Neurobiological Development in Foster Youth: Outcomes and Intervention

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Foster youth are exposed to various types of adversity, such as maltreatment and disruptions in relationships, which can significantly undermine healthy neurobiological development. Early in life, caregivers serve a key role in co-regulating children’s emotions, behaviors, and even physiology (Hofer, 1994). Developing neurobiological systems in a child depend on responsive and sensitive caregiving (Shonkoff, 2010). When there are threats to the caregiving system—such as parental substance abuse that leaves a parent disengaged and unresponsive to his or her infant’s cues, extreme poverty that prevents a parent from meeting his or her child’s basic needs for food, or domestic violence that interferes with a parent’s ability to keep his or her child safe—healthy neurobiological development is undermined in ways that have lasting consequences for the affected child (Gunnar & Fisher, 2006; Sullivan, 2012; Teicher & Samson, 2016). In addition to being exposed to such adversities with their biological parents, foster youth experience the added threat of separation.

Neurobiological consequences of maltreatment and foster care placement can be seen in regulation of the hypothalamus pituitary adrenal (HPA) axis, brain development, and immune system functioning. Disruptions to the neurobiology of foster youth may mediate pathways toward later mental and physical health problems, suggesting that these systems are important targets of interventions for foster youth. Indeed, a number of interventions for foster youth have been found to improve functioning of key neurobiological systems. In this chapter, we first present several theoretical perspectives on the ways that stress may undermine healthy neurobiological development. We then describe research regarding the neurobiological consequences of maltreatment and foster care placement on (1) HPA axis functioning, (2) brain development, and (3) immune system functioning. For each system, we consider typical and atypical neurobiological development, the behavioral outcomes associated with atypical development, and interventions that target each system in vulnerable youth. Finally, we discuss implications and considerations for future research.

Theoretical Perspectives on Neurobiological Consequences of Early Adversity

Several theories and frameworks have been proposed to help guide our understanding of how stressful experiences in a child’s environment may become biologically embedded. Here, we consider some of these theories as well as general considerations that are critical to informing how foster care and associated experiences may affect children on a neurobiological level.
When broadly considering the neurobiology of stress, “allostasis” and “allostatic load” are useful terms that help us to understand how overwhelming stress leads to physiological wear and tear on the body (McEwen, 2000). Allostasis refers to the process of maintaining stability (i.e., homeostasis) through adaptation and change. Allostatic load, then, refers to the physiological costs of repeatedly adapting to adverse environmental conditions. Allostatic load has been quantified by summing physiological indicators, such as atypical cortisol levels, high blood pressure, and elevated body mass index (Miller, Chen, & Parker, 2011). This has been a useful approach for examining the cumulative effects of stress, with studies showing evidence of allostatic load among samples exposed to neighborhood poverty (e.g., Brody, Lei, Chen, & Miller, 2014), family poverty (e.g., Chen, Miller, Kobor, & Cole, 2011), and child maltreatment (e.g., Danese et al., 2009). Allostatic load predicts age-related diseases and mortality, and thus can be considered an indicator of general physical health risk (Karlamangla, Singer, & Seeman, 2006).

Although much of this chapter will focus on the main effects of adversity on neurobiological outcomes, foster youth facing similar circumstances may show vastly different outcomes. Some theories have posed frameworks for understanding this phenomenon of individual differences. Boyce and Ellis (2005) proposed the biological sensitivity to context model, an evolutionary-developmental theory about stress responsiveness to the environment. This model suggests that physiological mechanisms (e.g., autonomic nervous system reactivity, cortisol regulation, immune reactivity) moderate the effects of environmental stress on outcomes. Thus, individual differences in the magnitude of stress responses modulate how susceptible a child is to environmental influences. Similarly, the differential susceptibility model suggests that some children are more susceptible to their caregiving environments than others—including both the negative effects of low-quality caregiving and the positive effects of high-quality caregiving (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007). Both temperamental and genetic factors have been considered in explaining children’s differential susceptibility to environmental influences. This model has implications for intervention effectiveness, as some children may be more or less responsive to parenting-focused interventions (Bakermans-Kranenburg, van IJzendoorn, Mesman, Alink, & Juffer, 2008).

