Published in final edited form as:

Annu Rev Psychol. 2011 ; 62: 501–530. doi:10.1146/annurev.psych.031809.130711.

Psychological Perspectives on Pathways Linking Socioeconomic Status and Physical Health

Karen A. Matthews¹ and Linda C. Gallo²

Karen A. Matthews: matthewska@upmc.edu; Linda C. Gallo: lcgallo@sciences.sdsu.edu

¹Department of Psychiatry, University of Pittsburgh, Pittsburgh, Pennsylvania 15213

²Department of Psychology, San Diego State University, San Diego, California 92120

Abstract

Low socioeconomic status (SES) is a reliable correlate of poor physical health. Rather than treat SES as a covariate, health psychology has increasingly focused on the psychobiological pathways that inform understanding why SES is related to physical health. This review assesses the status of research that has examined stress and its associated distress, and social and personal resources as pathways. It highlights work on biomarkers and biological pathways related to SES that can serve as intermediate outcomes in future studies. Recent emphasis on the accumulation of psychobiological risks across the life course is summarized and represents an important direction for future research. Studies that test pathways from SES to candidate psychosocial pathways to health outcomes are few in number but promising. Future research should test integrated models rather than taking piecemeal approaches to evidence. Much work remains to be done, but the questions are of great health significance.

Keywords

stress; resources; emotions; inflammation; metabolic factors; sleep; race; gender; life course; socioeconomic status

INTRODUCTION

Study of the relationships between socioeconomic status (SES) and health has had a long scientific history. Since the time of rapid industrialization and migration of the population into urban areas in Europe, observers have noted links between poor health and adverse living conditions associated with poverty. In the past few centuries, carefully conducted studies documented disease associations with poverty using early statistical approaches, e.g., mapping cases within geographical areas, comparing disease and healthy groups on living conditions. Medicine, epidemiology, sociology, demography, and economics are only some of the disciplines that participated in establishing the strong and consistent gradients between SES and health. Across diverse health outcomes, individuals who are less educated, have lower-status jobs, and earn less or no income are at greater risk for poor health than their higher-SES counterparts. The associations extend from relatively minor illnesses, e.g., headaches, to serious and life-threatening diseases, e.g., coronary heart disease, to early mortality, and are apparent across the life course. Furthermore, the associations are not

Copyright © 2011 by Annual Reviews. All rights reserved

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

solely due to poverty but rather have a monotonic or step-wise functional relationship with health. As an exemplar, Figure 1 shows the gradient: the higher the education, the lower the rates of osteoarthritis, chronic diseases, hypertension, and cervical cancer (Adler et al. 1994).

Only recently has psychology responded to the challenge of the SES gradient. In a seminal paper, Adler and colleagues (1994) described the associations of SES with diverse health outcomes and raised questions about possible pathways. Moving beyond the perspective that SES is a “nuisance variable” to control statistically, awareness of the importance of SES has led to an appreciation of what subgroups are most vulnerable to disease and to an explosion of research on pathways. As a better understanding of psychosocial pathways accumulates, health psychology has the potential to contribute to prevention and intervention efforts that help reduce the tremendous health burden created by low SES.

The overall goal of this review is to describe major new findings regarding psychobiological pathways connecting SES with health and to suggest avenues for investigation based on psychological theory. This review is restricted to examining physical health outcomes, for several reasons. First, psychological research on physical health in relation to SES is relatively recent and a review is timely. Second, the developmental literature has examined the role of SES and/or poverty extensively on children’s achievement and academic skills and socioemotional development and is available elsewhere (e.g., Bradley & Corwyn 2002, Evans 2004). The review also does not investigate the area of SES in relation to health behaviors, i.e., smoking, excessive eating and alcohol consumption, and physical inactivity, which are part of the pathways connecting SES and health. This literature is substantial and beyond the scope of this review.

The review begins with the formal definition and discussion of approaches to conceptualizing and measuring socioeconomic status. We then discuss the status of evidence on the role of stress and associated distress and social and personal resources, as most models consider these key pathways. For the most part, the issues discussed to that point emphasize adult SES–health pathways. The third major section emphasizes the burgeoning focus on SES and child health and how early SES sets a trajectory of risk over the life course. Then we discuss new directions in understanding the biological pathways underlying associations among SES, psychosocial factors, and health, with emphasis on biomarkers that compose the concept “allostatic load,” subclinical disease indicators, and the role of the brain. A final section discusses the challenges that face psychologists invested in understanding the SES gradient.

CONCEPTUALIZATION AND MEASUREMENT OF SOCIOECONOMIC STATUS

SES is a complex, multidimensional construct that has been conceptualized and measured in diverse ways (Krieger et al. 1997). In general, SES indicators represent access to material and social resources and assets, or rank within a social-economic hierarchy, or both. SES can be evaluated at various periods in the life course, from gestation to late adulthood, and the level of assessment may vary from the individual, to the household or family, to the community, to the national or global. Below we address key issues in SES measurement and describe the strengths and limitations of common assessment techniques as well as more recent, novel approaches.

Resource versus Status-Based Approaches

A fundamental distinction in SES measurement is whether indicators characterize an individual’s rank or prestige in relation to others (i.e., the “social” component of SES) or

access to material and social resources and goods (i.e., the economic component). Prestige-based measures refer to a person's relative status within a social hierarchy and provide information regarding his/her ability to obtain merchandise, services, and knowledge. Examples include occupational classification systems that provide clear, relative rankings—for example, military or civil service grades. In addition, measures that evaluate peoples' subjective perceptions of their status relative to others' tap rank and prestige facets of SES. In one common method of assessing "subjective social status," participants place themselves on a graphical 10-rung ladder, with the top rung representing those individuals with the highest status, e.g., according to typical SES indicators or individually defined characteristics, and the bottom rung representing those with the lowest status (Adler et al. 2000). Such measures are receiving increasing attention in public health research in the United States and elsewhere (Adler et al. 2000, Cohen et al. 2008, Manuck et al. 2010). Otherwise, strict, rank-based measures are less frequently employed in the United States, where class-driven social hierarchies may be less clear-cut than in other nations. In contrast, the most typical measures of SES in the United States capture material and social resources—for example, whether or not an individual has a college degree or owns a home, makes an income that is above the poverty line, or has access to health care (e.g., health insurance). Prestige and resource-based measures of SES are related, but not enough to be redundant.

Traditional SES Indicators

The most commonly used measures of SES in public health research are education, income, and occupation. In general, these factors capture both resource and prestige components of SES. Moderate correlations among them suggest that although related, these indicators also represent distinct socioeconomic information.

Education is a fundamental component of SES that precedes later markers such as occupation, income, and wealth. Its advantages include the fact that it can be used even in individuals who are not in the labor force and that it is typically completed prior to the onset of major health problems (excepting those in childhood); therefore, it is less vulnerable to the impact of reverse causation (meaning poor health leads to lower SES) than are other markers. In addition, education questions are typically answered accurately and with low refusal rates. Limitations of education include the fact that its meaning and implications for earnings potential may vary across demographic groups, e.g., as defined by age, gender, ethnicity/race, or national origins. In addition, education is a relatively crude indicator, with few categories, and does not include information about educational quality.

Income is another widely used SES indicator, especially in affluent, industrialized nations. Associations between income and health tend to be nonlinear and are steepest at the lowest SES levels. Household income estimates adjusted for the number of individuals supported provide more complete information regarding standard of living and ability to access material goods. In addition to absolute and adjusted income levels, income dynamics (i.e., changes in income across the lifespan) provide important information regarding health risks. Despite its utility, income information is subject to high nonresponse rates and biased reporting and can be less relevant to individuals who are not in the labor force (e.g., due to retirement or caretaking responsibilities). Likewise, income does not provide comprehensive information about buying power or standard of living since this may vary across community contexts and also demographic characteristics. To offset these latter limitations, information about wealth, especially among the elderly, can be a useful adjunct for research that seeks to determine income effects on health.

Finally, many studies have sought to examine the association between occupational status and physical health. In this approach, occupations are categorized hierarchically according to characteristics such as earned income, required education, associated prestige, skills, and

level of influence. Limitations of occupational measures include the fact that they cannot be used for individuals who are not working, that occupational status rankings are often highly subjective, and that occupation categories contain considerable variation in prestige, earnings, and other factors (Braveman et al. 2005).

Contextual Approaches

In another line of work, researchers have begun to examine how the socioeconomic contexts in which people live relate to health beyond person- or household-level markers. Such measures provide important supplementary information since people with similar SES can inhabit very diverse communities or neighborhoods. For example, when compared to non-Hispanic whites with similar income levels, ethnic minorities frequently reside in more disadvantaged communities (Williams & Jackson 2005). The pathways linking contextual SES with physical health may also be distinct. For instance, individuals who live in lower-SES neighborhoods typically have less access to healthy food and safe places to exercise (Lovasi et al. 2009), and they are also more vulnerable to environmental health risks, such as second-hand smoke, air pollution, ambient noise, suboptimal housing, crowding, and crime. Contextual SES is commonly indicated as the aggregate SES of individuals living in the area (e.g., percent of residents living below the poverty line, proportion with a college degree), with census tract or block-level data providing a more accurate representation than zip code information. A good deal of research suggests that neighborhood SES is predictive of health over and above the impact of individual SES (Pickett & Pearl 2001) even though these measures tend to be significantly correlated. On the other hand, since many studies account for only a single individual-level SES indicator when exploring area-based effects, person-level SES may still account for area effects to some extent (Pickett & Pearl 2001). Importantly, although researchers sometimes use community SES to estimate individual SES when the latter information is unavailable, this approach can lead to a substantial misestimation of true SES effects given the potential inaccuracy involved (Geronimus & Bound 1998). Furthermore, community-level SES is not a static variable and can be quite dynamic as individuals migrate to other communities or communities change dramatically with economic downturns or upturns. These and other limitations and difficulties in studying area-based health effects have been discussed in detail elsewhere (Diez Roux 2001).

Life Course Approaches

One recent direction in SES assessment is to examine how socioeconomic experiences across the life course relate to physical health outcomes (Pollitt et al. 2005). In this approach, knowledge concerning the timing and length of exposure to socioeconomic deprivation (or affluence) as experienced across various developmental stages is considered critical to fully understand the sociobiological impact of SES, particularly for chronic diseases with protracted etiologies. Many of the same adult and childhood SES indicators discussed above are used in life course-based research. However, most of the available studies use retrospective measures of SES in childhood and do not assess SES at multiple time points, such as infancy, childhood, adolescence, and/or various stages of adulthood. Nonetheless, this work is important to understanding the psychological pathways leading to adult health and is reviewed later in more detail.

Summary

Consistent with the multidimensional nature of the construct, SES measures vary widely along multiple dimensions including timing, level, whether they are objective or subjective, and whether they evaluate prestige, resources, or both. In general, measures should be selected to fit the population of interest and should be based on a clear theoretical articulation of how SES relates to the specific health outcome being addressed. In addition, studies that administer multiple measures of SES are best able to capture health

implications, given that individual indicators are typically only moderately correlated and may relate to health through divergent pathways.

APPROACHES TO UNDERSTANDING PSYCHOSOCIAL PATHWAYS

For a psychosocial pathway to be relevant to understanding the SES gradient, beyond the effects of poverty, three conditions should be met: SES should be related to the pathways in a step-wise or graded fashion; the putative pathway must be related to a health outcome in a step-wise fashion; and introducing the pathway into statistical models of the association between SES and health outcome must reduce the magnitude of the association. Note that a candidate mechanism does not need to eliminate the association because there are undoubtedly multiple contributing factors. In most conceptual models, possible psychosocial pathways connecting low SES with poor health can be distilled roughly into two categories: stress and concomitant psychological distress, and psychological and social resources that may be a mediational pathway or protect against stressful environments and the negative emotional responses they elicit. Then, in turn, these pathways are thought to connect to adverse biological processes and negative health behaviors (labeled biobehavioral), subclinical disease, and eventually to frank disease. Figure 2 illustrates these pathways.

Stress and Distress

A number of researchers have posited that low-SES environments foster negative health outcomes in part because they increase exposure to stress and adversity. Either as a function of increased stress exposure or as a direct correlate of disadvantage, individuals with low SES may also be more vulnerable to psychological distress (Gallo & Matthews 2003). There is a fair amount of indirect support for this perspective, with studies showing that low individual or community SES relates to higher scores on measures of traumatic and life events, chronic stress, perceived stress, and daily hassles (Gallo et al. 2005, Hatch & Dohrenwend 2007, McLeod & Kessler 1990). SES also demonstrates an inverse association with indicators of psychological distress, such as anxiety and depression symptoms or disorders, hostile cognition, and angry emotion (Gallo & Matthews 2003). In turn, stress and distress are increasingly recognized as important factors in major physical health outcomes, including cardiovascular diseases (CVDs) and all-cause mortality (Cohen et al. 2007, Everson-Rose & Lewis 2005).

