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Invited Review

Ameliorating the biological impacts of childhood adversity: A review of intervention programs

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ABSTRACT

Childhood adversity negatively impacts the biological development of children and has been linked to poor health outcomes across the life course. The purpose of this literature review is to explore and evaluate the effectiveness of interventions that have addressed an array of biological markers and physical health outcomes in children and adolescents affected by adversity. PubMed, CINAHL, PsychInfo, Sociological Abstracts databases and additional sources (Cochrane, WHO, NIH trial registries) were searched for English language studies published between January 2007 and September 2017. Articles with a childhood adversity exposure, biological health outcome, and evaluation of intervention using a randomized controlled trial study design were selected. The resulting 40 intervention studies addressed cortisol outcomes (n = 20) and a range of neurological, epigenetic, immune, and other outcomes (n = 22). Across institutional, foster care, and community settings, intervention programs demonstrated success overall for improving or normalizing morning and diurnal cortisol levels, and ameliorating the impacts of adversity on brain development, epigenetic regulation, and additional outcomes in children. Factors such as earlier timing of intervention, high quality and nurturant parenting traits, and greater intervention engagement played a role in intervention success. This study underlines progress and promise in addressing the health impacts of adversity in children. Ongoing research efforts should collect baseline data, improve retention, replicate studies in additional samples and settings, and evaluate additional variables, resilience factors, mediators, and long-term implications of results. Clinicians should integrate lessons from the intervention sciences for preventing and treating the health effects of adversity in children and adolescents.

1. Childhood adversity

Childhood adversities such as abuse, neglect, and additional household stressors, are highly prevalent, and are linked to poor child and adult health, and high societal costs (Bright, Alford, Hinojosa, Knapp, & Fernandez-Baca, 2015; Fang, Brown, Florence, & Mercy, 2012; Felitti et al., 1998; Flaherty et al., 2013; Wing, Gjelsvik, Nocera, & McQuaid, 2015). As many as 60% of children and adolescents in national population-based studies have experienced at least one category of Adverse Childhood Experiences (ACE)

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(Bethell, Newacheck, Hawes, & Halfon, 2014). Data on ACE exposures among youth in foster care, and community settings reveal that these populations demonstrate particularly high prevalence of early adversity. Foster care children are at between 1.5 and 7 times greater odds of having experienced any one of the 10 traditional ACEs compared to children not placed in foster care, even after controlling for race/ethnicity, parent education and employment, welfare services, and poverty status (Turney & Wildeman, 2017). Additionally, in an urban community sample, 92% of youth endorsed at least one stressor, including ACEs and additional community-specific adversities (Purewal Boparai et al., 2017).

Longitudinal research has demonstrated that children exposed to adversity are at risk for detrimental behavioral and mental health outcomes in youth and adulthood (Fergusson, McLeod, & Horwood, 2013; Flaherty et al., 2013; Lansford et al., 2002; Maniglio, 2009; Turner, Finkelhor, & Ormrod, 2006). An emerging body of data is beginning to shed light about the risk of physical health outcomes. A recent systematic review evaluating longitudinal child health outcomes associated with childhood adversity found that child outcomes, such as asthma, were consistently and negatively impacted by child adversity (Oh et al., 2018). A richer understanding of chronic and physical health outcomes stems from the literature on adult retrospective self-reports; researchers have found a dose-response relationship between the number of ACEs and many leading causes of death in the United States, including heart disease and cancer, as well as premature mortality (Brown et al., 2009; Felitti et al., 1998; Kalmakis & Chandler, 2015).

2. Toxic stress

The cumulative body of evidence on early life stress has generated the concept of toxic stress, which is the chronic or frequent activation of the stress response that results from exposure to severe or frequent adversity in childhood in the absence of support from a caregiver (National Scientific Council on the Developing Child, 2005). The human stress response is determined by a complex interplay between central and autonomic divisions of the nervous system, and interactions with endocrine, immune, and genetic regulatory mechanisms. The nervous system plays a major role in activating and regulating the stress response through the hypothalamic-pituitary-adrenal (HPA) and the sympathetic-adrenal-medullary (SAM) axes.

