

Understanding the Association Between Socioeconomic Status and Physical Health: Do Negative Emotions Play a Role?

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In this article, the authors evaluate the possible roles of negative emotions and cognitions in the association between socioeconomic status (SES) and physical health, focusing on the outcomes of cardiovascular diseases and all-cause mortality. After reviewing the limited direct evidence, the authors examine indirect evidence showing that (a) SES relates to the targeted health outcomes, (b) SES relates to negative emotions and cognitions, and (c) negative emotions and cognitions relate to the targeted health outcomes. The authors present a general framework for understanding the roles of cognitive–emotional factors, suggesting that low-SES environments are stressful and reduce individuals' reserve capacity to manage stress, thereby increasing vulnerability to negative emotions and cognitions. The article concludes with suggestions for future research to better evaluate the proposed model.

Health disparities associated with socioeconomic status (SES) have existed for centuries (G. D. Smith, Carroll, Rankin, & Rowan, 1992) and have been recognized by researchers for many decades (Chapin, 1924; Warren & Sydenstricker, 1916). Recent research within the United States and other industrialized countries demonstrates that SES is associated with diverse health outcomes (Adler, Marmot, McEwen, & Stewart, 1999), and some evidence suggests that SES inequalities in mortality may even be widening (e.g., Drever, Whitehead, & Roden, 1996; Pappas, Queen, Hadden, & Fisher, 1993; Phillimore, Beattie, & Townsend, 1994). Despite the consistent pattern of these findings, the mechanisms that underlie the graded relationship between SES and health have not been clearly elucidated. In part, SES disparities in health are clearly due to differences in the distribution of basic resources such as health care, nutrition, and sanitary living environments (e.g., Antonovsky, 1967; see also Lynch, Smith, Kaplan, & House, 2000). This focus may be particularly important to explaining poor health in groups characterized by poverty, but the impact of SES on health is not only at the poverty line. Rather, health discrepancies have a monotonic relationship with SES, so that even rela-

tively affluent groups exhibit worse health than their higher SES counterparts (e.g., Kitagawa & Hauser, 1973; Kraus, Borhani, & Franti, 1980). Thus, numerous interconnected factors appear to contribute to SES disparities in health, and researchers have therefore cast a wider net in attempting to explain the SES gradient.

One prominent explanation is that cognitive–emotional factors and disorders play a role in understanding how low SES results in risk for early death and disability (Adler et al., 1994; Kaplan & Keil, 1993; Matthews, 1989; Taylor, Repetti, & Seeman, 1997). Low-SES environments may kindle disproportionate levels of negative emotions and attitudes, and likewise, these variables may have deleterious effects on health. However, the literature has not been reviewed systematically to support or refute this hypothesis. In the current article, we evaluate the evidence for the tenet that cognitive–emotional factors may, in part, mediate the relationship between SES and health. First, we provide a brief overview of the conceptual issues important in examining socioeconomic and emotional factors. We then review the evidence that bears on the mediational hypothesis. For mediation by cognitive–emotional factors to be tenable, the research must show (a) that SES relates to health; (b) that SES relates to negative emotions and cognitions; (c) that negative emotions and cognitions relate to health; and finally (d) that when all factors are examined within a single methodological frame, the relationship between SES and health is attenuated if effects for negative cognitive–emotional factors are statistically controlled (Baron & Kenny, 1986). Unfortunately, few studies have evaluated these four criteria. The indirect evidence for mediation is more compelling, and we therefore discuss this research at length. Many detailed reviews concerning the associations between SES and health and between cognitive–emotional factors and health have been published recently. Rather than duplicate this work, we summarize and update it. We focus on cardiovascular diseases—the leading cause of mortality in the United States (American Heart Association, 2000)—and all-cause mortality because these outcomes have been examined with sufficient frequency and rigor to provide an estimate of the proposed

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links and because SES gradients are particularly strong for these health outcomes (e.g., M. A. Gonzalez, Rodriguez, & Calero, 1998; Kaplan & Keil, 1993; Lynch et al., 2000). The associations between SES and cognitive–emotional factors have not been presented in any recent, enumerative reviews (but see the review of SES and psychiatric disorders by Kohn, Dohrenwend, & Mirotnik, 1998), and we therefore analyze this research in more detail. Following our review and critical analysis, we present a framework for understanding the pathways that may dynamically link SES, cognitive–emotional factors, and health. Finally, we conclude with recommendations for future research to better address the proposed mediation hypothesis.

Conceptualizing SES

SES is an aggregate concept defined according to one's level of resources or prestige in relation to others (Krieger, Williams, & Moss, 1997; Lynch & Kaplan, 2000). Resource-based measures assess access to material and social assets, including income, wealth, and educational attainment. Prestige-based measures refer to an individual's rank or status in a social hierarchy, typically evaluated by access to and consumption of goods, services, and knowledge as linked to occupational prestige and education. Social class refers to groups defined by interdependent economic and legal relationships, based on an individual's structural location within the economy (e.g., employer vs. employee).

SES can be assessed at the level of the individual, household unit, and neighborhood or community. Most commonly, SES is assessed through single individual-level indicators, but these may not accurately characterize the status of the family or household. Moreover, research shows that neighborhood-level indicators (e.g., Eaton & Muntaner, 1999; Haan, Kaplan, & Camacho, 1987) and contextual income distribution (Daly, Duncan, Kaplan, & Lynch, 1998; Soobader & LeClere, 1999) predict health above the effects of individual-level indicators. Studies that evaluate SES at multiple levels can therefore provide a more accurate assessment of the association between SES and health outcomes (e.g., Diez-Roux et al., 1997; B. P. Kennedy, Kawachi, Glass, & Prothrow-Stith, 1998). Perhaps more important, they provide clues to the material, social, and psychological mechanisms that may account for the association between SES and health.

In research concerning health, the most commonly used indicators of SES include educational attainment, occupational prestige, and income. These indicators are related, but not fully overlapping, and they may impact health through disparate pathways. Furthermore, each is associated with distinct advantages and disadvantages for research (Krieger et al., 1997). For example, questions about income are prone to missing and distorted responses. Occupation-based measures cannot be used to indicate SES for individuals who are not working for reasons of retirement, unemployment, homemaking, or caretaking. Income and occupation may also be influenced by *reverse causation*—that is, the effects of psychiatric or medical illness on SES. In these respects, education affords some advantage because one's education is often completed before the onset of chronic illness, and individuals are typically willing and able to accurately represent their education. Overall, education may be the most appropriate assessment of SES for women because of the proportion of women who do not work outside the home (approximately 40%; Bureau of Labor Statistics,

2000). Their own income or occupation may therefore underrepresent the SES of the household. On the other hand, the implications of education for economic and health standing may differ by age, ethnicity, and gender (Krieger et al., 1997; Oliver & Shapiro, 1995). Thus, SES indicators should be chosen according to the specific research questions and populations of interest.

Another factor important in measuring SES is the temporal nature of the assessment. SES tends to be stable across the life span and across generations of family members (e.g., Lynch, Kaplan, & Salonen, 1997), but some indicators of SES are quite dynamic. For example, more than half of U.S. households sampled experienced an increase of 50% or a decrease of more than 33% in monthly income in 1984 (U.S. Bureau of the Census, 1996). Experiencing a single marked income drop during a 5-year period resulted in a 30% increase in mortality risk, whereas two or more decreases predicted a 70% increase in mortality risk (Duncan, 1996). Another recent study found a dose–response association between the frequency of exposure to low income and physical, psychological (i.e., depression, hostility, low optimism), and cognitive functioning (Lynch, Kaplan, & Shema, 1997). Similarly, accumulated exposure to low occupational prestige of parents and low SES at several points in young adulthood predicted self-rated health in midlife (Power, Manor, & Matthews, 1999). Thus, the common approach of measuring SES at a single time point may be inadequate to capture the full impact of exposure to low-SES environments.

An incorporation of time in assessing SES would also help elucidate directional effects in the association between SES and health, as proposed in the social causation and social drift hypotheses. According to the social causation perspective, socioeconomic standing has a causal role in determining health or emotional problems. Social drift interpretations assert that individuals with worse physical or emotional health may “drift down” the socioeconomic hierarchy or fail to rise in socioeconomic standing as would be expected on the basis of familial origins or changes in societal affluence. That is, the social drift model views health problems as exerting a causal influence on social status. These directional hypotheses have been the subject of considerable research and discussion in the literature (e.g., B. P. Dohrenwend et al., 1998; Eaton & Muntaner, 1999; Kessler, 1982; Lichtenstein, Harris, Pedersen, & McClearn, 1993; Marmot, Kogevinas, & Elston, 1987; J. P. Smith, 1999). In actuality, the associations are probably dynamic and reciprocal. We further discuss these issues below, but it is important to note that we view social drift as a phenomenon that, in part, maintains the adverse effects of SES.

To summarize, in evaluating research concerning the association between SES and mental health and the association between SES and physical health, the adequacy of the SES assessment is an important consideration. The fact that studies of SES and health incorporating a single indicator measured at one level and point in time have revealed statistically significant effects attests to the robust nature of the relationship. Nevertheless, this common measurement technique may underestimate the nature and scope of SES–health disparities and could impede efforts to delineate the numerous paths that create them. Future research should attempt to examine how the multiple levels of SES interrelate and affect health and should also consider the temporal and dynamic nature of socioeconomic position (for further discussion and review, see Anderson, 1999; Krieger et al., 1997; Robert & House, 2000).

Conceptualizing Emotion

The conceptual and theoretical description of emotion has been the source of considerable attention and disagreement in the literature (e.g., Ekman & Davidson, 1994). However, most researchers concur that emotions comprise affective, cognitive, and behavioral components, along with concomitant physiological changes that occur to ready the body for action. Moreover, many definitions and theories assert that emotional experiences reflect two underlying dimensions that characterize the valence, or pleasantness, and arousal, or attention, of the emotion (Plutchik, 1980; Russell, 1997; Watson & Tellegen, 1985). Emotions that are similar in terms of valence and/or arousal tend to cluster (e.g., L. A. Feldman, 1995). For example, individuals who report depressed affect also frequently report feeling anxious (Clark & Watson, 1991). Negative emotions and cognitions are also closely linked (Clare, 1994). For example, hostile cognition frequently occurs in conjunction with angry affect (T. W. Smith, 1994), and hopeless cognition often occurs with depressed affect (G. W. Brown & Harris, 1978).

Emotion researchers continue to disagree about whether positive affect and negative affect form two poles of a single dimension or distinct dimensions. Substantial evidence suggests that these constructs are orthogonal (Watson & Clark, 1997), yet a recent comprehensive review concluded that bipolarity represents the best fit to existing data (Russell & Carroll, 1999). Still other research has found support for both models of affectivity, showing that the structure of emotion is importantly influenced by individual-level variables (Reich, Zautra, & Potter, 2001). Resolving this issue is beyond the scope of this review. However, for our purposes, we consider the possibility that positive and negative affect represent discrete dimensions and, as such, that they may provide distinct information about the factors connecting SES with health.

Regardless of valence and arousal, all emotions are fundamentally adaptive inasmuch as they serve a communicative function (Lazarus, 1991). Maladaptive emotions are distinguished from adaptive emotions by their inappropriateness, frequency, intensity, and duration (Frijda, 1994). Although maladaptive emotional experiences form an integral part of psychiatric disorders (e.g., depressed mood is fundamental to clinical depression), they should be not be considered interchangeable (Santor & Coyne, 2001). Emotional disorders are broader syndromes that comprise a cluster of symptoms, behaviors, and cognitive–affective processes. Here, we summarize research that has analyzed cognitive–emotional states or symptoms as well as clinical disorders. This distinction is important because psychiatric disorders occur relatively infrequently in the population, whereas symptoms are fairly common.

In specific, our review addresses depression, anxiety, anger and hostility, and hopelessness. These constructs clearly overlap substantially with more general constructs such as psychological distress and with trait measures of negative affect (e.g., Clark & Watson, 1991; Clark, Watson, & Mineka, 1994). However, broader conceptualizations have not received rigorous attention in physical health research, perhaps in part because they may be too vague to contribute information regarding mechanisms or targets for intervention. In addition, broader conceptualizations of negative affectivity (e.g., Neuroticism) often relate to somatic complaints but not necessarily to objective health outcomes (Watson & Pennebaker, 1989). Thus, we focus explicitly on the cognitive–

emotional constructs that, at this point, have the greatest potential to help explain the health disadvantage experienced by people with low SES.

Definition and Measurement of Depression

Depression is an unpleasant emotion that is generally associated with low arousal. Clinical depression represents a constellation of emotional and behavioral symptoms forming several *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) diagnoses. A diagnosis of major depression entails depressed mood and/or a lack of interest or pleasure in most activities for at least 2 weeks. Four additional symptoms must be present, such as a change in appetite or sleeping habits, fatigue, psychomotor retardation or agitation, thoughts of guilt or worthlessness, problems thinking or concentrating, and suicidal ideation or intent. Over a 1-month period, approximately 5% of the U.S. population experiences a major depressive episode (Blazer, Kessler, McGonagle, & Swartz, 1994). Lifetime prevalence is considerably higher, at about 13% (Kessler et al., 1994). Prevalence is likely to be elevated in certain subgroups, including medically ill populations (e.g., Stevens, Merikangas, & Merikangas, 1995). For example, estimates of major depression prevalence rates among heart disease patients range from 15% to 23% (Carney, Freedland, Sheline, & Weiss, 1997; Frasure-Smith, Lespérance, & Talajic, 1993; M. B. Gonzalez et al., 1996). Other clinical depressive diagnoses include minor depression, depressive symptoms that are subthreshold in severity to major depression, and dysthymia, subthreshold depressive symptoms that endure for at least 2 years. Lifetime prevalence of dysthymia has been estimated at 5% (Kessler et al., 1994), and lifetime prevalence of subthreshold depressive symptoms is much higher, at approximately 23% (Horwath, Johnson, Klerman, & Weissman, 1992).

The research concerning depression and physical health has typically examined major depression or depressive symptoms. Research pertaining to socioeconomic predictors of depression has analyzed depressive symptoms as well as clinical depressive diagnoses. Studies that use measures of depressive symptoms can provide information concerning the dose–response relationship between depression and health outcomes, which occurs if the severity of health problems increases with the severity of the symptoms. This pattern of association suggests a gradient, or linear relationship, between depression and health. In contrast, a threshold interpretation of the depression and health relationship would be tenable if the research suggested that only those individuals with major depression suffered a health disadvantage. On the other hand, these patterns are difficult to discriminate because high levels of depressive symptoms portend risk for full-syndrome depression (Judd & Akiskal, 2000), which in turn may have a deleterious effect on health.

With some exceptions, most studies concerning clinical depression have used well-validated structured interview assessments, such as the Diagnostic Interview Schedule (Robins, Helzer, Croughan, & Ratcliff, 1981) or the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1979). Some of the research concerning depressive symptoms has used well-validated measures such as the Beck Depression Inventory (BDI; Beck & Beamesderfer, 1974) or the Center for Epidemiological

Studies Depression Scale (CES-D; Radloff, 1977), whereas other research has relied on study-specific measures, sometimes composed of one or only a few questions. Less confidence can be placed in findings derived from very few items, given the lower reliability typically associated with such assessment tools. Symptom measures can be examined on a continuum, with higher scores indicating higher levels of symptoms, or they can be dichotomized using a standard cutoff to form depressed and nondepressed groups.

Definition and Measurement of Hopelessness

Hopelessness describes negative cognition about the self and the future, which is likely to accompany severe negative emotions such as those associated with clinical depression. Along these lines, hopelessness is often conceptualized as a symptom of depression (G. W. Brown & Harris, 1978). However, some research suggests that this construct affects health beyond its association with depression (Everson et al., 1996). Furthermore, hopelessness and depression are uniquely associated with other psychological phenomena, including suicidal intent (Greene, 1989). Studies that have examined hopelessness have generally assessed this construct using a few items or even a single one. Despite the limitations associated with this type of assessment, the research has been fairly consistent in showing that hopelessness has important health implications.

Definition and Measurement of Anxiety

Anxiety is an emotion of negative valence and positive arousal. Fear or apprehension about the future represents the cardinal feature of all anxiety disorders, but corollary symptoms vary widely, consistent with the number of *DSM-IV* (American Psychiatric Association, 1994) anxiety diagnoses. Diagnostic criteria for generalized anxiety disorder—the least situation- and stimulus-specific diagnosis—include excessive anxiety and worry for at least 6 months; difficulty controlling the anxiety; and three additional symptoms, such as restlessness, fatigue, difficulty thinking or concentrating, irritability, muscle tension, and sleep disturbance. Panic disorder has frequently been examined in relationship to physical health. Panic disorder involves recurrent attacks of sudden intense fear that occur without an identifiable cause and that are accompanied by somatic (e.g., hot flashes, shortness of breath, sweating, gastrointestinal distress) and cognitive symptoms (e.g., fear of losing control or dying). Agoraphobia, or the enactment of behaviors designed to avoid triggers of panic attacks such as refusal to drive or leave one's house, may occur in association with panic disorder. Prevalence rates of anxiety disorders vary considerably. In a 1-year period, approximately 0.9% of the population meet criteria for panic disorder, whereas about 9.7% meet criteria for any phobic diagnosis (Eaton, Dryman, & Weissman, 1991). Most prior research concerning SES has focused on anxiety disorders, whereas many studies related to physical health have examined anxiety symptoms (e.g., phobic symptoms or generalized worry). A number of studies of physical health outcomes have applied the Crown-Crisp Experiential Index, a reliable and valid measure of somatic anxiety, phobic anxiety, and obsessiveness (see, e.g., M. W. Ross & Hafner, 1990). Other studies have used

less well-known measures of anxiety, impeding the evaluation of its health correlates.

Definition and Measurement of Hostility

Research concerning hostility involves analyzation of three interrelated emotional, behavioral, and cognitive constructs (T. W. Smith, 1994), consistent with the definition of emotion outlined above. Anger represents the emotional component and is characterized by negative valence and moderate to high arousal. Resentment, scorn, and derision are closely related emotional constructs. The behavioral component consists of verbal and physical aggressive acts, involving harmful intent. *Hostility* represents negative attitudes and beliefs about others, such as cynicism and mistrust. Studies of hostility have variously used interview ratings or questionnaire assessment methods, and the two strategies have produced only moderately related findings (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985). Interview ratings typically assess the behavioral aspects of this construct. Perhaps the most frequently used hostility self-report measure is the Cook-Medley hostility inventory (Ho; Cook & Medley, 1954), which is best described as a measure of hostile cognition (e.g., Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; T. W. Smith & Frohm, 1985). Despite widespread usage, the Ho has been criticized for questionable construct validity, heterogeneity of item content, and close association with Neuroticism (e.g., Barefoot, 1992; Barefoot et al., 1989; Carmody, Crossen, & Wiens, 1989).

SES and Health

The evidence for SES disparities in health is long-standing and compelling. More than 3 decades ago, Antonovsky (1967) found consistent support for an inverse relationship between SES and mortality in a review of more than 30 studies. The coherence of the association was notable given the diverse populations and methodologies used. More recently, many well-designed studies have identified a socioeconomic differential in morbidity and mortality in the United States (e.g., Backlund, Sorlie, & Johnson, 1996; J. J. Feldman, Makuc, Kleinman, & Cornoni-Huntley, 1989; Kitagawa & Hauser, 1973; Lantz et al., 1998; Pappas et al., 1993) and in other industrialized countries (e.g., Doornbos & Kromhout, 1990; Holme, Helgeland, Hjermand, & Leren, 1982; Marmot, Shipley, & Rose, 1984; Marmot et al., 1991; Salonen, 1982). A number of reviews have provided detailed and comprehensive analyses of this research and have presented the conclusion that SES has a profound influence on health (Adler et al., 1994; Adler, Boyce, Chesney, Folkman, & Syme, 1993; Carroll, Bennett, & Davey Smith, 1993; Lynch et al., 2000; Marmot et al., 1987; Williams, 1990; Williams & Collins, 1995).

The association between SES and health can be summarized as monotonic, so that as individuals or groups move up in the SES continuum, mortality and morbidity rates decrease, with the gradient steepest at the lowest levels (Backlund et al., 1996; Ecob & Davey Smith, 1999). This pattern emerges across socioeconomic indicators (e.g., occupation, income, education), suggesting that a general underlying social ordering is important (e.g., Adler et al., 1994). In addition, both community (e.g., census tract) and individual indicators of SES predict health outcomes (e.g., Adler et al., 1993; Robert & House, 2000). The gradient extends to mortality

from all causes (e.g., Adler et al., 1994; Anderson & Armstead, 1995) and to diverse specific health outcomes, including cardiovascular disease, renal disease, diabetes, cancer, arthritis, and infant mortality (e.g., Illsey & Baker, 1991; Pincus, Callahan, & Burkhauser, 1987). Two reviews have concluded that SES affects the development and progression of cardiovascular diseases (M. A. Gonzalez, Rodriguez, & Calero, 1998; Kaplan & Keil, 1993).

Researchers have examined a number of possible explanations for SES–health disparities, including access to health care, residential characteristics, environmental exposure, physiological processes, health behaviors, and psychosocial factors (e.g., Anderson & Armstead, 1995; Kaplan & Keil, 1993; Macintyre, 1997; Williams, 1990). What is clear is that none of these factors provides a complete explanation for the gradient. For example, SES relates to health behaviors and established biological health risk factors, but these variables do not account statistically for the gradient (e.g., Lantz et al., 1998; see also Adler et al., 1994; Evans, Barer, & Marmor, 1994; Marmot, Bobak, & Smith, 1995, for discussion and review). Inequalities in access to health care do not provide an exhaustive explanation, as evidenced by the fact that countries with nationally funded health care programs show a linear, albeit less steep, association between SES and health (Adler et al., 1993; Williams, 1990). SES gradients also exist for causes of death that are not amenable to medical treatment (Marmot et al., 1987). In addition, although health problems do affect social mobility, social drift does not appear to explain a substantial portion of the association between SES and health (e.g., Haan et al., 1987; Macintyre, 1997; Wilkinson, 1986). Thus, the paths linking SES to health are multifarious and not amenable to simple solutions.

Integrative Studies of SES, Cognitive–Emotional Factors, and Health: Evidence for Mediation?

Few studies meet the criteria outlined above as necessary to examine whether cognitive and emotional factors partially mediate the association between SES and health. Consequently, we do not limit this portion of our review to studies focusing on mortality and cardiovascular diseases, including any study that simultaneously examined SES, cognitive or emotional factors, and a physical health outcome (regardless of whether they tested all statistical criteria). Seven studies included all three types of variables.

Cohen, Kaplan, and Salonen (1999) examined the extent to which psychosocial characteristics (e.g., stress, personal control, social support, anger and hostility, depression and hopelessness) and health behaviors contributed to the association between SES and perceived health in samples from the United States (the Harris Poll Study) and Finland (the Kuopio Ischemic Heart Disease Study). Both studies revealed a graded, inverse association between SES and the odds of poor self-rated health. In the U.S. study, income and education were also significantly associated with personal control, social support, stress, life events, and anger, and these variables predicted the likelihood of perceived poor health. Statistical control for psychosocial variables attenuated the odds ratio (OR) for poor health for all lower education groups compared with the beyond college group as follows: from 5.0 to 3.6 for the 8th grade or less group, from 2.6 to 2.2 for the some high school or degree group, and from 1.6 to 1.4 for the some college or degree group. Excess risk for the two lower income groups compared with the highest income group (i.e., > \$45,000)

was also attenuated (ORs reduced from 4.6 to 2.6, \$10,000 or less group; from 3.1 to 2.2, \$10,000–\$20,000 group). In the Finnish study, income and education were strongly inversely associated with hostility, depression, and hopelessness. Social support was positively related to income but only marginally to education, and life events scores were unrelated to income and positively related to education. Increases in life events, depression, and hopelessness were positively associated with the risk of perceived poor health, and social support was inversely associated with the risk of perceived poor health. Hostility did not predict perceived health. Statistical control for psychosocial variables attenuated the OR for perceived poor health associated with less than elementary education from 4.4 to 2.5, for elementary or part junior high from 3.4 to 2.3, and for less than high school from 2.1 to 1.7 (with high school education serving as the comparison). Similarly, ORs for the lowest income group were attenuated from 5.3 to 3.4, the next lowest from 2.9 to 1.7, the middle income group from 2.2 to 1.5, and the next to highest income group from 1.2 to 0.9, when compared with the highest income group.

Thus, in both studies statistical control for the psychosocial variables reduced the excess risk for poor health associated with the lowest income and education groups by the greatest proportion but attenuated risk for all lower education and income groups to varying degrees. In commenting on this pattern of findings, Cohen et al. (1999) suggested that psychological characteristics might be more likely to covary with other sources of risk in lower SES groups (i.e., environmental factors, poor nutrition) and, therefore, that psychosocial factors capture multiple influences on health at low levels of SES. Limitations of this study include the use of a cross-sectional design, a self-reported health outcome, and some nonvalidated measures of psychosocial constructs. In addition, interactive associations were not tested statistically, nor were types of mediators examined individually (impeding identification of the relative impact of the cognitive and emotional variables). Nonetheless, the results support the view that psychosocial functioning in part mediates the association between SES and perceived health.

Levenstein and Kaplan (1998) investigated the degree to which psychological characteristics explained the association between SES and the occurrence of ulcer. For women, educational attainment was a significant predictor of new ulcers during a 9-year follow-up period. Compared with participants who attended at least some college, those with less than a high school education were more than three times as likely (OR = 3.3), and those with a high school degree were twice as likely (OR = 2.0), to develop an ulcer. For men, the association between educational attainment and incident ulcer was only marginally significant (OR = 1.9, less than high school; OR = 1.8, high school graduates). For women, adjustment for a composite psychological index comprising depression, hostility, anomie, and personal uncertainty reduced the OR for new ulcer associated with the less than high school group from 3.3 to 2.4 and from 2.0 to 1.5 for high school graduates. For men, adjustment for psychological factors had very little impact on the association between education and ulcer incidence (with ORs reduced from 1.9 to 1.8, less than high school; from 1.8 to 1.7, high school graduates), but given that the initial effects were nonsignificant, this lack of mediation effect is not particularly informative. The extent to which psychosocial factors related to SES, or were independently related to the occurrence of ulcer, was not reported. Levenstein and Kaplan concluded that overall, the results

suggest that psychosocial factors (in addition to other factors) might contribute to the association between SES and ulcer, at least in women.

Lynch, Kaplan, Cohen, Tuomilehto, and Salonen (1996) examined the effects of SES on incident myocardial infarction (MI), cardiovascular mortality, and all-cause mortality across a 4–10-year follow-up period and evaluated the degree to which biological, behavioral, and psychosocial risk factors explained the effects of SES. The associations between the psychosocial factors and the health outcomes and between the psychosocial factors and SES were not reported. Nonetheless, simultaneous adjustment for depression, hopelessness, marital status, participation in organizations, and social support reduced the excess risk associated with the lowest income group by 52% (from 3.14 to 2.03) for all-cause mortality and by 57% (from 2.66 to 1.71) for cardiovascular mortality. In contrast, very little of the relationship between SES and incident MI was explained by psychosocial factors (OR for the lowest income group was reduced from 4.34 to 4.25). Because social (i.e., resource) and cognitive–emotional factors were considered in a single block, the independent effects of depression and hopelessness cannot be determined. Furthermore, interactions among variables were not tested. The results of this study suggest that psychosocial factors contribute to the association between SES and all-cause or cardiovascular mortality but not to the association between SES and MI.

Another study (Gump, Matthews, & Rääkkönen, 1999) used structural equation modeling to examine the interrelationships among family and neighborhood SES, hostility, cardiovascular reactivity to stressful tasks, and left ventricular mass, in a group of children and adolescents. Low family and neighborhood SES predicted higher levels of hostility in Black but not White participants. Low SES was also associated with augmented cardiovascular reactivity, which, in turn, was related to left ventricular mass. For Black children, the association between SES and reactivity was mediated by individual differences in hostility. This study is limited by use of a cross-sectional design, with a relatively small and nonrandomly selected sample. In addition, children and adolescents were examined, and the results might not generalize to adults. Finally, clinical outcomes were not examined, although cardiovascular reactivity represents a possible physiological pathway through which psychosocial factors may affect cardiovascular health outcomes (Manuck, 1994), and left ventricular mass predicts cardiovascular morbidity and mortality (Casale, Devereux, & Milner, 1986; Levy, Garrison, Savage, Kannel, & Castelli, 1990). In summary, this study provides preliminary evidence that in Blacks, SES might influence the development of cardiovascular disease, in part, through its association with hostility.

