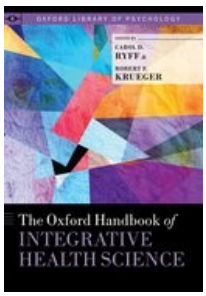

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CHAPTER

31 Socioeconomic Status and Health-Related Biology: Links Between Socioeconomic Disadvantage, Psychological Factors, and HPA Activity in MIDUS

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Abstract

Understanding causal pathways that explain socioeconomic-related health disparities is a long-standing challenge in health research. This chapter reviews research on links between socioeconomic status, ecology, and health, focusing on psychosocial factors that may influence these associations. The Socioeconomic Gradient Bow-Tie Model is introduced to connect socioeconomic status (SES) and SES-related physical and social environments to health. The activity of the hypothalamic–pituitary–adrenal (HPA) axis is highlighted as a key biological pathway through which psychosocial factors may influence the links between SES and health; these ideas are illustrated with findings from the Midlife in the United States study. Attention is given to those findings that showcase psychosocial factors that may help low-SES individuals "escape" the biological risks associated with socioeconomic disadvantage. The chapter concludes with a discussion of future research directions on the link between SES and health-related biology beyond the activity of the HPA axis.

Keywords: health, socioeconomic status, ecology, psychosocial factors, Socioeconomic Gradient Bow-Tie Model, hypothalamic-pituitary-adrenal axis, biological risks, health-related biology

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Introduction

Health disparities research is an intricate endeavor. Even in the presence of robust phenomena, such as the positive and monotonic relationship between socioeconomic status and health (i.e., socioeconomic gradient) (Adler et al., 1994), understanding the underlying multifactorial causal pathways and effects is complicated. Helpful in this endeavor is the formulation of conceptual frameworks that dynamically integrate relevant variables. A first section reviews prior research on socioeconomic status, ecology, and health. Next, we introduce the Socioeconomic Gradient Bow-Tie (SGBT) Model, a conceptual model that connects socioeconomic status (SES) and SES-related physical and social environments to health via intertwined biopsychological processes taking place at the individual level. This model, with particular emphasis on the endocrine intermediaries, is then illustrated with findings, mostly from the Midlife in the United States (MIDUS) study, that focus on the relationship between SES and diurnal activation on the hypothalamic–pituitary–adrenal (HPA) axis. The description of these findings highlights the strengths of the MIDUS study, which offers a rich set of data to test (a) the potential moderating/mediating role played by psychosocial factors for the SES-HPA activity link and (b) the hypothesized causal chain linking SES to clinical outcomes (e.g., onset medical morbidity, self-reported physical symptoms) via psychosocial factors and activity of the HPA axis. Because additional waves of data will ↳ be available from MIDUS, we conclude with a discussion of the directions for future research on the link between SES and health-related biology beyond the activity of the HPA axis.

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Prior Research on Socioeconomic Status, Ecology, and Health

Socioeconomic status is a multifaceted, multilevel, and dynamic construct (Braveman et al., 2005). SES is multifaceted because it refers to access to knowledge (e.g., education) and to financial resources (e.g., wealth); multilevel because it can characterize individuals or entire sections of society; and dynamic because it has the potential to change over time. Common social indicators of SES are education, income, and occupational status, which reflect individuals' social standing within a given hierarchy. Of these, income and wealth are also referred to as economic indicators that reflect the individual's ability to access resources. Economic indicators can be assessed not only at the individual level, but also at the family (e.g., household income) or neighborhood (e.g., neighborhood census tracks) level. These indicators are often referred to as objective and are usually distinct from subjective indexes of SES, which more closely reflect one's perception of their relative standing within a social hierarchy.

A person's socioeconomic position does not exist in a vacuum; rather, it is embedded in personal ecologies that continuously influence one another (Bronfenbenner, 1979). Both the physical and social environments in which individuals develop are affected by one's SES and can serve as pathways through which SES is linked to health outcomes. Although physical and social ecology often covary with SES and are dependent on each other, here we focus on social ecology and its influence on psychological, physiological, and behavioral processes with implications for health.

Social ecology refers to the social interactions and relationships that an individual has and develops with proximal (family, peer group, school, religious group, workplace) and distal (neighborhood and community level) relationship ties. Social ecology also includes the social processes experienced in the context of such social interactions and relationships (e.g., discrimination, conflict, ostracism, and community integration). One of the current challenges for research in health disparities is to clarify how these various social ecological factors may mediate the influence of SES on health and well-being.

