-as-usual group (Troller-Renfree et al. 2015). The magnitude of the positive bias was correlated with low levels of

externalizing problems, better social adjustment scores, higher levels of social engagement, and fewer emotionally withdrawn attachment disorder symptoms, although there was no formal test of mediation to determine whether a bias toward positive stimuli explained why intervention youth had more positive emotional and behavioral outcomes compared with youth who received care as usual (Troller-Renfree et al. 2015).

Not all studies of maltreated children find that their attention bias is toward threatening stimuli. At least two studies have found that children who have been maltreated have an attentional bias away from threat in the dot probe task, indicating that their reaction times were greater in trials when the dot appeared on the same side of the screen as the angry face versus trials when it appeared on the opposite side of the screen from the angry face (Kelly et al. 2015, Pine et al. 2005). Attention bias away from threat was also observed in a study of adult women, although only among those who carried at least one copy of the short form of the serotonin transporter polymorphism (Johnson et al. 2010). Finally, in a sample of adults, those who retrospectively reported childhood abuse showed a greater bias toward happy faces than adults without a history of abuse, but the groups did not differ in terms of attention toward angry faces (Fani et al. 2011).

The biased attention to threat involves limbic and prefrontal cortical circuitry (Williams et al. 2009). For example, maltreated children exhibit heightened amygdala reactivity to threat cues compared with nonmaltreated children, even when cues are presented preconsciously (McCrory et al. 2011, 2013; Tottenham et al. 2011). A childhood history of maltreatment is also associated with heightened right amygdala reactivity to angry and fearful versus neutral faces in psychiatrically healthy adults (Dannlowski et al. 2012) and bilateral amygdala reactivity to emotional faces (including angry, sad, fearful, and happy) versus neutral ones (van Harmelen et al. 2013). Activation of limbic regions (e.g., right amygdala and right insula) in response to fear stimuli and activation of left amygdala in response to anger stimuli has been shown to partially mediate the effect of child emotional maltreatment on symptoms of anxiety in adults (Fonzo et al. 2016). Moreover, in the same study, reduced activation of the right dorsolateral prefrontal cortex in response to fear and anger stimuli partially mediated the association between childhood emotional maltreatment and anxiety symptoms (Fonzo et al. 2016).

Although maltreated youth have been shown to be more reactive to negative stimuli than nonmaltreated youth, such responses can be modulated. For example, although adolescents who self-reported physical or sexual abuse showed greater left and right amygdala activation in response to negative versus neutral images compared with control youth, they also showed greater activation of prefrontal cortex during trials in which they were instructed to decrease their emotional response to negative stimuli, thus erasing maltreatment-related differences in amygdala activation in those trials (McLaughlin et al. 2015).

Most research focuses on hypervigilance to threat as a potential mechanism by which exposure to maltreatment increases risk for anxiety disorders, but it is equally possible that hypervigilance explains increased rates of aggressive behavior in maltreated youth. Maltreated youth are more likely than nonmaltreated youth to have social information processing styles characterized by a tendency to attribute hostile intent to others whose behavior is ambiguous (Crick & Dodge 1994, Dodge 1993, Keil & Price 2009; but for an exception, see Teisl & Cicchetti 2008). For example, when presented with a vignette in which a child's new radio is broken by a peer when the child is out of the room, a person who has a hostile attributional bias will assume that the radio was broken on purpose rather than by accident. Compared to nonmaltreated youth, physically abused children not only are more likely to make hostile attributions for a character's behavior in vignettes involving possible social provocation, but also generate more aggressive responses to the vignette than comparison youth (Keil & Price 2009). Neglected children, in contrast to physically abused youth, generate more aggressive responses than comparison youth to vignettes in which a child

tries to gain entry to a peer group and the group's response is positive, ambiguous, or negative (Keil & Price 2009).

A low threshold for perceiving anger and a tendency to allocate attention to anger may increase the likelihood that maltreated youth will attribute hostile intent to others' behavior and react accordingly. Indeed, physically abused children engage in higher rates of aggressive behavior partly because of their tendency to attribute hostile intent to others (Dodge et al. 1995). Although Teisl & Cicchetti (2008) did not find that maltreated youth made more hostile attributions than control youth, they did generate more aggressive responses to potential social provocations, and this explained why maltreated youth were perceived by peers as more aggressive than nonmaltreated youth. In addition to generating more aggressive responses, maltreated children generated fewer prosocial responses to potential social provocations. The inability to generate competent responses to social provocations (which is more characteristic of youth in out-of-home care than control youth) is also predictive of externalizing behavior problems (Kay & Green 2016).

The tendency to allocate more attention to angry faces (as measured by the P3b component during electroencephalography) has been shown to explain why physically abused youth engage in aggressive behavior. Shackman & Pollak (2014) studied children who played a video game with an online "partner." Physically abused youth displayed more negative affect and punished their partner more (by pressing a button to deliver an aversive noise) than control youth did during trials in which the partner was performing poorly in the game and reducing the chances that the pair would win a prize. Moreover, the abused children's tendency to allocate more attention to angry faces explained why they displayed more anger and aggressive behavior than control youth did (Shackman & Pollak 2014).

In summary, maltreated children and adults who were maltreated as children exhibit a biased attention to threatening stimuli, which partially explains their risk for anxiety and aggression. These findings are consistent with a body of research showing that maltreated (particularly physically abused) youth are more attentive to anger cues, have difficulty disengaging attention from anger cues, are faster to identify ambiguous cues as threatening, and recognize anger on the basis of less perceptual information than nonmaltreated children need. Although the majority of studies show that individuals who have a history of maltreatment exhibit biased attention toward threatening stimuli, several studies find the opposite—that children and adults with histories of maltreatment direct their attention away from threatening stimuli. Differences in findings could reflect the fact that samples vary in terms of their maltreatment exposure (i.e., the specific form of maltreatment varies across samples) and differ in terms of PTSD symptomatology. For instance, high rates of PTSD characterized two of the four samples that showed a bias away from threat or toward positive stimuli (Fani et al. 2011, Pine et al. 2005). Given that PTSD is characterized by both hypervigilance toward threatening stimuli and avoidance of threat (Armour et al. 2016), an attentional bias away from threat may be as likely in this group as an attentional bias toward threat.

Deficits in Emotion Recognition

The ability to accurately recognize and understand emotions is a key developmental task that facilitates social interactions. Children and adults who exhibit deficits in emotion understanding are more likely to suffer social rejection than individuals who are more skilled in this domain (Miller et al. 2005). Individual differences in children's recognition and understanding of emotions partly derives from parents' efforts to model and explain emotions (Brownell et al. 2013). The quality of emotion socialization is likely to be different in abusive or neglectful families versus those in which children are not maltreated. For example, physically abusive mothers have been shown to produce

less prototypical facial expression of anger and less prototypical vocal expressions of anger, fear, and happiness than nonabusive mothers (Shackman et al. 2010). Maltreating mothers have also been shown to engage in less validation, less coaching, and more invalidation of their children's emotions than nonmaltreating mothers—an effect that partially explains their children's increased risk for emotion regulation problems (Shipman et al. 2007). Finally, a meta-analysis of emotion recognition accuracy in parents who were at high and low risk for child physical abuse determined that high-risk parents were less accurate than low-risk parents—an effect size that was moderate in magnitude (Wagner et al. 2015). Thus, abusive parents not only model atypical facial expressions, but may also model ways of responding to children's emotions that increase children's risk for emotion regulation problems.

Consistent with the fact that abusive parents produce less prototypical expressions of emotions, a number of studies have shown that maltreated youth are poorer than nonmaltreated youth at accurately identifying facial expressions of emotion. For example, when presented with stories in which children are experiencing a certain emotion, maltreated youth are less accurate than nonmaltreated youth at matching the emotion in the story to the corresponding photo of the emotion when presented with three choices (Camras et al. 1983, Pollak et al. 2000). Other protocols present participants with photographs of prototypical emotion expressions and ask them to identify the person in the photo who is feeling a particular emotion or ask them what the person in the photo is feeling. Maltreated youth are less accurate at recognizing emotions than nonmaltreated youth (During & McMahon 1991, Pears & Fisher 2005; but for an exception see Masten et al. 2008), and this effect may be strongest when maltreated youth have relatively low IQ (Shenk et al. 2013). One study found that the specific form of the emotion recognition deficit corresponded to the type of maltreatment experienced by the child, with neglected children—whose interactions with caregivers are emotionally impoverished—less likely than control children to differentiate facial expressions of emotion, and physically abused children—whose interactions with caregivers are consistently aggressive—showing a bias to recognize emotions as angry (Pollak et al. 2000).

Maltreated youth have difficulties not only recognizing emotion expressions, but also matching emotional outcomes with their common elicitors. Although typically developing preschoolers can predict which kinds of situations will elicit positive versus negative emotions, maltreated preschoolers reported that positive, equivocal, and negative events were equally plausible elicitors of anger or fear in a task in which emotional outcomes were first presented to children (e.g., a happy, sad, or angry face) and children were asked how likely it was that a positive, negative, or equivocal event might have elicited that emotion (Perlman et al. 2008). A recent meta-analysis confirmed that maltreated youth have poorer emotion recognition and understanding than nonmaltreated youth; the magnitude of the effect is bigger for emotion recognition than understanding, and the largest effects are seen in early childhood (Luke & Banerjee 2013).

In summary, maltreated children have difficulty recognizing others' emotions, which may adversely affect their ability to respond appropriately when others express emotions (Klimes-Dougan & Kistner 1990). Moreover, maltreated children have difficulty understanding the sorts of situations that typically elicit positive and negative emotions—a deficit that may affect their ability to predict the reactions that their own negative behaviors elicit from others (Perlman et al. 2008) and may heighten the likelihood that they will be rejected by peers. Although it is clear that problems recognizing and understanding others' emotions lead to problems with peers, studies are needed to demonstrate that this is a pathway by which maltreated children are at risk for depression or aggressive behavior. For example, maltreated children have difficulty not only recognizing and understanding emotions but also regulating their own emotions (Kim & Cicchetti 2009). The externalizing problems exhibited by these youth elicit rejection by peers, which exacerbates their problem behaviors (Kim & Cicchetti 2009).

