For Better and Worse: Social Influences on Coronary Heart Disease Risk

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Abstract
From the neighborhoods we live in to those we marry, other people are a part of our everyday lives. Mounting evidence suggests that these social factors constitute risk and resilience influences for coronary heart disease (CHD). The current aim is to summarize the literature into four representative categories relating to CHD risk: social environments, social roles, social resources, and close relationships. The argument is made that these factors moderate and mediate stress and associated physiological responses constituting a psychosomatic pathway to disease.

Introduction
Family, friends, enemies, bosses, coworkers, mates, acquaintances, and strangers – from the neighborhoods we live in to the people we marry, other people are a part of our everyday experience of the world. Mounting evidence suggests that our social lives may also constitute an important source of risk for our health with the strongest evidence supporting a causal link with coronary heart disease (CHD), the leading cause of death in the United States (Rosamond et al., 2007).

In this article, we examine four classes of social factors and how they influence disease. Following the review, we discuss hypothesized psychological and biological pathways mediating the relationship and suggest alternative approaches to understanding how our social lives constitute a source of risk. Integral to this discussion is the role of stress. Strong epidemiological and laboratory evidence supports stress as a causal determinant of CHD. For example, primates exposed to repeated stress in the laboratory develop significantly more atherosclerosis than control animals, independent of diet and other factors (Kaplan et al., 1983; Manuck, Kaplan, Adams, & Clarkson, 1988). Evidence suggests that this relationship is mediated by acute physiological responses to stress such as sympathetic arousal (Manuck, Kaplan, & Clarkson, 1983). In humans, stress is prospectively associated with the greater incidence of CHD, faster disease progression, and more clinical events such as heart attacks and mortality (Krantz & McCeney, 2002). Thus, we argue that social factors
may be related to CHD to the extent that they moderate and mediate stress and disease-relevant physiological responses.

**Is there a Relationship between Social Factors and CHD?**

To what degree do social factors moderate CHD risk? The answer lies in part in which social factors we are referring to. There are innumerable ways by which we encounter other people, which has led to a vibrant proliferation of socially relevant concepts. Rather than conduct an exhaustive review of all these concepts or factors, we have organized the literature into four basic categories representing the ways by which we experience other people: (i) social environments; (ii) social roles; (iii) social resources; and (iv) close relationships. This organizational schema will help facilitate an examination of the larger questions regarding social influences on CHD rather than explore each individual construct. We feel that examining the literature at the broad category level is appropriate for discussing general relationships to health. However, other approaches can and should be used to answer specific questions regarding how these relationships occur.

**Social environments**

Social environments refer to any context in which one is exposed to other people. This exposure begins at birth when we enter into an environment filled with family or surrogates upon whom we rely on for all our physical and social needs. Like all social context, the affiliative quality of this early environment can vary from warm and friendly to cold and hostile. A handful of studies suggest that exposure to stressful, less affiliative environments may increase the chances of developing diseases such as CHD. For example, a study of 17,000 adults found that individuals who reported adverse childhood experiences such as neglect, abuse, or family violence were three times more likely to develop CHD compared to those without such experiences (Dong et al., 2004). Similarly, 87% of college students who rated both of their parents as interpersonally cold were subsequently diagnosed with serious health challenges (e.g., heart disease and hypertension, ulcers) 35 years later (Russek & Schwartz, 1997). In comparison, only 25% of adults who had rated both parents as warm and caring experienced subsequent serious disease.

