The Neurodevelopmental Impact of Stress, Adversity, and Trauma: *Implications for Social Work (part 1)*

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FEATURE

One of the most widely recognized applications of neuroscience as it pertains to clinical social work is our enhanced understanding of how early adversity, trauma, and toxic stress can negatively impact neurodevelopment and, in turn, health and mental health across the life span (Garland & Howard, 2009; Kaufman & Charney, 2001; National Scientific Council on the Developing Child, 2014; Shonkoff et al., 2009). For social workers interested in understanding mechanisms of risk and resiliency, it is important to ask the question, how does exposure to early adversity and stress precipitate neurodevelopmental changes that, in turn, are associated with increased risk behaviors and poor health outcomes? The answer to this question requires multidisciplinary awareness of research from fields such as epidemiology, developmental psychopathology, neuroscience, and, in particular, brain development in early life.

Practitioners who work with young children and their families know that a high percentage of young children are experiencing stress associated with poverty, child abuse and neglect, and separation from caregivers due to factors such as incarceration and substance abuse. The research we review in this chapter shows that these types of early experiences, particularly in the absence

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of supportive care, can set children on a trajectory of disrupted neurodevelopment and risks to psychosocial and health outcomes. Thus, children in particular social contexts (e.g., economically vulnerable families) face not only the challenges of income inequality in a direct sense but are also at increased risk for poor health outcomes, contributing to social disparities in health (Green & Darity, 2010; Jones Harden, Buhler, & Jiminez Parra, 2016).

For social work practitioners interested in the impact of stress on the developing brain, two areas of research are particularly important. First, the Adverse Childhood Experiences (ACE) studies demonstrate, from an epidemiological perspective, the connection between specific types of early adversity and developmental and health outcomes across time. Relatedly, research describes how long-term activation of the stress response system can create neurodevelopmental and neuroendocrine changes that link unmediated stress to health outcomes in particular ways (Gunnar, 1998, 2000; Hart, Gunnar, & Cicchetti, 1996).

This chapter begins with a brief overview of the ACE studies, a set of ground-breaking, epidemiological studies that demonstrated the connection between childhood stress and adversity, and risk for poor health outcomes later in life. Key to understanding this trajectory is an awareness of how stress and adversity precipitate neurodevelopmental and neuroendocrine changes, particularly during sensitive periods of brain development. For this reason, this chapter also provides an overview of the stress response system

and research on the impact of unmediated stress on the developing brain and nervous system. Finally, and from a more hopeful point of view, research on early adversity shows that supportive and secure attachment experiences are important buffers against stress exposure in early childhood (National Scientific Council on the Developing Child, 2007). For this reason, it is vitally important that we understand more about what happens when conditions exist that interfere with the caregiver's ability to provide attuned and responsive care (Perry, Ettinger, Mendelson, & Le, 2010). We briefly consider the issues of maternal depression and parental substance use from this perspective.

THE ACE STUDIES

The ACE studies are multidisciplinary publications that originally stemmed from an epidemiological study undertaken as a collaboration between the Centers for Disease Control and researchers from Kaiser Permanente (Felitti, 2009; Felitti et al., 1998). Based on a 10-item questionnaire that asked adults to retrospectively endorse whether they had experienced specific adverse events as a child, subjects were assigned an ACE score, which represented the number of adverse events endorsed. These events included experiences familiar to clinical social workers who work with vulnerable children, adults, and families and include abuse and neglect; witnessing intimate partner violence; having a parent who abused drugs, had a mental illness, or was incarcerated; and experiencing separation from a parent or primary caregiver. The strongest finding of the early ACE studies was that the higher an individual's ACE score, the more likely he or she was to experience a host of negative health outcomes as an adult, even when other sociodemographic factors were controlled for in data analyses (Dube, Felitti, Dong, Giles, & Anda, 2003).