Two articles based on the Beta Blocker Heart Attack Trial (BHAT) examined the extent to which psychosocial factors explained the association between SES and cardiovascular outcomes. In the first, Ruberman, Weinblatt, Goldberg, and Chaudhary (1984) examined psychosocial factors including social isolation, stress, depression, Type A behavior, and educational attainment on survival in men recovering from MI. Individuals with lower education had higher mortality rates following MI compared with participants with more education. In addition, less-educated individuals had higher levels of stress and social isolation. Type A behavior and depression were not related to educational attainment, and overall, the proportion of men with high depression

scores was quite low. Life stress and social isolation predicted mortality rates. When education, life stress, and social isolation were included simultaneously in a regression equation, education no longer predicted survival. It is notable that occupational status formed a component of the life stress assessment because this creates interpretive ambiguities. That is, social isolation and stress may have mediated the association between education and survival, or life stress and education could simply have represented confounded indicators of SES. In addition, as noted, depression rates were very low in this study; thus, range restriction may have limited power.

A later study (Ickovics, Viscoli, & Horwitz, 1997) from the BHAT examined the associations among SES, depression, and functional status in individuals 12 months after MI. The initial criteria for mediation were fulfilled—SES was related to depression, life stress, and social support, and SES was inversely associated with improvement in functional status. Likewise, life stress, social isolation, and depression predicted changes in functional status. However, after controlling for clinical features (i.e., medical history, health behaviors, treatment group, severity of MI) and demographic variables (i.e., age, ethnicity), the psychosocial factors explained only a small amount of the risk for no improvement associated with lower social class; the OR for showing no improvement for low versus high social class decreased from 1.62 to 1.51. One limitation of this study is that it did not allow for possible interactions among stress, social isolation, and depression. In addition, it would have been informative if the authors had presented information about potential discrepancies between analyses examining the mediational effects of psychosocial factors both before and after controlling for clinical features, thereby allowing for the possible indirect effects of psychosocial factors (e.g., through comorbid health problems, severity of MI, health behaviors).

Finally, Fiscella and Franks (1997) examined the extent to which psychological factors explained the association between SES and mortality over a 12–16-year follow-up period. Individuals with low income had significantly higher levels of depression, hopelessness, and life dissatisfaction at baseline compared with their higher SES counterparts, and these psychosocial constructs significantly predicted mortality risk. In multivariate analyses, statistical control for depression and hopelessness accounted for a small amount of the association between SES and mortality (3%–11%). Limitations of this research include the fact that psychological factors were measured over a brief period and that they were not reassessed during follow-up. In addition, the measures of depression and hopelessness consisted of only a few items.

In summary, few studies have integrated socioeconomic, cognitive or emotional, and health variables within the same methodological framework. The studies that have evaluated these factors concurrently provide inconclusive evidence for the model, given their conflicting findings and methodological limitations. None of the studies statistically tested interactions among psychosocial factors, or between SES and psychosocial factors, and they may therefore underestimate the true effects of negative emotions and cognitions (and other psychosocial paths). Therefore, we now turn to the more comprehensive indirect evidence from studies that have examined the associations between SES and negative emotions or cognitions and between negative emotions or cognitions and health. Cognitive and emotional factors have received consid-

enable attention as health risk factors in recent years, and a number of excellent reviews are available through which to evaluate this portion of the mediation hypothesis. Rather than duplicate this work, we provide a synopsis and update to these reviews. We address the associations between SES and cognitive–emotional factors in greater detail.

Negative Cognitive–Emotional Factors and SES

In the following sections, we review the research that has examined SES in relation to cognitive and emotional symptoms and to psychiatric disorders that have negative emotion as a primary component, which some evidence indicates could relate to health (i.e., major and minor depression, dysthymia, phobic and panic disorders, generalized anxiety disorder). We review works that have been published since 1990, as well as older studies that are frequently cited in the literature. Reviewed studies were identified through MEDLINE and PsycINFO searches, crossing the key words *hostility*, *anger*, *depression*, *anxiety*, *hopelessness*, *affective disorder*, and *anxiety disorder with socioeconomic status and social status* (searches were limited to studies that were published in the English language between 1990 and 2001 and that used an adult population sample). We identified additional studies through the ancestry method. An effort was made to avoid presenting redundant findings. Thus, when several studies from the same research were published, we attempted to present the most recent findings. Cross-sectional studies are reviewed summarily, and because of their ability to provide information concerning directionality, prospective studies are reviewed in detail. To examine the association between SES and emotional disorders, we rely primarily on evidence from the Epidemiologic Catchment Area (ECA; Robins & Regier, 1991) and National Comorbidity Studies (NCS; Kessler et al., 1994). These studies both involved the administration of structured psychiatric interviews to large probability samples of U.S. residents. The authors of the ECA administered the Diagnostic Interview Schedule for *DSM-III* (*Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed.; American Psychiatric Association, 1980) to approximately 20,000 U.S. residents over age 18. The authors of the NCS used the Composite International Diagnostic Interview for *DSM-III-R* (*Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed., rev.; American Psychiatric Association, 1987) to assess more than 8,000 U.S. residents between the ages of 15 and 54.

We rated all studies for methodological rigor on a scale of 0 to 6, according to the following: design (1 point for including a prospective component, thereby facilitating the determination of directionality), sample selection (1 point for random or population-based sampling, which would improve the generalizability of the findings), sample size (1 point for $N > 300$ and 0 points for smaller samples, which would have lower power), measure of SES (1 point for at least one continuous assessment of SES, 0 points if the study dichotomized SES, thereby attenuating power and preventing examination of a gradient effect), measure of emotion and/or cognition (1 point for use of a structured diagnostic interview or well-validated measure of symptoms and 0 points for study-specific—and hence potentially less reliable or valid—measures), and control or restriction of potential confounds (1 point for control of factors such as age, sex, and ethnicity—a procedure that would facilitate examining the “unique” effect of SES). We then grouped

studies according to whether they provided evidence of an inverse relationship between SES and the cognitive–emotional construct (i.e., a statistically significant association, at $p < .05$), mixed evidence (i.e., an inverse association in some groups but not others), or null evidence (i.e., no significant association between SES and the cognitive–emotional factor). To examine the possibility that associations depend on the type of socioeconomic indicator, we examined the proportion of positive, mixed, and null evidence separately for education, income, occupation, and other measures of SES.

Depression and Hopelessness and SES

Table 1 summarizes the research concerning the association between depression or hopelessness and SES. Nine of the studies reviewed examined the cross-sectional association between depressive or hopeless symptoms and indicators of SES. Of the five that examined education, two showed evidence of an inverse, linear association (Lynch, Kaplan, & Salonen, 1997; Salokangas & Putanen, 1998), and the remaining three identified mixed evidence (Comstock & Helsing, 1976; Craig & Van Natta, 1979; West, Reed, & Gildengorin, 1998). Three out of four studies identified an inverse association between income and depressive symptoms (Fiscella & Franks, 1997; Salokangas & Putanen, 1998; West et al., 1998), whereas one revealed mixed evidence (Comstock & Helsing, 1976). Four studies used composite measures of SES; of these, three identified an inverse association (Ickovics et al., 1997; Steele, 1978; Warheit, Holzer, & Arey, 1975), and one showed mixed evidence (Lynch, Kaplan, & Salonen, 1997). One study found an inverse association between occupational prestige and depressive and hopeless symptoms (Lynch, Kaplan, & Salonen, 1997). Thus, overall, 64% of the examined associations suggested an inverse relationship between SES and depressive symptoms, whereas 36% showed an inverse association for some groups or measures and nonsignificant associations for others. None of the studies identified completely null findings. In studies that identified an inverse relationship, the association tended to be linear, so that at each successive decrease in SES, depressive and hopeless symptoms increased. The obvious limitation of all these studies is their cross-sectional design, which impedes determination of directionality.

Seven of the reviewed studies examined SES and the prevalence of depressive disorders, as shown in Table 1. One of three that included assessments of education—Kessler et al. (1994), using the NCS—identified an inverse association, and two found no association: Bebbington, Hurry, Tennant, Sturt, and Wing (1981) and, using the ECA, Weissman, Bruce, Leaf, Florio, and Holzer (1991). The NCS also found an inverse association between income and prevalence of affective disorder (Kessler et al., 1994), whereas the ECA findings regarding income were again null (Weissman et al., 1991). Likewise, the ECA identified no association between occupation and depression prevalence (Weissman et al., 1991). Four out of six studies identified an inverse association between other indicators of SES and the prevalence of depressive disorders (Bebbington et al., 1981; Murphy et al., 1991; Weissman et al., 1991; Wilson, Chen, Taylor, McCracken, & Copeland, 1999), and two showed mixed evidence (Regier et al., 1993; Weissman & Myers, 1978). Thus, 50% of the studies identified an

(text continues on page 22)

Table 1
Research Addressing the Relationship Between Socioeconomic Status (SES) and Depression and/or Hopelessness

Study	Population/source	Design	SES measure	Depression/hopelessness measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies examining the cross-sectional association between SES and depressive or hopeless symptoms							
Constock & Helsing (1976)	3,845 residents of Kansas City, MO, and Washington County, MD, ≥ 18 yrs old	Cross-sectional	Income, education	Depressive symptoms, CES-D ≥ 16	Sex, age, marital status, employment status, income, or education	± Income: strength and pattern of association varies by ethnicity and location ± Education: strength and pattern of association varies by ethnicity and location	5
Craig & Van Natta (1979)	1,614 community residents and 45 psychiatric in-patients	Cross-sectional	Education	CES-D, prevalence (symptoms in prior week), persistence (symptoms 5-7 days in prior week)	Sex, age, marital status, clinical status	± For prevalence, inverse association in 2 of 16 symptoms; for persistence, inverse association in 10 of 16 symptoms	4/Clinical sample not selected randomly, symptoms examined individually
Fiscella & Franks (1997)	6,582 U.S. residents, 25-74 yrs old/ NHANES	Cross-sectional, 12-16-yr f/u for health outcomes only	Income (< \$10,000, ≥ \$10,000)	General Well Being Scale, high versus low depressive symptoms, hopeless affect in past month; high hopeless outlook in past week (n = 2,962)	Adjustment for oversampling and nonresponse	+ Rates of all outcomes 1.6-2.0 times higher for low income	2/Distress measured over brief time period and with minimal items; no adjustment for sex, age, ethnicity
Ickovics et al. (1997)	2,145 male post-MI patients, 29-69 yrs old Health Insurance Plan substudy of BHAT	Cross-sectional, 12-mo f/u for health outcomes only	Education and occupation composite	Three-item measure of depression, low versus high symptoms	None	+ Inverse, linear relationship	2/Brief depression measure; no controls for possible confounds; sample not selected randomly (post-MI patients)
Lynch, Krause, et al. (1997)	2,674 Finnish Men/Kuopio Ischemic Heart Disease Study	Cross-sectional	Aggregate index of recalled childhood SES Education (adolescent/early adulthood SES) Occupation (adult SES)	Depression: shortened MMPI Hopelessness: two items, study specific-measure	Age	± Childhood SES: poor- and middle-SES groups more likely to have high hopelessness, not depression + Education: gradient, inverse for hopelessness, depression + Occupation: blue collar workers (compared with white collar) with more high hopelessness, depression	5/Specific population (Finnish men); data cross-sectional, but capitalized on unique approach to capturing SES at different life stages

(table continues)

Table 1 (continued)

Study	Population/source	Design	SES measure	Depression/hopelessness measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies examining the cross-sectional association between SES and depressive or hopeless symptoms (<i>continued</i>)							
Salokangas & Poutanen (1998)	1,643 patients of community health centers, Finland, 18–64 yrs old	Cross-sectional	Education, income	Depressive symptoms, Depression Project Screening Instrument	Multivariate analysis with age, self and spouse health, alcohol, housing problems, other psychosocial factors	± Education: inverse, gradient association; nonsignificant in multivariate analysis, most likely because of strong correlation with physical health + Income: low earnings predicted higher depressive symptoms + Lowest class had higher scores compared with higher classes	4/Specific population (primary care patients); study-specific depression measure
Steele (1978)	134 upper- and upper-middle-class residents, New Haven, CT	Cross-sectional	Education and occupation composite	Zung Depression Scale and several lesser known measures	None		2/Participants not selected randomly and small sample; low social classes excluded; ethnicity confounded with social class; no controls
Warheit et al. (1975)	1,645 Florida residents	Cross-sectional	Income, occupation, education composite	18-item measure of depressive symptoms	Multivariate analysis with ethnicity, age, sex, and SES	+ Inverse, linear association	4/Study-specific measure used, but good psychometrics reported
West et al. (1998)	2,025 affluent Marin County, CA, residents, ≥ 55 yrs old	Cross-sectional	Household income, education	Depressive symptoms, CES-D ≥ 16	Age: in multivariate analysis: health conditions, behaviors, disability, social support	+ Income: inverse association up to income ≥ \$74,999; association nonsignificant in multivariate analysis, due to relationship between income and other psychosocial factors ± Education: inverse only; marginal in multivariate analysis	5
Studies examining the cross-sectional association between SES and depressive disorders							
Bebbington et al. (1981)	800 (Stage 1), 310 (Stage 2) Camberwell, England, community residents	Cross-sectional	Education, social class (occupation)	1-no prevalence of affective disorders, Present State Examination (no symptoms, minimal symptoms, below threshold, threshold disorder, definite disorder)	Sex-specific analyses, and no overall age differences were observed	- Education + Social class: significant association between total symptoms and social class, when both measured continuously	5

Table 1 (continued)

Study	Population/source	Design	SES measure	Depression/hopelessness measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies examining the cross-sectional association between SES and depressive disorders (continued)							
Kessler et al. (1994)	8,098 U.S. residents, 15–54 yrs old/NCS	Cross-sectional	Income, education	12-mo and lifetime prevalence of affective disorders (major depression, mania, dysthymia), CIDI for DSM-III-R (major depression accounted for 91% of 12-mo and 89% of lifetime prevalence)	Rates weighted for nonresponse, selection probabilities, deviation from population demographics	+ Income: for lifetime and 12-mo prevalence, lowest differed from highest income group (ORs = 1.56 and 1.73, respectively) + Education: for lifetime and 12-mo prevalence, all lower education groups differed from highest group (ORs = 1.86, 1.76, and 1.44, and 1.79, 1.38, and 1.37, respectively)	5/Lifetime diagnoses rely on memory and accuracy; affective disorders aggregated (includes mania, bipolar); rates for specific disorders not reported
Regier et al. (1993)	18,571 U.S. residents/ECA	Cross-sectional	Nam Index, composite of household income, own education, and own occupation, divided into quartiles	Major depression, dysthymia, DIS for DSM-III	Age, sex, ethnicity, marital status	± Linear inverse association between SES and OR for major depression, until highest SES when trend began to reverse; lowest differed significantly from next to highest (OR = 2.62); nonsignificant trend toward higher ORs for dysthymia in lower SES groups	5
Weissman et al. (1991)	19,182 U.S. residents/ECA	Cross-sectional	Occupation, income, education, unemployed (≥ 6 mo in past 5 yrs), financial dependence on government	1-yr prevalence of major depression, via DIS	Sex, age, ethnicity	- Occupation - Income + Unemployment (OR = 3.25) and financial dependence (OR = 2.56)	4/Crude distinctions in socioeconomic groupings (SES measures dichotomized)
Weissman & Myers (1978)	511 treated and untreated community residents	Cross-sectional (report on 8–9-yr follow-up of original sample)	Social class	Point prevalence, lifetime prevalence of major and minor depression, SADS	None	± For point prevalence, minor depression, not major depression, was more common in lower class groups. No consistent relationship for lifetime prevalence, and rates tended to be higher in upper classes	4/Many participants were lost from original cohort; differential attrition (e.g., mortality) could have affected results; no controls for possible confounds

(table continues)

Table 1 (continued)

Study	Population/source	Design	SES measure	Depression/hopelessness measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies examining the prospective association between SES and depressive disorders or symptoms							
Anthony & Petronis (1991)	5,969 U.S. residents, 18–44 yrs old/ECA	Prospective, 1-yr f/u	Employment, job prestige, education	Major depression, DIS for DSM-III	Analyzed in multiple regression with ethnicity, marital status, education, employment, job prestige; also stratified by age, census tract	+ Job prestige (inverse association) + Employment: working for pay associated with decreased incidence (RR = 0.60) – Education	6/Brief f/u period
Bruce et al. (1991)	3,497 New Haven, CT, community residents, \geq 18 yrs old/ECA	Prospective, 6-mo f/u	Poverty	Incident major depression, DIS for DSM-III	Sex, age, ethnicity, psychiatric history	+ Inverse association, with OR = 2.29 for poverty group; about 10% of cases attributable to poverty ± Education: inverse linear association for men, except the least educated had the lowest rates; positive association for women—the most educated had the highest rates – Occupation: No consistent relationship	5/Possible under reporting of past episodes (may overrepresent true incidence); brief f/u period 4/Sample nonrandomly selected, and 75% were first-degree relatives of individuals with affective disorders; completers were younger, better educated, and more often female and single than noncompleters
Coryell et al. (1992)	965 relatives (727), controls (150), and spouses (88) of persons with affective disorders who had no history of mental disorder/Collaborative Study	Prospective, 6-yr f/u	Education, occupation	SADS	Multiple logistic regression analysis with age, sex, marital status, place of residence and education or occupation		
J. J. Gallo et al. (1993)	7,737 ECA participants, \geq 40 yrs old	Prospective, 1-yr f/u	Education, unemployment	DIS for DSM-III (first-time occurrence of major depression)	Sex, marital status, minority status, neighborhood factors, employment or education	± Education: adults with < 12 yrs of education had higher risk (RR = 2.20) compared with those with \geq 12 yrs of education. F/u analyses showed an interaction with sex—association significant for women only – Unemployment: incidence not greater in unemployed	5/3,049 individuals lost to f/u (death, refusal, inability to attend the interview, or whereabouts unknown); differential attrition may have impacted the results; brief f/u period
Kaplan et al. (1987)	6,928 Alameda County, CA, residents/Human Population Laboratory	Prospective, 9-yr f/u	Education, income	Validated 18-item measure of depressive symptoms, incident “high” depression	Multivariate analyses with age, sex, education, income, ethnicity, baseline physical health, perceived health	+ Income: linear, inverse association; inadequate income group differed from very/adequate income group (RR = 1.46); nonsignificant in multivariate analysis + Education: linear, inverse association (RRs = 1.86 and 1.53 for low and medium vs. high education)	6/No interim depression assessment

Table 1 (continued)

Study	Population/source	Design	SES measure	Depression/hopelessness measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies examining the prospective association between SES and depressive disorders or symptoms (continued)							
Murphy et al. (1991)	593 rural Canadian residents	Prospective, 16-yr f/u	Material possessions (low, average, or high)	Presence of a depressive disorder (inferred through responses to a computer program)	Rates age and sex standardized	+ For prevalence at baseline and f/u, inverse association for men and women + For incidence across f/u, gradient inverse association for men and women	5/Study-specific assessment approach; no interim assessment of depression
Wilson et al. (1999)	5,222, ≥ 65 yrs old, Liverpool, England, community residents	Prospective cohort, 2-yr f/u	Townsend Index (higher scores mean more social deprivation according to census by postal codes)	Geriatric Mental State Examination (prevalent and incident depression "cases" and "subcases")	Age, sex	+ At baseline, depressive cases (OR = 5.57) and subcases (OR = 5.74) had higher Townsend Index scores compared with well participants (OR = 5.05) + Significant trend for higher odds of incident depression with increasing Townsend Index; however, most affluent group had highest odds of incident depression	6/District SES does not equate to individual SES—cannot directly link Townsend Index score and psychiatric diagnosis

Note. MO = Missouri; MD = Maryland; yr = year; CES-D = Center for Epidemiologic Studies Depression Scale; ± = mixed findings (i.e., significant inverse association for some groups but not all); U.S. = United States; NHANES = National Health and Nutrition Examination Study; f/u = follow-up; + = evidence for an inverse association at $p < .05$; MI = myocardial infarction; BHAT = Beta Blocker Heart Attack Trial; mo = month; MMPI = Minnesota Multiphasic Personality Inventory; CT = Connecticut; CA = California; - = no significant inverse association; NCS = National Comorbidity Study; CIDI = Composite International Diagnostic Interview; DSM-III-R = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); American Psychiatric Association, 1987); OR = odds ratio; ECA = Epidemiologic Catchment Area Study; DIS = Diagnostic Interview Schedule; SADS = Schedule for Affective Disorders and Schizophrenia; DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., American Psychiatric Association, 1980); RR = relative risk.

inverse association between SES and prevalence of depressive disorders, whereas 17% showed mixed evidence and 33% showed null evidence. It is notable that three of the four null associations derived from the ECA (see Weissman et al., 1991) and involved dichotomized assessments of education, income, and occupation.

As shown in Table 1, seven studies examined the association between SES and depression using prospective methods. Inasmuch as these studies demonstrate that low SES precedes the development of depressive symptoms or disorders, they provide support for the social causation hypothesis. Kaplan, Roberts, Camacho, and Coyne (1987) found a prospective relationship between SES and depressive symptoms in nearly 7,000 Alameda County, California, residents. For individuals who were not depressed at baseline, low and medium levels of education were associated with a greater risk of high depression after a 9-year follow-up period compared with rates associated with high levels of education. Inadequate income also predicted risk of high depression symptoms at follow-up. In a prospective epidemiologic study based in Stirling County (a pseudonym for the research site), Atlantic Canada, Murphy et al. (1991) used a computer algorithm to diagnose depressive disorders in 593 men and women who were then followed over 16 years. A composite assessment of material possessions served as the indicator of SES. Persons with low SES had a higher incidence of depression across follow-up. A trend for depression to predict downward social drift also emerged, but given the close association between depression and SES at baseline, the power to evaluate this effect was quite low. The level of social deprivation in participants' residential district also predicted incident major depression in a study of residents in Camberwell, England; however, the most affluent level had the highest rate of new depression cases (Wilson et al., 1999). Finally, Coryell, Endicott, and Keller (1992) reported findings from the Collaborative Study, in which the SADS was used to evaluate incident major depression in spouses, relatives, and controls (age- and sex-matched to relatives) of individuals with affective disorders. Education was inversely associated with incident depression in men but positively associated with depression in women. Occupation did not predict depression. It is important to note that the sampling strategy used in this study, as well as the differential attrition across gender and age, creates interpretive ambiguities.

The ECA also included a prospective component, which examined sociodemographic predictors of psychiatric disorder. In a 1-year follow-up of younger individuals (18–44 years old) enrolled in the ECA (Anthony & Petronis, 1991), unemployment and job prestige at the first assessment predicted new cases of major depression at follow-up, whereas education did not. Conversely, J. J. Gallo, Royall, and Anthony (1993) examined the older participants (> 40 years old) from the ECA across the same follow-up period and found that lower education predicted higher risk of first-time major depression after controlling for other factors (although this was statistically significant for women only), whereas unemployment at baseline did not. These contrasting findings suggest that distinct socioeconomic indicators could be of importance at different developmental stages or for different cohorts. In a third study from the ECA, a prospective analysis of the New Haven, Connecticut, sample showed that individuals reporting poverty-level income and no history of depression at baseline had higher rates of incident major depression across a 6-month follow-up period (Bruce, Takeuchi, & Leaf, 1991). In summary,

four of seven (57%) prospective studies found evidence of an inverse association between various indicators of SES and incident depression. The remaining three studies (43%)—two from the ECA—identified mixed findings.

In the studies that examined SES and depression using cross-sectional methods, 6 of 14 received a rating of 5 (out of a possible 6) according to our scheme for evaluating the strength of the evidence. Among the three methodologically rigorous studies that examined depressive symptoms, the evidence was mixed, with one identifying an inverse association for some subsamples but not others (Comstock & Helsing, 1976) and the remainder identifying mixed evidence across indicators (Lynch, Kaplan, & Salonen, 1997; West et al., 1998). The three rigorous studies that examined depressive disorders showed positive (Kessler et al., 1994) or mixed evidence (Bebbington et al., 1981; Regier et al., 1993). Thus, although few studies were rated as methodologically rigorous, the distribution of positive and mixed findings was roughly similar to that observed across all studies, and none of the rigorous studies identified null findings. All of the prospective studies were rated as methodologically rigorous.

In summary, the majority of the evidence suggests that individuals with low SES have higher levels of depressive symptoms and depressive disorders. The evidence is strongest for a cross-sectional association between depressive symptoms and SES and between incident depressive disorders and SES, although half of the reviewed studies also suggest that SES is associated with prevalent depressive disorders. The evidence seems most consistent for comparisons involving income or composite measures of SES, as opposed to education measures, although this pattern is by no means conclusive. Very few studies examined occupational prestige. Several studies suggest that higher SES is associated with decreasing rates of depressive disorders and symptoms only up to a high affluence level, at which point the effect appears to reverse (e.g., Regier et al., 1993; West et al., 1998; Wilson et al., 1999). Studies demonstrating that low SES precedes the development of depression suggest that social causation explains at least part of the association between the two.

Anxiety and SES

As shown in Table 2, our review revealed only two studies that examined the association between SES and anxiety symptoms. These studies suggest an inverse, linear association for education, income (Himmelfarb & Murrell, 1984), and composite social class (Warheit et al., 1975) with anxiety symptoms.

