1

Reducing Stress Disparities

Pathways to Equity Through the Study of Stress Biology

Emma Adam, Sarah Collier Villaume, and Emily Hittner

Research, intervention, and policy focused on identifying solutions for disparities in child health and developmental outcomes are gaining insight from a new source: the study of stress biology. Recent theoretical models have implicated disparities in stress exposure and the biological consequences of that exposure (Heissel, Levy, & Adam, 2017; Levy, Heissel, Richeson, & Adam, 2016; Miller & Chen, 2013; Miller, Chen, & Parker, 2011) as one pathway by which adverse circumstances for children and adolescents "get under the skin and into the mind," affecting the body and brain (Johnson, Riis, & Noble, 2016; McEwen, 2012). Differential exposure to stress, in turn, contributes to the emergence and maintenance of disparities in health, academic, and human capital outcomes.

Stress is not equally distributed in society; stress disparities exist along many dimensions and their intersections. Here, due to their pervasiveness, we focus on disparities by income (G. W. Evans & English, 2002) and by ethnic–racial group membership (Levy et al., 2016). Individuals of lower socioeconomic circumstances and historically disenfranchised ethnic and racial groups (e.g., Black, Hispanic, and Native American individuals in the United States) have been found to be exposed to higher levels of multiple sources of stress (G. W. Evans & English, 2002; Levy et al., 2016). Stress exposure activates a wide array of stress-sensitive biological systems that, in turn, have implications for health, cognition, and everyday functioning (Lupien, McEwen, Gunnar, & Heim, 2009; McEwen, 2012).

http://dx.doi.org/10.1037/0000187-002
Confronting Inequality: How Policies and Practices Shape Children's Opportunities, edited by L. Tach, R. Dunifon, and D. L. Miller

Disparities in exposure to both everyday and traumatic stressors, by way of their impact on stress biology, contribute to the emergence of disparities in developmental trajectories of health and human capital (Halfon & Hochstein, 2002; Heissel, Levy, & Adam, 2017). Differences in stress biology can serve as a marker for the presence of disparities in stress exposure and a pathway by which they are instantiated into health and developmental outcomes. More recently, however, stress science is also beginning to provide insights for potential solutions (McEwen, 2012). Through offering insights on how stress exposure can be reduced, coping promoted, and stress biology reregulated through social policy and other intervention efforts, stress science is providing clues to how to reduce stress disparities and gaps in health and academic outcomes.

In this chapter, we provide (a) an overview of the concept of stress disparities and evidence that stressors are unequally distributed by economic class and ethnic–racial group; (b) a description of stress biology, with a focus on biological systems that are both affected by stress and have implications for daily functioning, cognition, and health, along with some evidence that these systems vary by socioeconomic status (SES) or ethnicity–race; and (c) a description of how stress biology has been used to inform policy and intervention research, including evidence from existing studies and clinical trials demonstrating that stress-sensitive biological systems respond to either policy or behavioral interventions with potential positive implications for health and behavioral outcomes. We also provide our suggestions for future directions of stress disparities research to improve the potential of this research to provide not only an equal but also an equitable start for all children and adolescents.

STRESS DISPARITIES

In this section, we define stress disparities and describe stress disparities occurring by SES and by ethnic–racial group membership, as evidenced by differences in multiple types of stress exposure and differences of stress biology.

Defining Stress Disparities

Many past researchers have noted that living in low socioeconomic contexts is associated with higher levels of exposure to stressors, with resulting implications for stress biology, health, and developmental outcomes (Brooks-Gunn, Klebanov, & Liaw, 1995; G. W. Evans & English, 2002). A separate literature has focused on the increased stress exposure associated with being a member of a disenfranchised ethnic or racial group and the implications of this race-based stress for health and developmental outcomes (Adam et al., 2015; Levy et al., 2016). We coined the general term *stress disparities* to capture both of these (Heissel, Levy, & Adam, 2017) and, indeed, any circumstance in which (a) social or demographic factors are related to systematically different levels of exposure to stressors, and (b) differential stress exposure has resulting implications for differences in stress biology.

We believe that these stress disparities serve as plausible explanatory pathways or mechanisms for the emergence of disparities in health or developmental outcomes.[1] For example, we believe that stress disparities play roles in both ethnic–racial and socioeconomic disparities in health and developmental outcomes. Additionally, we hypothesize that individuals in other social categories experiencing significant social disenfranchisement, stigma, or reduced access to resources (e.g., LGBTQ individuals, undocumented immigrant populations) are also likely to suffer increased levels of stress exposure and altered stress biology, with stress disparities playing a potential role in negative health or attainment outcomes in these groups.

Types of Stress Exposures

Stress disparities can emerge from a variety of types of stressors, including traumatic stress (i.e., extreme stressors, either short- or long-term, that pose a fundamental threat to physical or social safety), acute or chronic major life events, and accumulated minor events and daily hassles (Grant et al., 2003, 2014). It has been shown that, often, stress disparities emerge from a combination of these stressor types, with stress disparities most evident and effect sizes on outcomes larger when exposure to multiple stressors is considered (G. W. Evans & English, 2002). For example, individuals living in poverty are more likely to encounter traumatic stressors (e.g., exposure to violence, severe abuse or neglect, or loss of or long-term separation from a caregiver), as well as chronic stressors (e.g., food insecurity, crowding, noise, substandard housing). When compared with higher income individuals, they are also more likely to encounter three or more of these stressor types simultaneously (G. W. Evans & English, 2002).

Stress disparities according to ethnicity and/or race include increased exposure to ethnic and racial discrimination. This involves structural forms of racism (e.g., residential segregation; discrimination in education, employment, and housing) that limit life chances and thereby disproportionately expose persons of color to the stress of unfair treatment, as well as the stresses of poverty noted above (Pager, Bonikowski, & Western, 2009; Pager & Shepherd, 2008). In addition, regardless of level of income, it has been shown that people of color face frequent everyday forms of discrimination including tokenism, stereotypes creating differing school and workplace expectations, and negative social interactions, including microaggressions, in which they are treated with differing expectations, disrespect, fear, or suspicion (Adam et al., 2015; Brondolo et al., 2008; Broudy et al., 2007; Jackson & Stewart, 2003). These

[1]We acknowledge that many other factors also contribute to the creation and maintenance of disparities in health and human capital, such as disparities in the quality of schooling and differences in access and the quality of health care. Our argument here is that disparities in stress exposure play a plausible additional role that has not received sufficient attention in the literature or in the generation of and policy solutions for disparities in health and developmental outcomes.

situations create social signals of exclusion that can precipitate feelings of sadness, shame, loneliness, or anger, which, in turn, precipitate changes in multiple aspects of stress biology (Adam et al., 2015; Brondolo, Rieppi, Kelly, & Gerin, 2003; Doane & Adam, 2010). These changes in stress biology, in turn, have been shown to impact the body and brain in ways that matter for health and human capital, completing the cycle by which stress disparities become instantiated as lifelong (and potentially intergenerational) disparities in health and developmental outcomes (Heissel, Levy, & Adam, 2017; Kuzawa & Sweet, 2009; Levy et al., 2016; Thayer & Kuzawa, 2015).

Timing of Stress Exposures

Past theory and research have revealed that the developmental timing of stress exposure matters greatly, modifying both the strength and nature of the effects of stress on stress biology and health, as well as on developmental outcomes. Stressors that occur during periods of rapid developmental change are thought to have larger impacts on developmental outcomes than those occurring during periods of slower developmental change; such periods are frequently referred to as *sensitive periods*. Stress researchers have focused primarily on two processes/timelines by which experience affects biology: (a) the biological embedding of experiences occurring during sensitive periods of development (especially early-life experiences, from the prenatal period through adolescence), and (b) the cumulative wear-and-tear effects of stressors impacting already-developed biological systems (especially during adulthood and aging) (McEwen, 2012). Some theorists have differentiated timing effects further, either (a) specifying differential processes taking place in prenatal, postnatal, adolescence, adulthood, and aging (Halfon & Hochstein, 2002; Lupien et al., 2009) or (b) describing the multiple time-scales over which experiential effects unfold and become evident in later functioning (Adam, 2012; Hertzman, 2012). The key distinction made in all theories, however, is between biological embedding of early stress experiences and the cumulative effects of later stress (Shonkoff, Boyce, & McEwen, 2009).

Biological Embedding of Early Experience

Past theory and research have suggested that early experiences of stress from the prenatal period through adolescence may be particularly important for the impact of traumatic experiences on stress biology. This research has highlighted that stressful experiences occurring during this time not only transiently affect stress biology but may permanently alter the structure and function of the biological stress systems still developing at each time point, thus becoming "built into" the structure and functioning of emerging biological systems. These effects of early experience on biology have been called *programming effects* (particularly used for the lifelong impact of prenatal experiences; Lupien et al., 2009) and *biological embedding* (Hertzman, 2012). Numerous reviews and empirical articles have detailed the dramatic effects of early social experience on stress biology

and lifelong health and cognitive functioning (Felitti et al., 1998; Lupien et al., 2009; Shonkoff et al., 2012).

Cumulative Impact of Acute and Chronic Stressful Events

Other theory and research suggest that cumulative impacts of acute and chronic stress over time are important, with these processes occurring to some extent during childhood and adolescence (Desantis, Kuzawa, & Adam, 2015) and being the most common manner in which stress exposure influences stress biology during adulthood (McEwen, 2012). The notion here is that the frequent or chronic activation of stress biology over time creates wear and tear on the biological systems being used, leading to less effective functioning of biological stress systems. This, in turn, has downstream implications for the parts of the body and brain that are regulated by those systems, as well as for long-term health and daily functioning. This cumulative wear and tear on multiple biological stress systems has been termed *allostatic load* (McEwen, 1998), a concept on which we elaborate later in this chapter.

Interactions Between Early and Later Stress Experiences

Importantly, interactions between early stress and ongoing and later cumulative stress may be important in maintaining and accentuating disparities in developmental trajectories that began with early adverse exposures (Desantis et al., 2015; Halfon & Hochstein, 2002; Hertzman, 2012; Lupien et al., 2009). Other theory and literature suggest that, from a life-course health and development perspective, additional sensitive periods occur across the life span whenever rapid biological and/or social changes are occurring. Potential sensitive periods postchildhood include adolescence, the transition to parenting, menopause/andropause, and older adulthood during the aging process (Halfon & Hochstein, 2002). Very little research has traced how developmental trajectories of health and well-being unfold through the combination of stress exposures during early sensitive periods, later sensitive periods, and the accumulation of acute and chronic stress across the life span. Understanding life-span histories of stress exposures across multiple developmental periods is likely the key to more fully understanding long-term trajectories of health and development (Adam et al., 2015; Halfon & Hochstein, 2002).

OVERVIEW OF STRESS-SENSITIVE BIOLOGICAL SYSTEMS

Having covered some key background concepts regarding stress disparities and the impact of experience on stress biology in different developmental periods, we describe several relevant biological stress systems in more detail, noting some of their impacts on health and developmental outcomes and whether disparities in these systems have been observed by income and/or ethnicity–race. We focus on primary stress mediators (McEwen & Seeman, 1999)—systems that are the "first responders" of stress experiences, helping

us to cope with the stressor at hand, primarily through mobilizing energy resources and helping to prevent or contain injury. For our purposes, these include the autonomic nervous system (ANS), the hypothalamic–pituitary–adrenal axis (HPA axis), the immune and inflammatory systems, and the alertness/sleep system.[2] These primary stress mediators regulate each other and have impacts on a variety of downstream neurological and biological systems that are important for long-term health and well-being. Although many nuanced indicators of these systems are available, we focus on measures of these systems that are most relevant for policy and intervention research, in that they can be noninvasively obtained in large scale studies in community settings.

Autonomic Nervous System

One of the fastest responding regulatory systems of the body is the ANS, which includes sympathetic and parasympathetic processes. The main function of the ANS is to mobilize energy to control internal bodily processes and deliver oxygenated blood to the body. Although many aspects of autonomic physiological activation can be examined (e.g., cardiac, vascular, respiratory), researchers and practitioners have highlighted blood pressure as one uncensored, prognostic, and mechanistic system that is an important index for physiological states of stress, threat, and effort (Blascovich, Vanman, Mendes, & Dickerson, 2011), with important health and developmental consequences.

Blood pressure measurement reflects the amount of pressure exerted against blood vessel walls during the phases of the cardiac cycle (Franklin & Mitchell, 2008). Such measurement is conducted either in clinical settings, typically by medical personnel, or by trained research staff in community or home-based settings, most often using automated blood pressure cuffs. For research purposes, multiple measures (at least three) are taken in a row, with the first measure usually discarded due to *white coat* effects (i.e., transient blood pressure increases in response to the novelty of anxiety associated with the blood pressure measurement process; see Den Hond, Celis, Vandenhoven, O'Brien, & Staessen, 2003), and the remaining measures are averaged.

Recent guidelines recommend measuring using ambulatory blood pressure monitoring (Flynn et al., 2017). Ambulatory blood pressure continuously monitors blood pressure throughout the day as individuals go about their daily activities and sleep, taking readings typically every 15 to 30 minutes (Pickering, Shimbo, & Haas, 2006). Ambulatory blood pressure not only gives an average blood pressure level but also provides information about the diurnal rhythm of blood pressure along with its variability without white coat effects

[2]Past definitions of primary stress mediators have focused on the ANS and the HPA axis, but recent evidence has revealed acute responding of the inflammatory system and alertness/sleep systems to stressors, with consequences for a wide range of downstream biological systems, which we believe meet the criteria for consideration as a primary stress mediator.

on the readings. Ambulatory blood pressure has well-documented normalized 24-hour values in children, adolescents, and adults (Wühl et al., 2002) that provide important naturalistic insights for large-scale policy research (Mancia et al., 1995).

Among others,[3] distinctions are typically made between systolic blood pressure (peak pressure in arteries during one heartbeat) and diastolic blood pressure (lowest pressure in arteries during one heartbeat) as, although correlated, these can index unique aspects of stress responses (Blascovich et al., 2011). For instance, increases in systolic blood pressure can be an adaptive response to acute stress (Brownley, Hurwitz, & Schneiderman, 2000), yet prolonged exposure to stress and persisting high levels of systolic blood pressure exert prolonged stress on the heart (Ayada, Toru, & Korkut, 2015; Vrijkotte, van Doornen, & de Geus, 2000).