However, a closer look at the literature reveals many inconsistent findings and limited available direct support for a mediating role of stress. For example, some published studies report no relationship between SES and stress, variously defined (Matthews et al. 2008, Prescott et al. 2007), or even a positive relationship so that higher SES predicts greater stress (MacLeod et al. 2005). In a recent review, Matthews et al. (2010) found that only four out of nine studies that examined stress (primarily life events or perceived stress) as an intermediate factor connecting SES with objective physical health outcomes identified supportive evidence. Different aspects of “job strain” (i.e., chronic work stress) contribute to the relationship between SES and health outcomes, such as coronary heart disease (Marmot et al. 1997, Wamala et al. 2000). However, a number of studies have identified null findings when attempting to investigate whether various forms of generalized stress (Avendano et al. 2006, Matthews et al. 2008, Prescott et al. 2007) or work stress in particular (Kuper et al. 2007) contribute to SES–health associations. The research concerning psychological distress is likewise mixed. Whereas some studies have identified support for an intermediate role of negative emotions, including depression or anxiety, in connecting SES with health outcomes such as stroke (Avendano et al. 2006) or the metabolic syndrome (Matthews et al. 2008), other studies have failed to provide evidence for these pathways (Cohen et al. 2008, Thurston et al. 2006). The inconsistency of available research is notable, but the small number of studies that have sought to test the role of stress empirically is even more striking,

especially given the considerable attention that this pathway has garnered in the SES–health literature.

The relatively weak support for an intermediary role of stress and distress must be considered in light of measurement limitations. Stress is a complex, multifaceted construct, and associations with SES could vary along a number of dimensions, including stressor domain, duration, severity, and whether stress exposures (e.g., objective events), experiences (e.g., perceptions of stress), or responses (i.e., subjective distress) are being assessed. Many large surveillance studies rely on brief, often nonvalidated measures of these constructs (Khang et al. 2009, Prescott et al. 2007), which may fail to adequately capture the contributions these variables make to SES–physical health associations. Moreover, recall biases associated with aggregate self-report measures can lead to under- (or over-) estimates of associations, especially when participants are asked to recollect the nature and severity of stress or psychological distress experienced across long periods of time.

New assessment approaches that examine contemporaneous accounts in daily life (Shiffman et al. 2008) may be more useful than retrospective assessments of past stressors. The notion is that the closer in time the report is to the event, the more accurate the report will be. Several studies using the concurrent methodology suggest that persons with low-status occupations experience more social conflict throughout the workday than persons with high-status positions (Gallo et al. 2005, Matthews et al. 2000a). In a large representative sample of middle-aged adults, individuals who had more education reported more stressors than those with less education on end-of-the-day interviews that measured stress exposure, stress severity, and symptoms (Almeida et al. 2005). However, less-educated individuals experienced more severe stressors and appraised them as posing greater risk to their financial situations and self-concepts than did those with more education. Findings also suggested that low education rendered participants more vulnerable to stress: On days that individuals experienced stressors, the less-educated reported greater distress and physical symptoms compared to their better-educated counterparts (Grzywacz et al. 2004). Nonetheless, the contention that stress is an important pathway underlying SES–physical health disparities requires additional empirical evidence. We are not suggesting that stress is not an important determinant of physical illness (Cohen et al. 2007). However, it may not be the primary mediator of SES–physical health associations.

Psychosocial Resources

In contrast to the literature concerning stress, the research that has examined the contribution of positive psychosocial factors provides greater support. These studies have examined psychological resources, such as a sense of mastery or control over important aspects of life, self-esteem, or optimism, and social variables, such as social support, integration in a social group, or social capital at the community level. Research suggests that these variables can exert salutary effects (Rasmussen et al. 2009, Uchino 2006), and when present in abundance, such resources may be able to protect individuals from the adverse health consequences of material deprivation (Lachman & Weaver 1998). Research shows that individuals with low SES typically report lower levels of resilient resources, including perceived control and social support or integration (Gallo & Matthews 2003), suggesting that these variables may indeed serve as one link in the chain connecting low SES with poor physical health.

A recent review of the literature examining the contribution of psychological or social resources to associations between SES and objective physical health outcomes identified mixed evidence for the roles of social variables, with 6 out of 10 studies identifying mediating effects (Matthews et al. 2010). For example, in a relatively young sample of women, general social support predicted higher stroke risk and, therefore, had little explanatory power in relation to the SES–stroke gradient (Kuper et al. 2007). In contrast,

social integration explained more than one-third of the excess stroke risk associated with low education in an older sample of men and women (Avendano et al. 2006). In other work, perceptions of support from the community, but not from family and friends, contributed to the relationship between income and heart disease and all-cause mortality in elderly men (Chaix et al. 2007). Although the populations, health outcomes, and specific SES indicators and social characteristics examined were quite variable, it is difficult to attribute the heterogeneous findings to any discernable pattern. Moreover, measurement limitations are again a concern, given that social resources, like stress, can vary along many dimensions, including qualitative or quantitative aspects of support or integration and individual-level or community-level aspects of support.

The evidence for the contribution of generalized psychological resources, although limited, has been consistent, with all four studies to date finding evidence of a mediating role for these variables (Matthews et al. 2010). Especially strong support was identified in a study of men performed by Bosma and colleagues (1999), which found that between one-third and two-thirds of the excess mortality risk attributable to low SES could be explained by variation in generalized control beliefs. Control beliefs also explained a substantial degree of the elevated coronary heart disease risk found in elderly persons with low SES (Bosma et al. 2005). In general, a great deal of interest has surrounded the potential contribution of control beliefs, which could contribute to health outcomes through a variety of pathways, such as problem solving (e.g., active attempts to cope), depression (i.e., since beliefs of low control can be equated with helplessness, a known correlate of depression), or adaptive health behaviors.

Integrative Models of Stress and Resources

A number of models similar to that illustrated in Figure 2 integrate stress and resources into unified frameworks that propose sequential relationships among SES, psychosocial pathways, and bio-behavioral mechanisms connecting them with health. For example, Adler and colleagues (1994) developed an overarching model that features environmental resources and constraints in one category and psychological affect and cognition in a second category. Environmental factors are thought to shape psychological processes, exposures to carcinogens and pathogens, and performance of health-relevant behaviors. In turn, psychological factors are believed to influence health-relevant behaviors and neuroendocrine responses to stress as intermediate pathways to physical disease. Marmot proposed a similar broad-based model that emphasizes the role of the work environment in the relationship between SES and physical health (e.g., Marmot et al. 1997; see Related Resources).

Another framework, the reserve capacity model, focuses on interpersonal and intrapersonal resources as modifiers of the impact of environmental demands and stresses on emotional and physiological responses (Gallo & Matthews 2003). In this perspective, low-SES environments are posited to foster greater exposure to frequent and intense harmful or threatening situations and fewer rewarding or potentially beneficial situations, which, in turn, are believed to have a direct, negative impact on emotional experiences. In addition, low-SES individuals are purported to maintain a smaller bank of resources—tangible, interpersonal, and intrapersonal—to deal with stressful events when compared to their higher-SES counterparts. Resource banks, termed reserve capacity, may be low in low-SES circumstances for two reasons: (a) Low-SES individuals are exposed to more situations over the life course that require the use of resources, and (b) their environments prevent the development and replenishment of resources to be kept in reserve. Hence, because individuals of low SES have fewer stress-dampening resources, which are further reduced by enduring or repeated stress exposures, they are likely to show increased responsiveness when faced with challenges and demands. In turn, elevated negative emotions and cognitions lead to alterations in health behaviors and intermediate physiological pathways,

e.g., sympathetic-adrenal-medullary (SAM) system and hypothalamic pituitary adrenal axis (HPA) system responses, and eventually to poor health. An important extension and elaboration of this model by Myers (2009) emphasizes the role of cultural resources in understanding minority health. In addition, he notes that race-specific stressors, when added to the impact of low SES, can lead to cumulative vulnerability and poor health over the life course.

In general, these models are attractive because they generate specific, theoretically driven hypotheses concerning relationships among categories of variables. However, testing them empirically has been challenging. Connections have been established between SES and model components, and between model components and physical health, but studies that move beyond piecemeal tests to integrative evaluations are relatively scarce. Given proposed temporal relationships and causal influences, analytic approaches that facilitate examination of longitudinal data and sequential pathways among constructs are best suited to testing these comprehensive conceptual models (see Related Resources).

In a recent study that applied these methods in an attempt to evaluate several tenets of the reserve capacity model, Matthews et al. (2008) reported that lower educational attainment was associated with the development of the metabolic syndrome in a sample of middle-aged women followed for 12 years. Using structural equation modeling, this study examined whether lower education was related to higher stress, which in turn was hypothesized to elicit greater emotional distress among those with low reserve capacity, and whether these pathways would connect SES with metabolic syndrome development. The relationships among the model components were not as predicted. Rather, the analyses revealed that lower education was related to metabolic syndrome through low levels of reserve capacity measured by a combined index of optimism, self-esteem, and social support, which in turn related to negative emotions, which subsequently connected with metabolic syndrome risk. Thus, stress did not play a primary role in the association between SES and the metabolic syndrome, contrary to the tenets of the reserve capacity model, even though stress was directly related to the development of the metabolic syndrome in another analysis performed in this sample (Räikkönen et al. 2007). A similar test in a sample of Latin American women showed that lower SES was related to higher waist circumference, a primary component of the metabolic syndrome, through low reserve capacity as measured by a sense of mastery, optimism, self-esteem, and social support (Gallo et al. 2007). These studies illustrate the iterative process of model development and testing. Revision of the reserve capacity model may be warranted as new evidence is evaluated; for example, resources may have a more direct role than originally posited, consistent with the conservation of resources model (Hobfoll 1989).

In another integrative approach, researchers have combined stress and psychological factors into a composite psychosocial index in order to compare their, and other possible, explanatory pathways. For example, in the Norwegian HUNT Study, low education and income were related to mortality in men (but not in women; Skalicka et al. 2009). A psychosocial index was composed of positive social relationships, negative emotions, and self-esteem. Other candidate pathways included material factors measured by employment status, financial difficulties, and public benefits; health behaviors as measured by smoking, activity, alcohol, and caffeine intake; and biomedical factors represented by cardiovascular risk factors. Analyses revealed that psychosocial variables and health behaviors helped explain the association of mortality with education, whereas material factors and to a lesser extent psychosocial variables contributed to the association of mortality with income. A similar approach was used in a sample of South Koreans, where material factors were measured by income, health insurance, and car ownership, and psychosocial factors were indicated by depression, stress, and marital status (Khang et al. 2009). Results showed that

the associations of all-cause mortality with education or occupational status, the markers of SES in these analyses, were substantially reduced with statistical control for material factors. In contrast, the percent reduction in low-SES related mortality risk resulting from control of psychosocial factors was small. Comparable results were obtained in a study performed in the Netherlands, which showed that effects of education on mortality were accounted for by material factors, including home ownership, financial difficulties, and health insurance (van Oort et al. 2005). Psychosocial factors reduced the risk of the lowest category of education by 40% when taken individually, but only by 7% when added to the model that included material factors.

These studies illustrate several points. First, some of the variables considered to be material factors, e.g., car ownership, income, and employment status, can also be conceptualized as indicators of SES. Thus, it is not surprising that material factors would account for a substantial amount of the relationship between SES and health, since SES (the predictor) and material factors (the candidate pathway) are potentially confounded. As such, after accounting for material factors, the amount of variance left to be explained by psychosocial factors or any other candidate pathway would be quite small. Second, some of these studies categorized SES into two to four discrete groups and compared the lowest to the higher SES categories. This approach obscures identification of pathways that may contribute to the step-wise, graded association between SES and most health outcomes. Despite these issues, some analyses do find that associations between SES and health are mediated in part by psychosocial pathways and that material factors also affect psychosocial pathways (van Oort et al. 2005), which would be expected if they are markers of SES.

