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CHAPTER

30 Social Inequalities, Psychological Risk and Resilience, and Health

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Abstract

Epidemiological evidence from the Midlife in the United States and other studies shows robust socioeconomic disparities in mental and physical health outcomes. Considerable heterogeneity exists in health within socioeconomic strata; not all socioeconomically disadvantaged individuals exhibit poor health. Evidence is presented supporting an integrative conceptual framework wherein psychological factors moderate the association between socioeconomic status and health, illuminating unique risk and resilience profiles. Regarding protective factors, distinctions between hedonic well-being and eudaimonic well-being are highlighted. Regarding psychological risk factors, the focus is on the experience and expression of anger. Several pathways through which socioeconomic and psychological factors may affect health, including health behaviors, emotion regulation, and physiological responses to stress are considered. The chapter concludes with directions for future research, including efforts to integrate psychological strengths and risk factors and the need for longitudinal and intervention approaches to address the public health issue of health disparities from a biopsychosocial perspective.

Keywords: [health](#), [socioeconomic status](#), [hedonic well-being](#), [eudaimonic well-being](#), [anger](#), [hostility](#), [health behaviors](#), [emotion stress](#)

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Introduction

The aim of this chapter is to present an integrative model that helps elucidate how individual differences in psychological functioning act as risk and resilience factors in the context of socioeconomic disadvantage, using evidence primarily from the Midlife in the United States (MIDUS) study. We begin by selectively reviewing the extant literature on socioeconomic disparities in health. We also discuss established associations between both positive and negative psychological factors and many health outcomes, as well as the biobehavioral mechanisms underlying such associations. MIDUS has contributed to these literatures extensively, and we separately summarize the evidence that has emerged from this study. However, the primary focus of this chapter is the integration of targeted individual differences in psychological factors with inequalities in health. There is notable heterogeneity in health and disease within socioeconomic strata, especially at the lower end of the distribution. That is, not all individuals of low socioeconomic status (SES) exhibit adverse health outcomes. We posit that individual variation in psychological factors helps to elucidate the differences between those vulnerable and the resilient.

We describe a *mitigation hypothesis*, which poses that several domains of psychological well-being and other psychological resources moderate (i.e., buffer) the association between low SES and poor health. Furthermore, the *exacerbation hypothesis* describes that psychological risk factors, especially anger, moderate (i.e., aggravate) the vulnerability of low-SES individuals, especially when racial and cultural factors are considered. We then turn to avenues for future research, emphasizing the importance of research that considers psychological strengths and risk factors together and employs longitudinal and intervention approaches to validate the constructs. There is a pressing need to link questions of *who* is especially vulnerable and resilient with *how* these profiles of risk and resilience unfold over time and in the context of the normal biology of aging.

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Socioeconomic and Psychosocial Determinants of Health

Emerging Evidence

Inequality is a major contemporary concern, with the reduction in socioeconomic disparities in health emerging as a top priority for public health policy. SES, characterized by one's access to material resources and relative position in the social hierarchy, is a robust predictor of morbidity and mortality. Individuals with relatively low SES have higher rates of most major health conditions (e.g., respiratory, cardiovascular, and metabolic disease) as well as higher mortality rates relative to those who are more socioeconomically advantaged (reviewed in Adler & Rehkopf, 2008; Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010; Marmot, Kogevinas, & Elston, 1987). Further, evidence for a link between socioeconomic context and health exists across the life course, beginning in childhood and adolescence (S. Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Hanson & Chen, 2007; Schreier & Chen, 2013) and continuing in adulthood and old age. Recent mortality data from the Social Security Administration death records revealed a 14.6-year difference in life expectancy between the top and bottom 1% of the income distribution. Moreover, this disparity in life expectancy has *increased* between 2001 and 2014 (Chetty et al., 2016). Importantly, disparities emerge at all levels of SES, not just when comparing the lowest and highest rungs of the socioeconomic ladder. Socioeconomic disparities are evident in mental health outcomes as well, including depression (Everson et al., 2002), anxiety disorders (Miech et al., 1999), and other psychiatric disorders (Dohrenwend et al., 1992; Gallo & Matthews, 2003).

In addition to SES, it is increasingly recognized that individual differences in psychological strengths and risk factors are likewise associated with various health outcomes, including biological risk factors,

morbidity, and mortality. The health-protective effects associated with psychological well-being and other psychological resources have been demonstrated to be independent of traditional health risk factors and are documented in both healthy and patient populations (reviewed in Boehm & Kubzansky, 2012; R. Cohen, Bavishi, & Rozanski, 2016; Pressman & Cohen, 2005; Ryff, 2014), although not all reports have found salubrious effects (cf. Freak-Poli, Wolfe, & Peeters, 2010; Liu et al., 2016). Conversely, it has been known for a long time that psychological distress can have negative health consequences. There is extensive prior research on anger and hostility as risk factors for cardiovascular disease and their relevance as common psychological experiences associated with socioeconomic disadvantage.