Of the eight studies reviewed that examined the cross-sectional association between SES and anxiety disorders, four derived from the NCS and three derived from the ECA. Two of four studies that included an assessment of education—both from the NCS—identified an inverse association with prevalent panic (Eaton, Kessler, Wittchen, & Magee, 1994) and phobic disorders (Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996). The ECA identified mixed evidence for an association between education and prevalent panic and phobic disorders (Eaton et al., 1991) and null evidence for education and generalized anxiety disorder (Blazer, Hughes, George, Swartz, & Boyer, 1991). Three of five studies, each from the NCS, identified an inverse, linear association between income and prevalence of various anxiety disorders (Kessler

(text continues on page 26)

Table 2
Research Addressing the Relationship Between Socioeconomic Status (SES) and Anxiety

Study	Population/source	Design	SES measure	Anxiety measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies addressing the cross-sectional association between SES and anxiety symptoms							
Himmelfarb & Murrell (1984)	2,051 Kentucky residents, ≥ 55 yrs old	Cross-sectional	Education income	Spielberger Trait Anxiety Scale	Sex-specific analyses performed; age range restricted	+ Education: inverse, linear association for men and women + Income: inverse correlation between income and anxiety + For phobic symptoms, inverse linear association leveled off at highest SES group; inverse linear association for general anxiety symptoms; for anxiety function, inverse association, but second lowest SES group had the highest score	5 4/Study-specific measures used, but good psychometrics reported
Warheit et al. (1975)	1,645 Florida residents	Cross-sectional	Income, occupation, and education composite	Phobic symptoms (10 items), general anxiety, symptoms (12 items), anxiety function (11 items)	Multivariate analysis with ethnicity, age, sex, and SES		
Studies addressing the cross-sectional association between SES and anxiety disorders							
Blazer et al. (1991)	8,205 U.S. residents/ECA	Cross-sectional	Income, occupation, education, financial dependence on government	1-yr prevalence of GAD, DIS	Rates weighted to site demographics	± Income: overall inverse association, but variable pattern - Occupation: inverse association, but nonsignificant - Education + Financial dependence: 3.8% versus 1% in Durham, NC, 4.6% versus 1% in Los Angeles	5/Data collected in only three ECA sites and during second study wave only; assessment of GAD varied across sites
Eaton et al. (1991)	19,498 U.S. residents (panic); 14,263 U.S. residents (phobia)/ECA	Cross-sectional	Education, occupation, financial dependence on government	1-yr prevalence of panic and phobic disorders, DIS for DSM-III	Sex-specific analyses performed; report that age adjustment did not affect education results	± Education: trend toward inverse association for men only, phobic disorders only; ethnicity-specific analyses show significant trend for Whites only - Occupation: no association for phobic disorder; for panic, trend toward inverse association in men, positive association in women + Financial dependence: inverse association for phobia and panic disorders, both men and women	5/Relatively low reliability assessments for panic diagnoses from DIS

(table continues)

Table 2 (continued)

Study	Population/source	Design	SES measure	Anxiety measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies addressing the cross-sectional association between SES and anxiety disorders (continued)							
Eaton et al. (1994)	8,098 U.S. residents, 15–54 yrs old/NCS	Cross-sectional	Education, income	Prevalence of panic attacks, disorder, and panic with agoraphobia in past month, CIDI	Rates weighted for nonresponse, selection, population demographics; Report that multivariate adjustment had negligible effect	+ Education: significant inverse association; least educated group > 4 times likely to have a panic attack, > 10 times likely to have panic disorder, > 7 times likely to have panic with agoraphobia compared with college educated – Income: no association + Income: for lifetime prevalence, all lower differed from highest income group ($\geq \$70,000$; ORs = 2.00, 1.52, and 1.48 for lowest to second highest income groups); similar for 12-mo prevalence (ORs = 2.12, 1.56, and 1.50) + Education: inverse, linear association for lifetime prevalence (ORs = 1.86, 1.76, and 1.44 for lowest to second highest educated groups); similar for 12-mo prevalence (ORs = 2.82, 2.10, and 1.60)	5
Kessler et al. (1994)	8,098 U.S. residents, 15–54 yrs old/NCS	Cross-sectional	Income, education	Lifetime and 12-mo prevalence of any anxiety disorder, CIDI	Rates weighted for nonresponse, selection probabilities, deviation from population demographics	– Income: no association + Income: for lifetime prevalence, all lower differed from highest income group ($\geq \$70,000$; ORs = 2.00, 1.52, and 1.48 for lowest to second highest income groups); similar for 12-mo prevalence (ORs = 2.12, 1.56, and 1.50) + Education: inverse, linear association for lifetime prevalence (ORs = 1.86, 1.76, and 1.44 for lowest to second highest educated groups); similar for 12-mo prevalence (ORs = 2.82, 2.10, and 1.60)	5/Lifetime diagnoses rely on memory and accuracy
Magee et al. (1996)	8,098 U.S. residents, 15–54 yrs old/NCS	Cross-sectional	Education, income	30-day prevalence of agoraphobia, social phobia, simple phobia, CIDI	Rates weighted for nonresponse, selection probabilities, deviation from population demographics	+ Education: inverse linear association for agoraphobia, simple phobia, and social phobia + Income: inverse linear association for agoraphobia, simple phobia, and social phobia + For phobia, inverse linear relationship between SES and OR of 1-mo phobia (lowest = 2.43; second lowest = 1.84); SES quartiles had higher OR compared with highest quartile; linear inverse relationship for panic but only lowest quartile differed significantly from highest (OR = 11.58)	5
Regier et al. (1993)	18,571 U.S. residents/ECA	Cross-sectional	Nam Index, composite of household income, own education, and occupation, divided into quartiles	1-mo prevalence of phobia, panic, DIS for DSM-III	Age, sex, ethnicity, marital status	+ For phobia, inverse linear relationship between SES and OR of 1-mo phobia (lowest = 2.43; second lowest = 1.84); SES quartiles had higher OR compared with highest quartile; linear inverse relationship for panic but only lowest quartile differed significantly from highest (OR = 11.58)	5/Relatively low reliability assessments for panic diagnoses from DIS

Table 2 (continued)

Study	Population/source	Design	SES measure	Anxiety measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies addressing the cross-sectional association between SES and anxiety disorders (continued)							
Witchen et al. (1994)	8,098 U.S. residents, 15–54 yrs old/NCS	Cross-sectional	Income	Lifetime prevalence of GAD, CIDI	Multivariate analysis controlling for sex, age, ethnicity, marital status, employment, and region	+ Inverse linear association; lowest differs from highest income group; effect nonsignificant in multivariate analysis	5/Lifetime diagnoses relied on memory, accuracy
Studies examining the prospective association between SES and anxiety disorders							
Bruce et al. (1991)	3,497 New Haven, CT residents, ≥ 18 yrs old/ECA	Prospective, 6-mo f/u	Poverty	Incident panic, phobia, DIS	Sex, age, ethnicity, psychiatric history	– Nonsignificant inverse association for both, with 1.7% and 6.7% of cases attributable to poverty for panic and phobia, respectively	5/Relatively low reliability assessments for panic diagnoses from DIS
Eaton & Keyl (1990)	11,756 U.S. residents/ECA	Prospective, 1-yr f/u	Education, occupation	1-yr incidence of agoraphobia, DIS for DSM-III, split among situational cases, classic cases (fear of going out alone), and cases with panic	Age, gender, ethnicity, marital status, living situation, education, or occupation	± Education: tendency for fewer education (1–9 yrs) to predict increased incidence of agoraphobic cases, but this was significant for “classic” agoraphobia only (OR = 1.77) ± Occupation: tendency for higher occupational rank to be protective, but again, this was significant for “classic” agoraphobia only (OR = 0.86)	6/Education was dichotomized; brief f/u period; possible underreporting of past episodes could have inflated incidence rates
Keyl & Eaton (1990)	12,823 U.S. residents/ECA	Prospective, 1-yr f/u	Occupation	Incident panic disorder, severe unexplained panic attacks, other panic attacks, DIS for DSM-III	Age, ethnicity, marital status	± Increasing occupational prestige was associated with lower odds of incident panic disorder (OR = 0.80), but the effects for other types of panic were nonsignificant	6/Brief f/u period; relatively low reliability assessments for panic diagnoses from DIS
Murphy et al. (1991)	593 rural Canadian residents	Prospective, 16-yr f/u	Material possessions (low, average, high)	Presence of an anxiety disorder (computer assessment)	Rates were standardized by age and sex	– No association for prevalence at baseline; a marginal association for prevalence at f/u; no association for incidence at f/u	5/Study-specific assessment; few incident cases
J. C. Wells et al. (1994)	9,437 U.S. residents/ECA	Case-control, 1-yr f/u	Education	1-yr incidence of social phobia, DIS for DSM-III	Gender, marital status, age (living alone, rural vs. nonrural residence did not predict social phobia)	+ Inverse relationship. RR for 0–8 yrs of education (vs. 13+) = 2.14. RR for 9–12 yrs of education (vs. 13+) = 2.93	6/Brief f/u period; site differences were observed, the meaning of which are unclear

Note. yr = year; + = evidence for an inverse association at $p < .05$; U.S. = United States; ECA = Epidemiologic Catchment Area Study; GAD = generalized anxiety disorder; DIS = Diagnostic Interview Schedule; ± = mixed findings (i.e., significant inverse association for some groups but not all); – = no significant inverse association; NC = North Carolina; DSM-III = Diagnostic and Statistical Manual of Mental Disorders (3rd ed.); American Psychiatric Association, 1980); NCS = National Comorbidity Study; CIDI = Composite International Diagnostic Interview; mo = month; OR = odds ratio; CT = Connecticut; f/u = follow-up; RR = relative risk.

et al., 1994; Magee et al., 1996; Wittchen, Zhao, Kessler, & Eaton, 1994); studies from the ECA identified mixed findings (Blazer et al., 1991) and null findings for the income and anxiety disorder association (Eaton et al., 1994). The two studies from the ECA also identified null associations between occupation and prevalent anxiety disorders (Blazer et al., 1991; Eaton et al., 1994). The ECA did find a positive association between financial dependence on the government and 1-year prevalence rates of generalized anxiety disorders (Blazer et al., 1991) and phobic and panic disorders (Eaton et al., 1991) and an association between a composite assessment of SES (i.e., the Nam index) and 1-month prevalence of panic and phobic disorders (Regier et al., 1993). Murphy and colleagues (1991) found no association between material possessions and prevalent anxiety disorders at a baseline assessment, and only a marginal inverse association at a follow-up assessment. Thus, the majority of the comparisons (53.3%) identified inverse associations between indicators of SES and prevalent anxiety disorders; 13.3% identified mixed findings, and 33.3% identified null findings.

As shown in Table 2, five studies examined whether SES predicts incident anxiety disorders, four of which represent findings from the ECA. In a 6-month longitudinal study of the New Haven ECA sample, poverty (i.e., very low income) did not significantly predict incident panic or phobic disorders (Bruce et al., 1991). However, few new cases were observed in this brief follow-up period, suggesting that low power might have contributed to the null results (Bruce et al., 1991). Eaton and Keyl (1990) examined the associations between occupation and education and 1-year incidence of agoraphobia in the ECA. Lower SES tended to predict a higher incidence of agoraphobia, but this was statistically significant only for "classic" agoraphobia (i.e., the fear of going out alone), which is the most debilitating type (Eaton & Keyl, 1990). Keyl and Eaton (1990) also found a prospective inverse association between occupational prestige and 1-year incidence of panic disorder but not other types of panic attacks. J. C. Wells, Tien, Garrison, and Eaton (1994) found that lower education was associated with higher incidence rates of social phobia over a 1-year follow-up of the ECA participants. Finally, Murphy and colleagues (1991) incorporated an assessment of anxiety disorders in their prospective study of SES and psychiatric status and found that SES did not show a clearly patterned association with incident anxiety disorders. In this longitudinal study, neither the social drift nor the social causation hypothesis received substantial support. Thus, of the six comparisons within, 17% (i.e., one) supported an inverse association between SES and incident anxiety disorders, 50% showed mixed findings, and 33% showed null findings. All but one of the mixed or null findings derived from studies associated with the ECA.

In summary, although based on only two studies, our review of this topic does suggest that lower levels of SES are associated with higher levels of anxiety symptoms. More than half of the reviewed comparisons also suggested an inverse association between SES and prevalent anxiety disorders. In contrast, only one of six comparisons suggested that lower SES leads to a higher incidence of anxiety disorders, with half of the comparisons providing mixed evidence. Thus, the extent to which a social causation versus a social drift interpretation explains the association between anxiety and SES is unclear. Overall, the mixed findings concerning SES and anxiety disorder are not easily explained in terms of type of

socioeconomic indicator or type of anxiety disorder. All but one of the studies concerning SES and anxiety received a strength of evidence rating of 5 or 6.

Hostility and SES

As shown in Table 3, all of the studies that examined the association between SES and hostility used cross-sectional approaches. Four studies examined the association between SES and scores on the Ho. The studies were consistent in identifying a linear, inverse relationship between education (Barefoot et al., 1991; Lynch, Kaplan, & Salonen, 1997; Scherwitz, Perkins, Chesney, & Hughes, 1991), income (Barefoot et al., 1991; Lynch, Kaplan, & Salonen, 1997), and occupation (Barefoot et al., 1991) and scores on the Ho full and individual subscales. In addition, Carmelli, Rosenman, and Swan (1988) found an inverse association between scores on the Hollingshead (1975) measure of SES and on the Ho, and Lynch, Kaplan, and Salonen (1997) showed that recalled childhood SES related to adult Ho scores. As noted above, the Ho is primarily a measure of the cognitive aspects of the hostility construct. Thus, this research suggests that individuals with lower SES tend to maintain attitudes of cynical distrust about others. In addition, the studies by Barefoot et al. (1991) and Scherwitz et al. (1991) showed that these inverse associations extended to subscales from the Ho that assess behavioral and affective components of hostility.

Four studies using measures of hostility besides the Ho also showed an inverse relationship with SES. Ranchor, Bouma, and Sanderman (1996) found inverse associations between education and occupation and scores on a multidimensional measure of anger and hostility. Matthews, Kelsey, Meilahn, Kuller, and Wing (1989) showed that women with lower levels of education had higher scores on measures of trait anger and the tendency to suppress—but not express—anger. Finally, Mittleman, Maclure, Nachnani, Sherwood, and Muller (1997) found that less-educated individuals were more likely to report experiencing an episode of anger prior to MI compared with their more highly educated counterparts.

In summary, 11 of 12, or 92%, of the comparisons concerning SES and hostility identified inverse and generally linear relationships, and the remaining study identified an inverse relationship for two of three anger scales. The strength of the evidence was mixed, with only three studies receiving a methodological rating of 5. However, given the consistency of the findings, the association is likely to be reliable. Thus, SES appears to be associated with the cognitive, affective, and behavioral correlates of hostility. Of note, two studies suggested that these associations might be stronger in ethnic minorities (Barefoot et al., 1991; Scherwitz et al., 1991). Because all of the reviewed studies concerning SES and hostility used cross-sectional methods, the direction of this association cannot be determined. However, some research suggests that the association between SES and hostility might begin in childhood (Gump et al., 1999; Lynch, Kaplan, & Salonen, 1997). Longitudinal research is needed to further explore the temporal association between SES and hostility.

Summary

The reviewed literature suggests that an association between SES and negative emotions and attitudes is likely. The evidence is particularly strong for an association between low SES and de-

Table 3
Research Addressing the Relationship Between Socioeconomic Status (SES) and Hostility

Study	Population/source	Design	SES measure	Hostility measure	Controls	Evidence for: inverse association	Strength of evidence/limitations
Studies examining the cross-sectional association between SES and hostility as assessed by the Cook–Medley Hostility Inventory (Ho)							
Barefoot et al. (1991)	2,536 U.S. residents, 18–90 yrs old	Cross-sectional	Income, occupation, education	Ho full scale and subscales	Multivariate analysis with age, sex, ethnicity, income, occupation, marital status, and education	+ Income: inverse linear association for full scale, Cynicism, Hostile Attribution, Aggressive Responding subscales, but weaker for Social Avoidance, Other; interaction between ethnicity and income for full scale: association for non-Whites only + Occupation: inverse linear association, but significant for full scale only + Education: inverse, linear association for full scale, Cynicism, Hostile Attribution, Aggressive Responding, but weaker for Social Avoidance, Other	5
Carmelli et al. (1988)	37 mono- and 60 dizygotic White male twin pairs, CA residents	Cross-sectional	Hollingshead	Ho full scale, Cynicism and Paranoid Alienation subscales	Age	+ Significant inverse association between Hollingshead ranking and full scale as well as subscale scores	3/Small sample, not selected randomly
Lynch, Krause, et al. (1997)	2,674 Finnish Men/Kuopio Ischemic Heart Disease Study	Cross-sectional	Aggregate index of recalled childhood SES, early adulthood SES), occupation (adult SES)	Abbreviated Ho	Age	+ Childhood SES: poor- and middle-SES groups have more high hostility + Education: Gradient, inverse association between education and hostility; least educated (compared with most educated) more likely to have high hostility + Occupation: blue collar workers (compared with white collar) more likely to have high hostility	5/Specific population (Finnish men); data cross-sectional, but capitalized on unique approach to capturing SES at different life stages

(table continues)

Table 3 (continued)

Study	Population/source	Design	SES measure	Hostility measure	Controls	Evidence for inverse association	Strength of evidence/limitations
Studies examining the cross-sectional association between SES and hostility as assessed by the Cook-Medley Hostility Inventory (Ho; <i>continued</i>)							
Scherwitz et al. (1991)	5,115 residents of Birmingham, AL, Chicago, IL, Minneapolis, MN, Oakland, CA, 18-30 yrs old/CARDIA	Cross-sectional	Education (high school or less, more than high school)	Ho full scale and subscales	Multivariate analysis with ethnicity, gender, age, education, all interaction effects	+ Inverse association for full scale and all subscales except Social Avoidance (i.e., Cynicism, Hostile Attributions, Hostile Affect, Aggressive Responding, Other); associations stronger for non-Whites than for Whites	4/Education dichotomized
Studies examining the cross-sectional association between SES and scores on other measures of hostility							
Matthews et al. (1989)	541 women, Pittsburgh, PA, 42-50 yrs old/Healthy Women Study	Cross-sectional	Education	Spielberger Trait Anger Scale, Anger-Out and Anger-In subscales	None	± Inverse association with linear trend for Spielberger Trait Anger and Anger-In; no association for Anger-Out	4/Sample was healthier and better educated than nonparticipants; no controls for possible confounds
Mittleman et al. (1997)	1,623 post-MI patients	Case crossover	Education	Onset Anger Scale (one item), Anger-In 2 hr preceding MI	Separate analyses of groups differing in aspirin use, hypertension, diabetes, overweight	+ Inverse, gradient association between education and anger triggering in all analyses	2/Retrospective report of anger; single-item assessment; specific population, not selected randomly (post-MI patients)
Ranchor et al. (1996)	6,989 men, the Netherlands, 30-70 yrs old	Cross-sectional	Education, occupation	Buss-Durkee Aggression, Resentment, and Suspicion Scales from factor analysis	Age, social desirability	+ Education: inverse, linear association for all three subscales + Occupation: inverse, linear association for all three subscales	5/Specific population (Dutch men)

Note. U.S. = United States; yr = year; + = evidence for an inverse association at $p < .05$; CA = California; AL = Alabama; IL = Illinois; MN = Minnesota; CARDIA = coronary artery risk development in young adults; PA = Pennsylvania; ± = mixed finding (i.e., significant inverse association for some groups but not all); MI = myocardial infarction.

pressive, hopeless, anxious, and hostile symptoms. Some evidence also suggests that SES exhibits a cross-sectional association with depressive and anxiety disorders, but these findings are less consistent. Likewise, some research suggests that low SES precedes the development of depressive symptoms and disorders and, to a lesser extent, anxiety disorders. These studies suggest the role of social causation in connecting SES with negative cognitive–emotional factors. Our review points to a number of areas for further research, including (a) additional studies examining the association between SES and anxiety symptoms, (b) further research concerning the prospective association between SES and cognitive and emotional symptoms, and (c) continued research into the socioeconomic correlates and consequences of emotional disorders.

It is interesting that the NCS provided substantially stronger evidence of an association between SES and anxiety disorders compared with the ECA. A similar difference, though perhaps not as marked, emerged in relation to depressive disorders. In brief, differences between the studies include the fact that the NCS examined a younger age distribution, presented all screening items at the outset of the interview to avoid participants' tendency to minimize pathology to shorten the interview, and included adjustments for nonparticipants' higher rates of disorder (Eaton et al., 1994). In addition, the NCS was based on *DSM-III-R* (American Psychiatric Association, 1987) criteria, whereas the ECA was based on *DSM-III* (American Psychiatric Association, 1980) criteria. The NCS also included more thorough probing, for example, in relation to phobias and in respect to lifetime recall, and more stem questions, for example, in relation to major depression (Kessler et al., 1994). Overall, the NCS identified higher prevalence rates for many disorders than the ECA (e.g., Kessler et al., 1994). Hence, increased power is probably one factor explaining the stronger associations observed in the NCS. Further research is needed to determine which study has provided the more accurate assessment of links between SES and prevalent and incident anxiety and depressive disorders.

Negative Emotions and Cognitions and Health

If negative emotions and cognitions are important in understanding the SES–health link, research should show that these factors predict verifiable health outcomes. In the following sections, we discuss this evidence, focusing on cardiovascular diseases and all-cause mortality. To demonstrate that these factors predict these outcomes, we draw on the work of other researchers who have provided recent, detailed reviews concerning the health correlates of depression (e.g., Glassman & Shapiro, 1998; Wulsin, Vaillant, & Wells, 1999), anxiety (e.g., Fleet & Beitman, 1998; Hayward, 1995; Kubzansky, Kawachi, Weiss, & Sparrow, 1998; Rozanski, Blumenthal, & Kaplan, 1999), and anger and hostility (e.g., Miller, Smith, Turner, Guijarro, & Hallet, 1996; Rozanski et al., 1999). In addition, we discuss prospective studies that have been performed since the publication of these reviews.

Depression and Hopelessness and Health

A number of reviews have summarized the evidence linking depression and hopelessness with all-cause mortality and with cardiovascular morbidity and mortality. It is not surprising that

these reviews overlap considerably. Rozanski et al. (1999) evaluated the prospective evidence assessing the effects of depression and hopelessness on cardiovascular outcomes. They limited their focus to studies that included a “hard” endpoint, such as MI or cardiac death. The studies varied according to whether they assessed depressive or hopeless symptoms or clinical depression (i.e., major depression, dysthymia). Depression or hopelessness predicted increased risk of future coronary events in six of eight studies of community populations (Anda et al., 1993; Aromaa et al., 1994; Barefoot & Schroll, 1996; Everson et al., 1996; Ford et al., 1998; Pratt et al., 1996). Relative risks for depressed versus nondepressed individuals ranged from 1.5 (for depressive symptoms; Anda et al., 1993) to 4.5 (for major depression; Pratt et al., 1996). Mixed findings were reported for a study by Wasserthal-Smoller and colleagues (1996), who found that depressive symptoms did not relate to cardiovascular outcomes during the relatively brief 4.5-year follow-up period but that increases in depressive symptoms predicted cardiovascular events. Negative findings were identified by Vogt, Pope, Mullooly, and Hollis (1994), who examined a study-specific measure of depression in relation to all-cause mortality. All reviewed studies that examined effects of depression on outcomes in coronary heart disease (CHD) patient populations found positive evidence for an association (Ahern et al., 1990; Barefoot et al., 1996; Carney, Rich, Freedland, & Sanai, 1988; Denollet & Brutsaert, 1998; Frasure-Smith, Lespérance, Juneau, Talajic, & Bourassa, 1999; Frasure-Smith, Lespérance, & Talajic, 1995a; Hermann et al., 1998; G. J. Kennedy et al., 1987). Rozanski and colleagues concluded that overall, there is support for an association between major depression as well as depressive symptoms and future coronary events. They noted that a number of studies suggest a dose–response relationship between hopelessness or depression and level of risk (e.g., Anda et al., 1993; Barefoot & Schroll, 1996; Everson et al., 1996; Pratt et al., 1996).

Musselman, Evans, and Nemeroff (1998) provided a detailed review of studies published between 1966 and 1997, which evaluated the prospective association of depressive symptoms, including hopeless cognition, or major depression, with cardiovascular outcomes. These studies generally concerned community populations, although one study examined hypertensive patients (Simonick, Wallace, Blazer, & Berkman, 1995). Major depression or depressive symptoms predicted incident cardiovascular disease or cardiac death in 10 of 13 studies. The consistency of these findings is notable given the rigorous methodological restrictions imposed by Musselman et al. Specifically, the review included only those studies that controlled for other cardiovascular risk factors (e.g., hypertension, hypercholesterolemia, smoking history) and socio-demographic characteristics (e.g., age, sex, SES). The authors concluded that depression is “an independent risk factor in the pathophysiologic progression of CVD [cardiovascular disease], rather than merely a secondary emotional response to the illness” (Musselman et al., 1998, p. 581).

In another review, Glassman and Shapiro (1998) reported that 9 of 10 studies comparing patients with major depression with general population samples found increased risk of cardiovascular mortality in depressed patients. Two community studies that lacked controls for smoking also found increased mortality in depressed individuals, and six of seven community studies that controlled for smoking and other cardiovascular risk factors found

increased cardiovascular morbidity and/or mortality in depressed individuals. Finally, six studies of cardiac patients found excess risk of death for depressed individuals, after controlling for other cardiovascular risk factors and medical and social variables. Glassman and Shapiro noted that the risk of cardiovascular mortality was about 1.5–2.0 times higher for depressed than for nondepressed “healthy” individuals and about double that for depressed versus nondepressed patients recovering from MI.

Finally, a detailed and comprehensive review by Wulsin et al. (1999) examined the association between depression and all-cause mortality in 57 studies published between 1966 and 1996. The authors rated each study in terms of methodological rigor, according to the following criteria: (a) sample size, (b) measure of depression (with a structured interview given the highest weight and a self-report measure the lowest weight), (c) chosen comparison group (with matched control given the highest weight, cohort the next highest, and a general population control group the lowest weight), and (d) factors controlled for, with the highest rating given to studies that accounted for age, sex, and at least two of the following: physical illness, smoking, alcohol, and suicide. Twenty-nine of the studies (51%) provided consistent positive evidence, 13 (23%) provided consistent negative evidence, and 15 (26%) provided mixed evidence for an association between depression and mortality. It is important to note, however, that only 37% of the examined studies met the authors’ criteria for “better” methodology. The relative percentage of positive (48%), negative (29%), and mixed findings (23%) was similar among these studies, though slightly less supportive of an independent effect of depression. Wulsin et al. also examined specific causes of death, including cardiovascular causes. They reported that 60% (15 of 25) of studies examining depression and cardiovascular mortality identified positive evidence for an association between them, with the remainder split between negative and mixed findings. Wulsin et al. concluded that suicide seemed to explain only a small amount of the association between depression and mortality and that mixed findings were not well explained, either in terms of an identifiable pattern or in terms of hypotheses put forth by the reviewed studies’ authors. Their overall conclusions were that depression has a substantial effect on mortality in certain populations and that it appears to be linked most closely with cardiovascular deaths. They recommended future research that follows a large sample using longitudinal design while controlling for important confounds.

Since the publication of these reviews, several studies have examined the association between depression and mortality or cardiovascular disease using a prospective design. Jonas and Mussolino (2000) examined stroke incidence in a probability sample of 6,095 Black and White U.S. residents. After adjustment for diverse potential confounds, individuals with high depressive symptoms had a higher risk (between 50% and 160%) of stroke over the next 22 years compared with individuals with low depressive symptoms. This association was present in all gender and ethnicity groups and was linear in nature. A study of 573 older hospitalized patients (Covinsky et al., 1999) found that patients who had more depressive symptoms when admitted were more likely to die during a 3-year follow-up than were patients with fewer symptoms. This excess risk was attenuated but remained statistically significant after controlling for potential confounding factors, including sociodemographics, indicators of illness severity, and functional status. Again, the association between depres-

sion and risk of death was linear in nature. Another study (Lespérance, Frasere-Smith, Juneau, & Thérioux, 2000) found that patients hospitalized with unstable angina and who had high BDI scores were more than four times as likely to experience a subsequent morbid or mortal coronary event compared with nondepressed patients, even after controlling for other identified predictors (e.g., left ventricular ejection fraction, number of diseased vessels, electrocardiographic evidence of ischemia). Finally, Penninx et al. (2001) found an increased risk of cardiac mortality in initially healthy, community-dwelling, depressed older persons and in those with diagnosed cardiac disease in a 50-month prospective study. Individuals with minor depression (as diagnosed according to CES-D score cutoffs) showed moderate levels of risk, and the associations persisted after control for diverse potential confounds. Contradictory data were presented in a recently published 12-month prospective study of a small sample of MI patients that found that depressive symptoms (and anxiety symptoms) failed to predict mortality (Lane, Carroll, Ring, Beevers, & Lip, 2001).

In conclusion, although mixed findings have appeared in the literature, the weight of the evidence supports the conclusion that depression and hopelessness predict negative health outcomes. The support for an association between depression and hopelessness and cardiovascular morbidity and mortality seems especially compelling, with more conflicting findings emerging in the literature assessing mortality from all causes. A number of more recent studies that used rigorous methodologies to examine depression or hopelessness and outcomes in relatively large community or patient populations provide especially convincing evidence that these constructs are important in the etiology and course of cardiovascular disease (e.g., Anda et al., 1993; Barefoot & Schroll, 1996; Everson et al., 1996; Everson, Roberts, Goldberg, & Kaplan, 1998; Frasere-Smith, et al., 1993, 1995a, 1999; Jonas & Mussolino, 2000; Penninx et al., 2001; Pratt et al., 1996). Nevertheless, as noted by Wulsin and colleagues (1999), mixed findings and a plethora of less methodologically sound research suggest the need for additional longitudinal research.