DEVELOPMENTAL PERSPECTIVES ON SOCIOECONOMIC STATUS OVER THE LIFE COURSE

Up to this point, we have focused on the pathways potentially connecting SES with health in adulthood. There is also a substantial body of work evaluating associations and pathways in children and adolescents (Chen et al. 2002). The studies in the young have typically relied on “softer” health indicators, such as parental reports of health quality or seeking medical treatment for health problems. However, in general, the literature does suggest that lower-SES children (based on family SES) are at greater risk for diverse health problems, such as multiple health complaints, physical symptoms, elevated ambulatory blood pressure, obesity, and poor overall self-rated health (Due et al. 2003, McGrath et al. 2006, Shrewsbury & Wardle 2008).

Recently, the adult and child literatures have been married in studies evaluating whether early exposure to low SES in childhood is linked to later physical health problems in adulthood. Using even crude indicators of retrospectively assessed SES in childhood, e.g., father’s occupation grouped into manual-nonmanual categories, early socioeconomic circumstances relate to premature mortality and cardiovascular morbidity in a majority of epidemiological investigations (Galobardes et al. 2006, 2008). Notwithstanding the largely consistent literature, several important cautionary notes must be stated. Perhaps most importantly, the adequacy of the empirical tests of these associations is sometimes limited (for a full discussion of methodological issues, see Cohen et al. 2010). Specifically, in addition to potential biases associated with retrospective reports, some studies do not statistically adjust for adult SES. Thus, associations of childhood SES with adult health may simply be due to correlations between childhood and adult SES. Another issue is identifying the appropriate statistical controls for health. Most studies account for adult health status, for example, smoking, obesity, and physical inactivity, at the start of the follow-up period in adulthood. However, other than birth cohort studies, most investigations do not have

information about childhood health status, and if they do, it is not clear which indicators of child health would provide appropriate controls for the adult health outcomes.

A number of conceptual models have been advanced to understand how SES across the life course might relate to health. The most commonly asserted model (Kuh et al. 2003), called the accumulation model, posits additive effects of exposure to low SES across the life span, i.e., the longer the duration of exposure to low SES, the greater the risk. Strictly speaking, this is not a developmental model because it presumes that life stage of exposure does not matter. A sensitive or critical period model suggests that the timing of exposure to low SES is important for some adult health outcomes and emphasizes early life development. Social mobility, or movement up and down the SES hierarchy within a lifetime, may also impact health. Substantial support has been garnered for life course models in general (see Related Resources), and in particular for the perspective that SES early in life is predictive of later physical health and that exposure to socioeconomic hardship can have a cumulative, dose-response relationship with morbidity and mortality (Carson et al. 2007, Singh-Manoux et al. 2004). However, as noted by other authors, it is extremely difficult to untangle the relative evidence for life course models (Hallqvist et al. 2004), in part due to substantial covariation in both SES and physical health across time and generations.

Psychosocial Perspectives in Life Course Models

From a psychosocial perspective on SES across the life course, the role of early family environment is an obvious place to begin. Repetti et al. (2002) propose that families that transmit high risk for poor health are characterized by conflictual interactions, manifested in recurrent episodes of anger and aggression, and deficient nurturing, resulting in relationships that are cold, unsupportive, and neglectful. Troxel & Matthews (2004) suggest that parental conflict is especially harmful because it leads to decreased monitoring of children, less expressed warmth and affection, and inconsistent communication and discipline style, and because marital dissolution often leads to a decline in SES, with the attendant financial insecurity. Evans and colleagues (2005) point to the role of chaotic, unpredictable family environments as key, which may lead to the greater threat appraisals documented in low-SES children. These types of family environments are less able to provide the kinds of experiences necessary for children to learn how to regulate emotions and to develop a sense of security with and attachment to important figures in their lives. Such deficits are thought to foster a propensity to experience chronic negative affect, to have difficulties in developing or maintaining supportive social networks, and to exhibit elevated SAM and HPA responses to threatening circumstances (Chen & Matthews 2001, Chen et al. 2003, Luecken et al. 2006). To the extent that these experiences occur repeatedly, they are believed to lead to poor health later in life.

This reasoning is consistent with evidence summarized elsewhere (Repetti et al. 2002, Troxel & Matthews 2004). For example, several analyses have explicitly tested the pathways connecting low childhood SES and cardiovascular risk factors in adulthood. Participants completed measures of an adverse early family environment (characterized by harsh, conflictual, and neglectful parenting) and adult psychosocial functioning. Structural equation models showed that low childhood SES was associated with an adverse family environment, which in turn, was related to a latent factor reflecting adult psychosocial functioning (comprising combinations of negative emotions and personal and/or social resources). Adult psychosocial functioning then related to poor metabolic functioning, elevations in C-reactive protein, and prevalence of high blood pressure and increases across five years in blood pressure (Lehman et al. 2009). Although early family environment was assessed retrospectively, these examples demonstrate both the promise of the perspective and the utility of explicitly testing life course models.

Another developmental perspective considers not only early psychosocial factors but also their interaction with adverse physical exposures common in low-SES neighborhoods (Clougherty & Kubzansky 2009). Gump and colleagues (2007) reported that children from lower-SES families exhibited greater cardiovascular reactivity to stress and that the association was mediated by heightened blood lead levels observed in the children, which were correlated with family SES. Among asthmatic children, low neighborhood SES (based on census tract indicators of poverty) was associated with immune markers correlated with asthma; chronic stress, appraisals of perceived threat, and beliefs about control of health mediated these associations (Chen et al. 2003). In another sample of asthmatics, neighborhood problems were related to daytime symptoms during a two-week monitoring period (Chen et al. 2007), and greater chronic family stress was related to both adverse biologic and clinical outcomes among those who lived in areas with low air pollution (Chen et al. 2008). Adolescent blood pressure, overnight catecholamines, and cortisol levels increased with a higher multiple risk factor index composed of housing quality, crowding, noise, family turmoil, child separation from family, and violence (Evans et al. 2007).

That the accumulation of psychosocial and physical risk factors plays a role in understanding SES–health relationships is promising. A large body of research indicates that poverty is related to multiple and diverse types of risk (Evans 2004). For example, among children living in rural areas, those from low- relative to middle-income families were more like to have substandard housing quality, high levels of noise, family turmoil, and exposure to violence (Evans 2004). However, as noted by Evans & Kim (2010), few studies have evaluated whether multiple risk exposures are graded across SES because typically the research using this approach compares only two groups. Taking a multiple risk factor approach has been profitable for understanding children’s emotional and social development, although at present few studies have examined multiple risk factors in the context of physical health.

In summary, life course perspectives have promise in informing the understanding of psychosocial and psychobiological pathways connecting SES and health. Developmental models are admittedly complex, but the focus solely on studies in adulthood misses the early life experiences that may be crucial to later health. Retrospective assessments of early life exposures can be useful additions to adult studies, but the most complete understanding of the life course requires longitudinal studies starting early in life, preferably during gestation. Coordination of study designs with scientists interested in early development may yield long-term benefits.

ALTERNATIVES TO CLINICAL DISEASE OUTCOMES IN STUDIES OF SOCIOECONOMIC STATUS

Given conceptual models suggesting that SES– health associations begin early in life and that risks accumulate within individuals, research does not need to wait until frank disease emerges to identify underlying mechanisms. Furthermore, some mechanisms may be relevant to more than one disease. As such, studies of SES–health gradients have begun to emphasize a number of novel and/or integrative conceptualizations of health and health risk. Pre-clinical endpoints may be useful for research that seeks to examine socioeconomic influences on health, since they can help address concerns regarding reverse causation, or the idea that poor health influences SES in a downward direction (Smith 1999). In addition, integrative models that assess risk across multiple physiological systems may facilitate efforts to determine how psychobiological risks accumulate within individuals and across time. Further, although uncommon, longitudinal studies that examine SES and indicators of health risk or subclinical disease at multiple time points create opportunities to understand how associations among SES, underlying pathways, and health evolve across the life course

and can help establish opportunities for early prevention and intervention efforts. Additional research has focused on how physiological parameters can be measured in the real world to better understand how psychosocial experiences in everyday life influence SES–health associations. Finally, a novel line of research has focused on the neurobiological implications of socioeconomic status.

Subclinical Measures of Atherosclerosis

A variety of methods are available to noninvasively estimate risk for a future cardiovascular event, even in the absence of clinically relevant symptoms or diagnoses. These include ultrasound measures of carotid artery atherosclerosis and computed tomography assessment of calcification in the coronary arteries and other arterial beds. Research using these tools has identified associations between adult, childhood, cumulative, and area-based SES markers with the prevalence or extent of atherosclerosis and with more rapid progression of carotid atherosclerosis (Diez Roux et al. 2005, Gallo et al. 2001, Lemelin et al. 2009). Importantly, null findings have appeared in the literature (Kop et al. 2005), and findings are somewhat varied across ethnic and gender groups (Diez Roux et al. 2005, Ranjit et al. 2006). Although psychosocial factors have also been related to subclinical outcomes in prior research, few studies have specifically addressed the role of these pathways in connecting SES with subclinical CVD. Gallo and colleagues (Gallo et al. 2001) showed that depression and anxiety explained a small amount of shared variance between education and atherosclerotic burden (specifically, aortic calcification) in a cohort of middle-aged women. Left ventricular mass is another marker of risk for later CVD events. In a sample of children and adolescents, neighborhood and family SES was related to greater cardiovascular reactivity to stress, which, in turn, was associated with left ventricular mass (Gump et al. 1999). Given their utility and accessibility, subclinical CVD measures can facilitate efforts to test integrative models of SES, psychobiological pathways, and early markers of CVD risk in future research.

Allostatic Load

The allostatic load model proposes that the stress-related physiological alterations that occur as the body attempts to restore allostasis following stress [i.e., the process of “maintaining stability through change” (McEwen 1998)] can over time lead to dysregulation in multiple interrelated physiological systems. Even if this dysregulation is only moderate, when it occurs across multiple systems, the summative effects are posited to be health damaging. Allostatic load is generally operationalized by a summary index reflecting the number of extreme values on neuroendocrine markers of the SAM and HPA systems and on markers of cardiovascular (e.g., blood pressure) and metabolic (e.g., waist circumference, glucose regulation) functioning—an approach used in the MacArthur Studies of Successful Aging (Karlman et al. 2002). Neuroendocrine markers are considered primary mediators of the cascade of physiological processes that, in turn, foster cardiovascular and metabolic dysfunction (secondary outcomes of allostatic load) and ultimately raise risk for morbidity, mortality, and functional decline (considered tertiary outcomes; McEwen & Seeman 1999). In recent work, elevations of inflammatory markers have been incorporated into models of allostatic load (Seeman et al. 2004).

Various conceptualizations of allostatic load predict risk of CVD, all-cause mortality, physical decline, and cognitive dysfunction (Karlman et al. 2002, 2006; Seeman et al. 1997). In addition, some research suggests that allostatic load is more predictive of varied health outcomes than are the individual risk components it comprises, i.e., the whole is greater than the sum of the parts (Karlman et al. 2002, Seeman et al. 1997), and that psychosocial pathways of interest to SES researchers, such as stress, depression, and social variables, relate to allostatic load (Clark et al. 2007, Seeman et al. 2002). However, this

model is still being tested and elaborated, and important outstanding issues remain to be resolved, e.g., which parameters to use, whether both static and dynamic measures should be included, and how they should be weighted and aggregated. In addition, the longitudinal research that would be necessary to verify sequential associations among stressors, alterations in primary mediators, emerging changes in cardiovascular and metabolic functioning, and distal health outcomes has yet to be performed.

Moreover, although the allostatic model may be a useful approach in efforts to examine how SES-related adversity fosters health risks over time, few studies to date have actually examined associations between SES and allostatic load. A recent review (Dowd et al. 2009) identified seven such studies and described mixed evidence both for the relationship between SES and allostatic load and for the hypothesis that allostatic load mediates associations between SES and various physical health outcomes. For example, in analyses based on the MacArthur Study of Successful Aging (Seeman et al. 2004), allostatic load explained more than one-third of the difference in mortality risk between high- and low-SES groups (with SES represented as a dichotomized index of education). In contrast, a study in Taiwan did not find evidence that allostatic load contributed to associations between SES and health outcomes, such as limitations in activities of daily living, and overall perceived health (Hu et al. 2007). In general, as further described below, Dowd et al. (2009) reported more consistent evidence for associations between SES and cardiovascular and metabolic facets than for relationships with neuroendocrine markers of allostatic load. These findings create doubt concerning the utility of the overall framework, given the primary role neuroendocrine factors are posited to have and the fact that cardiovascular and metabolic risk factors are already well-established predictors of CVD. Subsequent to this review, three papers have been published based on the National Health and Nutrition Examination Survey III. Two reported associations between neighborhood SES and allostatic load (e.g., Merkin et al. 2009), and the third showed that poverty status was associated with allostatic load, but only in participants younger than age 60 (Crimmins et al. 2009). To our knowledge, only one study has examined psychosocial pathways in the SES–allostatic load relationship. Kubzansky and colleagues (1999) found that the association between SES and allostatic load was partially explained by variations in hostile cognition. In sum, the current literature is mixed, and additional research is needed to determine the utility of this framework to understanding SES–health disparities and the roles that psychobiological pathways have in them.