A seminal article published by the cardiologists Friedman and Rosenman (1959) demonstrated that men with the Type A behavior pattern, which was characterized by hostility, aggression, time urgency, and competitive drive, were at greater risk for myocardial infarction than men exhibiting Type B behaviors, such as patience. Later, it was recognized that anger and hostility were cardiotoxic components of Type A behavior (Matthews, Glass, Rosenman, & Bortner, 1977). A large body of empirical evidence has been amassed to support this association between anger and hostility and more adverse profiles of cardiovascular risk markers (e.g., blood pressure) and disease, independent of traditional cardiovascular risk factors (Caska et al., 2009; Chida & Steptoe, 2009; Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2004; Everson et al., 1999; Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003; Steptoe & Kivimäki, 2013). Conversely, being able to manage anger has been associated with fewer cardiovascular events, quicker wound healing, and a better health-related quality of life (Gouin, Kiecolt-Glaser, Malarkey, & Glaser, 2008; Haukkala, 2014; Haukkala, Konttinen, Laatikainen, Kawachi, & Uutela, 2010; Julkunen & Ahlström, 2006). While psychological resources and risk factors have strong ties to health, they have not always been studied in contexts of inequality, which is an important limitation addressed by the current program of research.

Biobehavioral Pathways

poor health behaviors, less effective emotional regulation, and dysregulated stress responses have often been cited as primary processes through which SES and psychological factors “get under the skin” to affect health (Adler & Ostrove, 1999; Gallo & Matthews, 2003). Smoking, alcohol use, and physical inactivity are key behavioral targets that are known to play an important role in disease pathophysiology and also differ systematically by SES (Lantz et al., 1998; Pampel, Krueger, & Denney, 2010). Lower SES individuals are more likely to use alcohol excessively and have unhealthy diets, be sedentary, and less likely to get regular physical exercise (Barbeau, Krieger, & Soobader, 2004; Hanson & Chen, 2007; Lee et al., 2015; Matthews, Kelsey, Meilahn, Kuller, & Wing, 1989; Melchior, Moffitt, Milne, Poulton, & Caspi, 2007). These behaviors can affect health directly via known physiological pathways, such as insulin-related glucoregulation, and they may also promote obesity, which has independent negative health consequences (National Heart, Lung, and Blood Institute [NHLBI], 2013).

Lower SES environments are further believed to have an inhibitory influence on the development and maintenance of effective emotion regulation strategies, while also fostering negative emotions and diminishing positive psychosocial characteristics (Adler & Snibbe, 2003; Gallo & Matthews, 2003; Matthews, Gallo, & Taylor, 2010). Cross-sectional and prospective research has indicated that clinical and subclinical depression and anxiety, and anger and hostility, which all share some features of emotion dysregulation, follow SES gradients, with lower SES typically linked to worse psychosocial functioning (Gallo & Matthews, 2003; Johnson, Cohen, Dohrenwend, Link, & Brook, 1999; Lorant et al., 2003, 2007; Thurston, Kubzansky, Kawachi, & Berkman, 2006).

Finally, lower SES individuals experience more traumatic events and chronic stress than do higher SES individuals, which affects stress physiology and may lead to physiological dysregulation and disease (Baum, Garofalo, & Yali, 1999; Chandola & Marmot, 2011; Hatch & Dohrenwend, 2007). These biobehavioral

pathways underlie disparities in health, providing some insight into *how* risk and resilience may develop among the socioeconomically disadvantaged.

There is limited empirical evidence for SES differences in physiological dysregulation related to stress, however, and results have been mixed thus far. The functional integrity of many physiological systems may be best examined in response to challenge, as opposed to basal levels. Sustained physiological changes, such as shifting regulatory set points associated with allostasis, are thought to account for why sustained stress among low-SES individuals may lead to disease pathophysiology. Poor and slow recovery, in particular, is thought to reflect a failure to adapt and regulate physiological systems over time following chronic or repeated challenges (McEwen, 1998), and it is an especially important attribute that reflects a heightened vulnerability to downstream health effects (Dickerson & Kemeny, 2004). Blood pressure and heart rate reactivity and recovery to acute stress show relatively consistent associations with SES, such that lower SES has been linked with exaggerated cardiovascular reactivity and prolonged recovery (i.e., elevated blood pressure and heart rate following stressor cessation or increased duration in returning to baseline blood pressure and heart rate; Chida & Steptoe, 2010; Everson et al., 2001; Panaite, Salomon, Jin, & Rottenberg, 2015; Steptoe & Marmot, 2006; Steptoe et al., 2003), although there are inconsistent findings, especially related to cortisol (cf. Dowd, Simanek, & Aiello, 2009; Shaffer et al., 2012; Suchday, Krantz, & Gottdiener, 2005). In sum, while physiological responses to stress are an important pathway linking our social world and psychological experiences to physical health (Kristenson, Eriksen, Sluiter, Starke, & Ursin, 2004), more empirical research is needed to better understand how these mechanistic processes unfold over time. Such efforts will require integrating laboratory studies of physiological reactivity and recovery with evidence of differences in health as a function of SES and psychosocial factors.