A long tradition of research in social sciences has demonstrated that social ecological factors stemming from either the individual or broad social structures can have an important impact on health and longevity

(Holt-Lunstad, Smith, & Layton, 2010; House, Landis, & Umberson, 1988). For example, exposure to community violence, which is more commonly experienced in low-SES neighborhoods, has been associated with risk of coronary heart disease (Sundquist et al., 2006); pregnancy complications (Zapata, Rebolledo, Atalah, Newman, & King, 1992); and increased risk of asthma (Sternthal, Jun, Earls, & Wright, 2010). More proximally, conflicting, hostile, and neglectful family relationships—characteristics of so-called risky families (Repetti, Taylor, & Seeman, 2002)—constitute chronic social stressors that can have detrimental effects on physical health. Finally, examples of social processes that can be experienced at both the individual and more distal social levels and that might be implicated in connecting SES to health are discrimination and social isolation. For example, working with a sample of predominantly rural white youth, Fuller-Rowell and colleagues (Fuller-Rowell, Evans, & Ong, 2012) found that discrimination on the basis of social status partially explained the link between poverty and cumulative biological risk (i.e., allostatic load). Similarly, social isolation and loneliness, the measurable conditions of having a shrunken social network (i.e., few, infrequent, or unsatisfactory social contacts) have been found to increase with decreasing education and income (Hawkley et al., 2008) and to predict medical morbidity and mortality (Luo, Hawkley, Waite, & Cacioppo, 2012).

Despite the evidence that social ecological factors mediate the influence of SES on health, SES and social ecology do not completely overlap, as some social factors are independent of SES or have different implications for the link between SES and health depending on broader cultural factors. Therefore, it is possible that social factors can act not only as mediators, but also as moderators of the influence of SES on health. In the MIDUS study this has been illustrated through several findings that indicate that various aspects of social relationships and psychological experiences interact with SES in predicting biological and health outcomes. For example, Morozink and colleagues (2010) have shown that higher levels of purpose in life reduce the detrimental effect of low education on the pro-inflammatory marker interleukin 6 (IL-6). On the other hand, Park and colleagues (2013) have found that the expression of anger indicates frustration and
p. 433 has a negative association with SES ↳ in the United States, with the reverse being true in Japan, where the expression of anger is connected to high social status and privilege. Therefore, this study demonstrates how a distant aspect of social ecology such as culture can moderate the influence of SES on health-relevant negative emotions such as anger, leading to potentially different implications for the influence of emotions on the SES–health link among these populations. Finally, Fuller-Rowell and colleagues (2012) have also shown that experiences of discrimination can have a different impact on the daily cortisol activity of high- versus low-SES African Americans, with low-SES individuals actually showing healthier cortisol activity for higher levels of discrimination, given that awareness of racism may allow this group to protect themselves from negative self-attributions.

To summarize, SES is a multifaceted, multilevel, and dynamic construct embedded in a person's physical and social ecology. SES covaries with these ecological dimensions, which can be thought as the first line of mediators and moderators of the socioeconomic gradient. Ultimately, however, intertwined biological and psychological processes taking place at the individual level mediate the effects of SES and the surrounding ecology on health. In the next section, we briefly describe how these biopsychological mechanisms synthesize incoming inputs and give rise to heterogeneous health outcomes.

Psychological Pathways

Psychological factors represent more proximal mediators and moderators of the SES–health outcomes link. They can encompass affective pathways (e.g., negative affectivity, emotional reactivity, emotional regulation); cognitive pathways (e.g., cognitive coping, stressor appraisal, cognitive abilities); and behavioral pathways (e.g., behavioral coping, health behaviors). Various theories have proposed how these three factors relate to each other. For example, according to the transactional model of stress and coping proposed by Lazarus and Folkman (1984), the impact of environmental stressors on a person's well-being and functional status depends on how he or she appraises and copes with the situation. Events appraised as taxing and threatening lead to emotional responses that motivate the individual to marshal cognitive and behavioral energies necessary to cope with the situation at hand.

The relationship between cognitive appraisal and coping is bidirectional; for example, events appraised as stressful can lead to coping strategies that alter the person–environment relationship, engendering new appraisal (i.e., reappraisal). For example, moving to a neighborhood, which is a common occurrence among low-SES individuals, can be appraised as a threat that exceeds personal resources. In response to the emotional distress caused by this evaluation, some individuals might adopt coping strategies (e.g., denial) that can lead to further negative affect or coping strategies (e.g., positive reappraisal) that reduce negative affect.

