CHAPTER 13

# The Effects of Violent Experiences on Infants and Young Children

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There is no question that experiences of violence and maltreatment adversely and enduringly alter neurobiological development, psychological and social functioning, and subsequent expectations of the environment (Enlow, Egeland, Blood, Wright, & Wright, 2012; Kaufman, Plotsky, Nemeroff, & Charney, 2000; Udo, Sharps, Bronner, & Hossain, 2016). The more numerous, frequent, and enduring the exposure(s), the greater the number of behavioral and developmental difficulties children experience (Clarkson Freeman, 2014). The questions that now require our attention are rather to what degree, and in what way, is early development affected by exposure to violence? Which effects follow from specific types of events and the frequency of exposures to those events? What is the impact of a specific individual infant's or child's constitution? Is there a differential impact of adverse events dependent upon specific critical periods of development? What is the effect of the exposure in the context of specific relationships in which the meaning of the experience(s) is co-constructed? These questions are critical to the clinical assessment and effective treatment of the sequelae of violent experiences during early childhood and subsequently.

Following an overview of pertinent epidemiology regarding the scope of early childhood exposure to violence, we discuss the nature of the most prevalent types of traumatic exposure and the known sequelae. We continue with a review of select factors within the developmental context that impact the manner in which traumatic violence is experienced, including the construct of attachment. We then turn to neurobiology, namely, the mechanics of the stress response system, individual difference and gene–environment interaction, and current research findings on the relational neurobiology of violence exposure. The nature and sequelae of exposure, the developmental context of traumatic experiences, and neurobiological changes are all inextricably interwoven, although we consider each in turn. The metaphor of looking through a crystal, such that regardless of which side one gazes into, the other sides are all reflected back, seems apt when considering the effects of violence on very young children.

## Epidemiology of Exposure to Violence

In many ways, the past two decades across the globe have included a host of unfortunate naturalistic case studies in the multiple modalities by which children may be exposed to violence. Children can be the victims of, or witnesses to, interpersonal, familial, community, and international violence. They also can be witnesses many times over given the nearly universal ac-

cess to smartphones and Internet-transmitted media. We begin this section with the available data on U.S. national exposure rates, with particular attention to the experience of very young children, and continue to global statistics regarding exposure to international terror and violence.

According to the *Child Maltreatment 2014* report of the U.S. Department of Health and Human Services (2014), approximately 3.2 million children were the subject of CPS investigations nationally, an increase of 7.4% from 2010. From these reports, the total estimated number of children determined to be victims of abuse or neglect was 702,000, a rate of 9.4 per 100,000 same-age children in the general population. Overall, 75% of child maltreatment victims suffered neglect, 17% physical abuse, and 8.3% sexual abuse. Over half of the victims (58.7%) were 7 years old or younger, with 27.4% of the victims younger than 3 years of age. The highest victimization rates were found for children under the age of 1 year at 24.4% per 100,000 same-age children. This was an increase of 2.7% from 2010, and the largest increase for any age group over the 5 years analyzed. The rates for children in age groups 1–5, 6–10, and 11–17 remained largely consistent between 2010 and 2014.

For 2014, the estimate of child fatalities in the United States was 1,580. Approximately 70% of all child fatalities were children younger than 3 years of age. Consistent with risk being greatest for the youngest children, the rate of child fatalities under the age of 1 year was 17.96 per 100,000 same-age children, whereas child fatalities at 1 year of age was 6.51 per 100,000 same-age children. In comparison to the overall estimated rate of 2.17 deaths per 100,000 children, infant boys under the age of 1 year had a fatality rate of 20.23, while infant girls under the age of 1 year had a rate of 15.39 deaths. In 80% of 2014 child maltreatment cases, the parent was the abuser.

The National Survey of Children's Exposure to Violence (NatSCEV), supported by the U.S. Department of Justice, Office of Justice Programs, Office of Juvenile Justice and Delinquency Prevention (OJJDP) and the Centers for Disease Control and Prevention (CDC), was first conducted in 2008 (NatSCEV I: Finkelhor & Turner, 2016a), repeated in 2011 (NatSCEV II: Finkelhor & Turner, 2016b), and repeated again in 2014 (NatSCEV III: Finkelhor & Turner, 2016c). This comprehensive survey measures the national incidence and prevalence of multiple forms of violence exposure for children from birth to age 17 within the past year, and over the lifetime of the child. NatSCEV II (2011) expanded the range of violent acts, as well as forms of violence exposure for children, affording the most detailed picture of child violence exposure in the United States to date (Finkelhor, Turner, Shattuck, Hamby, & Kracke, 2015).

The NatSCEV III survey found that in the "past year," 67.5% of children had exposure to at least one type of direct, indirect, or witnessed violence (Finkelhor, Turner, Shattuck, & Hamby, 2015). The types of violence were physical assault, sexual victimization, maltreatment, property victimization, or witnessing family or community violence. Narrowing to past year direct exposure only, 60.8% of children had at least one direct experience, and 40.9% had more than one direct experience of abuse, violence, or crime. Nearly one-fourth of children (24.5%) had witnessed violence in their family or community over the past year.

Addressing violence exposure specifically within the home, the NatSCEV II survey investigated three categories of exposure: "psychological/emotional intimate partner violence" (IPV), including verbal threats and displaced aggression (breaking, punching, hitting something in the home rather than a person); exposure to "physical IPV"; and exposure to "any family violence," such as parental assault of a sibling or other family members engaging in violence within the home (Hamby, Finkelhor, Turner, & Ormrod, 2011). Within a national representative sample of 4,549 youth (birth to age 17), exposure to any of these three categories in the past year was 1 in 9 (11.1%), with a lifetime exposure rate of 1 in 4 (25.6%). When extrapolated to the national population, the data from this survey suggest that an estimated 8.2 million children were exposed to family violence in one year alone, with a higher lifetime exposure estimate of 18.8 million children. It is important to note that the 2011 NatSCEV survey found that 90% of children exposed to physical IPV were direct eyewitnesses, and that 76% of children exposed to other types of family violence were direct eyewitnesses. Given the estimate of 18.8 million children with lifetime exposure, the impact of violence within the home cannot be understated.

For children under the age of 5 years who were exposed to IPV ($N$ = 1,458), lifetime ex-

posure rates as reported by primary caregivers were the following: 5.4% exposed to verbal threats; 11.5% exposed to displaced aggression; 7.1% eyewitness to the assault of a parent; 10.5% exposed to a parent being pushed; 8.2% exposed to a parent being hit or slapped; and 4.6% exposed to the severe physical assault of a parent, such as being kicked, choked, or beaten up. For children under 5 years of age, lifetime exposure across all three categories of violence in the home was 17.2%.

Overall survey data from NatSCEV I found that more than 33.9% of all children who witnessed IPV were also maltreated in the past year, with a lifetime co-occurrence rate of 56.8% (Hamby, Finkelhor, Turner, & Ormrod, 2010). This is consistent with previous findings of a median co-occurrence rate of 41% (Appel & Holden, 1998) and 30–60% (Edleson, 1999).

In order to examine not only the frequency with which children witness interpersonal violence but also their age and level of sensory exposure, Fantuzzo and Fusco (2007) worked with a large Northeastern county police department to collect exposure data. As assessed by the responding police officers, children were present for 43% of domestic violence episodes, 92% of which involved violence against the children's mother. The authors reported that 81% of the children present either heard and/or saw the event, and that 60% of these directly exposed children were younger than 6 years old. The authors replicated their 2007 study 2 years later (Fusco & Fantuzzo, 2009). As assessed by the responding police officers, children were again present for 43% of domestic violence episodes, 94% of which involved violence against the children's mother; 86% of children heard and/or saw the event. The mean age of children in the sample was 4.8 years. Given the extrapolated national estimates for family violence exposure for all children and the specific exposure rates for young children, the immense adverse implications for young children are clear and sobering.

The World Trade Center attacks in 2001, and the start of the Iraq war in 2003, brought home the impact of terrorism and war for a generation of American children. However, since the publication of the third edition of the *Handbook of Infant Mental Health* in 2009, either directly or via media coverage, children around the world have been exposed to the horrors of the Syrian conflict and the brutality of terrorist attacks by groups such as Al-Qaeda, ISIL (Daesh), Hamas, and Boko Haram. In addition, there has been a relentless series of ideological "homegrown" terrorist violent events in places such as Boston, Orlando, Dallas, Louisiana, Baghdad, Istanbul, Nice, Paris, and Munich. The world's children have suffered the effects of terrorism, war-related physical and emotional injury, dislocation, and refugee related trauma.

The United Nations International Children's Emergency Fund (UNICEF; 2016) estimated that at the time of their 2016 report, approximately 28 million children had been forcibly displaced: 17 million children displaced within their country of origin due to conflict and violence, 1 million children seeking asylum in a foreign country, and 10 million refugee children. The decade 2005–2015 saw the doubling of child refugees under the protection of the United Nations High Commissioner for Refugees (UNHCR; 2015) from 4 million to over 8 million children. In 2015 alone, children comprised 31% of the total refugee arrivals in the European Union, many unaccompanied or separated. Unaccompanied children are particularly vulnerable in that they are challenged both by the adverse events they have experienced in the past and the demands of adjustment to an entirely new environment (Reed, Fazel, Jones, Panter-Brick, & Stein, 2012).

The media and the Internet represent additional forms of violence exposure that are increasingly interesting and important. Inability to distinguish media presentations from real and/or personal experience at an early age also raises questions in terms of the complexity of traumatic exposure for very young children. Behavioral effects of violent media on preschool-age children have been known for many years (Stein & Friedrich, 1972). Leiner and colleagues (2016) note that multiple studies have shown associations between media exposure to terrorist acts and adverse mental health effects in children. Recent studies of families with young children have demonstrated an association between familial preference for viewing violent media and history of actual and/or current traumatization in the primary caregiver who allows and/or promotes the violent media viewing (Schechter et al., 2009).

## Nature of the Exposure

Exposure to a traumatic event is generally defined in terms of proximity to the event, degree

of injury or exposure to injury and/or loss of life, as well as perceived threat of injury and/or loss of life. Since infants and very young children have a limited capacity to judge threat and rely on their caregivers for survival, exposure becomes a more complex issue for this age group. However, the immediate and longitudinal effects of violent trauma and maltreatment on psychopathology in infants and young children are well documented. A common finding regardless of age group is the association between violent trauma and risk for a broad range of resultant psychiatric conditions covering domains of functioning as varied as sleeping, feeding, elimination, depression, anxiety, mood, somatization, behavior, attentional regulation, language development, dissociative processes, self-endangering behaviors, and numerous autoimmune and other medical conditions (Driessen, Schroeder, Widmann, von Schonfeld, & Schneider, 2006; Dube et al., 2001; Harpur, Polek, & van Harmelen, 2015; Seng, Graham-Bermann, Clark, McCarthy, & Ronis, 2005; Sugaya et al., 2012; Wing, Gjelsvik, Nocera, & McQuaid, 2015).

