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CHAPTER 34

Early-Life Socioeconomic Status, Emotion Regulation, and the Biological Mechanisms of Disease across the Lifespan

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Health disparities-that is, differences in disease outcomes by socioeconomic status (SES)—remain one of the most pressing public health issues in our society. For example, low-SES individuals-meaning those who are low in education, income, or occupational status—are 2.7 times more likely to have repeated hospitalizations during a 1-year period than high-SES individuals (National Center for Health Statistics, 2010), and 3.5 times more likely to suffer activity limitations due to disease than high-SES individuals (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010). And by age 25, those from the lowest SES group are expected to live 6 fewer years compared to those in the highest SES group (Braveman et al., 2010).

This issue has become such a widespread concern that *Healthy People 2010*, the national health objectives from the U.S. Department of Health and Human Services (2000), lists eliminating health disparities as one of two overarching goals. In addition, the National Institutes of Health (NIH) ranked the issue of health disparities third among its top five priorities (Thomson, Mitchell, & Williams, 2006).

Explanations for why health disparities are so pervasive have been difficult to unearth, because commonly suggested fac-

tors, such as access to health care, have not sufficiently explained existing disparities (Adler, Boyce, Chesney, Folkman, & Syme, 1993). In addition, there is growing consensus that social, and not just biomedical, determinants of disease are important to identify (Dankwa-Mullan et al., 2010). In this chapter, we explore the role that emotion regulation may play in explaining health disparities that emerge early in life. We do this by first providing an overview of links between childhood SES and disease outcomes into adulthood. Second, we examine whether emotion regulation may serve as one explanation for these associations by discussing links between emotion regulation and physiological processes implicated in disease, as well as meditational evidence for emotion regulation strategies in relationships between low-SES and these physiological processes. Third, we discuss the question of moderation—that is, whether certain types of emotion regulation strategies could also serve as protective buffers for a subgroup of those who are low in SES. Throughout this chapter, our premise is that low early life SES fosters certain emotion regulation strategies that emerge during childhood and have implications for physiological processes during childhood and into adulthood; hence, we discuss findings that provide potential

explanations for links between childhood SES and both childhood and adult diseases.

Early Life Environments and Risk for Disease

We begin by discussing epidemiological evidence that low SES increases risk for disease. Low SES in childhood confers greater risk for disease, both throughout childhood and into adulthood. A number of reviews have documented that the effects of low SES start early, and that low SES during childhood is associated with a number of different adverse health outcomes, including greater asthma morbidity, obesity, and injury rates, and poorer self- and parent-reports of health (Chen, Matthews, & Boyce, 2002; Goodman, 1999; Starfield, Riley, Witt, & Robertson, 2002; Starfield, Robertson, & Wiley, 2002).

In addition, the effects of low SES persist into adulthood (Miller, Chen, & Parker, 2011). For example, two reviews of the literature reported that the vast majority of studies found an increased risk of all-cause mortality in individuals who grew up in lowversus high-SES households (Galobardes, Lynch, & Smith, 2004, 2008). Moreover, controlling for adult SES did not eliminate these associations, indicating that something specific to low SES in childhood confers risk for early mortality. Another review documented a heightened risk of cardiovascular disease morbidity associated with low SES in childhood (Galobardes, Shaw, Lawlor, Lynch, & Smith, 2006). Again, associations held up after researchers controlled for adult SES.

These studies are corroborated by quasiexperimental evidence, such as the viral challenge paradigm in humans (Cohen, Doyle, Turner, Alper, & Skoner, 2004), in which a sample of adults was quarantined and exposed to rhinoviruses that cause colds. Participants who came from low-SES households in childhood were significantly more likely to become infected with the rhinovirus and to develop cold symptoms compared to those who came from high childhood SES households. These associations held even after researchers controlled for adult SES, suggesting again that experiencing low SES specifically during the childhood years increases risk for adverse health outcomes later in life.

In summary, a large body of epidemiological evidence demonstrates that low SES during childhood is associated with a variety of poor health outcomes both during childhood and into adulthood. In the next section, we explore the idea that one psychological factor contributing to this association may be difficulties with emotion regulation.

The Role of Emotion Regulation as a Pathway Linking SES and Disease

In this section, we discuss the idea that emotion regulation may serve as one psychological pathway linking SES and disease outcomes. To make this argument, we (1) provide a brief overview of associations between SES and emotion regulation; (2) discuss what types of emotion regulation strategies are relevant to physiological outcomes; (3) discuss physiological markers relevant to disease; (4) provide an overview of previous research on links between emotion regulation and physiological outcomes; and (5) describe studies that have tested emotion regulation as a mediator of SES and physiology relationships.