The idea of bidirectional relationships between psychological pathways is also reported in the Reserve-Capacity Model (Gallo & Matthews, 2003), a seminal theoretical framework in health psychology that emphasizes the role of psychological factors as modulators of the socioeconomic gradient. Briefly, the Reserve-Capacity Model posits that threatening and harmful events are more severe and occur with more frequency in low-SES environments, causing low-SES individuals to experience more negative cognitive–emotional states and taking a toll on their psychosocial stress-buffering resources. Depleted resources and negative cognitive–emotional experiences reinforce each other and ultimately lead to deleterious health outcomes (i.e., clinical end points) via alterations of biobehavioral intermediate pathways, including adoption of dysfunctional health behaviors and dysregulation of various biological systems.

Empirical evidence in support of the mediating role played by psychological pathways emerges from the large literature on SES disparities in unhealthy behaviors (e.g., sedentary lifestyle, poor diet, substance abuse) (Pampel, Krueger, & Denney, 2010) and the growing literature showing that negative affect, perceived stress, and psychological resources (e.g., self-esteem, perceived control, optimism) contribute to explain the association between social status and health-related biological pathways (e.g., Chiang et al., 2015), surrogate end points, and clinical end points (for a review, seeMatthews, Gallo, & Taylor, 2010). Existing literature also bears the hypothesis that psychological pathways can change the strength and directionality of the SES–health link, thus acting as moderators (Chen & Miller, 2013).

Several studies in the MIDUS project illustrate this point quite clearly. For example, using data from the first cohort of MIDUS participants, Lachman and Weaver (1998) found that perceived control and SES interacted in predicting self-reported health (i.e., physical symptoms), such that individuals endorsing high levels of
p. 434 perceived ↳ control reported lower physical symptoms regardless of their SES background. Also looking at data from the first MIDUS cohort, Turiano and colleagues (Turiano, Chapman, Agrigoroaei, Infurna, & Lachman, 2014) found that greater perceived control safeguarded low-SES individuals from SES-driven mortality risk. Last, Chen and Miller (2012) found support for the idea that a coping strategy characterized by both high levels of positive reappraisal (i.e., shift) and high levels of optimism and sense of purpose (i.e., persist) may confer health benefits among the most disadvantaged.

Biological Pathways

Social ecological factors can influence the association between SES and health through several biological pathways involving the central nervous system, neuroendocrine circuits, and intercellular mechanisms. Among these factors, social processes and their corresponding cognitive and affective states have gained particular attention by research with regard to their ability to modulate physical regulatory influences. For example, engaging in a hostile verbal exchange, thinking of an upcoming stressful social interaction, or vicariously living pain expressed by a loved one can activate physiological pathways (e.g., neuroendocrine circuitries), with downstream effects on cellular processes.

Direct empirical support for this interaction emerges, for example, from psychoneuroimmunological studies showing the role of psychosocial factors in modulating immune responses to infectious agents, such as work on Epstein–Barr virus reactivation (Fagundes et al., 2012, 2014) and studies on lipopolysaccharide (Chen, McLean, & Miller, 2015) and rhinovirus (Cohen et al., 2012) challenge.

A good example of this process from the MIDUS study indicates that a low childhood SES individual who reported high levels of maternal nurturance during the childhood years indicated lower levels of metabolic risk in comparison to low childhood SES individuals who reported lower maternal nurturance (Miller et al., 2011). More broadly, this hypothesis is indirectly buttressed by studies on the negative impact of psychological stress and disease progression (S. Cohen, Janicki-Deverts, & Miller, 2007).

While emerging evidence suggests that social ecological factors are also associated with structural and functional characteristics in several brain regions, such as the amygdala or the prefrontal cortex (Kim et al., 2013; Noble, Houston, Kan, & Sowell, 2012), as well as with cellular mechanisms at the gene expression level (Miller et al., 2009; Powell et al., 2013; Stringhini et al., 2015), one of the most well-researched biological pathways remains the modulation of the HPA.

The HPA axis is a hormone response system that plays a critical role in coordinating physiological adaptation to challenges (McEwen, 2007). The primary function of the HPA response is to mobilize the organism in times of threat and to supply the metabolic resources that are necessary in order to fight or escape from danger. Although the HPA axis responds to a wide array of threats, a large body of research shows that this system is particularly sensitive to psychological stressors (McEwen, 2007; Miller, Chen, & Zhou, 2007). As a result, the HPA axis has attracted a lot of attention from researchers interested in understanding the biological mechanisms through which psychological stress can influence disease, and it is considered to be one of the most prominent pathways through which mind–body connections are materialized.