Like the early family environment, the neighborhood and community one lives in may also influence CHD risk. Socioeconomic status (SES) is an index characterizing the relative social standing of an individual in terms of resources or prestige. The concept can be used to characterize the relative standing of individuals or environments such as neighborhoods and communities. The relationship between lower neighborhood SES and CHD risk is among the most robust findings in social/health research.
Living in a lower SES neighborhood is typically associated with a 2- to 3-fold increased risk for hypertension [i.e., high blood pressure (BP)] and CHD including diagnosis, risk of myocardial infarction (MI: heart attack), and mortality due to cardiovascular causes (Steenland, Hu, & Walker, 2004). This relationship is partially mediated by non-social/structural factors including the availability of healthy food options, exercise options, and safety resources (Kivimaki et al., 2007). However, exposure to social stressors including overcrowding, drug use, and violent crime also increase risk (Sundquist et al., 2006). In contrast, less stressful neighborhoods characterized by a greater sense of trust, positive reciprocity, and communal participation are associated with lower mortality after adjusting for physical environment factors (Lochner, Kawachi, Brennan, & Buka, 2003).

Work environments are also a source of significant social exposure. The Bureau of Labor Statistics finds that most Americans spend between 33.7 and 46 hours at work each week depending on the specific industry of employment. This is roughly equal to the total awake time spent at home. A recent meta-analysis of 14 prospective cohort studies concluded that lower status jobs and higher work-related stress are associated with a 50% excess risk for CHD morbidity and mortality (Kivimaki et al., 2006). How does job stress translate into such dramatic health outcomes? Evidence suggests that jobs characterized by high demand coupled with either high or low control (Job strain model: Karasek, 1979) or with inadequate compensation (Effort-reward imbalance (ERI: Siegrist, 1996) contribute to CHD risk. For example, evidence from the Whitehall II study, a prospective investigation of 10,308 civil servants, supports higher ERI with CHD development and faster disease progression in initially healthy individuals (Bosma, Peter, Siegrist, & Marmot, 1998; Lynch, Krause, Kaplan, Salonen, & Salonen, 1997). In a related examination, perceived unfairness in the workplace was predictive of MI and angina (chest pain) risk after controlling for traditional risk factors (De Vogli, Ferrie, Chandola, Kivimaki, & Marmot, 2007).

In addition to these specific examples, a number of other factors can contribute to the stressful quality of social environment and may constitute sources of CHD risk. For example, institutionalized discrimination or experiences of bias or racism heighten tension and stress (Broudy et al, 2007). Interpersonal situations involving harassment, conflict, or situations where one may feel outnumbered or threatened are also associated with stress. Like stressful work environments or dangerous neighborhoods, chronic exposure to these environments or conditions may also heighten CHD risk.

Social roles

Exposure to stressful environments and circumstances is often a requisite part of our social roles. For example, occupational roles such as law
enforcement and military service require chronic exposure to often threatening and violent social environments. Persons in these roles must not only act in response to specific events but also must be vigilant for potential threats. Does this chronic exposure to social stress increase CHD risk? A study comparing retired male law enforcement officers and age-matched controls found that law enforcement officers had a higher incidence of heart disease as well as higher incidence of contributing risk factors including high cholesterol, diabetes, and tobacco use (Franke, Collins, & Hinz, 1998). Ancillary analyses found that perceived stress and time in the profession, controlling for age were significant predictors, suggesting that stress and duration of exposure contribute to CVD risk (Franke, Ramey, & Shelley, 2002).

Similar to law enforcement, military combat service involves chronic threat of exposure to violence and stressful interpersonal circumstances. Posttraumatic stress disorder, a clinical diagnosis characterized by persistent intrusive thoughts and re-experiencing of a traumatic event, is common among combat veterans. In a recent investigation, Kubzansky et al. (2007) presented the first prospective evidence that combat-related PTSD predicted CHD development in an initially healthy sample of men with military service. Higher levels of PTSD symptoms were associated with a 26% increased risk of angina, MI, and CHD-related mortality over the 11-year follow-up period. Two prior studies also found that combat veterans with PTSD had a higher incidence of CHD and early mortality (Boscarino, 2006). Together, these data suggest social roles requiring exposure to stressful social environments increase the risk of CHD development as well as associated morbidity and mortality.