Contributions From MIDUS

The MIDUS study has contributed significantly to the literature on differential health outcomes as a function of SES, including at the level of physiological dysregulation assessed with biomarkers. An early study (Lachman & Weaver, 1998) documented associations between income and self-reported health, such that those with higher incomes reported fewer depressive symptoms, better self-rated health, and fewer functional limitations, chronic conditions, and acute physical symptoms. Longitudinal follow-ups of the original cohort have consistently found increased risk for mortality among lower SES individuals, defined by a composite of income, education, assets, and occupational prestige (Chapman, Fiscella, Kawachi, & Duberstein, 2010), as well as among those with lower educational attainment (Turiano, Chapman, Agrigoroaei, Infurna, & Lachman, 2014). With regard to physiological dysregulation, SES gradients have been observed in circulating inflammatory markers (Friedman & Herd, 2010) and allostatic load (Cornman, Gleib, Goldman, Ryff, & Weinstein, 2015). Further, those who experienced socioeconomic adversity in childhood had higher rates of metabolic syndrome (Miller et al., 2011), greater risk for prediabetes and diabetes (Tsenkova, Pudrovska, & Karlamangla, 2014), increased allostatic load (Friedman, Karlamangla, Gruenewald, Koretz, & Seeman, 2015), and several markers of worse bone health (Crandall, Merkin, et al., 2012; Crandall, Miller-Martinez, et al., 2012; Crandall et al., 2015; Karlamangla et al., 2013) in midlife, independent of SES attained in adulthood.

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Building on this foundation of health disparities, the current program of research utilizes a moderation framework to test whether psychological factors confer health risk and resilience in the context of socioeconomic disadvantage. We hypothesize that individuals who possess resilience factors, such as psychological well-being, will show an attenuated association between SES and poor health given the salubrious benefits associated with psychological resilience (mitigation hypothesis). Conversely, individuals with psychological risk factors, such as anger, are expected to show evidence of a stronger link between SES and health, given that health risks associated with having a low SES may compound health risks associated with high anger (exacerbation hypothesis). With the case of anger, we also examine the relevance of these

moderating hypotheses in the context of ethnic minority status and within other sociocultural contexts, capitalizing on assessments from the Milwaukee, Wisconsin, sample of African American individuals and the sample of Japanese adults from the Midlife in Japan (MIDJA) study, respectively. Figure 30.1 provides a visual heuristic of the conceptual framework, and evidence for the mitigation and exacerbation hypotheses is reviewed in the following section.

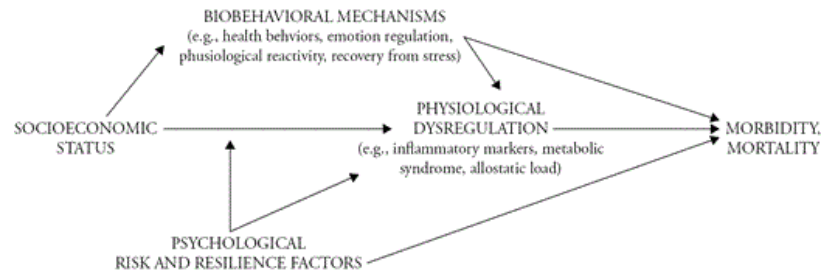


Figure 30.1 Conceptual model: associations among socioeconomic status, psychological risk and resilience factors, biobehavioral mechanisms, physiological dysregulation, and morbidity and mortality.

The MIDUS study is unique among population-based studies in the breadth and depth of psychological factors measured, especially those assessing psychological strengths and virtues. Multiple positive psychological factors have been associated with salubrious subjective and objective health measures, including fewer chronic conditions, less use of healthcare, fewer cardiometabolic conditions and risk (assessed with biomarkers), and lower allostatic load in the MIDUS cohort (Boehm, Chen, Williams, Ryff, & Kubzansky, 2016; Friedman & Ryff, 2012; Keyes, 2005; Keyes & Grzywacz, 2002; Zilioli, Slatcher, Ong, & Gruenewald, 2015). Supporting the mitigation hypothesis, evidence of positive psychological factors buffering associations between SES and health have been noted for self-rated health (Lachman & Weaver, 1998; Ryff, Radler, & Friedman, 2015); mortality (Turiano et al., 2014); chronic conditions (O'Brien, 2012); inflammatory markers (Elliot & Chapman, 2016); and diurnal cortisol (Zilioli, Imami, & Slatcher, 2015). These findings suggest that individuals have some capacity to counteract the usual health risks attendant to socioeconomic disadvantage, a key point to which we return in material that follows.

There are also several examples using MIDUS data that have addressed biobehavioral pathways, providing insight into mechanisms of how SES disparities are established and maintained. For example, Chapman and colleagues (2010) showed that a considerable portion of the SES disparity in mortality risk was accounted for by unhealthy behaviors, such as smoking, drinking alcohol, obesity, and physical inactivity. Related to emotion regulation as a potential pathway, Boehm, Chen, Williams, Ryff, and Kubzansky (2015) found that both lower dispositional optimism and life satisfaction were associated with socioeconomic disadvantage. However, statistical adjustment for negative emotions did not attenuate the association between SES and self-rated health; rather, SES and negative emotions both demonstrated independent associations with health (Barger, 2006). Addressing stress as a mediator, Almeida, Neupert, Banks, and Serido (2005) showed that individuals with less than a high school education experienced more severe daily stressors than those with a high school or college degree. Further, differences in the severity and appraisal of daily stressors appear to account for the influence of educational attainment on physical symptoms and psychological distress. In another MIDUS study, individuals without a college degree reported more physical symptoms and psychological distress on days they experienced stressors than did those with a college degree (Grzywacz, Almeida, Neupert, & Ettner, 2004). Taken together, the aforementioned research sets a solid foundation for the current program of studies, which aims to assimilate work on individual differences in psychological functioning and biobehavioral mechanisms with the extant knowledge of socioeconomic disparities in cardiometabolic risk factors.

