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# The Workplace and Cardiovascular Disease: Relevance and Potential Role for Occupational Health Psychology

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Cardiovascular disease (CVD) is the major cause of morbidity and mortality in the industrialized world. It is projected that CVD will become the most common cause of death worldwide by 2020, causing more than 36% of all deaths (Braunwald, 1997, p. 1364). However, based primarily on studies of nonindustrialized populations (Carvalho et al., 1989; Cooper, Rotimi, & Ward, 1999), CVD and hypertension appear to be of relatively recent historical origin (Schnall & Kern, 1981). A major cross-cultural study found virtually no rise in blood pressure (BP) with age and no hypertension among hunter–gatherers, herders, or traditional family farmers (Waldron et al., 1982). In contrast, men and women in urban industrial societies have steady rises of BP with age and hypertension is common (Schnall & Kern, 1981; Waldron et al., 1982). This study also found substantial ($r = 0.46–0.67$) and significant associations between BP and involvement in a money economy even after controlling for salt consumption and, for men, after controlling for body mass index (Waldron et al., 1982).

A number of individual risk factors for CVD have been identified, including cigarette smoking, total (and low density lipoprotein) cholesterol, hypertension, fibrinogen, overweight, diabetes, and sedentary behavior (Kannel, 1992). However, many cases do not occur in "high-risk" individuals (Whelton, He, & Klag, 1994)—for example, in the multiple risk factor intervention trial, more than 30% of the CHD deaths resulting from systolic

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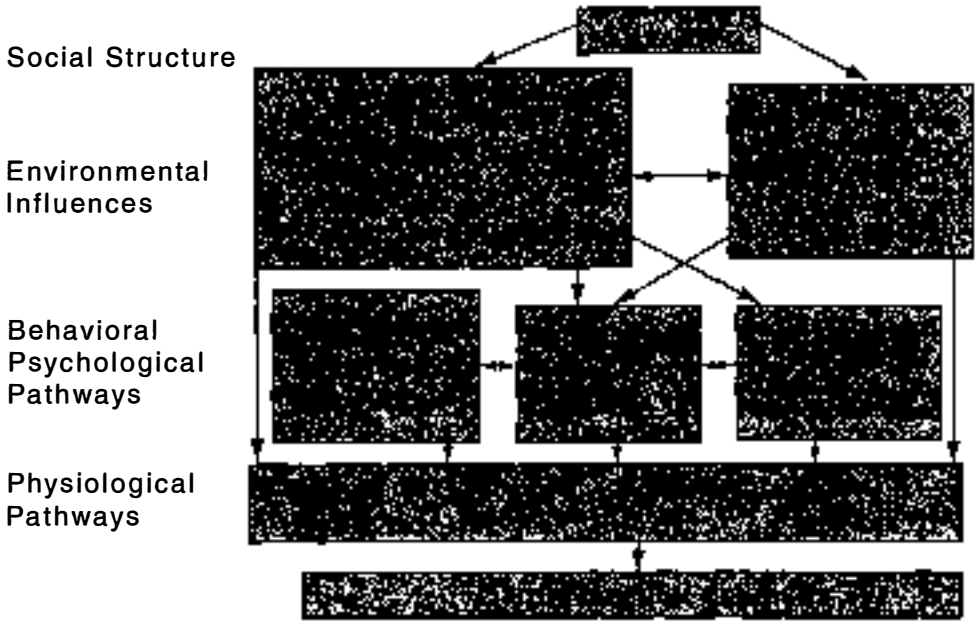


**Figure 13.1.** Levels of causation of CVD. Adapted with permission from McKinlay and Marceau (1999).

BP > 110 mm Hg were in the group with "normal" systolic BP (110–139 mm Hg; Stamler, 1991).

Over the past 20 years, an extensive body of evidence has documented that lower socioeconomic status (Kaplan & Keil, 1993) and work stressors (Karasek & Theorell, 1990; Schnall, Belkić, Landsbergis, & Baker, 2000) are important risk factors for hypertension and CVD. Job control was "the biggest factor contributing to the socioeconomic gradient in CHD risk across civil service employment grade" in the Whitehall study (Kawachi & Marmot, 1998; Marmot, Bosma, Hemingway, Brunner, & Stansfeld, 1997). A model of the impact of social structure and environment on behavior, psychological characteristics, physiology, and CVD is depicted in Figure 13.1. For example, sedentary behavior and smoking "often arise in the context of individuals trapped in low-control work environments" (Kawachi & Marmot, 1998, p. 162). More advantaged communities and individuals have greater resources for the promotion of "hygienic" life-styles and the "reduction of alienating living and working conditions which are conducive to initiating and maintaining unhealthful behaviors" (Wing, Casper, Riggan, Hayes, & Tyroler, 1988, p. 925).