Reward Responsiveness

Reduced responsiveness to reward is hypothesized to be a neural mechanism by which maltreatment increases risk for depression—particularly anhedonic symptoms. Behaviorally, maltreated children and adults who were maltreated as children are less sensitive to cues for reward than nonmaltreated controls (Dillon et al. 2009, Guyer et al. 2006). Mesolimbic dopamingeric circuitry that projects to the basal ganglia (including the ventral striatum and nucleus accumbens) is involved in responsivity to reward and particularly in anticipation of reward (Dunlop & Nemeroff 2007). Thus, in a task in which participants were presented with reward, loss, and no-incentive cues, young adults who had been maltreated in childhood rated reward cues less positively than control participants, although groups did not differ in their response to actual gains (Dillon et al. 2009). Moreover, although control participants showed greater left basal ganglia activation in response to reward versus loss or no-incentive cues, maltreated participants did not exhibit this difference (Dillon et al. 2009). Consistent with these findings, a small study of postinstitutionalized adolescents found that they showed less ventral striatum activation in anticipation of moderate to large rewards compared to control participants (Mehta et al. 2010).

Rates of depression tend to increase in adolescence (Hankin et al. 1998), and individual differences in the development of reward-related neural circuitry associated with early life stress may underlie this increase. In a study that followed adolescents from age 13 to 15 years, youth who experienced more emotional neglect in childhood showed less change in ventral striatal activity from age 13 to 15 in response to reward (Hanson et al. 2015). In addition, individual differences in ventral striatal activity mediated the association between childhood emotional neglect and adolescent symptoms of depression (Hanson et al. 2015). Importantly, the changes in ventral striatal activity associated with emotional maltreatment were observed before the onset of major depressive disorder, making it more plausible that reduced responsivity to reward is a potential risk mechanism, rather than a correlate, of clinically significant depression.

Other research in this area has focused on the anterior cingulate cortex (ACC), which is involved in reinforcement learning (Berridge & Robinson 2003). Researchers have hypothesized that the relatively slow maturation of the ACC leaves it vulnerable to the physiological sequelae of chronic stressors, such as maltreatment, that lead to excessive release of glucocorticoids as part of the stress response (Pechtel & Pizzagalli 2013). Women with histories of childhood sexual abuse and major depressive disorder responded more slowly and less accurately than women with histories of major depressive disorder alone or than controls to trials in which they had to learn which of two stimuli (A or B) was more consistently rewarded. For example, although participants received feedback on 70% of the training trials that A was the correct response and received feedback on only 30% of the training trials that B was the correct response, sexually abused women more often chose B as the correct response than women without sexual abuse histories (regardless of their depression status). In addition, sexually abused women were less accurate in picking the rewarded stimulus in novel trials, although they were equally accurate at avoiding the punished stimulus. That is, when A was paired with C, D, E, or F stimuli—all of which were rewarded less consistently than A in the learning phase of the study—sexually abused women less frequently chose A as the correct response than nonabused women. However, in trials in which B was paired with the other stimuli—all of which were rewarded more consistently than B in the learning phase—sexually abused women were as accurate at avoiding B as the nonabused women. These findings suggest that childhood sexual abuse affects development of the ACC, thus affecting the ability to learn from positive experiences and perpetuating maladaptive decision making that may increase risk for depression.

In summary, children and adults who have histories of maltreatment tend to be less responsive to reward—and particularly to the anticipation of reward—than individuals who do not have histories

of maltreatment. For example, they do not discriminate cues for high risk/high reward from cues for low risk/low reward in terms of reaction times (Guyer et al. 2006), and they rate cues for reward less positively than control participants (Dillon et al. 2009). Maltreated and nonmaltreated individuals also exhibit differences in activation of the neural circuitry underlying this behavior. Thus, the increased risk for depression observed in adolescents and adults who have histories of maltreatment may be partly explained by reduced responsivity to reward and by deficits in the ability to learn from positive experiences. Prospective, longitudinal data are needed to confirm that maltreatment is associated with deficits in reward responsiveness that are observable before the onset of clinically significant depression.

MODERATORS OF CHILD MALTREATMENT

Moderators of maltreatment can be conceptualized as factors that exacerbate effects of maltreatment on risk for psychopathology (e.g., in a diathesis-stress framework) or as factors that promote competence in mental health, academic, or interpersonal domains despite exposure to maltreatment (e.g., in a resilience framework). In studies that stringently define resilience to maltreatment as competence that is sustained over time across more than one domain, between 12% and 22% of individuals who were maltreated as children are defined as resilient (Cicchetti et al. 1993, Jaffee & Gallop 2007, Kaufman et al. 1994, McGloin & Widom 2001). The effect of maltreatment on risk for psychopathology is largely similar across demographic groups (e.g., sex, race, and ethnicity) (Jaffee & Maikovich-Fong 2013). Similarly, evidence that the association between maltreatment and risk for psychopathology varies as a function of maltreatment subtype is weak and inconsistent. In contrast, maltreatment that is chronic tends to be more strongly associated with risk for psychopathology than maltreatment that occurs sporadically or that is confined to a single developmental period (Jaffee & Maikovich-Fong 2011). With respect to age-at-onset, although maltreatment that is first experienced in childhood is more strongly associated with internalizing outcomes than maltreatment that is first experienced in adolescence, the reverse is true with respect to externalizing problems (Kaplow & Widom 2007, Thornberry et al. 2010).

The remainder of this section focuses on two literatures showing that the effects of maltreatment on risk for psychopathology vary as a function of (*a*) genotype and (*b*) other individual and environmental factors. These are reviewed in turn.

Genetic Moderators of Maltreatment Effects

Although there have been dozens of reports of genotype by maltreatment (G × E) interactions predicting psychopathology, this review is focused on the best-replicated findings. These include studies testing whether monoamine oxidase A (*MAOA*) genotype moderates effects of maltreatment on risk for antisocial behavior and studies testing whether the serotonin transporter–linked polymorphic region (5-HTTLPR; *SLC6A4*) genotype moderates effects of maltreatment on risk for depression. Readers interested in methodological critiques of the G × E literature should see papers by Duncan & Keller (2011), Caspi et al. (2010), and Aliev et al. (2014). Moreover, candidate G × E studies are likely to capture only a small fraction of the variation accounted for by the biological interactions among networks of genes and environments over time.

***MAOA* × maltreatment.** The *MAOA* gene is involved in the metabolism of dopamine, serotonin, and norepinephrine. Interest in this gene was piqued by a study showing that a mutation in the *MAOA* gene was associated with violent behavior among males in a large Dutch family (Brunner et al. 1993). A 30–base pair variable number tandem repeat polymorphism in this gene has been

identified and is characterized by a low activity variant (3 or 5 repeats) that leads to lower *MAOA* expression and a high activity variant (3.5 or 4 repeats) that leads to higher *MAOA* expression (Sabol et al. 1998). Because the *MAOA* gene is located on the X chromosome, males are hemizygous for the low or high activity variant whereas females can carry zero, one, or two copies of the low activity variant.

The first published report of an interaction between *MAOA* genotype and maltreatment came from Caspi et al. (2002), who showed that men in the Dunedin Longitudinal Study birth cohort who had experienced childhood maltreatment had elevated levels of childhood conduct and adult antisocial behavior problems if they carried the low activity variant of the *MAOA* gene. Men who carried the high activity variant of the *MAOA* gene were not at elevated risk for antisocial outcomes regardless of their exposure to childhood maltreatment. A recent meta-analysis of 27 studies tested whether the interaction was (*a*) specific to maltreatment versus adverse life events more generally and (*b*) specific to males versus females (Byrd & Manuck 2014). This meta-analysis reported that the association between early life adversity (specifically maltreatment) and antisocial outcomes was significantly stronger for males who carried the low versus the high activity variant of the *MAOA* gene. In the 11 female cohorts, maltreatment was more strongly associated with antisocial outcomes among carriers of the high versus the low activity variant, although this effect was not robust to sensitivity checks (Byrd & Manuck 2014). Consistent with the possibility that there are sex-specific effects of the *MAOA* variant on antisocial behavior, data from one prospective longitudinal study showed that, for women, increasing numbers of childhood adverse events were associated with increases in amygdala and hippocampal activity in response to angry or fearful faces if they carried the high activity variant and decreases if they carried the low activity variant, whereas the pattern of effects was reversed for men (Holz et al. 2016).

A future direction for research is to identify why males who carry the low activity variant are at elevated risk for antisocial outcomes when they are exposed to maltreatment. Imaging genetic studies suggest possible mechanisms. For example, individuals who carry the low activity variant exhibit low resting state activity in areas of the brain related to executive function and inhibitory control (Clemens et al. 2015) as well as reduced activation of the ventrolateral prefrontal cortex (Passamonti et al. 2006) and ACC (Fan 2003, Fossella et al. 2002) during tasks designed to measure response inhibition and executive function. Individuals who carry the low activity variant exhibit heightened activation in response to insult or to sad or angry faces in the regions of the brain involved in emotion regulation, including the amygdala, ACC, and hippocampus (Denson et al. 2014, Lee & Ham 2008, Meyer-Lindenberg et al. 2006). Moreover, activation of the left amygdala and posterior thalamus in response to hearing the word "no" (versus the word "up") is more highly correlated with trait anger reactivity in carriers of the low versus high activity variant (Alia-Klein et al. 2009). Finally, low *MAOA* activity in cortical and subcortical regions as measured by positron emission topography is associated with heightened levels of trait aggression (Alia-Klein et al. 2008), and hypermethylation in CpG sites in the promoter region of *MAOA* has been associated with decreased promoter activity and lower levels of circulating serotonin (Checknita et al. 2015). Studies are needed that show how maltreatment interacts with these processes to increase risk for antisocial behavior.