The HPA response starts with the activation of the paraventricular nucleus of the hypothalamus, which then gives rise to a cascade of biological intermediaries that culminate in the release of the glucocorticoid hormone cortisol by the adrenal cortex. Once cortisol levels reach a certain threshold in the circulation, they initiate a negative-feedback loop by communicating with the hippocampus, which then sends signals to the hypothalamus in order to terminate the HPA response (Tsigos & Chrousos, 2002). Given its role in the cascade, cortisol release is considered a reliable marker of HPA activity (and therefore an indicator of biological responses to psychological stress); however, its functions go way beyond the HPA axis and support many other systems in the body. For example, cortisol is involved in the consolidation of memory and learning; it regulates the storage of glucose and the mobilization of fat and amino acids from cells and orchestrates the inflammatory response and other functions of the immune system (Sapolsky, Romero, & Munck, 2000). Therefore, dysregulation of cortisol activity could have implications for a wide array of physiological functions.

Besides being released in response to stress, cortisol activity also follows a diurnal rhythm characterized by high morning levels that peak approximately 30 minutes after awakening and have a steady decline

p. 435 throughout the day, which reaches its lowest point shortly before bedtime (Tsigos & ↳ Chrousos, 2002). The sharp morning increase is known as the cortisol awakening response (CAR), and the rate of decline throughout the day is known as the cortisol slope. Both of these parameters can be easily estimated by assessing cortisol concentrations in saliva collected at several time points throughout the day. Another way of quantifying daily cortisol output is to calculate the total cortisol concentration over the day (area under the curve); however, the relation of this parameter to health outcomes is not very clear (Clements, 2013).

On the other hand, a large body of research shows that many negative health outcomes are observed when the rate of cortisol decline does not follow the expected circadian rhythm (Miller et al., 2007). More specifically, flatter diurnal cortisol slopes (which indicate abnormal HPA activity given that cortisol levels continue to remain high as the day advances) have been linked, among others, to onset of Type 2 diabetes (Hackett, Kivimäki, Kumari, & Steptoe, 2016); coronary artery calcification (K. Matthews, Schwartz, Cohen, & Seeman, 2006); and breast and lung cancer survival rates (Sephton et al., 2013; Spiegel, Giese-Davis, Taylor, & Kraemer, 2006). Flatter cortisol slopes have also been associated with increased mortality risk in large samples of older adults (i.e., Kumari, Shipley, Stafford, & Kivimaki, 2011) and are believed to mark a trajectory of future health risk among children growing up in adversity (Miller, Chen, & Parker, 2011).

Considering that many individuals of low SES are chronically exposed to multiple sources of stress, it is not surprising that research to date has uncovered robust associations between low SES and unhealthy cortisol profiles in several populations. The relationship between SES and cortisol secretion has been documented in several large studies involving community-dwelling adults. Although there are differences in results obtained for morning cortisol, the general finding is that low-SES individuals are more likely to show slower cortisol declines throughout the day (i.e., "unhealthy," flatter slopes).

One of the earliest attempts to investigate the association between SES and diurnal cortisol was by Rosmond and Björntorp (2000), who collected saliva samples from 284 Swedish men during an ordinary workday. SES in this sample was measured through a composite score of educational achievement and occupational grade. The findings revealed that although SES was not related to total cortisol output throughout the day, it was inversely related to the diurnal cortisol slope and to stress-related cortisol secretion. Similar findings also emerged for male participants from a cohort of British civil servants in the Whitehall study, where those who occupied the lowest employment grades showed the "flattest" cortisol curves (Kumari, Chandola, Brunner, & Kivimaki, 2010).

It should be noted that these associations withstand controlling for the influence of several health behaviors, such as smoking or alcohol consumption, although the effect sizes are sometimes attenuated once the contribution of these variables is accounted for (for examples, see S. Cohen, Doyle, & Baum, 2006, and Hajat et al., 2010). This pattern of results strongly suggests that the effect of SES on health-related biological mechanisms is transmitted through both behavioral and psychosocial intermediaries that characterize the ecology of individuals at each step of the SES gradient. Further, these studies provide strong evidence for the unique contribution of SES to cortisol secretion, which often goes above and beyond the influence of these other factors, particularly behavioral ones. For example, when researchers in the CARDIA (Coronary Artery Risk Development in Young Adults) study (S. Cohen et al., 2006) analyzed the data in a subsample that excluded smokers, the association between SES and cortisol slope was still present and became even stronger.