Anxiety and Health

Several recent reviews summarize the evidence concerning the health implications of anxiety. Rozanski and colleagues (1999) examined nine studies investigating the association between anxiety and cardiovascular outcomes. Three studies of community populations showed a positive association between phobic anxiety symptoms at baseline and risk of cardiac death during follow-up (Haines, Imeson, & Meade, 1987; Kawachi, Sparrow, Vokonas, & Weiss, 1994; Kawachi, Colditz, et al., 1994), even after control for a variety of potential confounds. Level of anxiety appears to exert a graded, or dose–response effect on mortality risk (Kawachi, Colditz, et al., 1994; Kawachi, Sparrow, et al., 1994). It is notable that these studies support an association between anxiety and sudden coronary death rather than nonfatal coronary heart disease, suggesting the possible role of fatal ventricular arrhythmia triggered by acute anxiety (e.g., Amsterdam, 1990; Lown, 1982). Another community study (Kubzansky et al., 1997) found that level of worry, a symptom of generalized anxiety disorder, predicted incident MI and marginally predicted incident CHD. Again, results suggest a dose–response association. Finally, Rozanski et

al. reviewed the findings of a community study (Weissman, Markowitz, Ouellette, Greenwald, & Kahn, 1990) that found increased risk of MI for people with panic disorder compared with those without panic disorder. Rozanski et al.'s review also showed that in three recent studies of patients recovering from MI, anxiety symptoms predicted physical outcomes (Denollet & Brutsaert, 1998; Frasure-Smith, Lespérance, & Talajic, 1995b; Moser & Dracup, 1996). In these studies, patients with high levels of anxiety had between 2.5 and 4.9 times the likelihood of negative outcomes including unstable angina, re-infarction, and cardiac death after controlling for other risk factors as compared with patients with lower anxiety. Finally, a study of medical in-patients (Hermann et al., 1998) showed that those with high levels of anxiety symptoms who were admitted with cardiopulmonary diagnoses were significantly more likely to die during follow-up than their less anxious counterparts. Rozanski et al. suggested that large-scale epidemiological studies of coronary artery disease patients should be attempted and that studies of women in particular are needed.

Fleet and Beitman (1998) provided a critical review and analysis of the six studies that, before 1997, examined the association between panic disorder or panic-like anxiety symptoms and death from cardiovascular causes. Three of these studies used retrospective methodologies. In the first, male but not female in-patients with probable panic disorder (according to chart review) had increased risk of death from "circulatory disease" across follow-up compared with age- and gender-matched population mortality rates (Coryell, Noyes, & Clancy, 1982). Similar results were identified by the same group of researchers in a retrospective study of out-patients with probable panic disorder (Coryell, Noyes, & House, 1986). As discussed by Fleet and Beitman, the methodological limitations of these studies include the following: (a) current diagnostic criteria for panic may not have been met; (b) *circulatory disease* was not defined, and the number of deaths by circulatory causes was quite small; (c) the contribution of medical and psychiatric disorders that developed across follow-up was not assessed; and (d) the contribution of other coronary risk factors, such as smoking, was not assessed. In the third retrospective study of a random sample of more than 18,000 adults (Weissman et al., 1990), panic disorder was assessed by structured interview, assuring adherence to *DSM-III* criteria. Patients with panic were more likely to have experienced hypertension, heart attack, or stroke, according to their own self-reports, after adjustment for a number of demographic variables. The use of self-reported outcomes is an important limitation of this study. In addition, the interview assessed lifetime diagnoses, making it impossible to identify the direction of any association between psychiatric problems and health outcomes.

Fleet and Beitman (1998) also reviewed three prospective studies (Haines et al., 1987; Kawachi, Colditz, et al., 1994; Kawachi, Sparrow, et al., 1994) that were likewise described by Rozanski and colleagues (1999). Consistent with this review, Fleet and Beitman concluded that the research suggests a dose-response relationship between symptoms of anxiety and future risk of sudden coronary death. Furthermore, they pointed out that associations persisted even after controlling for a wide range of potential confounds (e.g., smoking, blood pressure, family history of CHD). However, they also noted the possible limited clinical implications of the findings in light of the fact that very few sudden cardiac deaths occurred in any of the studies. In addition, the studies

assessed symptoms of anxiety, which do not necessarily constitute a panic disorder or any other anxiety disorder.

Finally, Kubzansky et al. (1998) provided a comprehensive review of studies published between 1980 and 1996 as well as especially relevant earlier reports of the association between anxiety and CHD. Many of the studies reviewed, including the three major prospective community studies, have already been discussed in this article. The authors also described the findings of Eaker, Pinsky, and Castelli's (1992) follow-up from the Framingham Study of factors predicting incident CHD (MI, cardiac death) in women across 20 years; anxiety and tension symptoms were associated with excess risk of incident CHD in homemakers, but not in employed women, after controlling for a wide range of additional risk factors. Kubzansky et al. (1998) also summarized three earlier prospective studies that found no relationship between anxiety and CHD (Allgulander & Lavori, 1991; Martin, Cloninger, Guze, & Clayton, 1985; Wheeler, White, Reed, & Cohen, 1950). All three of these examined psychiatric patient populations. However, the review authors delineated numerous methodological limitations of these studies. For example, standardized mortality rates were examined rather than deaths in appropriate control samples. One of the studies had a relatively high number of deaths due to suicide (Allgulander & Lavori, 1991), and another study had no CHD cases in patients with panic disorder (Martin et al., 1985). In concluding their review, Kubzansky et al. (1998) noted that anxiety is a potentially important risk factor for CHD and that further, interdisciplinary research is needed. In particular, the authors called for research including women and studies examining the effects of nonpathological levels of anxiety. In addition, they suggested research that allows the effects of interrelated negative emotions (i.e., depression, anxiety) to be distinguished.

To our knowledge, no prior reviews have aggregated the findings assessing the association between anxiety and all-cause mortality. Our examination of the literature suggested only limited available evidence. Murphy, Monson, Olivier, Sobol, and Leighton (1987) found no association between presence of an anxiety disorder at baseline and later mortality in either men or women. Two studies (Kubzansky et al., 1997; Vogt et al., 1994) reported no association between baseline levels of worry and mortality during follow-up. In contrast, in their analysis of the association between psychiatric status and 9-year mortality rates from the New Haven ECA, Bruce, Leaf, Rozal, Florio, and Hoff (1994) found that individuals with a history of panic disorder were approximately three times more likely to die during follow-up, after adjustment for age and sex, than individuals without a history of panic disorder. A history of phobia did not predict mortality risk. Denollet and Brutsaert (1998) reported that Type D personality (i.e., high trait anxiety and social inhibition) predicted an increased risk of death from noncardiac as well as cardiac mortality in patients with established CHD, even after control for severity of disease.

In summary, several well-designed, prospective studies of community populations suggest that higher levels of anxiety symptoms confer excess risk for negative cardiac outcomes, and in particular, sudden coronary death. The evidence for an association between anxiety and mortality from all causes is less compelling, as is the research concerning anxiety disorders and health outcomes. Additional research is needed to confirm whether the health effects of anxiety extend to women.

Hostility and Health

Like the research concerning depression and anxiety, the most recent review of hostility and health was performed by Rozanski and colleagues (1999). These authors discussed 11 studies that examined the association between hostility and cardiovascular outcomes or all-cause mortality in healthy samples and noted that the findings from this research are quite mixed. For example, of eight studies that administered the Ho to healthy samples, four (Hearn, Murray, & Luepker, 1989; Leon, Finn, Murray, & Bailey, 1988; Maruta et al., 1993; McCranie, Watkins, Brandsma, & Sisson, 1986) found no evidence for an association with cardiovascular health outcomes or all-cause mortality, whereas the other four found that Ho scores predicted cardiovascular outcomes (Barefoot, Larsen, von der Lieth, & Schroll, 1995), all-cause mortality (Barefoot et al., 1989), or both (Barefoot et al., 1983; Shekelle, Gale, Ostfeld, & Paul, 1983). Among the remaining three studies that used measures other than the Ho scale, one identified a marginal association between hostility and all-cause mortality (Koskenvuo et al., 1988), one found that cynical distrust predicted MI and cardiac death, but only before other risk factors were statistically controlled (Everson et al., 1997), and the final study found a positive and graded association between anger and likelihood of experiencing any coronary event during follow-up, even after control for potential confounds (Kawachi et al., 1996). The authors summarized four additional studies showing that higher levels of hostility predicted more negative outcomes in patients with known coronary artery disease (Dembroski et al., 1985; Hecker, Chesney, Black, & Frautchi, 1988; Koskenvuo et al., 1998; Mendes de Leon, Kop, de Swart, Bar, & Appels, 1996) and two showing that hostility (Julkunen, Salonen, Kaplan, Chesney, & Salonen, 1994) and anger (Matsumoto et al., 1993) predicted atherosclerotic progression. In discussing the varied findings, Rozanski and colleagues pointed out that the negative studies are of disparate quality. For example, in some cases high rates of attrition (Maruta et al., 1993) and brief follow-up periods (Koskenvuo et al., 1988) could have obscured underlying effects.

Perhaps the most comprehensive review of the literature addressing the association between hostility and physical health was performed by Miller et al. (1996). Forty-five studies were examined by these authors and subjected to both qualitative and quantitative (i.e., meta-analytic) analysis. Unique features of this review include efforts to disentangle the relative predictive utility of different facets of hostility and to examine methodological limitations contributing to contradictory findings. Twelve reviewed studies used interview-based ratings, which are most closely related with expressive aspects of the hostility construct (i.e., verbal or physical aggressive behaviors; Miller et al., 1996). Seven found positive evidence for a relationship with CHD outcomes, 2 showed null findings, and 3 identified mixed findings, such as an association for some measures and not others ($n = 2$) or in older participants only ($n = 1$). In a meta-analysis of studies that used interview-based assessments of hostility to predict CHD, the weighted mean effect size was .18. In contrast, scores on the Ho and on other self-report measures of the experiential aspects of hostility (i.e., hostile cognition, angry affect) were less consistently associated with CHD outcomes. Three out of 12 studies identified an association between Ho scores and CHD outcomes, and 9 identified null results. Fourteen out of 31 studies identified an

association between scores on other self-report measures of hostility CHD outcomes; 13 identified null results, 3 identified mixed results (e.g., positive findings for some measures only), and 1 identified contradictory results (i.e., an effect in the direction opposite to that predicted). The weighted mean effect size for the association across all studies including the Ho and CHD outcomes was .07, as was the weighted mean effect size representing the association between other self-report assessments of cognitive-experiential aspects of hostility and CHD outcomes. This represents a small but statistically significant effect. Additional analyses showed that self-report measures of emotional or expressive hostility did not consistently predict CHD. Miller and colleagues noted that overall, adjustment for CHD risk factors had little impact on the associations between hostility and CHD.

Fifty percent of studies reviewed by Miller et al. (1996) that administered the Ho (three of six) found positive evidence for an association with all-cause mortality. The studies that administered other self-report assessments of experiential aspects of hostility found either positive (three of five) or mixed (two of five) evidence for an association with all-cause mortality. The weighted mean effect size for all studies that examined hostility and all-cause mortality was .16. Statistical adjustment for other risk factors had little effect on the association between hostility and all-cause mortality. In summarizing their findings, the authors noted that research is most clearly indicative of an association between behavioral ratings of hostility and CHD and between cognitive-experiential self-report measures of hostility and all-cause mortality. Null findings appear to have been influenced by variability in measurements and samples and by the use of high-risk populations. That is, if hostility is a predictor of CHD, studies that include only persons with CHD or those at high risk for CHD will sample, on average, a higher and more limited range of hostility scores.

Recent studies provide additional evidence for the deleterious health effects of anger and hostility. For example, Everson et al. (1999) showed that high scores on a measure of expressed anger predicted incident stroke in Finnish men. Men with a history of cardiovascular disease who had high anger-expression scores were more than six times as likely to experience a stroke, when compared with their less angry counterparts, after adjustment for numerous other risk factors. The tendency to suppress and control anger was not significantly associated with stroke risk. Williams, Nieto, Sanford, Couper, and Tyroler (2002) showed that trait anger was associated with increased stroke incidence among younger participants and among those with less atherogenic lipid profiles (i.e., among individuals who were otherwise at lower risk for stroke). Similarly, trait anger was associated with an increased risk of CHD in the same cohort, but only among individuals who were not hypertensive (Williams et al., 2000). Two recent case-crossover studies (Mittleman et al., 1995; Möller et al., 1999) showed that episodes of intense angry affect increased the risk of suffering an acute MI in the following 1–2 hr. Finally, recent studies have shown that facets of hostility prospectively predict the severity and progression of atherosclerosis in healthy populations (Iribarren et al., 2000; Matthews, Owens, Kuller, Sutton-Tyrrell, & Jansen-McWilliams, 1998; Whiteman, Dreary, & Fowkes, 2000).

In summary, the weight of the evidence suggests that hostility and related constructs pose significant health risks. Research suggests an effect of hostility on health outcomes such as incident

CHD, atherosclerotic progression, stroke, and all-cause mortality. Detailed quantitative analyses indicate that distinct facets of the hostility construct could be important for different health outcomes (Miller et al., 1996)—an assertion that may help explain contradictory findings. In addition, studies concerning high-risk populations could impede the identification of hostility's health effects (cf. Miller et al., 1996), as demonstrated by studies showing that anger increases risk for cardiac events only in lower risk groups (e.g., Williams et al., 2000, 2002). Well-designed studies that have controlled for diverse potential confounds (e.g., Everson et al., 1999; Kawachi et al., 1996; see also Miller et al., 1996, for a review) provide especially compelling evidence that hostility poses health risks.

Summary

The reviewed research provides evidence that negative emotions and attitudes predict health outcomes. The evidence is most compelling for the effects of depression, hopelessness, and hostility on cardiovascular morbidity and mortality and for anxiety on sudden cardiac death. These associations have been observed in initially healthy populations and in individuals with diagnosed cardiovascular diseases. Some research also suggests that negative cognitive–emotional constructs may be important predictors of all-cause mortality. The evidence is particularly strong for aspects of hostility and all-cause mortality, although hopelessness and depression have also been shown to predict mortality in some research. The cited reviews suggest a number of methodological issues that need to be confronted before any definitive conclusions can be drawn, and they offer some possible explanations for inconsistencies observed in previous research.

It is important to note that a number of studies suggest a dose–response relationship between cognitive–emotional symptoms and health risks. Given that an individual diagnosed with a psychiatric disorder would tend to have more severe symptoms, one might infer that psychiatric disorders would be particularly strong predictors of morbidity and mortality. Moreover, although the existence of a dose–response relationship implies that even relatively small elevations of cognitive–emotional symptoms may be associated with worse health outcomes, methodological limitations of previous research limit definitive conclusions. For example, as noted in a recent editorial commentary (Carney, Freedland, & Jaffe, 2001), many studies include only a single, baseline assessment of depression. Because subclinical depressive symptoms are strongly predictive of future major depression (Judd & Akiskal, 2000), it is unclear whether negative health outcomes develop only in those individuals who eventually suffer a full-blown major depressive episode. Thus, further research that includes assessments of cognitive–emotional symptoms and disorders (e.g., Frasure-Smith et al., 1993, 1995a; Rovner et al., 1991), preferably at multiple time points, is clearly warranted. What we can safely conclude from the reviewed patterns of findings is that SES is consistently related to cognitive and emotional symptoms, elevations of which do predict worse health outcomes.

How Do Low-SES Environments Influence Negative Emotions and Cognitions?

If cognitive–emotional factors play a role in connecting SES with health, a key question becomes how the environments that

people with low SES inhabit lead them to experience negative emotions and cognitions, which, in turn, engender early morbidity and mortality. To address this question, we offer a general model outlining plausible psychosocial pathways among SES, cognitive–emotional factors, and health. The model and associated literature review are intended to serve two functions. First, if available evidence supports the posited pathways, the model would reinforce the primary mediational hypothesis, providing a potential framework for understanding the roles of cognitive–emotional factors in the SES and health association. Second, the model is intended to guide future research by more clearly identifying the circumstances under which low-SES environments may impact health through cognitive–emotional factors. The latter focus is developed in the final portion of the article.

In the current section, we begin our presentation of the model with consideration of the leading psychosocial candidate for connecting SES with negative emotions and cognitions: exposure and psychological reactivity to stress. Other pathways, such as environmental toxins or a lack of mental health services, are also likely to be important, but they are not considered here. We then articulate how low levels of tangible, interpersonal, and intrapersonal resources, that is, a deficient reserve capacity, may exacerbate the impact of SES-associated stress on negative emotions and attitudes, recognizing that low SES can both deplete resources and thwart the development and restoration of the resource bank. A challenge of any model of SES and cognitive–emotional factors is to consider the potential for bidirectional relationships; our model explicitly posits that emotions and cognitions can influence the availability of resources and that they can alter interpretations of stressful circumstances. However, the model does not specifically address the connection from health outcomes, or intermediate paths, back to emotions and cognitions. Clearly, health endpoints such as CHD can affect the intermediate psychosocial factors included in the model, as well as socioeconomic standing (e.g., Dew, 1998). However, CHD and premature mortality represent distal health outcomes that (typically) occur late in the natural history of disease, well after the usual establishment of socioeconomic position. In the final step of the model, we review the behavioral and physiological pathways connecting emotions and cognitions with health.

It is important to note that we do not intend to assert that psychosocial aspects of low-SES environments affect health exclusively through cognitive–emotional paths. Indeed, there is reasonable evidence that low-SES environments directly impact behavioral and biological mechanisms that in turn affect health (see Taylor et al., 1997, for further discussion). Similarly, Link and Phelan (1995, 1996) have pointed out that across time and changing patterns of disease, individuals with low SES are less able to avoid risks for health problems because they maintain less money, power, and information with which to adopt newly identified health-protective lifestyles and prevention strategies. However, in line with the primary focus of this review, our model centers on cognitive–emotional pathways.

SES, Exposure, and Reactivity to Stress

From a psychosocial perspective, the frequency and intensity of exposure to harmful or potentially threatening situations and to rewarding or potentially beneficial situations is the primary step

linking SES with negative emotions and attitudes (see Arrow A of Figure 1). Indeed, a number of studies indicate that individuals with low SES encounter more frequent negative life events and chronic stressors (B. S. Dohrenwend, 1973; Langer & Michael, 1963; McLeod & Kessler, 1990; Murrell & Norris, 1991; Myers, Lindenthal, & Pepper, 1974; Stansfeld, Head, & Marmot, 1998) and interpret ambiguous social events more negatively (Chen & Matthews, 2001; Flory, Matthews, & Owens, 1998). For example, Matthews, Räikkönen, et al. (2000) documented that, throughout 3 days of experiential sampling, men and women employed in positions with low occupational prestige experienced more interpersonal conflict relative to those with higher prestige positions. Exposure to chronic and acute stressors, in turn, has a direct negative impact on emotional experiences (Figure 1, Arrow B; e.g., Alec, 1996; Ensel & Lin, 1991; Paykel, 1994; Stansfeld, North, White, & Marmot, 1995) and a direct association with pathways affecting health outcomes (Figure 1, Arrow C; e.g., McEwan & Stellar, 1993). However, studies that account for initial differences in stress exposure suggest that at every level of stress, individuals with low SES report more emotional distress than those with higher SES (G. W. Brown & Harris, 1978; Kessler & Cleary, 1980; McLeod & Kessler, 1990; Turner & Noh, 1983).

Why might individuals residing in low-SES environments be more reactive to stress? Our framework suggests that low-SES individuals maintain a smaller bank of resources—tangible, inter-

personal, and intrapersonal—to deal with stressful events compared with their higher SES counterparts (Figure 1, Arrow D). Resources tend to occur in aggregate or to be absent in aggregate (e.g., Rini, Dunkel-Schetter, Wadhwa, & Sandman, 1999; Turner, Lloyd, & Roszell, 1999), suggesting the existence of a general protective influence, or resource bank (Hobfoll, 1998, 2001). Borrowing a concept from the aging literature, we label this *reserve capacity*. Low-SES persons' reserve capacity to deal with stressful environments may be low for two reasons: (a) Low-SES individuals are exposed to more situations in which they must use their resources and (b) their environments prevent the development and replenishment of resources to be kept in reserve.

Consistent with our model, having few tangible, interpersonal, or intrapersonal resources exacerbates the effects of stressful events on outcomes such as depression (e.g., G. W. Brown & Bifulco, 1990; G. W. Brown & Harris, 1978; G. W. Brown & Moran, 1997; Cohen & Wills, 1985; Hobfoll, 1988, 1989; Holahan & Moos, 1987, 1991). Furthermore, once an individual has been exposed to stress, resources tend to deteriorate, leaving the individual even more vulnerable to future strains (e.g., Bolger, Foster, Vinokur, & Ng, 1996; Ensel & Lin, 1991; path not shown in Figure 1). In fact, degradations in resources may help explain the effects of negative events on subsequent depressive symptoms (Holahan, Moos, Holahan, & Cronkite, 1999, 2000). Hence, having fewer stress-dampening resources, which are further reduced by stress

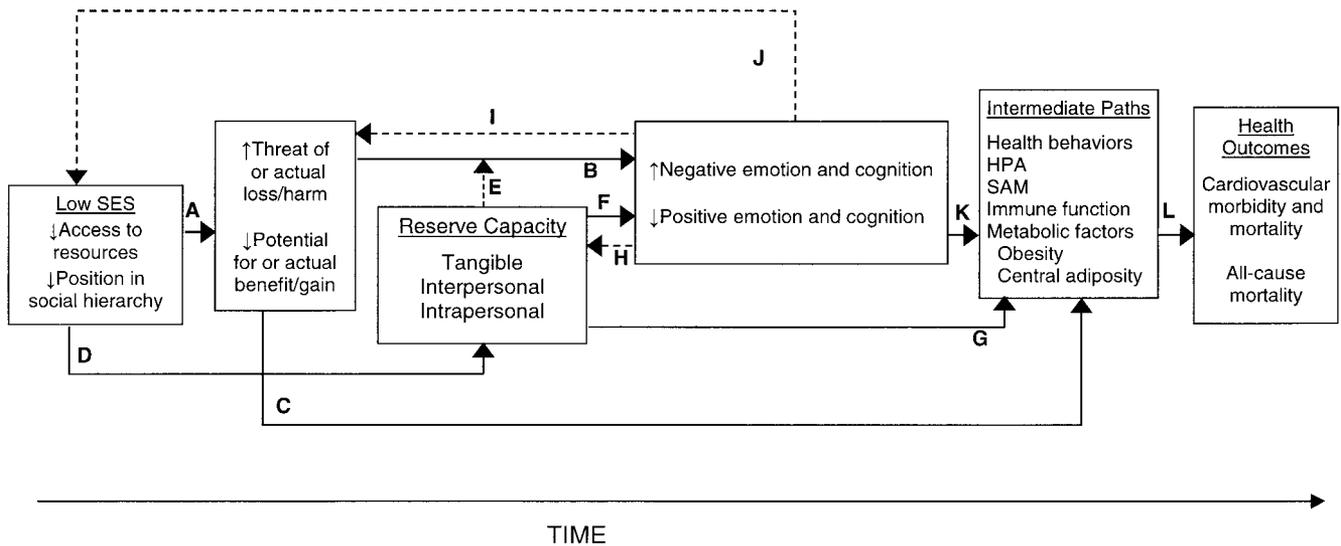


Figure 1. The reserve capacity model for the dynamic associations among environments of low socioeconomic status (SES), stressful experiences, psychosocial resources, emotion and cognition, and biological and behavioral pathways predicting cardiovascular disease and all-cause mortality over time. Dashed lines depict possible influences. Arrow A depicts the direct influence of SES on exposure to stressful experiences. Arrow B indicates the direct impact of stressful experiences on emotion and cognition. Arrow C shows the effects of stress on intermediate pathways hypothesized to affect health outcomes. Arrow D shows that socioeconomic environments condition and shape the bank of resources (i.e., the reserve capacity) available to manage stress. Arrow E (dashed line) shows that the reserve capacity represents a potential moderator of the association between stress and cognitive–emotional factors. Arrows F and G indicate the direct impact of cognitive–emotional factors and reserve capacity resources, respectively, on intermediate pathways. Arrows H, I, and J (dashed lines) depict the possible reverse influence of cognitive–emotional factors on reserve capacity resources, the experience of stress, and SES, respectively. Arrows K and L show that emotion and cognition are hypothesized to affect health outcomes through a variety of interrelated behavioral and physiological pathways. HPA = hypothalamic–pituitary–adrenocortical axis; SAM = sympathetic adrenal–medullary axis.

exposures, individuals with low SES are likely to show increased responsiveness when faced with stress (Figure 1, Arrow E).

SES and Reserve Capacity

What is the evidence that SES is associated with low reserve capacity (i.e., Figure 1, Arrow D)? Consistent with resource-based definitions, individuals with low SES have access to fewer financial and material goods, which might otherwise offset tangible stressors such as job loss, illness, or disability (Thoits, 1995). Low-SES environments also contain deficient community resources, such as safe neighborhoods, parks, transportation, and child care (Macintyre, Maciver, & Solomon, 1993; Sooman & Macintyre, 1995; Troutt, 1993). If present, these resources might reduce chronic or daily stress.

SES is also associated with diverse aspects of social functioning, including contact with others, network size, reciprocity in relationships, satisfaction with support, the tendency to seek social support, work support, and generalized support perceptions (e.g., Belle, 1982, 1990; Bosma, Van de Mheen, & Mackenbach, 1999; Cohen et al., 1999; House et al., 1994; Krause, 1991; Krause & Borawski-Clark, 1995; Matthews et al., 1989; Murrell & Norris, 1991; Oakley & Rajan, 1991; Ranchor, Bouma, & Sanderman, 1996; Stansfeld et al., 1998; Turner & Marino, 1994). Furthermore, neighborhood as well as individual SES appears to influence social experiences (e.g., Gracia, Garcia, & Musitu, 1995; Tigges, Browne, & Green, 1998). Social stressors typical of low-SES environments (e.g., crowding, violence, high crime rates) may interfere with the development of supportive contacts by discouraging interpersonal trust (Krause, 1991; Roschelle, 1997; C. E. Ross & Jang, 2000). In addition, individuals with low SES may be vulnerable to factors that could degrade social support and increase social stress, such as marital instability (Tzeng & Mare, 1995), domestic violence (Aldarondo & Sugarman, 1996; Christmas, Wodarski, & Smokowski, 1996; Lockhart, 1987; Straus, 1980), substance abuse (Kessler et al., 1994), and single parenting (Bianchi, 1995). Contradictory findings have been reported (e.g., Sokolovsky & Cohen, 1981; Stack, 1974), possibly reflecting the multidimensional nature of social support (e.g., Krause & Borawski-Clark, 1995), ethnic and cultural differences in the association between SES and social functioning (e.g., Stack, 1974), or methodological limitations such as inadequate sample sizes and time frames. However, overall, the weight of the evidence suggests that low-SES environments shape interpersonal experiences in a negative manner, although the effect sizes may be small.

Further research indicates an inverse relationship between SES and resilient intrapersonal characteristics such as self-efficacy, mastery, or a sense of perceived control (Cohen et al., 1999; Lachman & Weaver, 1998; Marmot, Ryff, Bumpass, Shipley, & Marks, 1997; Matthews et al., 1989; Mirowsky, Ross, & Willigen, 1996; Ranchor et al., 1996). A recent meta-analysis suggests that individuals with low SES have lower levels of self-esteem, with the effects stronger for occupation and education than for income (Twenge & Campbell, 2002). The association between SES and intrapersonal resources might originate through asymmetrical relationships associated with status hierarchies, which bring unpleasant feelings of inferiority for those low in the hierarchy and pleasant feelings of superiority for those high in the hierarchy (R. Brown, 1965). These interpersonal experiences are likely to begin

in early development (Kessler & Cleary, 1980; Repetti, Taylor, & Seeman, 2002) and may be maintained through socialization experiences related to low-SES work environments later in life (e.g., Gecas & Seff, 1989). For example, downward social mobility in employment status is related to lower levels of personal control and mastery (Pearlin, Lieberman, Menaghan, & Mullan, 1981), and individuals with low SES tend to report low control, decision-making latitude, skill discretion, and work support and high demands in their jobs (e.g., Marmot & Theorell, 1988; Stansfeld et al., 1998). Like the research regarding social support, that concerning SES and intrapersonal resources has been somewhat inconsistent, perhaps in part because of gender, cultural, and ethnic differences in the nature of the association (e.g., Richman, Clark, & Brown, 1985). For example, the association between SES and self-esteem has become stronger in women over time, is higher among Asian Americans than other groups, and increases with age, peaking in midlife (Twenge & Campbell, 2002).