Neuroendocrine Factors—A comprehensive review of the relationship between SES and cortisol in 26 studies concluded that research that takes into account the circadian rhythm of hormones may be most useful (Dowd et al. 2009). Specifically, although average cortisol levels across the 26 studies were unrelated to SES, lower SES was more consistently associated with a blunted pattern of diurnal secretion of salivary cortisol, though whether this corresponded to a higher or lower overall exposure to cortisol varied by study. This is not surprising, given that both hypo- and hyper-secretion of cortisol have been connected with poor health (Heim et al. 2000). Some research also suggests that the pattern of association between SES and cortisol differs by gender. In the Whitehall Study, cortisol output over the day was higher in low-SES men but lower in low-SES women (Steptoe et al. 2003). Additional analyses indicated that, at least in part, these differences might reflect gender by SES-related differences in job stress (i.e., demands and control; Kunz-Ebrecht et al. 2004). Similarly, in the same study, a blunted pattern of salivary cortisol secretion with lower employment grade and wealth was obtained in men but not women (Kumari et al. 2010). These associations were mediated by smoking and sleep duration. Thus, the nature of the relationship between SES and HPA regulation is complex, and moderating factors are an important focus for future research in the area.

Surprisingly few studies are available with regard to associations between SES and SAM activation. In a healthy volunteer sample, the lower a combined SES index based on income and education, the higher the urinary epinephrine and norepinephrine averaged across two 24-hour periods (S. Cohen et al. 2006). Smoking, not eating breakfast, and low social network diversity were the primary mediators of these associations. In the Coronary Artery Risk Development in Young Adults study sample, lower SES, whether measured by education, occupation, or income, was associated with higher overnight levels of norepinephrine and epinephrine (Janicki-Deverts et al. 2007). Smoking and depressive symptoms scores partially accounted for these associations. Among a large population-based sample of Taiwanese middle-aged and older men and women, neither overnight epinephrine nor norepinephrine was related to education or income in men or women, with the exception of an unexpected association of higher education with higher catecholamines in women (Dowd & Goldman 2006). Cultural factors may account for the difference in results across the two studies.

Another concept related to SAM activation is cardiovascular reactivity to psychological stressors, i.e., the magnitude of blood pressure and heart rate change in response to challenge. Individual differences in cardiovascular reactivity to stress are associated with future hypertension and atherosclerosis (Jennings et al. 2004, Matthews et al. 2004). The few studies of SES and reactivity to standardized psychological stressors show inverse associations in most but not all studies, perhaps due to the difficulty of employing tasks that are equally familiar to individuals of all SES levels (Stephoe & Marmot 2002). Interestingly, low SES, in conjunction with cardiovascular reactivity, predicts subclinical atherosclerosis (Lynch et al. 1998). We know of only one study that has traced psychosocial pathways connecting SES to cardiovascular reactivity; the findings indicated that hostility represented an important mediating pathway in black but not white adolescents (Gump et al. 1999). In Whitehall II, low employment grade was associated with low-frequency heart rate variability, an index of parasympathetic activation; job control and behavioral factors statistically explained the association (Hemingway et al. 2005).

Cardiovascular and Metabolic Markers—Additional research has addressed the association between SES and the metabolic syndrome, a well-established aggregation of metabolic and cardiovascular risk factors that cluster together more than expected based on chance alone and the presence of which portends high risk for negative health outcomes including CVD, diabetes, kidney disease, and all-cause mortality (Gami et al. 2007). Although measurements of the metabolic syndrome vary, it is generally defined as three or more of the following: elevated fasting glucose (or medicated for diabetes), waist circumference, and blood pressure (or medicated for hypertension), and lipid dysregulation, including lowered high-density lipoprotein cholesterol or elevated triglycerides (or medication for the same).

A large body of research shows that individuals with low SES are at high risk for the metabolic syndrome. This relationship holds for diverse measures of SES (Carnethon et al. 2004, Chichlowska et al. 2008, Manuck et al. 2010). Null findings have also appeared in the literature (Lucove et al. 2007), and the SES gradient may be stronger in women than in men, although some evidence has supported the association in men as well. Among other variables, health behaviors may contribute to social distributions of metabolic syndrome, particularly given the close ties to obesity, caloric intake, and physical activity (Ramsay et al. 2008). Furthermore, research has shown that psychosocial factors relate to metabolic syndrome (Goldbacher & Matthews 2007) and that they may be important factors connecting low SES with metabolic syndrome risk (Gallo et al. 2007, Lehman et al. 2005, Matthews et al. 2008), as elaborated elsewhere in this review.

AMBULATORY MEASURES OF BIOMARKERS

Additional research concerning psychobiological pathways measures standard biomarkers in home, work, and school environments as opposed to the laboratory or clinic. Advantages include greater ecological validity, although at the same time, the technologies can influence the processes being observed, and reliability could be reduced due to measurement in uncontrolled settings. One example of this approach is studies that measure levels of stress hormones in saliva across the day in relation to HPA axis research. Salivary measurements have been used to measure other hormones as well and may lead to an expansion of the types of biomarkers that can be studied in an ambulatory context of varying SES groups.

Other studies have focused on ambulatory measures of blood pressure and heart rate to evaluate cardiovascular responses in everyday life. Research suggests that 24-hour ambulatory blood pressure is a better predictor of clinical cardiovascular events than is clinic/doctor's office blood pressure (Conen & Bamberg 2008). Moreover, high nighttime blood pressure or a high night/day ratio of blood pressure is particularly prognostic of future events, especially among hypertensives (Fagard et al. 2008). When synchronized with diary entries of mood and stress, ambulatory data can be used to test specific hypotheses about the types of events that trigger elevations and in which individuals acute rises in blood pressure and heart rate are most likely to occur. Several studies that classified individuals by occupational groups showed that lower-SES individuals have elevated daytime blood pressure or heart rate (Gallo et al. 2004, Matthews et al. 2000a, Steptoe et al. 2003). Adolescents who are from lower-income neighborhoods have higher ambulatory blood pressure and heart rate than those from higher-income neighborhoods. Moreover, among adolescents from poorer families or neighborhoods, negative mood experienced at the time of the blood pressure assessment is related to higher blood pressure levels (McGrath et al. 2006). Furthermore, individuals with lower SES fail to exhibit the nighttime fall in blood pressure that is otherwise health protective (Campbell et al. 2008). However, associations are not always found in the expected direction. In a convenience sample of non-Hispanic whites and Hispanics, 24-hour blood pressure was higher in less-educated whites but lower in less-educated Hispanics (Steffen 2006). Steffan suggested that a lack of "Anglo" acculturation (i.e., low acculturation to mainstream U.S. culture) accounted for the unexpected results observed in Hispanics.

One possible explanation for the failure of blood pressure to fall at night in lower-SES individuals is sleep disruption, which has been proposed as a potential mediator of SES–health relationships. Sleep characteristics can also be measured reliably in the home by actigraphy or polysomnography (PSG) in combination with sleep logs. Actigraphy measures movement and infers sleep in the absence of significant movement; PSG bases its assessment of sleep on brain activity and respiration. In two multiethnic samples of middle-aged adults, lower individual SES related to less-efficient sleep (taking longer to fall asleep or being awake longer after sleep onset) based on actigraphy (Lauderdale et al. 2006, Mezick et al. 2008) and PSG (Mezick et al. 2008). Self-reported financial strain was also related to sleep inefficiency in a multiethnic sample of women (Hall et al. 2009). This pattern of results is consistent with the less-efficient sleep measured in the clinic setting among lower-income women (Friedman et al. 2007) and with self-reported poorer sleep quality shown in low-SES persons in a number of studies (Mezick et al. 2008, Sekine et al. 2006). It is noteworthy that this body of work has also revealed striking ethnic differences in sleep, with African Americans in general having shorter and less in-home sleep compared to non-Hispanic whites, usually independent of SES (Hall et al. 2009, Lauderdale et al. 2006, Mezick et al. 2008).

OTHER BIOMARKERS

Inflammation/Immune Function

Markers of inflammation and immune functioning are believed to be key processes in many infectious illnesses and chronic diseases. In addition, a growing body of literature links varied indicators of SES with inflammatory markers (for reviews, see Aiello & Kaplan, 2009 and Nazmi & Victora 2007), although some variability across age and ethnic groups has been noted (Gruenewald et al. 2009, Rathmann et al. 2006). A comparison of healthy adults matched on current SES who came from high- versus low-SES families of origin revealed that early-life low SES was associated with resistance to glucocorticoid signaling, which in turn facilitated exaggerated adrenocortical and inflammatory responses (Miller et al. 2009). This suggests a root biological pathway that develops early in life and may underlie the link between SES and inflammatory responses. Although the majority of the research in this area has focused on inflammatory markers, Cohen and his colleagues (2004) found that adults who experienced lower SES in childhood displayed decreased resistance to upper respiratory infections. Other research has identified socioeconomic gradients in seroprevalence of single pathogens and overall burden of pathogens that have previously been linked with CVD risk (Steptoe et al. 2007, Zajacova et al. 2009).

Although studies have also demonstrated that various psychological and social factors of interest in health disparities research relate to markers of inflammation and immune functioning (McDade et al. 2006, Ranjit et al. 2007), little research has explored psychosocial pathways in relation to SES. One study found that variables such as chronic stress, depression, and social support explained minimal covariation between SES and CRP and IL-6; behavioral variables such as smoking, BMI, and sleep quality seemed to play more important roles (Gruenewald et al. 2009). Additional research is needed, and although still in its early stages, this area of work provides a promising avenue for research that seeks to explore psychobiological mechanisms in the SES–health gradient.

Brain

Ultimately the brain must play a central role in understanding how SES influences physical health, and studies that take advantage of new methods to measure brain structure and function have promise (McEwen 2007). Brain regions relevant to psychological perspectives on SES include those involved in regulation of central and peripheral nervous system functions important for mood regulation, threat and award appraisals, and adaptation to chronic and repeated stressors. These areas include the hippocampus, hypothalamus, and prefrontal cortex and their subunits (Hackman & Farah 2009). With regard to brain structure, compared to healthy adults with higher scores on subjective SES, those with lower scores had reduced gray matter volume in the rostral area of the anterior cingulate cortex, a corticolimbic brain region involved in experiencing emotion and regulating cardiovascular reactivity to psychological stress (Gianaros et al. 2007a). Interestingly, in the same sample, individual-level (education, income) and community-level SES measures were unrelated to gray matter volume in the anterior cingulate. Healthy adults who had more adverse childhood events had smaller anterior cingulate cortex volumes (R.A. Cohen et al. 2006), and healthy elderly women with a history of chronic stress had smaller hippocampal volume (Gianaros et al. 2007b). With regard to brain function, healthy young adults whose parents had lower SES had greater amygdala activity in response to angry faces than did those whose parents had higher SES, whereas there were no differences in response to neutral or surprised faces (Gianaros et al. 2008). These findings were independent of the participants' own subjective SES. Taken together, these data suggest that SES may be related to structure and function of specific brain regions involved in emotional regulation and appraisal of

stressful circumstances, but that the effects of SES vary according to the specific measure being examined.

Another relevant line of research has focused on associations between SES and brain neurotransmitters, especially those related to health outcomes. One illustration of this approach examines the monoamine neurotransmitter, serotonin. Serotonin-releasing neurons originate in the raphe nuclei of the midbrain and project to nearly all areas of the central nervous system, including the cerebral cortex and its subunits. Research spearheaded by Manuck, Muldoon, and their colleagues shows that individuals low in central serotonergic responsivity are at elevated risk for hostile attributes, central adiposity, high blood pressure, metabolic syndrome, and carotid atherosclerosis (Manuck et al. 2006, Muldoon et al. 2004). Furthermore, healthy adults with less education and lower income have attenuated serotonergic responsivity (Matthews et al. 2000b), as do adults who reside in census tracts that have greater poverty and unemployment, lower-priced housing, and lower median education (Manuck et al. 2005). The latter effects for census tract remained after statistical adjustments for individual-level SES. Although few studies examine direct neurobiological pathways in relation to SES to date, this research area has potential.