SES and Emotion Regulation

Emotion regulation refers to strategies to increase, decrease, or maintain emotional responses (Gross, 2001). Gross's process model of emotion regulation states that there are various types of emotion regulation strategies, including antecedent-focused emotion regulation, that is, strategies employed before emotional responses become fully activated, and response-focused emotion regulation, that is, strategies employed after emotion response tendencies have been activated (Gross, 1998).

In the context of SES, extensive evidence documents that low-SES individuals are more prone to experience negative emotions, and in turn that these negative emotions are detrimental for health (for a review, see Gallo & Matthews, 2003; but note that the evidence of negative emotions actually serving as a mediator of the SES-health relationship is mixed; Matthews & Gallo, 2011). Nonetheless, in this context, effective

emotion regulation strategies should reduce experiences of negative emotions.

Implications of Emotion Regulation Strategies for Physiological Responses

Gross (1998) has documented that efforts that fall under antecedent-focused emotion regulation, such as reappraisal, have fewer physiological costs than response-focused emotion regulation strategies, such as suppression. Hence, in the next sections, we focus on the role that antecedent-focused emotion regulation plays in linking SES to physiological and health outcomes.

Antecedent-focused emotion regulation involves strategies such as reappraisal—that is, reevaluating a stressful situation in a way that seeks to reduce its emotional impact. It can also include strategies such as situation selection, situation modification, and attention deployment; together with reappraisal, all of these strategies occur temporally before emotional responses are generated and can alter behavioral, emotional, and physiological response tendencies. Reappraisal is the strategy that has been studied most frequently with respect to affective, cognitive, and social consequences (Gross, 2001), so in the section below we focus below largely on links between reappraisal and physiological processes implicated in disease.

Relevant Physiological Markers for Disease

Conceptualizing pathways to disease on the biological end entails consideration of both acute and longer-term physiological responses. Below we provide a brief overview of the types of systems and processes implicated in chronic diseases—with a focus on diseases linked to inflammation, such as cardiovascular disease and asthma, so that readers will be familiar with the outcomes we present later on in studies of emotion regulation and physiological processes. In this section, we focus on physiological systems that are capable of being altered by psychosocial factors (e.g., stress) and hence could be plausibly linked to variables such as emotion regulation.

Acutely the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic

nervous system (SNS) become activated with many psychosocial stressors, releasing hormones such as cortisol, epinephrine, and norepinephrine (Cannon, 1932; Kemeny, 2003). These hormones bind to receptors located on a variety of bodily tissues, exerting effects on the heart, vasculature, and metabolic and immune systems. With respect to acute physiological responses, profiles indicative of lower disease risk include a reduced magnitude of reactivity of these systems and/or a quicker recovery time (quicker return to baseline levels) (Krantz & Manuck, 1984; Linden, Earle, Gerin, & Christenfeld, 1997; Linden, Gerin, & Davidson, 2003; McEwen, 1998; Schwartz et al., 2003). We note that much of the literature reviewed below does not directly measure SNS and HPA activity, but instead focuses on the responses of end organs such as the heart and blood vessels. However, because these organs are influenced by SNS and HPA activity, and are the source of eventual manifestations of cardiovascular disease (CVD), their responses to acute stressors are relevant here.

Over the long term, with excessive and prolonged exposure to the hormones mentioned earlier, the structure and function of tissues and organs are thought to be altered, giving rise to pathogenic processes that drive CVD, such as obesity, insulin resistance, systemic inflammation, high blood pressure, endothelial dysfunction, and platelet activation (Brotman, Golden, & Wittstein, 2007; Everson-Rose & Lewis, 2005; Rozanski, Blumenthal, Davidson, Saab, & Kubzansky, 2005). Hence, we also review links between emotion regulation strategies and these longer-term mechanisms.

Longer-term cumulative physiological risk has sometimes been encapsulated in concepts such as allostatic load (McEwen, 1998), which is defined as instances when individuals experience stressors repeatedly and have more frequent activation of physiological systems over time; or when physiological systems do not show adaptation of responses after repeated stressors; or when shutdown mechanisms are delayed or insufficient, leading to prolonged physiological responses over time. The strain on these allostatic systems over years may eventually cause a breakdown of these systems that ultimately leads to disease. Empirically, high levels of obesity, insulin resistance, systemic

inflammation, blood pressure, endothelial dysfunction, and platelet activation all predict CVD morbidity and mortality (Danesh et al., 2005; Guh et al., 2009; Lindmark, Diderholm, Wallentin, & Siegbahn, 2001; Ridker, Hennedens, Buring, & Rifai, 2000; Vasan et al., 2001; Yeboah et al., 2009). In addition, the accumulation of these characteristics, in constellations such as allostatic load or metabolic syndrome, predicts even more strongly an increased risk of CVD later in life (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; Lakka et al., 2002; Morrison, Friedman, & Gray-McGuire, 2007; Ridker, Buring, Cook, & Rifai, 2003; National Cholesterol Education Program, 2002).