Evidence Supporting the Moderating Roles of Reserve Capacity Resources

Consistent with the reserve capacity model (Figure 1, Arrow E), studies showing that social support (e.g., Turner & Noh, 1983; Stansfeld et al., 1998; C. E. Ross & Mirowsky, 1989), negative work characteristics (Stansfeld et al., 1998), and intrapersonal resources (e.g., Lachman & Weaver, 1998; Mirowsky et al., 1996; C. E. Ross & Mirowsky, 1989; Turner, Lloyd, & Roszell, 1999) help explain low-SES individuals' excess vulnerability to distress provide direct support for the moderating roles of reserve capacity resources (although see contradictory evidence in Murrell & Norris, 1991; Thoits, 1982, 1984). For example, Turner and Noh (1983) found that individuals with low SES but high social support and perceived control did not display elevated emotional distress relative to those with high social status. Similarly, Lachman and Weaver (1998) found that the positive effects of control on health outcomes (i.e., perceived health, physical symptoms, depression, life satisfaction) were greater at lower levels of education or income; individuals with low SES and strong control beliefs had health outcomes similar to their higher SES counterparts. Thus, negative emotions and cognitions may be more likely to serve as mediators of the SES–health association in the context of low reserve capacity resources.

Evidence Supporting the Mediating Roles of Reserve Capacity Resources

Although less germane to our primary focus, it is important to note that reserve capacity resources may also serve mediating roles in the associations between SES and emotional and physical health. For example, prior research indicates an association between reserve capacity resources and cognitive–emotional states, as shown in Arrow F of Figure 1. Specifically, research has shown that low self-esteem predisposes individuals to depression (Hokanson, Rubert, Welker, Hollander, & Hedeem, 1989), although contradictory findings have been reported (Lewinsohn, Steinmetz, Larson, & Franklin, 1981; Robertson & Simons, 1989). Social support—and in particular social integration—also has a direct, positive association with mental health (Cohen & Wills, 1985; Thoits, 1995). Evidence for the mediating roles of resources was

provided in a study by Link, Lennon, and Dohrenwend (1993), in which the lack of control and planning associated with low-SES occupations related to lower levels of personal control, which in turn predicted depression and distress. Similarly, Ennis, Hobfoll, and Schröder (2000) showed that changes in material resources led to increased depression in inner-city women; this association was mediated by psychosocial resources (i.e., mastery, social support), which were inversely associated with SES.

Reserve capacity resources could also contribute directly to the association between low SES and physical health outcomes through intermediate pathways, as shown in Arrow G of Figure 1 (Taylor & Seeman, 1999). Substantial research suggests that the same types of interpersonal resources deficient in low-SES environments protect against negative health outcomes (e.g., Adler & Matthews, 1994; Krantz & McCeney, 2002; Rozanski et al., 1999). Some studies have also shown that intrapersonal resources predict physical health outcomes (Rodin, 1990; Seeman & Lewis, 1995). Arrow G is consistent with the reviewed mediation studies, which included resource variables such as social support and control (in addition to cognitive–emotional factors) to examine mediating roles of psychosocial factors in studies of SES and physical health (e.g., Cohen et al., 1999; Levenstein & Kaplan, 1998; Lynch et al., 1996). Further evidence is provided by a recent study (Marmot, 1998) showing that work characteristics (e.g., skill discretion, decision authority) and perceived efficacy and control partially mediated the association between SES and perceived health and the association between SES and waist-to-hip ratio. Thus, both mediating roles and moderating roles of psychosocial resources should be considered in future research.

Bidirectionality in the Associations Between Emotions and Cognitions and SES

Our framework considers that distress may negatively impact available resources, which would result in the perpetuation and intensification of negative cognitive–emotional experiences (Arrow H, Figure 1). For example, research has shown that hostile individuals experience less social support and more social conflict compared with their nonhostile counterparts (Barefoot, Dahlstrom, & Williams, 1983; Houston & Kelly, 1989; Scherwitz et al., 1991). Similarly, depressed persons experience greater interpersonal difficulties and lesser social support (e.g., Brugha, 1995), and this association appears to be reciprocal in nature (e.g., T. P. Johnson, 1991; see also discussions in Hammen, 1991; Joiner & Coyne, 1999). Likewise, depression (Beach & O’Leary, 1993; S. L. Johnson & Jacob, 1997) and hostility (Miller, Marksides, Chiriboga, & Ray, 1995; Newton & Kiecolt-Glaser, 1995; T. W. Smith, Sanders, & Alexander, 1990) predict poorer marital functioning.

As shown by Arrow I (Figure 1), cognitive–emotional states may also influence appraisals of external stimuli—a tenet that underlies cognitive theories of depression (Beck, 1971). Specifically, depressed individuals may distort processing of information in a manner that serves to reinforce negative mood (Haaga, Dyck, & Ernst, 1991). Similarly, hostile individuals may be more likely to interpret ambiguous stimuli in a negative or threatening light (Chen & Matthews, 2001; Flory, Matthews, & Owens, 1998). Thus, negative emotions and cognitions may increase the likelihood that ambiguous stimuli are viewed as threatening or harmful, thereby resulting in further degradations of the reserve capacity.

Negative emotional and cognitive experiences could also feed back to SES, in accord with the social drift, or social selection, hypotheses (Figure 1, Arrow J; e.g., Kessler, 1979). According to this view, psychological disorder impairs one’s ability to attain a higher social class and/or causes one to drift down the socioeconomic hierarchy. For example, a study by Kessler, Foster, Saunders, and Stang (1995) showed that an early history of conduct disorder, substance use, anxiety, and mood disorders predicted the likelihood that one would fail to complete high school. However, this study was not longitudinal, thereby creating interpretive ambiguities (J. G. Johnson, Cohen, Dohrenwend, Link, & Brook, 1999). In a longitudinal study by Caspi, Elder, and Bem (1987), the presence of temper tantrums in late childhood predicted later socioeconomic attainment. Specifically, men with a history of temper tantrums experienced downward occupational mobility, and women with a history of tantrums tended to marry men with lower status occupations. Another study found that individuals with panic disorder had an increased likelihood of initiating disability payments across a 1-year follow-up compared with those without a panic diagnosis (Kouzis & Eaton, 2000).

However, some research suggests that the social drift effect might be more relevant to very debilitating psychiatric disorders, such as schizophrenia (e.g., B. P. Dohrenwend, 1990; Wyatt & Clark, 1987) and substance problems (e.g., Kessler et al., 1995), and less so to depression and anxiety. For example, a recent study showed that SES in childhood was a strong prospective predictor of future depressive and anxiety disorders, but depression and anxiety did not predict further decrements in SES (J. G. Johnson et al., 1999). In contrast, alcohol- and substance-related diagnoses predicted downward socioeconomic drift. A recent prospective study of adolescents found no evidence of downward social selection, in respect to educational attainment, for participants with depression or anxiety at baseline (Miech, Caspi, Moffitt, Wright, & Silva, 1999). Overall, studies have supported the causation hypothesis over the drift, or selection hypothesis, in respect to depression (see also B. P. Dohrenwend, 2000; Moos, Cronkite, & Moos, 1998; Ritscher, Warner, Johnson, & Dohrenwend, 2001).

Ultimately, identifying directionality in the associations between SES and emotions and cognitions represents an exceedingly difficult goal, as evidenced by the fact that after many years of investigation, the primary questions remain unanswered (Fox, 1990). Considerable methodological obstacles exist. For example, in longitudinal studies, previous subclinical episodes or periodic increases in symptoms might have predated the initial study period and, therefore, impacted the socioeconomic standing of the participant as measured at baseline. Even past threshold episodes may be underestimated because of inaccurate memory or intentional misrepresentation in reporting lifetime occurrences. Furthermore, longitudinal studies may underemphasize the impact of psychological disorder on social standing by disregarding the influence of psychological distress on the “failure to rise” (i.e., to achieve a higher socioeconomic standing than one’s origins) or to keep up with societal increases in affluence (e.g., B. P. Dohrenwend & Dohrenwend, 1969). Studies that measure psychological dysfunction in relation to SES at a single point in time also cannot integrate the influence of previous exposures to low SES. Finally, research concerning reciprocal influences of SES and cognitive–emotional symptoms involves complexity in unraveling state and trait influences and in inferring datable onsets. The association between SES

and psychological functioning is sure to be complex and, most likely, dynamic. In aggregate, we interpret the literature as showing that to some extent, low SES can be considered causally antecedent to psychological distress and that psychological dysfunction and low SES may be mutually reinforcing.

Pathways From Cognitive–Emotional Factors to Health

As shown in Figure 1, Arrows K and L, negative emotions and cognitions appear to affect health through several interrelated behavioral and physiological pathways. These constructs influence health behavior choices and adherence to intervention and prevention regimens. For example, depressed individuals are more likely to display negative health behaviors, such as smoking (Anda et al., 1990; Hughes et al., 1986) and leading a sedentary lifestyle (Farmer et al., 1988). Other research suggests that depression leads to poor adherence to coronary treatment recommendations (Blumenthal, Williams, Wallace, Williams, & Needles, 1982; Carney, Freedland, Eisen, Rich, & Jaffe, 1995; Guiry, Conroy, Hickey, & Mulcahy, 1987). High levels of anxiety (Breslau, Kilbey, & Andreski, 1991; Pohl, Yeragani, Balon, Lycaki, & McBride, 1992) and hostility (Leiker & Hailey, 1988; Siegler, 1994) also increase the likelihood that young people will engage in negative health behaviors later in life. Negative cognitive and emotional processes could affect health behavior choices through their association with factors such as locus of control, health beliefs, or self-efficacy (Bandura, 1989; Lau, 1988; Strickland, 1978). In addition, negative health practices and poor adherence may represent maladaptive coping strategies associated with emotional distress. Consistent with this assertion, some research suggests that individuals experiencing stress are more likely to engage in negative health behaviors (Horowitz et al., 1979; Schachter, Silverstein, Kozlowski, Herman, & Liebling, 1977). However, studies that have examined health practices suggest that they do not account completely for associations of negative emotions and cognitions with health. Investigators have therefore begun to explore a number of plausible biological pathways.

Negative emotions and attitudes appear to produce diverse physiological alterations that are linked to increased risk of cardiovascular morbidity and mortality, including hyperactivity of the sympathetic adrenal–medullary (SAM) system, dysregulation of the hypothalamic–pituitary–adrenocortical axis, exaggerated platelet reactivity, reduced heart rate variability, heightened inflammatory processes, and ventricular instability and ischemia in response to stress (see reviews by Carney, Rich, & Jaffe, 1995; Ehlert & Straub, 1998; Glassman & Shapiro, 1998; Kop, 1999; Krantz & McCeney, 2002; Kubzansky et al., 1998; Markovitz & Matthews, 1991; Miller et al., 1996; T. W. Smith, & Gallo, 2001; Van Kanel, Mills, Fainman, & Dimsdale, 2001). More speculative is the possibility that negative emotions and cognitions, particularly hostility and depression, may be associated with altered central serotonergic function (Manuck et al., 1998), which, in turn, is associated with SES and several cardiovascular risk factors (Matthews, Flory, Muldoon, & Manuck, 2000; Muldoon et al., 1998). Some research suggests that negative emotions and attitudes might also influence cardiovascular morbidity and mortality through their association with established cardiovascular risk factors such as hypertension (Everson et al., 1998; Jonas, Franks, & Ingram, 1997; Markovitz, Matthews, Kannel, Cobb, & D’Agostino, 1993; Markovitz, Mat-

thews, Wing, Kuller, & Meilahn, 1991; Paterniti et al., 1999; K. B. Wells, Golding, & Burnam, 1989) and hyperlipidemia (Bajwa, Asnis, Sanderson, Irfan, & van Praag, 1992; Oxenkrug, Branconier, Harto-Traux, & Cole, 1983; Weidner, Sexton, McLellarn, Connor, & Matarazzo, 1987; see also Hayward, 1995, for a review) and metabolic factors such as diabetes (Eaton, Armenian, Gallo, Pratt, & Ford, 1996), obesity, and central adiposity (Räikkönen, Matthews, Kuller, Reiber, & Bunker, 1999). In addition, research showing that negative emotions and attitudes affect immune functioning—possibly through SAM activation (Cohen & Herbert, 1996)—provides evidence that they alter vulnerability to infectious diseases, cancer, and coronary artery disease (Appels, Bär, Bär, Bruggeman, & de Baets, 2000; Christensen, Edwards, Wiebe, Benetsch, & McKelvey, 1996; Herbert & Cohen, 1993; Weisse, 1992). Thus, negative emotions and attitudes could shape health and disease through a number of behavioral and biological pathways.

Summary and Implications

The presented model and supporting evidence provide a framework for integrating disparate literatures with the unique goal of evaluating a frequently posited psychosocial pathway connecting SES and health. Understanding whether and how cognitive–emotional factors play a role in SES–health links is of vast importance, and the model suggests specific ways to evaluate that role. More specifically, the model has heuristic value, leading to a number of testable predictions.

For example, the association between SES and negative emotions may begin in childhood as children develop attitudes about others and process information about potentially threatening or harmful environments (Chen, Matthews, & Boyce, 2002). The role of reserve capacity should increase with age because low-SES environments are proposed to cause increased use of available resources and to interfere with the development of a resource bank as one ages. Similarly, the association between SES and negative emotions should increase in size longitudinally, given the proposed effects of negative emotion feedback on social position. Thus, studies that examine associations among SES, cognitive–emotional factors, and intermediate pathways or health in multiple age groups or, ideally, with longitudinal methodologies may help elucidate when and how these relationships emerge. Indices of social position that are more dynamic in nature, such as income, should be more sensitive to the bidirectional relationships between SES and negative cognitive–emotional factors and, presumably, health. Finally, the influence of SES on negative emotions and cognitions may be enhanced with consideration of resources or reserve capacity. Cognitive–emotional factors and resources may exhibit systematic and potentially synergistic relationships. Hence, any study that considers either in isolation of the other may underestimate their impact on health. We further develop these assertions and their implications for future research below.

Future Research Directions

SES is inversely associated with anxiety and depression symptoms and negative cognitive styles, such as hopeless and hostile attitudes. Low SES may also be associated with higher rates of anxiety and depressive disorders, although the evidence for these

associations is less consistent. As suggested by a number of prospective studies (e.g., Bruce et al., 1991; Kaplan et al., 1987; J. C. Wells et al., 1994; Wilson et al., 1999), and as depicted in Figure 1, at least part of the discrepant vulnerability associated with low SES likely stems from social causation. Low-SES environments are associated with events and situations that have a direct impact on negative emotional and cognitive states. In addition, lower SES individuals do not appear to benefit from having as large a reserve capacity, developed over time from the aggregation of positive interpersonal experiences and intrapersonal characteristics, which might otherwise attenuate the effects of their stressful environments. Thus, low SES is associated with greater exposure and reactivity to stress (cf. Bolger & Zuckerman, 1995).

Previous research also suggests that negative emotions and attitudes have deleterious effects on health, and a number of behavioral and biological mechanisms could underlie these associations. In aggregate, the findings suggest that experiencing higher levels of negative emotions and cognitions represents one possible mediator of the association between SES and health—particularly cardiovascular health and all-cause mortality. Studies that have addressed mediation more directly provide limited evidence for the roles of cognitive–emotional factors. However, these studies are associated with important methodological limitations that create interpretive ambiguities. Specifically, prior studies of mediation have not considered the types of reciprocal associations between resources and cognitive–emotional factors that are depicted in Figure 1 and supported by previous research. Furthermore, previous mediation studies have not typically considered SES from a multilevel, dynamic perspective, nor have they addressed the aggregate influence of negative emotions and cognitions or the possible protective effects of positive emotions and attitudes. Prior research also has been limited by including homogeneous samples and by underutilizing developmental and lifestyle trajectory approaches. We now present what we consider to be the most comprehensive methods for future studies addressing the mediation framework. We also present suggestions regarding initial steps for preliminary tests of the proposed associations.

Comprehensive Tests of Mediation: What Types of Research Are Needed?

To further evaluate the tenet that negative emotions and cognitions contribute to the association between SES and health, additional integrative research is needed. Our review has uncovered very few studies that could be used to directly evaluate mediation, and studies that did include all pieces of the framework provide very limited evidence for the dynamic links suggested in our model. What characteristics of studies would allow a more comprehensive analysis of mediation in future research?

Consideration of aggregate effects among risk factors. First, consistent with the reserve capacity model presented in Figure 1, we recommend that future mediation studies consider joint effects among variables. Studies that control for all psychosocial factors simultaneously and then examine the degree to which the excess risk of low-SES individuals is attenuated may underestimate the true roles of psychosocial factors by neglecting additive, interactive, or synergistic effects. Risk factors tend to aggregate within lower SES individuals (e.g., Lynch, Kaplan, & Salonen, 1997), and attention to these aggregations might contribute to psychologists'

understanding of how SES affects health. For example, Kaplan (1995) described data from the Alameda County Study (Berkman & Breslow, 1983) in which low income, social isolation, and depression each independently predicted mortality from all causes over 9 years. Individuals who were isolated, depressed, and poor had a dramatically increased risk of death, which was nearly four times higher than the referent group (i.e., not poor, isolated, or depressed). In the Kuopio Ischemic Heart Disease Study (Kaplan et al., 1994; see also Kaplan & Salonen, 1990), low income, social isolation, and cynical distrust each increased risk for premature mortality. Again, individuals who experienced these risk factors in combination evidenced a markedly elevated risk of death (nearly four times as high) relative to the comparison group (Kaplan, 1995). Another recent study (Frasure-Smith et al., 2000) showed that depression and social support interacted to predict outcomes following MI. Individuals who had high depression but low social support were at higher risk of a recurrent event, whereas those who had high depression and high social support were not at increased risk. Thus, joint effects among psychosocial risk factors may be important to predicting health outcomes and should be considered in tests of mediation.

It is important to note that covariation in high-risk psychological (i.e., depression, anger, low perceived control) and social (i.e., social support, work environments) characteristics do not occur randomly. Rather, these factors are likely to be reciprocally determined through recurring transactions between individuals and their social contexts (e.g., Revenson, 1990; T. W. Smith, 1995). Well-articulated theories based on this transactional view have been advanced to explain the close association between depression (Joiner & Coyne, 1999) and hostility (T. W. Smith, 1995) and personal control (Krause, 1997) with features of the social environment. The socioeconomic context is likely to play an important role in shaping these recurring psychosocial patterns, as shown in Figure 1. Thus, through the common epidemiological practice of statistically controlling for other risk factors in an effort to identify the independent predictive value of a single variable, information about dynamic risk processes may be lost (see also L. C. Gallo & Smith, 1999; Revenson, 1990; T. W. Smith & Gallo, 2001). We suggest that a more comprehensive understanding of risk may be gained by examining psychosocial risk factors in their naturally occurring configurations. For example, cluster analytic techniques could be applied to existing data sets to describe specific patterns of person- and social-level risk factors (L. C. Gallo & Smith, 1999), and resulting groupings could then be included as predictors of subsequent health.

Previous studies that have examined mediation have also tended to control for other types of risk factors prior to examining the effects of psychosocial variables. This is logical in cases in which the goal is to determine whether psychosocial factors add to prediction over and above traditional risk factors. However, this approach may underestimate the true impact of psychosocial factors on the outcome. For example, in part, psychosocial factors are believed to affect health by influencing health behaviors. Statistically controlling for health behaviors to examine the independent effect of psychosocial factors eliminates consideration of these indirect effects (e.g., Siegler, 1994). This assertion is supported by a recent study (Whiteman et al., 2000) in which hostility directly predicted atherosclerotic progression and also indirectly predicted progression through smoking. Moreover, psychosocial factors are

believed to be importantly related to a variety of health outcomes; therefore, covarying out prior health history could have a similar effect. Likewise, psychosocial factors could contribute to disease severity. For example, individuals with high levels of negative emotions and cognitions and low levels of resources could have more severe disease because of the cumulative exposure to these psychosocial risk factors. Thus, additional studies should focus on modeling both the direct and indirect effects of psychosocial risk factors using path analysis or structural equation modeling. Alternatively, both psychosocial and behavioral or biological risk factors could be included in attempts to identify naturally occurring patterns of risk through cluster analytic techniques.

Dynamic conceptualization of SES and cognitive–emotional factors. Second, ideally future studies concerning the hypothesized associations would include multilevel assessments of socioeconomic variables, as contextual SES variables appear to have important health implications over and above the effects of individual factors (Diez-Roux et al., 2001; Lynch et al., 2000; Robert, 1998; Yen & Kaplan, 1999; see also Diez-Roux, 1998; Macintyre & Ellaway, 2000, for further discussion). In addition, SES should be assessed at multiple time points because, as noted above, previous research suggests that repeated exposure to low SES could generate cumulative effects (e.g., Lynch, Kaplan, & Shema, 1997). Multiple assessments would also allow an evaluation of the effects of changes in SES on psychological and physical health (e.g., Duncan, 1996; Matthews et al., 2001; McDonough, Duncan, Williams, & House, 1997). Although this type of dynamic evaluation is best suited to a longitudinal framework, exposures and changes in SES could also be assessed in cross-sectional research. For example, future studies could evaluate periods of unemployment in one's lifetime or the number of times one's income has fallen below the poverty line.

Likewise, having more than one psychological disorder or having high levels of many types of emotional and cognitive symptoms may pose even greater risk than having only one disorder or one type of symptoms. Thus, we recommend that future research also focus on the aggregate health impact of negative emotions, emotional disorders, and other psychiatric disorders. Furthermore, future research should consider the cumulative impact of exposure to negative emotions, cognitions, or emotional disorders across time, consistent with the recommendations for conceptualizing SES. In line with our discussion of conceptualizing emotion, we also note the importance of using well-validated and reliable self-report and interview measures of emotion and cognition.

Positive emotions and attitudes. Future studies of mediation should also focus explicitly on the health-protective effects of high levels of positive emotions and cognitions, which may be more likely to occur in high-SES environments. Some previous studies concerning SES and negative emotions or cognitions have used continuous assessments and identified inverse, approximately linear relationships between the variables (e.g., Barefoot et al., 1991; Himmelfarb & Murrell, 1984; Ickovics et al., 1997; Kessler et al., 1994; Scherwitz et al., 1991; Wilson et al., 1999; Wittchen et al., 1994). To the extent that individuals with high SES report lesser negative emotions and cognitions, they may be protected from deleterious health effects. However, if affect consists of two valence dimensions, as some research and theories proffer (e.g., Watson & Clark, 1997; Zautra, Potter, & Reich, 1997), then future

studies should also include distinct measure of positive cognition and emotion.

Reflecting a rather ubiquitous negative research bias (cf. Seligman & Csikszentmihalyi, 2000), researchers have not typically conceptualized the SES and health gradient from the perspective of positive emotions and cognitions. However, they may protect against health through mechanisms similar to those proposed to link negative emotions and cognitions with health. Resilient interpersonal and intrapersonal characteristics could increase the chances that one will adaptively cope, thereby reducing reliance on health-detrimental coping behaviors. In addition, at least preliminary evidence suggests that positive emotions produce beneficial physiological effects, including short-circuiting cardiovascular reactivity associated with negative emotions (Fredrickson & Levenson, 1998) and augmenting immune functioning (Stone, Cox, Valdimarsdottir, & Jandorf, 1987; Stone, Neale, Cox, & Napoli, 1994). Additional research suggests that positive cognition about interpersonal partners may dampen reactivity to stress (T. W. Smith, Ruiz, & Uchino, 2001). Thus, future research should include separate measures of positive and negative emotional and cognitive experiences (and resources) to examine whether these constructs make independent or aggregate contributions to SES–health discrepancies.

Inclusion of diverse samples. Also important for future studies of associations among SES, psychosocial resources, emotions and cognitions, and physical health is the inclusion of diverse samples. For example, many prior studies evaluating physical endpoints have included samples of men only, and additional research is therefore needed to examine the proposed mediational framework in women. This seems particularly important because women show consistently higher rates of depressive (Comstock & Helsing, 1976; Fiscella & Franks, 1997; Weissman & Myers, 1978) and anxiety symptoms (Himmelfarb & Murrell, 1984) than men. Women also experience major depression approximately twice as often as men (Kessler et al., 1994; Weissman et al., 1991), and they may also have higher rates of most anxiety disorders (Kessler et al., 1994). In contrast, men display higher rates of hostility (Barefoot et al., 1991; Scherwitz et al., 1991). Thus, we recommend that future research consider whether gender affects the nature of relationships among SES, cognitive–emotional factors, and health.

Future research should also include ethnically diverse samples to evaluate whether the proposed paths are influenced by ethnic or cultural differences. Several studies have shown that individuals of ethnic minority status may have higher rates of cognitive–emotional symptoms (e.g., Bruce et al., 1991; Comstock & Helsing, 1976; Warheit et al., 1975), although some have suggested that these differences could be attributed largely to the effects of social class (e.g., Comstock & Helsing, 1976; Warheit et al., 1975). On the other hand, some research indicates that members of ethnic minorities obtain higher scores on measures of hostile, mistrustful attitudes compared with non-Hispanic White respondents (Barefoot et al., 1991; Gump et al., 1999; Scherwitz et al., 1991). Hostile, mistrustful attitudes could therefore play a more important mediating role for members of ethnic minority groups than for Whites. Indeed, the study by Gump and colleagues (1999) found that hostility represented a mediating factor connecting low SES with cardiovascular reactivity and left ventricular hypertrophy for Black but not White adolescents.

In addition, psychosocial resources could have disparate meanings, structures, or roles for individuals of different ethnic or cultural backgrounds. For example, some research suggests that kin support may function differentially for Blacks and Hispanics when compared with non-Hispanic Whites, although the pattern of differences has varied (e.g., Hogan, Hao, & Parish, 1990; Jung, 1989; Roschelle, 1997). Furthermore, the association between racial discrimination and SES appears to be complex and varied. For example, Blacks with higher SES are sometimes more likely to report discrimination relative to Blacks with lower SES (e.g., Gary, 1995; Landrine & Klonoff, 1996). Nonetheless, discriminatory experiences deserve further attention as factors that could increase the stressful experiences associated with low-SES environments for non-White individuals (Krieger, 2000; Williams, 1999). Thus, we recommend that future research concerning the mediation hypothesis incorporate diverse samples to evaluate the roles of discrimination and to examine consistency in the hypothesized pathways.

Developmental research and life-course trajectory approaches. Given our focus on cardiovascular endpoints and all-cause mortality, the reserve capacity model has been discussed in the context of understanding the roles of psychosocial factors in adult health gradients. However, the gradient between SES and other health problems can be observed in samples of children aged 1 to 18 years (Goodman, 1999; Johnson et al., 1999; for reviews, see also Bradley & Corwyn, 2002; Chen et al., 2002). This finding suggests the importance of future longitudinal research that incorporates a consideration of developmental factors in the processes outlined in our model. Chen et al. (2002) pointed out that because of normal developmental changes in cognition and affect, the transition from childhood to adolescence is the period in which negative emotions and cognitions are likely to begin influencing the links between SES and health outcomes. Adolescence is an intriguing period because for some health outcomes (e.g., blood pressure), SES links are strong in early childhood, disappear by adolescence, and re-emerge by adulthood (Chen et al., 2002). For other health outcomes, such as physical activity and smoking, links with SES develop during adolescence (Chen et al., 2002). Our model suggests that increased exposure across the life span to the processes outlined in the reserve capacity model contribute to SES–health links considerably prior to midlife and perhaps in adolescence and young adulthood.

Developmentally focused approaches are also of value because they may facilitate an understanding of why childhood SES (i.e., SES of family of origin) is an important predictor of adult psychosocial characteristics (Bosma et al., 1999; Lynch, Kaplan, & Salonen, 1997), behavioral and biological health risk factors (Blane et al., 1996; Lynch, Kaplan, & Salonen, 1997; Van de Mheen, Stronks, Looman, & Mackenbach, 1998), and morbidity and mortality (Ben-Shlomo & Davey Smith, 1991; Davey Smith, Hart, Blane, Gillis, & Hawthorne, 1997; Davey Smith, Hart, Blane, & Hole, 1998; Kaplan & Salonen, 1990; Rahkonen, Lahelma, & Huuhka, 1997) independently of adult SES. Furthermore, they may provide another perspective about why biological factors, such as in-uterine stress and low infant weight and height, that are correlated with low SES may have a direct association with later health (i.e., one that is not mediated by psychosocial factors). Biological factors may represent markers for stressful psychosocial environments associated with being born into a lower SES environment,

and they may relate to health by indicating future adult socioeconomic circumstances (as discussed in Kaplan & Keil, 1993; Marmot, Shipley, Brunner, & Hemingway, 2001; Power & Hertzman, 1997).