CHALLENGES TO PSYCHOLOGICAL THEORIES OF SOCIOECONOMIC STATUS AND HEALTH

Demographic Variables as Moderators

We have painted a picture suggesting that SES is robustly related to health across many populations and settings. Although this is true, psychological perspectives must also take into account the variability in associations as models become more sophisticated. For example, a number of studies suggest that SES gradients in health outcomes such as mortality (Marmot & Shipley 1996) and biological risk (Crimmins et al. 2009) diminish in older ages. This pattern may be explained to some extent by survivor biases, whereby the remaining population (i.e., those who have survived into old age) is an especially resilient group of individuals who may be less vulnerable to the detrimental health effects of low SES. In addition, this trend may be most evident in relation to specific SES indicators (e.g., income, occupational status) with less relevance in the elderly. Research in younger populations also suggests that associations between SES and overall health remain relatively constant, whereas relationships of SES with specific health outcomes vary according to developmental phase (Chen et al. 2006).

Additional research suggests that the nature of SES–health disparities may be stronger for one gender than another. For example, steeper gradients were observed in some studies for women for CVD (Thurston et al. 2005), diabetes (Maty et al. 2008), and biological risk profiles (Phillips et al. 2009). On the other hand, the association between education and all-cause mortality may actually be larger in men than in women, although this difference may depend on contextual factors such as marital status (Montez et al. 2009). To the extent that there are gender differences in SES gradients for major health outcomes, they may reflect a similar social patterning of risk factor differences, e.g., the more salient association between SES and obesity in adult women than in men (McLaren 2007), resulting in a larger SES–CVD or SES–diabetes association, and a stronger contribution of smoking-related deaths to educational–mortality gradients in men than in women (Montez et al. 2009). Social influences may also contribute to these patterns. For instance, SES–mortality gradients could be steeper in men because they experience clearer gains in standard of living with higher educational attainment or occupational status relative to women. Conversely, women with low SES may experience compounded stress created by leading single-parent households or through social stressors such as domestic violence or gender discrimination.

Given the complexities involved and the fact that gender differences in SES disparities may depend on selected SES markers, specific health outcomes of interest, age of participants, and marital status (for discussion, see Thurston et al. 2005), the general recommendation is that researchers should consider gender a possible moderating factor and where appropriate consider different psychosocial pathways.

SES–health differentials have also varied by race and ethnicity (Smith 2000). From a conceptual perspective, this variability is not surprising. It seems logical that ethnic minority groups with low SES may experience a multiplicative health burden, reflecting a disproportionate burden of stress due to ethnic discrimination, residential segregation, etc., resulting in worse health outcomes at low SES levels for ethnic minorities than for whites and fostering steeper gradients. These trends may be especially salient for the most historically disadvantaged groups, such as African Americans (Williams & Jackson 2005). However, ethnic minorities might also experience “diminishing returns” or a “glass ceiling” effect that negates the health benefits associated with high socioeconomic attainment, leading to flattened gradients (Myers 2009). The situation is further complicated when research concerns ethnic minority populations with substantial numbers of immigrants, given intricate associations between health and migration trends. Moreover, sociocultural resources or protective behavioral tendencies could protect some ethnic or immigrant groups from the health impact of low SES, and immigrants may also demonstrate the socioeconomic health gradients of their countries of origin, which may be reversed in undeveloped settings. Indeed, SES–health gradients have been found to be flattened or inconsistent within and among Hispanic and Asian populations. The ability to untangle the influences of SES and ethnicity/race on health is complicated by the substantial covariation between these demographic constructs and by variability in the meaning and implications of SES indicators across ethnic and national origins groups, as discussed elsewhere (e.g., Krieger et al. 1997). Overall, it seems important to consider SES and ethnicity concurrently and for possible ethnic differences in SES gradients to be routinely explored. In addition, national origins must be attended to, since substantial heterogeneity is likely when large pan-ethnic groupings (e.g., Asian, Latino) are examined.

Study Design Issues

Most research concerning SES and health has been observational in design. By their nature, these studies cannot establish causation. Especially in the mental health arena, researchers have argued that health determines the ability to achieve and sustain a high SES. Observational studies, even if they are longitudinal, are not typically designed to evaluate the individual components of SES or to take a developmental perspective. Moreover, rarely do the studies allow for detailed assessments of psychobiological pathways. In consequence, strong conclusions are not easily drawn.

The most rigorous study designs are randomly assigned experiments or interventions. Experimental approaches can be useful to evaluate short-term effects of changes in rank or in access to resources on intermediate biomarkers of health. For example, experimentally induced subordinate or superior status can aid in understanding short-term changes in some of the proposed pathways. Several studies have used this approach. Induced subordinate status led to increases in negative affect and greater blood pressure reactivity to stressors in prior research (Mendelson et al. 2008, Mendes et al. 2001). A brain-imaging study showed that viewing a higher-status in comparison with a lower-status individual led to greater activation in the dorsolateral prefrontal cortex while playing an interactive economic game (Zink et al. 2008). In a condition where relative status was updated throughout the game, i.e., unstable status condition, showing a higher-status individual to the participant led to increases in activity in the right amygdala, posterior cingulate, and medial prefrontal cortex, which were not observed in the stable status condition.

Manipulating exposures to the types of situations thought to covary with socioeconomic environments may be fruitful as well. For example, in manipulated conditions where adolescents were provided information that would improve their performance on stressful tasks versus control and decision making over aspects of the tasks, findings showed that having information and control related to lower cardiovascular responses during stressful tasks, but only among adolescents from low-SES families (Chen 2007). Furthermore, in this study, having information was more important than having control in reducing cardiovascular reactivity. Another illustration comes from work on threat appraisals as a mediating factor. The rationale underlying this work is that lower-SES environments expose individuals to more events that are potentially or actually harmful. An adaptive response to such environments is to be vigilant for possible threat. This perspective also posits that to the extent that individuals perceive or anticipate threat in their environments, even where the situations are ambiguous, greater physiological costs result. To test these ideas, vignettes have been prepared where negative outcomes or ambiguous outcomes to social situations are presented, and individuals are interviewed about their interpretations of the events. In children and adults, lower-SES individuals make more appraisals of threat in ambiguous situations than do higher-SES individuals. Furthermore, the threat appraisals lead to greater physiological reactivity to stressors in laboratory and field experiments (Chen & Matthews 2001, Flory et al. 1998).

Natural experiments can also be informative. In a longitudinal study of the mental health of rural children, a local casino opened and hired a substantial number of unemployed American Indians in the community. Children who moved out of poverty over time due to parental hiring showed decreased signs of antisocial behavior (Costello et al. 2003). Although no physical health data were reported, this type of study demonstrates the utility of taking advantage of available naturalistic experiments. Such an opportunity is available currently, with the downturn in the economy and its impact on families. These examples demonstrate new ways to test psychological theory that have been underutilized.

CONCLUSIONS AND DIRECTIONS FOR FUTURE RESEARCH

The field of health psychology has much to offer to the understanding of how SES leads to poor health, and in this section, we summarize several key points and suggest future directions for research. As we have reviewed, SES is a rich concept that comprises not one construct, but many. SES may be viewed at the individual, family, and neighborhood levels; it may be conceptualized objectively or subjectively. SES may also be viewed in a more dynamic manner, i.e., by examining changes or variability over the life course. That variability in the SES gradient differs by age, gender, and ethnicity suggests that multiple approaches to measurement of SES would be useful. For example, subjective SES measures may be informative in minority groups where there are barriers to achieving higher education or attaining a very prestigious occupation. Neighborhood SES may be helpful in areas where the in- and out-migration is relatively minimal. We recommend that psychologists use multiple measures of SES to examine which aspects are more important for health, e.g., resources versus rank, and to better explore potential psychosocial mediators. In this way, more accurate theoretical models can be developed.

The life course perspective on SES is supported by a great deal of empirical evidence. It is clear that lower SES in childhood has a long-lasting influence into adulthood, even if the factors underlying these trends remain unclear. The current literature is sufficiently consistent to suggest that efforts to understand the influence of SES in adulthood must take into account the family origins of participants, or better yet, measure prospectively exposures to low SES throughout the life course. Developmental psychology has amassed a strong literature concerning the influence of family, neighborhood, and school environments

on the development of children's emotional, cognitive, and social skills (Bradley & Corwyn 2002). This research provides clues about the types of environments that may lead to poor health in later life and could assist in the design and testing of developmental hypotheses.

Conceptual models suggesting that SES–health associations begin early in life and that risks are cumulative illustrate the need to move beyond research that focuses on adult SES in relation to a single clinical disease. Novel measures of health and health risk are a useful adjunct to traditional approaches to studying SES–health gradients. We have reviewed evidence suggesting that SES is connected to subclinical CVD, metabolic syndrome, ambulatory blood pressure, sleep, inflammation, and immune function. Stress hormones are less clearly linked to SES. We also described the few studies concerning SES and brain morphology and function, which are important given that ultimately the brain must be involved in understanding psychosocial pathways. Currently very few of these investigations have included the next step of examining psychosocial mediators. Nonetheless, these outcomes would be well suited to serve as surrogate endpoints in experimental and longitudinal designs that examine individuals prior to the usual onset of chronic illnesses.

Despite strong interest, there are relatively few reports on psychosocial pathways connecting SES and health. The fairly minimal available evidence does suggest that a lack of positive interpersonal and intrapersonal resources in individuals with low SES may be a pathway, whereas less evidence supports the role of stress. We suggest that the weaker data for stress may be due to measurement issues, since stress exposures and responses may not be adequately captured through general measures of perceived stress or life events or that the specific types of stress exposures and responses differ by SES. Nonetheless, the more general point is that the field requires additional studies with multiple measures of SES to better establish the role of psychosocial factors. Fortunately, many large data sets are becoming publicly available and have at least some, albeit limited, psychosocial measures.

We have not touched upon the fact that beyond theories of stress and emotion, other psychological theories are relevant to understanding the pathways connecting SES and health. For example, theories of social power (control of others), social comparison (how one views oneself relative to others), social exclusion, and feelings of rejection by the majority are based on a large literature and are relevant to SES mechanisms. Although most of these theories have appropriately focused on cognitive and social skills, motivation, and social relationships as outcomes, they have the potential to provide more detailed information on the processes by which low SES impacts physical health.

We have highlighted a number of integrative approaches: the concept of allostatic load, indices that combine exposures to psychosocial and physical stressors, models of reserve capacity with the elaboration of cultural reserves, and early family environments described as risky, chaotic, and conflictual. Furthermore, research that incorporates experimental approaches or that takes advantage of natural experiments should yield important data. Incorporation of biomarkers into theory-based experimental research would demonstrate their utility more directly.

On the basis of existing evidence summarized here, we suggest that the study of the pathways connecting SES and health from a psychological perspective is promising. Associations between the candidate variables and either SES or health have been demonstrated. The field is now establishing which candidate variables may serve as mediators and which have limited utility. It has identified some novel biomarkers and approaches. Although more work is needed, it is clear that psychosocial factors are an important focus in efforts to understand the influence of SES on health. We need to continue to make progress so that the next steps can be pursued; i.e., designing and testing

interventions based on understanding psychosocial pathways so that the health disparities associated with SES can be ameliorated. Such efforts will require expanding the knowledge base and training health psychologists in other relevant disciplines to facilitate a transdisciplinary approach. These are exciting times for scientists who are or will become interested in unraveling the mystery of the gradient.

SUMMARY POINTS

1. SES is a multifaceted concept that can be measured at the individual, family, and community levels.
2. It should not be assumed that the associations between SES and health are the same in populations that vary by age, gender, and race/ethnicity.
3. SES begins to exert an influence early in life, with effects extending into adulthood.
4. The relatively few reports on psychosocial pathways suggest that a lack of positive interpersonal and intrapersonal resources in individuals with low SES may be a pathway connecting SES and health. There is less evidence supporting stress and stress hormones as a pathway.
5. Integrative approaches to understanding SES-related health disparities are being tested and refined: the concept of allostatic load, indices that combine exposures to psychosocial and physical stressors, model of reserve capacity with the elaboration of cultural reserves, and early risky family environments.
6. Novel measures of biomarkers, health, and health risk, e.g., subclinical CVD, sleep, inflammation, and brain function, are associated with SES and constitute a useful adjunct to traditional approaches of identifying pathways to frank disease.
7. Experimental approaches can facilitate testing the application of relevant psychological theory to inform factors underlying the SES gradient.