Emotion Regulation and Physiological and Disease Outcomes

We focus here on links specifically between the emotion regulation strategy of reappraisal and physiological responses, given the existing evidence with respect to this strategy. Physiologically, reappraisals reduce cardiovascular reactivity to acute stressors. For example, lower reappraisals of threat have been associated with reduced blood pressure reactivity during acute stressors in both children and adults (El Sheikh & Harger, 2001; Maier, Waldstein, & Synowski, 2003), and lower ambulatory blood pressure during daily life social interactions (Chen, Matthews, & Zhou, 2007). Similarly, individuals high in the ability to reappraise stressful situations show reduced vascular reactivity during acutely stressful tasks (Mauss, Cook, Chang, & Gross, 2007), and lower blood pressure and cortisol responses to an acute stressor (Salovey, Stroud, Woolery, & Epel, 2002). Experimental evidence shows that interventions aimed at changing appraisals in patient populations produce increases in benefit finding, as well as decreases in serum cortisol levels from pre- to postintervention (Cruess et al., 2000). Finally, consistent with the idea that underlying positive beliefs about others shape reappraisals and physiological responses, those who believe that the world is fair (high in just world beliefs) reappraise an acute stressor as less threatening and show less vascular reactivity to the stressor (Tomaka & Blascovich, 1994).

Emotion regulation also mitigates longerterm pathogenic processes implicated in CVD. For example, better emotion regulation abilities are linked to lower allostatic load, including higher high-density lipoprotein (HDL) cholesterol, lower triglycerides, and lower basal systolic blood pressure (Kinnunen, Kokkonen, Kaprio, & Pulkkinen, 2005). Similarly, reappraisals have been linked to longer-term markers of immune processes. For example, HIVpositive individuals who reported finding benefit after experiencing a major negative life event showed slower declines in cluster of differentiation 4 (CD4) T cell levels over 2-3 years (indicating a slower progression to the diagnosis of AIDS) (Bower, Kemeny, Taylor, & Fahey, 1998). Finally, functional indicators of poor emotion regulation, such as the experience of high levels of depression and anger, have been associated with higher levels of systemic inflammatory markers that are implicated in CVD, such as interleukin-6 (IL-6) and C-reactive protein (CRP) (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Miller, Maletic, & Raison, 2009).

Finally, emotion regulation strategies can also alter clinical disease outcomes. Reappraisals such as finding benefit after a lifethreatening event predicts a lower likelihood of having a future heart attack (Affleck, Tennen, Croog, & Levine, 1987). Conversely, the experience of high levels of negative affect (arguably an indicator of inadequate emotion regulation) has robust associations with CVD-related outcomes (Brosschot, Gerin, & Thayer, 2006; Everson-Rose & Lewis, 2005; Krantz & McCeney, 2002; Kubzansky, Kawachi, Weiss, & Sparrow, 1998). In addition, how effectively one can manage emotions (emotional intelligence) is linked to better general indicators of physical health (better self-reported health and fewer illnesses; Goldman, Kraemer, & Salovey, 1996; Schutte, Malouff, Thorsteinsson, Bhullar, & Rooke, 2007). Finally, experimental data suggest that interventions to help individuals process negative emotions effectively, such as through written disclosure, produce fewer symptoms and health center visits in healthy adults, and improve disease indicators in patient populations (Smyth, Stone, Hurewitz, & Kaell, 1999; Smyth, 1998).

Emotion Regulation as a Mediator

The previously discussed literature links emotion regulation strategies to physiological, endocrine, and immune processes implicated in chronic diseases such as CVD. But is there any evidence that emotion regulation strategies actually mediate the relationship between SES and these biological processes? In a series of studies, our research group has shown evidence for such mediation by focusing on how children and adolescents reappraise stressful life situations.

In healthy adolescents, we have shown that lower SES is associated with greater cardiovascular reactivity to acute laboratory stressors. We further showed that low-SES adolescents were less likely to reappraise ambiguous life situations (e.g., shopping with an overly attentive sales clerk nearby) in benign ways. Finally, we documented that reappraisals of threat statistically mediated the relationship between low SES and heightened cardiovascular reactivity (Chen, Langer, Raphaelson, & Matthews, 2004).