Social resources

In contrast to the risk associated with exposure to stressful environments, individual financial and social assets are important resources that may buffer the effects of stressful experiences and reduce associated CHD risk. Individual SES is a measure of individual assets including one’s education level, job status, and income. Higher individual SES may allow a person to live in a safer, more secure environment, may reflect a greater financial buffer against economic difficulties, and may afford greater opportunity for safer, more prestigious jobs and work environments. In their 1993 review, Kaplan and Keil assert that ‘during 40 years of study there has been a consistent inverse relation between cardiovascular disease, primarily coronary heart disease, and many of the indicators of individual SES. Evidence for this relation has been derived from prevalence, prospective and retrospective cohort studies.’ Additional data since the time of that review continue to support this graded relationship between lower SES and greater CHD risk (Lantz et al., 1998; Stamler et al., 1999).

In addition to financial resources, other people can be a form of social capital useful for preventing or reducing stressful experiences. Collectively
referred to as social support, it is measured in a variety of ways including the size and quality of one’s social network and the degree of social integration (Uchino, 2004). One creative study retrospectively examined the link between social ties and mortality by reviewing the social content of autobiographies written by now deceased authors (Pressman & Cohen, 2007). Those whose autobiographies contained the highest social content lived on average 6 years longer than those in the lowest tertile. Like SES, this relationship between greater social support and health-benefits, including reduced CHD risk, is among the most robust findings in the behavioral–health literature. In particular, lower levels of support following a heart attack are associated with a greater risk of subsequent cardiac morbidity and mortality (Berkman, Leo-Summers, & Horwitz, 1992; Burg et al., 2005), whereas higher levels of support are predictive of survival (Farmer et al., 1996). Importantly, the source of support need not be human, as pets seem to provide a similar benefit (Friedmann & Thomas, 1995).

Close relationships

Personal relationships such as family relations, friendships, and romantic attachments are associated with numerous health benefits including lower all-cause morbidity and mortality as well as greater longevity. Among the most important adult relationships is marriage. Marriage seems to provide a cardio-protective effect against CHD. For example, married individuals experience lower CHD incidence rates, slower disease progression when it does develop, fewer MIs, lower rehospitalization rates following an MI, and are more likely to survive 5 years following an MI than unmarried persons. However, like most relationships, marriage is a double-edged sword of social influence – a source of enjoyment and aid that can also bring stress and frustration (Ruiz, Hamann, Coyne, & Compare, 2006). Thus, it is not surprising that whereas positive marriages are associated with health benefits, individuals in more discordant marriages are at greater risk of cardiovascular morbidity and early mortality (Eaker, Sullivan, Kelly-Hayes, D’Agostino, & Benjamin, 2007; Matthews & Gump, 2002).

Although married adults experience less CHD than unmarried persons, the relative health effects differ for men and women (Kiecolt-Glaser & Newton, 2001). Unmarried men are at significantly greater risk of disease morbidity and mortality compared to unmarried women. Yet, within the context of marriage, women may be at greater risk. A recent study found that amongst happily married adults, more frequent interactions between spouses was associated with reduced risk of atherosclerosis in men but an increased risk in women (Janicki, Kamarck, Shiffman, Sutton–Tyrrell & Gwaltney, 2005). Although it is unclear as to why more interactions were associated with greater risk in women, this finding is consistent with the suggestion that marital status (married or not) is more important for men.
and marital quality is more important for women (Kiecolt-Glaser & Newton, 2001). What is the impact of marital quality? A study of 292 women with pre-existing heart disease found that marital stress was associated with a nearly 3-fold increased risk for subsequent cardiac events (Orth-Gomer et al., 2000).

It is important to note that beyond social support, there is relatively little research on the potential downside to friends and family. This lack of research may bias understanding of such relationships as they pertain to cardiac health. As Uchino (2004) points out, social networks are not homogenously positive. Most of us have friends who are predominantly friendly, supportive, and put little strain on us. There are others whose calls we avoid and with whom we try and create as much distance as possible because we simply find them aversive. We also probably know some people who have both positive and negative qualities such as your mother who loves and supports you yet expresses her disapproval of your romantic partner, career choice, or lifestyle. Thus, an emerging challenge in the area of close relationships is to follow the direction of marital research and move beyond questions of status to examine the effects of relationship quality as it pertains to CHD risk.