The ACE pyramid, depicts a trajectory, moving forward in time, that connects early experiences of adversity to neuro-developmental impacts, which in turn are associated with health risk behaviors, early onset of negative health outcomes, and, ultimately, mortality rates (Shonkoff et al., 2012). Specific associations have been documented between higher ACE scores and increased likelihood of neurodevelopmental alterations in areas such as memory (Brown et al., 2007), the emergence of depression and depressed affect (Chapman, Anda, Felitti, Dube, Edwards & Whitfield, 2004), health risk behaviors such as alcohol use and smoking (Dube et al., 2003), and the emergence of chronic disease such as autoimmune diseases and cancer (Brown, Thacker, & Cohen, 2014).

From a social work perspective, research on ACE points to the importance of primary prevention and protecting the developing child from exposure to toxic stress, as well as to the importance of intervention at various points along the ACE pyramid trajectory. The American Academy of Pediatrics argues that research on the connection between early adversity and later health disparities should be integrated with current work in neuroscience and neurodevelopment to create a holistic approach to prevention and intervention. The academy suggests that "research in molecular biology, genomics, immunology,

and neuroscience" must be integrated into our understanding of the processes by which early experiences shape later outcomes in order to inform "science-based strategies to build foundations for children's lifelong health" (Johnson et al., 2011, p. 319). In particular, these researchers point to the quality of early caregiving relationships as key to emerging neuroendocrine, neurobiological, and other body systems that build the foundation for lifelong health (National Scientific Council on the Developing Child, 2007).

THE STRESS RESPONSE SYSTEM

Most clinicians are aware that even age-related changes, normative in nature, create stress of a certain sort for the developing person. A key focus of assessment is often the differentiation of short-term difficulties, perhaps understood as stress reactions to maturational demands, from longer-term, more persistent concerns or exposure to acute or overwhelming stressors that may give rise to serious developmental and mental health vulnerabilities (Boyd-Webb, 2015; Davies, 2010). Embedded in this understanding is the notion that not all stress has long-lasting negative impact and that for development to move forward, some exposure to stress may be necessary and developmentally optimal. Thus, researchers have differentiated among types of stress (Shonkoff & Phillips, 2000).

In a working paper titled, "Excessive Stress Disrupts the Architecture of the Developing Brain" (National Scientific Council on the Developing Child, 2014), experiences of stress are divided into three categories associated with differential activation of the stress

response system. Positive stress is described as short lived in nature, moderate in intensity, and associated with an adaptive and brief activation of stress response systems followed by a timeline return to homeostasis, or baseline. These are the types of stressors that often accompany developmental transitions, such as starting school. With good-enough adult support, the developing child learns to negotiate new maturational tasks and potentially gains a sense of mastery and optimism. At the same time, an event that is manageable to one child may be in a different category for a child carrying preexisting vulnerabilities and experiences of traumatic stress. For a child who has experienced repeated attachment disruptions and lack of access to empathic care, the same event—starting at a new school—may be much more fraught. And, so an example of positive stress can become, for any given individual, more challenging. Tolerable stress refers to those stressors that are significant enough to potentially have longer-term neurobiological and neuroendocrine impacts if not offset by the provision of contingently responsive and empathically attuned support. These types of stressors, depending on the age of the child, will likely require adult intervention to reestablish a sense of homeostatic balance. For this level of stress, children will likely need attuned intervention provided by adults who have some insight into how moderately sustained activation of their stress response system may impact their physiology, behavior, cognition, affect, and even sense of identity. Toxic or traumatic stress refers to intense and sometimes frequent exposure to overwhelming stressors in the absence of supportive and responsive adult care. When children are exposed to toxic or traumatic stress, their stress response systems are activated and remain activated over long periods of time, potentially even after the stressor has been removed. This type of stress has the greatest potential to have negative impacts on the developing brain, such as (1) the volume of different parts of the brain, (2) neuroendocrine changes that may increase individuals' susceptibility to future stressors by increasing their overall level of reactivity to stress exposure, and (3) related impacts on development in multiple spheres, including growth and wellness, cognition and executive functioning, and indexes of emotional well-being and mental health (Loman & Gunnar, 2010; Lupien et al., 2009; Lupien, King, Meaney, & McEwen, 2001; McEwen, 2008; Shonkoff et al., 2009).