Integrative Objectives in the Current Program of Studies

Our research aims to integrate the previously described literatures to approach health disparities within an interdisciplinary, biopsychosocial approach. In the following section, we first describe the evidence that supports the mitigation and exacerbation hypotheses. Subsequently, we summarize our work on additional psychosocial factors that contribute cardiometabolic risk and describe efforts to extend our conceptual framework into additional cultural contexts, taking advantage of the unique opportunity afforded by the MIDJA study.

Psychological Well-Being and the Mitigation Hypothesis

Psychological well-being has been partitioned in two domains, hedonic and eudaimonic (Keyes, Shmotkin, & Ryff, 2002; Ryan & Deci, 2001). Hedonic well-being, focused on pleasurable aspects of life, is typically measured with scales of positive affect, happiness, quality of life, and life satisfaction. Eudaimonic well-being, on the other hand, is multifaceted and emphasizes living life to one's full potential. Extensive theoretical and construct validity efforts have supported a six-factor model for eudaimonic well-being, defined by autonomy, environmental mastery, personal growth, positive relations with others, purpose in life, and self-acceptance (Ryff, 1989, 2014; Ryff & Keyes, 1995). While dimensions of hedonic and eudaimonic well-being are correlated, they exhibit distinct trajectories over the life course and are patterned differently as a function of educational attainment (Lachman, Röcke, Rosnick, & Ryff, 2008; Ryff, 1989; Ryff & Keyes, 1995; Springer, Pudrovska, & Hauser, 2011). Additional psychological strengths, such as optimism and sense of control, are likewise associated with better health (Boehm & Kubzansky, 2012). Importantly, most prior research on positive psychological factors and health pursued these associations as *main effects*. We have, however, considered psychological strengths as both main effects and *buffers* (i.e., moderators) of the association between SES and health. Previous findings from MIDUS (e.g., Elliot & Chapman, 2016; Lachman & Weaver, 1998; O'Brien, 2012; Ryff et al., 2015; Turiano et al., 2014; Zilioli, Imami, et al., 2015) have followed this approach and documented that psychological factors function to mitigate the association between SES and various, primarily self-reported, health outcomes.

Additional efforts have sought to elucidate the intervening biological mechanisms accounting for the mitigation pathway, explaining how well-being can buffer against health disparities associated with SES. Using the MIDUS national sample who completed biological data collection, Morozink, Friedman, Coe, and Ryff (2010) first replicated the inverse association between education and a pro-inflammatory cytokine, interleukin 6 (IL-6). That is, individuals with higher educational status had, on average, lower (i.e., healthier) levels of IL-6. Inflammation plays a key role in the etiology and pathogenesis of several chronic diseases (Ross, 1999), and circulating levels of IL-6 are predictive of future adverse health events in both healthy and patient populations, independent of standard cardiovascular risk factors (Ershler & Keller, 2000; Ridker, Rifai, Stampfer, & Hennekens, 2009). However, this main effect of education on IL-6 was moderated by several components of both hedonic and eudaimonic well-being, including positive affect, environmental mastery, personal growth, positive relations with others, and self-acceptance. Individuals with a high school education or less with relatively high psychological well-being had comparable (lower) levels of IL-6 to those with a college degree, whereas well-being was not associated with IL-6 among the college educated. Put another way, educational gradients in IL-6 were only apparent among those with low well-being and were absent among those with high well-being (Figure 30.2). Importantly, the associations among well-being dimensions and IL-6 were independent of each other. That is, controlling for additional

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dimensions of well-being did not attenuate the interaction between education and any respective measure of well-being.

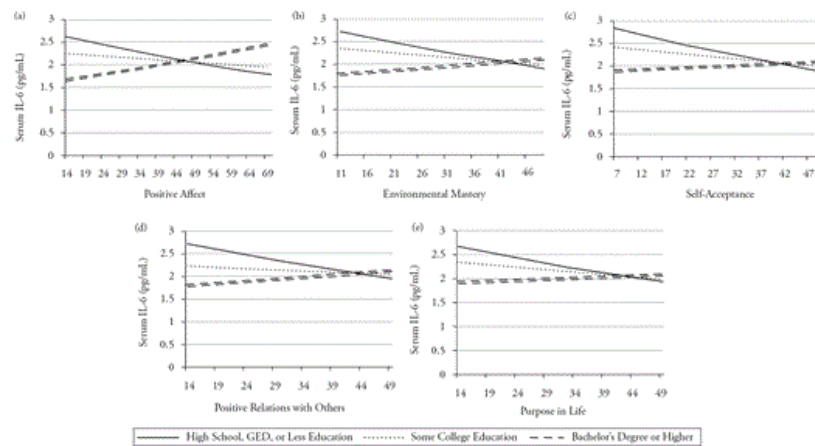


Figure 30.2 Hedonic and eudaimonic psychological well-being moderate educational gradients in circulating interleukin 6 (IL-6).

Reprinted from Morozink et al., 2010.