Although child maltreatment may vary in terms of type, severity, developmental stage, perpetrator, and chronicity, the deleterious effects have been conclusively established (Cicchetti & Lynch, 1995). For example, maltreated children generally exhibit greater internalizing and externalizing problems, lower levels of ego resiliency, and greater ego undercontrol. Maltreated children abused only in infancy and toddlerhood have been shown to exhibit specific negative sequelae during middle childhood, such as greater externalizing symptoms and greater perceived aggression than same-age nonmaltreated peers (Manly, Kim, Rogosch, & Cicchetti, 2001). Among a sample of adolescents with a history of maltreatment, Dunn, McLaughlin, Slopen, Rosand, and Smoller (2013) found that maltreatment during early childhood (first five years of life) was most strongly associated with depression and suicidal ideation in young adulthood as compared to abuse that occurred during adolescence. Interpersonal trauma exposure (physical, sexual, emotional abuse or neglect, witnessing IPV) between birth and age 5 years was significantly associated with decreased cognitive scores at 24, 64, and 96 months of age (Enlow et al., 2012).

When the caregivers on whom the infant or young child is dependent are themselves the source of threat, such as in the instances of maltreatment and family violence, profound effects are noted in virtually every area of subsequent development, including fundamental disturbances of relatedness (Cicchetti, Toth, Bush, & Gillespie, 1988). Maltreated infants as young as 15 months have demonstrated hyperresponsivity to angry facial affect as compared to happy facial affect (Curtis & Cicchetti, 2013). Maltreatment has been associated with language delay in vocabulary, production of syntactic structures, as well as internal state language (Beeghly & Cicchetti, 1994; Eigsti & Cicchetti, 2004). Using a child welfare dataset, Clarkson Freeman (2014) examined the association between adverse childhood experiences (ACEs) in children from birth to 6 years of age and later behavioral health outcomes. Approximately 70% of the sample children had experienced three or more ACEs by 6 years of age. Results suggested a dose–response effect. At 4–8 years following the close of the investigation, a history of three or more ACEs more than quadrupled the risk of internalizing problems and nearly quadrupled the risk of externalizing or total problems.

Witnessing IPV in childhood is linked to a wide range of pernicious outcomes. In a meta-analysis of over 100 studies of child witnesses to domestic violence, Kitzmann, Gaylord, Holt, and Kenny (2003) concluded that in childhood, there is a significant association between exposure and behavioral, social, and academic problems. IPV in childhood has been associated with conduct disorder during childhood and adolescence (Jouriles et al., 2016) and adult aggression in intimate relationships (Ehrensaft et al., 2003). Early childhood cognitive functioning has been shown to be impacted by exposure to IPV at 30 months of age, significantly predicting children's short-term, working, and deliberate memory capacities at 60 months of age (Gustafsson et al., 2013). Severity of maternal domestic violence experienced during pregnancy has been found to be associated with an increased risk of language and neurological delays in infants and toddlers (Udo et al., 2016). Preschool witnesses of IPV have been shown to suffer disturbances in multiple domains with severe internalizing and externalizing symptoms (Lieberman, van Horn, & Ozer, 2005).

Levendosky, Leahy, Bogat, Davidson, and von Eye (2006; see also Levendosky, Bogat, & Martinez-Torteya, 2013) have shown that infants exhibit posttraumatic stress symptoms

and externalizing behaviors (aggression, negative emotional reactivity, activity level) following exposure to IPV, and with mothers suffering from posttraumatic stress disorder (PTSD). Consistent with Scheeringa and Zeanah's (2001) model of relational PTSD, maternal functioning can serve as a mediator and/or moderator between domestic violence and infant externalizing behaviors, with maternal regulation either inhibiting or promoting infant regulation and resilience. Ahlfs-Dunn and Huth-Bocks (2014) found that infants whose mothers experienced IPV during the child's first year of life exhibited greater socioemotional problems at 12 months of age. Furthermore, maternal posttraumatic hyperarousal and reexperiencing symptoms served as moderators of the association between IPV in the child's first year of life and socioemotional problems at 12 months.

Terrorism and war erode the safety and predictability of a young child's world. In a study of 148 war-exposed children ages 1.5 to 5 years living in the vicinity of the Gaza Strip as compared to 84 controls, 37.8% were found to meet criteria for PTSD, with 60% of those 56 war-exposed children suffering from significant developmental regression (Feldman & Vengrober, 2011). Not surprisingly, in that same study, mothers of the traumatized children were found to have the highest rates of anxiety, depression, and PTSD, the least sensitive caregiving behavior, and the lowest social support, thus echoing other studies (Slone & Mann, 2016).

As supported by the Feldman and Vengrober (2011) study, critical variables are degree of exposure, amount of family support during and after, impact on primary caregivers, degree of life disruption, and degree of social chaos. In studies of direct exposure, a dose–response effect is found with greater exposure resulting in more severe risk of PTSD (Pine, Costello, & Masten, 2005). For preschoolers living in war zones, higher levels of traumatic exposure are related to severity of behavioral and emotional symptoms (Thabet, Karim, & Vostanis, 2006). Indirect exposure appears to have a more deleterious effect on children with prior trauma but does not necessarily result in PTSD for most children (Pfefferbaum et al., 2003). For young children, indirect exposure does, however, create an atmosphere and perception of danger, which can induce separation anxiety, new fears, and avoidant behaviors (Pynoos, Schreiber, Steinberg, & Pfefferbaum, 2005). Wang and colleagues (2006) found that 1- to 4-year-olds in Israel demonstrated differential patterns of association depending on type of trauma exposure: direct exposure to terrorism, media exposure to terrorism, and other traumas. Direct exposure was significantly associated with an increased risk of externalizing and internalizing problems. Exposure to television coverage for a minimum of 5 minutes daily or more was associated with a greater risk of emotional reactivity and sleep problems, as well as some externalizing and aggressive behaviors. Nonterrorism-related trauma (e.g., motor vehicle accidents, other accidents, dog bites, medical–surgical trauma) produced greater anxiety and other internalizing symptoms without notable externalizing symptoms.

For children who are forcibly displaced, each element of the migration path (premigration conflict-related violence, migration dislocation and loss, postmigration adaptation) may include a great number of traumatic events that put both children and adults at high risk for psychopathology, particularly PTSD, anxiety, and depression (Fasfous, Peralta-Ramirez, & Perez-Garcia, 2013; Kirmayer et al., 2011). Rates of PTSD have been found to be higher among displaced people living in camps than among the general population. Out of 14,000 displaced persons living in a Syrian refugee camp, 683 out of 820 (83.5%) randomly selected refugees met clinical criteria for PTSD (Acarturk et al., 2015). For children, postmigration adjustment depends on not only their primary experience but also the psychological adjustment of their parents (Panter-Brick, Grimon, & Eggerman, 2014). Thus, providing adequate support for refugee caregivers is critical to mitigating the effects of war and displacement on children.

A recent systematic review, comprising 35 studies that included a total of 4,365 young children, examined the effects of exposure to war, conflict, and terrorism on young children and the influence of parental factors on these effects (Slone & Mann, 2016). The review divided these studies into those in which there had been (1) media exposure; (2) personal exposure, such as witnessing firsthand; (3) severe personal exposure involving a sense of direct threat, with or without injury; (4) a single episode (i.e., the 9/11 attacks); (5) an acute period (i.e., the Gulf War); and (6) chronic ongoing exposure (i.e., the Israeli–Palestinian conflict). Results showed that effects include PTSD and posttraumatic stress symptoms, behavioral and emotional symptoms, sleep problems, disturbed

play, and psychosomatic symptoms. Correlations emerged between parental and children's psychopathology. Family environment and parental functioning further emerged as moderators of the exposure–outcome association for children.

Among young children who develop PTSD, a substantial number also have comorbid psychopathology, including oppositional defiant disorder and other anxiety disorders (Scheeringa & Zeanah, 2008; Scheeringa, Zeanah, Myers, & Putnam, 2003). Some traumatized preschool children develop no discernable psychopathology, yet they may display more subtle or subthreshold difficulties that defy presently used diagnostic categories. The nature of exposure, gene–environment interaction, developmental factors, and attachment relationships may steer the individual victim of violent trauma down multiple pathways of psychopathology without obvious PTSD.

PTSD diagnostic rates may actually be deceptively low in childhood and particularly in early childhood for several reasons. A challenging problem is that of the very young child meeting the DSM-5 (American Psychiatric Association, 2013) traumatic event criterion. The meaning of a given experience as "traumatic" may not be understood as such by infants and very young children, even though anxiety is generated in response to the sense of traumatization by caregivers and/or others in the environment. This is to say, infants and toddlers do not often have the capacity to accurately appraise threat and consequences of traumatic exposures, so they depend on their caregivers for this appraisal.

Additionally, the linking of PTSD symptoms temporally to the occurrence of a violent event is required for the diagnosis of PTSD. For many infants and preverbal children, this temporal link will not be possible to make. For example, a foster child may present with symptoms of full-blown PTSD but without any known record of clear physical or sexual abuse. In such cases, one may infer PTSD or rule out PTSD pending more information. Regardless of the age of the child, a discernable and enduring or frequently recurring change of behavior that is associated with impairment and/or distress following an index event, or coinciding with a prolonged exposure (i.e., to domestic violence or maltreatment), is the most likely indicator of need for evaluation of psychopathology, regardless of whether the behaviors or symptoms fit neatly into any particular diagnostic category.

## Developmental Context

In describing the effects of trauma on development, Fischer and colleagues (1997, p. 749) have stated that "contrary to the standard assumption that psychopathology stems from developmental immaturity," psychopathology is actually a form of "adaptation" to trauma, with the individual deviating from normative developmental frameworks. With respect to maltreatment, Fischer and colleagues note that children who are victims of maltreatment have normal developmental complexity but distinctive affective–cognitive organization, with specific features, such as negative attribution biases in play, and representations of interactions. Similarly, Cote, Vaillancourt, Barker, Nagin, and Tremblay (2007) have shown that hostile parenting interferes with the redirection of normative aggression to socially acceptable behavior and is significantly associated with higher levels of interpersonal difficulties, including hostile peer-directed aggression persisting beyond 2 years of age.

Development has also been shown to impact the risk for maltreatment (Horner-Johnson & Drum, 2006), as well as the expression of the effects of maltreatment and violent trauma by virtue of its being delayed or otherwise fundamentally or pervasively disturbed (Turk, Robbins, & Woodhead, 2005). Just as the preverbal infant has been shown to express adverse effects of violent traumatization, the language-delayed or disturbed-preschooler also has been shown to display behavioral signs of violent traumatization to the trained clinician (Cook, Kieffer, Charak, & Leventhal, 1993; Turk et al., 2005).