Just as not all stressors have negative developmental impacts, it is also true that activation of our stress response systems can be critical to our well-being and survival. An understanding of learning and memory is relevant to clinical social work practice in many ways. For example, research on adolescent brain development and drug use shows that during adolescence, the brain experiences a spurt of growth, resulting in many changes, including an enhanced capacity for learning and memory. While this process can support positive developmental outcomes such as knowledge and skill acquisition, it also makes adolescents particularly susceptible to addiction. The adolescent brain learns to be addicted more quickly than the mature, adult brain and so, drug usage during adolescence may have more immediate, severe, and long-

term consequences (Jensen & Nutt, 2015). LTP refers to the increase in synaptic strength, or neuronal connections, that usually occurs after repeated activation or stimulus exposure. Researchers in addiction have demonstrated in animal studies that molecular changes associated with LTP can be induced with repetitive drug exposure. These changes are termed "drug-evoked synaptic plasticity" (Luscher & Malenka, 2011, p. 1). Gunnar and Vasquez (2006) described the human body's stress response system as psychophysiological in nature, supporting the individual's ability to mobilize energy and resources needed to deal with perceived stressors. When working optimally, the stress response system not only turns on when threats or stressors are perceived, but also turns off, or returns to baseline, when threats have passed. Before detailing specifics of our stress response systems, consider some of the commonly recognized psychophysiological signs of stress. Fear responses, for example, are often accompanied by increased respiration and heart rate, and surges of adrenalin that cause people to become ashen or pale with fright. Other types of stressors may be associated with state dysregulation as evidenced by sleep, appetite, and digestive disruption. We may be familiar with the headaches or stomachaches that accompany stress. Our immune systems may also be negatively impacted by the activation of our stress response systems, giving empirical support to the experience of feeling run down or vulnerable to illness when stressed. These experiences can be

explained, in part, by consequences of our stress response systems being activated.

As with other aspects of neurodevelopment, the stress response system is shaped by many factors in the environment, including access to stable and empathically attuned care. When the developing child experiences consistent exposure to high levels of stress and adversity and lacks access to the buffering effects of responsive caregiving, chronic activation of the stress response system is associated with a range of risks to health and well-being. Below we provide a brief overview of two neurologically mediated pathways associated with the stress response system: the SAM system (sympathetic adrenal medullary) and the HPA axis.

The SAM system involves two aspects of the autonomic nervous system—the sympathetic nervous system and the parasympathetic nervous system. The sympathetic nervous system revs up when a stressor is perceived, resulting in a release of hormones and the fight-or-flight response we commonly recognize. The adrenal gland releases catecholamines from the central part of the adrenal gland, including adrenalin, sometimes referred to as epinephrine, as well as norepinephrine, sometimes referred to as noradrenaline. The release of these hormones is key to the fight-or-flight response. When they are released, we may observe an increase in heart rate, respiration, and dilation of pupils, all signs of increased adrenaline surge. The parasympathetic nervous system operates to counter the effects of the sympathetic nervous system (Ulrich-Lai & Herman, 2009). When the parasympathetic nervous system is activated, level of arousal is decreased, as may be evidenced by a decrease in heart and respiration rates, and the pupils becoming less dilated. Consider for a moment a young infant experiencing distress. When the SAM system is activated, the infant's cry becomes more intense, the legs kick, and the heart rate elevates. Infants and young children will likely need the aid of adult intervention to engage their parasympathetic system—as might be the case in the calming effect of being offered a pacifier for engaging the soothing of nonnutritive sucking, or the vestibular stimulation offered by being held closely and rocked. If effective, these strategies will support the infant's parasympathetic nervous system and speed the return to a state of homeostatic balance. As children move through the rst year of life, they become more able to engage in self-soothing behaviors as simple as sucking on their fingers or thumb.

The HPA axis involves the release of hormones to the rest of the body via their secretion into the bloodstream. The paraventricular nucleus of the hypothalamus secretes stress hormones, including corticotropin releasing hormone and vasopressin. These hormones, in turn, elicit the secretion of adrenocorticotrophic hormone from the adrenal cortex into the bloodstream. This, in turn, leads the adrenal gland to secrete glucocorticoids, such as cortisol.