Depressive symptoms were also considered as an exacerbating factor of the link between educational attainment and IL-6 in Morozink et al. While the pattern of effects supported hypotheses that there were the strongest links between depressive symptoms and IL-6 among the less educated, the interaction was at trend-level significance. Similarly, Lazzarino, Hamer, Stamatakis, and Steptoe (2013) reported the strongest associations between psychological distress and mortality among low-SES individuals in the large, representative Health Survey for England. It will be important for future research to elucidate if a similar pattern of findings for depressive symptoms and anger, as described in material that follows, reflects a general effect of negative affectivity, or whether each emotion domain contributes independent effects (Suls & Bunde, 2005). Further, how distinct psychological factors, both positive and negative, interact in their association with cardiovascular risk measures, and how these play out across different levels of SES, is described in greater detail in following material as a worthy future direction for research.

Anger and the Exacerbation Hypothesis

As just described, psychological vulnerabilities can also exacerbate the association between SES and health. The adverse effects of anger on health have been posited to be greater in the context of low SES given fewer economic, interpersonal, and intrapersonal resources among disadvantaged individuals (Gallo & Matthews, 2003). Anger is an especially relevant emotion in the context of inequality. Experiencing blocked goals and frustrations sparks anger (Berkowitz, 1989), and these may accrue to a greater extent among low-SES individuals (Henry, 2009; Markus, Ryff, Curhan, & Palmersheim, 2004). Indeed, data support that that lower, as compared to higher, SES individuals report experiencing more anger and expressing it more often (Eaker et al., 2004; Haukkala, 2002; Schieman, 2010). The goal of the moderation framework presented here tests whether the risks for poor health associated with lower SES are exacerbated by an emotional profile of high anger. We also consider potential race differences in the exacerbation hypothesis, as African Americans do not experience the same health “returns” associated with higher educational attainment as their white counterparts (Braveman et al., 2010; Lewis et al., 2005).

The MIDUS study is unique among population-based studies in its multidimensional assessments of anger experience and expression for participants who were also a part of the biomarker testing. The available

measures of anger included assessments of how frequently and intensely anger is felt (trait anger) as well as several modes of expression on experiencing anger, such as outward expressions (anger-out), suppressing angry feelings (anger-in), or taking efforts to actively control anger and calm down (anger-control). Using the MIDUS national sample with biomarker data, Boylan and Ryff (2013) tested whether these types of anger experience and expression were associated with inflammatory markers, including the cytokine IL-6, the acute phase protein C-reactive protein (CRP), and fibrinogen, a clotting factor, and whether the associations between anger and these inflammatory indicators were different as a function of educational status.

We (Boylan and Ryff, 2013) found no differences in anger as a function of educational status, with the exception that higher education was associated with higher anger-control among men only. There were also no main effects in associations between anger and the inflammatory markers, although anger-control was weakly and inversely correlated with IL-6 and CRP in bivariate models ($r_s = .10$). However, in line with the exacerbation hypothesis, among those with a high school education or less, trait anger and anger-out were associated with higher IL-6 and fibrinogen, after taking into account numerous sociodemographic, health status, and health behavior covariates. These patterns are depicted in the left panel of Figure 30.3. Supporting the mitigation hypothesis, women with a high school education or less who reported being able to control their anger when felt had comparable levels of IL-6 compared to college-educated women. Interestingly, there were also inverse associations between trait anger and anger-out with fibrinogen among college-educated individuals, perhaps related to health benefits of using anger as a sign of dominance, a finding that we return to in the discussion of culture.

An important limitation of the Boylan and Ryff (2013) study is that the analysis utilized a largely white sample (7.2% nonwhite). To address this shortcoming, Boylan, Lewis, Coe, and Ryff (2015) tested whether similar exacerbation hypothesis patterns were apparent among a sample of African Americans from Milwaukee. These participants were added to the MIDUS survey during the second wave of recruitment.

p. 420 Approximately 200 middle-aged African Americans completed both the survey and biomarker assessments, allowing for critical tests of race differences in the associations between anger, educational status, and inflammation. Indeed, three-way interactions emerged between race, anger (trait anger and anger-out), and education in the prediction of IL-6 and fibrinogen, depicted in Figure 30.3.

