What is Toxic Stress?

Exposure to Adverse Childhood Experiences (ACEs) without a positive buffer can cause a toxic stress response in the body.

The toxic stress framework was developed to bring to light decades of basic and clinical research conducted on the biological impacts of early life stress and childhood adversities. Since this initial work, the American Academy of Pediatrics (AAP) has published numerous policy statements calling for increased understanding and action to address toxic stress in pediatric populations impacted by adversity (Garner et al., 2012; Shonkoff 2012; Shonkoff 2010).

The toxic stress framework categorizes three types of stress responses:

- Positive stress response: A brief physiological state characterized by the activation of the hypothalamic-pituitary-adrenal (HPA) axis in response to a mild or moderate stressor. The body returns to homeostasis through the activation of the autonomic nervous system once the individual is no longer exposed to the stressor. Positive stress is a normal and important part of healthy development.
- Tolerable stress response: The physiological state resulting from exposure to a threat that is greater in severity or duration than positive stress. In the presence of the buffering effects of a supportive caregiver and effective coping mechanisms, the body can adapt to this type of stress and is able to return to homeostasis.
- Toxic stress response: An intense, frequent, and/or sustained activation of the body's stress response and autonomic nervous system, in the absence of buffering by a caring adult, results in a dysregulation of the neuroendocrine and immune (NEI) system. This dysregulation doesn't allow the body to return to homeostasis and therefore is considered "toxic" for the body, especially in children during their most critical years

Systemic Impacts

Toxic stress can have far-reaching implications on the body through cumulative effects of a chronically dysregulated stress response system. The chronic dysregulation of the neuroendocrine immune system via the hypothalamic-pituitary axis (HPA) has been documented to compromise nervous, immune, cardiovascular, reproductive, endocrine systems (Bucci et al, 2016). Multi-systemic alterations result in changes to the body's metabolic and epigenetic functioning (Bucci et al, 2016). Exposure to adversity early in life, particularly during sensitive periods of child and adolescent development, are especially problematic due to enhanced sensitivity and likelihood of permanent and long-term integration into regulatory biological processes (Johnson, 2013).

- Nervous system: Disruption to architecture and neurochemistry of the developing brain, including structural and functional changes to the hippocampus, prefrontal cortex, and amygdala, may lead to an increase in risk of cognitive impairment, attention deficits, learning disabilities, poor executive function, self-regulation, memory and attention, and anxiety (McEwen 2000; Shonkoff 2010; O'Connor 2000; Gallagher & Chiba 1996; LeDoux, 2007).
- Cardiovascular system: Activation of a chronic inflammatory response due to a chronically activated sympathetic response and persistent circulation of inflammatory

markers may result in high blood pressure, damaged arteries, early atherosclerosis, risk of cardiovascular disease, myocardial infarction, and stroke (McEwen 2007; McEwen 1998; McEwen 2004; Steptoe, Hamer & Chida 2007).

- Immune system: Alteration to the natural immune defense responses increase the risk of infections due to high levels of circulating cortisol, decreases in the T-helper mediated cellular immune response, chronic inflammation from persistent pro-inflammatory cytokines, and reduced capacity for white cells to respond to anti-inflammatory signals. These compromised functions also increase the risk for autoimmune diseases such as asthma and irritable bowel syndrome (Johnson 2013; Steptoe, Hamer & Chida 2007; Shonkoff 2014; O'Connor 2000; Segerstrom & Miller 2004).
- Reproductive system: Hormonal imbalances lead to a number of changes, including inhibition of synthesis and secretion of gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH) and follicle-stimulating hormone (FSH), decline in testosterone production, inhibition of steroids biosynthesis and action of LH on ovaries, apoptosis in testes and ovaries; these occurrences can ultimately result in infertility (Whirledge, 2001).
- Endocrine system: Persistently high cortisol levels impact several endocrine organs, including inhibitory effects on glucocorticoid secretion of growth hormone and somatomedin C, inhibition of thyroid prohormone T4 to T3, and inhibited secretion of thyroid-stimulating hormone due to increased levels of somatostatin resulting in metabolic disorders (McEwen 2000; O'Connor 2000; McEwen 1998; Johnson 2013; Pacak & Palkovits 2001; Bose 2009).

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