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Socioeconomic Status and Coronary Heart Disease Risk: The Role of Social Cognitive Factors

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Abstract

The aim of this study is to examine existing research on social cognitive factors that may, in part, mediate the relationship between socioeconomic status (SES) and coronary heart disease (CHD). We focus on how social status is ‘carried’ in the mental systems of individuals, and how these systems differentially affect CHD risk and associated behaviors. To this end, literatures documenting the association of various social cognitive factors (e.g., social comparison, perceived discrimination, and self-efficacy) with cardiovascular disease are reviewed as are literatures regarding the relationship of these factors to SES. Possible mechanisms through which social cognitions may affect health are addressed. In addition, directions for future research are discussed, and a model identifying the possible associations between social cognitive factors, SES, and coronary disease is provided.

Introduction

There is a well-established relationship between socioeconomic status (SES) and health in industrialized nations, with individuals lower in SES experiencing higher rates of morbidity and mortality than higher-SES individuals. This relationship is particularly evident in the case of coronary heart disease (CHD), the progressive narrowing of the coronary vessels manifested by angina pectoris (chest pain), myocardial infarction (MI), and coronary death (AHA, 2009). Despite the overall decline in CHD mortality over the past 30 years, CHD is the leading cause of death in the United States (CDC, 2009). Recent advancements in cardiovascular health have primarily benefitted wealthier, better-educated individuals, while progress among those of lower socioeconomic standing continues to lag (Cooper, Cutler, Desvigne-Nickens, et al., 2000; NHLBI, 1995). Variation in SES demonstrates a consistent, inverse relationship with CHD morbidity (Diez-Roux, Nieto, Tyroler, Crum, & Szklo, 1995; Rose & Marmot, 1981) and mortality (Keil, Sutherland, Knapp, & Tyroler, 1992; Seigel et al., 1987) and with CHD risk factors such as cigarette smoking (Zang & Wynder, 1998), obesity (Sobal & Stunkard, 1989) and physical inactivity (Evenson et al., 2002).

Despite this robust association, the underlying mechanisms by which social inequalities impair cardiovascular health are not well understood. These associations span multiple levels of SES and are not explained by poverty alone, illiteracy, or lack of health care resources (Adler et al., 1994), findings which have prompted researchers to identify other possible mechanisms for the SES–CHD gradient. Examinations of individual psychological attributes in relation to social health disparities have focused primarily on the influence of negative emotions (Gallo & Matthews, 2003).

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Here, we attempt to broaden the discussion of SES influences on health by suggesting a causal role for social psychological factors in the relationship between SES and CHD (see Figure 1) – particularly those related to self-perception and social perception. For example, living and working in lower SES environments may contribute to diminished self-esteem, lower sense of control, and a reduced orientation toward mastery and efficacy (Amato & Zuo, 1992; Baum, Garofalo, & Yali, 1999; McLoyd, 1998; Pearlin, Menaghan, Lieberman, & Mullan, 1981). Lower childhood SES has been associated with development of a pessimistic explanatory style (Finkelstein, Kubzansky, Capitman, & Goodman, 2007), suggesting that cognitive patterns linked to social origins may be carried throughout the life course. Accumulating evidence suggests that the influence of social standing upon these processes may impact behavioral and physiological mechanisms that, in turn, could affect cardiovascular health (Lynch, Davey Smith, Kaplan, & House, 2000; Taylor, Repetti, & Seeman, 1997).

Social Psychological Factors: Terminology

The field of social psychology has long been interested in how people perceive themselves, other individuals, and their social environments. Relevant terms include self-perception, social perception, social cognition, and interpersonal perception. For heuristic use, we refer here to ‘social cognitions’ as the constellation of beliefs and perceptions people hold for and about themselves and the world around them (e.g., self-efficacy, attitudes, and stereotype threat) as well as about other people in particular (e.g., norm perception, social comparison). In this study, we consider how these perceptions are related to SES and coronary disease, and more importantly, whether social cognitions could operate as mechanisms through which lower SES increases the CHD risk; i.e., whether they might *mediate* this association (Baron & Kenny, 1986). The factors of interest in this study are grouped according to conceptual similarity. As outlined by Moskowitz’s (2005) ‘interaction sequence’, when individuals’ negotiate the social environment, they do so through a prism of beliefs and perceptions regarding their reasons for achieving certain goals (*motivations*), a sense of *efficacy* in regard to navigating the environment, and an interpretation of the *social interactions* they encounter. In this study, we have adapted this ‘grouping’ of social cognitions to examine how individuals’ might negotiate the health-related sphere of their environment; specifically, how one’s motivations, one’s sense of efficacy, and one’s interpretation of social relationships might influence cardiovascular health.

Thus, the categories used throughout the article are as follows: Motivational factors are reasons to undergo improved health practices or forgo detrimental ones and include *attitudes* and *health beliefs* regarding a particular health behavior, as well as *intentions* to perform the behavior. (These factors are related to volitional behavior and, by definition, are addressed only within the ‘Behaviors’ section of the manuscript.) Human Agency factors refer to a sense of capacity to influence the environment, either in relation to a particular health behavior or in reference to negotiation of the broader social environment, and include *perceived control*, *self-efficacy*, and *attribution*. Further, social interactions and the ensuing interpretations of these interactions can serve both as facilitators and impediments of health practices and of psychological processes that may affect health, and the salient Interpersonal Factors examined here include *social norms*, *perceived discrimination*, *stereotype threat*, and *social comparison*.

Organization of the Article

In the following sections, evidence relating social cognitions to (i) coronary disease outcomes, (ii) biological risk markers, and (iii) CHD-related risk behaviors is reviewed. In each section, evidence linking SES to the specific social cognitive factors of interest in that

section (Columns A and B, Figure 1) is presented. Next, studies relating those factors to coronary disease outcomes, biological risk markers, or behaviors are discussed (Columns B and C/D, Figure 1).