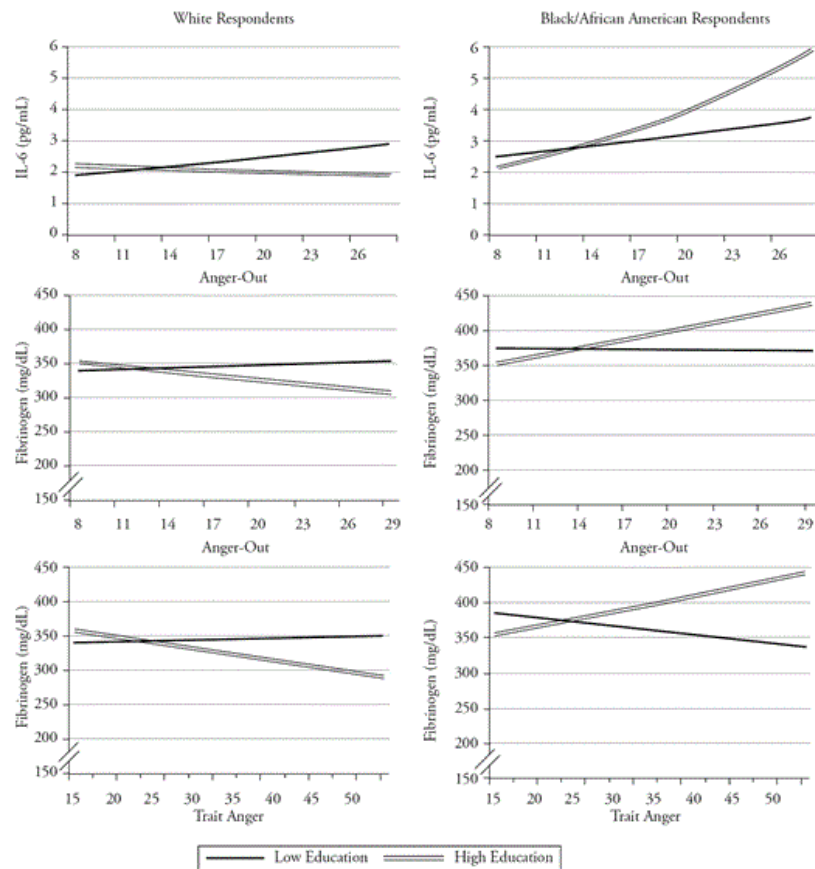


Figure 30.3 Anger moderates educational gradients in interleukin 6 (IL-6) and fibrinogen differently among black/African American and white MIDUS respondents.

Reprinted with permission from Boylan et al., 2015.

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While we found that trait anger and anger-out were associated with elevated inflammation among white adults with lower educational status, anger was clearly associated with elevated inflammatory physiology for African Americans with higher education, independent of health status and health behaviors. These racial differences are consistent with prior research demonstrating that African Americans do not accrue the same health benefits of higher educational attainment as whites. Further, prolonged experiences of discrimination and unfair treatment, despite educational success, may contribute to these indicators of sustained subclinical inflammation. Anger was a common response to inequitable treatment on the basis of race among African Americans who achieved educational and occupational success (Cose, 1993). For highly educated whites, anger expression can signal status and dominance (Tiedens, 2001), whereas there may be social sanctions against African Americans who express anger. Future research should interrogate the role of sociocultural stressors and perceived status as possible precursors to anger among African Americans as well as identify coping mechanisms that may offset their risk and burden on physical health. The previous examples demonstrate how imperative it is that health researchers consider psychosocial factors in context to better understand and ultimately reduce health disparities.

Anger has also been found to exacerbate associations between other key demographic factors, namely, age and health. Aging is known to increase risk for diabetes and cardiovascular diseases (Berry et al., 2012). At the same time, emotional well-being is generally preserved or improved with age (Carstensen et al., 2011). The expected life course pattern is for anger to decrease with age, paralleling enhanced emotion regulation and a normal shifting of focus toward a more positive emotional outlook (Carstensen, Isaacowitz, & Charles, 1999; John & Gross, 2004; Schieman, 2003). Indeed, older adults in the MIDUS biomarker sample reported less anger, on average, than younger adults, and older adults who had higher levels of anger expression

were at the highest risk for metabolic syndrome (Boylan & Ryff, 2015a). In contrast, older adults with low anger expression had a risk for metabolic syndrome comparable to adults several decades younger. These results indicate that age-related advantages in emotion regulation may be offset in high-arousal contexts, including those that evoke the outward expression of anger (Charles, 2010). The extent to which well-being and anger can be effectively modified through cognitive-behavioral interventions and yield physical health benefits, especially among vulnerable constituents, is an exciting avenue for future research.

Additional Protective Factors for Cardiometabolic Risk

Other biomarker analyses from MIDUS, as well as its sister project in Japan, MIDJA, convey that certain psychological resources are protective for health for all individuals, regardless of SES. Boylan and Ryff (2015b) extended the mitigation hypothesis findings by testing associations between hedonic and eudaimonic well-being and a clinical condition, metabolic syndrome. Metabolic syndrome is a composite of risk factors, including central obesity, dyslipidemia, hypertension, and poor gluco-regulation associated with insulin resistance (Grundy, 2005). The unadjusted prevalence of metabolic syndrome in the MIDUS biomarker sample was 36.6%. Hedonic well-being measures of life satisfaction and positive affect and a eudaimonic well-being measure of personal growth were associated with having fewer components of metabolic syndrome and lower risk of meeting diagnostic criteria for metabolic syndrome in cross-sectional models. Further, life satisfaction and a composite of eudaimonic well-being were associated with lower risk of metabolic syndrome in longitudinal models, using well-being measures from MIDUS 1. All associations were independent of sociodemographic (age, race, sex, marital status, education) and health factors, including smoking, physical activity, alcohol, and cholesterol, blood pressure, and glucose-lowering medication usage. Associations between well-being and metabolic syndrome were unchanged when depressive symptoms were added to regression models, and there was no evidence of moderation by age, sex, race, or SES.