5-HTTLPR × maltreatment. 5-HTTLPR is a 43-base pair insertion/deletion polymorphism (Lesch et al. 1996). The serotonin transporter (5-HTT) plays a vital role in the regulation of serotonin (5HT) reuptake (Lesch et al. 1996), and dysregulated 5-HT functioning is associated with both depression and aggression (Lucki 1998). Homozygosity for the long (L) allele is associated with increased transcriptional efficiency in human lymphoblast cells, whereas the short (S) allele is associated with diminished *5-HTT* gene transcription (Greenberg et al. 1999, Heils

et al. 2002). Although many studies focus on differences between S and L carriers, an additional insertion/deletion polymorphism (rs25531; A/G) within the serotonin transporter polymorphism modifies the transcriptional efficacy of the L allele such that only L_G carriers have high serotonin transporter messenger RNA levels whereas the transcriptional efficacy of L_A carriers is similar to that of S carriers (Hu et al. 2006).

The first published report of an interaction between 5-HTTLPR genotype (*SLC6A4*) and maltreatment also came from the Dunedin Longitudinal Study and showed that among individuals who were homozygous for the short form of the serotonin transporter allele, a childhood history of maltreatment was associated with elevated risk for depression and depressive symptomatology in adulthood (Caspi et al. 2003). Two of three subsequent meta-analyses have tested whether the effect of stressful life events (rather than maltreatment specifically) is moderated by 5-HTTLPR genotype and have concluded that evidence for an interaction effect is weak (Munafo et al. 2009, Risch et al. 2009). In contrast, the meta-analysis by Karg et al. (2011) stratified studies according to type of stressful life event and found that 5-HTTLPR genotype moderated the effect of maltreatment (as opposed to other stressful life events) on risk for depression.

Risk for depression may be elevated among individuals who carry the 5-HTTLPR S allele because these individuals have a more pronounced physiological response to stress than L allele carriers. For example, a meta-analysis showed that S/S homozygotes mount a significantly greater cortisol response to acute stressors than L-allele carriers (Miller et al. 2013), though it is not clear whether S and L allele carriers differ in their physiological response to chronic stress, such as ongoing abuse or neglect. In addition, S allele carriers show enhanced amygdala reactivity to negatively valenced stimuli compared with individuals who are homozygous for the L allele (Munafo et al. 2008). A handful of investigations have shown that elevated cortisol reactivity to acute stressors and amygdala reactivity to negative emotional stimuli in S allele carriers is most pronounced among those who have experienced numerous stressful life events (Alexander et al. 2009, 2012; Williams et al. 2009; but for an alternative interaction pattern, see Canli et al. 2006, Mueller et al. 2011), thus demonstrating that potential endophenotypes for depression are also predicted by G × E interactions.

Two studies of healthy adults have investigated whether interactions between 5-HTTLPR genotype and childhood maltreatment alter *SLC6A4* expression or methylation levels. Although one found that individuals who carried the S allele and who had experienced childhood maltreatment had 56% lower serotonin transporter messenger RNA expression levels compared with individuals who were homozygous for the L allele and who did not report childhood maltreatment (Wankerl et al. 2014); this additive effect was not replicated in an independent sample (Duman & Canli 2015). Moreover, alterations in *SLC6A4* expression are not consistently associated with alterations in *SLC6A4* methylation levels (Duman & Canli 2015). At this point, it is not clear what roles gene expression and methylation play in explaining why S allele carriers who are exposed to maltreatment are at elevated risk for depression.

Along with the genetic and imaging genetic study findings, there is evidence that the S allele is associated with cognitive vulnerabilities to depression, particularly under stressful conditions (Gibb et al. 2013). For example, compared with individuals who are homozygous for the L allele, healthy adults who carry the S allele take longer to disengage attention from facial expressions of emotions (Beevers 2009) and they appraise recent stressful life events as being more negative (Conway et al. 2012), with negative appraisals correlated with elevations in depressive symptoms (Conway et al. 2012). Another study found that healthy children who carried the S allele had enhanced memory for negative (versus positive) self-descriptive traits (Hayden et al. 2013), thus exhibiting a potential cognitive vulnerability for depression. A meta-analysis of 11 samples from 10 published studies showed that S/S, S/L_G, and L_G/L_G carriers possess a greater negative attention

bias (i.e., they showed a bias to negative versus neutral stimuli) than L_A/L_A carriers do (Pergamin-Hight et al. 2012).

A few investigations have shown that coping or perceptions of coping are associated with the 5-HTTLPR genotype. For example, among adults who were asked to recall recent situations in which they had felt strong emotions of fear, sadness, or joy, those who carried the S allele reported that they had felt less able to cope with situations that evoked strong feelings of sadness or fear than did individuals who were homozygous for the L allele (Szily et al. 2008). In another study, healthy young adults who were homozygous for the S allele less frequently endorsed the use of cognitive reappraisals to deal with negative emotions or events (e.g., "When I want to feel less negative emotion, I change what I'm thinking about" or "I look for the positive side of the matter") than L allele carriers. In turn, their less frequent use of cognitive reappraisal strategies explained why individuals who carried two copies of the S allele had increased symptoms of social anxiety (Miu et al. 2013). Finally, in a sample of 156 healthy adults, Wilhelm et al. (2007) reported that S allele carriers utilized fewer problem-solving coping strategies in response to a stressor. Consistent with these findings, Cline et al. (2015) showed that among youth who were homozygous for the S allele, those who were exposed to lower parental warmth, higher harsh discipline, and more traumatic events less frequently used distraction coping strategies, such as playing a video game or playing sports, and this partly explained their elevated symptoms of internalizing problems (Cline et al. 2015).

Psychosocial Moderators of Maltreatment

Children who are resilient to maltreatment tend to be characterized by high ego control and ego resiliency, high self-esteem, high self-reliance, and the tendency to attribute successes to their own efforts (Cicchetti et al. 1993, Feiring et al. 2002, Moran & Eckenrode 1992). Above-average intelligence has been identified as a protective factor in some studies (Herrenkohl et al. 1994, Jaffee et al. 2007), but not others (Cicchetti & Rogosch 1997, Dumont et al. 2007). These individual characteristics may be protective only as long as children are not exposed to a multitude of stressors in addition to maltreatment (Dumont et al. 2007, Jaffee et al. 2007).

Because there is a relatively large literature on the topic, the remainder of this section focuses on social support as a factor that potentially buffers children and adults from the adverse effects of maltreatment. Social support is defined in a variety of ways in the literature on child abuse and neglect, with some studies measuring perceived support in a variety of domains (e.g., material support, emotional support) and others measuring the quality of (or satisfaction with) relationships with parents, friends, intimate partners, or other relatives.

Children and adults with histories of maltreatment tend to experience lower levels of social support from friends and family members (Horan & Widom 2015, Lamis et al. 2014, Powers et al. 2009, Salazar et al. 2011, Sperry & Widom 2013, Vranceanu et al. 2007) and less stable social support across the life course (Horan & Widom 2015). Having low levels of social support partially explains why children and adults with histories of maltreatment have elevated rates of psychopathology (Lamis et al. 2014, Runtz & Schallow 1997, Sperry & Widom 2013, Vranceanu et al. 2007), although this mediation effect is not always observed (Powers et al. 2009, Tremblay et al. 1999).

When present, however, socially supportive relationships can buffer individuals with histories of maltreatment from risk for psychopathology, meaning that high levels of social support reduce rates of psychopathology to the same low levels as in children or adults without histories of maltreatment. At its most stringent, this definition requires that (high) social support be associated (or be more strongly associated) with reduced risk for psychopathology only for victims of abuse or neglect; nonvictims are expected to have low rates of psychopathology regardless of their social

support. For example, one cross-sectional, retrospective study showed that women with childhood histories of abuse and adult rape histories reported fewer symptoms of PTSD if they had high (versus low) levels of social support, but social support was unassociated with PTSD symptoms for women who had experienced neither or only one of those interpersonal traumas (Schumm et al. 2006). Similarly, incarcerated adolescents who reported higher levels of satisfaction with the amount of social support they received had lower rates of suicidal ideation than adolescents who reported lower levels of satisfaction; this association was observed for those with a history of sexual abuse, but not for those without (Esposito & Clum 2002). In some instances, the buffering effects of social support are gender specific, but findings are mixed as to whether social support specifically benefits women (Gayer-Anderson et al. 2015, Sperry & Widom 2013) or men (Folger & Wright 2013).

Statistical interactions between maltreatment and social support are not always consistent with the buffering hypothesis. Some studies have shown that social support only benefits individuals without histories of abuse (Folger & Wright 2013) or—counterintuitively—that exposure to violence is more strongly associated with poor psychological outcomes for youth who have high rather than low levels of social support (Gagne & Melancon 2013, Salazar et al. 2011). These findings may reflect the fact that youth with high levels of problem behaviors are encouraged to seek support (e.g., in the form of counseling) or that they elicit that support from adults who perceive they need it.

In contrast to studies that report buffering effects (i.e., statistical interactions between social support and maltreatment status), others have shown that having more socially supportive relationships is associated with lower levels of depressive symptoms (Henry et al. 2015) and other forms of psychopathology (Gagne & Melancon 2013) regardless of an individual's maltreatment history. Still other studies focus solely on maltreated children or adults in an effort to identify factors that differentiate those who are resilient (in terms of having good mental health and social functioning) from those who are not resilient. For example, normal peer relationships in adolescence, high-quality friendships in adulthood, and a stable relationship history predicted low rates of psychopathology among adults with childhood histories of sexual abuse (Collishaw et al. 2007). In a sample of maltreated girls, having a warm relationship with the nonmaltreating parent differentiated girls who were resilient (as defined by the absence of clinical symptomatology or by high levels of social adjustment) from girls who were nonresilient (Spaccarelli & Kim 1995). Finally, one study of nonparental mentors among youth in foster care found that youth who had been mentored before the age of 18 were more likely to participate in higher education and have better self-reported physical health, and they were less likely to have been diagnosed with a sexually transmitted infection, engage in suicidal ideation, or hurt someone in a fight than those who did not report the presence of a mentor (Ahrens et al. 2008).