The prevalence of hypertension (i.e., clinically diagnosed high levels of blood pressure) has increased among both children and adults since the 1990s, a concerning trend following several decades of decline (Elliott & Black, 2007; Falkner, Gidding, Portman, & Rosner, 2008; Muntner, He, Cutler, Wildman, & Whelton, 2004). In fact, the prevalence of high blood pressure in children and adolescents is estimated to be as high as to 19% in males and 13% in females (Rosner, Cook, Daniels, & Falkner, 2013). This is concerning given that elevated blood pressure during youth not only predicts adult hypertension (X. Chen & Wang, 2008) but also is associated with poor physical health (e.g., higher risk of stroke, heart attack, and chronic kidney failure; Chobanian et al., 2003), mental health (e.g., higher levels of anxiety, depression; Lande et al., 2009; Pickering, 2001), and cognition (e.g., decreased attention, concentration, and short-term memory; Lande, Kaczorowski, Auinger, Schwartz, & Weitzman, 2003). With worldwide estimates of high blood pressure at as much as 1.13 billion individuals (World Health Organization, 2019), understanding blood pressure as a key mechanism linking childhood stress and adult health is a public health priority (Chobanian et al., 2003; Falkner, Lurbe, & Schaefer, 2010).

Although a range of factors affect blood pressure, including genetic and lifestyle factors, it is also strongly affected by psychosocial stress (Spruill, 2010). Relevant to our arguments regarding stress disparities, blood pressure has been found to be higher in lower SES adults and adolescents (Conen, Glynn, Ridker, Buring, & Albert, 2009; Kaczmarek, Stawinska-Witoszynska, Krzyzaniak, Krzywinska-Wiewiorowska, & Siwinska, 2015; Leng, Jin, Li, Chen, & Jin, 2015), but there is little evidence as to whether BP differences exist by family SES in children (Colhoun, Hemingway, & Poulter, 1998). Higher blood pressure in low SES populations has been shown to be partly attributable to greater job strain (Landsbergis, Schnall, Pickering, Warren, &

[3]Other measures of blood pressure include pulse pressure (i.e., subtracting DBP from SBP), mean arterial pressure (i.e., weighted average of DBP and SBP), and total peripheral resistance (i.e., combination of mean arterial pressure and cardiac output). Each offer insights in the physiological bases of stress reactivity.

Schwartz, 2003; Schnall, Schwartz, Landsbergis, Warren, & Pickering, 1992). Differences in blood pressure also consistently have appeared by ethnicity–race, being higher in African American children and adults than in Whites (Bosworth et al., 2006; Levinson et al., 1985). Psychosocial stress related to ethnicity–race, such as the stress associated with negative stereotypes of one's ethnic–racial group, has predicted elevated blood pressure among African Americans (Blascovich, Spencer, Quinn, & Steele, 2001).

Immune System

When faced with stress, the body not only mobilizes the ANS but also triggers changes in the immune response. Evolutionary perspectives have suggested that threatening situations that stimulate ANS "fight or flight" behavior also carry risk of injury or infection (Maier, 2003; Segerstrom & Miller, 2004). As such, synchrony across the ANS and inflammatory system was not only considered evolutionarily adaptive but also continues to be relevant in understanding links between psychological stress and health. Indeed, there has been decades of accumulating evidence that link disparities in early-life stress and inflammation (Miller, Chen, & Cole, 2009; Segerstrom & Miller, 2004). Coupled with a lack of coping resources (e.g., lack of support in personal relationship, less self-efficacy; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Uchino, Cacioppo, & Kiecolt-Glaser, 1996), higher levels of perceived chronic stress have been linked with immune dysregulation; the immune system is less likely to adaptively engage in a successful response to viral challenges (Glaser et al., 1992; Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985; Keller, Shiflett, Schleifer, & Bartlett, 1994; Yang & Glaser, 2002). Stress-related changes in immune responsivity in turn have been shown to have downstream effects (Glaser & Kiecolt-Glaser, 2005) on mental health (e.g., depression, anxiety; Connor & Leonard, 1998; Kiecolt-Glaser, Page, Marucha, MacCallum, & Glaser, 1998; Maes, Ombelet, De Jongh, Kenis, & Bosmans, 2001; Reiche, Nunes, & Morimoto, 2004), physical health (e.g., infection, autoimmune disease, coronary artery disease; Cohen et al., 1998; Rozanski, Blumenthal, & Kaplan, 1999; Whitacre, Cummings, & Griffin, 1994), and cognition (e.g., memory consolidation and impairment; McKim et al., 2016; Rachal Pugh, Fleshner, Watkins, Maier, & Rudy, 2001).

In understanding mechanisms linking psychological stress, the immune response, and health, researchers have proposed that the endocrine system serves as one avenue through which psychological stress influences immunologic functioning (Kiecolt-Glaser et al., 2002; Rabin, 1999). In addition to sympathetic changes in the immune response, pituitary and adrenal hormones released in response to stress have been shown to mediate associations between stress and immune functioning (Cacioppo, Berntson, Sheridan, & McClintock, 2002; Miller, Cohen, & Ritchey, 2002), although more research elucidating these interactions is needed (Lovallo, 2016).

Inflammation has been found to vary systematically by SES in both adults and children, with lower SES typically linked with more heightened inflammatory responses (Gruenewald, Cohen, Matthews, Tracy, & Seeman, 2009;

Schmeer & Yoon, 2016). In addition, racial and ethnic differences in inflammation have been found, with Latinx and African American children at greater risk for low-grade inflammation than White children (Albert, 2007; Schmeer & Tarrence, 2018).

HPA Axis

Perceptions of psychosocial stress, particularly those that involve uncontrollable or social-evaluative experiences, have been shown to trigger changes in the HPA axis as evidenced by acute reactivity of cortisol, a key product of the HPA axis (Dickerson & Kemeny, 2004; Hankin, Badanes, Abela, & Watamura, 2010; Kirschbaum & Hellhammer, 1989). A larger and more extended HPA axis response to psychosocial stress has been associated with clinical depression (Burke, Davis, Otte, & Mohr, 2005; Lopez-Duran, Kovacs, & George, 2009).

In addition to acutely responding to stress, cortisol has been shown to follow a daily diurnal pattern that is characterized by high cortisol levels at waking, a sharp increase in cortisol (called the cortisol awakening response) reaching its highest point about 30 minutes after waking, and a decline across the rest of the day (Adam & Kumari, 2009; Linkowski et al., 1993; Schmidt-Reinwald et al., 1999). Extensive laboratory and naturalistic research have shown the responsivity of this diurnal cortisol rhythm to acute stressors (Adam, 2006; Doane & Adam, 2010) and the daily challenges (actual or anticipated) we face in the course of everyday life (Adam, Hawkley, Kudielka, & Cacioppo, 2006; Rohleder, Beulen, Chen, Wolf, & Kirschbaum, 2007; Stalder, Hucklebridge, Evans, & Clow, 2009). These acute changes typically have been shown to provide energy and attentional focus to help the individual cope with daily demands (Adam et al., 2006; P. D. Evans, Hucklebridge, Loveday, & Clow, 2012; Stalder, Evans, Hucklebridge, & Clow, 2010). However, repeated and sustained exposure to stress (i.e., chronic stress) has been shown to set into motion a cascade of physiological changes that disrupt the responsivity of cortisol and produce a flattened cortisol pattern (i.e., lower levels of cortisol at waking and higher evening levels of cortisol; Fries, Hesse, Hellhammer, & Hellhammer, 2005; Gunnar & Vazquez, 2001) that is associated with worse cognitive functioning and a wide range of mental and physical health outcomes (Adam et al., 2017; P. D. Evans et al., 2011).

Disparities in HPA-axis activity have been found by SES and by race, with lower income individuals and Black and Latinx individuals in the United States having altered basal cortisol levels (either notably higher or lower than typical) and flatter diurnal cortisol rhythms (Desantis et al., 2015; Lupien, King, Meaney, & McEwen, 2001). These forms of HPA axis dysregulation have been found to be typically indicative of stress exposure. Indeed, both the stress of poverty and experiences of racial discrimination have been found to predict these types of cortisol alterations (Adam et al., 2015; G. W. Evans & English, 2002; Zeiders, Doane, & Roosa, 2012; Zeiders, Hoyt, & Adam, 2014). Research on racial disparities in cortisol has, to our knowledge, not been conducted in young children, but disparities have been found to be present in adolescents, and differences in acute reactivity have even been found in infancy and are

predicted by maternal reports of discrimination during pregnancy (Thayer & Kuzawa, 2015). Racial differences in cortisol in adulthood have been found to be substantially explained by cumulative developmental histories of exposure to discrimination, with discrimination experiences during adolescence playing a particularly important role (Adam et al., 2015).

Sleep

Sleep is another system that is sensitive to social stressors and that is inhibited by activation of the sympathetic nervous system. Although sleep is affected by individual biological rhythms, daily schedules, and activity choices (Adam, Snell, & Pendry, 2007), sleep is also a stress-sensitive system, which is inhibited by activation of the sympathetic nervous system (Åkerstedt, 2006; Chrousos, 2009). Perceived stressors contribute to changes in both sleep quantity and quality (M. D. Hanson & Chen, 2010; Hicken, Lee, Ailshire, Burgard, & Williams, 2013; Sadeh, 1996; Sadeh, Raviv, & Gruber, 2000). In children, separation from or death of loved ones, child abuse, natural disasters, acute violence, and family stress all have been associated with poorer sleep, such as fewer sleep hours, more night awakenings, and lower sleep efficiency (Sadeh, 1996; Sadeh et al., 2000).

There are several aspects of sleep that can be studied in relation to health, including sleep duration, quality, latency (the time between getting in bed and falling asleep), and efficiency (total time sleeping divided by total time in bed). In experimental settings, sleep deprivation has been associated with an increase in levels of evening cortisol, circulating levels of proinflammatory cytokines, catecholamines, C-reactive protein, and elevations in blood pressure (Chrousos, 2009; Lusardi et al., 1999; McEwen, 2006; Meier-Ewert et al., 2004). As such, sleep deprivation has been shown to be a stressor that can both pose acute risk and inflict chronic damage to the organs and cardiovascular functioning (Lusardi et al., 1999). Short sleep has been associated with health consequences in adulthood, including hypertension, diabetes, cardiovascular disease, and increased mortality (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011; Gottlieb et al., 2006; Hale & Do, 2007; Lauderdale et al., 2006). In children and adolescents, shorter sleep duration has been associated with greater probability of being overweight (Lumeng et al., 2007; Snell, Adam, & Duncan, 2007), higher risk for depression and anxiety disorders (Dahl, 1996), behavioral disturbance, increased accidents, and impairments in attention, memory, and cognition, including executive functioning (Beebe, 2011; Sadeh, 2007; Sadeh, Gruber, & Raviv, 2003).

Socioeconomic and racial disparities in sleep have been found in children, adolescents, and adults—with low SES, Black, and Latinx children and adolescents showing shorter sleep than Whites (Buckhalt, 2011; Hale & Do, 2007). In nationally representative data, Blacks and Hispanics have been shown to have lower sleep hours (approximately 30 min; Adam et al., 2007), as well as lower sleep quality (Thomas, Bardwell, Ancoli-Israel, & Dimsdale, 2006) and lighter sleep (Tomfohr, Pung, Edwards, & Dimsdale, 2012) when compared

with non-Hispanic Whites. Perceived racial discrimination and, in particular, vigilance regarding racism has been found to be a predictor of lesser levels of deep sleep for Blacks (Tomfohr et al., 2012).

Multiple Stress-System Dysregulation: Allostatic Load

Importantly, there is feedback and coregulation across these and other stress-sensitive biological systems. The combined dysregulation across these systems, known as *allostatic load* (McEwen, 1998), has been linked with disparities in health and cognition across the lifespan (Adler & Stewart, 2010; Everson-Rose & Lewis, 2005; McEwen, 2004; Szanton, Gill, & Allen, 2005). Allostatic load is typically measured by constructing a count index of the number of different biological stress indicators that are elevated (or suppressed, for some indices) beyond a certain cut point (McEwen & Seeman, 1999). Disparities in allostatic load have been shown to exist by both SES and race, with lower SES individuals and ethnic and racial minorities having higher allostatic load in both adolescence and adulthood (McEwen & Seeman, 1999; Rainisch & Upchurch, 2013). In Black adolescents, allostatic load is predicted by higher perceived racial discrimination (Brody et al., 2014). Although allostatic load is rarely measured in childhood, early dysregulation of the elements of an allostatic load index are apparent (G. W. Evans & English, 2002), and cascading effects impacting related biological systems, resulting in allostatic load, are expected to be observed over time.

INTERVENTIONS DEMONSTRATED TO ALTER STRESS BIOLOGY

Evidence that early and ongoing disparities in stress exposure affect the body and brain in lasting ways does not immediately provide a hopeful message for those exposed to early or cumulative stress. Importantly, however, evidence of plasticity in biological systems, even into adulthood, and of the reversibility of previous negative impacts on biology is increasing (Lupien et al., 2009; Lupien et al., 2013). Researchers are investigating how they can change environments for children and adolescents (and adults) in ways that reduce stress exposure or reduce the psychological, behavioral, and biological impacts of stress (Lupien et al., 2013). Here, we reflect on social policies and interventions that we believe show promise of reducing stress disparities (i.e., disparities in stress exposure and stress biology) and their impact on disparities in health and developmental outcomes. We review how stress biology has been utilized in the development and evaluation of social policy and intervention research, focusing on studies that have: (a) intervened on poverty, race-based stress, or the social processes by which these types of stress are thought to have their effects on child and adolescent outcomes; and (b) measured biological outcomes relevant for health and/or developmental outcomes, including academic functioning, and prioritizing studies that consider impacts on children and adolescents.

It is worth noting that it is possible to reduce negative impacts of stress disparities by (a) reducing stress exposure itself (i.e., reducing material hardship or race-based social stress) or (b) helping individuals respond to and function more effectively in the face of the stressors that they do experience. Ultimately, we believe that responsibility to reduce stress disparities does not lie with those who are subjected to unjust treatment. To that end, we first consider how policy can reduce stress exposure in ways meaningful for stress biology. We also recognize that systemic change requires considerable time and commitment (on the part of policymakers and citizens alike) to redress inequities. In the meantime, interventions that help young people respond to the stressors they do experience are essential for reducing the consequences of stress disparities.