Largely missing from the literature on the health benefits associated with psychological well-being is a cultural perspective, given that most of the studies have examined such associations in North American or Western European samples. Previous work from the sister study of MIDUS in Japan, MIDJA, has demonstrated that there are cultural differences in the association between social status and anger (Park et al., 2013) as well as in associations between negative emotions, including anger, and health, assessed via IL-6 and other cardiometabolic risk markers (Kitayama et al., 2015; Miyamoto et al., 2013). Utilizing data from the MIDJA biomarker study, Boylan, Tsenkova, Miyamoto, and Ryff (2017) brought a sociocultural perspective to the study of associations between several varieties of well-being and gluco-regulation. Poor gluco-regulation and Type 2 diabetes are major public health concerns (Morimoto, Nishimura, R., & Tajima, 2010), despite relatively low rates of coronary heart disease in Japan. The MIDJA biomarker sample includes 382 middle-aged adults who reside in the Tokyo metropolitan area. Blood samples were taken to assess several biomarkers, including glycosylated hemoglobin (HbA_{1c}) as the key indicator of gluco-regulation. Given that positive emotions have been reported to be less valued and to be experienced less frequently in East Asian cultures as compared to the United States (Bastian, Kuppens, De Roover, & Diener, 2014; Miyamoto & Ma, 2011), hedonic well-being was not predicted, and not found, to be associated with HbA_{1c}. However, purpose in life was associated with lower HbA_{1c}, and peaceful disengagement, a measure of interdependent well-being developed within the Japanese cultural context, was associated with higher HbA_{1c}. Effect sizes were comparable to that between body mass index and HbA_{1c}. This study provided evidence that positive associations between well-being and physical health are especially strong for purpose in life, including in distinct cultural contexts. However, that associations between well-being and gluco-regulation were patterned differently in the United States as compared to Japan emphasizes the importance of considering the sociocultural context to fully understanding linkages between psychological resources and health.

Future Directions

The research presented in this chapter reflects the efforts of researchers from many different disciplines to address disparities in health from a biopsychosocial perspective. The guiding framework highlights psychological risk and resilience factors as moderators of socioeconomic gradients in health. In the final section, we consider some promising avenues for future inquiry, emphasizing the need to consider psychological strengths and risk factors together and the need for prospective, longitudinal studies. We need to consider the questions of *who* and *how* together and, ultimately, to validate these conclusions with interventions that can beneficially change the factors identified as mediators of risk.

Bringing positive and negative psychological factors together.

Much of the research modeling positive and negative psychological factors as predictors of health and as moderators of SES gradients in health has treated psychological resilience and risk factors independently. Future efforts should attempt to consider psychological risk and resilience factors in a more summative and unified manner, as well as address processes of stability and change over time. While positive and negative psychological factors tend to be moderately inversely correlated, they are not just opposite ends of a continuum (Keyes, 2005; Keyes et al., 2002). Indeed, individuals are composites of both positive and negative psychological traits, and these unique profiles emerge and are manifest differently depending on SES. Kashdan and Rottenberg (2010), for example, argued that psychological flexibility, not simply positivity, is a cornerstone for health. At this time, how positive and negative traits synergistically affect health is an open scientific question. Current evidence indicates that there are distinct, not simply opposing, associations between positive and negative psychological factors and health (Ryff et al., 2006).

Another important consideration for future research is whether the theoretical distinctions between hedonic and eudaimonic well-being translate at the level of biology. Relatively few studies consider hedonic and eudaimonic well-being simultaneously, making it difficult to discern whether they have overlapping or independent contributions to health, although the available evidence supports their independence (Fredrickson et al., 2013; Friedman, Hayney, Love, Singer, & Ryff, 2007; Morozink et al., 2010; Saloumi & Plourde, 2010). Additionally, research in this area has focused heavily on circulating inflammatory markers as the primary biological outcomes, and future research should look to extend the moderation findings to other clinically relevant outcomes.

Longitudinal approaches.

Much of the research described in this chapter on the psychological moderation of SES gradients in health is based on cross-sectional data. With the additional waves of MIDUS forthcoming, longitudinal analyses will be able to establish temporal precedence and examine how dynamics between SES and psychological risk and resilience factors emerge over time. When the biological assessments have been repeated in MIDUS 3, it will allow for prospective testing of the associations between biological risk factors and disease morbidity and mortality. Longitudinal studies should also assess dynamics over time in well-being and anger measures (i.e., gains, losses) to determine how these psychological factors change normally with age (e.g., Carstensen et al., 2011; Mroczek & Kolarz, 1998). It is also likely that the dimensions of stability and change will differ across socioeconomic strata. Prior research indicated that some dimensions of hedonic and eudaimonic well-being have unique age trajectories (Ryff & Keyes, 1995; Springer et al., 2011). It appears that persistently high eudaimonic well-being over a 10-year window is protective for subjective health, functional limitations, chronic conditions, and health symptoms, and these effects were stronger among less educated individuals (Ryff et al., 2015).

Integrating longitudinal and experimental methods.

p. 423 Modeling socioeconomic disadvantage, psychological functioning, biobehavioral mediators, and health is complex, with interactions unfolding at multiple levels over the life course. Indeed, much of our empirical evidence was obtained in fragmented form, with very few analyses capable of testing multiple aspects of the whole model. This limitation is an important constraint, and studies such as MIDUS, with assessments of life course SES and psychological functioning, daily diary assessments, and multiple tests of reactivity and recovery, will provide new opportunities to empirically validate the conceptual framework presented herein.

Schaefer and colleagues (2013) provided a noteworthy example of this type of effort within MIDUS, linking purpose in life with findings from affective neuroscience to elucidate neural circuitry underlying psychological well-being. In over 250 respondents, higher purpose in life was prospectively associated with faster recovery from negative emotional stimuli. Emotional recovery was assessed by an attenuated eye-blink rate (EBR) following exposure to negative emotional stimuli, a measure known to reflect amygdala activation (Davis, 2006). Further, effects were independent of age, sex, positive and negative affect, life satisfaction, and baseline and reactivity EBR. These results suggest that one way in which purpose in life may contribute to resilience is by enhancing emotion regulation and enabling reappraisal and faster recovery from negative experiences.