Finally, in understanding how adverse childhood experiences influence neurobiological development, it is critical to consider developmental timing. There may be sensitive periods (e.g., infancy and preadolescence), during which neurobiological systems are most open to environmental influences. The Bucharest Early Intervention Project used an experimental design to examine the effects of early institutional care on young children’s development. Findings suggested that timing is of the essence for remediating effects of early deprivation on children’s development across domains of cognitive development (Almas, Degnan, Nelson, Zeanah, & Fox, 2016), cellular aging (Drury et al., 2012), and brain function (Vanderwert, Marshall, Nelson, Zeanah, & Fox, 2010). Across these outcomes, children placed sooner into family foster care (usually under 24 months) tend to show better catch-up developmentally than children who experienced prolonged institutional care.

With these theoretical perspectives and other considerations in mind, we now turn to a discussion of how development is threatened by exposure to adversities commonly faced by foster youth. We describe the typical development of key neurobiological systems, how development may go awry in the context of maltreatment and foster care, and interventions that target issues at the level of neurobiological functioning.

**HPA Axis and Stress Response Systems**

**Typical Development: Stress Reactivity and Diurnal Rhythm**

The hypothalamic pituitary adrenal (HPA) axis serves as one of the body’s main stress response systems. When an individual encounters a stressor, the HPA axis initiates a cascade of hormonal
reactions, resulting in the output of cortisol, a steroid hormone. This release of cortisol leads to increased availability of glucose and directs resources away from processes less critical for survival (e.g., digestion, reproduction), which support an adaptive response to the stressor. As soon as children are born, they show increased cortisol when faced with stressors, such as physical examinations or heel lancets (Gunnar & Fisher, 2006). However, there is a developmentally normative shift in cortisol reactivity throughout the first year of life, such that stressors no longer cause an increase in cortisol. In typically-developing children, this hyporesponsive period (i.e., period of dampened reactivity) persists for much of childhood, with cortisol reactivity being reliably established again during adolescence (Gunnar & Donzella, 2002). It has been suggested that this prolonged period of hypo-responsivity may reflect an evolutionary adaptation that serves to protect the developing brain from high levels of glucocorticoids (Gunnar & Fisher, 2006).

In addition to its role in responding to a stressor (i.e., cortisol reactivity), the HPA axis also maintains a circadian pattern of cortisol production (i.e., cortisol diurnal rhythm). Typically, individuals show a distinct diurnal rhythm of cortisol secretion, in which levels are high when the individual wakes up, peak about 30 minutes after wake-up, and decrease steadily throughout the day, reaching a nadir around bedtime. This pattern develops around 3 months of age (Price, Close, & Fielding, 1983) and becomes fully established by preschool age (Gunnar & Donzella, 2002). This diurnal rhythm of cortisol supports regulatory functions in the body, such as metabolism, temperature regulation, and maintaining circadian sleep/wake patterns (Baum & Contrada, 2010).

**HPA Axis Functioning of Foster Youth**

Children in foster care often show atypical patterns of cortisol reactivity and diurnal cortisol rhythms. There are more studies characterizing diurnal cortisol activity among children in foster care than studies characterizing cortisol reactivity, so we will begin by describing the former. As described above, typical diurnal cortisol rhythms are characterized by high levels of cortisol in the morning, followed by a steep decline in cortisol throughout the day, and low levels of cortisol at bedtime. Research has shown that children who experience foster care are more likely to show a blunted diurnal rhythm of cortisol, characterized by low morning levels and less of a decline throughout the day (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010; Bruce, Fisher, Pears, & Levine, 2009; Dozier et al., 2006; Gunnar & Vazquez, 2001). This blunted pattern of cortisol dysregulation is also found among children who experience early maltreatment (i.e., abuse or neglect), but remain in the care of their biological families (Bernard, Zwerling, & Dozier, 2015; Doom, Cicchetti, & Rogosch, 2014).

In fact, some research shows that children who remain with their birth parents after investigation by child protective services show more dysregulated cortisol production than children who are removed from their parents’ care and placed in foster care (i.e., Bernard et al., 2010), suggesting that foster care may reflect an effective intervention for vulnerable children. Daily patterns of cortisol may vary based on the type of maltreatment the child experienced prior to being placed in foster care (Bruce, Fisher, et al., 2009; Cicchetti, Rogosch, Gunnar, & Toth, 2010). For instance, Bruce, Fisher, and colleagues (2009) found that foster children who experienced severe physical neglect (e.g., failure to provide adequate food or medical care) had low morning cortisol, whereas foster children who experienced severe emotional maltreatment (e.g., parental rejection, failure to protect from traumatic events) had high morning cortisol.