In patient populations, we have documented similar patterns with diseaserelevant markers. For example, in pediatric patients with asthma, we have demonstrated that low SES is associated with greater asthma inflammation (e.g., greater production of cytokines relevant to asthma, higher eosinophil counts). We further demonstrated that low SES is associated with being less likely to reappraise ambiguous life situations in benign ways in this patient population. Finally, we documented that reappraisals of threat statistically mediated the relationship between low SES and heightened asthma inflammation (Chen, Fisher, Bacharier, & Strunk, 2003; Chen et al., 2006).

We further documented that effects of low SES can be seen at the genomic level. Low SES children with asthma showed indications of increased activity of proinflammatory gene networks, and these associations between low SES and gene expression patterns were no longer significant once reappraisals of threat during ambiguous life situations were statistically controlled (Chen et al., 2009).

Taken together, this set of studies illustrates how low-SES individuals are, on average, less able to engage in reappraisals of life situations effectively. Furthermore, reappraisals of threat form one pathway explain-

ing why low-SES children show heightened inflammatory and cardiovascular responses. In turn, these inflammatory and cardiovascular profiles are predictive of later disease.

The Role of Emotion Regulation as Buffer

In the previous section we discussed how emotion regulation strategies serve as one psychological mediator explaining links between low SES and detrimental profiles of physiological processes relevant to disease. In this section, we turn to the question of whether emotion regulation could also serve as a moderator of SES and health outcomes. Despite the robust associations between low SES and disease, there remains a subset of individuals that displays physiologically healthy profiles despite living under adversity. What can explain this group of individuals? That is, what factors might naturally protect individuals who grow up in low-SES environments from the physiological toll and accumulation of health problems typically exacted by these environments? Our research group has articulated a theory about the psychological characteristics that may be specifically beneficial to low-SES individuals (Chen & Miller, 2012). Emotion regulation plays a key role in this theory; hence we discuss its role as a buffer for low-SES individuals in this chapter. In this section, we first provide an outline of the shiftand-persist theory; then we describe the empirical evidence in support of this theory.

Shift and Persist

The theory begins with the notion that a lifetime of facing constraints with limited options leads those living in a low-SES context to place value on the ability to adjust in response to stressors through emotion regulation strategies such as reappraisals (shifting). At the same time, in this context, successful adaptation entails enduring adversity with strength by finding meaning in difficult situations and maintaining optimism in the face of adversity (persisting). We proposed that this combination of approaches to dealing with adversity reduces physiological responses to stressful situations acutely, specifically among those who are low in SES, and over the long term mitigates the progression of pathogenic processes leading to chronic diseases such as CVD (Chen & Miller, 2012).

Hence, one of the key components of this beneficial psychological profile centers around the ability to regulate one's emotions through reappraisal strategies. Because low-SES individuals on average have fewer opportunities to select or modify their life situations (alternative forms of emotion regulation; Gross, 1998, 2001), reappraisals represent a realistic approach to emotion regulation in this group. That is, given the myriad day-to-day, largely uncontrollable stressors experienced by many low-SES individuals, in many instances their best option may be to control the one thing they can—the self rather than engage in what may turn out to be futile attempts to control their environment. By controlling the self, they engage in emotion regulation strategies in which they accept that a stressor has occurred and try to change the effect that stressor has on them. They do this by reappraising the meaning of an event, so that the implications for their lives become less negative. And they adjust their emotional reactions, so that the event evokes less distress in them. As they come to see events as having less serious implications and being less upsetting, the physiological responses they elicit are mitigated. Hence, we propose that low-SES individuals uphold as an ideal the goal of utilizing emotion regulation strategies related to reappraisals when dealing with stress. The ability to do this successfully comprises the "shift" part of our shift-and-persist model.

Shifting is hypothesized to be necessary but not sufficient for buffering low-SES individuals from stressors. In addition to shifting, we hypothesize that it will be important to persist-that is, to endure adversity with strength by finding meaning in life and maintaining optimism about the future. Finding meaning allows individuals to understand adversity and to grow from it. Optimism allows individuals to maintain hope about the future, and can be essentially thought of as reappraising the future (as opposed to shifting, which entails reappraisals of events that have already happened). Thus, reappraisals are an important component of both shifting and persisting.