In aggregate, these findings suggest that a comprehensive analysis of the paths creating socioeconomic disparities in health would ultimately require longitudinal research that examines resources, emotions, attitudes, behavioral and biological health risk factors, and psychological and physical health outcomes as they unfold across the life span. Life-course trajectory approaches have become more realistic with the accessibility of multilevel modeling statistical procedures, which facilitate analysis of clustered data and intrapersonal change. Even with careful longitudinal methodologies, identifying temporal precedence and causal relationships is a difficult task (e.g., Bradley & Corwyn, 2002; Kuh & Ben-Shlomo, 1997). Nevertheless, by comparing life-course trajectories in individuals with low versus high SES, future research can at least begin to elucidate the intricate pathways we have presented. Thus, we suggest that additional longitudinal studies that incorporate developmental-stage analyses be conducted.

What Intermediate Research Steps May Be Taken to Evaluate the Mediation Hypothesis?

The proposed framework for an ideal test to the mediation hypothesis is daunting given the complexity of addressing reciprocal relationships, the requirement of detailed measures taken repeatedly over an appropriate period of time, the need to assess symptoms, disorders, and multiple levels of SES, the analytic challenges, and the need to take into account the general changes in the population. Thus, we propose a number of initial research steps that may provide a foundation for future, more comprehensive research.

Analysis of existing data sets. Our review has uncovered very few studies that examined the degree to which negative emotions and cognitions account for SES and cardiovascular morbidity and mortality or all-cause mortality. Yet, many large-scale databases exist in which measures of each of these variables are included. Thus, we recommend that formal mediation tests that include analysis of each criterion presented by Baron and Kenny (1986) be applied to existing data sets. We also recommend that these analyses incorporate a consideration of interactive effects, especially when measures of stress or interpersonal and intrapersonal resources are included. Similarly, the procedures outlined above for examining naturally occurring aggregations of socioeconomic, psychosocial, and/or other types of risk factors could be applied to existing data sets to perform a more comprehensive analysis of the roles of emotions, cognitions, and other psychosocial factors.

Tests of mediation with intermediate health outcomes. As a further step toward testing the proposed model, we recommend that future research focus on the intermediate paths depicted in Figure 1. For example, mediational tests could be applied in studies that have examined the association between SES and cardiovascular reactivity, an approach adopted by Gump et al. (1999). Similarly, future research could examine whether cognitive and emotional factors help explain the association between SES and metabolic factors, health behaviors, and other risk factors (e.g., Kubzansky, Kawachi, & Sparrow, 1999; Marmot, 1998). Finally, not depicted in Figure 1, subclinical cardiovascular dis-

ease measures could be used as an intermediate outcome. Measures such as carotid artery disease, coronary calcification, or endothelial dysfunction that occur without clinical signs can be measured reliably and noninvasively, can predict subsequent clinical cardiovascular events, and recently have been used to examine psychosocial hypotheses (e.g., L. C. Gallo, Matthews, Kuller, Sutton-Tyrrell, & Edmundowicz, 2001; Matthews et al., 1998). These endpoints would allow a less time- and resource-intensive approach to evaluating the model, which could provide a basis for future research with more distal health endpoints. They also have the advantage of not being as subject to an interpretation of reverse causality, because intermediate disease outcomes occur without symptoms. Consistent with our recommendations for comprehensive mediation approaches, we suggest that such tests consider the roles of additive and synergistic effects among cognitive-emotional and resource variables.

Experimental research approaches. Experimental research approaches may also facilitate tests of associations represented in the reserve capacity model. For example, using social-psychological procedures, one could experimentally manipulate key components of SES—such as access to tangible resources or prestige—and then measure the impact on mood states and intermediate health outcomes (e.g., cardiovascular responses to acute stress). The moderating effect of provision of interpersonal resources could also be evaluated through experimental manipulation. There may also be opportunities to piggy-back onto randomized interventions designed to reduce negative affect to examine whether therapeutic techniques that improve symptoms also lead to improvement in intermediate health outcomes when compared with control groups. Finally, natural experiments, such as plant closings or planned layoffs, may provide the context for examining the impact of changing social status on negative emotions and attitudes and health outcomes.

Conclusion

Available evidence supports the plausibility of the hypothesis that the association between SES and health is mediated—at least in part—by cognitive-emotional factors. Cognitive-emotional factors may play a particularly salient role in the context of a low reserve capacity, and we therefore recommend further studies that adopt a more integrative approach to examining the roles of psychosocial factors. Cognitive-emotional factors are only one potential influence to consider in unraveling the links between low SES and poor health. The challenge is to identify those factors susceptible to intervention to promote better health in the population and reduce the substantial health variability that exists according to SES. This review points to the importance of cognitive and emotional factors in the quest to reduce health inequalities.

References

- Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health: The challenge of the gradient. *American Psychologist*, *49*, 15–24.
- Adler, N. E., Boyce, T., Chesney, M. A., Folkman, S., & Syme, L. (1993). Socioeconomic inequalities in health: No easy solution. *Journal of the American Medical Association*, *269*, 3140–3145.
- Adler, N. E., Marmot, M., McEwen, B. S., & Stewart, J. (Eds.). (1999). *Annals of the New York Academy of Sciences: Vol. 896. Socioeconomic status and health in industrialized nations*. New York: New York Academy of Sciences.
- Ahern, D. K., Gorkin, L., Anderson, J. L., Tierney, C., Hallstrom, A., Ewart, C., et al. (1990). Biobehavioral variables and mortality or cardiac arrest in the Cardiac Arrhythmia Pilot Study (CAPS). *American Journal of Cardiology*, *66*, 59–62.
- Aldarondo, E., & Sugarman, D. B. (1996). Risk marker analysis of the cessation and persistence of wife assault. *Journal of Consulting and Clinical Psychology*, *64*, 1010–1019.
- Alec, R. (1996). Psychosocial factors and depression. *Journal of Nervous and Mental Disease*, *184*, 509–510.
- Allgulander, C., & Lavori, P. W. (1991). Excess mortality among 3302 patients with “pure” anxiety neurosis. *Archives of General Psychiatry*, *48*, 599–602.
- American Heart Association. (2000). *Heart and stroke statistical update*. Dallas, TX: Author.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Amsterdam, E. A. (1990). Emotions, cardiac arrhythmias, and sudden death. In D. G. Byrne & R. H. Rosenmann (Eds.), *Anxiety and the heart: The series in health psychology and behavioral medicine* (pp. 251–258). New York: Hemisphere.
- Anda, R. F., Williamson, D. F., Escobedo, L. G., Mast, E. E., Giovino, G. A., & Remington, P. L. (1990). Depression and the dynamics of smoking: A national perspective. *Journal of the American Medical Association*, *264*, 1541–1545.
- Anda, R., Williamson, D., Jones, D., Macera, C., Eaker, E., Glassman, A., & Marks, J. (1993). Depressed affect, hopelessness, and the risk of ischemic heart disease in a cohort of U.S. adults. *Epidemiology*, *4*, 285–294.
- Anderson, N. B. (1999). Solving the puzzle of socioeconomic status and health: The need for integrated, multilevel, interdisciplinary research. In N. E. Adler, M. Marmot, B. S. McEwen, & J. Stewart (Eds.), *Annals of the New York Academy of Sciences: Vol. 896. Socioeconomic status and health in industrialized nations* (pp. 302–312). New York: New York Academy of Sciences.
- Anderson, N. B., & Armstead, C. A. (1995). Toward understanding the association of socioeconomic status and health: A new challenge for the biopsychosocial approach. *Psychosomatic Medicine*, *57*, 213–225.
- Anthony, J. C., & Petronis, K. R. (1991). Suspected risk factors for depression among adults 18–44 years old. *Epidemiology*, *2*, 123–132.
- Antonovsky, A. (1967). Social class, life expectancy and overall mortality. *Milbank Quarterly*, *45*, 31–73.
- Appels, A., Bär, F. W., Bär, J., Bruggeman, C., & de Baets, M. (2000). Inflammation, depressive symptomatology, and coronary artery disease. *Psychosomatic Medicine*, *62*, 601–605.
- Aromaa, A., Raitasalo, R., Reunanen, A., Impivaara, O., Heliövaara, M., Knekt, P., et al. (1994). Depression and cardiovascular diseases. *Acta Psychiatrica Scandinavica*, *89*(Suppl. 377), 77–82.
- Backlund, E., Sorlie, P. D., & Johnson, N. J. (1996). The shape of the relationship between income and mortality in the United States: Evidence from the National Longitudinal Mortality Study. *Annals of Epidemiology*, *6*, 12–20.
- Bajwa, W. K., Asnis, G. M., Sanderson, W. C., Irfan, A., & van Praag, H. M. (1992). High cholesterol levels in patients with panic disorder. *American Journal of Psychiatry*, *149*, 376–378.

- Bandura, A. (1989). Human agency in social cognitive theory. *American Psychologist*, *44*, 1175–1184.
- Barefoot, J. C. (1992). Developments in the measurement of hostility. In H. S. Friedman (Ed.), *Hostility, coping, and health* (pp. 13–31). Washington, DC: American Psychological Association.
- Barefoot, J. C., Dahlstrom, W. G., & Williams, R. B. (1983). Hostility, CHD incidence, and total mortality: A 25-year follow-up study of 255 physicians. *Psychosomatic Medicine*, *45*, 59–63.
- Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, W. G., & Williams, R. B., Jr. (1989). The Cook–Medley Hostility Scale: Item content and ability to predict survival. *Psychosomatic Medicine*, *51*, 46–57.
- Barefoot, J. C., Helms, M. S., Mark, D. B., Blumenthal, J. A., Califf, R. M., Haney, T. L., et al. (1996). Depression and long-term mortality risk in patients with coronary artery disease. *American Journal of Cardiology*, *78*, 613–617.
- Barefoot, J. C., Larsen, S., von der Lieth, L., & Schroll, M. (1995). Hostility, incidence of acute myocardial infarction, and mortality in a sample of older Danish men and women. *American Journal of Epidemiology*, *142*, 477–484.
- Barefoot, J. C., Peterson, B. L., Dahlstrom, W. G., Siegler, I. C., Anderson, N. B., & Williams, R. B., Jr. (1991). Hostility patterns and health implications: Correlates of Cook–Medley Hostility Scale scores in a national survey. *Health Psychology*, *10*, 18–24.
- Barefoot, J. C., & Schroll, M. (1996). Symptoms of depression, acute myocardial infarction, and total mortality in a community sample. *Circulation*, *93*, 1976–1980.
- Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182.
- Beach, S. R., & O’Leary, K. D. (1993). Dysphoria and marital discord: Are dysphoric individuals at risk for marital maladjustment? *Journal of Marital & Family Therapy*, *19*, 355–368.
- Bebbington, P., Hurry, J., Tennant, J. C., Sturt, E., & Wing, J. K. (1981). The epidemiology of mental disorders in Camberwell. *Psychological Medicine*, *11*, 561–580.
- Beck, A. T. (1971). Cognition, affect, and psychopathology. *Archives of General Psychiatry*, *24*, 495–500.
- Beck, A. T., & Beamesderfer, A. (1974). Assessment of depression: The Depression Inventory. In P. Pinchot (Ed.), *Psychological measurements in psychopharmacology* (pp. 151–169). Basel, Switzerland: Karger Press.
- Belle, D. E. (1982). The impact of poverty on social networks and supports. *Marriage & Family Review*, *5*, 89–103.
- Belle, D. (1990). Poverty and women’s mental health. *American Psychologist*, *45*, 385–389.
- Ben-Shlomo, Y., & Davey Smith, G. (1991). Deprivation in infancy or adult life: Which is more important for mortality risk? *Lancet*, *337*, 530–534.
- Berkman, L. F., & Breslow, L. (1983). Health and ways of living: The Alameda County Study. New York: Oxford University Press.
- Bianchi, S. (1995). The changing demographic and socioeconomic characteristics of single parent families. *Marriage & Family Review*, *20*, 71–97.
- Blane, D., Hart, C. L., Davey Smith, G., Gillis, C. R., Hole, D. J., & Hawthorne, V. M. (1996). Association of cardiovascular disease risk factors with socioeconomic position during childhood and during adulthood. *British Medical Journal*, *313*, 1434–1438.
- Blazer, D. G., Hughes, D., George, L. K., Swartz, M., & Boyer, R. (1991). Generalized anxiety disorder. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric disorders in America* (pp. 180–203). New York: Free Press.
- Blazer, D. G., Kessler, R. C., McGonagle, K. A., & Swartz, M. S. (1994). The prevalence and distribution of major depression in a national community sample: The National Comorbidity Survey. *American Journal of Psychiatry*, *151*, 979–986.
- Blumenthal, J. A., Williams, R. S., Wallace, A. G., Williams, R. B., & Needles, T. L. (1982). Physiological and psychological variables predict compliance to prescribed exercise therapy in patients recovering from myocardial infarction. *Psychosomatic Medicine*, *44*, 519–527.
- Bolger, N., Foster, M., Vinokur, A. D., & Ng, R. (1996). Close relationships and adjustment to a life crisis: The case of breast cancer. *Journal of Personality and Social Psychology*, *70*, 283–294.
- Bolger, N., & Zuckerman, A. (1995). A framework for studying personality in the stress process. *Journal of Personality and Social Psychology*, *69*, 890–902.
- Bosma, H., Van de Mheen, H. D., & Mackenbach, J. P. (1999). Social class in childhood and general health in adulthood: Questionnaire study of contribution of psychological attributes. *British Medical Journal*, *318*, 18–22.
- Bradley, R. H., & Corwyn, R. F. (2002). Socioeconomic status and child development. *Annual Review of Psychology*, *53*, 371–399.
- Breslau, N. M., Kilbey, M., & Andreski, P. (1991). Nicotine dependence, major depression, and anxiety in young adults. *Archives of General Psychiatry*, *48*, 1069–1074.
- Brown, G. W., & Bifulco, A. (1990). Motherhood, employment, and the development of depression: A replication of a finding. *British Journal of Psychiatry*, *156*, 169–179.
- Brown, G. W., & Harris, T. (1978). *Social origins of depression*. London: Tavistock.
- Brown, G. W., & Moran, P. M. (1997). Single mothers, poverty, and depression. *Psychological Medicine*, *27*, 21–33.
- Brown, R. (1965). *Social psychology*. New York: Free Press.
- Bruce, M. L., Leaf, P. J., Rozal, G. P., Florio, L., & Hoff, R. A. (1994). Psychiatric status and 9-year mortality data in the New Haven Epidemiologic Catchment Area Study. *American Journal of Psychiatry*, *151*, 716–721.
- Bruce, M. L., Takeuchi, D. T., & Leaf, P. J. (1991). Poverty and psychiatric status. *Archives of General Psychiatry*, *48*, 470–474.
- Brugha, T. (1995). *Social support and psychiatric disorder*. Cambridge, England: Cambridge University Press.
- Bureau of Labor Statistics. (2000). *Current population survey: Civilian employment–population ratio* (Series LFS1601702). Washington, DC: Author.
- Carmelli, D., Rosenman, R. H., & Swan, G. E. (1988). The Cook and Medley Ho Scale: A heritability analysis in adult male twins. *Psychosomatic Medicine*, *50*, 165–170.
- Carmody, T. P., Crossen, J. R., & Wiens, A. N. (1989). Hostility as a health risk factor: Relationships with neuroticism, Type A behavior, attentional focus, and interpersonal style. *Journal of Clinical Psychology*, *45*, 754–762.
- Carney, R. M., Freedland, K. E., Eisen, S. A., Rich, M. W., & Jaffe, A. S. (1995). Major depression and medication adherence in elderly patients with coronary artery disease. *Health Psychology*, *14*, 88–90.
- Carney, R. M., Freedland, K. E., & Jaffe, A. S. (2001). Depression as a risk factor for coronary heart disease mortality. *Archives of General Psychiatry*, *58*, 229–230.
- Carney, R. M., Freedland, K. E., Sheline, Y., & Weiss, E. (1997). Depression and coronary heart disease: A review for cardiologists. *Clinical Cardiology*, *20*, 196–200.
- Carney, R. M., Rich, M. W., Freedland, K. E., & Sanai, J. (1988). Major depressive disorder predicts cardiac events in patients with coronary artery disease. *Psychosomatic Medicine*, *50*, 627–633.
- Carney, R. M., Rich, M., & Jaffe, A. S. (1995). Depression as a risk factor for cardiac events in established coronary heart disease: A review of possible mechanisms. *Annals of Behavioral Medicine*, *17*, 142–149.
- Carroll, D., Bennett, P., & Davey Smith, G. (1993). Socio-economic health

- inequalities: Their origins and implications. *Psychological Health*, 8, 295–316.
- Casale, P. N., Devereux, R. B., & Milner, M. (1986). Value of echocardiographic measurement of left ventricular mass in predicting cardiovascular morbid events in hypertensive men. *Annals of Internal Medicine*, 105, 173–178.
- Caspi, A., Elder, G. H., & Bem, D. J. (1987). Moving against the world: Life-course patterns of explosive children. *Developmental Psychology*, 23, 308–313.
- Chapin, C. F. (1924). Deaths among taxpayers and non-taxpayers of income tax, Providence, 1865. *American Journal of Public Health*, 14, 647–651.
- Chen, E., & Matthews, K. A. (2001). Cognitive appraisal biases: An approach to understanding the relationship between socioeconomic status and cardiovascular reactivity in children. *Annals of Behavioral Medicine*, 23, 101–111.
- Chen, E., Matthews, K. A., & Boyce, W. T. (2002). Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychological Bulletin*, 128, 295–329.
- Christensen, A. J., Edwards, D. L., Wiebe, J. S., Benotsch, E. G., & McKelvey, L. (1996). Effect of verbal self-disclosure on NK cell activity: Moderating influence of cynical hostility. *Psychosomatic Medicine*, 58, 150–155.
- Christmas, A. L., Wodarski, J. S., & Smokowski, P. R. (1996). Risk factors for physical child abuse: A practice theoretical paradigm. *Family Therapy*, 23, 233–248.
- Clark, L. A., & Watson, D. (1991). Theoretical and empirical issues in differentiating depression from anxiety. In J. Becker & A. Kleinman (Eds.), *Psychosocial aspects of depression* (pp. 39–65). Hillsdale, NJ: Erlbaum.
- Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, 103, 103–116.
- Clore, G. L. (1994). Why emotions require cognition. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 181–191). New York: Oxford University Press.
- Cohen, S., & Herbert, T. B. (1996). Health psychology: Psychological factors and disease from the perspective of human psychoneuroimmunology. *Annual Review of Psychology*, 47, 113–142.
- Cohen, S., Kaplan, G. A., & Salonen, J. T. (1999). The role of psychological characteristics in the relation between socioeconomic status and perceived health. *Journal of Applied Social Psychology*, 29, 445–468.
- Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, 98, 310–357.
- Comstock, G. W., & Helsing, K. J. (1976). Symptoms of depression in two communities. *Psychological Medicine*, 6, 551–563.
- Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology*, 6, 414–418.
- Coryell, W., Endicott, J., & Keller, M. (1992). Major depression in a nonclinical sample: Demographic and clinical risk factors for first onset. *Archives of General Psychiatry*, 49, 117–125.
- Coryell, W., Noyes, R., & Clancy, J. (1982). Excess mortality in panic disorder. *Archives of General Psychiatry*, 39, 701–703.
- Coryell, W., Noyes, R., & House, D. (1986). Mortality among outpatients with anxiety disorders. *American Journal of Psychiatry*, 143, 608–610.
- Covinsky, K. A., Kahana, E., Chin, M., Palmer, R., Fortinsky, R., & Landefeld, S. (1999). Depressive symptoms and 3-year mortality in older hospitalized medical patients. *Annals of Internal Medicine*, 130, 563–569.
- Craig, T. J., & Van Natta, P. A. (1979). Influence of demographic characteristics on two measures of depressive symptoms. *Archives of General Psychiatry*, 36, 149–154.
- Daly, M., Duncan, G., Kaplan, G. A., & Lynch, J. W. (1998). Macro-to-micro linkages in the inequality-mortality relationship. *Milbank Quarterly*, 76, 315–339.
- Davey Smith, G., Hart, C., Blane, D., Gillis, C., & Hawthorne, V. (1997). Lifetime socioeconomic position and mortality: Prospective observational study. *British Medical Journal*, 314, 547–552.
- Davey Smith, G., Hart, C., Blane, D., & Hole, D. (1998). Adverse socioeconomic conditions in childhood and cause specific adult mortality: Prospective observational study. *British Medical Journal*, 316, 1631–1635.
- Dembroski, T. M., MacDougall, J. M., Williams, R. B., Haney, T. L., & Blumenthal, J. A. (1985). Components of Type A, hostility and anger-in: Relationship to angiographic findings. *Psychosomatic Medicine*, 47, 219–233.
- Denollet, J., & Brutsaert, D. L. (1998). Personality, disease severity, and the risk of long-term cardiac events in patients with a decreased ejection fraction after myocardial infarction. *Circulation*, 97, 167–73.
- Dew, M. A. (1998). Psychiatric disorder in the context of physical illness. In B. P. Dohrenwend (Ed.), *Adversity, stress, and psychopathology* (pp. 177–218). New York: Oxford University Press.
- Diez-Roux, A. V. (1998). Bringing context back into epidemiology: Variables and fallacies in multilevel analysis. *American Journal of Public Health*, 88, 216–222.
- Diez-Roux, A. V., Merkin, S. S., Arnett, D., Chambless, L., Massing, M., Nieto, F. J., et al. (2001). Neighborhood of residence and incidence of coronary heart disease. *New England Journal of Medicine*, 345, 99–106.
- Diez-Roux, A. V., Nieto, F. J., Muntaner, C., Tyroler, H. A., Comstock, G. W., Shahr, E., et al. (1997). Neighborhood environments and coronary heart disease: A multilevel analysis. *American Journal of Epidemiology*, 146, 48–63.
- Dohrenwend, B. P. (1990). Socioeconomic status (SES) and psychiatric disorders: Are the issues still compelling? *Social Psychiatry and Psychiatric Epidemiology*, 25, 41–47.
- Dohrenwend, B. P. (2000). The role of adversity and stress in psychopathology: Some evidence and its implications for theory and research. *Journal of Health and Social Behavior*, 41, 1–19.
- Dohrenwend, B. P., & Dohrenwend, B. S. (1969). *Social status and psychological disorder*. New York: Wiley.
- Dohrenwend, B. P., Levav, I., Shrout, P., Schwartz, S., Naveh, G., Link, B. G., et al. (1998). Ethnicity, socioeconomic status, and psychiatric disorders: A test of the social causation–social selection issue. In B. P. Dohrenwend (Ed.), *Adversity, stress, and psychopathology* (pp. 285–318). New York: Oxford University Press.
- Dohrenwend, B. S. (1973). Social status and stressful life events. *Journal of Personality and Social Psychology*, 28, 225–235.
- Doornbos, G., & Kromhout, D. (1990). Educational level and mortality in a 32-year follow-up study of 18-year-old men in the Netherlands. *International Journal of Epidemiology*, 19, 374–379.
- Drever, F., Whitehead, M., & Roden, M. (1996). Current patterns and trends in male mortality by social class (based on occupation). *Population Trends*, 86, 15–20.
- Duncan, G. J. (1996). Income dynamics and health. *International Journal of Health Services*, 26, 419–444.
- Eaker, E. D., Pinsky, J., & Castelli, W. P. (1992). Myocardial infarction and coronary death among women: Psychosocial predictors from a 20-year follow-up of women in the Framingham Study. *American Journal of Epidemiology*, 135, 854–864.
- Eaton, W. W., Armenian, H., Gallo, J., Pratt, L., & Ford, D. E. (1996). Depression and risk for onset of Type II diabetes: A prospective population-based study. *Diabetes Care*, 19, 1097–1102.
- Eaton, W. W., Dryman, A., & Weissman, M. M. (1991). Panic and phobia. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric disorders in America* (pp. 155–179). New York: Free Press.
- Eaton, W. W., Kessler, R. C., Wittchen, H. U., & Magee, W. J. (1994).