This pattern of low cortisol among foster youth shows longitudinal stability in childhood (Laurent, Gilliam, Bruce, & Fisher, 2014), but it is unclear if the pattern persists into adulthood. Some studies suggest that adults with a history of foster care or early maltreatment show increased cortisol output (Gonzalez, Jenkins, Steiner, & Fleming, 2012; Johnson & Tottenham, 2015; Nicolson, Davis, Kruszewski, & Zautra, 2010), whereas other studies suggest blunted cortisol patterns in adult samples (Monteleone et al., 2015; Shea et al., 2007; Weissbecker, Floyd, Dedert, Salmon, & Sephton, 2006).
Given that dysregulated cortisol rhythms have been linked to behavioral and health problems, as described next, it is important to understand how cortisol outcomes are affected longitudinally; future research in this area is needed.

Disruption of the HPA axis can have implications for children’s socioemotional and physical development. Numerous studies have shown that cortisol patterns are related to children’s risk for developing psychological disorders. Low levels of basal cortisol (McBurnett, Lahey, Rathouz, & Loeber, 2000; Shirtcliff, Granger, Booth, & Johnson, 2005) and blunted cortisol reactivity (Snoek, van Goozen, Matthys, Buitelaar, & van Engeland, 2004; van Goozen et al., 1998) have been associated with externalizing behavior problems (e.g., aggression, oppositional/defiant behavior). In contrast, patterns of high cortisol and hyper-reactivity have been linked to internalizing problems, such as depression (Burke, Davis, Otte, & Mohr, 2005; Lopez-Duran, Kovacs, & George, 2009). These associations may differ based on the age of the child. For instance, Alink and colleagues (2008) found that high basal cortisol was associated with externalizing problems in preschool children, but low basal cortisol was associated with these problems in elementary school children. Dysregulation of the HPA axis can also have implications for physical health. Some studies suggest that increased cortisol secretion contributes to increased risk for obesity (e.g., Warne, 2009) and mediates the relationship between psychosocial stress and cardiovascular disease (Chandola et al., 2008). In addition, low secretion of cortisol places children at risk for autoimmune and inflammatory disorders, including asthma (e.g., Wright, 2011).

Interventions that Enhance HPA Axis Regulation

Several interventions have been shown to impact HPA axis functioning among children who experience early adversity or are placed in foster care (Slopen, McLaughlin, & Shonkoff, 2010). Two such interventions, Multidimensional Treatment Foster Care for Preschoolers (MTFC-P; Fisher, Stoolmiller, Gunnar, & Burraston, 2007) and Attachment and Biobehavioral Catch-Up (ABC; Dozier, Meade, & Bernard, 2014), have integrated HPA regulation into their theories of change and specifically target cortisol as a marker of treatment efficacy.

Multidimensional Treatment Foster Care for Preschoolers (Fisher & Chamberlain, 2000) is a team-based therapeutic intervention delivered to foster children, foster parents, and permanent placement resources (e.g., birth parents, adoptive relatives). The intervention consists of intensive caregiver training in which parents are taught to cultivate a warm, responsive, and consistent environment at home. In addition, children take part in individualized behavioral treatment focusing on prosocial skills and problem-solving. Families typically receive services for 6 to 9 months, and children are followed across placements to promote consistency through each transition (Fisher & Chamberlain, 2000). In a longitudinal randomized control trial comparing MTFC-P participants to those in regular foster care and a community comparison group, Fisher and colleagues (2007) demonstrated that MTFC-P can impact diurnal rhythms of cortisol. Children receiving MTFC-P showed blunted diurnal cortisol at baseline, but their cortisol patterns became more typical over time such that they were comparable to the community sample by the end of 1 year. In contrast, children in regular foster care showed increasingly flattened diurnal cortisol rhythms over the course of the year (Fisher et al., 2007). In addition to these neurobiological outcomes, MTFC-P has been found to enhance placement stability and attachment-related behaviors, which may further support healthy HPA axis functioning (Fisher & Kim, 2007; Fisher, Kim, & Pears, 2009).