The causal role of social support as a buffering factor, direct protective factor, or mediator is difficult to establish, given that individual differences in temperament or personality could alter an individual's perception that social support is being provided, their satisfaction with the provision of support, or their actual receipt of support. One study used a propensity score weighting approach in an attempt to match maltreated youth who reported low versus high access to support from friends, family, and nonfamily members (Dingfelder et al. 2010). In unadjusted models, youth who reported that they had a higher (versus lower) number of friends, family, or nonfamily members to whom they could go for support had lower levels of depressive symptoms. Although the magnitude of this association was reduced in the propensity score weighting model, the association remained significant (Dingfelder et al. 2010). Intervention studies in which maltreated youth are randomized to receive formal or informal support would provide strong evidence for or against the causal role of social support in reducing rates of psychopathology.

In summary, there is substantial evidence that children and adults with histories of maltreatment have less social support than children and adults without histories of maltreatment. The evidence is mixed as to whether social support has a buffering effect or whether it has a direct protective effect (in the sense that having social support is beneficial regardless of a person's maltreatment history). Given the substantial heterogeneity in how social support is measured across studies, it is difficult to determine the reasons for mixed findings. In an effort to achieve consistency, future studies of social support may want to measure certain key dimensions, including (*a*) the size of a person's network, (*b*) the source of support (e.g., friends, family members, extrafamilial adults), and (*c*) satisfaction with receipt of support in specific domains.

CONCLUSIONS

Maltreatment is a significant public health problem. From a basic research perspective, there is a need for more prospective, longitudinal data on maltreatment to better understand the course of resilience and dysfunction over time and the long-term effects of maltreatment on mental and physical health. A mix of research strategies and research models is needed to understand the mechanisms by which neglect and abuse influence basic biological and psychological processes. Research on the biology of maltreatment would benefit from larger and more representative samples, whereas research on the psychological sequelae of maltreatment would benefit from research designs that allow for stronger causal inferences about potential mediators of maltreatment. An integrative, multilevel perspective is needed to trace the effects of maltreatment on pathways from genes to brain to behavior. From a clinical perspective, more research is needed to better evaluate treatment efficacy for maltreated children, to improve access to services and the quality of services for maltreated children, and to understand why some maltreated children respond better to treatment than others.

SUMMARY POINTS

- Although rates of abuse and neglect are on the decline in the United States, 9.4 per 1,000 children were found to be victims in 2014, the most recent year for which figures are available. Children under the age of 3 have the highest risk of victimization.
- Although having a childhood history of maltreatment increases the odds that an individual will engage in abuse or neglect as an adult, the majority of children who are maltreated do not grow up to perpetrate abuse or neglect.
- Child victims of maltreatment and adults who were maltreated in childhood are at risk for a range of mental health problems, including depression, anxiety, substance abuse, antisocial behavior, psychotic symptoms, and personality disorders. The risk for psychopathology associated with a history of abuse or neglect is not due to co-occurring sociodemographic risk factors.
- Maltreatment is likely to increase risk for different forms of psychopathology by increasing threat sensitivity, decreasing responsivity to reward, and producing deficits in emotion recognition and understanding.
- Mental health problems are not inevitable consequences of being maltreated. Access to socially supportive relationships can buffer individuals from the adverse effects of maltreatment, and some individuals carry gene variants that are associated with reduced susceptibility to maltreatment.

FUTURE ISSUES

- The field relies heavily on retrospective self-reports of maltreatment, which can be biased by current mental state and are prone to omission errors. Prospective, longitudinal studies could determine whether exposure to maltreatment is associated with the emergence of neurocognitive, cognitive, or affective deficits that produce vulnerability to psychopathology or whether chronic exposure to maltreatment is associated with increasing severity of such deficits.
- Although many studies show that victims of maltreatment are characterized by a range of neurocognitive and affective deficits, more studies are needed that demonstrate that these endophenotypes explain the risk for psychopathology associated with a history of maltreatment.
- For obvious ethical reasons, children cannot be randomly assigned to abusive versus nonabusive families, and thus, it can be difficult to determine whether maltreatment is a cause or a correlate of psychopathology. Research designs that allow for stronger causal inference include (*a*) case-control designs in which maltreated cases and controls are carefully matched for sociodemographic factors, (*b*) very large samples of twin or nontwin siblings who are discordant for their exposure to maltreatment, and (*c*) longitudinal studies of high-risk samples that assess an individual's functioning before and after exposure to maltreatment.

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LITERATURE CITED

Ahrens KR, DuBois DL, Richardson LP, Fan M-Y, Lozano P. 2008. Youth in foster care with adult mentors during adolescence have improved adult outcomes. *Pediatrics* 121(2):e246–52. **https://doi.org/10.1542/peds.2007-0508**

Alexander N, Klucken T, Koppe G, Osinsky R, Walter B, et al. 2012. Interaction of the serotonin transporter-linked polymorphic region and environmental adversity: increased amygdala-hypothalamus connectivity as a potential mechanism linking neural and endocrine hyperreactivity. *Biol. Psychiatry* 72(1):49–56

Alexander N, Kuepper Y, Schmitz A, Osinsky R, Kozyra E, Hennig J. 2009. Gene-environment interactions predict cortisol responses after acute stress: implications for the etiology of depression. *Psychoneuroendocrinology* 34(9):1294–303

Alia-Klein N, Goldstein RZ, Kriplani A, Logan J, Tomasi D, et al. 2008. Brain monoamine oxidase A activity predicts trait aggression. *J. Neurosci.* 28(19):5099–104

Alia-Klein N, Goldstein RZ, Tomasi D, Woicik PA, Moeller SJ, et al. 2009. Neural mechanisms of anger regulation as a function of genetic risk for violence. *Emotion* 9(3):385–96. **https://doi.org/10.1037/a0015904**

Aliev F, Latendresse SJ, Bacanu S-A, Neale MC, Dick DM. 2014. Testing for measured gene-environment interaction: problems with the use of cross-product terms and a regression model reparameterization solution. *Behav. Genet.* 44(2):165–81. **https://doi.org/10.1007/s10519-014-9642-1**

Armour C, Műllerová J, Elhai JD. 2016. A systematic literature review of PTSD's latent structure in the Diagnostic and Statistical Manual of Mental Disorders: DSM-IV to DSM-5. *Clin. Psychol. Rev.* 44:60–74. **https://doi.org/10.1016/j.cpr.2015.12.003**

Arseneault L, Cannon M, Fisher HL, Polanczyk G, Moffitt TE, Caspi A. 2011. Childhood trauma and children's emerging psychotic symptoms: a genetically sensitive longitudinal cohort study. *Am. J. Psychiatry* 168:65–72

Bar-Haim Y, Lamy D, Pergamin L, Bakermans-Kranenburg MJ, van IJzendoorn MH. 2007. Threat-related attentional bias in anxious and nonanxious individuals: a meta-analytic study. *Psychol. Bull.* 133(1):1–24. **https://doi.org/10.1037/0033-2909.133.1.1**

Beevers CG, Wells TT, Ellis AJ, McGeary JE. 2009. Association of the serotonin transporter gene promoter region (5-HTTLPR) polymorphism with biased attention for emotional stimuli. *J. Abnorm. Psychol.* 118(3):670–81

Berridge KC, Robinson TE. 2003. Parsing reward. *Trends Neurosci.* 26(9):507–13. **https://doi.org/10.1016/S0166-2236(03)00233-9**

Bolger KE, Patterson CJ. 2001. Pathways from child maltreatment to internalizing problems: perceptions of control as mediators and moderators. *Dev. Psychopathol.* 13(4):913–40

Breslau N, Koenen KC, Luo Z, Agnew-Blais J, Swanson S, et al. 2014. Childhood maltreatment, juvenile disorders and adult post-traumatic stress disorder: a prospective investigation. *Psychol. Med.* 44(9):1937–45. **https://doi.org/10.1017/S0033291713002651**

Briggs-Gowan MJ, Pollak SD, Grasso D, Voss J, Mian ND, et al. 2015. Attention bias and anxiety in young children exposed to family violence. *J. Child Psychol. Psychiatry* 56(11):1194–201. **https://doi.org/10.1111/jcpp.12397**

Brown J, Cohen P, Johnson JG, Smailes EM. 1999. Childhood abuse and neglect: specificity of effects on adolescent and young adult depression and suicidality. *J. Am. Acad. Child Adolesc. Psychiatry* 38(12):1490–96

Brownell CA, Svetlova M, Anderson R, Nichols SR, Drummond J. 2013. Socialization of early prosocial behavior: Parents' talk about emotions is associated with sharing and helping in toddlers. *Infancy* 18(1):91–119. **https://doi.org/10.1111/j.1532-7078.2012.00125.x**

Brunner HG, Nelen M, Breakefield XO, Ropers HH, van Oost BA. 1993. Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. *Science* 262:578–80

Byrd AL, Manuck SB. 2014. MAOA, childhood maltreatment, and antisocial behavior: meta-analysis of a gene-environment interaction. *Biol. Psychiatry* 75(1):9–17. **https://doi.org/10.1016/j.biopsych.2013.05.004**

Camras LA, Grow JG, Ribordy SC. 1983. Recognition of emotional expression by abused children. *J. Clin. Child Psychol.* 12(3):325–28. **https://doi.org/10.1207/s15374424jccp1203_16**

Canli T, Qiu M, Omura K, Congdon E, Haas BW, et al. 2006. Neural correlates of epigenesis. *PNAS* 103(43):16033–38