## *Current Economic and Workplace Trends*

Current economic and workplace trends, resulting from economic globalization, may be having dramatic effects on the health of lower socioeconomic groups (Cooper, 1996; Tuchsen, 1993). These trends include stagnant or

falling real income, downsizing, contingent and temporary work, and new management systems such as "lean production" that intensify work (Landsbergis, Cahill, & Schnall, 1999). Income inequality in the United States is at the highest level over the past 60 years (Wolff, 1995), and the United States "is the most unequal among developed countries" (U.S. Departments of Labor and Commerce, 1994, p. 19). Concurrently, substantial changes in job characteristics have been occurring. In Europe, "time constraints" (i.e., workload demands) increased between 1977 to 1996 (European Foundation, 1997), and in the United States, increases between 1977 to 1997 were reported for "never enough time to get everything done on my job" (from 40 to 60%) (Bond, Galinsky, & Swanberg, 1998). U.S. workers' average weekly work hours are now the longest in the developed world (International Labour Office, 1999). The proportion of "high strain" (high demand–low control) jobs in Europe increased from 25% to 30% between 1991 to 1996 (European Foundation, 1997).

### *Trends in CVD*

Although CVD *mortality* in industrialized countries has fallen over the past 40 years (Liao & Cooper, 1995), CVD *incidence* in the United States has shown little or no decline over the past 20 years (Rosamond et al., 1998; Wilson et al., 1991). The gap between higher and lower socioeconomic status in CVD mortality (Gonzalez, Artalejo, & Calero, 1998; Marmot & Theorell, 1991) and incidence (Hallqvist, Lundberg, Diderichsen, & Ahlbom, 1998; Tuchsen & Endahl, 1999) is *increasing*. Increases in work stressors produced by economic globalization may be playing an important role in maintaining incidence rates for CVD.

## Work Stressors and CVD: Epidemiological Data

Various models of work stressors have been investigated. The most highly studied construct is *job strain*—in other words, work that combines high psychological work demands with low job decision latitude, or job control (Karasek & Theorell, 1990). More recently, the *effort–reward* model defines deleterious job conditions as a "mismatch between high workload (high demand) and low control over long-term rewards" (Siegrist, Peter, Junge, Cremer, & Seidel, 1990, p. 1128). Low reward includes concepts such as low "esteem reward" (e.g., respect, support), low income, and low "status control" (e.g., poor promotion prospects, job insecurity, status inconsistency). A number of studies have examined *threat-avoidant vigilant work*—in other words, work that involves continuously maintaining a high level of vigilance to avoid disaster, such as loss of human life (Belkić, Landsbergis, et al., 2000). This is a feature of a number of occupations at high risk for CVD—for example, urban mass transit operators, truck drivers, air traffic controllers, and sea pilots. Long work hours (Falger & Schouten, 1992; Sokejima & Kagamimori, 1998) and shift work (Steenland, 2000) have

also been investigated (see chapter 11, this volume). Prolonged exposure to stressful working conditions appears to be particularly deleterious (Belkić, Schnall, Savic, & Landsbergis, 2000).

Several reviews have been published on the association between these psychosocial work stressors and CVD (Belkić, Landsbergis, et al., 2000; Kristensen, Kronitzer, & Alfedsson, 1998; Schnall, Landsbergis, & Baker, 1994). As of 2000, there have been 24 studies of job strain and CVD (primarily CHD) among men (Belkić, Landsbergis, et al., 2000), and 6 studies among women (Brisson, 2000), most with significant positive associations. Four CVD studies have tested the moderating effect of job control on job demands and two of these found significant interactions (Hallqvist, Diderichsen, et al., 1998; Johnson & Hall, 1988). Among men, the impact of job strain on CVD is more consistent and stronger among blue-collar workers (e.g., Johnson & Hall, 1988; Theorell et al., 1998), with risk ratios as high as 10.0 (Hallqvist, Diderichsen, et al., 1998), than among men in jobs with higher socioeconomic status.

Of five studies of effort–reward imbalance among men (Bosma, Peter, Siegrist, & Marmot, 1998; Lynch, Krause, Kaplan, Salonen, & Salonen, 1997; Lynch, Krause, Kaplan, Tuomilehto, & Salonen, 1997; Peter et al., 1999b; Siegrist, 1996; Siegrist et al., 1990), all found significant positive associations between exposure and CVD. No studies of effort–reward and CVD in women have been published to date.

Significant associations with death as a result of heart attacks were found for "economic and financial implications of decisions taken at work, as well as the relevance of possible damage and hazards both economic and for human life as a consequence of possible mistakes made at work" among Italian male railroad workers (Menotti & Seccareccia, 1985). In the United States, cardiovascular disability was associated with the job dimensions "alert to changing conditions" and "hazardous job situation" (Murphy, 1991). Swedish men whose jobs entailed risk of explosion had an increased risk of hospitalization for myocardial infarction (MI; Alfredsson, Spetz, & Theorell, 1985). However, only a few epidemiologic studies have specifically examined aspects of threat-avoidant vigilant activity and CVD outcomes. The strongest evidence for this risk factor comes from studies of single occupations, where professional drivers, particularly urban transport operators, emerge as the occupation with the most consistent evidence of elevated risk of CHD and hypertension (Belkić et al., 1998).