Attachment and Biobehavioral Catch-Up (Dozier et al., 2014) is a 10-week home-based intervention for parents and infants who are involved with foster care or at high risk for neglect. The ABC intervention aims to improve parent–child attachment by coaching parents to respond in nurturing ways to their child’s cues of distress, follow their child’s lead, and reduce their frightening or harsh behavior. In addition to showing effects on parent sensitivity (Bick & Dozier, 2013) and
child attachment (Bernard et al., 2012), a randomized control trial demonstrated that infants who receive this intervention show higher morning cortisol levels and a steeper decline in cortisol across the day than children who receive an educational intervention (Bernard, Dozier, Bick, & Gordon, 2015). At a preschool follow-up (approximately 3 years post-intervention), children who received ABC as infants still had more normalized cortisol than children who received the control intervention (Bernard, Hostinar, & Dozier, 2015), indicating that this intervention has lasting effects on children’s HPA axis functioning. These interventions illustrate the malleable nature of the HPA axis: the system is vulnerable to the damaging effects of early life stress, but also amenable to change in the context of well-timed interventions.

**Brain Structure and Function**

**Typical Development: Prefrontal Cortex and Amygdala**

Next, we turn to research suggesting that adverse experiences commonly faced by foster youth can alter the developing brain. Here, we focus specifically on the prefrontal cortex and the amygdala.

The prefrontal cortex is responsible for a number of executive functions, such as inhibitory control, cognitive flexibility, and problem-solving. Given its connections with other brain regions, it also has a role in many other aspects of behavioral and emotional regulation. With its development extending from birth through to adulthood (Rubia et al., 2006), the prefrontal cortex is susceptible to environmental experiences across development. However, there may be particular developmental windows when the prefrontal cortex has heightened sensitivity to environmental stress. During infancy and then again during adolescence, the density of glucocorticoid receptors in the prefrontal cortex peaks; thus, prolonged or frequent exposure to stressors may impact the developing brain during these times (Teicher & Samson, 2016).

Along with other structures in fronto-limbic networks, the amygdala plays an important role in perceiving emotions, evaluating threat, processing emotional information, and regulating emotion. Similar to the prefrontal cortex, the amygdala has a high density of glucocorticoid receptors. During the first several years of life, the amygdala goes through rapid development, making early childhood a possible sensitive period during which environmental stress may affect structural development; preadolescence may represent another sensitive period, as amygdala growth peaks at this time, followed by synaptic pruning, the selective elimination of neural connections (Uematsu et al., 2012).

**Brain Structure and Function of Foster Youth**

Although research on brain structure and function of foster youth is quite limited, research about the impact of maltreatment and early life stress on the brain provides relevant insights. For both the prefrontal cortex and amygdala, we describe structural and functional changes that may result from experiences common among foster youth, such as exposure to maltreatment, deprivation, and disruptions in care.

A number of studies have shown decreased volume in areas of the prefrontal cortex in children following experiences of maltreatment, such as physical abuse (e.g., Hanson et al., 2010), as well as in children with interpersonal trauma and symptoms of post-traumatic stress disorder (PTSD; e.g., Carrion, Garrett, Menon, Weems, & Reiss, 2008). Similar findings of reduced grey matter volume in areas of the prefrontal cortex have been found in adults who were exposed to childhood sexual abuse (Andersen et al., 2008) and physical abuse (Tomoda et al., 2009).

Given that the prefrontal cortex is primarily involved in executive functioning, functional magnetic resonance imaging (fMRI; i.e., a neuroimaging approach that measures brain activity by detecting changes in blood flow) studies have examined impairment of the prefrontal cortex by using
response inhibition tasks. For example, Mueller et al. (2010) examined cognitive control using a task that required adolescents to inhibit a dominant “go” response (i.e., pressing a button in response to seeing a letter) and switch to an alternative response (i.e., pressing a different button in response to that letter, when they were shown a signal to change the response). Adolescents (approximately 13 years old) in the “early life stress” group had been adopted following foster care or institutional care; on average, the sample spent approximately 28 months in out-of-home care, with several having experienced multiple placements. The adolescents in the early life stress group showed greater activation in regions involved in inhibitory control, such as the inferior prefrontal cortex, among other areas, relative to a control group. Additionally, behavioral evidence suggested impairment in switching from the dominant response to the alternative response when prompted to do so. Taken together, these deficits have implications for their behavioral functioning in areas of attention, inhibitory control, and learning.