In this section, we review evidence regarding (a) policy that has changed families' living circumstances or material resources to reduce exposure to neighborhood or family disadvantage; (b) family/parenting interventions that worked to increase warm and supportive parenting and a positive home environment in the context of adversity, past trauma, or economic disadvantage; and (c) school-based interventions that have aimed to help students cope with and function more effectively in the context of the stressful circumstances.

Policies Aimed at Reducing Exposure to Stress and Disadvantage

Changes in policy can increase resources and reduce exposure to stress in ways that are likely to affect stress biology in both parents and their children. Some policies have changed over time or varied across geographic boundaries in ways that allow for quasi-experimental estimates of their impacts. Other prospective new policies have been tested experimentally through demonstration projects or other studies. Here, we consider studies of policy changes in which investigators included measures of stress or stress biology as outcomes or as pathways by which the policy changes are thought to affect health or academic outcomes.

Although there are many ways in which income inequality can limit a person's opportunity, two key (and related) pathways are a person's physical environment (e.g., home, neighborhood) and material resources (including cash and near-cash resources). Many families are forced to make choices between spending on housing, neighborhood quality and safety, and spending on other goods (Edin & Lein, 1997)—tradeoffs that can have implications for the well-being of both parents and children, given the importance of each (Adam & Chase-Lansdale, 2002; Coley, Leventhal, Lynch, & Kull, 2013; Crowley, 2003; Heissel, Sharkey, Torrats-Espinosa, Grant, & Adam, 2018; McGrath, Matthews, & Brady, 2006). Here, we consider several policy-related experiments or quasi-experiments that have acted on families' housing and neighborhood quality (via voucher or relocation programs) or income (via cash or near-cash transfers) and measured their impacts on stress biology.

Housing Voucher/Relocation Programs

Several publicly funded programs were created during the 1980s and 1990s in response to concerns about the poor conditions experienced in public housing developments, including the Moving to Opportunity for Fair Housing Demonstration (MTO), the Yonkers Project, and HOPE VI. As a random assignment experiment that reached over 4,000 families across five large cities, MTO is one of the most well-studied, with its initial and long-term findings both receiving a great deal of attention (Chetty, Hendren, & Katz, 2016; DeLuca, Clampet-Lundquist, & Edin, 2016). It is also the only such project to directly measure health outcomes, and several relevant health impacts have been identified among female heads of household. Assignment to the experimental[4] condition was associated with a large, significant reduction in the prevalence of extreme obesity and in symptoms of psychological distress more than 10 years after random assignment (Kling, Liebman, & Katz, 2007). Blood-spot data also revealed lower levels of risk for diabetes (measured through hemoglobin A1c levels) and inflammatory risk for cardiovascular disease (measured through C-reactive protein levels) among adults in the experimental group (Sanbonmatsu et al., 2011).

These findings are consistent with improvements in health found in other relocation studies that asked respondents about their physical and mental health but did not collect biomarker data. Participants in the Yonkers relocation project reported fewer diagnosed health conditions two years after moving to lower poverty neighborhoods (Fauth, Leventhal, & Brooks-Gunn, 2004). At the 7-year follow-up, only movers who stayed in their relocation neighborhoods reported better physical health than those who left them during the follow-up period (Fauth, Leventhal, & Brooks-Gunn, 2008). The evaluation of these and other relocation projects, such as HOPE VI, suggest that vouchers supporting a move to a lower poverty neighborhood can be beneficial for the health of those who want to move (Keene & Geronimus, 2011) and are able to find stability in their new neighborhood (Fauth et al., 2008; Ludwig et al., 2011). From these data, it is difficult to identify the aspect(s) of lower-poverty neighborhoods that matter or the biological mechanisms through which they operate. It is also important to note that relocation projects have not resulted in universally positive impacts on health or academic attainment (Chetty et al., 2016; Sharkey & Faber, 2014). Further study is needed to understand how to reduce stress disparities experienced by those living in disadvantaged neighborhoods, including whether improvements to housing conditions or their material resources can improve stress biology.

Income Supplements

Evidence that increases in household cash (or near cash) resources improve stress biology comes from several sources, including the expansion of the

[4]For simplicity, the term *experimental* refers to those who received low-poverty vouchers.

Earned Income Tax Credit (EITC), the rollout of the Food Stamp Program, and evidence from several cash transfer experiments. The 1993 expansion of the EITC increased payments to families with two or more children. Considering changes in the health of women who received the increased payments, W. N. Evans and Garthwaite (2010) estimated that income supplementation led to a 23% reduction in the total number of risky biomarkers for metabolic or cardiovascular disease. In particular, recipients were significantly less likely to report elevated levels of diastolic blood pressure and markers of inflammation (measured through C-reactive protein and albumin). Additionally, recipient mothers were more likely to report being in good or excellent health and indicated fewer poor mental health days in the last month compared to nonrecipient mothers (W. N. Evans & Garthwaite, 2010).

Another policy change that offers insight into the impact of increased income comes from county-level variation in when the federal Food Stamp Program was implemented. By considering the duration of food stamp availability from conception through early childhood, researchers estimated that having access to food stamps for the entirety of the period between conception and age 5 leads to a significant reduction in the number of markers for metabolic syndrome, with an effect of about 0.4 standard deviations (*SD*s; Hoynes, Schanzenbach, & Almond, 2012). Although food stamp access in utero had previously been associated with reduced incidence of low birth weight (Almond, Hoynes, & Schanzenbach, 2011), finding an association with health later in life suggests there are additional mechanisms through which food stamp receipt improves health. As food stamps are a *near-cash* source of aid (i.e., they augment families' total budget for consumption), it is possible that access to food stamps improves health in part by acting as an income supplement, which in turn reduces income-related stress for families.

Other research has also found that greater income during pregnancy and early childhood impacts child well-being in part through improvements to stress biology. One study that followed mothers across multiple pregnancies observed higher prenatal cortisol during pregnancies in which they reported less household income (Aizer, Stroud, & Buka, 2009). Children carried during these pregnancies completed, on average, a half year less of schooling than their siblings who experienced relatively lower prenatal cortisol (regardless of birth order; Aizer et al., 2009). Additionally, children whose parents had received cash transfers through the Oportunidades program in Mexico had lower average cortisol levels than children in comparison villages, with the largest difference in child cortisol found for those whose mothers had high depressive symptoms before receiving the income supplements (Fernald & Gunnar, 2009). In studies in Kenya, changes in income arising from a cash transfer experiment (Haushofer & Shapiro, 2016) and annual variation in rainfall that affected agricultural yields (Chemin, de Laat, & Haushofer, 2013) have also provided evidence that average cortisol levels are impacted by changes in household income. Future income supplementation experiments should continue to examine cortisol and examine a broader range of stress biomarkers and/or measures of allostatic load.

Family and Parenting Interventions

Beyond programs that attempt to reduce the impact of poverty by targeting income itself, a number of programs have targeted the family context with the aim of supporting positive parenting of individuals exposed to economic disadvantage (Brooks-Gunn et al., 1995). One theoretical rationale for targeting the home or family context of individuals living in poverty is provided by the family stress model, which argues that the stress associated with poverty or financial strain causes stress in parents, which in turn impacts their parenting in ways that are detrimental for child and adolescent development (K. J. Conger, Rueter, & Conger, 2000; R. D. Conger et al., 2002). Thus, when targeting income directly through policy or experimental income supplementation is not an option, intervention efforts to improve parent–child interactions are thought to be one way to improve the well-being of children and adolescents living in poverty and other stressful contexts. When an extreme breakdown in parenting is judged to have occurred, such as in the presence of child abuse or neglect, experimental interventions target the quality of the parent–child relationship in an attempt to improve the home environment or support placements outside the familial home. These interventions also attempt to ameliorate the effects of early traumatic stress on children. Similar interventions have targeted enhancing parenting in families of children adopted from early institutional (orphanage) care; such children may require enriched parenting to help counter the stress effects of early social deprivation and trauma encountered in institutional settings. We focus here on those interventions that included measures of stress biology as part of their outcome assessments.

Interventions Reducing or Reversing the Effects of Early Traumatic Stress

Some of the earliest insights into the malleability of stress biology in response to intervention in human populations came from studies of children exposed to the trauma of child abuse and neglect. As evidence has accumulated that child abuse and neglect can leave lasting marks on biological stress systems (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010; Gunnar & Vazquez, 2001), several interventions have been developed to reach children involved with the child welfare system, who disproportionately come from families facing the stresses of poverty or other hardship. Although each intervention has its own approach and theory of change, the interventions typically last from 6 to 12 weeks, with components that address the needs of both caregiver and child. For example, the Attachment and Biobehavioral Catch-up (ABC) intervention is an 8-week program for families of children aged 0 to 2 years that have experienced maltreatment, domestic violence, or placement instability that is focused on increasing parent–child synchrony, promoting nurturing parenting, and reducing parental behavior that would frighten or alarm the child (Bernard, Dozier, Bick, & Gordon, 2015). The Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) program is a therapeutic intervention designed to promote attachment and behavior for

children in foster care and increase placement stability. The intervention includes both individual and group support for foster parents, individualized treatment for children, and weekly playgroup sessions focused on social and emotional development and school readiness (P. A. Fisher, Stoolmiller, Gunnar, & Burraston, 2007).

Finally, the Kids in Transition to School program focuses on promoting school readiness among children in foster care as they transition to kindergarten, with child group sessions that focus on increasing school readiness (e.g., early literacy) and promoting prosocial and self-regulatory skills and parent group sessions that address behavioral management and school involvement (Graham, Pears, Kim, Bruce, & Fisher, 2018). Of the studies that have considered biological outcomes, most have measured cortisol, with some considering impacts on other outcomes. Overall, these interventions have shown that HPA-axis functioning can be improved, or further dysregulation prevented, in young children who have experienced neglect or maltreatment. After intervention, children in the treatment group have shown better-regulated diurnal cortisol than control participants, with higher waking levels (Bernard, Dozier, et al., 2015; D. B.-D. Fisher et al., 2007) and steeper slopes across the day (Bernard, Dozier, et al., 2015; P. A. Fisher et al., 2007; Graham et al., 2018). There is evidence that improved cortisol regulation can persist for several years after intervention (Bernard, Hostinar, & Dozier, 2015). Of note, not *every* intervention has found an impact on diurnal cortisol (Nelson & Spieker, 2013), and no impacts on afternoon/evening cortisol have emerged from these studies.

Participation in interventions that improved cortisol regulation was also associated with other promising outcomes for treatment group members, including greater incidence of secure attachment and impacts on cognitive flexibility and theory of mind (Bernard et al., 2012); lasting reductions in caregiving stress (P. A. Fisher & Stoolmiller, 2008); and more typical patterns of neural activation during response inhibition 5 years after intervention (Jankowski et al., 2017). In contrast, many of the control group children showed a blunted cortisol diurnal rhythm several years after intervention (Bernard, Dozier, et al., 2015; P. A. Fisher et al., 2007), consistent with studies that have found flattened cortisol slopes in children who stay with their birth parents rather than being placed in foster care following contact with the child welfare system (Bernard et al., 2010; Bernard, Dozier, et al., 2015). Together, these studies provide evidence that stress-sensitive systems influenced by early traumatic stress, such as child maltreatment, can be responsive to early intervention.

Community-Based Interventions for Families Under Stress

Although programs targeting children in the child welfare system primarily reach children and families during early childhood, programs that reach families experiencing stress disparities (e.g., high poverty, exposure to race-based discrimination) in their communities are far broader in the type of intervention, ages at time of intervention, and biological stress markers studied, including cortisol, inflammatory markers, and risk markers for metabolic syndrome.

Cortisol outcomes. Several interventions considered impacts on cortisol, including a home visiting program for mothers of infants from primarily low income families at medical risk (Bugental, Schwartz, & Lynch, 2010) and a preventive intervention offered following the death of one of an adolescent's parents (Luecken et al., 2010). Both interventions were associated with impacts on average cortisol levels relative to control group youth. In infants, the intervention was associated with reductions in midmorning cortisol at 1 year of age that predicted better verbal short-term memory 2 years later. The bereavement program also showed long-term impacts of the intervention, with treatment participants showing an increase in average cortisol level relative to control youth during a conflict discussion task that occurred 6 years after participation. Treatment youth also reported lower externalizing problems, suggesting lower incidence of the attenuated cortisol/elevated mental health symptoms profile that might otherwise follow from experiencing the death of a parent at a young age (Adam, 2006; Shirtcliff & Essex, 2008).

Immune/inflammatory and other biological outcomes. The Strong African American Families Program (SAAF) was a preventive intervention that randomly assigned over 600 rural Georgia families of 11-year-old children between a family-focused parenting intervention and a literature-only control condition (Brody et al., 2004). The program focused on the promotion of a regulated and communicative home environment, which was expected to operate by increasing (a) involved/vigilant parenting, racial socialization, and discussion of behaviors like sex and alcohol use; and (b) protective factors among youth, including self-regulation, future orientation, more negative attitudes about risk behaviors, and acceptance of parental influence. The seven-week intervention included separate skill-building for parents and youth as well as joint sessions in which families practiced applying the skills they learned.

Those who participated in SAAF were followed through early adulthood, with several studies measuring biological stress outcomes. Studies conducted when they were 19 and 25 years old identified several associations in the control group that were not present for those who received the intervention. In particular, among control youth: (a) higher adolescent-reported unsupportive parenting was associated with having more risk factors for metabolic syndrome (E. Chen, Miller, Yu, & Brody, 2018), and (b) having lived more adolescent years in poverty was associated with lower volume of several regions of the brain's limbic system (Brody et al., 2017). There is some evidence that improvements in parenting (i.e., a reduction in *un*supportive parenting) in the treatment group partially accounted for the reduced risk observed relative to the control condition (E. Chen et al., 2018; J. L. Hanson et al., 2019). Those who participated in the intervention also had lower levels of inflammatory cytokines measured in late adolescence, with the lowest levels of inflammation found among those who experienced more nurturant and less harsh parenting following the intervention (Miller, Brody, Yu, & Chen, 2014).