Health Outcomes

Health outcomes are the product of the continuous synthesis of ecological inputs linked to SES and can be distinguished between surrogate end points and clinical end points. *Clinical end points* are defined as a "characteristic or variable that reflects how a patient feels, functions, or survives" (Biomarker Definitions Working Group, 2001, p. 91), while surrogate end points represent biomarkers that have been scientifically proven to predict clinical end points and, for this reason, can substitute for them (Colburn et al., 2001, p. 91). Thus, most surrogate end points are etiologically related to clinical end points (Temple, 1999). An extensive empirical literature has shown a strong monotonic relationship between SES and both surrogate and clinical end points (e.g., Adler & Ostrove, 1999). In MIDUS, lower SES has been associated with higher BMI (Chapman, Fiscella, Duberstein, Coletta, & Kawachi, 2009); lower bone strength (Karlamangla et al., 2013); and greater odds of being diagnosed with prediabetes and diabetes status (Tsenkova, Pudrovska, &
p. 436 Karlamangla, 2014). ↳

The Socioeconomic Gradient Bow-Tie Model and MIDUS

Given that SES is a dynamic construct that shapes health and well-being through often-intertwined psychological and behavioral factors, in our work we have formulated the SGBT Model, a guiding conceptual framework that integrates these pathways and tries to explain how SES-related ecological factors influence health via biopsychological processes taking place at the individual level. The term *bow tie* is commonly used in biology to describe the architecture of complex systems (Csete & Doyle, 2004). The core of bow-tie architectures is the knot, a small set of regulatory mechanisms that synthesizes heterogeneous inputs, giving rise to diverse outputs. We borrow the idea of bow-tie architecture to describe how the organism (the knot) translates influences associated with SES and SES-related environment (inputs) into health outcomes (outputs).

Figure 31.1 depicts the SGBT model. In brief, the model assumes that variations in SES (e.g., education, financial resources, perceived social status) correspond to variations in the physical environment (e.g., exposure to toxicants) and social dynamics (e.g., conflict, ostracism) to which people are likely to be exposed. The organism processes these diverse social and physical inputs via interconnected psychological (e.g., emotional reactivity) and biological (e.g., cortisol reactivity in response to stress) mechanisms. Health outcomes, regarding both surrogate end points (e.g., blood pressure) and clinical end points (e.g., physical symptoms), are the product of this continuous synthesis.

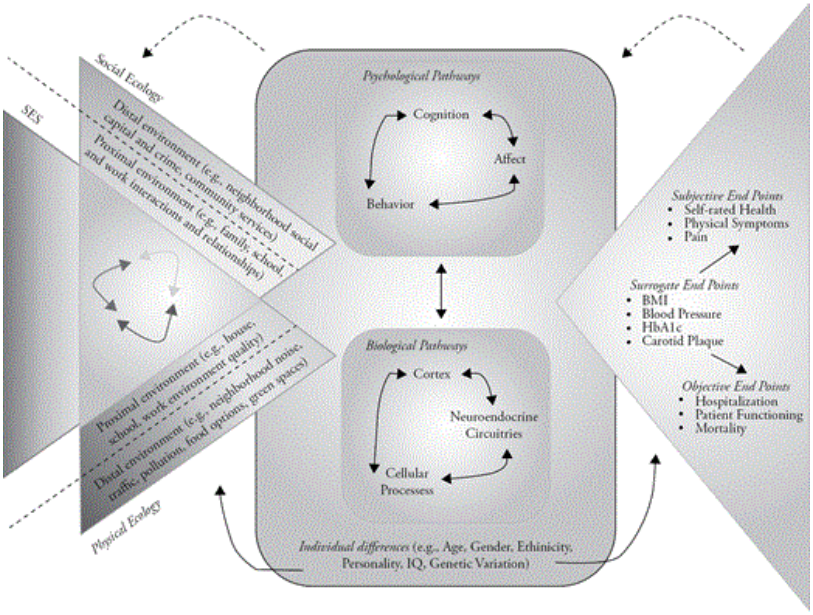

Figure 31.1 The socioeconomic gradient bow-tie (SGBT) model.

One of the strengths of the MIDUS study is that it offers a comprehensive dataset allowing researchers to test aspects of complex theoretical models such as the SGBT model. Originally designed to investigate the factors that promote successful aging, the MIDUS study has become one of the largest interdisciplinary projects on midlife development in the United States, containing detailed psychological assessments and multiple biological markers of health.