Children in foster care show elevated rates of attention deficit hyperactivity disorder (ADHD), difficulty with learning, and behavioral problems (Lewis, Dozier, Ackerman, & Sepulveda-Kozakowski, 2007; Pears & Fisher, 2005), which all reflect deficits in processes largely mediated by the prefrontal cortex (Booth et al., 2005). These deficits, in turn, have implications for children’s school readiness and academic success. Indeed, children in foster care tend to show poorer academic competence and poorer math and reading achievement than peers (Berger, Cancian, Han, Noyes, & Rios-Salas, 2015; Pears, Fisher, Bruce, Kim, & Yoerger, 2010).

In general, studies have not found evidence that the structure of the amygdala is affected by abuse and neglect in the context of biological families (Tottenham & Sheridan, 2009). However, institutional care, which reflects a more extreme form of deprivation, has been associated with increased amygdala volume. Tottenham et al. (2010) found that children adopted from institutional care showed enlarged amygdala volume years after adoption, with the length of time spent in institutional care positively correlated with amygdala volume; further, amygdala volume was associated with greater child anxiety (Tottenham et al., 2010).

Maheu et al. (2010) examined amygdala functioning in children in foster care and adopted from institutional care. Children with histories of foster or institutional care showed greater amygdala activation to fearful and angry faces than to neutral faces, relative to comparison children (Maheu et al., 2010). Further, more changes of placement and less time with the adoptive family were associated with greater magnitude of the angry versus neutral contrast in amygdala activation, suggesting that out-of-home placement history has an influence on amygdala function. Similar findings have been seen among institutionalized children, with heightened amygdala activation to threatening stimuli has been associated with poorer social competence and less eye contact (Tottenham et al., 2011). In addition to fMRI studies, studies using electroencephalogram (EEG) methodology have also shown functional differences in how the brain processes emotion following experiences of early adversity. Event-related potentials (ERPs) can be extracted from ongoing EEG recorded from the scalp, and reflect changes in electrical activity in the brain that occur in response to a specific event, such as seeing a face on a computer screen. In studies of physically abused children, Pollak and colleagues found that abused children showed larger ERP responses to angry target faces than to neutral faces (Pollak, Klorman, Thatcher, & Cicchetti, 2001). Behaviorally, physically abused children also show enhanced attention to and processing of threatening information, such as angry faces (Pollak, 2008).

Children in foster care show elevated rates of internalizing problems, such as anxiety, difficulty regulating emotions, and poorer socioemotional competence (Kaplow & Widom, 2007; Slopen et al., 2010), which probably, at least in part, reflect altered functioning of the amygdala. Indeed, greater amygdala activation to threatening stimuli has been associated with poorer social competence and less eye contact (Tottenham et al., 2011). Further, heightened electrophysiological responses of maltreated children to cues of anger may contribute to maladaptive threat detection, leading to hostile attribution biases, elevated aggressive behavior, and difficulty perceiving, expressing, and regulating emotions.
Taken together, findings across studies highlight structural and functional impairment in brain regions of the prefrontal cortex and amygdala following experiences of deprivation, maltreatment, and foster care. Other brain regions not reviewed here, such as the hippocampus, also show impairments following early adversity (for a review, see Teicher & Samson, 2016).