In two of the family-focused interventions, MTFC-P and SAAF, long-term follow-up has included brain scans of the young people who had experienced

either the intervention or control conditions. Both studies found that control, but not intervention, youth showed reduced connectivity between subcortical and cortical regions of the brain involved in self-regulation, attention, and behavior (J. L. Hanson et al., 2018). These differences in connectivity have been associated with attention and behavior problems more common among control children in the MTFC-P study (Bruce et al., 2013; Pollak et al., 2010) and have been partially accounted for by improvements in self-regulation and parental support that developed via the SAAF intervention (Brody et al., 2017; Jankowski et al., 2017). Differences in connectivity and volume were identified in a region of the hippocampus that is sensitive to corticosteroid levels and influenced by both stress exposure and parental support (Fa et al., 2014; J. L. Hanson et al., 2019; Luby et al., 2012; Pagliaccio et al., 2014; Teicher, Anderson, & Polcari, 2012). Together, these results provide evidence that young people randomly assigned to the control condition went on to develop markers of chronic stress exposure that are less prevalent in the intervention group, some of which in turn predicted cognitive and behavioral functioning and longer term health outcomes.

School-Based Interventions

Disparities in stress exposure occur not only at home but also through social and academic stressors faced in school. Schools are also where young people spend a substantial proportion of their waking hours. Student choices (including whether to participate in physical activity, consume healthy food/drink, or engage in risky behaviors) and experiences (including experiences of social support or social exclusion from teachers or peers) during this time can affect their health and well-being in the short term and contribute to the formation of habits and health trajectories that will affect their health later in life (Kimm et al., 2005). The school context is, therefore, an optimal space in which to implement interventions that aim to reduce student stress, improve health behaviors, and improve young people's health, performance, and well-being.

Although there are multiple aspects of children's and adolescents' school experiences that could be targeted to reduce stress disparities through interventions, very few published studies have done so with attention to effects on stress biology. For example, numerous rigorous randomized control studies have attempted to manipulate aspects of children's school contexts, such as bullying reduction programs, class size experiments, teacher training programs, or manipulations of per-pupil budgets, but we are not aware of school context-based interventions that have measured impacts on stress biology.

Rather, most school-based interventions incorporating biological measures have focused on modifying student perceptions of and responses to a wide range of stressors (including poverty-related stressors and stressors associated with discrimination and social exclusion). The most commonly employed approaches involve training in meditation or mindfulness. We focus first on school-based meditation or mindfulness interventions as an example of a well-studied approach with promising links to stress biology; we then focus

on a new study of our own designed to promote positive functioning and better regulation of stress biology, specifically in the face of exposure to race-based social stress.

Mindfulness and Meditation Interventions

With a rich history in Hindu and Buddhist traditions, meditation and mindfulness can be broadly understood as a practice of training attention to the present moment (Chambers, Gullone, & Allen, 2009; Kabat-Zinn, 2005). Although there are multiple traditions out of which many modern approaches have been developed, the main distinction has been drawn between *focused attention* practices, in which one concentrates on a single object (e.g., an image, body part, or the breath) and *open monitoring*, which promotes nonjudgmental awareness and observation. More recently, some scholars have argued for understanding these attributes as orthogonal, with beginners primarily engaged in learning to concentrate without letting the mind wander (Chambers et al., 2009; Chiesa, 2013). Interventions based on these principles have been implemented in schools, with wide variety in the approach and intensity of training and practice time that students experience (Esch, 2014). As such, our aim is not to draw inferences about the effects of specific practices or programs but rather to review relevant studies that have evaluated impacts on stress biology. We denote the type of practice where possible and otherwise use the general term *meditation* or *mindfulness* to refer to the broader category of included studies. Literature on connections between mindfulness, meditation, the stress response, and student biology and behavior suggests that school-based mindfulness or meditation practice may benefit young people through reductions in perceived stress and improved stress biology (Hölzel et al., 2011; Lane, Seskevich, & Pieper, 2007; Turakitwanakan, Mekseepralard, & Busarakumtragul, 2013).

Cortisol outcomes. Among adolescents, a school-based behavioral stress-education program was found to decrease cortisol levels and depressive symptomatology in adolescents making the transition to high school, with effect sizes on the order of 0.3 *SD*s for youth high in anger at baseline (Lupien et al., 2013). Completion of a mindfulness-based social emotional learning program was associated with steeper school-day cortisol slopes than peers in the control condition for fourth- and fifth-grade students (Schonert-Reichl et al., 2015). Studies outside the school context also found lower average cortisol after 2 to 4 months of meditation intervention, with effects ranging from 0.75 *SD* to 1.25 *SD* (MacLean et al., 1997). Others have found decreased acute cortisol levels following meditation sessions (Carlson, Speca, Faris, & Patel, 2007; Jevning, Wilson, & Davidson, 1978; Sudsuang, Chentanez, & Veluvan, 1991). A school-based yoga and mindfulness intervention for teachers led to lower waking cortisol levels after 16 weeks, compared with teachers at control schools (Harris, Jennings, Katz, Abenavoli, & Greenberg, 2016). Although these results are promising, findings have been inconsistent. Some studies have found no effects on cortisol outcomes (Sieverdes et al., 2014), and many

have employed data collection or analysis methods that are not adequate for modeling diurnal cortisol rhythms, which is problematic given the important associations between daily cortisol slopes and a wide range of health outcomes (Adam et al., 2017).

Blood pressure outcomes. Meditation interventions have also improved blood pressure among adolescents and adults. Several school-based interventions have reached students at elevated risk for hypertension (i.e., in the upper third or half of the distribution for their age or school) and led to reductions of approximately 0.3 to 0.4 *SD*s in systolic and diastolic blood pressure (Barnes, Pendergrast, Harshfield, & Treiber, 2008; Barnes, Treiber, & Johnson, 2004; Gregoski, Barnes, Tingen, Harshfield, & Treiber, 2011). As few studies have enrolled students across the blood pressure distribution, there is limited evidence of the extent to which meditation can be expected to change blood pressure levels in young people with healthy baseline levels. A meta-analysis of meditation studies with mostly adult populations finds that those with healthy blood pressure see reductions comparable in magnitude with those with elevated blood pressure (Anderson, Liu, & Kryscio, 2008). In contrast, a study in college students found a reduction in systolic blood pressure of 0.4 *SD*s for those at elevated risk of hypertension, but a (nonsignificant) reduction of less than 0.2 *SD*s for the full sample (Nidich et al., 2009). Further study will be needed to better estimate how meditation affects stress biology in healthy children and adolescents.

Sleep outcomes. Although there is not much literature on meditation or mindfulness and sleep among adolescents, several studies with adults have found improvements in sleep quality (Hülsheger, Feinholdt, & Nübold, 2015) and duration (Carlson & Garland, 2005) following a brief mindfulness or meditation intervention. Results from a small pilot study suggest that a school-based mindfulness intervention can improve sleep quality among high school women who reported poor sleep (Bei et al., 2013). These studies are fairly preliminary, and additional evidence is needed to identify the mechanisms through which meditation improves sleep.

Inflammation outcomes. Very little work has considered impacts of mindfulness or meditation on inflammation in adolescents, but several studies in adults provide evidence of impacts on certain inflammatory markers. Specifically, randomized mindfulness interventions have found reductions in proinflammatory gene expression (measured through NF-κB levels; Black & Slavich, 2016; Creswell et al., 2012) and small reductions in levels of C-reactive protein (Creswell et al., 2012; Malarkey, Jarjoura, & Klatt, 2013). A meta-analysis concluded that there is insufficient evidence to determine whether mindfulness impacts other common inflammatory markers including antibodies and interleukins (Black & Slavich, 2016). There is a need for additional research that meets standards for rigorous causal research design including the use of an appropriate control condition. Thus, preliminary

evidence suggests that meditation and mindfulness interventions can improve regulation of stress biology, particularly for populations under current stress, but much additional research is needed, especially studies on the long-term impacts of these interventions.

Racial and Ethnic Identity Promotion Interventions

In addition to these more general approaches designed to reduce the emotional and biological effects of stress exposure, our research group has been focused on testing interventions designed specifically to reduce the social, emotional, and biological effects of race-based stress and especially the stress of racial discrimination. Past theory and research have suggested that having a strong ethnic and racial identity, and in particular positive affect and pride in one's ethnic or racial heritage, tends to buffer or reduce the stress of discrimination and to be associated with positive outcomes, particularly for children and youth of color (Rivas-Drake et al., 2014). Correlational evidence has suggested that ethnic pride is also associated with better regulated stress biology in the form of steeper diurnal cortisol rhythms (Zeiders, Causadias, & White, 2018). In the Biology, Identity and Opportunity study, we are conducting a randomized controlled trial to test whether an 8-week ethnic and racial identity promotion intervention for high school freshman (see Umaña-Taylor & Douglass, 2017; Umaña-Taylor, Douglass, Updegraff, & Marsiglia, 2018; Umaña-Taylor, Kornienko, Douglass Bayless, & Updegraff, 2018) has positive effects on stress biology, as well as emotional, cognitive, physical, and academic outcomes. Ideally, we hope that such an intervention is positive for all youth but are hoping that it has particularly strong effects for youth of color, in that it could help to close ethnic and racial disparities in stress biology and developmental outcomes.

SUMMARY OF STUDIES OF POLICIES AND INTERVENTIONS AFFECTING STRESS BIOLOGY

Overall, we have provided evidence that the reduction of biological stress disparities need not rely only on increases in individual coping; indeed, the preliminary evidence reviewed here suggests that policies designed to reduce levels of economic and race-based stress in children in families can have positive effects on stress biology. Given the pervasiveness of stress disparities, a combination of policy interventions that reduce stress exposure and interventions designed to promote better coping with stress may hold the most promise. Future random assignment studies will preferably (a) measure and attempt to reduce disparities in stress exposure and/or increase coping with stress, (b) include a comprehensive battery of stress biology measures, and (c) follow children and adolescents longitudinally postintervention to observe not only short-term but also long-term effects on stress biology and developmental outcomes.

SUGGESTIONS FOR FUTURE RESEARCH AND CONCLUSION

In this chapter, we have argued that greater attention is needed to the disparities in stress exposure, biological responses to that stress exposure, and in understanding the origins of both economic and ethnic–racial disparities in health, developmental, and human-capital outcomes. Future research attempting to understand the origins of disparities should consider adding a "stress disparities" lens to their work, including both measures of various types of stress exposure and measures of stress biology. As the field advances, possible studies should move beyond examining single stress biomarkers to the inclusion of multiple aspects of the biological stress response and its downstream effects on related biological systems. Researchers should be aware that cumulative measures of stress exposure may have greater impact on stress biology and cumulative cross-system measures of stress biology may be better predictors of long-term outcomes. Studies should, if possible, gather multiple waves of stress biology data, in order to allow changes in stress biology can be observed in relation to changes in stress exposure, or in response to an intervening policy or program. In addition, studies would be advised to pay attention to the possibility of sensitive period effects, being particularly attentive to the possible embedding effects of early life stress, but not forget the possibility that ongoing, cumulative stress, and the interactions between early and ongoing stress may play an important role.

In choosing measures of stress, and of biology, researchers should engage in close conversation with community partners, who are aware both of the types of stressors typically encountered in their communities (which may be different than those expected a priori by researchers), and also are sensitive to the types of biological measures that will be perceived as acceptable for measurement within that community (which is in turn strongly based on the level of community trust the researcher has established through past interactions). Certain biological markers, such as DNA gathered to examine genetic polymorphisms, may be rightfully rejected or regarded with suspicion, as they can be perceived to locate the source of the stress-related negative outcomes as emerging from within the individual.

Overall, we believe that taking a stress disparities framing is an important new way to shed light on the origins and consequences of the vastly unequal distribution of resources, adversities, and stress within U.S. society and across the world. The addition of stress measures (including measures of stressor exposure and stress biomarkers) to intervention and policy studies designed to reduce disparities helps to shed light on the efficacy of social interventions and social policies and the pathways by which they have their effects on health and human capital outcomes. Additional policy research should add measures of stress exposure and stress biology as both potential outcomes of policy change and plausible mechanisms for the maintenance of inequality and consider the reduction in inequalities of perceived and biological stress as an important target and indicator by which we can measure progress toward reducing inequality and promoting equity.

REFERENCES

Adam, E. K. (2006). Transactions among adolescent trait and state emotion and diurnal and momentary cortisol activity in naturalistic settings. *Psychoneuroendocrinology*, *31*, 664–679. http://dx.doi.org/10.1016/j.psyneuen.2006.01.010

Adam, E. K. (2012). Emotion–cortisol transactions occur over multiple time scales in development: Implications for research on emotion and the development of emotional disorders. *Monographs of the Society for Research in Child Development, 77*, 17–27. http://dx.doi.org/10.1111/j.1540-5834.2012.00657.x

Adam, E. K., & Chase-Lansdale, P. L. (2002). Home sweet home(s): Parental separations, residential moves, and adjustment problems in low-income adolescent girls. *Developmental Psychology*, *38*, 792–805. http://dx.doi.org/10.1037/0012-1649.38.5.792

Adam, E. K., Hawkley, L. C., Kudielka, B. M., & Cacioppo, J. T. (2006). Day-to-day dynamics of experience–cortisol associations in a population-based sample of older adults. *Proceedings of the National Academy of Sciences*, *103*, 17058–17063. http://dx.doi.org/10.1073/pnas.0605053103

Adam, E. K., Heissel, J. A., Zeiders, K. H., Richeson, J. A., Ross, E. C., Ehrlich, K. B., . . . Eccles, J. S. (2015). Developmental histories of perceived racial discrimination and diurnal cortisol profiles in adulthood: A 20-year prospective study. *Psychoneuroendocrinology*, *62*, 279–291. http://dx.doi.org/10.1016/j.psyneuen.2015.08.018

Adam, E. K., & Kumari, M. (2009). Assessing salivary cortisol in large-scale, epidemiological research. *Psychoneuroendocrinology*, *34*, 1423–1436. http://dx.doi.org/10.1016/j.psyneuen.2009.06.011

Adam, E. K., Quinn, M. E., Tavernier, R., McQuillan, M. T., Dahlke, K. A., & Gilbert, K. E. (2017). Diurnal cortisol slopes and mental and physical health outcomes: A systematic review and meta-analysis. *Psychoneuroendocrinology*, *83*, 25–41. http://dx.doi.org/10.1016/j.psyneuen.2017.05.018

Adam, E. K., Snell, E. K., & Pendry, P. (2007). Sleep timing and quantity in ecological and family context: A nationally representative time-diary study. *Journal of Family Psychology*, *21*, 4–19. http://dx.doi.org/10.1037/0893-3200.21.1.4

Adler, N. E., & Stewart, J. (2010). Health disparities across the lifespan: Meaning, methods, and mechanisms. *Annals of the New York Academy of Sciences*, *1186*, 5–23. http://dx.doi.org/10.1111/j.1749-6632.2009.05337.x

Aizer, A., Stroud, L., & Buka, S. (2009). *Maternal stress and child well-being: Evidence from siblings*. Unpublished manuscript, Brown University, Providence, RI.