Caspi A, Hariri AR, Holmes A, Uher R, Moffitt TE. 2010. Genetic sensitivity to the environment: the case of the serotonin transporter gene and its implications for studying complex diseases and traits. *Am. J. Psychiatry* 167(5):509–27

Caspi A, McClay J, Moffitt TE, Mill J, Martin J, et al. 2002. Role of genotype in the cycle of violence in maltreated children. *Science* 297:851–54

Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, et al. 2003. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science* 31:386–89

Checknita D, Maussion G, Labonté B, Comai S, Tremblay RE, et al. 2015. Monoamine oxidase A gene promoter methylation and transcriptional downregulation in an offender population with antisocial personality disorder. *Br. J. Psychiatry* 206(3):216–22. **https://doi.org/10.1192/bjp.bp.114.144964**

Cicchetti D, Curtis WJ. 2005. An event-related potential study of the processing of affective facial expressions in young children who experienced maltreatment during the first year of life. *Dev. Psychopathol.* 17(3):641–77

Cicchetti D, Rogosch FA. 1997. The role of self-organization in the promotion of resilience in maltreated children. *Dev. Psychopathol.* 9:797–815

Cicchetti D, Rogosch FA, Lynch M, Holt KD. 1993. Resilience in maltreated children: processes leading to adaptive outcome. *Dev. Psychopathol.* 5(4):629–47

Clemens B, Voß B, Pawliczek C, Mingoia G, Weyer D, et al. 2015. Effect of MAOA genotype on resting-state networks in healthy participants. *Cereb. Cortex* 25(7):1771–81. **https://doi.org/10.1093/cercor/bht366**

Cline JI, Belsky J, Li Z, Melhuish E, Lysenko L, et al. 2015. Take your mind off it: coping style, serotonin transporter linked polymorphic region genotype (*5-HTTLPR*), and children's internalizing and externalizing problems. *Dev. Psychopathol.* 27(4 Part 1):1129–43. **https://doi.org/10.1017/S0954579415000723**

Cohen P, Brown J, Smailes E. 2001. Child abuse and neglect and the development of mental disorders in the general population. *Dev. Psychopathol.* 13(4):981–99

Collishaw S, Pickles A, Messer J, Rutter M, Shearer C, Maughan B. 2007. Resilience to adult psychopathology following childhood maltreatment: evidence from a community sample. *Child Abus. Negl.* 31(3):211–29. **https://doi.org/10.1016/j.chiabu.2007.02.004**

Colman I, Kingsbury M, Garad Y, Zeng Y, Naicker K, et al. 2016. Consistency in adult reporting of adverse childhood experiences. *Psychol. Med.* 46(3):543–49. **https://doi.org/10.1017/S0033291715002032**

Conger RD, Schofield TJ, Neppl TK, Merrick MT. 2013. Disrupting intergenerational continuity in harsh and abusive parenting: the importance of a nurturing relationship with a romantic partner. *J. Adolesc. Health* 53(4):S11–17. **https://doi.org/10.1016/j.jadohealth.2013.03.014**

Conway CC, Hammen C, Espejo EP, Wray NR, Najman JM, Brennan PA. 2012. Appraisals of stressful life events as a genetically-linked mechanism in the stress-depression relationship. *Cogn. Ther. Res.* 36(4):338–47

Crick NR, Dodge KA. 1994. A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychol. Bull.* 115(1):74–101

Crusto CA, Whitson ML, Walling SM, Feinn R, Friedman SR, et al. 2010. Posttraumatic stress among young urban children exposed to family violence and other potentially traumatic events. *J. Trauma. Stress* 23(6):716–24

Curtis WJ, Cicchetti D. 2011. Affective facial expression processing in young children who have experienced maltreatment during the first year of life: an event-related potential study. *Dev. Psychopathol.* 23(2):373–95

Danese A, McEwen BS. 2012. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol. Behav.* 106(1):29–39

Dannlowski U, Stuhrmann A, Beutelmann V, Zwanzger P, Lenzen T, et al. 2012. Limbic scars: long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. *Biol. Psychiatry* 71(4):286–93

Denson TF, Dobson-Stone C, Ronay R, von Hippel W, Schira MM. 2014. A functional polymorphism of the *MAOA* gene is associated with neural responses to induced anger control. *J. Cogn. Neurosci.* 26(7):1418–27. **https://doi.org/10.1162/jocn_a_00592**

Dillon DG, Holmes AJ, Birk JL, Brooks N, Lyons-Ruth K, Pizzagalli DA. 2009. Childhood adversity is associated with left basal ganglia dysfunction during reward anticipation in adulthood. *Biol. Psychiatry* 66(3):206–13

Dingfelder HE, Jaffee SR, Mandell DS. 2010. The impact of social support on depressive symptoms among adolescents in the child welfare system: a propensity score analysis. *Child. Youth Serv. Rev.* 32(10):1255–61

Dodge KA. 1993. Social-cognitive mechanisms in the development of conduct disorder and depression. *Annu. Rev. Psychol.* 44:559–84

Dodge KA, Pettit GS, Bates JE, Valente E. 1995. Social information-processing patterns partially mediate the effect of early physical abuse on later conduct problems. *J. Abnorm. Psychol.* 104:632–43

Duman EA, Canli T. 2015. Influence of life stress, 5-HTTLPR genotype, and *SLC6A4* methylation on gene expression and stress response in healthy Caucasian males. *Biol. Mood Anxiety Disord.* 5:2. **https://doi.org/10.1186/s13587-015-0017-x**

Dumont KA, Widom CS, Czaja SJ. 2007. Predictors of resilience in abused and neglected children grown-up: the role of individual and neighborhood characteristics. *Child Abus. Negl.* 31(3):255–74

Duncan LE, Keller MC. 2011. A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. *Am. J. Psychiatry* 168(10):1041–49

Dunlop BW, Nemeroff CB. 2007. The role of dopamine in the pathophysiology of depression. *Arch. Gen. Psychiatry* 64(3):327–37. **https://doi.org/10.1001/archpsyc.64.3.327**

During SM, McMahon RJ. 1991. Recognition of emotional facial expressions by abusive mothers and their children. *J. Clin. Child Psychol.* 20(2):132–39

Esposito CL, Clum GA. 2002. Social support and problem-solving as moderators of the relationship between childhood abuse and suicidality: applications to a delinquent population. *J. Trauma. Stress* 15(2):137–46. **https://doi.org/10.1023/A:1014860024980**

Famularo R, Kinscherff R, Fenton T. 1992. Psychiatric diagnoses of maltreated children: preliminary findings. *J. Am. Acad. Child Adolesc. Psychiatry* 31(5):863–67

Fan J, Fossella J, Sommer T, Wu Y, Posner MI. 2003. Mapping the genetic variation of executive attention onto brain activity. *PNAS* 100(12):7406–11. **https://doi.org/10.1073/pnas.0732088100**

Fani N, Bradley-Davino B, Ressler KJ, McClure-Tone EB. 2011. Attention bias in adult survivors of childhood maltreatment with and without posttraumatic stress disorder. *Cogn. Ther. Res.* 35(1):57–67. **https://doi.org/10.1007/s10608-010-9294-2**

Feiring C, Taska L, Lewis M. 2002. Adjustment following sexual abuse discovery: the role of shame and attributional style. *Dev. Psychol.* 38(1):79–92

Folger SF, Wright MO. 2013. Altering risk following child maltreatment: family and friend support as protective factors. *J. Fam. Violence* 28(4):325–37. **https://doi.org/10.1007/s10896-013-9510-4**

Fonzo GA, Ramsawh HJ, Flagan TM, Simmons AN, Sullivan SG, et al. 2016. Early life stress and the anxious brain: evidence for a neural mechanism linking childhood emotional maltreatment to anxiety in adulthood. *Psychol. Med.* 46(5):1037–54. **https://doi.org/10.1017/S0033291715002603**

Fossella J, Sommer T, Fan J, Wu Y, Swanson JM, et al. 2002. Assessing the molecular genetics of attention networks. *BMC Neurosci.* 3:14. **https://doi.org/10.1186/1471-2202-3-14**

Gagne M-H, Melancon C. 2013. Parental psychological violence and adolescent behavioral adjustment: the role of coping and social support. *J. Interpers. Violence* 28(1):176–200. **https://doi.org/10.1177/0886260512448842**

Gayer-Anderson C, Fisher HL, Fearon P, Hutchinson G, Morgan K, et al. 2015. Gender differences in the association between childhood physical and sexual abuse, social support and psychosis. *Soc. Psychiatry Psychiatr. Epidemiol.* 50(10):1489–500. **https://doi.org/10.1007/s00127-015-1058-6**

Gibb BE, Beevers CG, McGeary JE. 2013. Toward an integration of cognitive and genetic models of risk for depression. *Cogn. Emot.* 27(2):193–216

Gibb BE, Schofield CA, Coles ME. 2009. Reported history of childhood abuse and young adults' information-processing biases for facial displays of emotion. *Child Maltreat.* 14(2):148–56. **https://doi.org/10.1177/1077559508326358**

Greenberg BD, Tolliver TJ, Huang S-J, Li Q, Bengel D, Murphy DL. 1999. Genetic variation in the serotonin transporter promoter region affects serotonin uptake in human blood platelets. *Am. J. Med. Genet.* 88(1):83–87. **https://doi.org/10.1002/(SICI)1096-8628(19990205)88:1<83::AID-AJMG15>3.0.CO;2-0**

Gulley LD, Oppenheimer CW, Hankin BL. 2014. Associations among negative parenting, attention bias to anger, and social anxiety among youth. *Dev. Psychol.* 50(2):577–85. **https://doi.org/10.1037/a0033624**

Guyer AE, Kaufman J, Hodgdon HB, Masten CL, Jazbec S, et al. 2006. Behavioral alterations in reward system function: the role of childhood maltreatment and psychopathology. *J. Am. Acad. Child Adolesc. Psychiatry* 45(9):1059–67