Social Cognitions and Coronary Disease Outcomes

Substantial evidence links socioeconomic position with coronary morbidity and mortality (Diez-Roux et al., 1995; Keil et al., 1992; Rose & Marmot, 1981; Seigel et al., 1987). This section reviews research linking social cognitions with specific coronary disease outcomes (i.e., angina, MI, and coronary mortality), a body of work which has focused primarily on human agency factors. The theoretical basis for, and evidence supporting, a relation between SES and human agency is discussed first. Next, the evidence documenting positive associations between agentic factors and CHD is reviewed. Finally, the three studies that have examined SES, social cognitions, and CHD outcomes within a single model are presented and are summarized in Table 1.

Self-efficacy and control

Marxist theories of social class and alienated labor have focused on how the nature of work affects self-efficacy (Gecas, 1989), suggesting that prolonged lack of control is inherent to lower social status (Mirowsky & Ross, 1983). Education (Aalto et al., 2007; Cohen, Kaplan, & Salonen, 1999), income (Cohen et al., 1999; Lachman & Weaver, 1998), and occupation (Bosma, Schrijvers, & Mackenbach, 1999; Chandola, Kuper, Singh-Manoux, Bartley, & Marmot, 2004; Marmot, Bosma, Hemingway, Brunner, & Stansfield, 1997) are related to mastery and personal control as are measures combining socioeconomic indices (Bosma et al., 1999, 2005; Marmot et al., 1997). SES has generally shown positive relations with self-efficacy (Ribisl, Winkleby, Fortmann, & Flora, 1998; Winkleby, Flora, & Kraemer, 1994) and self-esteem (Twenge & Campbell, 2002). According to the 'job strain' model of psychosocial work characteristics (Karasek, 1979), coronary risk is heightened by the distress resulting from an imbalance between workers' low control and high conflicting task demands. Lack of occupational control has been consistently associated with CHD risk (see reviews by Hemingway & Marmot, 1998 and Schnall & Landsbergis, 1994), and higher control outside the workplace has been linked to lower incidence of CHD (Bosma et al., 2005).

In contrast, having a stronger belief in personal control has also been related to greater coronary atherosclerosis in adults undergoing angiography (Seeman, 1991). This finding may reflect participants' unrealistic expectations of control (Taylor & Seeman, 1999) or hostile tendencies, which are associated with increased coronary risk (Barefoot, Dahlstrom, & Williams, 1983; Dembroski, MacDougall, Costa, & Grandits, 1989). The locus of control (LOC) construct has yielded inconsistent results when examining recovery from coronary events. In one investigation, patients recovering from acute MI who were classified as having an internal control locus demonstrated better prognoses than those reporting external control perceptions (Cromwell, Butterfield, Brayfield, & Curry, 1977), while Flowers (1994) found no such association for cardiac patients.

Attributions

Attributional or explanatory style (Abramson, Seligman, & Teasdale, 1978) adds to the LOC concept of internality–externality by including the dimensions of stability–instability and globality–specificity (i.e. whether attributions are maintained across time/in varied situations). Considering the imbalance between resources and demands in low-SES environments, pessimistic attributions (tendencies toward expecting negative outcomes for future events) may be activated more frequently and with greater magnitude in lower-SES

individuals (Grewen et al., 2000). Kohn and Schooler (1982) report positive associations between SES and optimism, while Taylor and Seeman (1999) show negative outcome expectations (pessimism) to relate to low SES. Low occupational status is correlated with 'fatalistic' causal perceptions (i.e., emphasis of environmental rather than personal causation of behavior) (Wheaton, 1980). Optimism covaries positively with educational attainment (Giltay, Geleijnse, Zitman, Hoekstra, & Schouten, 2004; Radcliffe & Klein, 2002), whereas pessimistic explanatory style is inversely related to education (Kubzansky, Sparrow, Vokonas, & Kawachi, 2001).

In regard to CHD, Kubzansky et al. (2001) demonstrated that veterans' optimistic explanatory style predicted lower risk of angina, nonfatal MI, and fatal CHD. In two separate studies of older Dutch persons, optimism was found to have a protective effect against CHD mortality (Giltay, Kamphuis, Kalmijn, Zitman, & Kromhout, 2006; Giltay et al., 2004).

Mediating social cognitions

The three studies investigating social cognitions as mediators between SES and CHD outcomes have focused on perceived control (Table 1). In one analysis of 5-year CHD incidence from the Whitehall II study of British civil servants, Marmot et al. (1997) examined the contribution of control perceptions to disease risk. Compared with those in the highest occupational grade, those in the lowest grade had significantly higher age-adjusted odds ratio of developing new CHD, and adjustment for perceived control provided the largest contribution to reducing the odds ratios for newly reported CHD in the lowest grade. Results from a second Whitehall II study indicated that low control at home predicted CHD among women, but not among men. Furthermore, adjustment for control accounted for a significant portion of the relation between household social position and women's CHD risk (Chandola et al., 2004). In a third, population-based prospective investigation, incident heart disease (acute MI) at 5 years was recorded in middle- and older-aged participants without baseline heart disease. As expected, low SES predicted higher risk of CHD (Bosma et al., 2005), and adjustment for control beliefs and coronary risk factors (e.g. smoking, hypertension) showed control to account for substantially greater variance in coronary risk (30%) than did traditional risk factors (4%).

In sum, human agency factors such as self-efficacy, control beliefs, and attributional style have been related both to coronary disease outcomes and to indices of socioeconomic position. Further, perceived control appears to play a mediational role in the relation between SES and CHD. Conceivably, coronary risk in lower SES individuals may be heightened, in part, by the inherent distress of having relatively little control over demanding social and economic environments.