FUTURE ISSUES

1. Examine multiple facets of SES simultaneously to better understand their independent and interactive relationships with health.
2. Develop and examine models of SES and health that accommodate potential differences according to age, gender, and race/ethnicity.
3. Formulate and explicitly test integrative models so that they can be refuted or refined by evidence.
4. Mine population-based studies for evaluation of psychobiological pathways.
5. Employ different, more comprehensive approaches to stress assessment, given the weak evidence in the current literature concerning stress as a pathway in SES and health.
6. Develop and test evidence-based interventions to reduce health-damaging psychobiological processes and to enhance health-promoting processes involved in the SES-health links.

Glossary

SES	socioeconomic status
CVDs	cardiovascular diseases
SAM	sympathetic-adrenal-medullary
HPA	hypothalamic pituitary adrenal
PSG	polysomnography

Acknowledgments

We regret that many scientists whose work is relevant to this review were not cited because of mandatory space limitations. We extend our thanks to Sheldon Cohen and Susan Everson-Rose for commenting on an earlier version of this review and to members of the MacArthur Foundation Network on Socioeconomic Status and Health for their collegueship. This work was supported in part by NIH grants HL076852, HL076858, HL025767, HL081604, and HL101649.

LITERATURE CITED

- Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, et al. Socioeconomic status and health. The challenge of the gradient. *Am. Psychol.* 1994; 49:15–24. [PubMed: 8122813]
- Adler NE, Epel ES, Castellazzo G, Ickovics JR. Relationship of subjective and objective social status with psychological and physiological functioning: preliminary data in healthy white women. *Health Psychol.* 2000; 19(6):586–592. [PubMed: 11129362]
- Aiello AE, Kaplan GA. Socioeconomic position and inflammatory and immune biomarkers of cardiovascular disease: applications to the panel study of income dynamics. *Biodemogr. Soc. Biol.* 2009; 55:178–205.
- Almeida DM, Neupert SD, Banks SR, Serido J. Do daily stress processes account for socioeconomic health disparities? *J. Gerontol. B Psychol. Sci. Soc. Sci.* 2005; 60(Spec. No. 2):34–39. [PubMed: 16251588]
- Avendano M, Kawachi I, Van LF, Boshuizen HC, Mackenbach JP, et al. Socioeconomic status and stroke incidence in the US elderly: the role of risk factors in the EPESE study. *Stroke.* 2006; 37(6): 1368–1373. [PubMed: 16690902]
- Bosma H, Schrijvers C, Mackenbach JP. Socioeconomic inequalities in mortality and importance of perceived control: cohort study. *Br. Med. J.* 1999; 319(7223):1469–1470. [PubMed: 10582929]
- Bosma H, Van Jaarsveld CH, Tuinstra J, Sanderman R, Ranchor AV, et al. Low control beliefs, classical coronary risk factors, and socio-economic differences in heart disease in older persons. *Soc. Sci. Med.* 2005; 60(4):737–745. [PubMed: 15571892]
- Bradley RH, Corwyn RF. Socioeconomic status and child development. *Annu. Rev. Psychol.* 2002; 53:371–399. [PubMed: 11752490]
- Braveman PA, Cubbin C, Egerter S, Chideya S, Marchi KS, et al. Socioeconomic status in health research: One size does not fit all. *JAMA.* 2005; 294(22):2879–2888. [PubMed: 16352796]
- Campbell TS, Key BL, Ireland AD, Bacon SL, Ditto B. Early socioeconomic status is associated with adult nighttime blood pressure dipping. *Psychosom. Med.* 2008; 70(3):276–281. [PubMed: 18256336]
- Carnethon MR, Loria CM, Hill JO, Sidney S, Savage PJ, Liu K. Risk factors for the metabolic syndrome: the Coronary Artery Risk Development in Young Adults (CARDIA) study, 1985–2001. *Diabetes Care.* 2004; 27(11):2707–2715. [PubMed: 15505009]
- Carson AP, Rose KM, Catellier DJ, Kaufman JS, Wyatt SB, et al. Cumulative socioeconomic status across the life course and subclinical atherosclerosis. *Ann. Epidemiol.* 2007; 17(4):296–303. [PubMed: 17027292]
- Chaix B, Isacson SO, Rastam L, Lindstrom M, Merlo J. Income change at retirement, neighbourhood-based social support, and ischaemic heart disease: results from the prospective cohort study “Men born in 1914”. *Soc. Sci. Med.* 2007; 64(4):818–829. [PubMed: 17126973]

- Chen E. The impact of socioeconomic status on physiological health: an experimental manipulation of psychosocial factors. *Psychosom. Med.* 2007; 69:348–355. [PubMed: 17510293]
- Chen E, Chim LS, Strunk RC, Miller GE. The role of the social environment in children and adolescents with asthma. *Am. J. Respir. Crit. Care Med.* 2007; 176(7):644–649. [PubMed: 17556714]
- Chen E, Fisher EB, Bacharier LB, Strunk RC. Socioeconomic status, stress, and immune markers in adolescents with asthma. *Psychosom. Med.* 2003; 65(6):984–992. [PubMed: 14645776]
- Chen E, Martin AD, Matthews KA. Socioeconomic status and health: Do gradients differ within childhood and adolescence? *Soc. Sci. Med.* 2006; 62(9):2161–2170. [PubMed: 16213644]
- Chen E, Matthews KA. Cognitive appraisal biases: an approach to understanding the relation between socioeconomic status and cardiovascular reactivity in children. *Ann. Behav. Med.* 2001; 23(2): 101–111. [PubMed: 11394551]
- Chen E, Matthews KA, Boyce WT. Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychol. Bull.* 2002; 128(2):295–329. [PubMed: 11931521]
- Chen E, Schreier HM, Strunk RC, Brauer M. Chronic traffic-related air pollution and stress interact to predict biologic and clinical outcomes in asthma. *Environ. Health Perspect.* 2008; 116(7):970–975. [PubMed: 18629323]
- Chichlowska KL, Rose KM, Diez-Roux AV, Golden SH, McNeill AM, Heiss G. Individual and neighborhood socioeconomic status characteristics and prevalence of metabolic syndrome: the Atherosclerosis Risk in Communities (ARIC) Study. *Psychosom. Med.* 2008; 70(9):986–992. [PubMed: 18799428]
- Clark MS, Bond MJ, Hecker JR. Environmental stress, psychological stress and allostatic load. *Psychol. Health Med.* 2007; 12(1):18–30. [PubMed: 17129930]
- Clougherty JE, Kubzansky LD. A framework for examining social stress and susceptibility to air pollution in respiratory health. *Environ. Health Perspect.* 2009; 117(9):1351–1358. [PubMed: 19750097]
- Cohen RA, Grieve S, Hoth KF, Paul RH, Sweet L, et al. Early life stress and morphometry of the adult anterior cingulate cortex and caudate nuclei. *Biol. Psychiatry.* 2006; 59(10):975–982. [PubMed: 16616722]
- Cohen S, Alper CM, Doyle WJ, Adler N, Treanor JJ, Turner RB. Objective and subjective socioeconomic status and susceptibility to the common cold. *Health Psychol.* 2008; 27(2):268–274. [PubMed: 18377146]
- Cohen S, Doyle WJ, Baum A. Socioeconomic status is associated with stress hormones. *Psychosom. Med.* 2006; 68(3):414–420. [PubMed: 16738073]
- Cohen S, Doyle WJ, Turner RB, Alper CM, Skoner DP. Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosom. Med.* 2004; 66(4):553–558. [PubMed: 15272102]
- Cohen S, Janicki-Deverts D, Chen E, Matthews KA. Childhood socioeconomic status and adult health. *Ann. N.Y. Acad. Sci.* 2010; 1186:37–55. [PubMed: 20201867]
- Cohen S, Janicki-Deverts D, Miller GE. Psychological stress and disease. *JAMA.* 2007; 298:1685–1687. [PubMed: 17925521]
- Conen D, Bamberg F. Noninvasive 24-h ambulatory blood pressure and cardiovascular disease: a systematic review and meta-analysis. *J. Hypertens.* 2008; 26(7):1290–1299. [PubMed: 18550999]
- Costello EJ, Compton SN, Keller G, Angold A. Relationships between poverty and psychopathology: a natural experiment. *JAMA.* 2003; 290:2023–2029. [PubMed: 14559956]
- Crimmins EM, Kim JK, Seeman TE. Poverty and biological risk: the earlier “aging” of the poor. *J. Gerontol. A Biol. Sci. Med. Sci.* 2009; 64(2):286–292. [PubMed: 19196637]
- Diez Roux AV. Investigating neighborhood and area effects on health. *Am. J. Public Health.* 2001; 91(11):1783–1789. [PubMed: 11684601]
- Diez Roux AV, Detrano R, Jackson S, Jacobs DR Jr, Schreiner PJ, et al. Acculturation and socioeconomic position as predictors of coronary calcification in a multiethnic sample. *Circulation.* 2005; 112(11):1557–1565. [PubMed: 16144996]
- Dowd JB, Goldman N. Do biomarkers of stress mediate the relation between socioeconomic status and health? *J. Epidemiol. Community Health.* 2006; 60(7):633–639. [PubMed: 16790837]

- Dowd JB, Simanek AM, Aiello AE. Socioeconomic status, cortisol and allostatic load: a review of the literature. *Int. J. Epidemiol.* 2009; 38(5):1297–1309. [PubMed: 19720725]
- Due P, Lynch J, Holstein B, Modvig J. Socioeconomic health inequalities among a nationally representative sample of Danish adolescents: the role of different types of social relations. *J. Epidemiol. Community Health.* 2003; 57:692–698. [PubMed: 12933775]
- Evans GW. The environment of childhood poverty. *Am. Psychol.* 2004; 59(2):77–92. [PubMed: 14992634]
- Evans GW, Gonnella C, Marcynyszyn LA, Gentile L, Salpekar N. The role of chaos in poverty and children's socioemotional adjustment. *Psychol. Sci.* 2005; 16(7):560–565. [PubMed: 16008790]
- Evans GW, Kim P. Multiple risk exposure as a potential explanatory mechanism for the socioeconomic status-health gradient. *Ann. N.Y. Acad. Sci.* 2010; 1186:174–189. [PubMed: 20201873]
- Evans GW, Kim P, Ting AH, Tesher HB, Shannis D. Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Dev. Psychol.* 2007; 43(2):341–351. [PubMed: 17352543]
- Everson-Rose SA, Lewis TT. Psychosocial factors and cardiovascular diseases. *Annu. Rev. Public Health.* 2005; 26:469–500. [PubMed: 15760298]
- Fagard RH, Celis H, Thijs L, Staessen JA, Clement DL, et al. Daytime and nighttime blood pressure as predictors of death and cause-specific cardiovascular events in hypertension. *Hypertension.* 2008; 51:55–61. [PubMed: 18039980]
- Flory JD, Matthews KA, Owens JF. A social information processing approach to dispositional hostility: relationships with negative mood and blood pressure elevations at work. *J. Soc. Clin. Psychol.* 1998; 17:491–504.
- Friedman EM, Love GD, Rosenkranz MA, Urry HL, Davidson RJ, et al. Socioeconomic status predicts objective and subjective sleep quality in aging women. *Psychosom. Med.* 2007; 69(7):682–691. [PubMed: 17766692]
- Gallo LC, Bogart LM, Vranceanu AM, Matthews KA. Socioeconomic status, resources, psychological experiences, and emotional responses: a test of the reserve capacity model. *J. Personal. Soc. Psychol.* 2005; 88(2):386–399.
- Gallo LC, Bogart LM, Vranceanu AM, Walt LC. Job characteristics, occupational status, and ambulatory cardiovascular activity in women. *Ann. Behav. Med.* 2004; 28(1):62–73. [PubMed: 15249260]
- Gallo LC, de Los Monteros KE, Ferent V, Urbina J, Talavera G. Education, psychosocial resources, and metabolic syndrome variables in Latinas. *Ann. Behav. Med.* 2007; 34(1):14–25. [PubMed: 17688393]
- Gallo LC, Matthews KA. Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychol. Bull.* 2003; 129(1):10–51. [PubMed: 12555793]
- Gallo LC, Matthews KA, Kuller LH, Sutton-Tyrrell K, Edmundowicz D. Educational attainment and coronary and aortic calcification in postmenopausal women. *Psychosom. Med.* 2001; 63(6):925–935. [PubMed: 11719631]
- Galobardes B, Lynch JW, Davey-Smith G. Is the association between childhood socioeconomic circumstances and cause-specific mortality established? Update of a systematic review. *J. Epidemiol. Community Health.* 2008; 62(5):387–390. [PubMed: 18413449]
- Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Ann. Epidemiol.* 2006; 16:91–104. [PubMed: 16257232]
- Gami AS, Witt BJ, Howard DE, Erwin PJ, Gami LA, et al. Metabolic syndrome and risk of incident cardiovascular events and death: a systematic review and meta-analysis of longitudinal studies. *J. Am. Coll. Cardiol.* 2007; 49:403–414. [PubMed: 17258085]
- Geronimus AT, Bound J. Use of census-based aggregate variables to proxy for socioeconomic group: evidence from national samples. *Am. J. Epidemiol.* 1998; 148:475–486. [PubMed: 9737560]