The first MIDUS cohort included a nationally representative sample of 7,108 adults between the ages of 25
p. 437 and 75 years (Brim, Ryff, & Kessler, 2004). These participants were assessed ↳ again 9–10 years later, and a subsample of them completed a study of daily experiences over 8 days (the National Study of Daily Experiences [NSDE]), during which they provided saliva samples for daily cortisol assessment on 4 of the 8 days (for more details, see Almeida, Wethington, & Kessler, 2002). Another subsample of participants (the Biomarker project) also participated in an overnight clinic visit to one of three General Clinic Research Centers, where they provided detailed anthropometric measures and blood samples for additional biomarker assessments and also participated in several cognitive challenges.

This design allows researchers to test hypotheses about not only the psychological factors that may connect SES to various clinical end points and health-relevant biological responses at the daily level, but also the longitudinal links between SES, psychological factors, and health throughout midlife. As a result, the MIDUS study provides a unique opportunity to test models that highlight the mediating and moderating role of psychological factors on the relation between SES and health. In our work, we have utilized this opportunity to test various aspects of the SGBT model, with a particular emphasis on the association between social ecological factors and alterations in daily HPA axis functioning.

SES and HPA Axis in the MIDUS Study: Mediators and Moderators

Although the association between SES and HPA activity is a well-established one, more research is needed in order to gain a better understanding of the psychological pathways that underlie this association. Although only a small number of studies have investigated the links between SES and daily cortisol in the MIDUS NSDE project, current work already shows some promise in this direction. First, it should be noted that the negative association between SES and the cortisol slope is also found in MIDUS (Karlamangla et al., 2013). In agreement with previous investigations, MIDUS participants do not show differences based on SES with regard to total cortisol output; however, less educated participants tend to show the typical pattern of flatter slopes.

Surprisingly, not many studies have tested for psychological mediators/moderators with regard to cortisol, but findings on inflammation (a process orchestrated by cortisol activity) show that psychological factors act as intermediaries of the link between SES and health-related biology. For example, Boylan and Ryff (2013) found that anger expression moderated the relation between SES and pro-inflammatory markers such that lower SES individuals who reported higher levels of anger showed higher levels of inflammation than higher SES individuals. Furthermore, Chen and Miller (2012) showed that low-SES individuals who engaged in positive reappraisal of stressors while remaining optimistic about the future displayed lower levels of inflammation in comparison to low-SES individuals who did not adopt this coping pattern.

Based on this work, our group has started to investigate how psychological factors may mediate or moderate the relationship between SES and daily cortisol. One aspect that distinguishes our work from previous work on SES and cortisol is our approach to measure SES as a cumulative indicator of several areas of socioeconomic disadvantage, from income and education to difficulty in paying bills and the availability of money to meet basic needs. In this regard, our approach reflects the main premise of the SGBT model that multiple characteristics of one's ecology tend to accumulate and intertwine in order to affect health.

One of the first tests of this premise was in a study where we investigated how various aspects of physical and social ecology during childhood were linked to daily cortisol activity in adulthood (Zilioli, Slatcher, Chi, Li, Zhao, & Zhao, 2016). In this investigation we created an index of childhood adversity that included several experiences typical of risky family environments, from the experience of physical abuse by family members, to experiences of neglect, and to experiences of problems in school. Although this index did not include any SES indicators, its content covered many characteristics of physical and social ecology that share a strong association with SES.

In testing the mediators of childhood adversity and HPA activity, we focused on overall perception of self-worth in adulthood, as indicated by a measure of self-esteem. Based on the sociometer hypothesis (Leary, Tambor, Terdal, & Downs, 1995), we reasoned that perceptions of low self-esteem would reflect individuals' perceptions of a threat to their current social status and would therefore have the potential to modulate HPA activity. Our findings showed that both childhood adversity and self-esteem were associated with various parameters of cortisol activity. In line with our hypotheses, we found that greater childhood adversity was associated with lower levels of cortisol at awakening and that this link was partially mediated by self-esteem. In addition, we found that greater childhood adversity ↳ was indirectly linked to flatter cortisol slope through lower levels of self-esteem. These findings obtained from MIDUS were also mirrored in a sample of youth in rural China, lending strong support to the idea that psychological factors, particularly those related to the ways by which one perceives one's worth within the context of both intimate and more distal social networks (e.g., self-esteem, depression, perceptions that one's spouse or friends care and value oneself), can constitute important pathways through which early experiences can modulate HPA activity in adulthood.