Hankin BL, Abramson LY, Moffitt TE, Silva PA, McGee R, Angell KE. 1998. Development of depression from preadolescence to young adulthood: emerging gender differences in a 10-year longitudinal study. *J. Abnorm. Psychol.* 107(1):128–40

Hanson JL, Nacewicz BM, Sutterer MJ, Cayo AA, Schaefer SM, et al. 2015. Behavioral problems after early life stress: contributions of the hippocampus and amygdala. *Biol. Psychiatry* 77(4):314–23. **https://doi.org/10.1016/j.biopsych.2014.04.020**

Hayden EP, Olino TM, Bufferd SJ, Miller A, Dougherty LR, et al. 2013. The serotonin transporter linked polymorphic region and brain-derived neurotrophic factor valine to methionine at position 66 polymorphisms and maternal history of depression: associations with cognitive vulnerability to depression in childhood. *Dev. Psychopathol.* 25(3):587–98

Heils A, Teufel A, Petri S, Stöber G, Riederer P, et al. 2002. Allelic variation of human serotonin transporter gene expression. *J. Neurochem.* 66(6):2621–24. **https://doi.org/10.1046/j.1471-4159.1996.66062621.x**

Henry KL, Thornberry TP, Lee RD. 2015. The protective effects of intimate partner relationships on depressive symptomatology among adult parents maltreated as children. *J. Adolesc. Health* 57(2):150–56. **https://doi.org/10.1016/j.jadohealth.2015.02.015**

Herrenkohl EC, Herrenkohl RC, Egolf B. 1994. Resilient early school-age children from maltreating homes: outcomes in late adolescence. *Am. J. Orthopsychiatry* 64:301–9

Herrenkohl TI, Klika JB, Brown EC, Herrenkohl RC, Leeb RT. 2013. Tests of the mitigating effects of caring and supportive relationships in the study of abusive disciplining over two generations. *J. Adolesc. Health* 53(4):S18–24. **https://doi.org/10.1016/j.jadohealth.2013.04.009**

Holz N, Boecker R, Buchmann AF, Blomeyer D, Baumeister S, et al. 2016. Evidence for a sex-dependent *MAOA* × childhood stress interaction in the neural circuitry of aggression. *Cereb. Cortex* 26(3):904–14. **https://doi.org/10.1093/cercor/bhu249**

Horan JM, Widom CS. 2015. From childhood maltreatment to allostatic load in adulthood: the role of social support. *Child Maltreat*. 20(4):229–39. **https://doi.org/10.1177/1077559515597063**

Hu X-Z, Lipsky RH, Zhu G, Akhtar LA, Taubman J, et al. 2006. Serotonin transporter promoter gain-of-function genotypes are linked to obsessive-compulsive disorder. *Am. J. Hum. Genet.* 78(5):815–26. **https://doi.org/10.1086/503850**

Jaffee SR. 2017. Child maltreatment and risk for psychopathology. In *Child and Adolescent Psychopathology*, ed. TP Beauchaine, pp. 144–177. Hoboken, NJ: Wiley. 3rd ed.

Jaffee SR, Bowes L, Ouellet-Morin I, Fisher HL, Moffitt TE, et al. 2013. Safe, stable, nurturing relationships break the intergenerational cycle of abuse: a prospective nationally representative cohort of children in the United Kingdom. *J. Adolesc. Health* 53(4):S4–S10

Jaffee SR, Caspi A, Moffitt TE, Polo-Tomas M, Taylor A. 2007. Individual, family, and neighborhood characteristics promote resilience to physical maltreatment. *Child Abus. Negl.* 31:231–53

Jaffee SR, Caspi A, Moffitt TE, Taylor A. 2004. Physical maltreatment victim to antisocial child: evidence of an environmentally mediated process. *J. Abnorm. Psychol.* 113(1):44–55

Jaffee SR, Gallop R. 2007. Social, emotional, and academic competence among children who have had contact with Child Protective Services: prevalence and stability estimates. *J. Am. Acad. Child Adolesc. Psychiatry* 46(6):757–65

Jaffee SR, Maikovich-Fong AK. 2011. Effects of chronic maltreatment and maltreatment timing on children's behavior and cognitive abilities. *J. Child Psychol. Psychiatry* 52(2):184–94

Jaffee SR, Maikovich-Fong AK. 2013. Child maltreatment and risk for psychopathology. In *Child and Adolescent Psychopathology*, ed. TP Beauchaine, SP Hinshaw, pp. 171–96. Hoboken, NJ: Wiley. 2nd ed.

Johnson AL, Gibb BE, McGeary J. 2010. Reports of childhood physical abuse, 5-HTTLPR genotype, and women's attentional biases for angry faces. *Cogn. Ther. Res.* 34(4):380–87. **https://doi.org/10.1007/s10608-009-9269-3**

Johnson JG, Cohen P, Brown J, Smailes EM, Bernstein DP. 1999. Childhood maltreatment increases risk for personality disorders during early adulthood. *Arch. Gen. Psychiatry* 56(7):600–6

Jonson-Reid M, Presnall N, Drake B, Fox L, Bierut L, et al. 2010. Effects of child maltreatment and inherited liability on antisocial development: an official records study. *J. Am. Acad. Child Adolesc. Psychiatry* 49(4):321–32

Kaplow JB, Widom CS. 2007. Age of onset of child maltreatment predicts long-term mental health outcomes. *J. Abnorm. Child Psychol.* 116:176–87

Karg K, Burmeister M, Shedden K, Sen S. 2011. The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: evidence of genetic moderation. *Arch. Gen. Psychiatry* 68(5):444–54

Kaufman J, Cook A, Arny L, Jones B, Pittinsky T. 1994. Problems defining resiliency: illustrations from the study of maltreated children. *Dev. Psychopathol.* 6:215–29

Kaufman J, Yang B-Z, Douglas-Palumberi H, Crouse-Artus M, Lipschitz D, et al. 2007. Genetic and environmental predictors of early alcohol use. *Biol. Psychiatry* 61(11):1228–34. **https://doi.org/10.1016/j.biopsych.2006.06.039**

Kay CL, Green JM. 2016. Social cognitive deficits and biases in maltreated adolescents in UK out-of-home care: relation to disinhibited attachment disorder and psychopathology. *Dev. Psychopathol.* 28(1):73–83. **https://doi.org/10.1017/S0954579415000292**

Keil V, Price JM. 2009. Social information-processing patterns of maltreated children in two social domains. *J. Appl. Dev. Psychol.* 30(1):43–52. **https://doi.org/10.1016/j.appdev.2008.10.003**

Kelly PA, Viding E, Puetz VB, Palmer AL, Mechelli A, et al. 2015. Sex differences in socioemotional functioning, attentional bias, and gray matter volume in maltreated children: a multilevel investigation. *Dev. Psychopathol.* 27(4 Part 2):1591–609. **https://doi.org/10.1017/S0954579415000966**

Kim J, Cicchetti D. 2009. Longitudinal pathways linking child maltreatment, emotion regulation, peer relations, and psychopathology: pathways linking maltreatment, emotion regulation, and psychopathology. *J. Child Psychol. Psychiatry* 51(6):706–16. **https://doi.org/10.1111/j.1469-7610.2009.02202.x**

Klimes-Dougan B, Kistner J. 1990. Physically abused preschoolers' responses to peers' distress. *Dev. Psychol.* 26(4):599–602. **https://doi.org/10.1037/0012-1649.26.4.599**

Lamis DA, Wilson CK, King NM, Kaslow NJ. 2014. Child abuse, social support, and social functioning in African American children. *J. Fam. Violence* 29(8):881–91. **https://doi.org/10.1007/s10896-014-9639-9**

Lansford JE, Dodge KA, Pettit GS, Bates JE. 2010. Does physical abuse in early childhood predict substance use in adolescence and early adulthood? *Child Maltreat*. 15(2):190–94

Lansford JE, Dodge KA, Pettit GS, Bates JE, Crozier J, Kaplow J. 2002. Long-term effects of early child physical maltreatment on psychological, behavioral, and academic problems in adolescence: a 12-year prospective study. *Arch. Pediatr. Adolesc. Med.* 156(8):824–30

Lansford JE, Miller-Johnson S, Berlin LJ, Dodge KA, Bates JE, Pettit GS. 2007. Early physical abuse and later violent delinquency: a prospective longitudinal study. *Child Maltreat*. 12(3):233–45

Lee B-T, Ham B-J. 2008. Monoamine oxidase A–uVNTR genotype affects limbic brain activity in response to affective facial stimuli. *NeuroReport* 19(5):515–19. **https://doi.org/10.1097/WNR.0b013e3282f94294**

Leeb RT, Paulozzi L, Melanson C, Simon T, Arias I. 2008. *Child Maltreatment Surveillance: Uniform Definitions for Public Health and Recommended Data Elements*, Version 1.0. Atlanta, GA: Cent. Dis. Control Prev., Natl. Cent. Inj. Prev. Control

Lesch KP, Bengel D, Heils A, Sabol S, Greenberg B, et al. 1996. Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science* 274:1527–31

Li M, D'Arcy C, Meng X. 2016. Maltreatment in childhood substantially increases the risk of adult depression and anxiety in prospective cohort studies: systematic review, meta-analysis, and proportional attributable fractions. *Psychol. Med.* 46(4):717–30. **https://doi.org/10.1017/S0033291715002743**

Lucki I. 1998. The spectrum of behaviors influenced by serotonin. *Biol. Psychiatry* 44:151–62

Luke N, Banerjee R. 2013. Differentiated associations between childhood maltreatment experiences and social understanding: a meta-analysis and systematic review. *Dev. Rev.* 33(1):1–28. **https://doi.org/10.1016/j.dr.2012.10.001**

Luntz BK, Widom CS. 1994. Antisocial personality disorder in abused and neglected children grown up. *Am. J. Psychiatry* 151(5):670–74