- Gianaros PJ, Horenstein JA, Cohen S, Matthews KA, Brown SM, et al. Perigenual anterior cingulate morphology covaries with perceived social standing. *Soc. Cogn. Affect. Neurosci.* 2007a; 2(3): 161–173. [PubMed: 18418472]
- Gianaros PJ, Horenstein JA, Hariri AR, Sheu LK, Manuck SB, et al. Potential neural embedding of parental social standing. *Soc. Cogn. Affect. Neurosci.* 2008; 3(2):91–96. [PubMed: 18594696]
- Gianaros PJ, Jennings JR, Sheu LK, Greer PJ, Kuller LH, Matthews KA. Prospective reports of chronic life stress predict decreased gray matter volume in the hippocampus. *NeuroImage.* 2007b; 35:795–802. [PubMed: 17275340]
- Goldbacher EM, Matthews KA. Are psychological characteristics related to risk of the metabolic syndrome? A review of the literature. *Ann. Behav. Med.* 2007; 34(3):240–252. [PubMed: 18020934]
- Gruenewald TL, Cohen S, Matthews KA, Tracy R, Seeman TE. Association of socioeconomic status with inflammation markers in black and white men and women in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Soc. Sci. Med.* 2009; 69(3):451–459. [PubMed: 19524346]
- Grzywacz JG, Almeida DM, Neupert SD, Ettner SL. Socioeconomic status and health: a microlevel analysis of exposure and vulnerability to daily stressors. *J. Health Soc. Behav.* 2004; 45(1):1–16. [PubMed: 15179904]
- Gump BB, Matthews KA, Räikkönen K. Modeling relationships among socioeconomic status, hostility, cardiovascular reactivity, and left ventricular mass in African American and white children. *Health Psychol.* 1999; 18(2):140–150. [PubMed: 10194049]
- Gump BB, Reihman J, Stewart P, Lonky E, Darvill T, Matthews KA. Blood lead (Pb) levels: a potential environmental mechanism explaining the relation between socioeconomic status and cardiovascular reactivity in children. *Health Psychol.* 2007; 26(3):296–304. [PubMed: 17500616]
- Hackman DA, Farah MJ. Socioeconomic status and the developing brain. *Trends Cogn. Sci.* 2009; 13(2):65–73. [PubMed: 19135405]
- Hall MH, Matthews KA, Kravitz HM, Gold EB, Buysse DJ, et al. Race and financial strain are independent correlates of sleep in midlife women: the SWAN Sleep Study. *Sleep.* 2009; 32(1):73–82. [PubMed: 19189781]
- Hallqvist J, Lynch J, Bartley M, Lang T, Blane D. Can we disentangle life course processes of accumulation, critical period and social mobility? An analysis of disadvantaged socio-economic positions and myocardial infarction in the Stockholm Heart Epidemiology Program. *Soc. Sci. Med.* 2004; 58(8):1555–1562. [PubMed: 14759698]
- Hatch SL, Dohrenwend BP. Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and age: a review of the research. *Am. J. Community Psychol.* 2007; 40(3–4):313–332. [PubMed: 17906927]
- Heim C, Ehler U, Hellhammer DH. The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology.* 2000; 25:1–35. [PubMed: 10633533]
- Hemingway H, Shipley M, Brunner E, Britton A, Malik M, Marmot M. Does autonomic function link social position to coronary risk? The Whitehall II study. *Circulation.* 2005; 111(23):3071–3077. [PubMed: 15939818]
- Hobfoll SE. Conservation of resources: a new attempt at conceptualizing stress. *Am. Psychol.* 1989; 44(3):513–524. [PubMed: 2648906]
- Hu P, Wagle N, Goldman N, Weinstein M, Seeman TE. The associations between socioeconomic status, allostatic load and measures of health in older Taiwanese persons: Taiwan social environment and biomarkers of aging study. *J. Biosoc. Sci.* 2007; 39(4):545–556. [PubMed: 17052381]
- Janicki-Deverts D, Cohen S, Adler NE, Schwartz JE, Matthews KA, Seeman TE. Socioeconomic status is related to urinary catecholamines in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Psychosom. Med.* 2007; 69(6):514–520. [PubMed: 17636147]
- Jennings JR, Kamarck TW, Everson-Rose SA, Kaplan GA, Manuck SB, Salonen JT. Exaggerated blood pressure responses during mental stress are prospectively related to enhanced carotid atherosclerosis in middle-aged Finnish men. *Circulation.* 2004; 110:2198–2203. [PubMed: 15451789]

- Karlamangla AS, Singer BH, McEwen BS, Rowe JW, Seeman TE. Allostatic load as a predictor of functional decline. *MacArthur studies of successful aging. J. Clin. Epidemiol.* 2002; 55(7):696–710. [PubMed: 12160918]
- Karlamangla AS, Singer BH, Seeman TE. Reduction in allostatic load in older adults is associated with lower all-cause mortality risk: MacArthur studies of successful aging. *Psychosom. Med.* 2006; 68(3):500–507. [PubMed: 16738085]
- Khang YH, Lynch JW, Yang S, Harper S, Yun SC, et al. The contribution of material, psychosocial, and behavioral factors in explaining educational and occupational mortality inequalities in a nationally representative sample of South Koreans: relative and absolute perspectives. *Soc. Sci. Med.* 2009; 68(5):858–866. [PubMed: 19121885]
- Kop WJ, Berman DS, Gransar H, Wong ND, Miranda-Peats R, et al. Social network and coronary artery calcification in asymptomatic individuals. *Psychosom. Med.* 2005; 67(3):343–352. [PubMed: 15911895]
- Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu. Rev. Public Health.* 1997; 18:341–378. [PubMed: 9143723]
- Kubzansky LD, Kawachi I, Sparrow D. Socioeconomic status, hostility, and risk factor clustering in the Normative Aging Study: any help from the concept of allostatic load? *Ann. Behav. Med.* 1999; 21(4):330–338. [PubMed: 10721441]
- Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. *J. Epidemiol. Community Health.* 2003; 57(10):778–783. [PubMed: 14573579]
- Kumari M, Badrick E, Chandola T, Adler NE, Epel E, et al. Measures of social position and cortisol secretion in an aging population: findings from the Whitehall II study. *Psychosom. Med.* 2010; 72:27–34. [PubMed: 19995885]
- Kunz-Ebrecht SR, Kirschbaum C, Steptoe A. Work stress, socioeconomic status and neuroendocrine activation over the working day. *Soc. Sci. Med.* 2004; 58:1523–1530. [PubMed: 14759695]
- Kuper H, Adami HO, Theorell T, Weiderpass E. The socioeconomic gradient in the incidence of stroke: a prospective study in middle-aged women in Sweden. *Stroke.* 2007; 38(1):27–33. [PubMed: 17138948]
- Lachman ME, Weaver SL. The sense of control as a moderator of social class differences in health and well-being. *J. Personal. Soc. Psychol.* 1998; 74(3):763–773.
- Lauderdale DS, Knutson KL, Yan LL, Rathouz PJ, Hulley SB, et al. Objectively measured sleep characteristics among early-middle-aged adults: the CARDIA study. *Am. J. Epidemiol.* 2006; 164(1):5–16. [PubMed: 16740591]
- Lehman BJ, Taylor SE, Kiefe CI, Seeman TE. Relation of childhood socioeconomic status and family environment to adult metabolic functioning in the CARDIA study. *Psychosom. Med.* 2005; 67:846–854. [PubMed: 16314588]
- Lehman BJ, Taylor SE, Kiefe CI, Seeman TE. Relationship of early life stress and psychological functioning to blood pressure in the CARDIA study. *Health Psychol.* 2009; 28(3):338–346. [PubMed: 19450040]
- Lemelin ET, Diez Roux AV, Franklin TG, Carnethon M, Lutsey PL, et al. Life-course socioeconomic positions and subclinical atherosclerosis in the multi-ethnic study of atherosclerosis. *Soc. Sci. Med.* 2009; 68(3):444–451. [PubMed: 19081660]
- Lovasi GS, Hutson MA, Guerra M, Neckerman KM. Built environments and obesity in disadvantaged populations. *Epidemiol. Rev.* 2009; 31:7–20. [PubMed: 19589839]
- Lucove JC, Kaufman JS, James SA. Association between adult and childhood socioeconomic status and prevalence of the metabolic syndrome in African Americans: the Pitt County Study. *Am. J. Public Health.* 2007; 97(2):234–236. [PubMed: 17194854]
- Luecken LJ, Appelhans BM, Kraft A, Brown A. Never far from home: a cognitive affect model of the impact of early-life family relationships on physiological stress responses in adulthood. *J. Soc. Personal. Relat.* 2006; 23:189–203.
- Lynch JW, Everson SA, Kaplan GA, Salonen R, Salonen JT. Does low socioeconomic status potentiate the effects of heightened cardiovascular responses to stress on the progression of carotid atherosclerosis? *Am. J. Public Health.* 1998; 88(3):389–394. [PubMed: 9518969]

- MacLeod J, Davey-Smith G, Metcalfe C, Hart C. Is subjective social status a more important determinant of health than objective social status? Evidence from a prospective observational study of Scottish men. *Soc. Sci. Med.* 2005; 61(9):1916–1929. [PubMed: 15916842]
- Manuck SB, Bleil ME, Petersen KL, Flory JD, Mann JJ, et al. The socioeconomic status of communities predicts variation in brain serotonergic responsivity. *Psychol. Med.* 2005; 35:519–528. [PubMed: 15856722]
- Manuck, SB.; Kaplan, JR.; Lotrich, FE. Brain serotonin and aggressive disposition in humans and nonhuman primates. In: Nelson, RJ., editor. *Biology of Aggression*. New York: Oxford Univ. Press; 2006. p. 65-113.
- Manuck SB, Phillips JE, Gianaros PJ, Flory JD, Muldoon MF. Subjective socioeconomic status and presence of the metabolic syndrome in midlife community volunteers. *Psychosom. Med.* 2010; 72(1):35–45. [PubMed: 19933505]
- Marmot MG, Bosma H, Hemingway H, Brunner E, Stansfeld S. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet.* 1997; 350(9073):235–239. [PubMed: 9242799]
- Marmot MG, Shipley MJ. Do socioeconomic differences in mortality persist after retirement? A 25 year follow up of civil servants from the first White hall study. *Br. Med. J.* 1996; 313(7066):1177–1180. [PubMed: 8916748]
- Matthews KA, Flory JD, Muldoon MF, Manuck SB. Does socioeconomic status relate to central serotonergic responsivity in healthy adults? *Psychosom. Med.* 2000b; 62(2):231–237. [PubMed: 10772403]
- Matthews, KA.; Gallo, LC.; Taylor, SE. Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. In: Adler, NE.; Stewart, J., editors. *Biology of Disadvantage: Socioeconomic Status and Health*. Vol. 1186. Ann. N.Y. Acad. Sci.; 2010. p. 146-173.
- Matthews KA, Katholi CR, McCreath H, Whooley MA, Williams DR, et al. Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. *Circulation.* 2004; 110(1):74–78. [PubMed: 15210592]
- Matthews KA, Rikknen K, Everson SA, Flory JD, Marco CA, et al. Do the daily experiences of healthy men and women vary according to occupational prestige and work strain? *Psychosom. Med.* 2000a; 62(3):346–353. [PubMed: 10845348]
- Matthews KA, Rikknen K, Gallo L, Kuller LH. Association between socioeconomic status and metabolic syndrome in women: testing the reserve capacity model. *Health Psychol.* 2008; 27(5):576–583. [PubMed: 18823184]
- Maty SC, Lynch JW, Raghunathan TE, Kaplan GA. Childhood socioeconomic position, gender, adult body mass index, and incidence of type 2 diabetes mellitus over 34 years in the Alameda County Study. *Am. J. Public Health.* 2008; 98(8):1486–1494. [PubMed: 18556612]
- McDade TW, Hawkey LC, Cacioppo JT. Psychosocial and behavioral predictors of inflammation in middle-aged and older adults: the Chicago Health, Aging, and Social Relations Study. *Psychosom. Med.* 2006; 68(3):376–381. [PubMed: 16738067]
- McEwen BS. Protective and damaging effects of stress mediators. *N. Engl. J. Med.* 1998; 338:171–179. [PubMed: 9428819]
- McEwen BS. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiol. Rev.* 2007; 87(3):873–904. [PubMed: 17615391]
- McEwen BS, Seeman T. Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Ann. N.Y. Acad. Sci.* 1999; 896:30–47. [PubMed: 10681886]
- McGrath JJ, Matthews KA, Brady SS. Individual versus neighborhood socioeconomic status and race as predictors of adolescent ambulatory blood pressure and heart rate. *Soc. Sci. Med.* 2006; 63(6):1442–1453. [PubMed: 16733078]
- McLaren L. Socioeconomic status and obesity. *Epidemiol. Rev.* 2007; 29:29–48. [PubMed: 17478442]
- McLeod JD, Kessler RC. Socioeconomic status differences in vulnerability to undesirable life events. *J. Health Soc. Behav.* 1990; 31(2):162–172. [PubMed: 2102495]