**How Does Social Stress Translate to CHD Risk?**

One way by which social factors can influence CHD risk is through the pathway of stress and associated biological mechanisms. Figure 1 represents

![Figure 1](image-url)
one approach to conceptualizing the relationships between social factors, stress, and disease risk. The clearest path is between environments or circumstances and stress where the frequency or chronicity of exposure is proportional to risk (Pathway A). On the other hand, relationships exert direct effects on stress although these effects can be harmful or positive, depending on the quality of the experience (Pathway B). As discussed, social roles primarily influence stress indirectly through exposure to environments (Pathway C). For example, being a police officer may not be inherently stressful. However, a police officer who patrols (is exposed to) a violent neighborhood is more likely to experience stress. Social resources are unique in that they can mediate as well as moderate stress. Having more resources reduces the chances of exposure to stressful circumstances or the magnitude of threat associated with certain environments (i.e., direct effects hypothesis: Pathway D). Persons with more wealth can afford to live in safer neighborhoods, having more education is a pathway to more job opportunities, and having a larger social network reduces the threat of interpersonal violence. Second, social resources may reduce the impact of stressful circumstances (i.e., stress buffering hypothesis: Pathway E). After a stressful car accident a friend may help to calm you with a hug, with money, or with a ride home. Finally, stress may be a function of fit between individual differences in behavioral preference and social environment/social condition characteristics (Christenfeld, Glynn, Kulik, & Gerin, 1998; Pathway F). For example, an introverted person may be comfortable in a quiet social setting such as a library or coffee shop but experience increased stress in loud or raucous social environments such as a concert or sporting event.

**Biological Mediators**

There are two general classes of biological mechanisms linking stress to CHD risk: Reactivity responses and markers. Although the relationship between these mechanisms and CHD is primarily speculative, they are the leading candidate risk factors hypothesized to link psychological and social factors to CHD. Reactivity typically refers to acute physiological changes in response to psychological stress. Cardiovascular reactivity (CVR) includes almost instantaneous changes in BP, heart rate (HR), and the autonomic nervous system. The frequency, magnitude, and duration of these responses are hypothesized to contribute to wear-and-tear on the body leading to disease (Manuck, 1994). Although chronic stress is assumed to cause prolonged activation of the cardiovascular system, these physiological changes are not bound to the duration of the acute event. Salient events such as an argument with a loved one, an incident of discrimination, or traumatic experiences such as assault may evoke repeated reactions through intrusive thoughts and rumination long after the event (Glynn, Christenfeld, & Gerin, 2002). In addition, vigilant monitoring for potential threats is
associated with heightened vascular reactivity independent of any specific event (Smith, Ruiz, & Uchino, 2000).

The second class of biological mechanisms are so-called ‘risk markers’ whose status is often interpreted as a sign of disease. Perhaps the hottest topic (no pun intended) in current psychosomatic research is the causal role of inflammation in CHD. In response to injury or infection, the body mounts an inflammatory response mediated by pro-inflammatory cytokines (Miller & Blackwell, 2006). Although these cytokines are typically associated with immune system activity directed toward overcoming infection and promoting healing, they also stimulate the development of atherogenic plaques underlying CHD. Consequently, the presence of inflammation, typically assessed by the presence of C-reactive protein (CRP), is interpreted as a risk marker for CHD.

Individual differences in patterns of CVR may also be interpreted as a risk marker. Evidence suggests that some individuals are so-called ‘hot reactors’ meaning that they experience larger and longer lasting CVR to stress, possibly potentiating risk. Reactivity may also be a marker of disease. For example, persons with hypertension or with extensive atherosclerosis may experience larger acute changes in BP and slower recovery times based on disease severity.

Are Social Factors Associated with These Biological Pathways?