Cortisol, broadly recognized as a stress hormone, is essential to our functioning in everyday life. Cortisol plays a regulatory role in functions such as our sleep-wake

cycle and state regulation with regard to experiences of hunger and satiation, as well as to maintenance of blood pressure and even our ability to process information and both form and retain memories. Cortisol is always present, to some degree, in our bloodstream. Typically, the blood levels of cortisol vary, in a particularized pattern, throughout the day, giving rise to patterns of diurnal variation. Cortisol can be measured in the blood, but also through a saliva swab, making it easier and less invasive to measure in both normative and at-risk populations (Dozier, 2014; Gunnar et al., 2011).

As described above, when we experience stress, cortisol levels may rise and contribute to our ability to mount a stress response. Working in tandem with the SAM system, blood sugar may increase so that our bodies have more energy to focus our attention on immediate threats. Thus, a rise in cortisol supports adaptation in a short-term sense. A well-functioning stress response system is one that turns on or is activated when a stressor is perceived but also turns off when it is no longer needed. Research on cortisol empirically demonstrates what we have seen clinically, namely, that chronic activation of the stress response will, over time, impair the ability of the system to turn off, even when an immediate threat has passed.

Chronic activation of the stress response system can damage brain development, organization, and function. For example, researchers have noted alterations in the volume in specific structures within the brain, including the hippocampus and the amygdala, in both children and adults

associated with chronic activation of the stress response system (Gunnar & Quevedo, 2007). The hippocampus is central to information processing and memory, two functional areas also sometimes observed to be impaired by chronic stress. Likewise, researchers have noted negative impacts on those parts of the brain associated with executive functioning skills such as selective attention and effortful control, two areas of functioning key to overall well-being (Hostinar et al., 2012).

Research on early adversity sometimes emphasizes child abuse and neglect, because this type of adversity often puts children in double jeopardy. When the adversity or toxic stress occurs in the context of children's primary caregiving relationships, they are impacted directly by the experience of abuse and neglect, because they may lack access to the types of supportive care shown to protect the developing nervous system and brain. Research on the impact of abuse and neglect on brain development has identified several outcomes that are, in turn, associated with psychosocial and behavioral outcomes. For example, severe childhood neglect has been associated with reduced volume in the orbitofrontal cortex, which is associated with the capacities for emotion and self-regulation. Similarly, decreased volume in the hippocampus, cerebellum, and corpus callosum have also been documented in adolescents and adults who experienced abuse and neglect in early childhood (Wilson, Hansen, & Li, 2011). These brain structures are associated with competencies such as memory and executive functioning. From a neuroendocrine perspective, as described above in our discussion of the stress response system, children who have experienced child abuse and neglect have been shown to have atypical patterns of blood cortisol, which in turn may be associated with alterations in their immune system, sleep-wake cycles, and multiple indicators of psychosocial well-being relationships (Bruce, Fisher, Pears, & Levine, 2009).

Prevention scientists have identified access to consistent, empathically attuned care as a key ingredient in neurodevelopment and a key protective factor for children who experience early adversity and stress (National Scientific Council on the Developing Child, 2014). Many parent-child dyads are deemed vulnerable because various risk factors, or sets of factors, combine to inhibit the caregiver's ability to accurately perceive or respond to the emotional or physical needs of the developing child. In addition to a range of contextual and social factors, such as social isolation and poverty, other factors may interfere with (1) the caregiver's own capacity for emotion regulation, (2) the ability to take the point of view of the child, and (3) the ability to use language to represent either emotional experience or to reflect back to the child the parental perception of the child's internal state of mind and experience. These kinds of obstacles interfere with the child's developing sense of being known or understood by the caregiver, resulting in critical problems in the child's emerging capacity to identify affective states, relate particular states of affect to known experience, and to regulate affect in a manner sufficient to help the child reinstate homeostasis when his or her

affective experience becomes dysregulated. As has been discussed elsewhere in this volume, the relational environment of infants and young children is a primary context for early brain development. One of the reasons that researchers describe the early years as a critical or sensitive period with regard to brain development is because both the organization and function of the child's neural systems are so affected during this time by the amount and quality of interactive experience. In particular, human infants are uniquely dependent on their adult caregivers for an environment that provides sufficient contingent responsivity (Weder & Kaufmann, 2011). These studies highlight the need for initial and early assessment, prevention, and intervention for at-risk parents and children (Dozier & Fisher, 2014).