The activation of these two axes has been also described as “fight or flight” response and exerts its action through a cascade of responses that involves the secretion of epinephrine and norepinephrine, and glucocorticosteroids, such as cortisol and dehydroepiandrosterone sulfate (DHEA-S) (Bucci, Marques, Oh, & Harris, 2016). The HPA axis upregulates and downregulates the cascade of biological events that prepare the body to respond to a stressor; cortisol, the end product of the HPA axis, has been extensively studied in the field of early life stress (Gunnar & Donzella, 2002). Humans have a typical diurnal cortisol pattern that spikes 30 min after waking and slopes toward nadir in the evening; infants do not show an established pattern until about three months of age and approach a more predictable pattern over the first two years of life (Gunnar & Donzella, 2002; Larson, White, Cochran, Donzella, & Gunnar, 1998; Sippell, Becker, Versmold, Bidlingmaier, & Knorr, 1978). When young children are exposed to significant or frequent adversity, cortisol levels can either become hyper- or hypoactive due to a dysregulated feedback loop (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010; Bruce, Fisher, Pears, & Levine, 2009; Gunnar & Vazquez, 2001).

The dysregulation of the stress response can result in a persistent elevation of circulating immune markers such as C-reactive protein (CRP) and proinflammatory cytokines (Bucci et al., 2016). Childhood adversities such as maltreatment, social isolation, and economic disadvantage have been linked to high levels of inflammation in adulthood (Danese et al., 2009). Moreover, altered levels of inflammatory markers and cortisol have been documented to impact brain structure and function (Bucci et al., 2016). Additionally, numerous studies have shown the impacts of early life stress on the developing brain (Carrion, Weems, & Reiss, 2007; Choi, Jeong, Rohan, Polcari, & Teicher, 2009; Glaser, 2000). Finally, epigenetic changes due to exposure to early adversity have also been identified (Bucci et al., 2016; Drury, Theall, et al., 2012; Shalev et al., 2013). Physiological dysregulation can alter brain architecture and other organ systems during sensitive periods of childhood development when children are most vulnerable to social and environmental context; the negative health outcomes that follow later in adult life may be a result of cumulative physiological, epigenetic, and cognitive responses to chronic stress due to early exposure to adversity (Bucci et al., 2016).

3. Clinical advocacy and action

Given the evidence of effects of adversity on children’s health, it is critical to develop best practices to reverse the biologically damaging impacts of early life stress. The American Academy of Pediatrics (AAP) has called on scientists and practitioners to explore solutions to address early adversity and associated outcomes, including novel and effective preventative treatments (Garner, Shonkoff, Committee on Psychosocial Aspects of Child and Family Health, Committee on Early Childhood, Adoption, and Dependent Care, & and Section on Developmental and Behavioral Pediatrics, 2012; Johnson, Riley, Granger, & Riis, 2013; Shonkoff, Garner, & Committee on Psychosocial Aspects of Child and Family Health, Committee on Early Childhood, Adoption, and Dependent Care, & Section on Developmental and Behavioral Pediatrics, 2012). Understanding progress on intervention research is essential to identifying effective intervention components and outcome targets, highlighting study limitations, and improving research with the ultimate goal of informing translation and development of standard practices of care for children and adolescents exposed to adversity.

To date, a number of interventions have successfully targeted cortisol outcomes in an effort to regulate maltreated children’s physiological response to stress (Fisher, Gunnar, Dozier, Bruce, & Pears, 2006; Gunnar, Fisher, Early Experience, Stress, & and Prevention Network, 2006). Slopen, McLaughlin, and Shonkoff (2014) reviewed 19 interventions that addressed regulation of cortisol secretion among children. Interventions drew from both healthy and negatively-impacted samples and focused exclusively on cortisol outcomes. Researchers found that cortisol levels can be modified through interventions yet variation across studies in cortisol