We further postulate that there is something important about the combination—that is, it is not sufficient to be able to engage

in emotion regulation to deal with current adversities; one also needs also to find broader meaning in life and be able to reappraise the future. Hence the label that we use, "shift and persist," is intended to connote the fact that it is this combination of characteristics that will be beneficial to low-SES individuals with respect to their health. In the next section, we discuss evidence supporting the notion that when low-SES individual engage in shift and persist, there are benefits to the physiological mechanisms that underlie disease.

Empirical Evidence for Shift and Persist

In two studies from our research group, we have documented the benefits of shift and persist specifically for low-SES individuals. In the first study, we assessed childhood SES in a national sample of adults. We measured cumulative physiological risk via allostatic load, based on 24 different measures across seven physiological systems. Shift and persist was measured using questionnaires probing reappraisal-related coping styles (shift) and future orientation (persist). We found a three-way interaction between childhood SES, shift, and persist in predicting allostatic load in this sample. Breaking down this three-way interaction revealed that there was a significant two-way interaction between shift and persist in those from low childhood SES backgrounds, but no two-way interaction of shift and persist among those from high childhood SES backgrounds. The two-way interaction revealed that those participants with low childhood SES backgrounds who were high on both shifting and persisting had the lowest allostatic load. In contrast, the combination of shift and persist did not predict allostatic load among those from high-SES childhood backgrounds (Chen, Miller, Lachman, Gruenewald, & Seeman, 2012).

In a second study, using a clinical sample, we investigated the effects of shift and persist among children diagnosed with asthma, using questionnaires that tapped both reappraisal styles of coping (for shifting) and optimism about the future (for persisting). Among those low in SES, the higher their shift-and-persist scores, the lower their asthma inflammation. Also, among low-SES children, higher shift-and-persist scores pro-

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spectively predicted less functional impairment (fewer school absences, less rescue inhaler use) 6 months later, when we controlled for baseline levels. In fact, low-SES children who scored high on shift and persist had inflammatory and clinical profiles more similar to high-SES children with asthma than to low-SES children who scored low in shift and persist. Shift and persist was not related to inflammatory or clinical profiles in high-SES children with asthma (Chen et al., 2011).

In summary, we find that low-SES individuals who are able to engage in emotion regulation strategies involving reappraisals, in combination with being optimistic and future oriented, are the ones who show the most beneficial physiological profiles and the least clinical disease impairment. Shift and persist is only beneficial to those who are low in SES, not to those who are high in SES, suggesting that there are contextspecific determinants of what types of strategies will be beneficial physiologically for whom. In particular, for low-SES individuals, engaging in reappraisals and focusing on the future when encountering current daily stressors that are largely uncontrollable may be beneficial. In contrast, for those who are high in SES, proactive attempts to eliminate or mitigate stressful situations may be a more beneficial approach given the greater resources these individuals tend to have.

In addition, it is important to note that the combination of shift and persist is critical to physiological profiles among low-SES individuals. That is, neither shifting nor persisting alone was predictive of physiological outcomes; rather, it is only when individuals combine shifting and persisting that one sees physiological benefits. This suggests that emotion regulation strategies of reappraisals on their own are not sufficient; rather, they need to be combined with a focus on the future that emphasizes optimism and meaning in order to derive physiological benefits among those who are low in SES.

Conclusions

In summary, health disparities are a pressing issue in our society, and researchers have been working to understand what factors account for such striking differences in health outcomes across the SES gradient. One possibility we suggest here is that on the psychological end, low-SES children may experience greater difficulties with emotion regulation. In particular, low-SES children may find themselves less able to reappraise stressful situations in positive ways. In turn this leads them to be more likely to experience negative emotions and physiological costs, both acutely and cumulatively, that may contribute to risk for disease over the long term. We note, however, that much of this work is correlational, and cannot be used to draw conclusions about causality.

We also document that emotion regulation serves an important function in terms of buffering low-SES children from detrimental physiological profiles. That is, those low-SES children who are able to engage in shift and persist (utilizing effective emotion regulation strategies, such as reappraisals in combination with persisting with hopes and finding meaning with respect to one's future exhibit physiological profiles that are more similar to profiles of high-SES children than to those of low-SES children who do not engage in shift and persist. These children also showed less clinical disease impairment compared to low-SES children who did not engage in shift and persist. These findings suggest that shift and persist serves as a natural protective factor in low- but not high-SES children.

Emotion regulation strategies are an important factor to consider when investigating individual-level psychological mechanisms underlying SES disparities in health. These strategies provide an interface between children and their broader social environments, and play an important role in shaping hormonal and inflammatory responses to stress. In turn, these acute responses appear to have longer-term implications for the pathogenic processes that underlie chronic diseases across the lifespan.

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