It is therefore not only what happened and when it happened to the young child that determines long-term outcome, it matters quite clearly who that infant is, constitutionally, in terms of how effects will manifest. We now address several developmental factors that impact the shaping of what constitutes a traumatic exposure for the very young child: perception as a function of development, the developing capacity for memory, and the parent–child attachment/relational context.

### *Perception*

In reviewing the language of DSM-5, one can appreciate that infants and young children may not be aware that an event has posed to them

"actual or threatened death, serious injury, or sexual violence," even though most children are exposed to one or more of these events from infancy onward (Copeland, Keeler, Angold, & Costello, 2007; Costello, Erklani, Fairbank, & Angold, 2002). Similarly, an infant or toddler might perceive an event as life threatening that would not be life threatening to an older child or adult, for example, a parent pushing another parent violently during an argument or a sibling pushing the toddler down in a bathtub, resulting in the child's head being briefly submerged in very shallow water. Rather, very young children may show fear for other reasons. As Eth and Pynoos (1994) have described in reviewing cases of children who have witnessed the homicide of a parent, young children are more likely to find an unanticipated aspect most disturbing, such that the clinician needs to ask in an open-ended way what was scary to the child. For example, the removal of the parent's corpse by strangers in an ambulance may be more disturbing than the actual murder.

Additionally, concepts of human malevolence and death are not fathomable to children in the first 3 years of life, but emerge between ages 3 and 5 years (Barrett & Behne, 2005). A man wielding a gun in the near vicinity may not be inherently frightening to the infant or toddler. However, beginning with the developmental achievement of secondary intersubjectivity at approximately 8–10 months, infants are able to experience the fear felt by others around them and become frightened. Even infants younger than 8 months are able to sense their caregivers' fear and hyperarousal, and may become distressed in response to the reaction of the caregiver to the impending trauma rather than to the threat itself.

By 18 months, as toddlers develop increased representational capacity that allows them to compare mental representations of expected appearances and behaviors based on prior relational experience with new input, they attain greater capacity to appreciate, reenact, and remember real-life experiences, including a potential threat to themselves and their caregivers (Lukowski et al., 2005; Simcock & DeLoache, 2006). Scheeringa and Zeanah (1995) found that the most powerful trauma factor for young children following exposure to a traumatic event was threat to the caregiver. This was replicated in a study of children one to 18 years old (Scheeringa, Wright, Hunt, & Zeanah, 2006).

### *Memory*

The psychological and neurobiological implications of exposure to traumatic events also involve infants' and young children's developmentally determined capacity to encode, remember, and recall those events in order to subsequently make meaning of their experience. Recent evidence suggests that even prior to 1 year of age, infants' capacity to recall events is well under way. By the end of the second year of life, long-term memory is reliably and clearly present, especially when there have been reinforcing memories (i.e., repeated exposures or explicit reminders) that unfortunately are all too common in cases of maltreatment and family violence (Bauer, 2006; Hartshorn & Rovee-Collier, 2003). Based on her review of the literature, Fivush (1998) has written that traumatic events perceived before the age of 18 months are frequently not verbally accessible, whereas events experienced between ages 18 and 36 months can often be coherently recounted and retained as long-term memories.

Two important case studies have raised the issue of memory and recall in infancy and early childhood. Two young children endured severe direct exposure to the murder of their primary caregiver, one at 12 months (Gaensbauer, Chatoor, Drell, Siegel, & Zeanah, 1995) and the other at 19 months (Kaplow, Saxe, Putnam, Pynoos, & Lieberman, 2006). Both cases suggest that under such extreme circumstances, traumatic memories are encoded in detail and consolidated, although they may not yet be readily accessible to verbal narrative memory. Such individuals with early exposure remain vulnerable to triggers of their traumatic memories. As is found in many children who were maltreated prior to 2 and 3 years of age, they may display difficulty in regulating their emotional responses when confronted with high degrees of negative emotion in themselves or others. They may also have difficulty in developing coherent, balanced self-representations, consistent behavioral organization, and trust leading to the development of new relationships (Hartman & Burgess, 1989).

Early chronic and/or severe exposure to violence and/or maltreatment has also been noted to lead to greater pervasive insult to memory functions and to promote dissociative processes that can interfere with memory retrieval (Howe, Cicchetti, & Toth, 2006; Nelson & Carver, 1998). One mechanism for this biological in-

sult to memory function is thought to be the effect primarily of an excess of glucocorticoids damaging the developing structures involved in memory contextualization and storage, such as the hippocampus (Sapolsky, 2000; Sapolsky, Uno, Rebert, & Finch, 1990). It is clear that over the course of formative development, exposure to violent trauma and maltreatment can affect the degree and nature of changes in the neurobiology of the brain.

### *Parent–Child Relationship*

In support of the notion that exposure is extensively shaped by relational experience, Freud and Burlingham (1943) said the following about young children's experience during the London Blitz in World War II:

> The war acquires comparatively little significance for children so long as it only threatens their lives, disturbs their material comfort, or cuts their food rations.
>
> It becomes enormously significant the moment it breaks up family life and uproots the first emotional attachments of the child within the family group. London children, therefore were on the whole much less upset by bombing than by evacuation to the country as a protection from it. (p. 37)

The violent traumatization of an infant or very young child, whether due to maltreatment or exposure to family, community, war, or terroristic violence, is most significantly a breach in safety. Unlike older children or adults, very young children experience their world contextually, from within the embrace of the primary attachment relationship. Their sense and expectation of safety is therefore inherently bound to the caregiver. To appreciate the effects of violence on young children requires an understanding of the goals and mechanisms involved in the attachment relationship, as well as the ways in which trauma impacts attachment.

The anchoring concept of attachment theory is the ethological wisdom of a caregiver–infant behavioral system that ensures species survival (Bowlby, 1969). The infant's drive to maintain safety is paramount and is expressed in attachment behaviors that may phenotypically change over time but serve the same purposeful goal of achieving "felt security." Perturbations in the infant's ability to achieve felt-security necessarily result in adaptations that may be more or less pernicious depending on the quality and degree of frustration. Based on the primary attachment figure's track record of providing felt security, the infant constructs an "internal working model" of self and other. This internal representation consolidates over the first 3 years of life and guides the infant's expectations and behaviors in times of stress.

The experience of violence with its attendant physiological "felt anxiety" might therefore be conceptualized as the exact affective opposite of felt security. The young child does not yet have the cognitive ability to mediate feelings of fear that result when exposed to violence, either as victim or witness. For young children, the caregiver's role is to function as external regulator of negative or overwhelming internal affect and sensation. Several scenarios involving violence may be imagined in which the caregiver is unavailable to soothe infant anxiety: when the caregiver is being victimized, when the caregiver is a witness to violence and becomes too hyperaroused or too dissociated/avoidant to provide safety, or when the caregiver is the source of the violence—as in the case of parental child abuse. A toddler who has internalized a working model in which he or she is unprotected and repeatedly subjected to overwhelming fear—one of the definitional criterion for trauma—may develop what has been termed distortions in secure-base behavior (Lieberman & Pawl, 1990). Such distortions are in fact attempts by the child to manage unmanageable anxiety without the actual or internalized assistance of the caregiver.

If early childhood is characterized by this relational context in which the child's ability to manage stress is determined by caregiver response, then the mental health status of the caregiver becomes a vital concern. Fraiberg, Adelman, and Shapiro (1975) called attention to the profound effects of maternal mental health on the developing child. The *ghosts in the nursery* that they described were malevolent internalized attachment figures who had subjected the caregiver to various forms of maltreatment during his or her own childhood. Fraiberg and colleagues observed that caregiver traumatization in the past resulted in the caregiver's present-day inability to respond appropriately to infant anxiety, or engagement in behavior that actually induced anxiety. From an attachment perspective, the infant's "working model" of self and other is thereby shaped, impacted by the caregiver's disturbed attachment representations.

Exploring representational models, Fonagy Moran, Steele, Steele, and Higgitt (1991; see

also Fonagy, Steele, Moran, Steele, & Higgitt, 1993) identified the capacity for "reflective functioning," the awareness of a meaningful relationship between underlying mental states (feelings, thoughts, motivations, intentions) and behavior in and between both self and others. Fonagy's group found that caregiver reflective functioning was significantly predictive of infant attachment classification. The caregiver's capacity to read infant mental states accurately, and with inference of meaning, allows for sensitively attuned responses that create a subjective experience of security/safety and support the infant's developing capacity for self-regulation (Bretherton & Munholland, 1999). However, when engaging in reflective functioning leads to the experiencing of highly negative affect, certain aspects of mental functioning may be defensively inhibited (Fonagy, Steele, Steele, Higgitt, & Target, 1994) or excluded (Bretherton, 1990). Indeed, a distressed young child can activate maternal mental representations and affect linked to traumatic memory traces (Schechter et al., 2005, 2006). A caregiver in a state of defensive inhibition will be incapable of accurately responding to and reflecting the child's mental state, leaving the child to manage states of arousal and anxiety on their own. Consistent with this is the finding that young children assessed as having a disorganized attachment have caregivers who are often unresolved with respect to past traumatic experience (Lyons-Ruth & Jacobvitz, 1999). In short, caregiver history of attachment relationships and of trauma exposure, via reflective functioning, may determine not only the dyad's quality of attachment but also the manner in which trauma exposure will be processed.

Thus, traumatic violence can interfere with the initial development of a secure and organized attachment or derail a previously secure attachment if the caregiver is sufficiently adversely affected. Disturbances in attachment in turn confer increased risk for (1) recovery from trauma exposure by the child and/or caregiver (Fisher, Gunnar, Dozier, Bruce, & Pears, 2006), (2) enactment of maltreatment by the traumatized caregiver (Cicchetti, Rogosch, & Toth, 2006), (3) child exposure to trauma via inadequate monitoring by the caregiver (Schechter, Brunelli, Cunningham, Brown, & Baca, 2002) and (4) subsequent repetition and transmission of risk by the traumatized child and/or caregiver (Weinfield, Whaley, & Egeland, 2004). Such evidence supports the contention that we must view infant mental health disturbances through the dual lenses of attachment and trauma (Lieberman, 2004).

The recognition of the importance of the relational context of trauma in clinical assessment and treatment has emerged in several applications, one that described mediating, moderating, and mixed mediating–moderating effects on PTSD within the parent–child relationship (Scheeringa & Zeanah, 2001) and another that considers the importance of assessing the parental awareness of child states following terrorism or disaster (Coates, Schechter, & First, 2003).