Åkerstedt, T. (2006). Psychosocial stress and impaired sleep. *Scandinavian Journal of Work, Environment & Health*, *32*, 493–501. http://dx.doi.org/10.5271/sjweh.1054

Albert, M. A. (2007). Inflammatory biomarkers, race/ethnicity and cardiovascular disease. *Nutrition Reviews*, *65*(12, Pt. 2), S234–S238. http://dx.doi.org/10.1111/j.1753-4887.2007.tb00369.x

Almond, D., Hoynes, H. W., & Schanzenbach, D. W. (2011). Inside the war on poverty: The impact of food stamps on birth outcomes. *The Review of Economics and Statistics*, *93*, 387–403. http://dx.doi.org/10.1162/REST_a_00089

Anderson, J. W., Liu, C., & Kryscio, R. J. (2008). Blood pressure response to transcendental meditation: A meta-analysis. *American Journal of Hypertension*, *21*, 310–316. http://dx.doi.org/10.1038/ajh.2007.65

Ayada, C., Toru, Ü., & Korkut, Y. (2015). The relationship of stress and blood pressure effectors. *Hippokratia*, *19*, 99–108.

Barnes, V. A., Pendergrast, R. A., Harshfield, G. A., & Treiber, F. A. (2008). Impact of breathing awareness meditation on ambulatory blood pressure and sodium handling in prehypertensive African American adolescents. *Ethnicity & Disease*, *18*, 1–5.

Barnes, V. A., Treiber, F. A., & Johnson, M. H. (2004). Impact of transcendental meditation on ambulatory blood pressure in African-American adolescents. *American Journal of Hypertension*, *17*, 366–369. http://dx.doi.org/10.1016/j.amjhyper.2003.12.008

Beebe, D. W. (2011). Cognitive, behavioral, and functional consequences of inadequate sleep in children and adolescents. *Pediatric Clinics of North America*, *58*, 649–665. http://dx.doi.org/10.1016/j.pcl.2011.03.002

Bei, B., Byrne, M. L., Ivens, C., Waloszek, J., Woods, M. J., Dudgeon, P., . . . Allen, N. B. (2013). Pilot study of a mindfulness-based, multi-component, in-school group sleep intervention in adolescent girls. *Early Intervention in Psychiatry*, *7*, 213–220. http://dx.doi.org/10.1111/j.1751-7893.2012.00382.x

Bernard, K., Butzin-Dozier, Z., Rittenhouse, J., & Dozier, M. (2010). Cortisol production patterns in young children living with birth parents vs. children placed in foster care following involvement of Child Protective Services. *Archives of Pediatrics & Adolescent Medicine*, *164*, 438–443. http://dx.doi.org/10.1001/archpediatrics.2010.54

Bernard, K., Dozier, M., Bick, J., & Gordon, M. K. (2015). Intervening to enhance cortisol regulation among children at risk for neglect: Results of a randomized clinical trial. *Development and Psychopathology*, *27*, 829–841. http://dx.doi.org/10.1017/S095457941400073X

Bernard, K., Dozier, M., Bick, J., Lewis-Morrarty, E., Lindhiem, O., & Carlson, E. (2012). Enhancing attachment organization among maltreated children: Results of a randomized clinical trial. *Child Development*, *83*, 623–636. http://dx.doi.org/10.1111/j.1467-8624.2011.01712.x

Bernard, K., Hostinar, C. E., & Dozier, M. (2015). Intervention effects on diurnal cortisol rhythms of Child Protective Services-referred infants in early childhood: Preschool follow-up results of a randomized clinical trial. *JAMA Pediatrics*, *169*, 112–119. http://dx.doi.org/10.1001/jamapediatrics.2014.2369

Black, D. S., & Slavich, G. M. (2016). Mindfulness meditation and the immune system: A systematic review of randomized controlled trials. *Annals of the New York Academy of Sciences*, *1373*, 13–24. http://dx.doi.org/10.1111/nyas.12998

Blascovich, J., Spencer, S. J., Quinn, D., & Steele, C. (2001). African Americans and high blood pressure: The role of stereotype threat. *Psychological Science*, *12*, 225–229. http://dx.doi.org/10.1111/1467-9280.00340

Blascovich, J., Vanman, E. J., Mendes, W. B., & Dickerson, S. (2011). *Social psychophysiology for social and personality psychology*. Thousand Oaks, CA: Sage. http://dx.doi.org/10.4135/9781446287842

Bosworth, H. B., Dudley, T., Olsen, M. K., Voils, C. I., Powers, B., Goldstein, M. K., & Oddone, E. Z. (2006). Racial differences in blood pressure control: Potential explanatory factors. *The American Journal of Medicine, 119*(1), 70.e9–70.e15. http://dx.doi.org/10.1016/j.amjmed.2005.08.019

Brody, G. H., Gray, J. C., Yu, T., Barton, A. W., Beach, S. R. H., Galván, A., . . . Sweet, L. H. (2017). Protective prevention effects on the association of poverty with brain development. *JAMA Pediatrics*, *171*, 46–52. http://dx.doi.org/10.1001/jamapediatrics.2016.2988

Brody, G. H., Lei, M.-K., Chae, D. H., Yu, T., Kogan, S. M., & Beach, S. R. H. (2014). Perceived discrimination among African American adolescents and allostatic load: A longitudinal analysis with buffering effects. *Child Development*, *85*, 989–1002. http://dx.doi.org/10.1111/cdev.12213

Brody, G. H., Murry, V. M., Gerrard, M., Gibbons, F. X., Molgaard, V., McNair, L., . . . Neubaum-Carlan, E. (2004). The Strong African American Families Program: Translating research into prevention programming. *Child Development*, *75*, 900–917. http://dx.doi.org/10.1111/j.1467-8624.2004.00713.x

Brondolo, E., Brady, N., Thompson, S., Tobin, J. N., Cassells, A., Sweeney, M., . . . Contrada, R. J. (2008). Perceived racism and negative affect: Analyses of trait and state measures of affect in a community sample. *Journal of Social and Clinical Psychology*, *27*, 150–173. http://dx.doi.org/10.1521/jscp.2008.27.2.150

Brondolo, E., Rieppi, R., Kelly, K. P., & Gerin, W. (2003). Perceived racism and blood pressure: A review of the literature and conceptual and methodological

critique. *Annals of Behavioral Medicine*, *25*, 55–65. http://dx.doi.org/10.1207/S15324796ABM2501_08

Brooks-Gunn, J., Klebanov, P. K., & Liaw, F.-R. (1995). The learning, physical, and emotional environment of the home in the context of poverty: The Infant Health and Development Program. *Children and Youth Services Review*, *17*, 251–276. http://dx.doi.org/10.1016/0190-7409(95)00011-Z

Broudy, R., Brondolo, E., Coakley, V., Brady, N., Cassells, A., Tobin, J. N., & Sweeney, M. (2007). Perceived ethnic discrimination in relation to daily moods and negative social interactions. *Journal of Behavioral Medicine*, *30*, 31–43. http://dx.doi.org/10.1007/s10865-006-9081-4

Brownley, K. A., Hurwitz, B. E., & Schneiderman, N. (2000). Cardiovascular Psychophysiology. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (2nd ed., pp. 224–263). New York, NY: Cambridge University Press.

Bruce, J., Fisher, P. A., Graham, A. M., Moore, W. E., III, Peake, S. J., & Mannering, A. M. (2013). Patterns of brain activation in foster children and nonmaltreated children during an inhibitory control task. *Development and Psychopathology*, *25*, 931–941. http://dx.doi.org/10.1017/S095457941300028X

Buckhalt, J. A. (2011). Insufficient sleep and the socioeconomic status achievement gap. *Child Development Perspectives*, *5*, 59–65. http://dx.doi.org/10.1111/j.1750-8606.2010.00151.x

Bugental, D. B., Schwartz, A., & Lynch, C. (2010). Effects of an early family intervention on children's memory: The mediating effects of cortisol levels. *Mind, Brain and Education*, *4*, 159–170. http://dx.doi.org/10.1111/j.1751-228X.2010.01095.x

Burke, H. M., Davis, M. C., Otte, C., & Mohr, D. C. (2005). Depression and cortisol responses to psychological stress: A meta-analysis. *Psychoneuroendocrinology*, *30*, 846–856. http://dx.doi.org/10.1016/j.psyneuen.2005.02.010

Cacioppo, J. T., Berntson, G. G., Sheridan, J. F., & McClintock, M. K. (2002). Multilevel integrative analyses of human behavior: Social neuroscience and the complementing nature of social and biological approaches. In J. T. Cacioppo, G. G. Berntson, R. Adolphs, C. S. Carter, R. J. Davidson, M. McClintock, . . . S. E. Taylor (Eds.), *Foundations in social neuroscience* (pp. 21–46). Cambridge, MA: MIT Press.

Cappuccio, F. P., Cooper, D., D'Elia, L., Strazzullo, P., & Miller, M. A. (2011). Sleep duration predicts cardiovascular outcomes: A systematic review and meta-analysis of prospective studies. *European Heart Journal*, *32*, 1484–1492. http://dx.doi.org/10.1093/eurheartj/ehr007

Carlson, L. E., & Garland, S. N. (2005). Impact of mindfulness-based stress reduction (MBSR) on sleep, mood, stress and fatigue symptoms in cancer outpatients. *International Journal of Behavioral Medicine*, *12*, 278–285. http://dx.doi.org/10.1207/s15327558ijbm1204_9

Carlson, L. E., Speca, M., Faris, P., & Patel, K. D. (2007). One year pre–post intervention follow-up of psychological, immune, endocrine and blood pressure outcomes of mindfulness-based stress reduction (MBSR) in breast and prostate cancer outpatients. *Brain, Behavior, and Immunity*, *21*, 1038–1049. http://dx.doi.org/10.1016/j.bbi.2007.04.002

Chambers, R., Gullone, E., & Allen, N. B. (2009). Mindful emotion regulation: An integrative review. *Clinical Psychology Review*, *29*, 560–572. http://dx.doi.org/10.1016/j.cpr.2009.06.005

Chemin, M., de Laat, J., & Haushofer, J. (2013, July). *Negative rainfall shocks increase levels of the stress hormone cortisol among poor farmers in Kenya* (Working paper). http://dx.doi.org/10.2139/ssrn.2294171

Chen, E., Miller, G. E., Yu, T., & Brody, G. H. (2018). Unsupportive parenting moderates the effects of family psychosocial intervention on metabolic syndrome in African American youth. *International Journal of Obesity*, *42*, 634–640. http://dx.doi.org/10.1038/ijo.2017.246

Chen, X., & Wang, Y. (2008). Tracking of blood pressure from childhood to adulthood: A systematic review and meta-regression analysis. *Circulation*, *117*, 3171–3180. http://dx.doi.org/10.1161/CIRCULATIONAHA.107.730366

Chetty, R., Hendren, N., & Katz, L. F. (2016). The effects of exposure to better neighborhoods on children: New evidence from the Moving to Opportunity Experiment. *The American Economic Review*, *106*, 855–902. http://dx.doi.org/10.1257/aer.20150572

Chiesa, A. (2013). The difficulty of defining mindfulness: Current thought and critical issues. *Mindfulness*, *4*, 255–268. http://dx.doi.org/10.1007/s12671-012-0123-4

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., Jr., . . . National High Blood Pressure Education Program Coordinating Committee. (2003). Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*, *42*, 1206–1252. http://dx.doi.org/10.1161/01.HYP.0000107251.49515.c2

Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews: Endocrinology*, *5*, 374–381. http://dx.doi.org/10.1038/nrendo.2009.106

Cohen, S., Frank, E., Doyle, W. J., Skoner, D. P., Rabin, B. S., & Gwaltney, J. M., Jr. (1998). Types of stressors that increase susceptibility to the common cold in healthy adults. *Health Psychology*, *17*, 214–223. http://dx.doi.org/10.1037/0278-6133.17.3.214

Coley, R. L., Leventhal, T., Lynch, A. D., & Kull, M. (2013). Relations between housing characteristics and the well-being of low-income children and adolescents. *Developmental Psychology*, *49*, 1775–1789. http://dx.doi.org/10.1037/a0031033

Colhoun, H. M., Hemingway, H., & Poulter, N. R. (1998). Socio-economic status and blood pressure: An overview analysis. *Journal of Human Hypertension*, *12*, 91–110. http://dx.doi.org/10.1038/sj.jhh.1000558

Conen, D., Glynn, R. J., Ridker, P. M., Buring, J. E., & Albert, M. A. (2009). Socioeconomic status, blood pressure progression, and incident hypertension in a prospective cohort of female health professionals. *European Heart Journal*, *30*, 1378–1384. http://dx.doi.org/10.1093/eurheartj/ehp072

Conger, K. J., Rueter, M. A., & Conger, R. D. (2000). The role of economic pressure in the lives of parents and their adolescents: The Family Stress Model. In L. J. Crockett & R. K. Silbereisen (Eds.), *Negotiating adolescence in times of social change* (pp. 201–223). Cambridge, England: Cambridge University Press.

Conger, R. D., Wallace, L. E., Sun, Y., Simons, R. L., McLoyd, V. C., & Brody, G. H. (2002). Economic pressure in African American families: A replication and extension of the family stress model. *Developmental Psychology*, *38*, 179–193. http://dx.doi.org/10.1037/0012-1649.38.2.179

Connor, T. J., & Leonard, B. E. (1998). Depression, stress and immunological activation: The role of cytokines in depressive disorders. *Life Sciences*, *62*, 583–606. http://dx.doi.org/10.1016/S0024-3205(97)00990-9

Creswell, J. D., Irwin, M. R., Burklund, L. J., Lieberman, M. D., Arevalo, J. M. G., Ma, J., . . . Cole, S. W. (2012). Mindfulness-based stress reduction training reduces loneliness and pro-inflammatory gene expression in older adults: A small randomized controlled trial. *Brain, Behavior, and Immunity*, *26*, 1095–1101. http://dx.doi.org/10.1016/j.bbi.2012.07.006

Crowley, S. (2003). The affordable housing crisis: Residential mobility of poor families and school mobility of poor children. *The Journal of Negro Education*, *72*, 22–38. http://dx.doi.org/10.2307/3211288

Dahl, R. E. (1996). The regulation of sleep and arousal: Development and psychopathology. *Development and Psychopathology*, *8*, 3–27. http://dx.doi.org/10.1017/S0954579400006945

DeLuca, S., Clampet-Lundquist, S., & Edin, K. (2016). *Coming of age in the other America*. New York, NY: Russell Sage Foundation.