Among women, in the Framingham Heart Study, 8-year heart disease incidence for *clerical* workers was double the rate for white-collar workers (Haynes & Feinleib, 1980) and 14-year incidence among *blue-collar* women was more than triple the rate for white-collar women (Eaker, Packard, & Thom, 1989). Twenty-year risk of MI or coronary death showed little difference between these occupational categories. However, other measures of socioeconomic status, such as low education or having a husband with a clerical or blue-collar occupation (versus white-collar), were associated with increased 20-year risk (Eaker, 1992). The association between job strain and CHD among Framingham clerical women was odds ratio = 5.2, substantially higher than the $OR = 2.9$ for all women (LaCroix, 1984). The combina-

tion of work and home stressors may help to explain increased CHD risk in lower socioeconomic-status women (Haynes & Feinleib, 1980).

### *Work Stressors and Hypertension*

A number of studies have explored whether the increased risk of CVD because of job strain is mediated by high BP.

*Effect of blood pressure measurement techniques and where measurements are taken.* Few studies of job strain and casual clinic BP have shown significant associations (Schnall et al., 1994); however, strong evidence of an association is found in studies where BP is measured by an ambulatory (portable) monitor (Belkić, Landsbergis, et al., 2000). This difference in findings may be explained, in part, by the imprecision and possible bias of taking "casual" BP measurements. For example, relaxation can occur when people are away from work, resulting in lower BP. There are also various atypical psychosocial stimuli present in a clinic that can affect BP, such as anxiety as a result of seeing a doctor. An alternative method of measuring BP is ambulatory monitoring, in which a person wears an automatic monitor on his or her arm throughout the work day (Pickering, 1991). Compared to casual BP measurements, ambulatory monitoring provides a more reliable measure of BP, because there is no observer bias and the number of readings is increased. It is also a more valid measure of average BP, because BP is measured during a person's normal daily activities and exposure to the daily stressors that influence persistent increases in BP. Ambulatory BP is a better predictor of target organ damage, such as increases in the size of the heart's left ventricle, and CVD, than are casual clinic BP readings (Verdecchia, Clement, Fagard, Palatini, & Parati, 1999).

*Occult (hidden) work-related hypertension.* The widespread application of ambulatory BP (AmBP) monitoring has focused attention on "white coat" hypertension (elevated casual clinic BP with normal AmBP), an entity of low predictive value with respect to hypertensive sequelae. Meanwhile, occult work-related hypertension, which is often characterized by the *opposite* pattern (normal casual clinic BP and elevated work AmBP), remains underdetected despite its potential clinical importance. In the Work Site BP Study (Schnall et al., 1990), 86 men with elevated casual clinic BP and 181 with normal casual clinic BP underwent AmBP on a working day. Twenty-seven men had "white coat" hypertension, and 36 had occult workplace hypertension. Thus, among working populations, the problem of occult workplace hypertension could be of even greater magnitude than that of "white coat" hypertension (Belkić et al., 2001).

*Epidemiological data.* Of 10 cross-sectional studies of job strain (or its components) and AmBP among men, many indicate significant positive associations with work BP (Belkić, Landbergis, et al., 2000). In the five studies in which measurements were also made outside of work, job strain

was associated with leisure time systolic BP. Both cohort studies of ambulatory BP among men found significant associations with job strain (Schnall, Landsbergis, Schwartz, Warren, & Pickering, 1998; Theorell et al., 1988). Of the six cross-sectional studies of job strain and AmBP among women, four indicate a significant positive association with work systolic BP (Brisson, 2000). Work systolic BP in men facing job strain is typically 4 to 8 mm Hg higher than those without job strain. In studies of women, estimates of the magnitude of effect have been even higher. One AmBP study tested the moderating effect of job control on job demands and found a significant interaction (Landsbergis, Schnall, Warren, Pickering, & Schwartz, 1994). Studies of high effort–low reward at work have shown significant positive associations with hypertension in men (Peter & Siegrist, 1997), in men and women (Peter et al., 1998), and with a comanifestation of hypertension and high LDL-cholesterol in men (Siegrist, Peter, Georg, Cremer, & Seidel, 1991).