## Neurobiology

### *Early Childhood Neurobiology*

Evidence has accumulated that violent trauma early in life—particularly when involving repeated and severe exposure—is tantamount to the effect of an environmental toxin to the developing central nervous system and causes enduring adverse effects across a range of brain structures and functions, including mental *and* physical health and susceptibility to illness (McEwen, 2003). Threats to the infant's survival, whether they be directly to his or her person, or to the caregiving system, result in activation of the fight–flight–freeze response associated with fear conditioning. Failure to extinguish the fear response and the psychobiological cost of that failure are thought to lead to posttraumatic psychopathology. Activation of the two principal stress-reactivity mechanisms associated with fear conditioning are most often prompted by the limbic system of the central nervous system, most prominently the amygdala, leading to activation of the sympathetic branch of the autonomic nervous system (i.e., increased heart rate) via quick-acting noradrenergic emission and the hypothalamic–pituitary–adrenal (HPA) axis via the slower-acting central glucocorticoid secretion (i.e., leading to increased circulating cortisol, binding at central nervous system sites such as the hippocampus and medial prefrontal cortex). Increased cortisol levels also result in an elevation of blood glucose that sustains the organism during the fight–flight–freeze response and that helps to quell the sympathetic nervous system response (see Thompson, Kiff, & McLaughlin, Chapter 5, this volume).

Preclinical studies have shown that areas of the brain that are particularly prone to adverse

effects of maltreatment and violent trauma during the first 3–5 years of life include those that have a prolonged postnatal developmental period, those with a high density of glucocorticoid receptors, and those that have the potential for postnatal neurogenesis (Teicher et al., 2003). These areas include, most prominently, the hippocampus and amygdala, corpus callosum, cerebellar vermis, and the cerebral cortex.

When a rat infant undergoes severe stress such as repeated footshocks, the hippocampus fails to form the expected density of synaptic connections. Normative pruning of these connections nonetheless occurs later in the prepubertal period, and adult animals who were repeatedly stressed in infancy end up with far fewer synaptic connections in this region (Andersen & Teicher, 2004). These results support Carrion, Weems, and Reiss's (2007) findings that differences in hippocampal volume among patients with PTSD are more likely due to the effects of neurotoxicity to stress hormones than to a constitutional size difference. Clinical implications of hippocampal and amygdala damage due to stress hormones may include increased propensity for confusion of past and present, flashbacks, and dissociative symptoms (Sakamoto et al., 2005). Moreover, given the role of the hippocampus in the extinction of traumatic memories, it is not surprising that a recent study has shown that larger hippocampal volume among adult patients with PTSD is associated with a better response to psychotherapy (Rubin et al., 2016).

The corpus callosum is a heavily myelinated region of the brain that is associated with hemispheric integration. High levels of stress hormones during infancy and early childhood have been associated with suppressed glial cell division that is critical for myelination (Berrebi et al., 1988). DeBellis and colleagues (2002) observed that reduced corpus callosum size was the most significant structural finding noted in children with a history of maltreatment and PTSD. Disturbances in the myelination of the corpus callosum and cortex due to excessive exposure to glucocorticoids during the first 3 years of life may explain some of the difficulties that maltreated preschool-age children have in integrating cognitive and emotional information and in taking others' perspective in comparison to nonmaltreated age-matched controls (Pears & Fisher, 2005). A more recent study has linked decreased corpus callosum size with disturbance in associative memory that may account for integration difficulties noted in previous studies (Saar-Ashkenazy et al., 2014).

Among the most exciting research that illustrates the interaction of development and traumatic experience is that regarding the differential effects of specific types of maltreatment and violent trauma on the brain at different critical periods of development through early adulthood in animal and human models (Hall, 1998; Teicher, Tomoda, & Andersen, 2006). For example, repeated episodes of child sexual and physical abuse were associated in the same group of subjects with reduced hippocampal volume if the abuse was reported to occur in early childhood, but with reduced prefrontal cortex volume if the abuse occurred during adolescence (Teicher, 2005). Similar exposure during different, temporally discrete windows of development may have very different clinical implications. That being said, individuals who had exposure to maltreatment and violence during early childhood had higher levels of PTSD symptom severity than those exposed in middle childhood or adolescence, even after controlling for a number of covariates (Dunn, Nishimi, Powers, & Bradley, 2017).

### *Relational Neurobiology*

In the past 5 years, researchers have made important gains toward understanding the interplay of the experience of violence, related psychopathology in the child, related psychopathology in the parent(s), and the parent–child relationship (i.e., attachment), all in the context of neurobiological development. As noted, more than two-thirds of children in the United States age 17 and younger were exposed to violence within the past year either directly as victims or indirectly as witnesses (Finkelhor, Turner, Shattuck, & Hamby, 2015), with over 40% of children ages 2–5 and a conservative estimate of over 10% under 2 years of age. So, what accounts for the variability of effects of violence at any point in time and across development? We established earlier in this chapter that both the nature and the severity of the violence (the number of events, the chronicity, the degree of injury, the perpetrator's closeness of kinship to the victim, etc.) matter in terms of sequelae to physical and mental health. Similarly, research has repeatedly supported the notion that parental psychological functioning does strongly affect the child's psychological and biological functioning whether or not the child him- or her-

self was directly or indirectly exposed (Schechter et al., 2011; Scheeringa, Myers, Putnam, & Zeanah, 2015; Scheeringa & Zeanah, 2001). Parental stress physiology and neural functioning are known both to subserve and to mirror, as it were, parental psychological functioning in the wake of violence exposure and its consequences (Moser, Aue, Suardi, Kutlikova, et al., 2015; Schechter, Moser, McCaw, & Myers, 2014), so we consider below recent research on their impact on the parent–child relationship and child developmental psychopathology.

What has also become clearer in recent studies is how the child's own biology interacts with environmental factors to shape individual differences in the child's response to his or her caregiving environment and to violent events he or she has experienced. For example, out of the nearly 70% of children who experience violence, why do only a relatively small percentage (i.e., an estimated 5–10% on average) develop PTSD? A number of studies have identified gene × environment interactions that may predispose certain individuals to developing PTSD versus major depression or their comorbidity in the wake of trauma (Donaldson et al., 2016; Wingo et al., 2015) and may augment a vulnerable individual's development of a psychotic disorder (Powers, Fani, Cross, Ressler, & Bradley, 2016). Alleles of several specific genes involved in the regulation of the HPA axis, such as the chaperone molecule *FKBP5* and the glucocorticoid receptor (*NR3c1*), have been identified as risk factors for the development of PTSD, while other gene polymorphisms have been identified as risk factors for MDD (Nemoda et al., 2015). Genetics researchers have become aware that they have only seen the tip of the iceberg with respect to PTSD, long thought to be purely an environmentally based disorder. A large-scale genomewide association study (GWAS) has begun in an effort to get at the complexity of genetic risk for PTSD in much the same way as researchers have studied bipolar disorder and schizophrenia (Logue et al., 2015).

We know that direct and indirect violence exposure and maltreatment (i.e., physical abuse perpetrated by a primary attachment figure) across development affects stress physiology in all relevant systems, most saliently, those of the autonomic nervous system and the HPA axis, in addition to the cytokine system that is involved in inflammation and therefore contributes to overall health outcomes (Tursich et al., 2014). When the violence exposure and maltreatment have an early onset and are repetitive and chronic, the outpouring of glucocorticoids, which is meant as a facilitator of the fight–flight response, can become noxious to developing brain structures, in particular, the hippocampus (Frodl & O'Keane, 2013; Sapolsky et al., 1990; Zhao et al., 2016). Strikingly, individuals with early-onset and otherwise chronic early exposures to maltreatment and violence often have lower levels of cortisol and decreased reactivity to stress (Elzinga et al., 2008; Rogosch, Dackis, & Cicchetti, 2011). This has been found to be particularly the case for maltreated individuals with a particular polymorphism of the corticotropin-releasing hormone receptor Type I gene *RS110402* (Sumner, McLaughlin, Walsh, Sheridan, & Koenen, 2014). Low cortisol levels and polymorphisms functionally linked to the glucocorticoid receptor such as that of the chaperone molecule *FKBP5* that is important to get the glucocorticoids to receptors in the cell nucleus have been posited to be interrelated risk factors for the development of PTSD (Binder et al., 2008).

In terms of neural activity independent of physiological activity, one study has shown that even for 6- to 12-month-old infants, higher levels of interparental verbal conflict are significantly associated with changes in salience of circuit regions (i.e., rostral anterior cingulate cortex, caudate, thalamus) upon exposure to angry versus neutral auditory speech stimuli while asleep in the MRI scanner (Graham, Fisher, & Pfeifer, 2013). The latter suggests that, beginning in infancy, the developing brain is sensitive to even less noxious stimuli than what the research literature has traditionally defined as "toxic" or "traumatic," such as maltreatment—and this, even when not awake!

Furthermore, many children of mothers diagnosed with IVP-related PTSD have been exposed to varying levels of adult conflict in the home, ranging from verbal disputes to verbal and psychological abuse, to physical violence. These children, by the age of 12–42 months indeed showed altered (i.e., decreased) stress reactivity to separation and exposure to novelty when compared to children of mothers without PTSD, illustrating the important role of maternal psychological functioning in processing their early experience (Cordero et al., 2017). The question of "how early" and "how severe" the exposure was may be more important in more ways than we previously realized. The notion that low glucocorticoid levels, flattened circa-

dian rhythms, and lower reactivity to validated laboratory stressors are associated with PTSD has raised the question of whether they are in fact risk factors for the disorder and, again, whether genetic or epigenetic vulnerabilities are at play and/or whether adaptations of "hypocortisolism" may serve a protective function in a high-risk for violence environment (Yehuda, Bell, Bierer, & Schmiedler, 2008; Yehuda et al., 2015). Studies have begun to examine the phenomenon of fetal programming and the notion that if a woman is exposed to significant stress while pregnant, and while the fetus is in a sensitive period of brain development (e.g., during the development of the CA1, and less so, CA3 layers of the hippocampus), more glucocorticoid receptors are produced to buffer the effects of cortisol on the developing brain, perhaps so as to prepare the fetus for adaptation to a stressful postnatal environment (McCabe, Marash, Li, & Matthews, 2001; Murthy, George, Ramasamy, & Mustapha, 2013; O'Connor, Bergman, Sarkar, & Glover, 2013; Buss et al., 2017). In addition, CA2 and CA3 continue to be affected by postnatal cortisol administration (or production) in the child (Bos, Montoya, Terburg, & van Honk, 2014). Decreased telomere length in newborns, which is associated with increased incidence of age-related diseases and mortality, has been associated with increased maternal psychosocial stress despite this possible buffering of effects (Entringer et al., 2013) as well as increased postnatal anxiety (Bennett, Palliser, Shaw, Walker, & Hirst, 2015). Relevant to violence exposure, infants of pregnant women who were at Ground Zero during the 9/11 attacks on the World Trade Center in New York were shown to have an upregulation of the glucocorticoid receptor set point by 9 months of life, with lower salivary cortisol and greater distressed behavior in response to novelty than infants of nonexposed mothers (Yehuda et al., 2005).