Den Hond, E., Celis, H., Vandenhoven, G., O'Brien, E., & Staessen, J. A. (2003). Determinants of white-coat syndrome assessed by ambulatory blood pressure or

self-measured home blood pressure. *Blood Pressure Monitoring*, *8*, 37–40. http://dx.doi.org/10.1097/00126097-200302000-00008

Desantis, A. S., Kuzawa, C. W., & Adam, E. K. (2015). Developmental origins of flatter cortisol rhythms: Socioeconomic status and adult cortisol activity. *American Journal of Human Biology*, *27*, 458–467. http://dx.doi.org/10.1002/ajhb.22668

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*, 355–391. http://dx.doi.org/10.1037/0033-2909.130.3.355

Doane, L. D., & Adam, E. K. (2010). Loneliness and cortisol: Momentary, day-to-day, and trait associations. *Psychoneuroendocrinology*, *35*, 430–441. http://dx.doi.org/10.1016/j.psyneuen.2009.08.005

Edin, K., & Lein, L. (1997). Work, welfare, and single mothers' economic survival strategies. *American Sociological Review*, *62*, 253–266. http://dx.doi.org/10.2307/2657303

Elliott, W. J., & Black, H. R. (2007). Prehypertension. *Nature Reviews: Cardiology*, *4*, 538–548. http://dx.doi.org/10.1038/ncpcardio0989

Esch, T. (2014). The neurobiology of meditation and mindfulness. In S. Schmidt & H. Walach (Eds.), *Meditation—Neuroscientific approaches and philosophical implications* (pp. 153–173). Heidelberg, Germany: Springer International. http://dx.doi.org/10.1007/978-3-319-01634-4_9

Evans, G. W., & English, K. (2002). The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development*, *73*, 1238–1248. http://dx.doi.org/10.1111/1467-8624.00469

Evans, P. D., Fredhoi, C., Loveday, C., Hucklebridge, F., Aitchison, E., Forte, D., & Clow, A. (2011). The diurnal cortisol cycle and cognitive performance in the healthy old. *International Journal of Psychophysiology*, *79*, 371–377. http://dx.doi.org/10.1016/j.ijpsycho.2010.12.006

Evans, P. D., Hucklebridge, F., Loveday, C., & Clow, A. (2012). The cortisol awakening response is related to executive function in older age. *International Journal of Psychophysiology*, *84*, 201–204. http://dx.doi.org/10.1016/j.ijpsycho.2012.02.008

Evans, W. N., & Garthwaite, C. L. (2010, August). *Giving mom a break: The impact of higher EITC payments on maternal health* (NBER Working Paper No. 16296). Cambridge, MA: NBER.

Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health*, *26*, 469–500. http://dx.doi.org/10.1146/annurev.publhealth.26.021304.144542

Fa, M., Xia, L., Anunu, R., Kehat, O., Kriebel, M., Volkmer, H., & Richter-Levin, G. (2014). Stress modulation of hippocampal activity—Spotlight on the dentate gyrus. *Neurobiology of Learning and Memory*, *112*, 53–60. http://dx.doi.org/10.1016/j.nlm.2014.04.008

Falkner, B., Gidding, S. S., Portman, R., & Rosner, B. (2008). Blood pressure variability and classification of prehypertension and hypertension in adolescence. *Pediatrics*, *122*, 238–242. http://dx.doi.org/10.1542/peds.2007-2776

Falkner, B., Lurbe, E., & Schaefer, F. (2010). High blood pressure in children: Clinical and health policy implications. *Journal of Clinical Hypertension*, *12*, 261–276. http://dx.doi.org/10.1111/j.1751-7176.2009.00245.x

Fauth, R. C., Leventhal, T., & Brooks-Gunn, J. (2004). Short-term effects of moving from public housing in poor to middle-class neighborhoods on low-income, minority adults' outcomes. *Social Science & Medicine*, *59*, 2271–2284. http://dx.doi.org/10.1016/j.socscimed.2004.03.020

Fauth, R. C., Leventhal, T., & Brooks-Gunn, J. (2008). Seven years later: Effects of a neighborhood mobility program on poor Black and Latino adults' well-being. *Journal of Health and Social Behavior*, *49*, 119–130. http://dx.doi.org/10.1177/002214650804900201

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., . . . Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine*, *14*, 245–258. http://dx.doi.org/10.1016/S0749-3797(98)00017-8

Fernald, L. C. H., & Gunnar, M. R. (2009). Poverty-alleviation program participation and salivary cortisol in very low-income children. *Social Science & Medicine*, *68*, 2180–2189. http://dx.doi.org/10.1016/j.socscimed.2009.03.032

Fisher, D. B.-D., Serbin, L. A., Stack, D. M., Ruttle, P. L., Ledingham, J. E., & Schwartzman, A. E. (2007). Intergenerational predictors of diurnal cortisol secretion in early childhood. *Infant and Child Development*, *16*, 151–170. http://dx.doi.org/10.1002/icd.474

Fisher, P. A., & Stoolmiller, M. (2008). Intervention effects on foster parent stress: Associations with child cortisol levels. *Development and Psychopathology*, *20*, 1003–1021. http://dx.doi.org/10.1017/S0954579408000473

Fisher, P. A., Stoolmiller, M., Gunnar, M. R., & Burraston, B. O. (2007). Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. *Psychoneuroendocrinology*, *32*, 892–905. http://dx.doi.org/10.1016/j.psyneuen.2007.06.008

Flynn, J. T., Kaelber, D. C., Baker-Smith, C. M., Blowey, D., Carroll, A. E., Daniels, S. R., . . . Subcommittee on Screening and Management of High Blood Pressure in Children. (2017). Clinical practice guideline for screening and management of high blood pressure in children and adolescents. *Pediatrics*, *140*, e20171904. http://dx.doi.org/10.1542/peds.2017-1904

Franklin, S. S., & Mitchell, G. F. (2008). Aging, arterial function, and systolic hypertension. In J. L. Izzo, Jr., D. A. Sica, & H. R. Black (Eds.), *Hypertension primer* (4th ed., pp. 144–148). Philadelphia, PA: Lippincott, Williams, & Wilkins.

Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D. H. (2005). A new view on hypocortisolism. *Psychoneuroendocrinology*, *30*, 1010–1016. http://dx.doi.org/10.1016/j.psyneuen.2005.04.006

Glaser, R., & Kiecolt-Glaser, J. K. (2005). Stress-induced immune dysfunction: Implications for health. *Nature Reviews: Immunology*, *5*, 243–251. http://dx.doi.org/10.1038/nri1571

Glaser, R., Kiecolt-Glaser, J., Bonneau, R., Malarkey, W., Kennedy, S., & Hughes, J. (1992). Stress-induced modulation of the immune response to recombinant hepatitis B vaccine. *Psychosomatic Medicine*, *54*, 22–29. http://dx.doi.org/10.1097/00006842-199201000-00005

Glaser, R., Kiecolt-Glaser, J. K., Speicher, C. E., & Holliday, J. E. (1985). Stress, loneliness, and changes in herpesvirus latency. *Journal of Behavioral Medicine*, *8*, 249–260. http://dx.doi.org/10.1007/BF00870312

Gottlieb, D. J., Redline, S., Nieto, F. J., Baldwin, C. M., Newman, A. B., Resnick, H. E., & Punjabi, N. M. (2006). Association of usual sleep duration with hypertension: The Sleep Heart Health Study. *Sleep*, *29*, 1009–1014. http://dx.doi.org/10.1093/sleep/29.8.1009

Graham, A. M., Pears, K. C., Kim, H. K., Bruce, J., & Fisher, P. A. (2018). Effects of a school readiness intervention on hypothalamus-pituitary-adrenal axis functioning and school adjustment for children in foster care. *Development and Psychopathology*, *30*, 651–664. http://dx.doi.org/10.1017/S0954579417001171

Grant, K. E., Compas, B. E., Stuhlmacher, A. F., Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003). Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin*, *129*, 447–466. http://dx.doi.org/10.1037/0033-2909.129.3.447

Grant, K. E., McMahon, S. D., Carter, J. S., Carleton, R. A., Adam, E. K., & Chen, E. (2014). The influence of stressors on the development of psychopathology. In M. Lewis & K. Randolph (Eds.), *Handbook of developmental psychopathology*

(pp. 205–223). Boston, MA: Springer. http://dx.doi.org/10.1007/978-1-4614-9608-3_11

Gregoski, M. J., Barnes, V. A., Tingen, M. S., Harshfield, G. A., & Treiber, F. A. (2011). Breathing awareness meditation and LifeSkills Training programs influence upon ambulatory blood pressure and sodium excretion among African American adolescents. *Journal of Adolescent Health*, *48*, 59–64. http://dx.doi.org/10.1016/j.jadohealth.2010.05.019

Gruenewald, T. L., Cohen, S., Matthews, K. A., Tracy, R., & Seeman, T. E. (2009). Association of socioeconomic status with inflammation markers in Black and White men and women in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Social Science & Medicine*, *69*, 451–459. http://dx.doi.org/10.1016/j.socscimed.2009.05.018

Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology*, *13*, 515–538. http://dx.doi.org/10.1017/S0954579401003066

Hale, L., & Do, D. P. (2007). Racial differences in self-reports of sleep duration in a population-based study. *Sleep*, *30*, 1096–1103. http://dx.doi.org/10.1093/sleep/30.9.1096

Halfon, N., & Hochstein, M. (2002). Life Course Health Development: An integrated framework for developing health, policy, and research. *The Milbank Quarterly*, *80*, 433–479, iii. http://dx.doi.org/10.1111/1468-0009.00019

Hankin, B. L., Badanes, L. S., Abela, J. R. Z., & Watamura, S. E. (2010). Hypothalamic–pituitary–adrenal axis dysregulation in dysphoric children and adolescents: Cortisol reactivity to psychosocial stress from preschool through middle adolescence. *Biological Psychiatry*, *68*, 484–490. http://dx.doi.org/10.1016/j.biopsych.2010.04.004

Hanson, J. L., Gillmore, A. D., Yu, T., Holmes, C. J., Hallowell, E. S., Barton, A. W., . . . Brody, G. H. (2019). A family focused intervention influences hippocampal–prefrontal connectivity through gains in self-regulation. *Child Development*, *90*, 1389–1401. http://dx.doi.org/10.1111/cdev.13154

Hanson, M. D., & Chen, E. (2010). Daily stress, cortisol, and sleep: The moderating role of childhood psychosocial environments. *Health Psychology*, *29*, 394–402. http://dx.doi.org/10.1037/a0019879

Harris, A. R., Jennings, P. A., Katz, D. A., Abenavoli, R. M., & Greenberg, M. T. (2016). Promoting stress management and wellbeing in educators: Feasibility and efficacy of a school-based yoga and mindfulness intervention. *Mindfulness*, *7*, 143–154. http://dx.doi.org/10.1007/s12671-015-0451-2

Haushofer, J., & Shapiro, J. (2016). The short-term impact of unconditional cash transfers to the poor: Experimental evidence from Kenya. *The Quarterly Journal of Economics*, *131*, 1973–2042. http://dx.doi.org/10.1093/qje/qjw025

Heissel, J. A., Levy, D. J., & Adam, E. K. (2017). Stress, sleep, and performance on standardized tests: Understudied pathways to the achievement gap. *AERA Open.* http://dx.doi.org/10.1177/2332858417713488

Heissel, J. A., Sharkey, P. T., Torrats-Espinosa, G., Grant, K., & Adam, E. K. (2018). Violence and vigilance: The acute effects of community violent crime on sleep and cortisol. *Child Development*, *89*, e323–e331. http://dx.doi.org/10.1111/cdev.12889

Hertzman, C. (2012). Putting the concept of biological embedding in historical perspective. *Proceedings of the National Academy of Sciences*, *109*, 17160–17167. http://dx.doi.org/10.1073/pnas.1202203109

Hicken, M. T., Lee, H., Ailshire, J., Burgard, S. A., & Williams, D. R. (2013). "Every shut eye, ain't sleep": The role of racism-related vigilance in racial/ethnic disparities in sleep difficulty. *Race and Social Problems*, *5*, 100–112. http://dx.doi.org/10.1007/s12552-013-9095-9

Hölzel, B. K., Carmody, J., Vangel, M., Congleton, C., Yerramsetti, S. M., Gard, T., & Lazar, S. W. (2011). Mindfulness practice leads to increases in regional brain gray matter density. *Psychiatry Research: Neuroimaging*, *191*, 36–43. http://dx.doi.org/10.1016/j.pscychresns.2010.08.006

Hoynes, H. W., Schanzenbach, D. W., & Almond, D. (2012). Long run impacts of childhood access to the safety net. *American Economic Review*, *106*, 903–934.

Hülsheger, U. R., Feinholdt, A., & Nübold, A. (2015). A low-dose mindfulness intervention and recovery from work: Effects on psychological detachment, sleep quality, and sleep duration. *Journal of Occupational and Organizational Psychology*, *88*, 464–489. http://dx.doi.org/10.1111/joop.12115

Jackson, P. B., & Stewart, Q. T. (2003). A research agenda for the Black middle class: Work stress, survival strategies, and mental health. *Journal of Health and Social Behavior*, *44*, 442–455. http://dx.doi.org/10.2307/1519789

Jankowski, K. F., Bruce, J., Beauchamp, K. G., Roos, L. E., Moore, W. E., III, & Fisher, P. A. (2017). Preliminary evidence of the impact of early childhood maltreatment and a preventive intervention on neural patterns of response inhibition in early adolescence. *Developmental Science*, *20*, e12413. http://dx.doi.org/10.1111/desc.12413

Jevning, R., Wilson, A. F., & Davidson, J. M. (1978). Adrenocortical activity during meditation. *Hormones and Behavior*, *10*, 54–60. http://dx.doi.org/10.1016/0018-506X(78)90024-7

Johnson, S. B., Riis, J. L., & Noble, K. G. (2016). State of the art review: Poverty and the developing brain. *Pediatrics*, *137*, e20153075. http://dx.doi.org/10.1542/peds.2015-3075

Kabat-Zinn, J. (2005). *Wherever you go, there you are: Mindfulness meditation in everyday life*. New York, NY: Hachette Books.