Interventions that Enhance Brain Structure and Function

Few interventions for foster children, or even more generally for children exposed to early adversity, have examined effects on the brain. There are a couple of notable exceptions. In the Bucharest Early Intervention Project (Zeanah et al., 2003), institutionalized children were randomly assigned to care as usual (i.e., initially staying in the institution) or high-quality family foster care. At baseline, when examining resting state EEG activity, institutionalized children showed greater EEG power in the theta band and reduced power in the alpha and beta bands (Marshall, Fox, & BEIP Core Group, 2004), a pattern similar to children diagnosed with ADHD. Activity in higher frequency bands (i.e., alpha and beta) is associated with better attention and cognitive performance; thus, low power in these bands may reflect impaired or delayed development. At 8 years old, however, children placed into family foster care had greater high frequency (i.e., alpha) EEG power than children in the care-as-usual (i.e., institutional care) group (Vanderwert et al., 2010). Further, placement into family foster care before the age of 2 was associated with the most pronounced effects. These findings suggest that the brain is responsive to improvements in the social environment, but may be less responsive to such recovery as children get older. Along with other findings from the Bucharest Early Intervention Project which show that earlier removal from institutional care is associated with better cognitive, social, and biological outcomes (e.g., Almas et al., 2016; Drury et al., 2012), these findings suggest that timing is of the essence when finding stable and responsive family placements for children.

As described above, MTFC-P is an intervention for preschoolers in foster care that aims to provide a consistent and responsive interpersonal environment in order to reduce behavior problems and improve self-regulation. Foster children who received MTFC-P and foster children who received services as usual were compared on their behavioral and electrophysiological performance on a flanker task requiring cognitive control and response monitoring (Bruce, McDermott, Fisher, & Fox, 2009). The flanker task required children to identify the color of a middle circle in a row of circles, which was either the same color as the other circles (i.e., congruent trials) or a different color (i.e., incongruent trials). Following their response, children received feedback: a smiling face for a correct response, or a frowning face for an incorrect response. Foster children that received MTFC-P showed differences in feedback-locked ERP components (i.e., brain responses to receiving feedback following correct and incorrect trials) compared to foster children who received services as usual. Specifically, MTFC-P foster children showed different neural responses to negative versus positive feedback, similar to non-maltreated children; whereas comparison foster children did not show differences in brain activity between positive versus negative feedback. These results suggest that MTFC-P enhanced children’s responsiveness to external feedback, suggesting potential intervention effects on a key aspect of behavioral regulation. Given that a key aspect of behavioral regulation in early childhood is shifting behavior and attention in response to external feedback (e.g., rules, reinforcement, consequences), these changes in brain activity may affect social and academic functioning.

Immune System Functioning

Typical Development: Immune System

Finally, we turn to the immune system, which is also found to be highly susceptible to environmental input. The basic role of the immune system is to protect the individual from injury or illness by
mounting a response to pathogens. Host defense, or the role of the immune system in protecting the individual from illness and injury, works largely through dispatching both specific and all-purpose cells to protect against pathogens. Host defense can either take the form of natural immunity, in which broad, all-purpose immune cells respond to the antigen, or specific immunity, in which cells with antigen-specific receptor sites mount a full, specialized defense (Cooper et al., 2009; Litman, Cannon, & Dishaw, 2005). In both cases, natural or specific immune cells rely on communication molecules called cytokines to amplify and facilitate immune response, either by promoting inflammation at the site of injury or illness or through directing the production of antibodies to neutralize pathogens (Cannon, 2000).

Although it was previously thought that the immune system’s primary role was to defend against pathogens, current evidence suggests that the immune system may also fill important regulatory or homeostatic functions (Schmitz & Chew, 2008). More specifically, it has been found that inflammatory cytokines are expressed in healthy adult brains without the presence of injury or illness, and experimental evidence suggests that these cytokines may regulate metabolism and sleep behavior (Schmitz & Chew, 2008; Vitkovic, Bockaert, & Jacque, 2001). From a developmental perspective, immune cells are believed to play an important role in normal development of the nervous system, and are crucial in a number of neurodevelopmental processes, such as cell migration and differentiation, synapse formation, and synaptic pruning (Meyer, Feldon, Schedlowski, & Yee, 2006; Nawa & Takei, 2006). At the other end of the developmental spectrum, the immune system also plays a role in supporting healthy aging, particularly through its role in recognizing and eliminating aging, or senescent, cells (Sagiv et al., 2016). Cellular aging typically results from the shortening of caps at the end of each strand of DNA, called telomeres, which function to protect chromosomes.

Immune Functioning of Foster Youth

It has been well documented that the developing immune system is vulnerable to a number of environmental and psychological influences, and that early immune activity may have significant consequences for later health (Bilbo & Schwarz, 2009; Miller et al., 2009; Nusslock & Miller, 2016; Segerstrom & Miller, 2004). Although research on immune system functioning specifically among youth in foster care is lacking, many studies on prenatal adversity and chronic stress offer insights into outcomes we might expect to see for foster youth.