VULNERABLE DYADS

Many groups of at-risk parents and children are characterized by atypical patterns of interaction and disruptions in early caregiving relationships. Research in the cognitive neurosciences has provided a new window into the clinical meaning of disorganized or traumatic early care experiences by delineating how disrupted early care precipitates change in the early wiring of the brain in neurobiological and neurochemical processes that have import for the experience and regulation of affect. It is now well understood that the human brain triples in size by 3 years of age and that the first year of life is a critical period with regard to the development of the prefrontal cortex, an important element of the biological basis of attachment behavior.

As Schore and Schore (2010) point out, early attachment and caregiving patterns directly in uence the development of the frontal limbic system in the brain's right hemisphere, a key neurobiological substrate of the emerging capacity for affect regulation, self-understanding, and the understanding of others (Cozolino, 2010; Fonagy & Higgit, 2004; Fonagy & Allison, 2012; Hofer, 1995). Thus, any factors or characteristics that are associated with the parental capacity for affect regulation or the child's developing capacity to regulate affect are understood as appropriate targets for support and intervention.

As previously noted, research on brain development and attachment shows that impaired early relational experience can create psychological and neurobiological vulnerability in the child that is sustained over time. Additionally, this research sharpens our understanding of how psychological and developmental vulnerability in a caregiver can be transferred from parent to child and sustained across generations (Fraiberg, 1980), an outcome that has also been examined from an epigenetic perspective (National Scientific Council on the Developing Child, 2011). As discussed elsewhere in this volume, disruptions in early caregiving experience are among the most damaging kinds of stress experienced by infants and young children.

For many families, pregnancy and early parenthood is a time of tremendous stress in ways that can impede even a well-intentioned caregiver's capacity to foster secure attachment and the child's experience of feeling psychologically held. Lack of access to consistent and empathically attuned relational care constitutes deprivation for the infant and young child because it poses great risk to developmental well-being. As Gunnar (2000) points out, deprivation can be characterized by a range of biopsychosocial factors including lack of access to health care and nutrition, age-appropriate stimulation and learning opportunities, and, of particular focus here, a lack of consistent access to stable and contingently responsive relationships. Relational deprivation in infancy confers a series of risks on the developing infant associated with deficits as measured by psychosocial characteristics, such as impaired attachment relationships, and the infant's underlying neurological well-being, particularly in terms of the stress response system (Gunnar, Bruce, & Grotevant, 2000; Gunnar, Morrison, Chisholm, & Schuder, 2001). This research is particularly relevant for practitioners working with caregivers and children in situations where a combination of parental, child, and environmental characteristics may combine to pose challenges to the development of secure caregiver-child attachment or disrupt existing attachment relationships by limiting the caregiver's capacity for empathic attunement and contingently responsive caregiving.

Why are some parents better able to provide empathic care that meets the child's developmental needs? A range of biopsychosocial factors can combine to inhibit the caregiver's ability to accurately perceive and respond to the emotional, social, and physical needs of the developing child. Factors such as social isolation, poverty, and stress can combine with lack of knowledge of

child development and parental mental health challenges such as maternal depression to interfere with the provision of sensitive and attuned care, thereby creating a context of risk and vulnerability for the developing child (Cohn & Tronick, 1989; Crandall, Fitzgerald, & Whipple, 1997). A capacity for reflective functioning supports parental sensitivity to the infant and thus attachment security because caregivers are more able to respond accurately not only to physical needs but to emotional needs as well. This, in turn, helps the child to "make meaning of feelings and internal experience and states of psychophysiological arousal associated with feelings without becoming overwhelmed and shutting down" (Slade, 2002, p. 11).

– Stay tuned for part 2 next month –

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