The Work Site Blood Pressure (BP) Study, a longitudinal study of psychosocial factors and ambulatory BP, was begun at Cornell Medical College in 1985 in New York City. At the first round of data collection (Time 1), men employed in "high strain" jobs were at increased risk of hypertension ($OR$ = 2.7), had increased left ventricular mass index (7.3 g/$m^2$; Schnall et al., 1990), and had higher levels of work (6.7 mm Hg systolic, 2.7 mm Hg diastolic), home and sleep ambulatory BP (AmBP), controlling for potential risk factors (Landsbergis et al., 1994; Schnall, Schwartz, Landsbergis, Warren, & Pickering, 1992).

Examining data from Time 1 and a second round of data collection 3 years later (Time 2), it was possible to construct a measure of repeated or cumulative exposure to job strain, which exhibited a consistent pattern of associations with work, home, and sleep AmBP . The chronically exposed group exhibited an 11 to 12 mm Hg higher systolic and 6 to 9 mm Hg higher diastolic work AmBP than the group unexposed at both times. These effect sizes are substantial, more than twice the difference between Black and White individuals in this sample and more than the estimated effect of aging 25 years or gaining 50 pounds in weight (Schnall et al., 1998).

Examining the 3-year change in AmBP between Time 1 and Time 2, those reporting job strain at Time 1 but no job strain at Time 2 exhibited a decrease in systolic AmBP of 5.3 mm Hg at work and 4.7 mm Hg at home ($p < .05$; Schnall et al., 1998). The decrease in AmBP associated with a decrease in job strain over time suggests that early detection and prevention strategies should be effective. The cross-sectional association between job strain and AmBP in men was also substantially greater for men with lower socioeconomic status (Landsbergis, Schnall, Warren, Pickering, & Schwartz, 1999), implying that increased efforts at early detection and prevention are required in this group.

## *Population Attributable Risk of Psychosocial Work Factors*

The population attributable risk (PAR%) of CVD as a result of psychosocial work factors may be substantial. Karasek and Theorell estimated a prevalence of exposure of 15 to 25% in Sweden and a resulting PAR% of 7 to

16% for men (Karasek & Theorell, 1990). A relative risk of 2.0 and a prevalence estimate for job strain of 15 to 25% (a conservative estimate given current economic trends and current European surveys) produces a PAR% of 13 to 20%. In Denmark, the PAR% for monotonous high-paced work (a conservative proxy measure for job strain) was estimated at 6% for men and 14% for women (Kristensen et al., 1998). However, few studies have examined the combined or synergistic effect of workplace stressors, which would increase estimates of the proportion of CVD as a result of work. One Swedish study (Peter et al., 1999) did find that the combined effects of exposure to job strain and to effort–reward imbalance on CVD were much stronger than the separate effects of each model.

## Mechanisms by Which Work Stressors Can Lead to CVD Outcomes

There are "intimate connections between the social environment and the central nervous system (CNS), and the CNS and the cardiovascular system via the autonomic and neuroendocrine systems" (Schnall, Belkić, Landsbergis, & Baker, 2000b, p. 2). This interrelation has been conceptualized as "econeurocardiology," the biological paradigm by which social factors, such as work stressors, are perceived and processed by the CNS, resulting in pathophysiological changes that increase risk of cardiovascular disease (Belkić, Schnall, Landsbergis, & Baker, 2000; Wolf, 2000). Although the mechanisms remain to be fully elucidated, at least four possible pathways exist by which job stressors may influence CVD risk (Schnall et al., 1994). The first three pathways, reviewed in this section, involve known cardiac risk factors, increased risk of cardiac events among vulnerable individuals, and atherosclerosis. Possible social psychological pathways are reviewed in the next section.

### *Known CVD Risk Factors*

We review the evidence on the association between work stressors and known CVD risk factors, such as hypertension, serum cholesterol, cigarette smoking, sedentary behavior, and atheroschlerosis in this section.

*Hypertension.* The strongest empirical evidence for a role for job stressors in the promotion of known CVD risk factors is for hypertension (Belkić, Schnall, Landsbergis, et al., 2000). Sympathetic nervous system overactivity (associated with job stressors) is also implicated in the clustering of hypertension and various atherogenic biochemical abnormalities, together known as the cardiovascular metabolic syndrome (CVM). The CVM includes hypertension, increased total cholesterol, triglycerides and insulin; decreased high-density lipoprotein (HDL) cholesterol; central obesity; insulin resistance and glucose intolerance; and hypercoagulability and reduced fibrinolysis (Fossum, Hoieggen, Moan, Rostrup, & Kjeldsen, 2000).