As the hypothalamic–midbrain–limbic–paralimbic–cortical circuits in the caregiver respond jointly to infant stimuli, as has been found in recent neuroimaging studies among normative mother–infant dyads (Swain, Lorberbaum, Kose, & Strathearn, 2007), one can imagine a cycle of dysregulation in which unquelled infant distress becomes a stressor, particularly for a traumatized parent. Indeed, while watching video clips of their children during separation and other stressful moments, group differences between violence-exposed mothers of toddlers and nonexposed mothers have been noted with respect to behavioral and brain activation (Moser, Aue, Suardi, Manini, et al., 2015; Schechter et al., 2012; Schechter, Moser, Paoloni-Giacobino, et al., 2015). In functional magnetic resonance imaging (fMRI), mothers with IPV-related PTSD (IPV-PTSD), when exposed to silent film excerpts showing (1) child–parent separation versus play or (2) adult male–female menacing versus neutral or prosocial interactions, display decreased activation of the medial prefrontal cortex in both ventral and dorsal areas and anterior cingulate cortex, as well as increased activation in limbic regions (i.e., hippocampus, parahippocampus, and entorhinal cortex), both when symptoms are analyzed by group (mothers with PTSD vs. those without PTSD) and as continuous ratings of severity of maternal PTSD symptoms (Schechter et al., 2012; Schechter, Moser, Paoloni-Giacobino, et al., 2015). This "cortical–limbic dysregulation" is thus consistent with multiple functioning neuroimaging studies of adult patients with PTSD exposed to trauma-related stimuli over the past 15 years (Bremner et al., 1999; Frewen, Pain, Dozois, & Lanius, 2006; Gold et al., 2011; Milad et al. 2009; Schreiber, Lu, Baynes, Richardson, & Gilpin, 2017; Shin et al., 2001). This suggests importantly that mothers with IPV-PTSD may well react to the helpless, distressed state of mind of their toddlers as they would to a traumatic trigger, since the child's helplessness and dysregulated negative emotion may remind the mother of her own and/or the perpetrator's state of mind during the experience of IPV (Schechter, Moser, Reliford, et al., 2015).

Schechter and colleagues have begun to examine the functional neuroimaging of peripheral (salivary DNA) epigenetic signatures related to stress physiology impacting gene promoter regions, and these epigenetic signatures' association to maternal IPV-PTSD, neural activity in response to relational stimuli, and individual differences in maternal and child behavior (Moser, Paoloni-Giacobino, et al., 2015; Schechter, Moser, Paoloni-Giacobino, et al., 2015; Schechter et al., 2017). "Imaging epigenetics" is a new frontier that extends work on "imaging genomics" (Nikolova & Hariri, 2015). Recently, significant associations have been identified between methylation of the promoter regions of the genes for the glucocorticoid receptor *NR3c1,* the ionotropic serotonin receptor 3a (*HTR3A*) and maternal IPV-PTSD and neural activity in the medial prefrontal cortex (mPFC)

(Schechter, Moser, Paoloni-Giacobino, et al., 2015; Schechter et al., 2017). The significant negative correlation between methylation of the maternal *NR3c1* receptor, maternal IPV-PTSD severity, and mPFC activity in response to video stimuli of mother–child separation versus play (Schechter, Moser, Paoloni-Giacobino, et al., 2015) supports findings of hypocortisolism among moathers with IPV-PTSD (Cordero et al., 2017). The positive correlation between methylation of maternal *HTR3A,* maternal and child aggression, and increased activity in the mPFC in response to adult male–female menacing versus neutral and prosocial interactions supports the notion that decreased available serotonin in saliva may be associated with an endophenotype among mothers with IPV-PTSD and their children that is more likely to display aggressive behavior (Schechter et al., 2017). Finally, methylation of the promoter region of brain-derived neurotrophic factor (BDNF), implying less available BDNF, was associated with maternal anxiety, childhood exposure to domestic violence, and neural activity in corticolimbic regions, including the ventral mPFC (Moser, Paoloni-Giacobino, et al., 2015).

With the identification of biomarkers such as those described earlier, researchers can now consider how interventions might leave traces via neural activity, epigenetic signatures, and changes in stress physiology as well as what works best for whom (Bakermans-Kranenburg, van IJzendoorn, Mesman, Alink, & Juffer, 2008; Ryan, Booth, Spathis, Mollart, & Clow, 2016). For example, treatment intervention while in foster care has already been most elegantly shown to dramatically affect the psychological and biological effects of early maltreatment and neglect in terms of psychopathology, glucocorticoid levels, and electroencephalographic (EEG) resting state activity—the latter with sustained improvement over years (Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008; Humphreys & Zeanah, 2015; Vanderwert, Zeanah, Fox, & Nelson, 2016; see also Coyne, Powell, Hoffman, & Cooper, Chapter 30, and Smyke & Breidenstine, Chapter 34, this volume).

## Conclusions

We have provided in this chapter a framework for the consideration of several dimensions that are essential to assessing and treating the infant or young child who may have been exposed to violent trauma and/or maltreatment: the nature and sequelae of the exposure, the developmental context of the exposure, and the individual and relational neurobiology of the exposure. We hope that clinicians and investigators reading this chapter will now ask, "What happened to the infant?"; "Who is the traumatized infant in psychobiological terms?"; "When did the trauma occur in the life course of the infant?"; and "Who is in the infant's relational world who can either help or hinder making sense of what happened?"

## REFERENCES


Acarturk, C., Konuk, E., Cetinkaya, M., Senay, I., Sijbrandij, M., Cuijpers, P., et al. (2015). EMDR for Syrian refugees with posttraumatic stress disorder symptoms: Results of a pilot randomized controlled trial. *European Journal of Psychotraumatology, 6,* Article No. 27414.

Ahlfs-Dunn, S. M., & Huth-Bocks, A. C. (2014). Intimate partner violence and infant socioemotional development: The moderating effects of maternal trauma symptoms. *Infant Mental Health Journal, 35*(4), 322–335.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Andersen, S. L., & Teicher, M. H. (2004). Delayed effects of early stress on hippocampal development. *Neuropsychopharmacology, 29*(11), 1988–1993.

Appel, A. E., & Holden, G. W. (1998). The co-occurance of spouse and physical child abuse: A review and appraisal. *Journal of Family Psychology, 12*(4), 578–599.

Bakermans-Kranenburg, M. J., van IJzendoorn, M. H., Mesman, J., Alink, L. R., & Juffer, F. (2008). Effects of an attachment-based intervention on daily cortisol moderated by dopamine receptor D4: A randomized trial on 1- to 3-year-olds screened for externalizing behavior. *Developmental Psychopathalogy, 20*(3), 805–820.

Barrett, H. C., & Behne, T. (2005). Children's understanding of death as the cessation of agency: A test using sleep versus death. *Cognition, 96*(2), 93–108.

Bauer, P. J. (2006). Constructing a past in infancy: A neuro-developmental account. *Trends in Cognitive Sciences, 10*(4), 175–181.

Beeghly, M., & Cicchetti, D. (1994). Child maltreatment, attachment, and the self system: Emergence of an internal state lexicon in toddlers at high social risk. *Development and Psychopathology, 6,* 5–30.

Bennett, G. A., Palliser, H. K., Shaw, J. C., Walker, D., & Hirst, J. J. (2015). Prenatal stress alters hippocampal neuroglia and increases anxiety in childhood. *Developmental Neuroscience, 37*(6), 533–545.

Berrebi, A. S., Fitch, R. H., Ralphe, D. L., Denenberg,

J. O., Friedrich, V. L. J., & Denenberg, V. H. (1988). Corpus callosum: Region-specific effects of sex, early experience and age. *Brain Research, 438*(1–2), 216–224.

Binder, E. B., Bradley, R. G., Liu, W., Epstein, M. P., Deveau, T. C., Mercer, K. B., et al. (2008). Association of FKBP5 polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults. *Journal of the American Medical Association, 299*(11), 1291–1305.

Bos, P. A., Montoya, E. R., Terburg, D., & van Honk, J. (2014). Cortisol administration increases hippocampal activation to infant crying in males depending on childhood neglect. *Human Brain Mapping, 35*(10), 5116–5126.

Bowlby, J. (1969). *Attachment and loss: Vol. 1. Attachment*. New York: Basic Books

Bremner, J. D., Narayan, M., Staib, L. H., Southwick, S. M., McGlashan, T., & Charney, D. S. (1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. *American Journal of Psychiatry, 156*(11), 1787–1795.

Bretherton, I. (1990). Communication patterns, internal working models, and the intergenerational transmission of attachment relationships. *Infant Mental Health Journal, 11*(3), 237–252.

Bretherton, I., & Munholland, K. A. (1999). Internal working models in attachment relationships: A construct revisited. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 89–114). New York: Guilford Press.

Buss, C., Entringer, S., Moog, N. K., Toepfer, P., Fair, D. A., Simhan, H. N., et al. (2017). Intergenerational transmission of maternal childhood maltreatment exposure: Implications for fetal brain development. *Journal of the American Academy of Child and Adolescent Psychiatry, 56*(5), 373–382.

Carrion, V. G., Weems, C. F., & Reiss, A. L. (2007). Stress predicts brain changes in children: A pilot longitudinal study on youth stress, posttraumatic stress disorder, and the hippocampus. *Pediatrics, 119*(3), 509–516.

Cicchetti, D., & Lynch, M. (1995). Failures in the expectable environment and their impact on individual development: The case of child maltreatment. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Risk, disorder, and adaptation* (Vol. 2, pp. 32–71). New York: Wiley.

Cicchetti, D., Rogosch, F. A., & Toth, S. L. (2006). Fostering secure attachment in infants in maltreating families through preventive interventions. *Development and Psychopathology, 18*(3), 623–649.

Cicchetti, D., Toth, S. L., Bush, M. A., & Gillespie, J. F. (1988). Stage-salient issues: A transactional model of intervention. *New Directions for Child Development, 39,* 123–145.

Clarkson Freeman, P. A. (2014). Prevalence and relationship between adverse childhood experiences and child behavior among young children. *Infant Mental Health Journal, 35*(6), 544–554.

Coates, S. W., Schechter, D. S., & First, E. (2003). Brief interventions with traumatized children and families after September 11. In S. W. Coates, J. L. Rosenthal, & D. S. Schechter (Eds.), *September 11: Trauma and human bonds* (pp. 23–50). Hillside, NJ: Analytic Press.

Cook, E. H. J., Kieffer, J. E., Charak, D. A., & Leventhal, B. L. (1993). Autistic disorder and posttraumatic stress disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*(6), 1292–1294.

Copeland, W. E., Keeler, G., Angold, A., & Costello, E. J. (2007). Traumatic events and posttraumatic stress in childhood. *Archives of General Psychiatry, 64*(5), 577–584.

Cordero, M. I., Moser, D. A., Manini, A., Suardi, F., Sancho-Rossignol, A. S., Torrisi, R., Rossier, M. F., et al. (2017). Effects of interpersonal violence-related post-traumatic stress disorder (PTSD) on mother and child diurnal cortisol rhythm and cortisol reactivity to a laboratory stressor involving separation. *Hormones and Behavior, 90,* 15–24.