Manly JT, Kim JE, Rogosch FA, Cicchetti D. 2001. Dimensions of child maltreatment and children's adjustment: contributions of developmental timing and subtype. *Dev. Psychopathol.* 13:759–82

Masten CL, Guyer AE, Hodgdon HB, McClure EB, Charney DS, et al. 2008. Recognition of facial emotions among maltreated children with high rates of post-traumatic stress disorder. *Child Abus. Negl.* 32(1):139–53. **https://doi.org/10.1016/j.chiabu.2007.09.006**

Maxfield MG, Widom CS. 1996. The cycle of violence—revisited 6 years later. *Arch. Pediatr. Adolesc. Med.* 150(4):390–95

McCrory EJ, De Brito SA, Kelly PA, Bird G, Sebastian CL, et al. 2013. Amygdala activation in maltreated children during pre-attentive emotional processing. *Br. J. Psychiatry* 202(4):269–76. **https://doi.org/10.1192/bjp.bp.112.116624**

McCrory E, De Brito SA, Sebastian CL, Mechelli A, Bird G, et al. 2011. Heightened neural reactivity to threat in child victims of family violence. *Curr. Biol.* 21(23):R947–48

McGloin JM, Widom CS. 2001. Resilience among abused and neglected children grown up. *Dev. Psychopathol.* 13:1021–38

McLaughlin KA, Peverill M, Gold AL, Alves S, Sheridan MA. 2015. Child maltreatment and neural systems underlying emotion regulation. *J. Am. Acad. Child Adolesc. Psychiatry* 54(9):753–62. **https://doi.org/10.1016/j.jaac.2015.06.010**

Mehta MA, Gore-Langton E, Golembo N, Colvert E, Williams SCR, Sonuga-Barke E. 2010. Hyporesponsive reward anticipation in the basal ganglia following severe institutional deprivation early in life. *J. Cogn. Neurosci.* 22(10):2316–25

Mersky JP, Berger LM, Reynolds AJ, Gromoske AN. 2009. Risk factors for child and adolescent maltreatment: a longitudinal investigation of a cohort of inner-city youth. *Child Maltreat.* 14(1):73–88. **https://doi.org/10.1177/1077559508318399**

Meyer-Lindenberg A, Buckholtz JW, Kolachana B, Hariri AR, Pezawas L, et al. 2006. Neural mechanisms of genetic risk for impulsivity and violence in humans. *PNAS* 103(16):6269–74

Miller AL, Gouley KK, Seifer R, Zakriski A, Eguia M, Vergnani M. 2005. Emotion knowledge skills in low-income elementary school children: associations with social status and peer experiences. *Soc. Dev.* 14(4):637–51. **https://doi.org/10.1111/j.1467-9507.2005.00321.x**

Miller R, Wankerl M, Stalder T, Kirschbaum C, Alexander N. 2013. The serotonin transporter gene-linked polymorphic region (5-HTTLPR) and cortisol stress reactivity: a meta-analysis. *Mol. Psychiatry* 18(9):1018–24

Milot T, Ethier LS, St-Laurent D, Provost MA. 2010. The role of trauma symptoms in the development of behavioral problems in maltreated preschoolers. *Child Abus. Negl.* 34(4):225–34

Miu AC, Vulturar R, Chis A, Ungureanu L, Gross JJ. 2013. Reappraisal as a mediator in the link between 5-HTTLPR and social anxiety symptoms. *Emotion* 13(6):1012–22

Moran PB, Eckenrode J. 1992. Protective personality characteristics among adolescent victims of maltreatment. *Child Abus. Negl.* 16:743–54

Moylan CA, Herrenkohl TI, Sousa C, Tajima EA, Herrenkohl RC, Russo MJ. 2010. The effects of child abuse and exposure to domestic violence on adolescent internalizing and externalizing behavior problems. *J. Fam. Violence* 25(1):53–63

Mueller A, Armbruster D, Moser DA, Canli T, Lesch KP, et al. 2011. Interaction of serotonin transporter gene-linked polymorphic region and stressful life events predicts cortisol stress response. *Neuropsychopharmacology* 36(7):1332–39

Munafo MR, Brown SM, Hariri AR. 2008. Serotonin transporter (5-HTTLPR) genotype and amygdala activation: a meta-analysis. *Biol. Psychiatry* 63(9):852–57

Munafo MR, Durrant C, Lewis G, Flint J. 2009. Gene × environment interactions at the serotonin transporter locus. *Biol. Psychiatry* 65(3):211–19

Nikulina V, Widom CS, Czaja S. 2011. The role of childhood neglect and childhood poverty in predicting mental health, academic achievement and crime in adulthood. *Am. J. Community Psychol.* 48(3–4):309–21. **https://doi.org/10.1007/s10464-010-9385-y**

Noll JG, Trickett PK, Harris WW, Putnam FW. 2009. The cumulative burden borne by offspring whose mothers were sexually abused as children: descriptive results from a multigenerational study. *J. Interpers. Violence* 24:424–49

Passamonti L, Fera F, Magariello A, Cerasa A, Gioia MC, et al. 2006. Monoamine oxidase-A genetic variations influence brain activity associated with inhibitory control: new insight into the neural correlates of impulsivity. *Biol. Psychiatry* 59(4):334–40

Pears K, Fisher PA. 2005. Developmental, cognitive, and neuropsychological functioning in preschool-aged foster children: associations with prior maltreatment and placement history. *Dev. Behav. Pediatr.* 26(2):112–22

Pechtel P, Pizzagalli DA. 2013. Disrupted reinforcement learning and maladaptive behavior in women with a history of childhood sexual abuse: a high-density event-related potential study. *JAMA Psychiatry* 70(5):499–507. **https://doi.org/10.1001/jamapsychiatry.2013.728**

Pergamin-Hight L, Bakermans-Kranenburg MJ, van IJzendoorn MH, Bar-Haim Y. 2012. Variations in the promoter region of the serotonin transporter gene and biased attention for emotional information: a meta-analysis. *Biol. Psychiatry* 71(4):373–79. **https://doi.org/10.1016/j.biopsych.2011.10.030**

Perlman SB, Kalish CW, Pollak SD. 2008. The role of maltreatment experience in children's understanding of the antecedents of emotion. *Cogn. Emot.* 22(4):651–70

Pine DS, Mogg K, Bradley BP, Montgomery L, Monk CS, et al. 2005. Attention bias to threat in maltreated children: implications for vulnerability to stress-related psychopathology. *Am. J. Psychiatry* 162(2):291–96
Pollak SD. 2003. Experience-dependent affective learning and risk for psychopathology in children. *Ann. N. Y. Acad. Sci.* 1008(1):102–11. **https://doi.org/10.1196/annals.1301.011**
Pollak SD, Cicchetti D, Hornung K, Reed A. 2000. Recognizing emotion in faces: developmental effects of child abuse and neglect. *Dev. Psychol.* 36:679–88
Pollak SD, Kistler DJ. 2002. Early experience is associated with the development of categorical representations for facial expressions of emotion. *PNAS* 99(13):9072–76. **https://doi.org/10.1073/pnas.142165999**
Pollak SD, Klorman R, Thatcher JE, Cicchetti D. 2001. P3b reflects maltreated children's reactions to facial displays of emotion. *Psychophysiology* 38(2):267–74
Pollak SD, Messner M, Kistler DJ, Cohn JF. 2009. Development of perceptual expertise in emotion recognition. *Cognition* 110(2):242–47. **https://doi.org/10.1016/j.cognition.2008.10.010**
Pollak SD, Sinha P. 2002. Effects of early experience on children's recognition of facial displays of emotion. *Dev. Psychol.* 38(5):784–91
Pollak SD, Tolley-Schell SA. 2003. Selective attention to facial emotion in physically abused children. *J. Abnorm. Psychol.* 112(3):323–38
Powers A, Ressler KJ, Bradley RG. 2009. The protective role of friendship on the effects of childhood abuse and depression. *Depress. Anxiety* 26(1):46–53. **https://doi.org/10.1002/da.20534**
Putnam FW, Helmers K, Horowitz LA. 1995. Hypnotizability and dissociativity in sexually abused girls. *Child Abus. Negl.* 19(5):645–55
Risch N, Herrell R, Lehner T, Liang KY, Eaves L, et al. 2009. Interaction between the serotonin transporter gene (*5-HTTLPR*), stressful life events, and risk of depression: a meta-analysis. *JAMA* 301(23):2462–71
Rogosch FA, Oshri A, Cicchetti D. 2010. From child maltreatment to adolescent cannabis abuse and dependence: a developmental cascade model. *Dev. Psychopathol.* 22(4):883–97. **https://doi.org/10.1017/S0954579410000520**
Runtz MG, Schallow JR. 1997. Social support and coping strategies as mediators of adult adjustment following childhood maltreatment. *Child Abus. Negl.* 21(2):211–26. **https://doi.org/10.1016/S0145-2134(96)00147-0**
Sabol S, Hu S, Hamer D. 1998. A functional polymorphism in the monoamine oxidase A gene promoter. *Hum. Genet.* 103:273–79
Salazar AM, Keller TE, Courtney ME. 2011. Understanding social support's role in the relationship between maltreatment and depression in youth with foster care experience. *Child Maltreat.* 16(2):102–13
Schofield TJ, Lee RD, Merrick MT. 2013. Safe, stable, nurturing relationships as a moderator of intergenerational continuity of child maltreatment: a meta-analysis. *J. Adolesc. Health* 53(4):S32–38. **https://doi.org/10.1016/j.jadohealth.2013.05.004**
Schumm JA, Briggs-Phillips M, Hobfoll SE. 2006. Cumulative interpersonal traumas and social support as risk and resiliency factors in predicting PTSD and depression among inner-city women. *J. Trauma. Stress* 19(6):825–36. **https://doi.org/10.1002/jts.20159**
Scott KM, Smith DR, Ellis PM. 2010. Prospectively ascertained child maltreatment and its association with DSM-IV mental disorders in young adults. *Arch. Gen. Psychiatry* 67(7):712–19
Sedlak AJ, Mettenburg J, Basena M, Petta I, McPherson K, et al. 2010. *Fourth national incidence study of child abuse and neglect* (NIS–4): Report to Congress, Executive Summary. Washington, DC: US Dept. Health Hum. Serv., Adm. Child. Fam.
Shackman JE, Fatani S, Camras LA, Berkowitz MJ, Bachorowski JA, Pollak SD. 2010. Emotion expression among abusive mothers is associated with their children's emotion processing and problem behaviours. *Cogn. Emot.* 24(8):1421–30
Shackman JE, Pollak SD. 2014. Impact of physical maltreatment on the regulation of negative affect and aggression. *Dev. Psychopathol.* 26(4 Part 1):1021–33. **https://doi.org/10.1017/S0954579414000546**
Shackman JE, Shackman AJ, Pollak SD. 2007. Physical abuse amplifies attention to threat and increases anxiety in children. *Emotion* 7(4):838–52
Shenk CE, Putnam FW, Noll JG. 2013. Predicting the accuracy of facial affect recognition: the interaction of child maltreatment and intellectual functioning. *J. Exp. Child Psychol.* 114(2):229–42. **https://doi.org/10.1016/j.jecp.2012.08.007**