The association between stress and CVR is well supported in non-human, primate models and human laboratory and field research (Kamarck & Lovallo, 2003). The past several years have also seen a maturing of the data supporting chronic stress as a moderator of pro-inflammatory states (Segerstrom & Miller, 2004). However, application to social factors has been slower in coming. For example, there is little mechanism data linking early social environment challenges with biological mechanisms. A few studies have shown that adults raised in low affiliative environments exhibit exaggerated cardiovascular responses to stress (Luecken, 1998; Luecken, Rodriguez, & Appelhans, 2005), and one recent study found these individuals are more likely to have elevated levels of CRP (Taylor, Lehman, Kiefe, & Seeman, 2006). Childhood individual and neighborhood-level SES are also predictive of subsequent inflammation, independent of adulthood SES (Miller & Chen, 2007; Turrell, Lynch, Leite, Raghunathan, & Kaplan, 2007), although findings are mixed regarding which is more important. In contrast, the relationship between stressful work environments and larger, more frequent elevations in BP during the work day is well supported (Pickering, 1997). However, there is little data examining its association with inflammation (Clays et al., 2005).

Interestingly, there are almost no published studies linking any major social roles (caregiving, law enforcement, military service) with inflammation.
or CVR. However, our model suggests that we should expect such links through the pathway of exposure to stressful environments. Social resources, particularly individual SES and social support, are strongly associated with both reactivity and markers. Recent evidence links individual SES in adulthood to elevated daily BP (McGrath, Matthews, & Brady, 2006) and heightened inflammatory markers (Hong, Nelesen, Krohn, Mills, & Dimsdale, 2006), suggesting a general association between SES and chronic activation of psychobiological pathways hypothesized to influence CHD risk. Several reviews and meta-analyses also support lower social support with heightened cardiovascular, neuroendocrine, and immunological functioning consistent with psychobiological risk models of CHD (Seeman, Berkman, Blazer, & Rowe, 1994; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). In addition, at least two recent studies extend these physiological risk markers to inflammation variables (Marsland, Sathanoori, Muldoon, & Manuck, 2007; Steptoe, Owen, Kunz-Ebrecht, & Brydon, 2004).

Evidence also suggests that the relationship between marital quality and CHD risk is underscored by alterations in hypothesized psychobiological mechanisms. For example, Barnett et al. (2005) found that people with more marital concerns reported more daily stress and experience higher daily BP than individuals who are more satisfied with their marriage. Similarly, couples who are more hostile during laboratory interaction studies exhibit larger and longer lasting CVR as well as greater immune suppression than couples who were less abrasive in their interactions (Kiecolt-Glaser et al., 2005; Malarkey, Kiecolt-Glaser, Pearl, & Glaser, 1994). Suppressed immune functioning opens the door to greater damage to the internal environment and may set the stage for more pronounced inflammation. In summary, the data suggest that although marital status confers a general health benefit, marital strain can increase CHD risk, in part through stress and associated alterations in underlying endocrine and immune functioning. However, the relationship between health outcomes and these mechanisms is hypothetical due to the current lack of any longitudinal data showing a prospective relationship.

Summary

Current evidence supports a relationship between a number of social factors and CHD morbidity and mortality. Although some specific factors are clearly more established than others, the four broad categories reviewed each have ample support to suggest some level of influence on CHD. Notably, social factors are not universally associated with risk. Rather, there is some evidence to suggest that social resources not only reduce risk but may promote resilience. Moreover, close relationships such as marriage can have both positive and negative consequences for CHD. Progress is also being made with regard to establishing links between social factors, stress, and the biological mechanisms proposed in our conceptual
model. Here, we offered one possible conception of how social factors influence CHD. We suggest that these broad categories moderate and or mediate experiences of stress resulting in a cascade of biological alterations. However, more work needs to be done to relate the various categories of social factors with these hypothesized biological mechanisms in order to establish the models overall utility.