Costello, E. J., Erkanli, A., Fairbank, J. A., & Angold, A. (2002). The prevalence of potentially traumatic events in childhood and adolescence. *Journal of Traumatic Stress, 15*(2), 99–112.

Cote, S. M., Vaillancourt, T., Barker, E. D., Nagin, D., & Tremblay, R. E. (2007). The joint development of physical and indirect aggression: Predictors of continuity and change during childhood. *Development and Psychopathology, 19*(1), 37–55.

Curtis, W. J., & Cicchetti, D. (2013). Affective facial expression processing in 15-month-old infants who have experienced maltreatment: An event-related potential study. *Child Maltreatment, 18*(3), 140–154.

De Bellis, M. D., Keshavan, M. S., Shifflett, H., Iyengar, S., Beers, S. R., Hall, J., et al. (2002). Brain structures in pediatric maltreatment-related posttraumatic stress disorder: A sociodemographically matched study. *Biological Psychiatry, 52*(11), 1066–1078.

Donaldson, Z. E., le Francois, B., Santos, T. L., Almli, L. M., Boldrini, M., Champagne, F. A., et al. (2016). The functional serotonin 1a receptor promoter polymorphism, rs6295, is associated with psychiatric illness and differences in transcription. *Translational Psychiatry, 1*(6), e746.

Dozier, M., Peloso, E., Lewis, E., Laurenceau, J. P., & Levine, S. (2008). Effects of an attachment-based intervention on the cortisol production of infants and toddlers in foster care. *Developmental Psychopathology, 20*(3), 845–859.

Driessen, M., Schroeder, T., Widmann, B., von Schonfeld, C. E., & Schneider, F. (2006). Childhood trauma, psychiatric disorders, and criminal behavior in

prisoners in Germany: A comparative study in incarcerated women and men. *Journal of Clinical Psychiatry, 67*(10), 1486–1492.

Dube, S. R., Anda, R. F., Felitti, V. J., Chapman, D. P., Williamson, D. F., & Giles, W. H. (2001). Childhood abuse, household dysfunction, and the risk of attempted suicide throughout the life span: Findings from the Adverse Childhood Experiences Study. *Journal of the American Medical Association, 286*(24), 3089–3096.

Dunn, E. C., McLaughlin, K. A., Slopen, N., Rosand, J., & Smoller, J. W. (2013). Developmental timing of child maltreatment and symptoms of depression and suicidal ideation in young adulthood: Results from the National Longitudinal Study of Adolescent Health. *Depression and Anxiety, 30*(10), 955–964.

Dunn, E. C., Nishimi, K., Powers, A., & Bradley, B. (2017). Is developmental timing of trauma exposure associated with depressive and post-traumatic stress disorder symptoms in adulthood? *Journal of Psychiatric Research, 84,* 119–127.

Edleson, J. L. (1999). The overlap between child maltreatment and woman battering. *Violence Against Women, 5*(2), 134–154.

Ehrensaft, M. K., Cohen, P., Brown, J., Smailes, E., Chen, H., & Johnson, J. G. (2003). Intergenerational transmission off partner violence: A 20-year prospective study. *Journal of Consulting and Clinical Psychology, 71*(4), 741–753.

Eigsti, I. M., & Cicchetti, D. (2004). The impact of child maltreatment on expressive syntax at 60 months. *Developmental Science, 7*(1), 88–102.

Elzinga, B. M., Roelofs, K., Tollenaar, M. S., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse events a study among healthy young subjects. *Psychoneuroendocrinology, 33*(2), 227–237.

Enlow, M. B., Egeland, B., Blood, E. A., Wright, R. O., & Wright, R. J. (2012). Interpersonal trauma exposure and cognitive development in children to age 8 years: A longitudinal study. *Journal of Epidemiology and Community Health, 66*(11), 1005–1010.

Entringer, S., Epel, E. S., Lin, J., Buss, C., Shahbaba, B., Blackburn, E. H., et al. (2013). Maternal psychosocial stress during pregnancy is associated with newborn leukocyte telomere length. *American Journal of Obstetrics and Gynecology, 208*(2), 134. el–134.e7.

Eth, S., & Pynoos, R. S. (1994). Children who witness the homicide of a parent. *Psychiatry, 57*(4), 287–306.

Fantuzzo, J., & Fusco, R. (2007). Children's direct sensory exposure to substantiated domestic violence crimes. *Violence and Victims, 22*(2), 158–171.

Fasfous, A. F., Peralta-Ramirez, I., Perez-Garcia, M. (2013). Symptoms of PTSD among children living in war zones in same cultural context and different situations. *Journal of Muslim Mental Health, 7*(2), 47–61.

Feldman, R., & Vengrober, A. (2011). Post-traumatic stress disorder in infants and young children exposed to war-related trauma. *Journal of the American Academy of Child and Adolescent Psychiatry, 50*(7), 645–658.

Finkelhor, D., & Turner, H. (2016a). *National Survey of Children's Exposure to Violence I, 1990–2008* [United States]. Ann Arbor, MI: Interuniversity Consortium for Political and Social Research.

Finkelhor, D., & Turner, H. (2016b). *National Survey of Children's Exposure to Violence II, 1993–2012* [United States]. Ann Arbor, MI: Interuniversity Consortium for Political and Social Research.

Finkelhor, D., & Turner, H. (2016c). *National Survey of Children's Exposure to Violence III, 1997–2014* [United States]. Ann Arbor, MI: Interuniversity Consortium for Political and Social Research.

Finkelhor, D., Turner, H. A., Shattuck, A., & Hamby, S. L. (2015). Prevalence of childhood exposure to violence, crime, and abuse: Results from the National Survey of Children's Exposure to Violence. *JAMA Pediatrics, 169*(8), 746–754.

Finkelhor, D., Turner, H., Shattuck, A., Hamby, S., & Kracke, K. (2015). *Children's exposure to violence, crime, and abuse: An update* (Publication No. NCJ 248547). Washington, DC: Office of Juvenile Justice and Delinquency Prevention, U.S. Department of Justice.

Fischer, K. W., Ayoub, C., Singh, I., Noam, G., Maraganore, A., & Raya, P. (1997). Psychopathology as adaptive development along distinctive pathways. *Development and Psychopathology, 9*(4), 749–779.

Fisher, P. A., Gunnar, M. R., Dozier, M., Bruce, J., & Pears, K. C. (2006). Effects of therapeutic interventions for foster children on behavioral problems, caregiver attachment, and stress regulatory neural systems. *Annals of the New York Academy of Sciences, 1094,* 215–225.

Fivush, R. (1998). Children's recollections of traumatic and nontraumatic events. *Development and Psychopathology, 10*(4), 699–716.

Fonagy, P., Moran, G., Steele, M., Steele, H., & Higgitt, A. (1991). The capacity for understanding mental states: The reflective self in parent and child and its significance for security of attachment. *Infant Mental Health Journal, 13,* 200–216.

Fonagy, P., Steele, M., Moran, G. S., Steele, H., & Higgitt, A. (1993). Measuring the ghost in the nursery: An empirical study of the relation between parents' mental representations of childhood experiences and their infants' security of attachment. *Journal of American Psychoanalytic Association, 41,* 957–989.

Fonagy, P., Steele, M., Steele, H., Higgitt, A., & Target, M. (1994). Theory and practice of resilience. *Journal of Child Psychology and Psychiatry, 35,* 231–257.

Fraiberg, S., Adelson, E., & Shapiro, V. (1975). Ghosts in the nursery: A psychoanalytic approach to the problems of impaired infant–mother relationships.

*Journal of the American Academy of Child Psychiatry, 14*(3), 387–421.

Freud, A., & Burlingham, D. T. (1943). *War and children*. New York: Medical War Books.

Frewen, P. A., Pain, C., Dozois, D. J., & Lanius, R. A. (2006). Alexithymia in PTSD: Psychometric and FMRI studies. *Annals of the New York Academy of Sciences, 1071,* 397–400.

Frodl, T., & O'Keane, V. (2013). How does the brain deal with cumulative stress?: A review with focus on developmental stress, HPA axis function and hippocampal structure in humans. *Neurobiology of Disease, 52,* 24–37.

Fusco, R. A., & Fantuzzo, J. W. (2009). Domestic violence crimes and children: A population-based investigation of direct sensory exposure and the nature of involvement. *Children and Youth Services Review, 31*(2), 249–256.

Gaensbauer, T., Chatoor, I., Drell, M., Siegel, D., & Zeanah, C. H. (1995). Traumatic loss in a one-year-old girl. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*(4), 520–528.

Gold, A. L., Shin, L. M., Orr, S. P., Carson, M. A., Rauch, S. L., Macklin, M. L., et al. (2011). Decreased regional cerebral blood flow in medial prefrontal cortex during trauma-unrelated stressful imagery in Vietnam veterans with post-traumatic stress disorder. *Psychological Medicine, 41*(12), 2563–2572.

Graham, A. M., Fisher, P. A., & Pfeifer, J. H. (2013). What sleeping babies hear: A functional MRI study of interparental conflict and infants' emotion processing. *Psychological Science, 24*(5), 282–289.

Gustafsson, H. C., Coffman, J. L., Harris, L. S., Langley, H. A., Ornstein, P. A., & Cox, M. J. (2013). Intimate partner violence and children's memory. *Journal of Family Psychology, 27*(6), 937–944.

Hall, F. S. (1998). Social deprivation of neonatal, adolescent, and adult rats has distinct neurochemical and behavioral consequences. *Critical Reviews in Neurobiology, 12,* 129–162.

Hamby, S., Finkelhor, D., Turner, H., & Ormrod, R. (2010). The overlap of witnessing partner violence with child maltreatment and other victimizations in a nationally representative survey of youth. *Child Abuse and Neglect, 34*(10), 734–741.

Hamby, S., Finkelhor, D., Turner, H., & Ormrod, R. (2011). *Children's exposure to intimate partner violence and other family violence* (Publication No. NCJ 232272). Washington, DC: Office of Juvenile Justice and Delinquency Prevention, U.S. Department of Justice.

Harpur, L. J., Polek, E., & van Harmelen, A. L. (2015). The role of timing of maltreatment and child intelligence in pathways to low symptoms of depression and anxiety in adolescence. *Child Abuse and Neglect, 47,* 24–37.

Hartman, C. R., & Burgess, A. W. (1989). Sexual abuse of children: Causes and consequences. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment* (pp. 95–128). New York: Cambridge University Press.

Hartshorn, K., & Rovee-Collier, C. (2003). Does infant memory expression reflect age at encoding or age at retrieval? *Developmental Psychobiology, 42*(3), 283–291.