Building on the work of Henry and Stephens (Henry, 1992), Frankenhauser and colleagues confirmed the involvement of two neuroendocrine systems in the stress response—the *sympathoadrenal medullary* system (which secretes the catecholamines, epinephrine, and norepinephrine), and the *hypothalamic-pituitary-adrenal cortical* system (which secretes corticosteroids such as cortisol). Under demanding conditions in which organisms can exert control, epinephrine levels increase, and some studies suggest that cortisol levels may decline (Frankenhaeuser & Johansson, 1986). However, in demanding but low-control situations (analogous to "job strain"), both epinephrine and cortisol are elevated (Frankenhaeuser, 1989; Schwartz, Belkić, Schnall, & Pickering, 2000). Elevated levels of both catecholamines and cortisol have severe consequences for myocardial pathology (Fredriksson, Sundin, & Frankenhaeuser, 1985; Steptoe, 1981). Cortisol enhances and prolongs the effect of epinephrine (Theorell, 2000). The combination of these hormones appears to promote BP elevation (Schwartz et al., 2000), dyslipidemia (Theorell, 2000), and the CVM (Fossum et al., 2000). The physiology of the stress response contradicts the standard advice given in stress management programs—that stress depends only on a person's *perception* of environmental *demands.* In fact, levels of *control* available to individuals in the environment is key to the type of stress response elicited.

These simple models may not capture the complexity of the stress response, and profound regulatory cortisol disturbances resulting from chronic arousal (Theorell, 2000). Short-term cortisol and adrenalin elevation has been associated with healthier coping in stressful situations (Karasek & Theorell, 1990). However, chronic elevation of cortisol appears in clinical depression, an apparent risk factor for CHD (Rozanski, Blumenthal, & Kaplan, 1999), and low cortisol levels reflect exhausted function, which has also been associated with cardiac death (Appels & Otten, 1992).

Personal control may reduce the duration of the stress response (Frankenhaeuser, 1989). Repetitive and machine-paced jobs and excessive overtime tend to prolong "unwinding," the return of neuroendocrine levels to baseline (Frankenhaeuser & Johansson, 1986). AmBP studies indicate a "carryover" effect in which the work, home, and sleep BP of "high strain" workers is elevated above levels of other workers (Belkić, Landsbergis, et al., 2000). Another obstacle to "unwinding" may be the dual role (the additional responsibility for household and children) that many workers (particularly women) face when they return home (Hall, 1992). A Canadian study found evidence of synergism between family responsibilities and job strain in their effect on BP among college-educated women (Brisson et al., 1999).

*Known risk factors other than hypertension.* Research on the association of work stressors with serum cholesterol and behavioral CVD risk factors has produced inconsistent results. Effort–reward imbalance was associated with LDL/HDL cholesterol ratio in several studies (Peter et al., 1998; Siegrist, Matschinger, Cremer, & Seidel, 1988) and with the combined occurrence of hypertension and hyperlipidemia (Siegrist et al., 1991). Some evidence exists for an association of job strain with smoking intensity and

cessation (Green & Johnson, 1990; Hellerstedt & Jeffery, 1997; Kawakami, Haratani, & Araki, 1998) although null studies have also been reported. In the Work Site BP Study, among men, an increase in job decision latitude over 3 years was associated with quitting smoking (Landsbergis et al., 1998). Job strain has been associated with BMI (Netterstrom, Kristensen, Damsgaard, Olsen, & Sjol, 1991) and skinfold thickness (Georges, Wear, & Mueller, 1992), but other studies of job strain and other job stressors have found no association with overweight. Several studies found sedentary behavior to be associated with low latitude for men and women (Hellerstedt & Jeffery, 1997; Johansson, Johnson, & Hall, 1991) and high job demands for women (Johansson et al., 1991), however, null studies also exist.

*Risk of cardiac events in vulnerable persons.* The influence of sympathoadrenal activity on cardiovascular function includes increased myocardial oxygen demand and decreased myocardial oxygen supply that can lead in vulnerable individuals to myocardial ischemia (Belkić, 2000b), destabilization of the cardiac electrical substrate (Belkić, 2000a), as well as increased risk of clot formation and disruption of unstable plaques (Steptoe & Marmot, 2000). Platelet activation and the concentration of fibrinogen also play a role in acute thrombosis (Steptoe & Marmot, 2000). Job strain may inhibit anabolic (regenerative) processes, which may contribute to an adverse, atherogenic metabolic profile (Theorell, 2000). Environmental stressors may act as potential triggers of life-threatening arrhythmias and sudden cardiac death (see Belkić, 2000a; Willich, Maclure, Mittleman, Arntz, & Muller, 1993). Young men who have suffered a heart attack and return to high strain jobs may be particularly at risk for CHD-related mortality (Theorell, Perski, Orth-Gomer, Hamsten, & de Faire, 1991). However, the evidence to date is not as clear with respect to women who return to high strain jobs after cardiac events (Orth-Gomer et al., 2000).

*Atherosclerosis.* Hypertension contributes to atherosclerosis (Steptoe & Marmot, 2000). In addition, low decision latitude is associated with high plasma fibrinogen, suggesting a link with coagulation and, thus, atherosclerosis (Brunner et al., 1996; Markowe et al., 1985). This pathway is consistent with the association between epinephrine levels and coagulation (Gertler & White, 1976; Haft, 1974), for example, the stimulation of platelet adhesiveness by epinephrine. Progression of coronary atherosclerosis over time has been associated with low job decision latitude (Langosch, Brodner, & Borcherding, 1983) and with high demands and low economic rewards at work (Lynch, Krause, Kaplan, Tuomilehto, et al., 1997).