- Panic and panic disorder in the United States. *American Journal of Psychiatry*, *151*, 413–420.
- Eaton, W. W., & Keyl, P. M. (1990). Risk factors for the onset of diagnostic interview schedule/DSM-III agoraphobia in a prospective population-based study. *Archives of General Psychiatry*, *47*, 819–824.
- Eaton, W. W., & Muntaner, C. (1999). Socioeconomic stratification and mental disorder. In A. V. Horwitz & T. L. Scheid (Eds.), *A handbook for the study of mental health: Social contexts, theories, and systems* (pp. 259–283). New York: Cambridge University Press.
- Ecob, R., & Davey Smith, G. (1999). Income and health: What is the nature of the relationships? *Social Science & Medicine*, *48*, 693–705.
- Ehlert, U., & Straub, R. (1998). Physiological and emotional response to psychological stressors in psychiatric and psychosomatic disorders. In P. Csermely (Ed.), *Annals of the New York Academy of Sciences: Vol. 851. Stress of life: From molecules to man* (pp. 477–486). New York: New York Academy of Sciences.
- Ekman, P., & Davidson, R. (1994). *The nature of emotion: Fundamental questions*. New York: Oxford University Press.
- Endicott, J., & Spitzer, R. L. (1979). Use of the Research Diagnostic Criteria and the Schedule for Affective Disorders and Schizophrenia to study affective disorders. *American Journal of Psychiatry*, *136*, 52–56.
- Ennis, N. E., Hobfoll, S. E., & Schröder, K. E. E. (2000). Money doesn't talk, it swears: How economic stress and resistance resources impact inner-city women's depressive mood. *American Journal of Community Psychology*, *28*, 149–173.
- Ensel, W. M., & Lin, N. (1991). The life stress paradigm and psychological distress. *Journal of Health and Social Behavior*, *32*, 321–341.
- Evans, R. G., Barer, M. L., & Marmor, T. R. (Eds.). (1994). *Why are some people healthy and others not? The determinants of health of populations*. New York: de Gruyter.
- Everson, S. A., George, K. A., Goldberg, D. E., Lakka, T. A., Sivenius, J., & Salonen, J. T. (1999). Anger expression and incident stroke: Prospective evidence from the Kuopio Ischemic Heart Disease Study. *Stroke*, *30*, 523–528.
- Everson, S. A., Goldberg, D. E., Kaplan, G. A., Cohen, R. D., Pukkala, E., Tuomilehto, J., & Salonen, J. T. (1996). Hopelessness and risk of mortality and incidence of myocardial infarction and cancer. *Psychosomatic Medicine*, *58*, 113–121.
- Everson, S. A., Kauhanen, J., Kaplan, G. A., Goldberg, D. E., Julkunen, J., Tuomilehto, J., & Salonen, J. T. (1997). Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *American Journal of Epidemiology*, *146*, 142–152.
- Everson, S. A., Roberts, R. E., Goldberg, D. E., & Kaplan, G. A. (1998). Depressive symptoms and increased risk of stroke mortality over a 29-year period. *Archives of Internal Medicine*, *158*, 1133–1138.
- Farmer, M. E., Locke, B. Z., Moscicki, E. K., Dannenberg, A. L., Larson, D. B., & Radloff, L. S. (1988). Physical activity and depressive symptoms: The NHANES I epidemiologic follow-up study. *American Journal of Epidemiology*, *128*, 1340–1351.
- Feldman, J. J., Makuc, D. M., Kleinman, J. C., & Cornoni-Huntley, J. (1989). National trends in educational differentials in mortality. *American Journal of Epidemiology*, *129*, 919–933.
- Feldman, L. A. (1995). Variations in the circumplex structure of mood. *Personality and Social Psychology Bulletin*, *21*, 806–817.
- Fiscella, K., & Franks, P. (1997). Does psychological distress contribute to racial and socioeconomic disparities in mortality? *Social Science & Medicine*, *45*, 1805–1809.
- Fleet, R. P., & Beitman, B. D. (1998). Cardiovascular death from panic disorder and panic-like anxiety: A critical review of the literature. *Journal of Psychosomatic Research*, *44*, 71–80.
- Flory, J. D., Matthews, K. A., & Owens, J. F. (1998). A social information processing approach to dispositional hostility: Relationships with negative mood and blood pressure elevations at work. *Journal of Social and Clinical Psychology*, *17*, 491–504.
- Ford, D. E., Mead, L. A., Chang, P. P., Cooper-Patrick, L., Wang, N., & Klag, M. J. (1998). Depression is a risk factor for coronary artery disease in men. *Archives of Internal Medicine*, *158*, 1422–1426.
- Fox, J. W. (1990). Social class, mental illness, and social mobility: The social selection-drift hypothesis for serious mental illness. *Journal of Health and Social Behavior*, *31*, 344–353.
- Frasure-Smith, N., Lespérance, F., Gravel, G., Masson, A., Juneau, M., Talajic, M., & Bourassa, M. G. (2000). Social support, depression, and mortality during the first year after myocardial infarction. *Circulation*, *101*, 1919–1924.
- Frasure-Smith, N., Lespérance, F., Juneau, M., Talajic, M., & Bourassa, M. G. (1999). Gender, depression, and one-year prognosis after myocardial infarction. *Psychosomatic Medicine*, *61*, 26–37.
- Frasure-Smith, N., Lespérance, F., & Talajic, M. (1993). Depression following myocardial infarction: Impact on 6-month survival. *Journal of the American Medical Association*, *270*, 1819–1825.
- Frasure-Smith, N., Lespérance, F., & Talajic, M. (1995a). Depression and 18-month prognosis after myocardial infarction. *Circulation*, *91*, 999–1005.
- Frasure-Smith, N., Lespérance, F., & Talajic, M. (1995b). The impact of negative emotions on prognosis following myocardial infarction: Is it more than depression? *Health Psychology*, *14*, 388–398.
- Fredrickson, B. L., & Levenson, R. W. (1998). Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cognition & Emotion*, *12*, 191–220.
- Frijda, N. H. (1994). Emotions are functional most of the time. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 112–122). New York: Oxford University Press.
- Gallo, J. J., Royall, D. R., & Anthony, J. C. (1993). Risk factors for the onset of depression in middle age and later life. *Social Psychiatry and Psychiatric Epidemiology*, *28*, 101–108.
- Gallo, L. C., & Matthews, K. A. (1999). Do negative emotions mediate the association between socioeconomic status and health? In N. E. Adler, M. Marmot, B. S. McEwen, & J. Stewart (Eds.), *Annals of the New York Academy of Sciences: Vol. 896. Socioeconomic status and health in industrialized nations* (pp. 226–245). New York: New York Academy of Sciences.
- Gallo, L. C., Matthews, K. A., Kuller, L., Sutton-Tyrrell, K., & Edmondowicz, D. (2001). Educational attainment and coronary and aortic calcification in post-menopausal women. *Psychosomatic Medicine*, *63*, 925–935.
- Gallo, L. C., & Smith, T. W. (1999). Patterns of hostility and social support: Conceptualizing psychosocial risk factors as characteristics of the person and the environment. *Journal of Research in Personality*, *33*, 281–310.
- Gary, L. (1995). African American men's perceptions of racial discrimination. *Social Work Research*, *19*, 207–217.
- Gecas, V., & Seff, M. (1989). Social class, occupational conditions, and self-esteem. *Sociological Perspectives*, *32*, 353–364.
- Glassman, A. H., & Shapiro, P. A. (1998). Depression and the course of coronary artery disease. *American Journal of Psychiatry*, *155*, 4–11.
- Gonzalez, M. A., Rodriguez, A. F., & Calero, J. R. (1998). Relationship between socioeconomic status and ischemic heart disease in cohort and case-control studies: 1960–1993. *International Journal of Epidemiology*, *27*, 350–358.
- Gonzalez, M. B., Snyderman, T. B., Colket, J. T., Arias, R. M., Jiang, J. W., O'Connor, C. M., & Krishnan, K. R. (1996). Depression in patients with coronary artery disease. *Depression*, *4*, 57–62.
- Goodman, E. (1999). The role of socioeconomic status gradients in explaining differences in U.S. adolescents' health. *American Journal of Public Health*, *89*, 1522–1528.
- Gracia, E., Garcia, F., & Musitu, G. (1995). Macrosocial determinants of social integration: Social class and area effect. *Journal of Community & Applied Social Psychology*, *5*, 105–119.
- Greene, S. M. (1989). The relationship between depression and hopeless-

- ness: Implications for current theories of depression. *British Journal of Psychiatry*, 154, 650–659.
- Guiry, E., Conroy, R. M., Hickey, N., & Mulcahy, R. (1987). Psychological response to an acute coronary event and its effect on subsequent rehabilitation and lifestyle change. *Clinical Cardiology*, 10, 256–260.
- Gump, B. B., Matthews, K. A., & Räikkönen, K. (1999). Modeling relationships among socioeconomic status, hostility, cardiovascular reactivity, and left ventricular mass in African American and White children. *Health Psychology*, 18, 140–150.
- Haaga, D. A. F., Dyck, M. J., & Ernst, D. (1991). Empirical status of cognitive theory of depression. *Psychological Bulletin*, 110, 215–236.
- Haan, M., Kaplan, G. A., & Camacho, T. (1987). Poverty and health: Prospective evidence from the Alameda County Study. *American Journal of Epidemiology*, 125, 989–998.
- Haines, A. P., Imeson, J. D., & Meade, T. W. (1987). Phobic anxiety and ischemic heart disease. *British Medical Journal*, 295, 297–299.
- Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology*, 100, 555–561.
- Hayward, C. (1995). Psychiatric illness and cardiovascular disease risk. *Epidemiologic Reviews*, 17, 129–138.
- Hearn, M. D., Murray, D. M., & Luepker, R. V. (1989). Hostility coronary heart disease, and total mortality: A 33-year follow-up study of university students. *Journal of Behavioral Medicine*, 12, 105–121.
- Hecker, M. H. L., Chesney, M. A., Black, G. W., & Frautchi, N. (1988). Coronary-prone behaviors in the Western Collaborative Group Study. *Psychosomatic Medicine*, 50, 153–164.
- Herbert, T. B., & Cohen, S. (1993). Depression and immunity: A meta-analytic review. *Psychological Bulletin*, 113, 472–486.
- Hermann, C., Brand-Driehorst, S., Kaminsky, B., Leibing, E., Staats, H., & Ruger, U. (1998). Diagnostic groups and depressed mood as predictors of 22-month mortality in medical inpatients. *Psychosomatic Medicine*, 60, 570–577.
- Himmelfarb, S., & Murrell, S. A. (1984). The prevalence and correlates of anxiety symptoms in older adults. *Journal of Psychology*, 116, 159–167.
- Hobfoll, S. E. (1988). *The ecology of stress*. New York: Hemisphere.
- Hobfoll, S. E. (1989). Conservation of resources: A new attempt at conceptualizing stress. *American Psychologist*, 44, 513–524.
- Hobfoll, S. E. (1998). *Stress, culture, and community: The psychology and philosophy of stress*. New York: Plenum Press.
- Hobfoll, S. E. (2001). The influence of culture, community, and the nested-self in the stress process: Advancing conservation of resources theory. *Applied Psychology*, 50, 337–370.
- Hogan, D. P., Hao, L. X., & Parish, W. L. (1990). Race, kin networks, and assistance to mother-headed families. *Social Forces*, 68, 797–812.
- Hokanson, J. E., Rubert, M. P., Welker, R. A., Hollander, G. R., & Hedeem, C. (1989). Interpersonal concomitants and antecedents of depression among college students. *Journal of Abnormal Psychology*, 98, 209–217.
- Holahan, C. J., & Moos, R. H. (1987). Risk, resistance, and psychological distress: A longitudinal analysis with adults and children. *Journal of Abnormal Psychology*, 96, 3–13.
- Holahan, C. J., & Moos, R. H. (1991). Life stressors, personal and social resources, and depression: A 4-year structural model. *Journal of Abnormal Psychology*, 100, 31–38.
- Holahan, C. J., Moos, R. H., Holahan, C. K., & Cronkite, R. C. (1999). Resource loss, resource gain, and depressive symptoms: A 10-year model. *Journal of Personality and Social Psychology*, 77, 620–629.
- Holahan, C. J., Moos, R. H., Holahan, C. K., & Cronkite, R. C. (2000). Long-term posttreatment functioning among patients with unipolar depression: An integrative model. *Journal of Consulting and Clinical Psychology*, 68, 226–232.
- Hollingshead, A. B. (1975). *Four Factor Index of Social Status*. New Haven, CT: Hollingshead.
- Holme, I., Helgeland, A., Hjermann, I., & Leren, P. (1982). Socio-economic status as a coronary risk factor: The Oslo study. *Acta Medica Scandinavica*, 660(Suppl.), 147–151.
- Horowitz, M. J., Berfari, R., Hulley, S., Blair, S., Alvarez, W., Borhani, N., et al. (1979). Life events, risk factors, and coronary disease. *Psychosomatics*, 20, 586–592.
- Horwath, E., Johnson, J., Klerman, G. L., & Weissman, M. M. (1992). Depressive symptoms as relative and attributable risk factors for first-onset major depression. *Archives of General Psychiatry*, 49, 817–823.
- House, J. S., Lepkowski, J. M., Kinney, A. M., Mero, R. P., Kessler, R. C., & Herzog, A. R. (1994). The social stratification of aging and health. *Journal of Health and Social Behavior*, 35, 213–234.
- Houston, B. K., & Kelly, K. E. (1989). Hostility in employed women: Relation to work and marital experiences, social support, stress, and anger expression. *Personality and Social Psychology Bulletin*, 15, 175–182.
- Hughes, J. R., Hatsukami, D. K., Mitchell, J. E., & Dahlgren, L. A. (1986). Prevalence of smoking among psychiatric outpatients. *American Journal of Psychiatry*, 143, 993–997.
- Ickovics, J. R., Viscoli, C. M., & Horwitz, R. I. (1997). Functional recovery after myocardial infarction in men: The independent effects of social class. *Annals of Internal Medicine*, 127, 518–525.
- Ilsey, R., & Baker, D. (1991). Contextual variations in the meaning of health inequality. *Social Science & Medicine*, 32, 359–365.
- Iribarren, C., Sidney, S., Bild, D. E., Liu, K., Markovitz, J. H., Roseman, J. M., & Matthews, K. (2000). Association of hostility with coronary artery calcification in young adults: The CARDIA study. Coronary artery risk development in young adults. *Journal of the American Medical Association*, 283, 2546–2551.
- Johnson, J. G., Cohen, P., Dohrenwend, B. P., Link, B. G., & Brook, J. S. (1999). A longitudinal investigation of social causation and social selection processes involved in the association between socioeconomic status and psychiatric disorders. *Journal of Abnormal Psychology*, 108, 490–499.
- Johnson, S. L., & Jacob, T. (1997). Marital interactions of depressed men and women. *Journal of Consulting and Clinical Psychology*, 65, 15–23.
- Johnson, T. P. (1991). Mental health, social relations, and social selection: A longitudinal analysis. *Journal of Health and Social Behavior*, 32, 408–423.
- Joiner, T., & Coyne, J. (1999). *The interactional nature of depression: Advances in interpersonal approaches*. Washington, DC: American Psychological Association.
- Jonas, B. S., Franks, P., & Ingram, D. D. (1997). Are symptoms of anxiety and depression risk factors for hypertension? Longitudinal evidence from the National Health and Nutrition Examination Survey I epidemiologic follow-up study. *Archives of Family Medicine*, 6, 43–49.
- Jonas, B. S., & Mussolino, M. E. (2000). Symptoms of depression as a prospective risk factor for stroke. *Psychosomatic Medicine*, 62, 463–471.
- Judd, L. L., & Akiskal, H. S. (2000). Delineating the longitudinal structure of depressive illness: Beyond clinical subtypes and duration thresholds. *Pharmacopsychiatry*, 33, 3–7.
- Julkunen, J., Salonen, R., Kaplan, G. A., Chesney, M. A., & Salonen, J. T. (1994). Hostility and the progression of carotid atherosclerosis. *Psychosomatic Medicine*, 56, 519–525.
- Jung, J. (1989). The relationship of daily hassles, social support, and coping to depression in Black and White students. *Journal of General Psychology*, 116, 407–417.
- Kaplan, G. A. (1995). Where do shared pathways lead? Some reflections on a research agenda. *Psychosomatic Medicine*, 57, 208–212.
- Kaplan, G. A., & Keil, J. E. (1993). Socioeconomic factors and cardiovascular disease: A review of the literature. *Circulation*, 88, 1973–1998.
- Kaplan, G. A., Roberts, R. E., Camacho, T. C., & Coyne, J. (1987). Psychosocial predictors of depression. *American Journal of Epidemiology*, 125, 206–220.

- Kaplan, G. A., & Salonen, J. T. (1990). Socioeconomic conditions in childhood and ischemic heart disease during middle age. *British Medical Journal*, *301*, 1121–1123.
- Kaplan, G. A., Wilson, T. W., Cohen, R. D., Kauhanen, J., Wu, M., & Salonen, J. T. (1994). Social functioning and overall mortality: Prospective evidence from the Kuopio Ischemic Heart Disease Risk Factor Study. *Epidemiology*, *5*, 495–500.
- Kawachi, I., Colditz, G. A., Ascherio, A., Rimm, E. B., Giovannucci, E., Stampfer, M. J., & Willett, W. C. (1994). Prospective study of phobic anxiety and risk of coronary heart disease in men. *Circulation*, *89*, 1992–1997.
- Kawachi, I., Sparrow, D., Spiro, A., Vokonas, P., & Weiss, S. T. (1996). A prospective study of anger and coronary heart disease: The normative aging study. *Circulation*, *94*, 2090–2095.
- Kawachi, I., Sparrow, D., Vokonas, P. S., & Weiss, S. T. (1994). Symptoms of anxiety and risk of coronary heart disease: The normative aging study. *Circulation*, *90*, 2225–2229.
- Kennedy, B. P., Kawachi, I., Glass, R., & Prothrow-Stith, D. (1998). Income distribution, socioeconomic status, and self-rated health in the United States: Multilevel analysis. *British Medical Journal*, *317*, 917–921.
- Kennedy, G. J., Hofer, M. A., Cohen, D., Shindldecker, R., & Fisher, J. D. (1987). Significance of depression and cognitive impairment in patients undergoing programmed stimulation of cardiac arrhythmias. *Psychosomatic Medicine*, *49*, 410–421.
- Kessler, R. C. (1979). Stress, social status, and psychological distress. *Journal of Health and Social Behavior*, *20*, 259–272.
- Kessler, R. C. (1982). A disaggregation of the relationship between socioeconomic status and psychological distress. *American Sociological Review*, *47*, 752–764.
- Kessler, R. C., & Cleary, P. D. (1980). Social class and psychological distress. *American Sociological Review*, *45*, 463–478.
- Kessler, R. C., Foster, C. L., Saunders, W. B., & Stang, P. E. (1995). Social consequences of psychiatric disorders, I: Educational attainment. *American Journal of Psychiatry*, *152*, 1026–1032.
- Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. *Archives of General Psychiatry*, *51*, 8–19.
- Keyl, P. M., & Eaton, W. W. (1990). Risk factors for the onset of panic disorder and other panic attacks in a prospective, population-based study. *American Journal of Epidemiology*, *131*, 301–311.
- Kitagawa, E. M., & Hauser, P. M. (1973). *Differential mortality in the United States: A study in socioeconomic epidemiology*. Cambridge, MA: Harvard University Press.
- Kohn, R., Dohrenwend, B. P., & Mirotznik, J. (1998). Epidemiological findings on selected psychiatric disorders in the general population. In B. P. Dohrenwend (Ed.), *Adversity, stress, and psychopathology* (pp. 235–284). New York: Oxford University Press.
- Kop, W. J. (1999). Chronic and acute psychological risk factors for clinical manifestations of coronary artery disease. *Psychosomatic Medicine*, *61*, 476–487.
- Koskenvuo, M., Kaprio, J., Rose, R. J., Kesaniemi, A., Sarna, S., Heikkilä, K., & Langinvaio, H. (1988). Hostility as a risk factor for mortality and ischemic heart disease in men. *Psychosomatic Medicine*, *50*, 330–340.
- Kouzis, A. C., & Eaton, W. W. (2000). Psychopathology and the initiation of disability payments. *Psychiatric Services*, *51*, 908–913.
- Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology*, *53*, 341–369.
- Kraus, J. F., Borhani, N. O., & Franti, C. E. (1980). Socioeconomic status, ethnicity, and risk of coronary heart disease. *American Journal of Public Health*, *82*, 703–710.
- Krause, N. (1991). Stress and isolation from close ties in later life. *Journal of Gerontology: Social Sciences*, *46*, 183–195.
- Krause, N. (1997). Social support and feelings of personal control in later life. In G. R. Pierce & B. Lakey (Eds.), *Sourcebook of social support and personality* (pp. 335–355). New York: Plenum Press.
- Krause, N., & Borawski-Clark, E. (1995). Social class differences in social support among older adults. *Gerontologist*, *35*, 498–508.
- Krieger, N. (2000). Discrimination and health. In L. F. Berkman & I. Kawachi (Eds.), *Social epidemiology* (pp. 36–75). New York: Oxford University Press.
- Krieger, N., Williams, D. R., & Moss, N. E. (1997). Measuring social class in U.S. public health research: Concepts, methodologies, and guidelines. *Annual Review of Public Health*, *18*, 341–378.
- Kubzansky, L. D., Kawachi, I., & Sparrow, D. (1999). Socioeconomic status, hostility, and risk factor clustering in the Normative Aging Study: Any help from the concept of allostatic load? *Annals of Behavioral Medicine*, *21*, 330–338.
- Kubzansky, L. D., Kawachi, I., Spiro, A., Weiss, S. T., Vokonas, P. S., & Sparrow, D. (1997). Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the Normative Aging Study. *Circulation*, *95*, 818–824.
- Kubzansky, L. D., Kawachi, I., Weiss, S. T., & Sparrow, D. (1998). Anxiety and coronary heart disease: A synthesis of epidemiological, psychological, and experimental evidence. *Annals of Behavioral Medicine*, *20*, 47–58.
- Kuh, D., & Ben-Shlomo, Y. (1997). *A life course approach to chronic disease epidemiology*. Oxford, England: Oxford University Press.
- Lachman, M. E., & Weaver, S. L. (1998). The sense of control as a moderator of social class differences in health and well-being. *Journal of Personality and Social Psychology*, *74*, 763–773.
- Landrine, H., & Klonoff, E. A. (1996). The schedule of racist events: A measure of racial discrimination and a study of its negative physical and mental health consequences. *Journal of Black Psychology*, *22*, 144–168.
- Lane, D., Carroll, D., Ring, C., Beevers, D. G., & Lip, G. Y. (2001). Mortality and quality of life 12 months after myocardial infarction: Effects of depression and anxiety. *Psychosomatic Medicine*, *63*, 221–230.
- Langer, T. S., & Michael, S. T. (1963). *Life stress and mental health*. New York: Free Press.
- Lantz, P. M., House, J. S., Lepowski, J. M., Williams, D. R., Mero, R. P., & Chen, J. (1998). Socioeconomic factors, health behaviors, and mortality: Results from a nationally representative prospective study of U.S. adults. *Journal of the American Medical Association*, *279*, 1703–1708.
- Lau, R. R. (1988). Beliefs about control and health behavior. In D. S. Gochman (Ed.), *Health behavior: Emerging research perspectives* (pp. 43–63). New York: Plenum Press.
- Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.
- Leiker, M., & Hailey, B. J. (1988). A link between hostility and disease: Poor health habits? *Behavioral Medicine*, *3*, 129–133.
- Leon, G. R., Finn, S. E., Murray, D., & Bailey, J. M. (1988). The inability to predict cardiovascular disease from hostility scores or MMPI items related to Type A behavior. *Journal of Consulting and Clinical Psychology*, *56*, 597–600.
- Lespérance, F., Frasure-Smith, N., Juneau, M., & Thérioux, P. (2000). Depression and 1-year prognosis in unstable angina. *Archives of Internal Medicine*, *160*, 1354–1360.
- Levenstein, S., & Kaplan, G. A. (1998). Socioeconomic status and ulcer. A prospective study of contributory risk factors. *Journal of Clinical Gastroenterology*, *26*, 14–27.
- Levy, D., Garrison, R. J., Savage, D. D., Kannel, W. B., & Castelli, W. P. (1990). Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *New England Journal of Medicine*, *322*, 1561–1566.

- Lewinsohn, P. M., Steinmetz, J. L., Larson, D. W., & Franklin, J. (1981). Depression-related cognitions: Antecedent or consequence? *Journal of Abnormal Psychology, 90*, 213–219.
- Lichtenstein, P., Harris, J. R., Pedersen, N. L., & McClearn, G. E. (1993). Socioeconomic status and physical health, how are they related? An empirical study based on twins reared apart and twins reared together. *Social Science & Medicine, 36*, 441–450.
- Link, B. G., Lennon, M. C., & Dohrenwend, B. P. (1993). Socioeconomic status and depression: The role of occupations involving direction, control, and planning. *American Journal of Sociology, 98*, 1351–1387.
- Link, B. G., & Phelan, J. C. (1995). Social conditions as fundamental causes of disease. *Journal of Health and Social Behavior, 92*, 80–94.
- Link, B. G., & Phelan, J. C. (1996). Understanding sociodemographic differences in health—the role of fundamental social causes. *American Journal of Public Health, 86*, 471–473.
- Lockhart, L. L. (1987). A reexamination of the effects of race and social class on the incidence of marital violence: A search for reliable difference. *Journal of Marriage and the Family, 49*, 603–610.
- Lown, B. (1982). Mental stress, arrhythmias and sudden death. *American Journal of Medicine, 72*, 177–180.
- Lynch, J., & Kaplan, G. (2000). Socioeconomic position. In L. F. Berkman & I. Kawachi (Eds.), *Social epidemiology* (pp. 13–35). New York: Oxford University Press.
- Lynch, J. W., Kaplan, G. A., Cohen, R. D., Tuomilehto, J., & Salonen, J. T. (1996). Do cardiovascular risk factors explain the relation between socioeconomic status, risk of all-cause mortality, cardiovascular mortality, and acute myocardial infarction? *American Journal of Epidemiology, 144*, 934–942.
- Lynch, J. W., Kaplan, G. A., & Salonen, J. T. (1997). Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse. *Social Science & Medicine, 44*, 809–819.
- Lynch, J. W., Kaplan, G. A., & Shema, S. J. (1997). Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. *New England Journal of Medicine, 337*, 1889–1895.
- Lynch, J. W., Krause, N., Kaplan, G. A., Tuomilehto, J., & Salonen, J. T. (1997). Workplace conditions, socioeconomic status, and the risk of mortality and acute myocardial infarction: The Kuopio Ischemic Heart Disease Risk Factor Study. *American Journal of Public Health, 87*, 617–622.
- Lynch, J. W., Smith, G. D., Kaplan, G. A., & House, J. S. (2000). Income inequality and mortality: Importance to health of individual income, psychosocial environment, or material conditions. *British Medical Journal, 320*, 1200–1204.
- Macintyre, S. (1997). The Black Report and beyond: What are the issues? *Social Science & Medicine, 44*, 723–745.
- Macintyre, S., & Ellaway, A. (2000). Ecological approaches: Rediscovering the role of the physical and social environment. In L. F. Berkman & I. Kawachi (Eds.), *Social epidemiology* (pp. 332–348). New York: Oxford University Press.
- Macintyre, S., Maciver, S., & Solomon, A. (1993). Area, class and health: Should we be focusing on places or people? *Social Policy, 22*, 213–234.
- Magee, W. J., Eaton, W. W., Wittchen, H. U., McGonagle, K. A., & Kessler, R. C. (1996). Agoraphobia, simple phobia, and social phobia in the National Comorbidity Survey. *Archives of General Psychiatry, 53*, 159–168.
- Manuck, S. B. (1994). Cardiovascular reactivity in cardiovascular disease: “Once more unto the breach.” *International Journal of Behavioral Medicine, 1*, 4–31.
- Manuck, S. B., Flory, J. D., McCaffery, J. M., Matthews, K. A., Mann, J. J., & Muldoon, M. F. (1998). Aggression, impulsivity, and central nervous system serotonergic responsivity in a nonpatient sample. *Neuropsychopharmacology, 19*, 287–299.
- Markovitz, J. H., & Matthews, K. A. (1991). Platelets and coronary heart disease: Potential psychophysiological mechanisms. *Psychosomatic Medicine, 53*, 643–668.
- Markovitz, J. H., Matthews, K. A., Kannel, W. B., Cobb, J. L., & D’Agostino, R. B. (1993). Psychological predictors of hypertension in the Framingham Study: Is there tension in hypertension? *Journal of the American Medical Association, 6*, 2439–2443.
- Markovitz, J. H., Matthews, K. A., Wing, R. R., Kuller, L. H., & Meilahn, E. N. (1991). Psychological, biological and health behavior predictors of blood pressure changes in middle-aged women. *Journal of Hypertension, 9*, 399–406.
- Marmot, M. G. (1998). Contribution of psychosocial factors to socioeconomic differences in health. *Milbank Quarterly, 76*, 403–448.
- Marmot, M., Bobak, M., & Smith, D. G. (1995). Explanations for social inequalities in health. In B. Amick III, S. Levine, A. R. Tarov, & D. Walsh (Eds.), *Society and health* (pp. 172–210). New York: Oxford University Press.
- Marmot, M. G., Kogevinas, M., & Elston, M. A. (1987). Social/economic status and disease. *Annual Review of Public Health, 8*, 111–135.
- Marmot, M., Ryff, C. D., Bumpass, L. L., Shipley, M., & Marks, N. F. (1997). Social inequalities in health: Next questions and converging evidence. *Social Science & Medicine, 44*, 901–910.
- Marmot, M., Shipley, M., Brunner, E., & Hemingway, H. (2001). Relative contribution of early life and adult socioeconomic factors to adult morbidity in the Whitehall II Study. *Journal of Epidemiology and Community Health, 55*, 301–307.
- Marmot, M. G., Shipley, M. J., & Rose, G. (1984). Inequalities in death: Specific explanations of a general pattern? *Lancet, 1*, 1003–1006.
- Marmot, M. G., Smith, G. D., Stansfeld, S., Patel, C., North, F., Head, J., et al. (1991). Health inequalities among British civil servants: The Whitehall II Study. *Lancet, 337*, 1387–1393.
- Marmot, M., & Theorell, T. (1988). Social class and cardiovascular disease: The contribution of work. *International Journal of Health Services, 18*, 659–674.
- Martin, R. L., Cloninger, R., Guze, S. B., & Clayton, P. J. (1985). Mortality in a follow-up of 500 psychiatric outpatients. *Archives of General Psychiatry, 42*, 58–66.
- Maruta, T., Hamburg, M. E., Jennings, C. A., Offord, K. P., Colligan, R. C., Frye, R. L., & Malincho, M. (1993). Keeping hostility in perspective: Coronary heart disease and the Hostility Scale on the Minnesota Multiphasic Personality Inventory. *Mayo Clinic Proceedings, 68*, 192–193.
- Matsumoto, Y., Uyama, O., Shimizu, S., Michishita, H., Mori, R., Owada, T., & Sugita, M. (1993). Do anger and aggression affect carotid atherosclerosis? *Stroke, 24*, 983–986.
- Matthews, K. A. (1989). Are sociodemographic variables markers for psychological determinants of health? *Health Psychology, 8*, 641–648.
- Matthews, K. A., Flory, J. D., Muldoon, M. F., & Manuck, S. B. (2000). Does socioeconomic status relate to central serotonergic responsivity in healthy adults? *Psychosomatic Medicine, 62*, 231–237.
- Matthews, K. A., Kelsey, S. F., Meilahn, E. N., Kuller, L. H., & Wing, R. R. (1989). Educational attainment and behavioral and biologic risk factors for coronary heart disease in middle-aged women. *American Journal of Epidemiology, 129*, 1132–1144.
- Matthews, K. A., Kiefe, C. I., Lewis, C. E., Liu, K., Sidney, S., & Yunis, C. (2001). Socioeconomic trajectories in a biracial cohort of young adults: Risk for incident hypertension. *Psychosomatic Medicine, 63*, 110.
- Matthews, K. A., Owens, J. F., Kuller, L. H., Sutton-Tyrrell, K., Jansen-McWilliams, L. (1998). Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? *Psychosomatic Medicine, 60*, 633–638.
- Matthews, K. A., Rääkkönen, K., Everson, S. A., Flory, J. D., Marco, C. A., Owens, J. F., & Lloyd, C. E. (2000). Do the daily experiences of healthy