Another important goal of our work within MIDUS has been to document those cases when experiences of SES disadvantage do not necessarily translate into biological risk. Although a strong focus of the current literature has been to identify the mediating links through which SES is embedded under the skin, a parallel line of work (particularly within MIDUS) has also focused on understanding when and how individuals of low SES are able to "escape" the detrimental health consequences of socioeconomic adversity.

Within this domain, we first focused on investigating the role of subjective well-being as a moderator of the influence of SES on cortisol activity (Zilioli, Imami, & Slatcher, 2015). Previous work has demonstrated that subjective well-being, as indexed by evaluations of life satisfaction, tends to show a small-to-moderate (although consistent) association with SES (Lawless & Lucas, 2011) and is a significant predictor of morbidity and mortality (Siahpush et al., 2008). In the MIDUS survey, life satisfaction is measured by having participants rate the quality of several important domains in their life, such as work, health, and family (Prenda & Lachman, 2001). In our own previous work in MIDUS, we had already found strong evidence that satisfaction within several important domains of one's life, such as the work–family interface (Zilioli, Imami, & Slatcher, 2016) or the ways in which one is perceived by one's partner (Slatcher, Selcuk, & Ong, 2015), was associated with healthier cortisol slopes both cross-sectionally and longitudinally.

Based on these findings, we reasoned that the overall affective evaluation of the quality of one's life on each of these domains could be related to biological functioning as well and could serve as an indicator of social and psychological resources being used by low-SES individuals in order to maintain good health in the face of adversity (Gallo & Matthews, 2003). Our findings revealed that greater life satisfaction was associated with steeper cortisol slopes, and that it served as a protective factor against the negative effects of low-SES on cortisol activity. More specifically, our findings showed that low-SES individuals who reported high levels of life satisfaction had cortisol slopes that were similar to those of high-SES individuals. On the other hand, low-SES individuals who reported low levels of life satisfaction showed the typical pattern of flatter slopes. In addition, low-SES individuals with low levels of life satisfaction also indicated lower levels of morning cortisol. These findings not only provide the first empirical evidence that diurnal cortisol activity may be one of the physiological pathways through which life satisfaction is linked to health effects, but also suggest that perceptions of well-being in areas of one's life other than material resources may buffer the negative consequences associated with low SES.

Theories of resilience emphasize that being able to maintain a sense of purpose and control over one's environment allows individuals to function well in the face of adversity (Luthar, Cicchetti, & Becker, 2000; Seligman, 2008), even when these perceptions are illusory in nature (Taylor, 1989). Based on these ideas, research has already shown that high levels of control and mastery contribute to higher well-being (Lachman & Weaver, 1998) as well as lower levels of allostatic load (Chen, Milller, Lachman, Gruenewald, & Seeman, 2012) among those facing socioeconomic disadvantage. In another investigation in MIDUS, we extended this research by examining if feelings of perceived control would moderate the relationship between SES, daily cortisol activity, and daily experiences of physical symptoms (Zilioli et al., 2017).

Previous work in MIDUS has already shown evidence that individual traits that promote adaptive functioning in the face of life challenges are associated with lower biological risk. For example, Turiano and colleagues (2013) have shown that individuals characterized by high levels of neuroticism and conscientiousness (a trait reflecting higher levels of self-regulation and industriousness) indicated lower levels of the pro-inflammatory marker IL-6, while work by Bogg and Slatcher (2015) has shown that higher levels of conscientiousness were associated with a steeper diurnal cortisol slope. In our results, we did not see a direct association between perceived control and diurnal cortisol activity. Nonetheless, in line with our moderation hypothesis, we found that SES differences in cortisol slope, number of daily physical symptoms,
p. 439 and severity of symptoms were stronger among individuals reporting low levels of perceived ↳ control. Further, our findings also revealed that for individuals reporting lower levels of perceived control, flatter cortisol slopes mediated the link between lower SES and greater number and severity of physical symptoms.

These associations, however, did not emerge for low-SES individuals reporting higher levels of perceived control.

These findings mirror previous results reported by Lachman and Weaver (1998) with regard to psychological well-being, as well as results by Turiano and colleagues (2014) with regard to mortality risk. To our knowledge, however, this is one of the first investigations in the MIDUS study to suggest a plausible biological pathway through which higher levels of perceived control protect low-SES individuals from higher risk of mortality. In addition, we also show evidence, for the first time, of a link between cortisol activity and self-reported health, as well as the contingency of these links on SES.