Horner-Johnson, W., & Drum, C. E. (2006). Prevalence of maltreatment of people with intellectual disabilities: A review of recently published research. *Mental Retardation and Developmental Disabilities Research Reviews, 12*(1), 57–69.

Howe, M. L., Cicchetti, D., & Toth, S. L. (2006). Children's basic memory processes, stress, and maltreatment. *Development and Psychopathology, 18*(3), 759–769.

Humphreys, K. L., & Zeanah, C. H. (2015). Deviations from the expectable environment in early childhood and emerging psychopathology. *Neuropsychopharmacology, 40*(1), 154–170.

Jouriles, E. N., Rosenfield, D., McDonald, R., Vu, N. L., Rancher, C., & Mueller, V. (2016). Children exposed to intimate partner violence: Conduct problems, interventions, and partner contact with the child. *Journal of Clinical Child and Adolescent Psychology, 30,* 1–13.

Kaplow, J. B., Saxe, G. N., Putnam, F. W., Pynoos, R. S., & Lieberman, A. F. (2006). The long-term consequences of early childhood trauma: A case study and discussion. *Psychiatry, 69*(4), 362–375.

Kaufman, J., Plotsky, P. M., Nemeroff, C. B., & Charney, D. S. (2000). Effects of early adverse experiences on brain structure and function: Clinical implications. *Biological Psychiatry, 48*(8), 778–790.

Kirmayer, L. J., Narasiah, L., Munoz, M., Rashid, M., Ryder, A. G., Guzder, J., et al. (2011). Common mental health problems in immigrants and refugees: General approach in primary care. *Canadian Medical Association Journal, 183*(12), E959–E967.

Kitzmann, K. M., Gaylord, N. K., Holt, A. R., & Kenny, E. D. (2003). Child witness to domestic violence: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 71,* 741–753.

Leiner, M., Peinado, J., Malazo Villanos, M. T., Lopez, I., Uribe, R., & Pathak, I. (2016). Mental and emotional health of children exposed to news media of threats and acts of terrorism: The cumulative and pervasive effects. *Frontiers in Pediatrics, 4,* 26.

Levendosky, A. A., Bogat, G. A., & Martinez-Torteya, C. (2013). PTSD symptoms in young children exposed to intimate partner violence. *Violence Against Women, 19*(2), 187–201.

Levendosky, A. A., Leahy, K. L., Bogat, G. A., Davidson, W. S., & von Eye, A. (2006). Domestic violence, maternal parenting, maternal mental health, and infant externalizing behavior. *Journal of Family Psychology, 20*(4), 544–552.

Lieberman, A. F. (2004). Traumatic stress and quality of attachment: Reality and internalization in disor-

ders of infant mental health. *Infant Mental Health Journal, 24*(4), 336–351.

Lieberman, A. F., & Pawl, J. H. (1990). Disorders of attachment and secure-base behavior in the second year of life. In E. T. Greenberg, D. Cicchetti, & E. M. Cummings (Eds.), *Attachment in the preschool years: Theory, research, and interventions* (pp. 375–397). Chicago: University of Chicago Press.

Lieberman, A. F., van Horn, P., & Ozer, E. J. (2005). Preschooler witnesses of marital violence: Predictors and mediators of child behavior problems. *Development and Psychopathology, 17,* 385–396.

Logue, M. W., Amstadter, A. B., Baker, D. G., Duncan, L., Koenen, K. C., Liberzon, I., et al. (2015). The Psychiatric Genomics Consortium Posttraumatic Stress Disorder Workgroup: Posttraumatic stress disorder enters the age of large-scale genomic collaboration. *Neuropsychopharmacology, 40*(10), 2287–2297.

Lukowski, A. F., Wiebe, S. A., Haight, J. C., DeBoer, T., Nelson, C. A., & Bauer, P. J. (2005). Forming a stable memory representation in the first year of life: Why limitation is more than child's play. *Developmental Science, 8*(3), 279–298.

Lyons-Ruth, K., & Jacobvitz, D. (1999). Attachment disorganization: Unresolved loss, relational violence, and lapses in behavioral and attentional strategies. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 520–555). New York: Guilford Press.

Manly, J. T., Kim, J. E., Rogosch, F. A., & Cicchetti, D. (2001). Dimensions of child maltreatment and children's adjustment: Contributions of developmental timing and subtype. *Development and Psychopathology, 13,* 759–782.

McCabe, L., Marash, D., Li, A., & Matthews, S. G. (2001). Repeated antenatal glucocorticoid treatment decreases hypothalamic corticotropin releasing hormone mRNA but not corticosteroid receptor mRNA expression in the fetal guinea-pig brain. *Journal of Neuroendocrinology, 13*(5), 425–431.

McEwen, B. S. (2003). Early life influences on life-long patterns of behavior and health. *Mental Retardation and Developmental Disabilities Research Reviews, 9*(3), 149–154.

Milad, M. R., Pitman, R. K., Ellis, C. B., Gold, A. L., Shin, L. M., Lasko, N. B., et al. (2009). Neurobiological basis of failure to recall extinction memory in posttraumatic stress disorder. *Biological Psychiatry, 66*(12), 1075–1082.

Moser, D. A., Aue, T., Suardi, F., Kutlikova, H., Cordero, M. I., Rossignol, A. S., et al. (2015). Violence-related PTSD and neural activation when seeing emotionally charged male–female interactions. *Social Cognitive and Affective Neuroscience, 10*(5), 645–653.

Moser, D. A., Aue, T., Suardi, F., Manini, A., Rossignol, A. S, Cordero, M. I., et al. (2015). The relation of general socio-emotional processing to parenting specific behavior: A study of mothers with and without posttraumatic stress disorder. *Frontiers in Psychology, 6,* 1575.

Moser, D. A., Paoloni-Giacobino, A., Stenz, L., Adouan, W., Manini, A., Suardi, F., et al. (2015). BDNF Methylation and maternal brain activity in a violence-related sample. *PLOS ONE, 10*(12), e0143427.

Murthy, K. D., George, M. C., Ramasamy, P., & Mustapha, Z. A. (2013). Housing under the pyramid reduces susceptibility of hippocampal CA3 pyramidal neurons to prenatal stress in the developing rat offspring. *Indian Journal of Experimental Biology, 51*(12), 1070–1078.

Nelson, C. A., & Carver, L. J. (1998). The effects of stress and trauma on brain and memory: A view from developmental cognitive neuroscience. *Development and Psychopathology, 10*(4), 793–809.

Nemoda, Z., Massart, R., Suderman, M., Hallett, M., Li, T., Coote, M., et al. (2015). Maternal depression is associated with DNA methylation changes in cord blood T lymphocytes and adult hippocampi. *Translational Psychiatry, 7*(5), e545.

Nikolova, Y. S., & Hariri, A. R. (2015). Can we observe epigenetic effect on human brain function? *Trends in Cognitive Sciences, 19*(7), 366–373.

O'Connor, T. G., Bergman, K., Sarkar, P., & Glover, V. (2013). Prenatal cortisol exposure predicts infant cortisol response to acute stress. *Developmental Psychobiology, 55*(2), 145–155.

Panter-Brick, C., Grimon, M. P., & Eggerman, M. (2014). Caregiver–child mental health: A prospective study in conflict and refugee settings. *Journal of Child Psychology and Psychiatry, 55*(4), 313–327.

Pears, K. C., & Fisher, P. A. (2005). Emotion understanding and theory of mind among maltreated children in foster care: Evidence of deficits. *Development and Psychopathology, 17*(1), 47–65.

Pfefferbaum, B., Pfefferbaum, R. L., Gurwitch, R. H., Nagumalli, S., Brandt, E. N., Robertson, M. J., et al. (2003). Children's response to terrorism: A critical review of the literature. *Current Psychiatry Reports, 5*(2), 95–100.

Pine, D. S., Costello, J., & Masten, A. (2005). Trauma, proximity, and developmental psychopathology: The effects of war and terrorism on children. *Neuropsychopharmacology, 30*(10), 1781–1792.

Powers, A., Fani, N., Cross, D., Ressler, K. J., & Bradley, B. (2016). Childhood trauma, PTSD, and psychosis: Findings from a highly traumatized, minority sample. *Child Abuse and Neglect, 58,* 111–118.

Pynoos, R. S., Schreiber, M. D., Steinberg, A. M., & Pfefferbaum, B. J. (2005). Impact of terrorism on children. In B. J. Sadock & V. A. Sadock (Eds.), *Comprehensive textbook of psychiatry* (Vol. 2, pp. 3551–3564). Philadelphia: Lippincott, Williams & Wilkins.

Reed, R. V., Fazel, M., Jones, L., Panter-Brick, C., &

Stein, A. (2012). Mental health of displaced and refugee children resettled in low-income and middle-income countries: Risk and protective factors. *Lancet, 379,* 250–265.

Rogosch, F. A., Dackis, M. N., & Cicchetti, D. (2011). Child maltreatment and allostatic load: Consequences for physical and mental health in children from low-income families. *Developmental Psychopathology, 23*(4), 1107–1124.

Rubin, M., Shvil, E., Papini, S., Chhetry, B. T., Helpman, L., Markowitz, J. C., et al. (2016). Greater hippocampal volume is associated with PTSD treatment response. *Psychiatry Research: Neuroimaging, 252,* 36–39.

Ryan, R., Booth, S., Spathis, A., Mollart, S., & Clow, A. (2016). Use of salivary diurnal cortisol as an outcome measure in randomized controlled trials: A systematic review. *Annals of Behavioral Medicine, 50*(2), 210–236.

Saar-Ashkenazy, R., Cohen, J. E., Guez, J., Gasho, C., Shelef, I., Friedman, A., et al. (2014). Reduced corpus callosum volume in posttraumatic stress disorder highlights the importance of interhemispheric connectivity for associative memory. *Journal of Traumatic Stress, 27*(1), 18–26.

Sakamoto, H., Fukuda, R., Okuaki, T., Rogers, M., Kasai, K., Machida, T., et al. (2005). Parahippocampal activation evoked by masked traumatic images in posttraumatic stress disorder: A functional MRI study. *NeuroImage, 26*(3), 813–821.

Sapolsky, R. M. (2000). Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. *Archives of General Psychiatry, 57*(10), 925–935.

Sapolsky, R. M., Uno, H., Rebert, C. S., & Finch, C. E. (1990). Hippocampal damage associated with prolonged glucocorticoid exposure in primates. *Journal of Neuroscience, 10,* 2897–2902.

Schechter, D. S., Brunelli, S. A., Cunningham, N., Brown, J., & Baca, P. (2002). Mother–daughter relationships and child sexual abuse: A pilot study of 35 mothers and daughters (ages 1–9 years). *Bulletin of the Menninger Clinic, 66*(1), 39–60.

Schechter, D. S., Gross, A., Willheim, E., McCaw, J., Turner, J. B., Myers, M. M., et al. (2009). Is Maternal PTSD associated with greater exposure of very young children to violent media? *Journal of Traumatic Stress, 22*(6), 658–662.