**Looking Forward**

Our approach in this article was to focus on the broad categories of social risk factors. The literature would also be well informed by examinations using other levels of analyses to address questions of interpersonal and individual aspects of social functioning. One possibility is to examine the interpersonal underpinnings of social constructs. From social environments to social roles to relationships, all social factors involve interactions with other people. In some cases, these interpersonal interactions are friendly and warm and in others they are cold and potentially hostile. The interpersonal approach is an emerging viewpoint in psychosocial health research (see Ruiz et al., 2006 for comprehensive review). A key aspect of the approach is to characterize constructs in terms of their interpersonal attributes. This approach has been useful for integrating and comparing research on health-relevant personality traits (Ruiz, Smith, & Rhodewalt, 2001; Smith Ruiz, Uchino, 2004). It also provides a process model and posits that personality or social conditions reflect patterns of social interactions and that these interactions evoke physiological responses hypothesized to contribute to CHD risk. We would hypothesize that all social environments, social roles, social resources, and relationships can be compared in terms of their interpersonal qualities. For example, a high crime, low SES neighborhood would reflect cold/hostile interpersonal interactions, whereas high social support would reflect warm/friendly interactions. By identifying and organizing the social factors literature in terms of social behaviors, we may find that it is not the individual factor but exposure to specific social characteristics that represent the real risk.

It is also important to acknowledge that while labeling some factors as inherently stressful will account for a significant proportion of variance in risk there will be exceptions. For example, why do some people react one way to certain social conditions and others react in a totally different way? The answer may lie in the interaction or degree of match between the person and the environment. Christensen (2000) has used this approach to examine differences in response to dialysis treatment. Renal failure patients who characteristically prefer to engage in active coping fare poorly when given a passive treatment such as clinic dialysis (a person × situation mismatch) but thrive when given a more active, patient-involved treatment such as home dialysis where they perform their own treatment (match). In contrast, people who prefer a more passive
coping style do better with the clinic dialysis (match) and more poorly with the active, patient-involved treatments (mismatch). Similarly, mismatches in several contexts including between people with different behavioral preferences, participant sex and gender characteristics of jobs, and behavioral preferences and social constraints are shown to evoke greater CVR (c/f., Christenfeld et al., 1998, Smith & Ruiz 2007).

In closing, the current review finds that social factors are generally associated with CHD risk. Stress and associated physiological response is one pathway that may underlie this relationship, although it is likely that other pathways also exist independently or in concert with those highlighted here. Moreover, other conceptual perspectives may be useful for explicating our findings and helping to answer questions regarding how our social lives constitute both risk and resilience influences for CHD.

Short Biographies

John M. Ruiz is currently Assistant Professor of Clinical Psychology at Washington State University where he runs a cardiovascular behavioral medicine laboratory. His program of research focuses on understanding the health effects of the person in the social environment. He is particularly interested in the relationship between personality, social behaviors, and coronary heart disease (CHD) risk and the mechanisms by which these relationships may occur. Dr. Ruiz’s recent works include the development and validation of the Transitive Model of interpersonal risk and studies of the acute cardiovascular effects of social vigilance. He has authored or co-authored papers in several peer-reviewed publications including the *Journal of Personality, Journal of Personality and Social Psychology, Journal of Consulting and Clinical Psychology*, and *Health Psychology*. Dr. Ruiz completed his PhD in clinical psychology at the University of Utah under the guidance of Dr. Timothy Smith, his clinical internship at Western Psychiatric Institute and Clinics and a 2-year post-doctoral fellowship in cardiovascular behavioral medicine with Dr. Karen Matthews at the University of Pittsburgh School of Medicine.

James G. Hutchinson is currently a doctoral student in clinical psychology at Washington State University. His primary interests concern the relationship between higher order personality factors and health. He is particularly interested in the role of neuroticism in perceptual bias towards threat and its effects on acute cardiovascular responses. James has accepted an internship position at London Health Sciences Centre in Ontario and expects to graduate in 2009.

Alexandra L. Terrill is a second year doctoral student in clinical psychology at Washington State University. Her primary interests are in trait positive affect as a potential health resilience factor. Alex’s master’s thesis examines trait positive affect serves a stress buffering role for social and/or non-social challenges.
Endnotes
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