Social Cognitions and Biological Risk Markers

In addition to coronary disease morbidity and mortality, several 'preclinical' indicators of CHD have been related to socioeconomic position. Exaggerated cardiovascular reactivity, defined as physiologic responses to a psychological or physical stressor (Manuck, Kasprovicz, Monroe, Larkin, & Kaplan, 1989), has been implicated in the development of hypertension (Treiber et al., 2003), and research demonstrates inverse relationships between SES and both blood pressure (Sorel, Ragland, Syme, & Davis, 1992; Vargas, Ingram, & Gillum, 2000) and cardiovascular reactivity (Gump, Matthews, & Räikkönen, 1999; Lynch, Everson, Kaplan, Salonen, & Salonen, 1998). Significant inverse relations between SES and triglycerides (Brunner et al., 1997), plasma glucose concentrations (Brunner et al., 1997; Ko et al., 2001), and the metabolic syndrome (Brunner et al., 1997; Lawlor, Ebrahim, & Davey Smith, 2002; Park et al., 2003) have also been documented. As described previously (see

'CHD Outcomes' section), evidence supports an association between social standing and self-efficacy. Similarly, SES has been associated with interpersonal social cognitive factors which, in turn, are related to several biological markers of coronary risk. These relationships are discussed later, as are the three studies that have examined SES, social cognitions, and preclinical disease markers within a single model (summarized in Table 2).

Self-efficacy

Cardiovascular reactivity is inversely related to subjects' task-specific self-efficacy (Bandura, Reese, & Adams, 1982). Autonomic nervous system (ANS) activation plays an influential role in the pathophysiology of cardiovascular disease (Corti et al., 2000), and catecholamine reactivity (a measure of ANS activation) has been inversely related to performance self-efficacy on stressful tasks (Bandura, Taylor, Williams, Mefford, & Barchas, 1985; Gerin, Litt, Deich, & Pickering, 1996; Wright & Dismukes, 1995).

Social comparison

When asked to compare themselves with others in a society, individuals of lower occupational status, less education, and lower incomes rank themselves as lower in social standing (Adler, Epel, Castellazzo, & Ickovics, 2000; Gianaros et al., 2007; Singh-Manoux et al., 2005), and a frequently cited correlate of low SES is the distress or demoralization stemming from this form of 'upward' social comparison (Wilkinson, 1999). Consistent with this, adverse cardiovascular responses, including increased ventricular contractility and vasoconstriction, have been related to engagement in upward social comparison (Mendes, Blascovich, Major, & Seery, 2001). Evidence of socioeconomic variation in health outcomes even among those who are not materially disadvantaged has prompted the examination of *relative* social standing in SES-related variability in disease risk (Marmot & Wilkinson, 1999; Wilkinson, 1999). One measure of perceived social ranking, termed subjective SES, asks individuals to engage in a form of social comparison by indicating their standing in relation to others on a visual 'ladder' of ascending SES (Adler et al., 2000). People reporting lower subjective SES exhibit several physiological risk factors for CHD, including higher central adiposity (Adler et al., 2000), higher BMI (Goodman et al., 2001, 2003), a more pronounced rise in morning cortisol (Wright & Steptoe, 2005), greater adrenocortical responses to stress (Adler et al., 2000), and higher basal heart rate (Adler et al., 2000).

Stereotype threat and perceived discrimination

Steele and colleagues (Steele & Aronson, 1995; Steele, Spencer, & Aronson, 2002) have demonstrated the concept of *stereotype threat*, or the fear that one's behavior will confirm the existing stereotype of one's identified group. Although most research on stereotyping has examined categories based on racial categorization, class-based stereotypes have been documented (Feldman & Hilterman, 1974; Weeks & Lupfer, 2004). Because students from lower SES families generally perform worse on tests of intellectual ability (Neisser et al., 1996), the concept of stereotype threat has been proposed to contribute to differences in scholastic achievement between high- and low-SES individuals. After inducing stereotype threat for low SES (by citing poor performance in low SES students on standardized tests), Croizet and Claire (1998) found that SES was inversely related to performance when a test was presented as a measure of verbal ability, despite equal performance between groups when no mention of intellectual function was included. Similarly, another sample of lower-income students performed worse than their high-SES counterparts on an academic test when an SES-based stereotype threat was activated (Harrison, Stevens, Monty, & Coakley, 2006).

The activation of stereotype threat may heighten stress in stereotyped group members, bringing about alterations in neurobiological systems. Although no studies have yet

investigated physiological correlates of stereotype threat in reference to SES, African Americans under stereotype threat have been found to exhibit larger blood pressure increases during academic tests (Blascovich, Spencer, Quinn, & Steele, 2001).

Another consequence of social class stereotyping may be the perception of SES-related discrimination. However, despite documented negative beliefs relevant to social standing, such as perceptions of low-SES individuals as being lazy (Leahy, 1981), dishonest (Desmond, Price, & Eoff, 1989), and uninterested in education (Bullock, 1999), few investigations have included a measure of mistreatment on the basis of income level. In one study, low-SES patients reported higher rates of being discriminated against by health care providers, an effect that was independent of subjects' race (Trivedi & Ayanian, 2006). Although several investigations among the US African American population document links between self-reported experiences of discrimination and elevated blood pressure (see review by Brondolo, Rieppi, Kelly, & Gerin, 2003), no studies of class-based discrimination have included a measure of CHD-related risk.