Prenatal infection, particularly during key fetal brain development periods, has been linked to abnormal fetal brain development and increased risk for a number of neurodevelopmental and autoimmune disorders, such as autism, Alzheimer’s, and Parkinson’s (Bilbo & Schwarz, 2009; Brown, 2012; Meyer et al., 2006; Owen, O’Donovan, Thapar, & Craddock, 2011). These associations between early infection and later disorder are consistent with research indicating that immune cells are crucial in neurodevelopment. Many children in foster care have been exposed to poor maternal prenatal health, including infection, illness, and substance use (Simms, Dubowitz, & Szilagyi, 2000). Thus, the inflammatory correlates of poor maternal prenatal health, especially infection, have the potential to significantly alter brain development, placing children at risk for neurodevelopmental and immune-linked disorders over the course of life.

A growing body of research has indicated that chronic psychological stress, such as maltreatment and disruptions in care, may also lead to immune dysfunction in a similar way to infection or illness. Children who experience chronic stress have greater levels of pro-inflammatory cytokines in adulthood (Danese et al., 2009; Danese, Pariente, Caspi, Taylor, & Poulton, 2007; Miller & Cole, 2012), indicating that inflammation may be a major mechanism through which adversity confers later risk for mental and physical health problems. Further, prior to foster care placement, foster children may have experienced homes characterized by limited financial resources, food insecurity, and chaotic and unpredictable schedules (Ehrle, Geen, & Geen, 2002), all of which have the potential
to contribute to immune dysfunction. Additionally, telomeres, the protective caps on the end of chromosomes, have been found to shorten faster in children exposed to maltreatment and other adversities than in non-exposed children (for a meta-analysis, see Ridout et al., 2017). In sum, there is reason to believe that children and adolescents in foster care are at a substantially increased risk for experiencing immune dysfunction secondary to childhood adversity.

Immune dysfunction may exacerbate risk for a number of immune-linked disorders later in life, including metabolic syndrome, obesity, autoimmune disease, and some cancers, as well as psychiatric disorders such as depression and substance abuse, which are thought to have immune correlates (Danese et al., 2009; Nusslock & Miller, 2016; Shonkoff, Boyce, & McEwen, 2009). Bidirectional crosstalk between brain and body suggests that chronic medical conditions may affect threat- and reward-linked brain circuitry, increasing the likelihood of risky health behaviors that perpetuate inflammation and place individuals at risk for a host of negative outcomes.

Interventions Aimed at Enhancing Immune System Functioning

There is much that is yet to be discovered about immune system functioning and early adversity, particularly when considering special populations such as children and adolescents in foster care. However, the growing body of research examining immune correlates of early adversity offers several avenues for interventions that may enhance immune functioning among foster care youth. From a large-scale perspective, interventions providing access to quality prenatal care for at-risk families stand to have significant downstream benefits for children entering foster care. Homeless women, for example, are both more likely to have prenatal infections and be less likely to receive prenatal care (Rimawi, Mirdamadi, & John, 2014). Organizations that support provision of housing services to pregnant homeless women—particularly those that facilitate access to healthcare—may mitigate potentially detrimental effects of prenatal infection on children’s later neurodevelopment and risk for immune dysfunction, even if children are later placed in foster care.

Increasing the quality of medical attention and general care provided in foster care settings may also help to reduce some of the consequences of immune dysfunction resulting from adversity. Some researchers have suggested that pediatricians could play a critical role in ensuring consistent care for children who change placements frequently, perhaps by taking part in a larger team of healthcare workers who track the child’s whereabouts and monitor health conditions over time (Szilagyi, Rosen, Rubin, & Zlotnik, 2015). Relatedly, there is some evidence to suggest that, compared to non-relative foster care, children who are placed with extended family members experience greater placement stability and are healthier (Dubowitz et al., 1992; Rubin et al., 2008). Efforts to prioritize placement of children with extended family may result in more consistent healthcare, thereby curbing some of the immunological consequences of chronic medical conditions.