Together, our studies on the psychological moderators of SES on cortisol activity showcase not only how psychological factors can play a very important role in the relationship between SES and health-related biology, but also how the MIDUS study can provide an ideal framework for testing complex theoretical models on the relationship between SES and health.

Conclusion and Future Directions

The body of work reviewed in this chapter indicates that the association between SES and health is robust, and that SES gradients in health can be found in all segments of society and regardless of the ways in which SES is operationalized. The exciting challenge ahead is to increase our understanding of the psychobiological mechanisms through which this association takes place. As noted at the beginning of this chapter, this can be quite an intricate endeavor. However, although it is difficult to test a theoretical model like ours in its entirety—especially given the multiplicity of pathways involved, the bidirectionality of relationships, the temporal component (emergence of certain pathways), sample sizes, and longitudinal design that are required—research so far has started to showcase several promising pathways through which to move forward. For example, most of the present studies have focused on assessing single outcomes such as cortisol or various indicators of inflammatory activity. With the help of comprehensive projects like MIDUS, future research will be well positioned to investigate how multiple biological pathways may work in an orchestrated fashion in order to give rise to the SES–health gradient. A prime opportunity in this area will be the addition of a new wave of longitudinal data containing salivary cortisol and other biomarkers that index biological risk.

From a psychobiological perspective, one of the most interesting questions that can be investigated with the addition of these new data is the longitudinal association between SES and inflammatory risk through the mediating activity of the HPA axis. Although many studies in MIDUS have established links between SES and inflammatory markers (e.g., Elliot & Chapman, 2016; Friedman & Herd, 2010), none of them has been able to test for the potential intermediary association of cortisol in these links despite the fact that cortisol is responsible for coordinating inflammatory activity in times of challenge and threat. Furthermore, this new line of data will allow researchers to gain better insights on the role of brain mechanisms involved in the regulation of HPA and inflammatory activity and how these mechanisms may change with alterations in SES over time. More broadly speaking, this new wave of data will allow researchers to gain stronger evidence of the impact of cumulative SES adversity on psychobiological functioning and understand the ways in which exposure to macrolevel events, such as the 2008 US recession, may influence those who were already burdened by socioeconomic disadvantage in earlier waves of the MIDUS study.

Advancement in new statistical tools will allow researchers to test some of the complex pathways delineated in these models by assessing multiple mediation as well as moderated mediation in order to test for buffering and intermediary effects at the same time. For example, one underexploited area of research in SES-related health disparities is the investigation of the links between physical and social ecology and the ways in which they may influence each other as well as biological pathways related to health. Previous

research has shown that proximal physical stressors that covary with SES such as house crowding (i.e., persons per room) or poor quality of residential space (e.g., exposure to toxins; Evans & Kantrowitz, 2002) are associated with several important health outcomes. House crowding has been associated with increased cardiovascular risk (Schreier & Chen, 2010; see also Vijayaraghavan et al., 2013), while exposure to toxins, such as lead and polychlorinated biphenyls, has been associated with low birth weight (Baibergenova,
p. 440 Kudyakov, Zdeb, & Carpenter, 2003); neuroendocrine and cardiovascular dysregulation (Gump et al., 2007, ↳ 2009); and volume loss of certain brain areas (Cecil et al., 2008). The alteration of these regulatory systems by aspects of physical ecology could also influence many aspects of social ecology, with important implications for psychological well-being as well as for the way in which psychological and biological pathways influence one another in order to determine health outcomes.

Some of this work is already at the initial stages in MIDUS, with researchers looking at census track data in order to capture aspects of neighborhood disadvantage in an effort to understand racial disparities in health behaviors such as sleep (Fuller-Rowell et al., 2016). These opportunities, however, could be exploited even further by examining specific aspects of the physical ecology of neighborhoods (i.e., proximity to industrial complexes or residing in a highly urbanized or "food desert" area) and the way in which they influence biopsychosocial variables longitudinally.

Finally, the wealth of data that has been accumulated over the years in the MIDUS project will allow researchers to conduct comprehensive reviews on the findings that have emerged up to this point regarding the association between socioeconomic disadvantage and specific biological pathways, such as HPA activity, inflammatory activity, and indicators of autonomic system functioning. We look forward to a new wave of researchers embarking on these exciting new avenues for research and to the emergence of new insights on the SES disparities' challenge in the years to come.

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