Schechter, D. S., Coots, T., Zeanah, C. H., Davies, M., Coates, S. W., Trabka, K. A., et al. (2005). Maternal mental representations of the child in an inner-city clinical sample: violence-related posttraumatic stress and reflective functioning. *Attachment and Human Development, 7*(3), 313–331.

Schechter, D. S., Moser, D. A., McCaw, J. E., & Myers, M. M. (2014). Autonomic functioning in mothers with interpersonal violence-related posttraumatic stress disorder in response to separation–reunion. *Developmental Psychobiology, 56*(4), 748–760.

Schechter, D. S., Moser, D. A., Paoloni-Giacobino, A., Stenz, L., Gex-Fabry, M., Aue, T., et al. (2015). Methylation of NR3C1 is related to maternal PTSD, parenting stress and maternal medial prefrontal cortical activity in response to child separation among mothers with histories of violence exposure. *Frontiers in Psychology, 6,* 690.

Schechter, D. S., Moser, D. A., Pointet, V. C., Aue, T., Stenz, L., Paoloni-Giacobino, A., et al. (2017). The association of serotonin receptor 3A methylation with maternal violence exposure, neural activity, and child aggression. *Behavioural Brain Research, 325,* 268–277.

Schechter, D. S., Moser, D. A., Reliford, A., McCaw, J. E., Coates, S. W., Turner, J. B., et al. (2015). Negative and distorted attributions towards child, self, and primary attachment figure among posttraumatically stressed mothers: What changes with clinician assisted videofeedback exposure sessions (CAVES). *Child Psychiatry and Human Development, 46*(1), 10–20.

Schechter, D. S., Moser, D. A., Wang, Z., Marsh, R., Hao, X., Duan, Y., et al. (2012). An fMRI study of the brain responses of traumatized mothers to viewing their toddlers during separation and play. *Social Cognitive and Affective Neuroscience, 7*(8), 969–979.

Schechter, D. S., Myers, M. M., Brunelli, S. A., Coates, S. W., Zeanah, C. H., Davies, M., et al. (2006). Traumatized mothers can change their minds about their toddlers: Understanding how a novel use of video-feedback supports positive change of maternal attributions. *Infant Mental Health Journal, 27*(5), 429–447.

Schechter, D. S., Willheim, E., McCaw, J., Turner, J. B., Myers, M. M., & Zeanah, C. H. (2011). The relationship of violent fathers, posttraumatically stressed mothers, and symptomatic children in a preschool-age inner-city pediatrics clinic sample. *Journal of Interpersonal Violence, 26*(18), 3699–3719.

Scheeringa, M. S., Myers, L., Putnam, F. W., & Zeanah, C. H. (2015). Maternal factors as moderators or mediators of PTSD symptoms in very young children: A two-year prospective study. *Journal of Family Violence, 30*(5), 633–642.

Scheeringa, M. S., Wright, M. J., Hunt, J. P., & Zeanah, C. H. (2006). Factors affecting the diagnosis and prediction of PTSD symptomatology in children and adolescents. *American Journal of Psychiatry, 163,* 644–651.

Scheeringa, M. S., & Zeanah, C. H. (1995). Symptom expression and trauma variables in children under 48 months of age. *Infant Mental Health Journal, 16,* 259–270.

Scheeringa, M. S., & Zeanah, C. H. (2001). A relational perspective on PTSD in early childhood. *Journal of Traumatic Stress, 14*(4), 799–815.

Scheeringa, M., & Zeanah, C. (2008). Reconsideration of harm's way: Onsets and comorbidity patterns in preschool children and their caregivers following Hurricane Katrina. *Journal of Clinical Child and Adolescent Psychology, 37,* 508–518.

Scheeringa, M., Zeanah, C. H., Myers, L., & Putnam, F. (2003). New findings on alternative criteria for PTSD in preschool children. *Journal of the American Academy of Child and Adolescent Psychiatry, 42,* 561–570.

Schreiber, A. L., Lu, Y. L., Baynes, B. B., Richardson, H. N., & Gilpin, N. W. (2017). Corticotropin-releasing factor in ventromedial prefrontal cortex mediates avoidance of a traumatic stress-paired context. *Neuropharmacology, 113*(Pt. A), 323–330.

Seng, J. S., Graham-Bermann, S. A., Clark, M. K., McCarthy, A. M., & Ronis, D. L. (2005). Posttraumatic stress disorder and physical comorbidity among female children and adolescents: Results from service-use data. *Pediatrics, 116*(6), e767–e776.

Shin, L. M., Whalen, P. J., Pitman, R. K., Bush, G., Macklin, M. L., Lasko, N. B., et al. (2001). An fMRI study of anterior cingulate function in posttraumatic stress disorder. *Biological Psychiatry, 50*(12), 932–942.

Simcock, G., & DeLoache, J. (2006). Get the picture?: The effects of iconicity on toddlers' reenactment from picture books. *Developmental Psychology, 42*(6), 1352–1357.

Slone, M., & Mann, S. (2016). Effects of war, terrorism and armed conflict on young children: A systematic review. *Child Psychiatry and Human Development, 47*(6), 950–965.

Stein, A. H., & Friedrich, L. K. (1972). Television content and young children's behavior. In J. P. Murray, E. A. Rubinstein, & G. A. Comstock (Eds.), *Television and social behavior: Vol. 2. Television and social learning* (pp. 202–317). Washington, DC: U.S. Government Printing Office.

Sugaya, L., Hasin, D. S., Olfson, M., Lin, K. H., Grant, B. F., & Blanco, C. (2012). Child physical abuse and adult mental health: A national study. *Journal of Traumatic Stress, 25*(4), 384–392.

Sumner, J. A., McLaughlin, K. A., Walsh, K., Sheridan, M. A., & Koenen, K. C. (2014). CRHR1 genotype and history of maltreatment predict cortisol reactivity to stress in adolescents. *Psychoneuroendocrinology, 43,* 71–80.

Swain, J. E., Lorberbaum, J. P., Kose, S., & Strathearn, L. (2007). Brain basis of early parent–infant interactions: Psychology, physiology, and in vivo functional neuroimaging studies. *Journal of Child Psychology and Psychiatry, 48*(3–4), 262–287.

Teicher, M. H. (2005, October). *Childhood abuse and regional brain development: Evidence for sensitive periods.* Paper presented at the American Academy of Child and Adolescent Psychiatry, Toronto, Ontario, Canada.

Teicher, M. H., Andersen, S. L., Polcari, A., Anderson, C. M., Navalta, C. P., & Kim, D. M. (2003). The neurobiological consequences of early stress and childhood maltreatment. *Neuroscience and Biobehavioral Reviews, 27*(1–2), 33–44.

Teicher, M. H., Tomoda, A., & Andersen, S. L. (2006). Neurobiological consequences of early stress and childhood maltreatment: Are results from human and animal studies comparable? *Annals of the New York Acadamy of Sciences, 1071,* 313–323.

Thabet, A. A. M., Karim, K., & Vostanis, P. (2006). Trauma exposure in preschool children in a war zone. *British Journal of Psychiatry, 188,* 154–158.

Turk, J., Robbins, I., & Woodhead, M. (2005). Posttraumatic stress disorder in young people with intellectual disability. *Journal of Intellectual Disability Research, 49,* 872–875.

Tursich, M., Neufeld, R. W., Frewen, P. A., Harricharan, S., Kibler, J. L., Rhind, S. G., Lanius, R. A. (2014). Association of trauma exposure with proinflammatory activity: A transdiagnostic meta-analysis. *Translational Psychiatry, 22*(4), e413.

Udo, I. E., Sharps, P., Bronner, Y., & Hossain, M. B. (2016). Maternal intimate partner violence: Relationships with language and neurological development of infants and toddlers. *Maternal and Child Health Journal, 20*(7), 1424–1431.

United Nations International Children's Emergency Fund (UNICEF). (2016). *Uprooted: The growing crisis for refugee and migrant children.* New York: Author.

United Nations Refugee Agency (UNHCR). (2015). Global trends: Forced displacement in 2015. Retrieved from *www.unhcr.org/en-us/statistics/unhcrstats/576408cd7/unhcr-global-trends-2015.html.*

U.S. Department of Health and Human Services, Administration on Children, Youth, and Families. (2014). *Child Maltreatment 2014.* Washington, DC: U.S. Government Printing Office.

Vanderwert, R. E., Zeanah, C. H., Fox, N. A., & Nelson, C. A., III. (2016). Normalization of EEG activity among previously institutionalized children placed into foster care: A 12-year follow-up of the Bucharest Early Intervention Project. *Developmental Cognitive Neuroscience, 17,* 68–75.

Wang, Y., Nomura, Y., Pat-Horenczyk, R., Doppelt, O., Abramovitz, R., Brom, D., et al. (2006). Association of direct exposure to terrorism, media exposure to terrorism, and other trauma with emotional and behavioral problems in preschool children. *Annals of the New York Acadamy of Sciences, 1094,* 363–368.

Weinfield, N. S., Whaley, G. J., & Egeland, B. (2004). Continuity, discontinuity, and coherence in attachment from infancy to late adolescence: Sequelae of organization and disorganization. *Attachment and Human Development, 6*(1), 73–97.

Wing, R., Gjelsvik, A., Nocera, M., & McQuaid, E. L. (2015). Association between adverse childhood experiences in the home and pediatric asthma. *Annals of Allergy, Asthma and Immunology, 114*(5), 379–384.

Wingo, A. P., Almli, L. M., Stevens, J. S., Klengel, T., Uddin, M., Li, Y., et al. (2015). DICER1 and microRNA regulation in post-traumatic stress disorder with comorbid depression. *Nature Communications, 3*(6), 10106.

Yehuda, R., Bell, A., Bierer, L. M., & Schmeidler, J. (2008). Maternal, not paternal, PTSD is related to increased risk for PTSD in offspring of Holocaust survivors. *Journal of Psychiatric Research, 42*(13), 1104–1111.

Yehuda, R., Engel, S. M., Brand, S. R., Seckl, J., Marcus, S. M., & Berkowitz, G. S. (2005). Transgenerational effect of posttraumatic stress disorder in babies of mothers exposed to the World Trade Center attacks during pregnancy. *Journal of Clinical Endocrinology and Metabolism, 90*(7), 4115–4118.

Yehuda, R., Flory, J. D., Bierer, L. M., Henn-Haase, C., Lehrner, A., Desarnaud, F., et al. (2015). Lower methylation of glucocorticoid receptor gene promoter 1F in peripheral blood of veterans with posttraumatic stress disorder. *Biological Psychiatry, 77*(4), 356–364.

Zhao, M., Yu, Z., Zhang, Y., Huang, X., Hou, J., Zhao, Y., et al. (2016). Iron-induced neuronal damage in a rat model of post-traumatic stress disorder. *Neuroscience, 330,* 90–99.