Mediating social cognitions

A small number of investigations have addressed the influence of socioeconomic position on biological markers of coronary disease risk via interpersonal social cognitions (Table 2). Although they did not explicitly examine the mediational role of pessimism in the SES–CHD relationship, Grewen et al. (2000) showed that low-SES/high-pessimism women had significantly higher systolic blood pressure than women in three other groups (high SES/low pessimism, high SES/high pessimism, and low SES/low pessimism), demonstrating that the combination of lower social status and negative expectations regarding future events may enhance CHD risk. More direct evidence comes from two studies that tested cognitive appraisal biases among adolescents of diverse socioeconomic position (Chen, Langer, Raphaelson, & Matthews, 2004; Chen & Matthews, 2001). In these investigations, lower-SES participants displayed greater perceptions of hostile intent and threat appraisals to neutral events, and these cognitions mediated associations between social standing and total peripheral resistance, heart rate, and diastolic blood pressure reactivity in the lower-SES individuals. The authors concluded that, although SES contributed only a small portion of the variance in cardiovascular reactivity, cognitive appraisal biases explained a substantial portion of SES effects.

Altogether, these results suggest that individuals' cognitions regarding their social environment may influence coronary disease risk, perhaps through stress-related physiological responses (see Figure 1). Several social cognitive factors, including self-efficacy, social comparison processes, and perceived discrimination, have been related to measures of social status. Similarly, these factors have been related to various 'preclinical' indicators of CHD, such as higher blood pressure, heart rate, and central adiposity. In addition, preliminary evidence demonstrates a mediational role for cognitions (specifically cognitive appraisal biases) in the association between SES and CHD risk indicators.

Social Cognitions and CHD-related Behaviors

Consistent evidence links physical inactivity, poor diet, and smoking with CHD (American Heart Association, 2009; Wannamethee, Whincup, Shaper, & Walker, 1996), behaviors which have also been associated with social standing. The prevalence of cigarette smoking increases at successively lower levels of SES (Zang & Wynder, 1998), and direct associations have been found between physical activity levels and SES (Ford et al., 1991; Evenson et al., 2002). This section reviews research linking SES, social cognitions, and CHD-related behaviors (see Figure 1). This body of work has focused largely on motivational factors; thus, the theoretical basis for, and evidence supporting, a relation

between SES and motivational factors is discussed first. Next, evidence documenting associations between social cognition and behavior is reviewed. Finally, the nine studies that have examined SES, social cognitions, and CHD-related behavior within a single study are presented and summarized in Table 3.

Motivational factors

Many standard predictors of health behaviors in models like the Theory of Planned Behavior (Ajzen, 1991), which aims to explain and predict a variety of voluntary behaviors, characterize motivations for adopting health behaviors. *Attitudes* (positive or negative evaluations) toward healthy behavior predict consumption of low-fat food (Armitage & Conner, 2001; Paisley & Sparks, 1998), physical activity (Courneya, Friedenreich, Arthur, & Bobick, 1999; Trafimow & Trafimow, 1998), and smoking behavior (Morrison, Gillmore, Simpson, & Wells, 1996; Norman, Conner, & Bell, 1999). Attitudes may be determined, in part, by beliefs regarding the consequences of a behavior, termed *outcome expectancies*. Direct relations between outcome expectancies and exercise behavior have been found in many (though not all) studies (e.g., Dzewaltowski, Noble, & Shaw, 1990; Resnick, Orwig, Magaziner, & Wynne, 2002; Rovniak, Anderson, Winett, & Stephens, 2002). *Beliefs* regarding how lifestyle habits affect health, combined with attitudes toward health behaviors, motivate individuals to implement changes. Behavioral *intentions* indicate the degree to which one has formulated conscious plans to perform a behavior (Warshaw & Davis, 1985). Evidence supports an association between intention and actual performance of physical activity (Hausenblas, Carron, & Mack, 1997) and smoking (Hanson, 1999; O'Callaghan, Callan, & Baglioni, 1999).

Participants with fewer years of education report less favorable attitudes toward engagement in healthy behavior (Winkleby et al., 1994), and education is positively associated with intention to reduce CHD risk (Ribisl et al., 1998). Wardle, Waller, and Rapoport (2001), Wardle and Steptoe (2003) report more positive attitudes toward maintaining a healthy weight in British civil servants of higher occupational grade and found that lower grade predicted less 'health consciousness' and less concern for the future. Manfredi, Lacey, Warnecke, and Buis (1992) examined female smokers' beliefs about the health risks of smoking, and found that low-SES women are less concerned about smoking-related risks (i.e. cancer, heart disease) than higher-SES women.

Human agency

As described previously (see 'CHD Outcomes' section), evidence supports an association between social standing and human agency factors, and a similar relationship has been documented for these factors and specific health behaviors. For example, internal LOC was related to the performance of a higher number of health behaviors in a sample of over 11,000 adults (Norman, Bennett, Smith, & Murphy, 1998) and has been associated with higher physical activity (Sonstroem & Walker, 1973) and a healthier diet (Bennett, Moore, Smith, Murphy, & Smith, 1995). Some evidence suggests that 'internals' are more likely to reduce (Best & Steffy, 1971) or stop smoking (James, Woodruff, & Werner, 1965), whereas other research shows no relationship between LOC and cessation (Brod & Hall, 1984; Carlisle-Frank, 1991). Low self-efficacy ratings predict smoking initiation (Conrad et al., 1992), while high self-efficacy is related to smoking cessation (Barrios & Niehaus, 1985; Carey, Snel, Carey, & Richards, 1989; Coletti, Supnick, & Payne, 1985; Mudde, de Vries, & Dolders, 1995).