- Mendelson T, Thurston RC, Kubzansky LD. Affective and cardiovascular effects of experimentally-induced social status. *Health Psychol.* 2008; 27(4):482–489. [PubMed: 18643006]
- Mendes WB, Blascovich J, Major B, Seery MD. Challenge and threat responses during downward and upward social comparisons. *Eur. J. Soc. Psychol.* 2001; 31:477–497.
- Merkin SS, Basurto-Davila R, Karlamangla A, Bird CE, Lurie N, et al. Neighborhoods and cumulative biological risk profiles by race/ethnicity in a national sample of U.S. adults: NHANES III. *Ann. Epidemiol.* 2009; 19(3):194–201. [PubMed: 19217002]
- Mezick EJ, Matthews KA, Hall M, Strollo PJ Jr, Buysse DJ, et al. Influence of race and socioeconomic status on sleep: Pittsburgh SleepSCORE project. *Psychosom. Med.* 2008; 70(4):410–416. [PubMed: 18480189]
- Miller GE, Chen E, Fok AK, Walker H, Lim A, et al. Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proc. Natl. Acad. Sci. USA.* 2009; 106(34):14716–14721. [PubMed: 19617551]
- Montez JK, Hayward MD, Brown DC, Hummer RA. Why is the educational gradient of mortality steeper for men? *J. Gerontol. B. Psychol. Sci. Soc. Sci.* 2009; 64(5):625–634. [PubMed: 19307285]
- Muldoon MF, Mackey RH, Williams KV, Korytkowski MT, Flory JD, Manuck SB. Low central nervous system serotonergic responsivity is associated with the metabolic syndrome and physical inactivity. *J. Clin. Endocrinol. Metab.* 2004; 89:266–271. [PubMed: 14715860]
- Myers HF. Ethnicity- and socio-economic status-related stresses in context: an integrative review and conceptual model. *J. Behav. Med.* 2009; 32(1):9–19. [PubMed: 18989769]
- Nazmi A, Victora CG. Socioeconomic and racial/ethnic differentials of C-reactive protein levels: a systematic review of population-based studies. *BMC Public Health.* 2007; 7(1):212. [PubMed: 17705867]
- Phillips JE, Marsland AL, Flory JD, Muldoon MF, Cohen S, Manuck SB. Parental education is related to C-reactive protein among female middle-aged community volunteers. *Brain Behav. Immun.* 2009; 23(5):677–683. [PubMed: 19486653]
- Pickett KE, Pearl M. Multilevel analyses of neighbourhood socioeconomic context and health outcomes: a critical review. *J. Epidemiol. Community Health.* 2001; 55(2):111–122. [PubMed: 11154250]
- Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. *BMC Public Health.* 2005; 5:7. [PubMed: 15661071]
- Prescott E, Godtfredsen N, Osler M, Schnohr P, Barefoot J. Social gradient in the metabolic syndrome not explained by psychosocial and behavioural factors: evidence from the Copenhagen City Heart Study. *Eur. J. Cardiovasc. Prev. Rehabil.* 2007; 14(3):405–412. [PubMed: 17568240]
- Räikkönen K, Matthews KA, Kuller LH. Depressive symptoms and stressful life events predict metabolic syndrome among middle-aged women: a comparison of World Health Organization, Adult Treatment Panel III, and International Diabetes Foundation definitions. *Diabetes Care.* 2007; 30(4):872–877. [PubMed: 17392548]
- Ramsay SE, Whincup PH, Morris R, Lennon L, Wannamethee SG. Is socioeconomic position related to the prevalence of metabolic syndrome? Influence of social class across the life course in a population-based study of older men. *Diabetes Care.* 2008; 31(12):2380–2382. [PubMed: 18809625]
- Ranjit N, Diez-Roux AV, Chambless L, Jacobs DR Jr, Nieto FJ, Szklo M. Socioeconomic differences in progression of carotid intima-media thickness in the Atherosclerosis Risk in Communities study. *Arterioscler. Thromb. Vasc. Biol.* 2006; 26(2):411–416. [PubMed: 16322533]
- Ranjit N, Diez-Roux AV, Shea S, Cushman M, Ni H, Seeman T. Socioeconomic position, race/ethnicity, and inflammation in the multi-ethnic study of atherosclerosis. *Circulation.* 2007; 116(21):2383–2390. [PubMed: 18025402]
- Rasmussen HN, Scheier MF, Greenhouse JB. Optimism and physical health: a meta-analytic review. *Ann. Behav. Med.* 2009; 37(3):239–256. [PubMed: 19711142]

- Rathmann W, Haastert B, Giani G, Koenig W, Imhof A, et al. Is inflammation a causal chain between low socioeconomic status and type 2 diabetes? Results from the KORA Survey 2000. *Eur. J. Epidemiol.* 2006; 21(1):55–60. [PubMed: 16450207]
- Repetti RL, Taylor SE, Seeman TE. Risky families: family social environments and the mental and physical health of offspring. *Psychol. Bull.* 2002; 128(2):330–366. [PubMed: 11931522]
- Seeman TE, Crimmins E, Huang MH, Singer B, Bucur A, et al. Cumulative biological risk and socioeconomic differences in mortality: MacArthur studies of successful aging. *Soc. Sci. Med.* 2004; 58(10):1985–1997. [PubMed: 15020014]
- Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation—allostatic load and its health consequences. *MacArthur studies of successful aging. Arch. Intern. Med.* 1997; 157(19):2259–2268. [PubMed: 9343003]
- Seeman TE, Singer BH, Ryff CD, Dienberg LG, Levy-Storms L. Social relationships, gender, and allostatic load across two age cohorts. *Psychosom. Med.* 2002; 64(3):395–406. [PubMed: 12021414]
- Sekine M, Chandola T, Martikainen P, McGeoghegan D, Marmot M, Kagamimori S. Explaining social inequalities in health by sleep: the Japanese civil servants study. *J. Public Health (Oxf.)*. 2006; 28(1):63–70. [PubMed: 16287708]
- Shiffman S, Stone AA, Hufford MR. Ecological momentary assessment. *Annu. Rev. Clin. Psychol.* 2008; 4:1–32. [PubMed: 18509902]
- Shrewsbury V, Wardle J. Socioeconomic status and adiposity in childhood: a systematic review of cross-sectional studies 1990–2005. *Obesity*. 2008; 16(2):275–284. [PubMed: 18239633]
- Singh-Manoux A, Ferrie JE, Chandola T, Marmot M. Socioeconomic trajectories across the life course and health outcomes in midlife: evidence for the accumulation hypothesis. *Int. J. Epidemiol.* 2004; 33(5):1072–1079. [PubMed: 15256527]
- Skalicka V, Van LF, Bambra C, Krokstad S, Mackenbach J. Material, psychosocial, behavioural and biomedical factors in the explanation of relative socio-economic inequalities in mortality: evidence from the HUNT study. *Int. J. Epidemiol.* 2009; 38(5):1272–1284. [PubMed: 19661280]
- Smith GD. Learning to live with complexity: ethnicity, socioeconomic position, and health in Britain and the United States. *Am. J. Public Health*. 2000; 90(11):1694–1698. [PubMed: 11076232]
- Smith JP. Healthy bodies and thick wallets: the dual relation between health and economic status. *J. Econ. Perspect.* 1999; 13(2):144–166. [PubMed: 15179962]
- Steffen PR. The cultural gradient: Culture moderates the relationship between socioeconomic status (SES) and ambulatory blood pressure. *J. Behav. Med.* 2006; 29(6):501–510. [PubMed: 17082972]
- Stephens A, Kunz-Ebrecht SR, Owen N, Feldman PJ, Willemssen G, et al. Socioeconomic status and stress-related biological responses over the working day. *Psychosom. Med.* 2003; 65:461–471. [PubMed: 12764220]
- Stephens A, Marmot M. The role of psychobiological pathways in socio-economic inequalities in cardiovascular disease risk. *Eur. Heart J.* 2002; 23(1):13–25. [PubMed: 11741358]
- Stephens A, Shamaei-Tousi A, Gylfe A, Henderson B, Bergstrom S, Marmot M. Socioeconomic status, pathogen burden and cardiovascular disease risk. *Heart*. 2007; 93(12):1567–1570. [PubMed: 17488763]
- Thurston RC, Kubzansky LD, Kawachi I, Berkman LF. Is the association between socioeconomic position and coronary heart disease stronger in women than in men. *Am. J. Epidemiol.* 2005; 162(1):57–65. [PubMed: 15961587]
- Thurston RC, Kubzansky LD, Kawachi I, Berkman LF. Do depression and anxiety mediate the link between educational attainment and CHD. *Psychosom. Med.* 2006; 68(1):25–32. [PubMed: 16449408]
- Troxel WM, Matthews KA. What are the costs of marital conflict and dissolution to children's physical health. *Clin. Child Fam. Psychol. Rev.* 2004; 7(1):29–57. [PubMed: 15119687]
- Uchino BN. Social support and health: a review of physiological processes potentially underlying links to disease outcomes. *J. Behav. Med.* 2006; 29(4):377–387. [PubMed: 16758315]

- van Oort FV, van Lenthe FJ, Mackenbach JP. Material, psychosocial, and behavioural factors in the explanation of educational inequalities in mortality in The Netherlands. *J. Epidemiol. Community Health*. 2005; 59(3):214–220. [PubMed: 15710599]
- Wamala SP, Mittleman MA, Horsten M, Schenck-Gustafsson K, Orth-Gomer K. Job stress and the occupational gradient in coronary heart disease risk in women. The Stockholm Female Coronary Risk Study. *Soc. Sci. Med.* 2000; 51(4):481–489. [PubMed: 10868664]
- Williams DR, Jackson PB. Social sources of racial disparities in health. *Health Aff. (Millwood.)*. 2005; 24(2):325–334. [PubMed: 15757915]
- Zajacova A, Dowd JB, Aiello AE. Socioeconomic and race/ethnic patterns in persistent infection burden among U.S. adults. *J. Gerontol. A. Biol. Sci. Med. Sci.* 2009; 64(2):272–279. [PubMed: 19196638]
- Zink CF, Tong Y, Chen Q, Bassett DS, Stein JL, Meyer-Lindenberg A. Know your place: neural processing of social hierarchy in humans. *Neuron*. 2008; 58(2):273–283. [PubMed: 18439411]

RELATED RESOURCES

- Adler NE, Rehkopf DH. U.S. disparities in health: descriptions, causes, and mechanisms. *Annu. Rev. Public Health*. 2008; 29:235–252. [PubMed: 18031225]
- Report of the APA Task Force on Socioeconomic Status. Washington, DC: Am. Psychol. Assoc.; 2007. Am. Psychol. Assoc. Task Force Socioecon. Status.
- Lynch J, Smith GD. A life course approach to chronic disease epidemiology. *Annu. Rev. Public Health*. 2005; 26:1–35. [PubMed: 15760279]
- Marmot, M. *The Status Syndrome: How Social Standing Affects Our Health and Longevity*. New York: Henry Holt; 2004.
- Sampson RJ, Morenoff JD, Gannon-Rowley T. Assessing “neighborhood effects”: social processes and new directions in research. *Annu. Rev. Sociol.* 2002; 28:443–478.

Figure 1. Morbidity rate by socioeconomic status level. (a) Percent diagnosed osteoarthritis, (b) relative prevalence of chronic disease, (c) prevalence of hypertension, and (d) rate of cervical cancer per 100,000. Original figure from Adler et al. (1994).

Figure 2.
Psychobiological influences on the socioeconomic status–health gradient.