## Insights From Behavioral Medicine–Social Psychological Mechanisms

Although much research has focused on negative affect as a CVD risk factor, there has been a tendency to view behavioral–psychological factors in isolation, without conceptualizing them in relation to objective stressors,

in the work environment and elsewhere. Figure 13.1 presented a model of affect, behavior, and personality as outcomes of social conditions and as mediators of the effects of social stressors on CVD risk. Behavioral–psychological factors may also interact with social stressors in increasing CVD risk.

### *Role of Job Characteristics*

Additional research is needed on the hypothesis that job stressors may increase the risk of hypertension and CVD, in part by shaping personality or increasing negative affect. Karasek's job demands–control model describes the adult socialization of personality traits and behavior patterns that occur at work. Chronic adaptation to low control–low demand situations ("passive" jobs) can result in reduced self-efficacy, greater external locus of control, reduced ability to solve problems or tackle challenges (Karasek & Theorell, 1990), and feelings of depression (Karasek, 1979) or "learned helplessness"—yet these jobs are not associated with increases in BP. Conversely, when high (but not overwhelming) job demands are matched with greater authority and skill, more active learning and greater internal locus of control develop. This can enable individuals to develop a broader range of coping strategies. For example, in Sweden, workers whose jobs became more "passive" over six years reported less participation in political and leisure activities. In contrast, workers in jobs that became more "active" participated more in these activities (Karasek & Theorell, 1990, p. 53). In a U.S. study, evidence was seen for increased intellectual flexibility, nonauthoritarianism, capacity to take responsibility for one's actions, and intellectually demanding leisure time after 10 years among those with greater occupational self-direction, a concept similar to decision latitude (Kohn & Schooler, 1982). This research points to the organization of work not only as a potential source of stress and increased disease risk, but also potentially as a key factor in the promotion of physical and mental health and the development of one's creative potential, effective coping, and social involvement outside work.

### *Role of Personality–Negative Affect*

The precise role played by personality traits (e.g., Type A behavior, overcommitment, or hostility) or negative affect (e.g., anxiety, anger, or depression) in the development of hypertension and CVD remains unclear. Current evidence is more consistent for an association with CVD than with hypertension.

*Hypertension.* Empirical support for the concept of the "hypertensive personality" remains equivocal (Shapiro, 1988). Hypertension has been associated with internalized aggression (Perini et al., 1990) and with anxiety (Jonas, Franks, & Ingram, 1997; Markovitz, Matthews, Kannel, Cobb,

& D'Agostino, 1993), although other studies have failed to find these associations (Jonas et al., 1997; Markovitz et al., 1993; Sparrow, Garvey, Rosner, & Thomas, 1982). However, methodological problems have plagued studies of this association (see Friedman et al., 2001). Another limitation is the lack of a model that predicts *specific* interactions or mediation between work stressors and personality characteristics in the development of hypertension. For example, in one study, suppressed anger was associated with the prevalence of hypertension in male hourly workers only among those reporting job stress (Cottington, Matthews, Talbott, & Kuller, 1986). Other studies have shown that asymptomatic participants with hypertension (Knox, Svensson, Waller, & Theorell, 1988), as well as those with normal BP with a family history of hypertension (Theorell, 1990), seem to express fewer emotions and have a noncomplaining attitude (Karasek & Theorell, 1990). Theorell hypothesizes that such personality characteristics may result in part from a stressful work environment that "enforces a noncomplaining attitude and prevents development of active emotional coping" (Theorell, 1990). Thus, he observed an association between *underreporting* of stress and an *increased* physiologic response. Data among urban transport operators corroborates these premises: Operators with borderline or definite hypertension were distinguished from those with normal BP by having a low admitted fear during driving, while showing increased BP rise together with heightened selective attention during laboratory paradigms that mimicked stressful aspects of the traffic environment (Belkić et al., 1996; Emdad et al., 1997)

*CVD.* Job stressors may also increase the risk of CVD, in part by influencing personality characteristics or negative affect. Many emotions "are responses to power and status differentials embedded within social situations" (Kubzansky, Kawachi, Weiss, & Sparrow, 1998, p. 55). Positive associations have been found between anxiety and job stressors in a number of studies (Bourbonnais, Brisson, Moisan, & Vezina, 1996; Bourbonnais, Comeau, & Vezina, 1999; Stansfeld, North, White, & Marmot, 1995). Depression has also been associated prospectively with CVD (Anda et al., 1993; Aromaa et al., 1994), and linked with stressful job characteristics (Karasek, 1979; Lennon, 1987; Mausner-Dorsch & Eaton, 2000; Stansfeld et al., 1995).