Higher self-efficacy is also associated with successful weight loss attempts (Chambliss & Murray, 1979; Weinberg, Hughes, Critelli, England, & Jackson, 1984) weight loss maintenance (Blair, Booth, Lewis, & Wainwright, 1989), and initiation and maintenance of

exercise programs (e.g., Brawley & Rogers, 1993; Dziewaltowski et al., 1990; McAuley & Courneya, 1993; Poag-DuCharme & Brawley, 1993). Higher self-efficacy scores are associated with better self monitoring of blood glucose, better diet, and higher physical activity in patients with diabetes (Sarkar, Fisher, & Schillinger, 2006), and some (though not all) self-efficacy interventions have been shown to enhance diabetes management (Corbett, 1999). The single study relating coronary risk behavior and attributional style showed that internal explanatory style for negative events (i.e. 'self-blame') predicted a lapse in dietary restriction during weight loss attempts (Ogden & Wardle, 1990).

Interpersonal factors

Subjective norms are a subset of beliefs indicative of the desire to comply with social norms, either to avoid rejection and criticism or to receive gratification from others. Social norms are thought to provide a context in which an individual makes health-relevant decisions (Emmons, Barbeau, Gutheil, Stryker, & Stoddard, 2007). Christakis and Fowler's (2007) examination of social influences on obesity found that a person's chances of becoming obese increased significantly if one had an obese friend, sibling, or spouse, regardless of geographic separation. The authors suggest that having obese social contacts may change a person's tolerance for becoming obese and may, in turn, influence his or her adoption of specific consumption or activity patterns. Thus, although the strength of social norms has not been explicitly examined in relation to SES, the accepted behavioral norms of one's home, workplace, or community (i.e., indices of socioeconomic position) will likely influence individual health behavior. In adolescent boys (Trost, Pate, Ward, Saunders, & Riner, 1999) and college students (Okun et al., 2003), ratings of significant others' value of physical activity are positively associated with participants' own activity levels, and seeing physically active community members is related to exercise among African American women (Ainsworth et al., 2003; King et al., 2000). Emmons et al. (2007) found that those whose family and friends ate more fruit and vegetables and exercised daily demonstrated more favorable dietary and physical activity scores.

Perception of the *favorability* of other people's behavior also appears to influence one's own actions. The Prototype/Willingness Model for risk behavior (Gibbons & Gerrard, 1995) assumes that people maintain images of the type of person who engages in a particular behavior, with more favorable images (smokers are rebellious and cool) resulting in increased willingness to engage in the behavior. The model has been effective in predicting willingness to engage in smoking behavior; as participants' ratings of the 'typical smoker' image became more favorable, smoking behavior increased, and vice-versa (Thornton, Gibbons, & Gerrard, 2002). Because smoking is substantially more common in low-SES individuals (Zang & Wynder, 1998), more frequent exposure to smokers within one's social network may play a role in increasing the favorability of smokers' images in low-SES environments.

Social comparisons play an influential role in how people judge their own disease risk (Klein & Weinstein, 1997), and comparisons with 'the average person' have been associated with behavioral intentions (Klein, 2002). Because higher education has been associated with better CHD-related knowledge (Gans, Assmann, Sallar, & Lasater, 1999), higher-SES individuals may be more accurate judges of CHD risk than those of lower-social status. Higher perceived risk has been related to both desire to engage in CHD risk reducing behaviors and actual dietary and smoking behavior change (Silagy, Muir, Coulter, Thorogood, & Roe, 1993; Winkleby et al., 1994). The related phenomenon of unrealistic optimism (optimistic bias) is the belief that one is at less risk for negative health events than are others. Smokers, for instance, report a belief that the risk of smoking-related diseases applies more to other smokers than to themselves (Weinstein, 1998), and optimistically biased individuals exhibit higher rates of smoking (Strecher, Kreuter, & Koblin, 1995). The

one study that examined risk perception in the context of cardiovascular disease found that unrealistically optimistic individuals exhibited higher systolic blood pressure and higher serum cholesterol (Radcliffe & Klein, 2002).

Mediating social cognitions

Despite numerous investigations linking social cognitive factors with health behavior, only nine studies were found which included measures of both socioeconomic position and CHD-related health behaviors along with relevant social cognitions (Table 3). Steptoe and Wardle (1999) examined relations of educational attainment and attitudes toward dietary habits in a cross-sectional population survey. After dividing the sample into low- and high-SES groups, adjustment for attitudes toward food in a logistic regression analysis reduced the odds ratio for having a high-fiber diet to nonsignificance, suggesting that educational influences on diet are largely accounted for by attitudes. Consistent with data showing that people of lower SES, despite generally higher body mass, tend to be more satisfied with their body size than people of higher SES (Lynch et al., 2007; McLaren & Kuh, 2004; Wardle et al., 2001), Wardle and Griffith's (2001) British survey of weight attitudes found that those with higher civil service grades held stronger weight concerns (despite having lower body mass than those in lower social classes) and were more likely to participate in weight control practices. Another survey of health practices in a group of over 6000 Italian men showed that, along with holding more favorable attitudes, the highest SES groups smoked less, reported more physical activity, and attended blood pressure and cholesterol screenings more frequently than those in lower occupational grades (Seccareccia, Menotti, & Prati, 1991). Similarly, Ribisl et al. (1998) showed an inverse association between education and cardiovascular risk scores, while intention to alter risk was directly related to educational attainment.

Using data from the Stanford Five-City project, two studies examined self-efficacy in relation to coronary disease risk behavior scores, though neither directly tested for mediation. In the first, education was positively associated with self-efficacy scores and inversely related to change in CHD risk scores over 6 years (Winkleby et al., 1994). A later cross-sectional survey similarly documented low self-efficacy in those with less education, along with higher coronary risk scores (Ribisl et al., 1998). The one study to test for mediation of a health behavior via human agency factors showed that self-efficacy did not mediate the relation between smokers' educational attainment and cessation attempts (Manfredi, Cho, Crittenden, & Dolecek, 2007). In the two studies exploring interpersonal factors, Manfredi et al. (1992, 2007) theorized that, because smoking is more prevalent in low-SES groups, it may be a valued part of lifestyle among low-SES individuals and that, owing to weaker 'quitting' norms, low-SES women may be less motivated to quit. Results supported the authors' claim in both studies, showing that more highly educated women smokers lived in environments more supportive of quitting and this social pressure to quit mediated the effects of education on cessation.