Shipman KL, Schneider R, Fitzgerald MM, Sims C, Swisher L, Edwards A. 2007. Maternal emotion socialization in maltreating and non-maltreating families: implications for children's emotion regulation. *Soc. Dev.* 16(2):268–85. **https://doi.org/10.1111/j.1467-9507.2007.00384.x**

Spaccarelli S, Kim S. 1995. Resilience criteria and factors associated with resilience in sexually abused girls. *Child Abus. Negl.* 19(9):1171–82. **https://doi.org/10.1016/0145-2134(95)00077-L**

Sperry DM, Widom CS. 2013. Child abuse and neglect, social support, and psychopathology in adulthood: a prospective investigation. *Child Abus. Negl.* 37(6):415–25. **https://doi.org/10.1016/j.chiabu.2013.02.006**

Stouthamer-Loeber M, Loeber R, Homish DL, Wei E. 2001. Maltreatment of boys and the development of disruptive and delinquent behavior. *Dev. Psychopathol.* 13:941–55

Szily E, Bowen J, Unoka Z, Simon L, Keri S. 2008. Emotion appraisal is modulated by the genetic polymorphism of the serotonin transporter. *J. Neural Transm.* 115(6):819–22

Teisl M, Cicchetti D. 2008. Physical abuse, cognitive and emotional processes, and aggressive/disruptive behavior problems. *Soc. Dev.* 17(1):1–23. **https://doi.org/10.1111/j.1467-9507.2007.00412.x**

Thornberry TP, Henry KL, Ireland TO, Smith CA. 2010. The causal impact of childhood-limited maltreatment and adolescent maltreatment on early adult adjustment. *J. Adolesc. Health* 46(4):359–65

Thornberry TP, Henry KL, Smith CA, Ireland TO, Greenman SJ, Lee RD. 2013. Breaking the cycle of maltreatment: the role of safe, stable, and nurturing relationships. *J. Adolesc. Health* 53(4):S25–31. **https://doi.org/10.1016/j.jadohealth.2013.04.019**

Thornberry TP, Matsuda M, Greenman SJ, Augustyn MB, Henry KL, et al. 2014. Adolescent risk factors for child maltreatment. *Child Abus. Negl.* 38(4):706–22. **https://doi.org/10.1016/j.chiabu.2013.08.009**

Tottenham N, Hare TA, Millner A, Gilhooly T, Zevin JD, Casey BJ. 2011. Elevated amygdala response to faces following early deprivation. *Dev. Sci.* 14(2):190–204. **https://doi.org/10.1111/j.1467-7687.2010.00971.x**

Tremblay C, Hébert M, Piché C. 1999. Coping strategies and social support as mediators of consequences in child sexual abuse victims. *Child Abus. Negl.* 23(9):929–45. **https://doi.org/10.1016/S0145-2134(99)00056-3**

Troller-Renfree S, McDermott JM, Nelson CA, Zeanah CH, Fox NA. 2015. The effects of early foster care intervention on attention biases in previously institutionalized children in Romania. *Dev. Sci.* 18(5):713–22. **https://doi.org/10.1111/desc.12261**

US Dep. Health Hum. Serv., Adm. Child. Fam., ACF (Adm. Child. Youth Fam.), Child. Bur. 2016. *Child Maltreatment 2014*. Washington, DC: US Dep. Health Hum. Serv. **http://www.acf.hhs.gov/programs/cb/research-data-technology/statistics-research/child-maltreatment**

van Harmelen A-L, van Tol M-J, Demenescu LR, van der Wee NJA, Veltman DJ, et al. 2013. Enhanced amygdala reactivity to emotional faces in adults reporting childhood emotional maltreatment. *Soc. Cogn. Affect. Neurosci.* 8(4):362–69. **https://doi.org/10.1093/scan/nss007**

Varese F, Smeets F, Drukker M, Lieverse R, Lataster T, et al. 2012. Childhood adversities increase the risk of psychosis: a meta-analysis of patient-control, prospective- and cross-sectional cohort studies. *Schizophr. Bull.* 38(4):661–71. **https://doi.org/10.1093/schbul/sbs050**

Vranceanu A-M, Hobfoll SE, Johnson RJ. 2007. Child multi-type maltreatment and associated depression and PTSD symptoms: the role of social support and stress. *Child Abus. Negl.* 31(1):71–84. **https://doi.org/10.1016/j.chiabu.2006.04.010**

Wagner MF, Milner JS, McCarthy RJ, Crouch JL, McCanne TR, Skowronski JJ. 2015. Facial emotion recognition accuracy and child physical abuse: an experiment and a meta-analysis. *Psychol. Violence* 5(2):154–62. **https://doi.org/10.1037/a0036014**

Wankerl M, Miller R, Kirschbaum C, Hennig J, Stalder T, Alexander N. 2014. Effects of genetic and early environmental risk factors for depression on serotonin transporter expression and methylation profiles. *Transl. Psychiatry* 4(6):e402. **https://doi.org/10.1038/tp.2014.37**

Widom C. 1989a. Does violence beget violence? A critical examination of the literature. *Psychol. Bull.* 106(1):3–28. **https://doi.org/10.1037/0033-2909.115.2.287**

Widom C. 1989b. The cycle of violence. *Science* 244(4901):160–66

Widom C. 1999. Posttraumatic stress disorder in abused and neglected children grown up. *Am. J. Psychiatry* 156(8):1223–29. **https://doi.org/10.1176/ajp.156.8.1223**

Widom C, Czaja SJ, Paris J. 2009. A prospective investigation of borderline personality disorder in abused and neglected children followed up into adulthood. *J. Pers. Disord.* 23(5):433–46

Widom C, DuMont K, Czaja S. 2007. A prospective investigation of major depressive disorder and comorbidity in abused and neglected children grown up. *Arch. Gen. Psychiatry* 64(1):49–56. **https://doi.org/10.1001/archpsyc.64.1.49**

Widom C, Ireland T, Glynn PJ. 1995. Alcohol abuse in abused and neglected children followed-up: Are they at increased risk? *J. Stud. Alcohol* 56(2):207–17. **https://doi.org/10.15288/jsa.1995.56.207**

Widom C, Marmorstein NR, Raskin White H. 2006. Childhood victimization and illicit drug use in middle adulthood. *Psychol. Addict. Behav.* 20(4):394–403. **https://doi.org/10.1037/0893-164X.20.4.394**

Widom CS, Czaja S. 2012. Childhood trauma, psychopathology, and violence: disentangling causes, consequences, and correlates. In *Trauma, Psychopathology, and Violence: Causes, Consequences, or Correlates*? ed. CS Widom, pp. 291–317. Oxford, UK: Oxford Univ. Press

Widom CS, Czaja SJ, DuMont KA. 2015. Intergenerational transmission of child abuse and neglect: real or detection bias? *Science* 347(6229):1480–85. **https://doi.org/10.1126/science.1259917**

Widom CS, Morris S. 1997. Accuracy of adult recollections of childhood victimization, part 2: childhood sexual abuse. *Psychol. Assess.* 9(1):34–46. **https://doi.org/10.1037/1040-3590.9.1.34**

Widom CS, Weiler BL, Cottler LB. 1999. Childhood victimization and drug abuse: a comparison of prospective and retrospective findings. *J. Consult. Clin. Psychol.* 67(6):867–80. **https://doi.org/10.1037/0022-006X.67.6.867**

Wilhelm K, Siegel JE, Finch AW, Hadzi-Pavlovic D, Mitchell PB, et al. 2007. The long and short of it: associations between *5-HTT* genotypes and coping with stress. *Psychosom. Med.* 69(7):614–20. **https://doi.org/10.1097/PSY/.0b013e31814cec64**

Williams JH, Van Dorn RA, Bright CL, Jonson-Reid M, Nebbitt VE. 2010. Child maltreatment and delinquency onset among African American adolescent males. *Res. Soc. Work Pract.* 20(3):253–59

Williams LM, Gatt JM, Schofield PR, Olivieri G, Peduto A, Gordon E. 2009. "Negativity bias" in risk for depression and anxiety: brain–body fear circuitry correlates, 5-HTTLPR and early life stress. *NeuroImage* 47(3):804–14. **https://doi.org/10.1016/j.neuroimage.2009.05.009**

Yates TM, Carlson EA, Egeland B. 2008. A prospective study of child maltreatment and self-injurious behavior in a community sample. *Dev. Psychopathol.* 20(2):651–71. **https://doi.org/10.1017/S0954579408000321**