Kaczmarek, M., Stawinska-Witoszynska, B., Krzyzaniak, A., Krzywinska-Wiewiorowska, M., & Siwinska, A. (2015). Who is at higher risk of hypertension? Socioeconomic status differences in blood pressure among Polish adolescents: A population-based ADOPOLNOR study. *European Journal of Pediatrics*, *174*, 1461–1473. http://dx.doi.org/10.1007/s00431-015-2554-0

Keene, D. E., & Geronimus, A. T. (2011). "Weathering" HOPE VI: The importance of evaluating the population health impact of public housing demolition and displacement. *Journal of Urban Health*, *88*, 417–435. http://dx.doi.org/10.1007/s11524-011-9582-5

Keller, S. E., Shiflett, S. C., Schleifer, S. J., & Bartlett, J. A. (1994). Stress, immunity, and health. In R. Glaser & J. K. Kiecolt-Glaser (Eds.), *Handbook of human stress and immunity* (pp. 217–244). San Diego, CA: Academic Press. http://dx.doi.org/10.1016/B978-0-12-285960-1.50013-7

Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Psychoneuroimmunology: Psychological influences on immune function and health. *Journal of Consulting and Clinical Psychology*, *70*, 537–547. http://dx.doi.org/10.1037/0022-006X.70.3.537

Kiecolt-Glaser, J. K., Page, G. G., Marucha, P. T., MacCallum, R. C., & Glaser, R. (1998). Psychological influences on surgical recovery. Perspectives from psychoneuroimmunology. *American Psychologist*, *53*, 1209–1218. http://dx.doi.org/10.1037/0003-066X.53.11.1209

Kimm, S. Y., Glynn, N. W., Obarzanek, E., Kriska, A. M., Daniels, S. R., Barton, B. A., & Liu, K. (2005). Relation between the changes in physical activity and body-mass index during adolescence: A multicentre longitudinal study. *Lancet*, *366*, 301–307. http://dx.doi.org/10.1016/S0140-6736(05)66837-7

Kirschbaum, C., & Hellhammer, D. H. (1989). Salivary cortisol in psychobiological research: An overview. *Neuropsychobiology*, *22*, 150–169. http://dx.doi.org/10.1159/000118611

Kling, J. R., Liebman, J. B., & Katz, L. F. (2007). Experimental analysis of neighborhood effects. *Econometrica*, *75*, 83–119. http://dx.doi.org/10.1111/j.1468-0262.2007.00733.x

Kuzawa, C. W., & Sweet, E. (2009). Epigenetics and the embodiment of race: Developmental origins of U.S. racial disparities in cardiovascular health. *American Journal of Human Biology*, *21*, 2–15. http://dx.doi.org/10.1002/ajhb.20822

Lande, M. B., Adams, H., Falkner, B., Waldstein, S. R., Schwartz, G. J., Szilagyi, P. G., . . . Palumbo, D. (2009). Parental assessments of internalizing and externalizing behavior and executive function in children with primary hypertension. *The Journal of Pediatrics*, *154*, 207–212.e1. http://dx.doi.org/10.1016/j.jpeds.2008.08.017

Lande, M. B., Kaczorowski, J. M., Auinger, P., Schwartz, G. J., & Weitzman, M. (2003). Elevated blood pressure and decreased cognitive function among school-age children and adolescents in the United States. *The Journal of Pediatrics*, *143*, 720–724. http://dx.doi.org/10.1067/S0022-3476(03)00412-8

Landsbergis, P. A., Schnall, P. L., Pickering, T. G., Warren, K., & Schwartz, J. E. (2003). Lower socioeconomic status among men in relation to the association between job strain and blood pressure. *Scandinavian Journal of Work, Environment & Health*, *29*, 206–215. http://dx.doi.org/10.5271/sjweh.723

Lane, J. D., Seskevich, J. E., & Pieper, C. F. (2007). Brief meditation training can improve perceived stress and negative mood. *Alternative Therapies in Health and Medicine*, *13*, 38–44.

Lauderdale, D. S., Knutson, K. L., Yan, L. L., Rathouz, P. J., Hulley, S. B., Sidney, S., & Liu, K. (2006). Objectively measured sleep characteristics among early-middle-aged adults: The CARDIA study. *American Journal of Epidemiology*, *164*, 5–16. http://dx.doi.org/10.1093/aje/kwj199

Leng, B., Jin, Y., Li, G., Chen, L., & Jin, N. (2015). Socioeconomic status and hypertension: A meta-analysis. *Journal of Hypertension*, *33*, 221–229. http://dx.doi.org/10.1097/HJH.0000000000000428

Levinson, S., Liu, K., Stamler, J., Stamler, R., Whipple, I., Ausbrook, D., & Berkson, D. (1985). Ethnic differences in blood pressure and heart rate of Chicago school children. *American Journal of Epidemiology*, *122*, 366–377. http://dx.doi.org/10.1093/oxfordjournals.aje.a114117

Levy, D. J., Heissel, J. A., Richeson, J. A., & Adam, E. K. (2016). Psychological and biological responses to race-based social stress as pathways to disparities in educational outcomes. *American Psychologist*, *71*, 455–473. http://dx.doi.org/10.1037/a0040322

Linkowski, P., Van Onderbergen, A., Kerkhofs, M., Bosson, D., Mendlewicz, J., & Van Cauter, E. (1993). Twin study of the 24-h cortisol profile: Evidence for genetic control of the human circadian clock. *The American Journal of Physiology, Endocrinology and Metabolism*, *264*, E173–E181. http://dx.doi.org/10.1152/ajpendo.1993.264.2.E173

Lopez-Duran, N. L., Kovacs, M., & George, C. J. (2009). Hypothalamic–pituitary–adrenal axis dysregulation in depressed children and adolescents: A meta-analysis. *Psychoneuroendocrinology*, *34*, 1272–1283. http://dx.doi.org/10.1016/j.psyneuen.2009.03.016

Lovallo, W. R. (2016). *Stress and health: Biological and psychological interactions* (3rd ed.). Thousand Oaks, CA: Sage.

Luby, J. L., Barch, D. M., Belden, A., Gaffrey, M. S., Tillman, R., Babb, C., . . . Botteron, K. N. (2012). Maternal support in early childhood predicts larger hippocampal volumes at school age. *Proceedings of the National Academy of Sciences*, *109*, 2854–2859. http://dx.doi.org/10.1073/pnas.1118003109

Ludwig, J., Sanbonmatsu, L., Gennetian, L., Adam, E., Duncan, G. J., Katz, L. F., . . . McDade, T. W. (2011). Neighborhoods, obesity, and diabetes—A randomized social experiment. *The New England Journal of Medicine*, *365*, 1509–1519. http://dx.doi.org/10.1056/NEJMsa1103216

Luecken, L. J., Hagan, M. J., Sandler, I. N., Tein, J. Y., Ayers, T. S., & Wolchik, S. A. (2010). Cortisol levels six-years after participation in the Family Bereavement Program. *Psychoneuroendocrinology*, *35*, 785–789. http://dx.doi.org/10.1016/j.psyneuen.2009.11.002

Lumeng, J. C., Somashekar, D., Appugliese, D., Kaciroti, N., Corwyn, R. F., & Bradley, R. H. (2007). Shorter sleep duration is associated with increased risk for being overweight at ages 9 to 12 years. *Pediatrics*, *120*, 1020–1029. http://dx.doi.org/10.1542/peds.2006-3295

Lupien, S. J., King, S., Meaney, M. J., & McEwen, B. S. (2001). Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. *Development and Psychopathology*, *13*, 653–676. http://dx.doi.org/10.1017/S0954579401003133

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews: Neuroscience*, *10*, 434–445. http://dx.doi.org/10.1038/nrn2639

Lupien, S. J., Ouellet-Morin, I., Trépanier, L., Juster, R. P., Marin, M. F., Francois, N., . . . Plusquellec, P. (2013). The DeStress for Success Program: Effects of a stress education program on cortisol levels and depressive symptomatology in adolescents making the transition to high school. *Neuroscience*, *249*, 74–87. http://dx.doi.org/10.1016/j.neuroscience.2013.01.057

Lusardi, P., Zoppi, A., Preti, P., Pesce, R. M., Piazza, E., & Fogari, R. (1999). Effects of insufficient sleep on blood pressure in hypertensive patients: A 24-h study. *American Journal of Hypertension*, *12*, 63–68. http://dx.doi.org/10.1016/S0895-7061(98)00200-3

MacLean, C. R. K., Walton, K. G., Wenneberg, S. R., Levitsky, D. K., Mandarino, J. P., Waziri, R., . . . Schneider, R. H. (1997). Effects of the Transcendental Meditation program on adaptive mechanisms: Changes in hormone levels and responses to stress after 4 months of practice. *Psychoneuroendocrinology*, *22*, 277–295. http://dx.doi.org/10.1016/S0306-4530(97)00003-6

Maes, M., Ombelet, W., De Jongh, R., Kenis, G., & Bosmans, E. (2001). The inflammatory response following delivery is amplified in women who previously suffered from major depression, suggesting that major depression is accompanied by a sensitization of the inflammatory response system. *Journal of Affective Disorders*, *63*, 85–92. http://dx.doi.org/10.1016/S0165-0327(00)00156-7

Maier, S. F. (2003). Bi-directional immune–brain communication: Implications for understanding stress, pain, and cognition. *Brain, Behavior, and Immunity*, *17*, 69–85. http://dx.doi.org/10.1016/S0889-1591(03)00032-1

Malarkey, W. B., Jarjoura, D., & Klatt, M. (2013). Workplace based mindfulness practice and inflammation: A randomized trial. *Brain, Behavior, and Immunity*, *27*, 145–154. http://dx.doi.org/10.1016/j.bbi.2012.10.009

Mancia, G., Sega, R., Bravi, C., Vito, G., Valagussa, F., Cesana, G., & Zanchetti, A. (1995). Ambulatory blood pressure normality: Results from the PAMELA study. *Journal of Hypertension*, *13*, 1377–1390. http://dx.doi.org/10.1097/00004872-199512000-00003

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *The New England Journal of Medicine*, *338*, 171–179. http://dx.doi.org/10.1056/NEJM199801153380307

McEwen, B. S. (2004). Protection and damage from acute and chronic stress: Allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Annals of the New York Academy of Sciences*, *1032*, 1–7. http://dx.doi.org/10.1196/annals.1314.001

McEwen, B. S. (2006). Sleep deprivation as a neurobiologic and physiologic stressor: Allostasis and allostatic load. *Metabolism: Clinical and Experimental*, *55*(10, Suppl. 2), S20–S23. http://dx.doi.org/10.1016/j.metabol.2006.07.008

McEwen, B. S. (2012). Brain on stress: How the social environment gets under the skin. *Proceedings of the National Academy of Sciences*, *109*, 17180–17185. http://dx.doi.org/10.1073/pnas.1121254109

McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *896*, 30–47. http://dx.doi.org/10.1111/j.1749-6632.1999.tb08103.x

McGrath, J. J., Matthews, K. A., & Brady, S. S. (2006). Individual versus neighborhood socioeconomic status and race as predictors of adolescent ambulatory blood pressure and heart rate. *Social Science & Medicine*, *63*, 1442–1453. http://dx.doi.org/10.1016/j.socscimed.2006.03.019

McKim, D. B., Niraula, A., Tarr, A. J., Wohleb, E. S., Sheridan, J. F., & Godbout, J. P. (2016). Neuroinflammatory dynamics underlie memory impairments after repeated social defeat. *The Journal of Neuroscience*, *36*, 2590–2604. http://dx.doi.org/10.1523/JNEUROSCI.2394-15.2016

Meier-Ewert, H. K., Ridker, P. M., Rifai, N., Regan, M. M., Price, N. J., Dinges, D. F., & Mullington, J. M. (2004). Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk. *Journal of the American College of Cardiology*, *43*, 678–683. http://dx.doi.org/10.1016/j.jacc.2003.07.050

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology*, *60*, 501–524. http://dx.doi.org/10.1146/annurev.psych.60.110707.163551

Miller, G. E., Brody, G. H., Yu, T., & Chen, E. (2014). A family-oriented psychosocial intervention reduces inflammation in low-SES African American youth. *Proceedings of the National Academy of Sciences*, *111*, 11287–11292. http://dx.doi.org/10.1073/pnas.1406578111

Miller, G. E., & Chen, E. (2013). The biological residue of childhood poverty. *Child Development Perspectives*, *7*, 67–73. http://dx.doi.org/10.1111/cdep.12021

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*, 959–997. http://dx.doi.org/10.1037/a0024768

Miller, G. E., Cohen, S., & Ritchey, A. K. (2002). Chronic psychological stress and the regulation of pro-inflammatory cytokines: A glucocorticoid-resistance model. *Health Psychology*, *21*, 531–541. http://dx.doi.org/10.1037/0278-6133.21.6.531

Muntner, P., He, J., Cutler, J. A., Wildman, R. P., & Whelton, P. K. (2004). Trends in blood pressure among children and adolescents. *JAMA*, *291*, 2107–2113. http://dx.doi.org/10.1001/jama.291.17.2107

Nelson, E. M., & Spieker, S. J. (2013). Intervention effects on morning and stimulated cortisol responses among toddlers in foster care. *Infant Mental Health Journal*, *34*, 211–221. http://dx.doi.org/10.1002/imhj.21382

Nidich, S. I., Rainforth, M. V., Haaga, D. A., Hagelin, J., Salerno, J. W., Travis, F., . . . Schneider, R. H. (2009). A randomized controlled trial on effects of the Transcendental Meditation program on blood pressure, psychological distress, and coping in young adults. *American Journal of Hypertension*, *22*, 1326–1331. http://dx.doi.org/10.1038/ajh.2009.184

Pager, D., Bonikowski, B., & Western, B. (2009). Discrimination in a low-wage labor market: A field experiment. *American Sociological Review*, *74*, 777–799. http://dx.doi.org/10.1177/000312240907400505

Pager, D., & Shepherd, H. (2008). The sociology of discrimination: Racial discrimination in employment, housing, credit, and consumer markets. *Annual Review of Sociology*, *34*, 181–209. http://dx.doi.org/10.1146/annurev.soc.33.040406.131740

Pagliaccio, D., Luby, J. L., Bogdan, R., Agrawal, A., Gaffrey, M. S., Belden, A. C., . . . Barch, D. M. (2014). Stress-system genes and life stress predict cortisol levels and

amygdala and hippocampal volumes in children. *Neuropsychopharmacology*, *39*, 1245–1253. http://dx.doi.org/10.1038/npp.2013.327

Pickering, T. G. (2001). Mental stress as a causal factor in the development of hypertension and cardiovascular disease. *Current Hypertension Reports*, *3*, 249–254. http://dx.doi.org/10.1007/s11906-001-0047-1

Pickering, T. G., Shimbo, D., & Haas, D. (2006). Ambulatory blood-pressure monitoring. *The New England Journal of Medicine*, *354*, 2368–2374. http://dx.doi.org/10.1056/NEJMra060433

Pollak, S. D., Nelson, C. A., Schlaak, M. F., Roeber, B. J., Wewerka, S. S., Wiik, K. L., . . . Gunnar, M. R. (2010). Neurodevelopmental effects of early deprivation in postinstitutionalized children. *Child Development*, *81*, 224–236. http://dx.doi.org/10.1111/j.1467-8624.2009.01391.x

Rabin, B. S. (1999). *Stress, immune function, and health: The connection*. New York, NY: Wiley-Liss.