Overall, substantial evidence demonstrates an association between social status and CHD-related behavioral risk factors such as physical inactivity, poor diet, and smoking. Moreover, these health-impairing practices have been related to social cognitions (primarily motivational factors), although data supporting a mediating role for these factors in the relationship between SES and CHD risk behavior is limited. Further research is needed in this area and may benefit from inclusion of more well-defined health behavioral outcomes, as well as from more standardized measures for motivational factors across studies.

Summary

Overall, the moderate but growing evidence base to date supports a role for social cognitions in the relationship between socioeconomic position and risk of CHD (Figure 1). Fifteen

studies indirectly or directly addressed social cognitions as mediators of the SES-health gradient. Although the majority of studies examining links between both social status and social cognition with CHD and health-relevant behaviors were correlational in nature and did not explicitly test for mediation, those that did utilize more sophisticated analytical techniques demonstrated evidence for a causal role of social cognitive factors, specifically for control and threat perceptions, in the SES-CHD gradient. The fact that independent effects for these cognitive factors were shown after adjusting for behavioral risk factors (Bosma et al., 2005) further strengthens this assertion, given that behavior serves as a potentially key mediator of the SES-CHD relationship.

Despite evidence supporting a role for social cognitive factors in the development of coronary disease, several limitations were evident in this body of research. In the case of motivational factors, intention may be a useful predictor of future health behavior, but because studies often do not measure individuals' intentions close in time to the observed behavior, the changing nature of intentions over time may limit predictive power (Sutton, 1998). Also, considering some individuals' tendencies toward internal control beliefs in some circumstances (e.g., career goals) but externality regarding a target health behavior (e.g., weight management), a unidimensional and generalized scale of perceived control may fail to detect such distinctions (Carlisle-Frank, 1991).

Another potential limitation is the lack of standard self-efficacy measures, and the fact that many investigations employ study-specific self-efficacy questionnaires (Coletti et al., 1985; DiClemente, 1981; DiClemente, Fairhurst, & Piotrowski, 1995). Further, most investigations utilized questionnaires and survey techniques, and the primarily cross-sectional analyses prohibit causal inferences, both for the associated SES and CHD outcomes as well as for cognitive concepts and behavioral and physiological factors (Weinstein, Rothman, & Sutton, 1998). Results support continued exploration of the role of social cognitions in the SES-CHD relationship via prospective study designs, and novel concepts such as stereotype threat and cognitive biases bear much more investigation. Finally, inclusion of more well-defined health outcomes, particularly in the health behavior realm, is encouraged as a parallel to the growing sophistication of physiological measurement techniques (e.g. ambulatory blood pressure monitoring).

Future research on social cognitions that have not yet been applied to SES is encouraged. Possible directions include examinations of relationship perceptions (beliefs about marriage partner, which could be affected by SES and in turn affect behavior) and perceived norms about the value of good health vis-à-vis other goals. More comprehensive assessment of SES, either on an individual (wealth, subjective SES) or community level, is also essential. Also, considering that most investigations were conducted with ethnically and racially limited university populations, future studies should include more heterogeneous samples and should specifically examine gender or racial differences. The association of social status with health appears to be particularly strong among African Americans and women, for whom the burden of discrimination may be more powerful (Adler et al., 1994), underscoring the importance of disentangling the multiple effects of social class, gender, and ethnicity.

Although the aim here has been to present evidence for a causal role of social cognitive factors in the SES-CHD relationship, alternate explanations for the sequential nature of these associations exist. Poorer cardiovascular health may influence social status via vascular impairments in higher-order brain function (Haley et al., 2007), which could, in turn, diminish opportunities for educational and occupational attainment. However, evidence for the 'social drift' hypothesis of the SES-health relationship is relatively scarce (Adler et al., 1994). A second explanation suggests that the link between SES and health represents a spurious association stemming from the relationships of both social status and health

outcomes to underlying genetic vulnerabilities. These genetic factors may similarly influence cognitive processes, resulting in associations among SES, health, and cognitions which appear to be causally linked but are, in reality, unrelated. The most likely explanation for the inverse relationship of SES with cardiovascular risk lies in a complex interaction of genes, environment, and behavior. This is clearly an important area of future research.

If social cognitions serve merely as bridges to emotional factors, these affective experiences may be the 'true' mediators. Hostility, a disposition reflecting anger proneness and a distrustful view of others (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989), is predictive of CHD (Barefoot et al., 1983; Dembroski et al., 1989) and has been inversely related to SES (Barefoot, Peterson, Dahlström, & Williams, 1991; Scherwitz, Perkins, Chesney, & Hughes, 1991). Depression has also been linked with acute coronary events (Rosengren et al., 2004) and has been hypothesized to contribute to variation in SES–health disparities (Anderson & Armstead, 1995; Wilkinson, 1997). However, some evidence suggests an independent role for cognitions in the development of coronary disease. The protective effect of optimism against CHD demonstrated by Kubzansky et al. (2001) was not reduced upon adjustment for anger and hostility. Chen and Matthews (2001) examined both cognitive appraisals and anger as pathways from low SES to higher cardiovascular reactivity and showed an effect of threat appraisal above and beyond anger. It is possible that additional emotional factors that have not been examined, such as anxiety, may account for the observed associations between cognitive factors and disease. More likely, it is the interplay of cognitive and affective dimensions of psychological functioning (Forgas, 2001) which influences physical well-being (Salovey, Detweiler, Steward, & Bedell, 2001).