Finally, sensitive caregiving may mitigate the neurobiological consequences of stress and early adversity (referred to as “social buffering”), including pro-inflammatory signaling and stress responsiveness (Chen et al., 2011; Gunnar & Hostinar, 2015). Indeed, evidence suggests that alterations to caregiving sensitivity may have neurobiological payoffs. For example, Asok, Bernard, Roth, Rosen, & Dozier (2013) found that high-risk children involved with child protective services showed shorter telomeres than low-risk peers, which is considered an important indicator of physical health risk as it is associated with a number of age-related diseases (Epel et al., 2006). However, this effect was moderated by maternal sensitivity; that is, telomere length among high-risk children with highly responsive parents was comparable to that of low-risk children. Given these findings along with previous research that has established a link between maternal warmth and inflammation (e.g., Chen et al., 2011), there is good reason to believe that interventions aimed at enhancing caregiving may be similarly effective in addressing immune dysfunction. The ABC and MTFC-P interventions, described above, have been shown to enhance cortisol regulation, which may have implications for
immune system functioning. Taken together, there are a number of promising intervention avenues that may curb some of the immunological consequences of adversity in foster children and youth.

**Future Directions**

There is much to be discovered about how foster youth are affected on a neurobiological level by their adverse experiences, offering many important directions for future research. First, much of the research reviewed here focused broadly on samples of children or adults who experienced environmental stressors such as maltreatment and poverty. Although such approaches offer strong evidence that adversity leads to maladaptive development, we lack a more nuanced understanding of how the type, severity, chronicity, and developmental timing of such adverse exposures contribute differentially to negative outcomes. Further, and important for understanding the unique experience of foster youth, few studies have examined the specific effects of separations, multiple placement disruptions, and caregiving context (e.g., kinship versus non-relative care) on children’s neurobiological development.

Second, few studies have examined the neurobiological effects of interventions, and even fewer have done so for children in foster care. Given that intervention studies often employ an experimental design (i.e., randomly assigning children to different conditions), they offer key insights into causal mechanisms implicated in positive and negative developmental pathways. By understanding how children’s neurobiology is influenced by changing the environment (e.g., enhancing sensitive parenting) or changing the child’s capacities (e.g., improving self-regulation), researchers can also identify potential targets for intervention.

Finally, it will be important to continue to examine individual differences in neurobiological outcomes. As suggested by theoretical perspectives like the differential susceptibility model, some foster children seem remarkably resilient in the face of challenges, whereas others quickly follow risky trajectories. It will be important to examine how outcomes of youth in foster care vary based on gender, race and ethnicity, and socioeconomic status. Further, understanding temperamental, genetic, or environmental factors that contribute to risk and resilience may help with identifying the children most in need of intervention and tailoring intervention approaches to maximize effects for different children.

Although there are clear benefits to understanding the neurobiological consequences of foster care, there are a number of challenges relating to this work. In addition to the high cost of neuroscience research, it can be difficult for researchers to access vulnerable populations, such as youth in foster care. Thus, collaborative efforts between researchers, care providers, and child welfare agencies are critical for advancing knowledge in this area. Further, researchers should aim to engage families as active scientific partners, rather than passive participants; such efforts to connect through community-based outreach efforts may improve retention in longitudinal studies and enrich the quality and accuracy of data obtained from families. Finally, research on foster youth would benefit from the use of multi-method designs, combining physiological/neurobiological, observational, and self-report assessments from multiple individuals (e.g., foster youth, birth parents, and foster parents) across multiple time-points. Collectively, such methodologically rigorous and community-informed research efforts have the potential to significantly advance theoretical perspectives and bridge science-to-practice/practice-to-science gaps in our knowledge.

**Conclusion**

Foster youth experience a number of threats, such as maltreatment, deprivation, and separation, which interfere with their healthy socioemotional, behavioral, and physical development. Examining neurobiological changes that result from such threats can highlight mechanisms that lead to
psychopathology and physical health conditions. In particular, changes to stress response systems (e.g., HPA axis and cortisol regulation), brain development (e.g., structural and functional changes to the prefrontal cortex and amygdala), and immune system functioning (e.g., inflammatory processes) may offer targets for interventions that aim to prevent long-term consequences among foster youth. Much more research is needed to understand how foster youth, in particular, are affected on a neurobiological level.

References


Neurobiological Development in Foster Youth


53