Type A behavior has been described as a stable personality trait, however, Matthews and Haynes pointed out that

> this behavior pattern is thought to be encouraged by Western society because it appears to offer special rewards and opportunities to those who can think, perform, and even play more rapidly and aggressively than their peers. . . . it is seen as the outcome of a set of predispositions interacting with specific types of eliciting situations, including those that are stressful or challenging. (1986, p. 924)

However, the assumption that Type A is a stable personality characteristic contributed to researchers measuring it only once at baseline, resulting

in significant misclassification and bias in many studies. Evidence exists that the hostility component of Type A is a risk factor for CHD (Smith, 1992), and some studies have found associations between hostility and job stressors (Bosma, Stansfeld, & Marmot, 1998; Landsbergis, Schnall, Deitz, Friedman, & Pickering, 1992). "Overcommitment" in the effort–reward imbalance model (reminiscent of the work overinvolvement component of Type A behavior) is considered to be "a personal characteristic which is rather stable over time" (Siegrist et al., 1990, p. 1128), however, it has been associated with job strain (Peter, 1997).

Finally, negative affectivity (NA) has been proposed as a confounder of the stress–illness association (McCrae, 1990). However, it has not been associated with MI (Costa & McCrae, 1985) or BP (Landsbergis et al., 1992), and, in the Whitehall study, controlling for NA barely affected the association between low job control and CHD (Bosma, Peter, et al., 1998). In short, contrary to widely held beliefs, there is currently little evidence supporting an etiologic role for psychological or personality factors in essential hypertension (Friedman et al., 2001) and limited evidence for a role in CVD.

## Occupational Health Psychology in the Prevention and Management of Work-Related Hypertension and CVD

The occupational health psychologist can play a key role in preventing and managing work-related hypertension and CVD. As part of a public health strategy, we recommend a team approach in which occupational health psychologists work together with clinicians, health educators, ergonomists, epidemiologists, and other health professionals to identify high-risk workplaces and occupations, facilitate the provision of clinical care, and design and implement workplace interventions (as in Herbert et al., 1997).

### *Risk Assessment*

The first step in this process is *worksite surveillance.* Experts in Japan, Europe, and the United States have called for a program of surveillance at individual workplaces and monitoring at national and regional levels to identify the extent of work-stress related health problems and to provide baselines against which to evaluate efforts at amelioration. They recommended that workplaces assess both workplace stressors and health outcomes known to result from such exposures ("The Tokyo Declaration," 1998). First, the surveillance team needs to ascertain whether the current occupation is high-risk; whether workers are exposed to any physical, chemical, work schedule, or psychosocial CVD risk factors at work; and if any of these been increasing over time (Belkić, Schnall, & Ugljesic, 2000). Questionnaires, such as the Job Content Questionnaire (Karasek et al., 1985), the Effort-Reward Imbalance Questionnaire (Siegrist & Peter, 1996), and the Occupational Stress Index (Belkić, Savic, Theorell, & Cizinsky, 1995)

can help assess job characteristics and job stressors. Second, workplace screening should be conducted for biomedical CVD risk factors, including worksite point estimates of BP (Schnall & Belkić, 2000). Such surveillance can help to identify clusters of work-related hypertension and help target worksites for primary and secondary prevention programs.

Another modality of risk assessment is *an occupational history* of individual workers. The occupational health psychologist would have the expertise to identify those workplace exposures that may affect cardiovascular well-being and could prepare a succinct narrative to become part of the clinical record. We have developed hands-on tools that effectively trained graduate students to acquire this practical skill (Schnall, Belkić, Landsbergis, & Baker, 2001).

## *Preventing CVD and Improving the Cardiovascular Health of Working People*

Both individual health promotion and workplace protection–prevention programs are needed to combat the epidemic of CVC. However, because of the limitation of health promotion programs, primary prevention strategies, namely job redesign, are fundamental. The occupational health psychologist can also play an important role in secondary and tertiary prevention by working together with clinicians.

*Health promotion.* One important role for the occupational health psychologist is counseling patients to reduce their levels of unhealthy behaviors, such as smoking. However, cardiac risk factor counseling in isolation may have poor efficacy, particularly among occupational groups with a heavy burden of exposure to occupational stressors. For example, Fisher and Belkić stated,

> Despite devotion of substantial time and the use of state-of-the-art methods . . . our efforts applied systematically among professional drivers were, at best, only minimally effective, unless there was a concomitant amelioration in stressful working conditions. (Fisher & Belkić, 2000)

Although stress management interventions may have positive effects, if employees return to an unchanged work environment and high levels of job stressors, those beneficial effects are likely to be eroded (Nowack, 2000).