Interventions

The challenge of addressing socioeconomic disparities in health remains a prominent theme in public health research, and varied approaches for reducing disparities have been proposed. Recent work emphasizes promoting cleaner and safer living environments (Vlahov & Galea, 2002), improving access to quality care for disadvantaged groups (Weech-Maldonado, Dreachslin, & Dansky, 2002), and reducing societal income inequalities (Kaplan, 2000). Relevant to the present review, educational and health promotion approaches utilizing cognitive strategies may be a valuable intervention technique. Gollwitzer and colleagues (Gollwitzer & Brandstätter, 1997; Gollwitzer & Schaal, 1998) suggest that formulating goal-directed plans in a specific context more effectively facilitates the carrying out of intentions, such as going on a healthier diet (Verplanken & Faes, 1999). Investigations into attitude change demonstrate that directed-thinking tasks may increase the probability of engaging in a specific health behavior (McGuire & McGuire, 1996; Ratcliff et al., 1999), and self-efficacy manipulations (Chambless & Murray, 1979) have been shown to assist in losing weight.

Risk-stratification based on socioeconomic position may enable health care professionals to target these interventions where they are needed most. School programs may promote resilience to stressful social events by incorporating components to reduce pessimism and increase optimism (Gillham & Reivich, 1999) or to minimize cognitive biases (Chen & Matthews, 2001) possibly reducing the physiological toll of cardiovascular responses.

Additionally, raising individuals' educational status through cognitive interventions may demonstrate protective health effects. Work on stereotype threat has indicated that disidentification with academic pursuits among stereotyped groups contributes to reinforcement of the SES hierarchy through resulting low academic performance (Major, Spencer, Schmader, Wolf, & Crocker, 1997). Supporting this finding, children with higher perceptions of school-specific control perform better academically than children who attribute school performance to external causes (Bandura, Barbaranelli, Caprara, &

Pastorelli, 1996; Butler & Orion, 1990; Harter, 1992). Perceived control may enhance academic performance via associations with positive mood, curiosity, pride, and persistence (Skinner, 1995).

In sum, myriad factors influence SES differences in coronary disease and associated health behaviors, including the price of healthy foods, access to physical fitness resources, and barriers to medical care. Additionally, social cognitive resources, by influencing how events are perceived, may exacerbate or ameliorate the behavioral and physiological responses to stress that lead to social gradients in health (Taylor & Seeman, 1999). Although the social cognitive factors reviewed here may not fully explain the SES–CHD relationship, evidence supports a potentially key role.

Short Biographies

Jennifer E. Phillips holds an MS in Psychology from the University of Pittsburgh, where she is in the process of completing her dissertation, entitled *Socioeconomic Status, Amygdala Reactivity, and Selective Attention to Threat*. Throughout her graduate career Ms. Phillips' research has focused on examining the associations between socioeconomic factors and cardiovascular disease and its associated risk factors, with particular emphasis on the role of psychological factors in this relationship. She has co-authored papers in this area for *Brain, Behavior, and Immunity*, *Obesity*, and *Psychosomatic Medicine*. As her research interests have evolved over the course of graduate training, she has identified obesity as one primary area of focus. She recently collaborated on a chapter on disordered eating for the *International Handbook of Behavior, Diet, and Nutrition*.

Dr. William M. P. Klein is Associate Director of the Behavioral Research Program in the Division of Cancer Control and Population Sciences at the National Cancer Institute (NCI). He joined NCI from the Psychology Department at the University of Pittsburgh, where he was a member of the faculty in the Social Psychology and the Biological and Health Psychology graduate programs. Dr. Klein completed his PhD in Social Psychology at Princeton University in 1991. His research interests fall largely under the areas of self-judgment, risk perception, and risk communication. For example, he has been interested in how risk perception biases are related to the processing of health communications, to health decision-making, and to health behavior; how social processes (e.g., social comparison, self-affirmation, peer influence, self-evaluation, self-presentation) influence responses to personalized feedback and risk communication; the influence of affective factors such as worry on risk appraisal and health decisions; the impact of ambiguity on responses to feedback and risk messages; the role of optimism in health behavior and psychological functioning; and applications of theory to risk communication and health behavior intervention. Dr. Klein's work has been supported by the NCI, National Science Foundation, and several private foundations.

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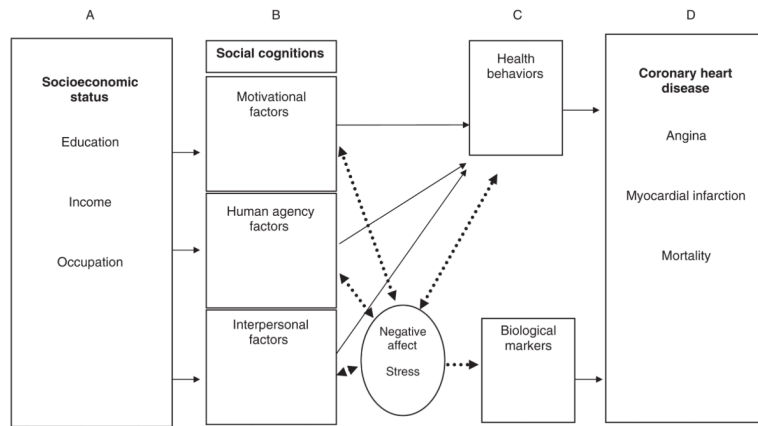


Figure 1. Model of social cognitive pathways through which socioeconomic status influences coronary heart disease.