Psychological factors may similarly mitigate and exacerbate physiological reactivity and recovery to stress. In one laboratory study, African Americans exhibited delayed cardiovascular recovery in response to an anger instigation task, regardless of whether anger was expressed or inhibited during the task. In contrast, Americans from European family backgrounds did not evince delayed recovery when they were allowed to express their anger (Dorr, Brosschot, Sollers, & Thayer, 2007). These results suggest there may be fewer options available for regulating anger that do not have negative health consequences for African Americans. If confirmed, it emphasizes how critical it is to consider how race and ethnicity differentially influence experiences following similar exposures. It will also help us to better understand how historical and contemporary experiences of discrimination may affect the associations between psychosocial factors and health, elevating the risk for certain racial and ethnic subgroups.

As another example, low childhood SES was associated with higher systolic blood pressure and heart rate in the recovery following an acute laboratory stressor (Boylan, Jennings, & Matthews, 2016). However, psychological resources moderated the link between childhood SES and systolic blood pressure in this community sample of black and white men, such that low-SES men with high psychological well-being did not exhibit elevated blood pressure during recovery. These low-SES men with greater psychological resources had blood pressure levels during recovery that were comparable to their higher SES counterparts.

These findings convey that the capacity to recover quickly after a stressful event is as an important component of the cascade linking childhood SES, psychological factors, and health. Future efforts need to address these temporal and dynamic aspects of mediation and moderation in order to better understand the pathways underlying SES disparities in health.

In sum, integrative models that bring longitudinal approaches together with experimental assessments of reactivity and recovery in real time offer promise to assess the multiple pathways leading to risk and resilience simultaneously. However, such models cover wide methodological territories and demand an interdisciplinary approach. Future efforts will face the challenge of maintaining methodological rigor while tracking these complicated processes as they play out differently across the life course and across socioeconomic strata.

Intervention approaches.

Efforts are already under way to translate and apply these findings, testing interventions to improve psychological well-being and teach anger management. The initial results have been very promising, indicating that this type of cognitive-behavioral training will be effective for improving both mental and physical health. Specifically, as a new form of cognitive-behavioral therapy, “well-being therapy” has been effective at reducing relapse rates for major depression (Fava et al., 2004) and as a treatment modality for generalized anxiety disorder (Fava et al., 2005; Ruini & Fava, 2009). Well-being therapy fosters engagement with positive experiences in one’s life, and patients have found it effective at promoting recovery in the context of major mental illness. Targeting well-being in a group setting has also been effective at reducing distress among adolescents and older adults, respectively (Friedman et al., 2017; Ruini, Belaise, Brombin, Caffo, & Fava, 2006). Older adults likewise showed decreases in depressive symptoms, fewer physical symptoms, and fewer sleep complaints as a function of increased well-being when evaluated in community-based participatory research (Friedman et al., 2017). In a more definitive randomized control trial, it was demonstrated that increasing certain dimensions of eudaimonic well-being, specifically purpose in life, was effective in improving quality of life among cancer patients (Breitbart et al., 2012; van der Spek et al., 2014), although the extent to which it affected disease course and recovery is not known. Interventions to enhance hedonic well-being in undergraduate students by writing about one’s life goals were associated with reductions in visits to student health facilities over a 5-month period (King, 2001).

Several interventions have already been developed for teaching better anger management strategies. Among both children and adults, interventions that reduced anger led to fewer depressive symptoms and increased self-efficacy, as well as improved long-term educational outcomes (e.g., Coon, Thompson, Steffen, Sorocco, & Gallagher-Thompson, 2003; Lochman & Lampron, 1988). Further, programs that helped to reduce the feelings of hostility and anger among cardiac patients were especially effective in the recovery from cardiovascular events (Blumenthal et al., 1997; Friedman et al., 1986). There are still many open questions about the sustained duration and effectiveness of these interventions, but the likelihood they will enhance quality of life and accrue benefits for physical health appears promising. The biological mediators of these improvements are not yet known, but changes in health behaviors, improved emotion regulation, and more adaptive recovery are likely to be key mediating pathways. One further challenge for this type of intervention research will be to evaluate the effectiveness in socioeconomically and racially diverse samples given that we are already aware of the significant disparities among the more vulnerable members of our society. It is likely that the interventions will have to consider the unique cultural and familial perspectives, as well as economic constraints of individuals from disadvantaged backgrounds.

Conclusion

In conclusion, inequality is a pressing issue with far-reaching economic, social, and health-related implications. Integrative approaches to studying health disparities have proven useful for elucidating key factors in buffering the impact of SES on health. In this chapter, we considered the role of psychological risk and resilience factors as moderators of SES gradients in health. The findings convey that both positive and negative psychological experiences can mitigate or exacerbate, respectively, the association between SES and health. We also identified a number of key biobehavioral pathways that account for how socioeconomic and psychosocial factors get under the skin to affect disease risk. The findings highlight promising avenues for future research and intervention. Ultimately, the hope is to reduce health disparities and improve the health and well-being of all segments of society.

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