Rachal Pugh, C., Fleshner, M., Watkins, L. R., Maier, S. F., & Rudy, J. W. (2001). The immune system and memory consolidation: A role for the cytokine IL-1beta. *Neuroscience and Biobehavioral Reviews*, *25*, 29–41. http://dx.doi.org/10.1016/S0149-7634(00)00048-8

Rainisch, B. K. W., & Upchurch, D. M. (2013). Sociodemographic correlates of allostatic load among a national sample of adolescents: Findings from the National Health and Nutrition Examination Survey, 1999–2008. *Journal of Adolescent Health*, *53*, 506–511. http://dx.doi.org/10.1016/j.jadohealth.2013.04.020

Reiche, E. M. V., Nunes, S. O. V., & Morimoto, H. K. (2004). Stress, depression, the immune system, and cancer. *The Lancet: Oncology*, *5*, 617–625. http://dx.doi.org/10.1016/S1470-2045(04)01597-9

Rivas-Drake, D., Syed, M., Umaña-Taylor, A., Markstrom, C., French, S., Schwartz, S. J., . . . Ethnic and Racial Identity in the 21st Century Study Group. (2014). Feeling good, happy, and proud: A meta-analysis of positive ethnic–racial affect and adjustment. *Child Development*, *85*, 77–102. http://dx.doi.org/10.1111/cdev.12175

Rohleder, N., Beulen, S. E., Chen, E., Wolf, J. M., & Kirschbaum, C. (2007). Stress on the dance floor: The cortisol stress response to social-evaluative threat in competitive ballroom dancers. *Personality and Social Psychology Bulletin*, *33*, 69–84. http://dx.doi.org/10.1177/0146167206293986

Rosner, B., Cook, N. R., Daniels, S., & Falkner, B. (2013). Childhood blood pressure trends and risk factors for high blood pressure: The NHANES Experience 1988–2008. *Hypertension*, *62*, 247–254. http://dx.doi.org/10.1161/HYPERTENSIONAHA.111.00831

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*, *99*, 2192–2217. http://dx.doi.org/10.1161/01.CIR.99.16.2192

Sadeh, A. (1996). Stress, trauma, and sleep in children. *Child and Adolescent Psychiatric Clinics of North America*, *5*, 685–700. http://dx.doi.org/10.1016/S1056-4993(18)30356-0

Sadeh, A. (2007). Consequences of sleep loss or sleep disruption in children. *Sleep Medicine Clinics*, *2*, 513–520. http://dx.doi.org/10.1016/j.jsmc.2007.05.012

Sadeh, A., Gruber, R., & Raviv, A. (2003). The effects of sleep restriction and extension on school-age children: What a difference an hour makes. *Child Development*, *74*, 444–455. http://dx.doi.org/10.1111/1467-8624.7402008

Sadeh, A., Raviv, A., & Gruber, R. (2000). Sleep patterns and sleep disruptions in school-age children. *Developmental Psychology*, *36*, 291–301. http://dx.doi.org/10.1037/0012-1649.36.3.291

Sanbonmatsu, L., Ludwig, J., Katz, L. F., Gennetian, L. A., Duncan, G. J., Kessler, R. C., . . . Lindau, S. T. (2011, November). *Moving to Opportunity for fair housing demonstration program: Final impacts evaluation*. Washington, DC: U.S. Department of Housing and Urban Development, Office of Policy Development and Research.

Schmeer, K. K., & Tarrence, J. (2018). Racial–ethnic disparities in inflammation: Evidence of weathering in childhood? *Journal of Health and Social Behavior*, *59*, 411–428. http://dx.doi.org/10.1177/0022146518784592

Schmeer, K. K., & Yoon, A. (2016). Socioeconomic status inequalities in low-grade inflammation during childhood. *Archives of Disease in Childhood*, *101*, 1043–1047. http://dx.doi.org/10.1136/archdischild-2016-310837

Schmidt-Reinwald, A., Pruessner, J. C., Hellhammer, D. H., Federenko, I., Rohleder, N., Schürmeyer, T. H., & Kirschbaum, C. (1999). The cortisol response to awakening in relation to different challenge tests and a 12-hour cortisol rhythm. *Life Sciences*, *64*, 1653–1660. http://dx.doi.org/10.1016/S0024-3205(99)00103-4

Schnall, P. L., Schwartz, J. E., Landsbergis, P. A., Warren, K., & Pickering, T. G. (1992). Relation between job strain, alcohol, and ambulatory blood pressure. *Hypertension*, *19*, 488–494. http://dx.doi.org/10.1161/01.HYP.19.5.488

Schonert-Reichl, K. A., Oberle, E., Lawlor, M. S., Abbott, D., Thomson, K., Oberlander, T. F., & Diamond, A. (2015). Enhancing cognitive and social-emotional development through a simple-to-administer mindfulness-based school program for elementary school children: A randomized controlled trial. *Developmental Psychology*, *51*, 52–66. http://dx.doi.org/10.1037/a0038454

Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin*, *130*, 601–630. http://dx.doi.org/10.1037/0033-2909.130.4.601

Sharkey, P., & Faber, J. W. (2014). Where, when, why, and for whom do residential contexts matter? Moving away from the dichotomous understanding of neighborhood effects. *Annual Review of Sociology*, *40*, 559–579. http://dx.doi.org/10.1146/annurev-soc-071913-043350

Shirtcliff, E. A., & Essex, M. J. (2008). Concurrent and longitudinal associations of basal and diurnal cortisol with mental health symptoms in early adolescence. *Developmental Psychobiology*, *50*, 690–703. http://dx.doi.org/10.1002/dev.20336

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *JAMA*, *301*, 2252–2259. http://dx.doi.org/10.1001/jama.2009.754

Shonkoff, J. P., Garner, A. S., Siegel, B. S., Dobbins, M. I., Earls, M. F., McGuinn, L., . . . Wegner, L. M. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, *129*, e232–e246. http://dx.doi.org/10.1542/peds.2011-2663

Sieverdes, J. C., Mueller, M., Gregoski, M. J., Brunner-Jackson, B., McQuade, L., Matthews, C., & Treiber, F. A. (2014). Effects of Hatha yoga on blood pressure, salivary α-amylase, and cortisol function among normotensive and prehypertensive youth. *Journal of Alternative and Complementary Medicine*, *20*, 241–250. http://dx.doi.org/10.1089/acm.2013.0139

Snell, E. K., Adam, E. K., & Duncan, G. J. (2007). Sleep and the body mass index and overweight status of children and adolescents. *Child Development*, *78*, 309–323. http://dx.doi.org/10.1111/j.1467-8624.2007.00999.x

Spruill, T. M. (2010). Chronic psychosocial stress and hypertension. *Current Hypertension Reports*, *12*, 10–16. http://dx.doi.org/10.1007/s11906-009-0084-8

Stalder, T., Evans, P., Hucklebridge, F., & Clow, A. (2010). Associations between psychosocial state variables and the cortisol awakening response in a single case study. *Psychoneuroendocrinology*, *35*, 209–214. http://dx.doi.org/10.1016/j.psyneuen.2009.06.006

Stalder, T., Hucklebridge, F., Evans, P., & Clow, A. (2009). Use of a single case study design to examine state variation in the cortisol awakening response: Relationship with time of awakening. *Psychoneuroendocrinology*, *34*, 607–614. http://dx.doi.org/10.1016/j.psyneuen.2008.10.023

Sudsuang, R., Chentanez, V., & Veluvan, K. (1991). Effect of Buddhist meditation on serum cortisol and total protein levels, blood pressure, pulse rate, lung volume and

reaction time. *Physiology & Behavior*, *50*, 543–548. http://dx.doi.org/10.1016/0031-9384(91)90543-W

Szanton, S. L., Gill, J. M., & Allen, J. K. (2005). Allostatic load: A mechanism of socioeconomic health disparities? *Biological Research for Nursing*, *7*, 7–15. http://dx.doi.org/10.1177/1099800405278216

Teicher, M. H., Anderson, C. M., & Polcari, A. (2012). Childhood maltreatment is associated with reduced volume in the hippocampal subfields CA3, dentate gyrus, and subiculum. *Proceedings of the National Academy of Sciences*, *109*, E563–E572. http://dx.doi.org/10.1073/pnas.1115396109

Thayer, Z. M., & Kuzawa, C. W. (2015). Ethnic discrimination predicts poor self-rated health and cortisol in pregnancy: Insights from New Zealand. *Social Science & Medicine*, *128*, 36–42. http://dx.doi.org/10.1016/j.socscimed.2015.01.003

Thomas, K. S., Bardwell, W. A., Ancoli-Israel, S., & Dimsdale, J. E. (2006). The toll of ethnic discrimination on sleep architecture and fatigue. *Health Psychology*, *25*, 635–642. http://dx.doi.org/10.1037/0278-6133.25.5.635

Tomfohr, L., Pung, M. A., Edwards, K. M., & Dimsdale, J. E. (2012). Racial differences in sleep architecture: The role of ethnic discrimination. *Biological Psychology*, *89*, 34–38. http://dx.doi.org/10.1016/j.biopsycho.2011.09.002

Turakitwanakan, W., Mekseepralard, C., & Busarakumtragul, P. (2013). Effects of mindfulness meditation on serum cortisol of medical students. *Journal of the Medical Association of Thailand*, *96*, S90–S95.

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin*, *119*, 488–531. http://dx.doi.org/10.1037/0033-2909.119.3.488

Umaña-Taylor, A. J., & Douglass, S. (2017). Developing an ethnic–racial identity intervention from a developmental perspective: Process, content, and implementation of the Identity Project. In N. J. Cabrera & B. Leyendecker (Eds.), *Handbook on positive development of minority children and youth* (pp. 437–453). Cham, Switzerland: Springer International. http://dx.doi.org/10.1007/978-3-319-43645-6_26

Umaña-Taylor, A. J., Douglass, S., Updegraff, K. A., & Marsiglia, F. F. (2018). A small-scale randomized efficacy trial of the Identity Project: Promoting adolescents' ethnic–racial identity exploration and resolution. *Child Development*, *89*, 862–870. http://dx.doi.org/10.1111/cdev.12755

Umaña-Taylor, A. J., Kornienko, O., Douglass Bayless, S., & Updegraff, K. A. (2018). A universal intervention program increases ethnic–racial identity exploration and resolution to predict adolescent psychosocial functioning one year later. *Journal of Youth and Adolescence*, *47*, 1–15. http://dx.doi.org/10.1007/s10964-017-0766-5

Vrijkotte, T. G. M., van Doornen, L. J. P., & de Geus, E. J. C. (2000). Effects of work stress on ambulatory blood pressure, heart rate, and heart rate variability. *Hypertension*, *35*, 880–886. http://dx.doi.org/10.1161/01.HYP.35.4.880

Whitacre, C. C., Cummings, S. O., & Griffin, A. C. (1994). The effects of stress on autoimmune disease. In R. Glaser & J. K. Kliecolt-Glaser (Eds.), *Handbook of human stress and immunity* (pp. 77–100). San Diego, CA: Academic Press. http://dx.doi.org/10.1016/B978-0-12-285960-1.50008-3

World Health Organization. (2019, September). *Hypertension* (Fact sheet). Geneva, Switzerland: Author. Retrieved from https://www.who.int/news-room/fact-sheets/detail/hypertension

Wühl, E., Witte, K., Soergel, M., Mehls, O., & Schaefer, F. (2002). Distribution of 24-h ambulatory blood pressure in children: Normalized reference values and role of body dimensions. *Journal of Hypertension*, *20*, 1995–2007. http://dx.doi.org/10.1097/00004872-200210000-00019

Yang, E. V., & Glaser, R. (2002). Stress-induced immunomodulation and the implications for health. *International Immunopharmacology*, *2*, 315–324. http://dx.doi.org/10.1016/S1567-5769(01)00182-5

Zeiders, K. H., Causadias, J. M., & White, R. M. B. (2018). The health correlates of culture: Examining the association between ethnic–racial identity and diurnal cortisol slopes. *Journal of Adolescent Health*, *62*, 349–351. http://dx.doi.org/10.1016/j.jadohealth.2017.09.020

Zeiders, K. H., Doane, L. D., & Roosa, M. W. (2012). Perceived discrimination and diurnal cortisol: Examining relations among Mexican American adolescents. *Hormones and Behavior*, *61*, 541–548. http://dx.doi.org/10.1016/j.yhbeh.2012.01.018

Zeiders, K. H., Hoyt, L. T., & Adam, E. K. (2014). Associations between self-reported discrimination and diurnal cortisol rhythms among young adults: The moderating role of racial–ethnic minority status. *Psychoneuroendocrinology*, *50*, 280–288. http://dx.doi.org/10.1016/j.psyneuen.2014.08.023