Table 1
 Studies examining social cognitive variables in relation to socioeconomic status (SES) and coronary heart disease (CHD) outcomes

| SC variable | Author/date | Subjects | SES outcome | CHD outcome | Results |
|---|---------------------------|--|---|-----------------------------|---|
| <i>Human agency</i> | | | | | |
| Control Pearlin and Schooler's mastery scale and measure of 'general' self-efficacy (When trying to learn new things I usually give up if I'm not initially successful) | Bosma et al. (2005) | 3888 older adults from GLAS study (Netherlands) Prospective (5 year f/u) cohort study | SES index of combined education, occupation, and income | Acute MI | HRs for MI in medium/low SES groups decreased by 4% when contr. for classic risk fx. 30% for low general control beliefs (SES effects on AMI lost significance) |
| Control | Marmot et al. (1997) | Whitehall II cohort | Civil service grade | Adult mortality | Higher childhood SES = higher levels of perceived control in adulthood, perceived control sig. mediated association of SES w/ later mortality |
| Control | Chandola et al. (2004) | Whitehall II phases 3-5, 7470 males and females | Civil service grade 'Household position' = presence of financial problems, difficulty paying bills, etc. | Fatal CHD, non- fatal MI | Low control at home predicted CHD among women but not men. Women - low SES predicted CHD (HR = 2.7). HR significantly reduced after adjusting for control (HR = 2.5) and risk fx |

MI, myocardial infarction.

Table 2
 Studies examining social cognitive variables in relation to socioeconomic status (SES) and coronary heart disease-related biological risk markers

| SC variable | Author/date | Subjects | SES outcome | Biological marker | Results |
|--|--------------------------|--|---|--|--|
| <i>Human agency</i> | | | | | |
| Attributional style measured by ASQ | Grewen et al. (2000) | 37 postmenopausal females (39–64 years) No HRT | Composite of income, educ, and race to create 'social disadvantage' score | Blood pressure | Mean comparisons – low SES/high pessimism women = higher SBP |
| <i>Interpersonal</i> | | | | | |
| Threat perception Controlling for anger did not account sig for SES effects on SBP reactivity | Chen and Matthews (2001) | 201 (50% AA/50%CA) children (8–10 years) and adolescents (15–17 years) Prospective study – lab sessions 3 years apart | Family SES, paternal SES (Hollingshead) (subst maternal SES if no father in home) | CV reactivity (BP, HR, cardiac output, total peripheral resistance) Time 1 CVR controlled for at Time 2 | Low SES assoc. w/higher TPR reactivity and > perceptions of hostile intent. Cog bias mediated relationship in low SES AAs |
| Threat perception | Chen et al. (2004) | 47 AA and 53 CA male and female adolescents Interviews and lab stressors + BP/HR measured (cross sectional) | Family SES, paternal SES (Hollingshead) | CV reactivity (BP, HR) | Lower SES assoc. w/> threat appraisals to neutral events, which mediated associations w/DBP and HR 2 tests of mediation |

Table 3

Studies examining social cognitive variables in relation to socioeconomic status (SES) and coronary heart disease (CHD)-related health behaviors

| SC variable | Author/date | Subjects | SES outcome | Behavior | Results |
|-------------------------------------|----------------------------|---|--------------------------|--|--|
| <i>Motivational</i> | | | | | |
| Attitudes ('important to me') | Steptoe and Wardle (1999) | 374 females, 290 males Postal survey, South London | Education (low vs. high) | Dietary intake | Controlling for dietary attitudes reduced effect of higher education on better dietary habits to non-significance |
| Attitudes | Wardle and Griffith (2001) | 1894 males and female participants Omnibus survey, Great Britain | Civil Service Grade | Weight control practices | Higher SES = more concerned about weight-led to weight control, perhaps leading to lower BMIs |
| Attitudes | Seccareccia et al. (1991) | 6074 Italian males 46 y.o. Postal survey | Occupation | Smoking, physical activity, diet, blood pressure and cholesterol checks | Highest of 3 SES groups held most favorable health attitudes, smoked less, reported most leisure PA, and had BP and cholesterol checks most frequently |
| Intentions ('motivation to change') | Ribisi et al. (1998) | 243 Hispanic, 1786 white males (25-64 years) Stanford Five City Project, cross sectional survey | Education | Framingham risk score (combination of age, cig/day, total cholesterol, SBP, BMI) | Lower Ed = lower change intentions, higher CVD risk Did not test for mediation |
| <i>Human agency</i> | | | | | |
| Self-efficacy | Winkleby et al. (1994) | 221 females/190 males (25-74 years) Stanford Five City Project Baseline + prospective 6 year follow up survey | Education | Framingham risk score (combination of age, cig/day, total cholesterol, SBP, BMI) | Low SES group = lowest self-efficacy scores, lowest CHD change scores – not a mediation model |
| Self-efficacy | Ribisi et al. (1998) | 243 Hispanic, 1786 White males (25-64 years) Stanford Five City Project, cross sectional survey | Education | Framingham risk score (combination of age, cig/day, total cholesterol, SBP, BMI) | Lower Ed = lower Self-efficacy, and higher CVD risk, but did not test for mediation |
| Self-efficacy | Manfredi et al. (2007) | 644 female smokers Path analysis examining mediators of education-smoking behavior | Education | Smoking | Self-efficacy did not directly influence quit attempts in low SES women |
| <i>Interpersonal</i> | | | | | |
| Social norms | Manfredi et al. (1992) | 256 female smokers (18-39 years) Evaluation of a smoking cessation intervention, baseline telephone interviews | Education | Smoking cessation | Higher ed. predictive of living in 'cessation supporting environments' w/fewer smokers |
| Social norms | Manfredi et al. (2007) | 644 female smokers Path analysis examining mediators of education-smoking behavior | Education | Smoking | Social pressure to quit mediated effects of education on attempts to quit |