- men and women vary according to occupational prestige and work strain? *Psychosomatic Medicine*, 62, 346–353.
- McCranie, E. W., Watkins, L. O., Brandsma, J. M., & Sisson, B. D. (1986). Hostility, coronary heart disease (CHD) incidence, and total mortality: Lack of association in a 25-year follow-up study of 478 physicians. *Journal of Behavioral Medicine*, 9, 119–125.
- McDonough, P., Duncan, G. J., Williams, D., & House, J. (1997). Income dynamics and adult mortality in the United States, 1972–1989. *American Journal of Public Health*, 87, 1476–1483.
- McEwan, B. S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine*, 153, 1093–1101.
- McLeod, J. D., & Kessler, R. C. (1990). Socioeconomic status differences in vulnerability to undesirable life events. *Journal of Health and Social Behavior*, 31, 162–172.
- Mendes de Leon, C. F., Kop, W. J., de Swart, H. B., Bar, F. W., & Appels, A. P. (1996). Psychosocial characteristics and recurrent events after percutaneous transluminal coronary angioplasty. *American Journal of Cardiology*, 77, 252–255.
- Miech, R. A., Caspi, A., Moffitt, T. E., Wright, B. R. E., & Silva, P. A. (1999). Low socioeconomic status and mental disorders: A longitudinal study of selection and causation during young adulthood. *American Journal of Sociology*, 104, 1096–1131.
- Miller, T. Q., Marksides, K. S., Chiriboga, D. A., & Ray, L. A. (1995). A test of the psychosocial vulnerability and health behavior models of hostility: Results from an 11-year follow-up study of Mexican Americans. *Psychosomatic Medicine*, 57, 572–581.
- Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin*, 119, 322–348.
- Mirowsky, J., Ross, C. E., & Willigen, M. V. (1996). Instrumentalism in the land of opportunity: Socioeconomic causes and emotional consequences. *Social Psychology Quarterly*, 59, 322–337.
- Mittleman, M. A., Maclure, M., Nachnani, M., Sherwood, J. B., & Muller, J. E. (1997). Educational attainment, anger, and the risk of triggering myocardial infarction onset. The determinants of myocardial infarction onset study investigators. *Archives of Internal Medicine*, 157, 69–75.
- Mittleman, M. A., Maclure, M., Sherwood, J. B., Mulry, R. P., Tofler, G. H., Jacobs, S. C., et al. (1995). Triggering of acute myocardial infarction onset by episodes of anger. *Circulation*, 92, 1720–1725.
- Möller, J., Hallqvist, J., Diderichsen, F., Theorell, T., Reuterwall, C., & Ahlbom, A. (1999). Do episodes of anger trigger myocardial infarction? A case-crossover analysis in the Stockholm Heart Epidemiology Program (SHEEP). *Psychosomatic Medicine*, 61, 842–849.
- Moos, R. H., Cronkite, R. C., & Moos, B. S. (1998). The long-term interplay between family and extrafamily resources and depression. *Journal of Family Psychology*, 12, 326–434.
- Moser, D. K., & Dracup, K. (1996). Is anxiety early after myocardial infarction associated with subsequent ischemic and arrhythmic events? *Psychosomatic Medicine*, 58, 395–401.
- Muldoon, M. F., Sved, A. F., Flory, J. D., Perel, J. M., Matthews, K. A., & Manuck, S. B. (1998). Inverse relationship between fenfluramine-induced prolactin release and blood pressure in humans. *Hypertension*, 32, 972–975.
- Murphy, J. M., Monson, R. R., Olivier, D. C., Sobol, A. M., & Leighton, A. H. (1987). Affective disorders and mortality: A general population study. *Archives of General Psychiatry*, 44, 473–480.
- Murphy, J. M., Olivier, D. C., Monson, R. R., Sobol, A. M., Federman, E. B., & Leighton, A. H. (1991). Depression and anxiety in relation to social status. *Archives of General Psychiatry*, 48, 223–229.
- Murrell, S. A., & Norris, F. H. (1991). Differential social support and life change as contributors to the social class-distress relationship in older adults. *Psychology and Aging*, 6, 223–231.
- Musselman, D. L., Evans, D. L., & Nemeroff, C. B. (1998). The relationship of depression to cardiovascular disease. *Archives of General Psychiatry*, 55, 580–592.
- Myers, J. K., Lindenthal, J. L., & Pepper, M. P. (1974). Social class, life events, and psychiatric symptoms: A longitudinal study. In B. S. Dohrenwend & B. P. Dohrenwend (Eds.), *Stressful life events: Their nature and effects* (pp. 191–206). New York: Wiley.
- Newton, T. L., & Kiecolt-Glaser, J. K. (1995). Hostility and marital quality during early marriage. *Journal of Behavioral Medicine*, 18, 601–619.
- Oakley, A., & Rajan, L. (1991). Social class and social support: The same or different? *Sociology*, 25, 31–59.
- Oliver, M. L., & Shapiro, T. M. (1995). *Black wealth/White wealth*. New York: Routledge.
- Oxenkrug, G. F., Brannonier, R. J., Harto-Truax, N., & Cole, J. O. (1983). Is serum cholesterol a biological marker for major depressive disorder? *American Journal of Psychiatry*, 140, 920–921.
- Pappas, G., Queen, S., Hadden, W., & Fisher, G. (1993). The increasing disparity in mortality between socioeconomic groups in the U.S., 1960–1986. *New England Journal of Medicine*, 329, 103–109.
- Paterniti, S., Alperovitch, A., Ducimetière, P., Dealberto, M. J., Lépine, J. P., & Bissler, J. C. (1999). Anxiety but not depression is associated with elevated blood pressure in a community group of French elderly. *Psychosomatic Medicine*, 61, 77–83.
- Paykel, E. S. (1994). Life events, social support, and depression. *Acta Psychiatrica Scandinavica*, 879(Suppl. 377), 50–58.
- Pearlin, L. I., Lieberman, M. A., Menaghan, E. G., & Mullan, J. T. (1981). The stress process. *Journal of Health and Social Behavior*, 18, 2–21.
- Penninx, B. W. J. H., Beekman, A. T. F., Honig, A., Deeg, D. J. H., Schoevers, R. A., van Eijk, T. M., & van Tilburg, W. (2001). Depression and cardiac mortality: Results from a community-based longitudinal study. *Archives of General Psychiatry*, 58, 221–227.
- Phillimore, P., Beattie, A., & Townsend, P. (1994). Widening inequality of health in Northern England, 1981–1991. *British Medical Journal*, 308, 1125–1128.
- Pincus, T., Callahan, L. F., & Burkhauser, R. V. (1987). Most chronic diseases are reported more frequently by individuals with fewer than 12 years of education in the 18–64 United States population. *Journal of Chronic Diseases*, 40, 865–874.
- Plutchik, R. (1980). *Emotion: A psychoevolutionary synthesis*. New York: Harper & Row.
- Pohl, R., Yeragani, R., Balon, R., Lycaki, H., & McBride, R. (1992). Smoking in patients with panic disorder. *Psychiatry Research*, 43, 253–262.
- Power, C., & Hertzman, C. (1997). Social and biological pathways linking early life and adult disease. *British Medical Bulletin*, 53, 210–221.
- Power, C., Manor, O., & Matthews, S. (1999). The duration and timing of exposure: Effects of socioeconomic environment on adult health. *American Journal of Public Health*, 89, 1059–1065.
- Pratt, L. A., Ford, D. E., Crum, R. M., Armenian, H. K., Gallo, J. J., & Eaton, W. W. (1996). Depression, psychotropic medication, and risk of myocardial infarction: Prospective data from the Baltimore ECA follow-up. *Circulation*, 94, 3123–3129.
- Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 3, 385–401.
- Rahkonen, O., Lahelma, E., & Huuhka, M. (1997). Past or present? Childhood living conditions and current socioeconomic status as determinants of adult health. *Social Science & Medicine*, 44, 327–336.
- Räikkönen, K., Matthews, K. A., Kuller, L. H., Reiber, C., & Bunker, C. H. (1999). Anger, hostility, and visceral adipose tissue in healthy postmenopausal women. *Metabolism*, 48, 1146–1151.
- Ranchor, A. V., Bouma, J., & Sanderman, R. (1996). Vulnerability and social class: Differential patterns of personality and social support over the social classes. *Personality and Individual Differences*, 20, 229–237.
- Regier, D. A., Farmer, M. E., Rao, D. S., Myers, J. K., Kramer, M., Robins,

- L. N., et al. (1993). One-month prevalence of mental disorders in the United States and sociodemographic characteristics: The Epidemiologic Catchment Area study. *Acta Psychiatrica Scandinavica*, *88*, 35–47.
- Reich, J. W., Zautra, A. J., & Potter, P. T. (2001). Cognitive structure and the independence of positive and negative affect. *Journal of Social & Clinical Psychology*, *20*, 99–115.
- Repetti, R. L., Taylor, S. E., & Seeman, T. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, *128*, 330–366.
- Revenson, T. A. (1990). All other things are not equal: An ecological approach to personality and disease. In H. S. Freidman (Ed.), *Personality and disease* (pp. 65–96). New York: Wiley.
- Richman, C. L., Clark, M. L., & Brown, K. P. (1985). General and specific self-esteem in late adolescent students: Race \times Gender \times SES effects. *Adolescence*, *20*, 555–556.
- Rini, C. K., Dunkel-Schetter, C., Wadhwa, P. D., & Sandman, C. A. (1999). Psychological adaptation and birth outcomes: The role of personal resources, stress, and sociocultural context in pregnancy. *Health Psychology*, *18*, 333–345.
- Ritsher, J. E. B., Warner, V., Johnson, J. G., & Dohrenwend, B. P. (2001). Inter-generational study of social class and depression: A test of social causation and social selection models. *British Journal of Psychiatry*, *178*, S84–S90.
- Robert, S. A. (1998). Community-level socioeconomic status effects on adult health. *Journal of Health and Social Behavior*, *39*, 18–37.
- Robert, S. A., & House, J. S. (2000). Socioeconomic inequalities in health: Integrating individual-, community-, and societal-level theory and research. In G. L. Albrecht & R. Fitzpatrick (Eds.), *The handbook of social studies in health and medicine* (pp. 115–135). London: Sage.
- Robertson, J. F., & Simons, R. L. (1989). Family factors, self-esteem, and adolescent depression. *Journal of Marriage and the Family*, *51*, 125–138.
- Robins, L. N., Helzer, J. E., Croughan, J. L., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry*, *38*, 381–389.
- Robins, L. N., & Regier, D. A. (1991). *Psychiatric disorders in America*. New York: Free Press.
- Rodin, J. (1990). Control by any other name: Definitions, concepts, and processes. In J. Rodin, C. Schoer, & K. W. Schaie (Eds.), *Self-directedness: Cause and effects throughout the life course* (pp. 1–17). Hillsdale, NJ: Erlbaum.
- Roschelle, A. R. (1997). *No more kin. Exploring race, class, and gender in family networks*. Thousand Oaks, CA: Sage.
- Ross, C. E., & Jang, S. J. (2000). Neighborhood disorder, fear, and mistrust: The buffering role of social ties with neighbors. *American Journal of Community Psychology*, *28*, 401–420.
- Ross, C. E., & Mirowsky, J. (1989). Explaining the social patterns of depression: Control and problem solving—or support and talking? *Journal of Health and Social Behavior*, *30*, 206–219.
- Ross, M. W., & Hafner, R. J. (1990). A comparison of the factor structure of the Crown-Crisp Experiential Index across sex and psychiatric status. *Personality and Individual Differences*, *11*, 733–739.
- Rovner, B. W., German, P. S., Brant, L. J., Clark, R., Burton, L., & Folstein, M. F. (1991). Depression and mortality in nursing homes. *Journal of the American Medical Association*, *265*, 993–996.
- Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*, *99*, 2192–2217.
- Ruberman, W., Weinblatt, E., Goldberg, J. D., & Chaudhary, B. S. (1984). Psychosocial influences on mortality after myocardial infarction. *New England Journal of Medicine*, *311*, 552–559.
- Russell, J. A. (1997). How shall an emotion be called? In R. Plutchik & H. Conte (Eds.), *Circumplex models of personality and emotion* (pp. 205–220). Washington, DC: American Psychological Association.
- Russell, J. A., & Carroll, J. M. (1999). On the bipolarity of positive and negative affect. *Psychological Bulletin*, *125*, 3–30.
- Salokangas, R. K. R., & Putanen, O. (1998). Risk factors for depression in primary care findings of the TADEP project. *Journal of Affective Disorders*, *48*, 171–180.
- Salonen, J. T. (1982). Socioeconomic status and risk of cancer, cerebral stroke, and death due to coronary heart disease and any disease: A longitudinal study in eastern Finland. *Journal of Epidemiology and Community Health*, *36*, 294–297.
- Santor, D. A., & Coyne, J. C. (2001). Evaluating the continuity of symptomatology between depressed and nondepressed individuals. *Journal of Abnormal Psychology*, *110*, 216–225.
- Schachter, S., Silverstein, B., Kozlowski, E. T., Herman, L. P., & Liebling, B. (1977). Effects of stress on cigarette smoking and urinary pH. *Journal of Experimental Psychology: General*, *106*, 24–30.
- Scherwitz, L., Perkins, L., Chesney, M., & Hughes, G. (1991). Cook-Medley Hostility Scale and subsets: Relationship to demographic and psychosocial characteristics in young adults in the CARDIA study. *Psychosomatic Medicine*, *53*, 36–49.
- Seeman, M., & Lewis, S. (1995). Powerlessness, health and mortality: A longitudinal study of older men and mature women. *Social Science & Medicine*, *41*, 517–525.
- Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist*, *55*, 5–14.
- Shekelle, R. B., Gale, M., Ostfeld, A. M., & Paul, O. (1983). Hostility, risk of coronary heart disease, and mortality. *Psychosomatic Medicine*, *45*, 109–114.
- Siegler, I. C. (1994). Hostility and risk: Demographic and lifestyle variables. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility, and the heart* (pp. 199–214). Hillsdale, NJ: Erlbaum.
- Simonsick, E. M., Wallace, R. B., Blazer, D. G., & Berkman, L. F. (1995). Depressive symptomatology and hypertension-associated morbidity and mortality in older adults. *Psychosomatic Medicine*, *57*, 427–435.
- Smith, G. D., Carroll, D., Rankin, S., & Rowan, D. (1992). Socioeconomic differentials in mortality: Evidence from Glasgow graveyards. *British Medical Journal*, *305*, 1554–1557.
- Smith, J. P. (1999). Healthy bodies and thick wallets: The dual relation between health and economic status. *Journal of Economic Perspectives*, *13*, 145–166.
- Smith, T. W. (1994). Concepts and methods in the study of anger, hostility, and health. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility and the heart* (pp. 23–42). Hillsdale, NJ: Erlbaum.
- Smith, T. W. (1995). Assessment and modification of coronary-prone behavior: A transactional view of the person in social context. In A. J. Goreczny (Ed.), *Handbook of health and rehabilitation psychology* (pp. 197–217). New York: Plenum Press.
- Smith, T. W., & Frohm, K. D. (1985). What's so unhealthy about hostility? Construct validity and psychosocial correlates of the Cook and Medley Ho Scale. *Health Psychology*, *4*, 503–520.
- Smith, T. W., & Gallo, L. C. (2001). Personality traits as risk factors for physical illness. In A. Baum, T. Revenson, & J. Singer (Eds.), *The handbook of health psychology* (pp. 139–174). Hillsdale, NJ: Erlbaum.
- Smith, T. W., Ruiz, J. M., & Uchino, B. N. (2001). Mental activation of supportive ties reduces blood pressure reactivity to stress [Abstract]. *Psychosomatic Medicine*, *63*, 114.
- Smith, T. W., Sanders, J. D., & Alexander, J. F. (1990). What does the Cook and Medley Hostility Scale Measure? Affect, behavior, and attributions in the marital context. *Journal of Personality and Social Psychology*, *58*, 699–708.
- Sokolovsky, J., & Cohen, C. I. (1981). Measuring social interaction of the urban elderly: A methodological synthesis. *International Journal of Aging and Human Development*, *13*, 233–244.

- Soobader, M. J., & LeClere, F. B. (1999). Aggregation and the measurement of income inequality: Effects on morbidity. *Social Science & Medicine*, *48*, 733–744.
- Soomon, A., & Macintyre, S. (1995). Health and perceptions of the local environment in socially contrasting neighborhoods in Glasgow. *Health & Place*, *1*, 15–26.
- Stack, C. (1974). *All our kin: Strategies for survival in a Black community*. New York: Harper & Row.
- Stansfeld, S. A., Head, J., & Marmot, M. G. (1998). Explaining social class differences in depression and well-being. *Social Psychiatry and Psychiatric Epidemiology*, *33*, 1–9.
- Stansfeld, S. A., North, F., White, I., & Marmot, M. G. (1995). Work characteristics and psychiatric disorder in civil servants in London. *Journal of Epidemiology and Community Health*, *49*, 124–130.
- Steele, R. E. (1978). Relationship of race, sex, social class, and social mobility to depression in normal adults. *Journal of Social Psychology*, *104*, 37–47.
- Stevens, D. E., Merikangas, K. R., & Merikangas, J. R. (1995). Comorbidity of depression and other medical conditions. In E. E. Beckham & W. R. Leber (Eds.), *Handbook of depression* (2nd ed., pp. 147–199). New York: Guilford Press.
- Stone, A. A., Cox, D. S., Valdimarsdottir, H., & Jandorf, L. (1987). Evidence that secretory IgA antibody is associated with daily mood. *Journal of Personality and Social Psychology*, *52*, 988–993.
- Stone, A. A., Neale, J. M., Cox, D. S., & Napoli, A. (1994). Daily events are associated with secretory immune response to an oral antigen in men. *Health Psychology*, *13*, 440–446.
- Straus, M. (1980). Social stress and marital violence in a national sample of American families. In F. Wright, C. Bahn, & W. Rieber (Eds.), *Annals of the New York Academy of Sciences: Vol. 347. Forensic psychology and psychiatry* (pp. 229–250). New York: New York Academy of Sciences.
- Strickland, B. R. (1978). Internal–external expectancies and health-related behaviors. *Journal of Consulting and Clinical Psychology*, *46*, 1192–1211.
- Taylor, S. E., Repetti, R. L., & Seeman, T. (1997). What is an unhealthy environment and how does it get under the skin? *Annual Review of Psychology*, *48*, 411–447.
- Taylor, S. E., & Seeman, T. (1999). Psychosocial resources and the SES–health relationship. In N. E. Adler, M. Marmot, B. S. McEwen, & J. Stewart (Eds.), *Annals of the New York Academy of Sciences: Vol. 896. Socioeconomic status and health in industrialized nations* (pp. 210–225). New York: New York Academy of Sciences.
- Thoits, P. A. (1982). Life stress, social support, and psychological vulnerability: Epidemiological considerations. *Journal of Community Psychology*, *10*, 341–362.
- Thoits, P. A. (1984). Explaining distributions of psychological vulnerability: Lack of social support in the face of life stress. *Social Forces*, *63*, 453–481.
- Thoits, P. A. (1995). Stress, coping, and social support processes: Where are we? What next? [Special issue]. *Journal of Health and Social Behavior*, *36*, 53–79.
- Tigges, L. M., Browne, I., & Green, G. P. (1998). Social isolation of the urban poor: Race, class, and neighborhood effects on social resources. *Sociological Quarterly*, *39*, 53–77.
- Troutt, D. D. (1993). *The thin red line: How the poor still pay more*. San Francisco: Consumers Union of United States, West Coast Regional Office.
- Turner, R. J., Lloyd, D. A., & Roszell, P. (1999). Personal resources and the social distribution of depression. *American Journal of Community Psychology*, *27*, 643–672.
- Turner, R. J., & Marino, F. (1994). Social support and social structure: A descriptive epidemiology. *Journal of Health and Social Behavior*, *35*, 193–212.
- Turner, R. J., & Noh, S. (1983). Class and psychological vulnerability among women: The significance of social support and personal control. *Journal of Health and Social Behavior*, *24*, 2–15.
- Twenge, J. M., & Campbell, W. K. (2002). Self-esteem and socioeconomic status: A meta-analytic review. *Personality and Social Psychology Review*, *6*, 59–71.
- Tzeng, J. M., & Mare, R. D. (1995). Labor market and socioeconomic effects on marital stability. *Social Science Research*, *24*, 329–351.
- U.S. Bureau of the Census. (1996). *Current population reports (Series P-60): Income, poverty, and valuation of non-cash benefits: 1993* (Publication No. P-60-188). Washington, DC: U.S. Government Printing Office.
- Van de Mheen, H., Stronks, K., Looman, C. W. N., & Mackenbach, J. P. (1998). Does childhood socioeconomic status influence health through behavioural factors? *International Journal of Epidemiology*, *27*, 431–437.
- Van Kanel, R., Mills, P. J., Fainman, C., & Dimsdale, J. E. (2001). Effects of psychological stress and psychiatric disorders on blood coagulation and fibrinolysis: A biobehavioral pathway to coronary artery disease. *Psychosomatic Medicine*, *63*, 531–544.
- Vogt, T., Pope, C., Mullooly, J., & Hollis, J. (1994). Mental health status as a predictor of morbidity and mortality: A 15-year follow-up of members of a health maintenance organization. *American Journal of Public Health*, *84*, 227–231.
- Warheit, G. J., Holzer, C. E., & Arey, S. A. (1975). Race and mental illness: An epidemiologic update. *Journal of Health and Social Behavior*, *16*, 243–256.
- Warheit, G. J., Holzer, C. E., III, & Schwab, J. J. (1973). An analysis of social class and racial differences in depressive symptomatology: A community study. *Journal of Health and Social Behavior*, *14*, 291–299.
- Warren, B. S., & Sydenstricker, E. (1916). Health of garment workers: The relation of economic status to health. *Public Health Report*, *31*, 1298–1305.
- Wasserthal-Smoller, S., Applegate, W. B., Berge, K., Chang, C. J., Davis, B., Grimm, R., et al. (1996). Changes in depression as a precursor of cardiovascular events. *Archives of Internal Medicine*, *156*, 553–561.
- Watson, D., & Clark, L. A. (1997). Measurement and mismeasurement of mood: Recurrent and emergent issues. *Journal of Personality Assessment*, *68*, 267–296.
- Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review*, *96*, 234–254.
- Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin*, *98*, 219–235.
- Weidner, G., Sexton, G., McLellarn, R., Connor, S. L., & Matarazzo, J. D. (1987). The role of Type A behavior and hostility in an elevation of plasma lipids in adult women and men. *Psychosomatic Medicine*, *49*, 136–145.
- Weisse, C. S. (1992). Depression and immunocompetence: A review of the literature. *Psychological Bulletin*, *111*, 475–489.
- Weissman, M. M., Bruce, M. L., Leaf, P. J., Florio, L. P., & Holzer, C. (1991). Affective disorders. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric disorders in America* (pp. 53–80). New York: Free Press.
- Weissman, M. M., Markowitz, J. S., Ouellette, R., Greenwald, S., & Kahn, J. P. (1990). Panic disorder and cardiovascular/cerebrovascular problems: Results from a community survey. *American Journal of Psychiatry*, *147*, 1504–1508.
- Weissman, M. M., & Myers, J. K. (1978). Affective disorders in a U.S. urban community: The use of research diagnostic criteria in an epidemiological survey. *Archives of General Psychiatry*, *35*, 1304–1311.
- Wells, J. C., Tien, A. Y., Garrison, R., & Eaton, W. W. (1994). Risk factors for the incidence of social phobia as determined by the Diagnostic Interview Schedule in a population-based study. *Acta Psychiatrica Scandinavica*, *90*, 84–90.

Wells, K. B., Golding, J. M., & Burnam, M. A. (1989). Chronic medical conditions in a sample of the general population with anxiety, affective, and substance use disorders. *American Journal of Psychiatry*, *146*, 1440-1446.

West, C. G., Reed, D., & Gildengorin, G. (1998). Can money buy happiness? Depressive symptoms in an affluent older population. *American Geriatrics Society*, *46*, 49-57.

Wheeler, E. O., White, P. D., Reed, E. W., & Cohen, M. E. (1950). Neurocirculatory asthenia (anxiety neurosis, effort syndrome, neurasthenia): A twenty-year follow-up study of one hundred and seventy-three patients. *Journal of the American Medical Association*, *142*, 878-889.

Whiteman, M. C., Dreary, I. J., & Fowkes, G. R. (2000). Personality and social predictors of atherosclerotic progression: Edinburgh Artery Study. *Psychosomatic Medicine*, *62*, 703-714.

Wilkinson, R. G. (1986). Socioeconomic differentials in mortality: Interpreting the data on their size and trends. In R. G. Wilkinson (Ed.), *Class and health* (pp. 1-20). London: Tavistock.

Williams, D. R. (1990). Socioeconomic differentials in health: A review and redirection. *Social Psychology Quarterly*, *53*, 81-99.

Williams, D. R. (1999). Race, socioeconomic status, and health: The added effects of racism and discrimination. In N. E. Adler, M. Marmot, B. S. McEwen, & J. Stewart (Eds.), *Annals of the New York Academy of Sciences: Vol. 896. Socioeconomic status and health in industrialized nations* (pp.173-188). New York: New York Academy of Sciences.

Williams, D. R., & Collins, C. (1995). U.S. socioeconomic and racial differences in health: Patterns and explanations. *Annual Review of Sociology*, *21*, 349-386.

Williams, J. E., Nieto, F. J., Sanford, C. P., Couper, D. J., & Tyroler, H. A.

(2002). The association between trait anger and incident stroke risk: The Atherosclerosis Risk in Communities (ARIC) Study. *Stroke*, *33*, 13-20.

Williams, J. E., Paton, C. C., Siegler, I. C., Eigenbrodt, M. L., Nieto, F. J., & Tyroler, H. A. (2000). Anger proneness predicts coronary heart disease risk: Prospective analysis from the Atherosclerosis Risk in Communities (ARIC) study. *Circulation*, *101*, 2034-2039.

Wilson, K. C. M., Chen, R., Taylor, S., McCracken, C. F. M., & Copeland, J. R. M. (1999). Socio-economic deprivation and the prevalence and prediction of depression in older community residents: The MRC-ALPHA study. *British Journal of Psychiatry*, *175*, 549-553.

Wittchen, H. U., Zhao, S., Kessler, R. C., & Eaton, W. W. (1994). *DSM-III-R* generalized anxiety disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, *51*, 355-364.

Wulsin, L. R., Vaillant, G. E., & Wells, V. E. (1999). A systematic review of the mortality of depression. *Psychosomatic Medicine*, *61*, 6-17.

Wyatt, R. J., & Clark, K. (1987). Calculating the cost of schizophrenia. *Psychiatric Annals*, *17*, 586-592.

Yen, I. H., & Kaplan, G. A. (1999). Neighborhood social environment and risk of death: Multilevel evidence from the Alameda County Study. *American Journal of Epidemiology*, *149*, 898-907.

Zautra, A. J., Potter, P. T., & Reich, J. W. (1997). The independence of affects is context-dependent: An integrative model of the relationship between positive and negative affect. In M. P. Lawton & K. W. Schaie (Eds.), *Annual Review of Gerontology and Geriatrics*, *17*, 75-102.

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