*Worksite health promotion.* Few health promotion programs have "focused on the physical, psychosocial or policy work environment," including job strain. Many "programs have emphasized risk-factor reduction strategies . . . but have not integrated disease prevention and safety programs with organizational policies to enhance the physical and social quality of the workplace" (Stokols, Pelletier, & Fielding, 1995, p. 1137). Another limitation is the tendency for less participation by higher risk—for example,

lower socioeconomic status employees (Lewis, Huebner, & Yarborough, 1996).

A number of researchers have recommended integrating workplace health promotion and occupational health, to develop complementary behavioral and environmental interventions (Dejoy & Southern, 1993; Heaney & Goetzel, 1997). One example of such a program is the WellWorks Project conducted in 24 worksites in Massachusetts. A "significant association was observed between participation in nutrition and [environmental] exposure-related activities, suggesting that participation in programs to reduce exposures to occupational hazards might contribute to blue-collar workers' participation in health promotion activities." In addition, "when workers were aware of change their employer had made to reduce exposures to occupational hazards, they were more likely to participate in both smoking control and nutrition activities" (Sorensen, Stoddard, Ockene, Hunt, & Youngstrom, 1996, p. 191). Barriers to participation, such as blue-collar workers' time constraints and job responsibilities, were addressed, for example, through negotiation of time-off for participation in health promotion activities (Sorensen et al., 1995).

*Tertiary prevention: return to work after cardiac events.* Cardiologists are called on to judge the cardiovascular work fitness of patients who have suffered cardiac events. Complicating the issue is that jobs in which public safety could be compromised with the occurrence of an acute cardiac event (deGaudemaris, 2000) are often those with high exposure to potentially cardio-deleterious factors (e.g., urban transit operators, air traffic controllers; Fisher & Belkić, 2000). On the other hand, advances in cardiovascular therapy permit the cardiovascular function of many patients to be restored to make returning to work *potentially* possible (deGaudemaris, 2000). In the study of Theorell et al. (Theorell et al., 1991), among men who had suffered a first MI below age 45, the predictive strength for five-year CHD mortality of returning to a high strain job was of similar magnitude to the degree of coronary atherosclerosis and more powerful than left ventricular ejection fraction. The expertise of the occupational health psychologist could be indispensable in helping clinicians to tackle this most delicate issue: "Should heart attack patients return to stressful jobs?" Namely, the occupational health psychologist could identify potentially modifiable cardionoxious stressors in the patient's work environment, and then together with the clinician formulate and implement a plan to provide a safer return to work after cardiac events.

## *Promoting Healthy Work: Job Redesign and Cardiovascular Health*

The effectiveness of a limited number of interventions to improve work organization and job design, reduce job stressors, and create a more healthy work organization have been documented (International Labor Office, 1992;

Landsbergis et al., 1993; Murphy, Hurrell, Sauter, & Keita, 1995). For example, an intervention among Swedish civil servants included worker committees that developed and carried out action plans to reduce work stressors. A significant decrease in apolipoprotein B/AI ratio occurred in the intervention group, but not in the control group. Stimulation from and autonomy over work significantly increased in the intervention group but remained the same in the control group (Orth-Gomer, Eriksson, Moser, Theorell, & Fredlund, 1994). An intervention on an inner-city bus line in Stockholm was designed to diminish time pressure and promote traffic flow. There was a significant decline in systolic BP (–10.7 mm Hg) in the intervention group that was greater than in the comparison group (–4.3; Rydstedt, Johansson, & Evans, 1998). A Swedish field study also showed that systolic BP, heart rate, epinephrine, and self-reported tiredness increased significantly from the start to the end of a day shift at a traditional auto assembly line, but not at a more flexible work organization with small autonomous groups having greater opportunities to influence the pace and content of their work (Melin, Lundberg, Soderlund, & Granqvist, 1999).

There have been no published job redesign studies in the United States that have examined CVD outcomes per se. However, a number of job redesign programs in the United States have been conducted that have examined other stress-related health outcomes and that provide valuable guidance for current efforts. These programs have included efforts to reduce symptoms of "burnout" among employees of a state child protection agency through a labor–management committee and new technology (Cahill & Feldman, 1993), increase participation in decision making through labor–management committees at an automobile manufacturer (Israel, Schurman, & House, 1989), and reduce work-related musculoskeletal disorders through job redesign at a meat-packing plant (Smith & Zehel, 1992).

In addition to job redesign, legislative or regulatory approaches have been used. However, efforts to regulate (Warren, 2000) or collectively bargain (Landsbergis, 2000) over work organization and psychosocial stressors such as job strain have met with limited success in the United States. A promising development in this area is recent legislation in states such as California and New Jersey, which provide minimum staffing levels and limits on mandatory overtime for health care workers. Valuable models for the United States include legislation in Scandinavia, the European Union (Levi, 2000), and Japan (Shimomitsu & Odagiri, 2000) that regulate work organization and job stressors as health hazards.

Occupational health psychologists can also work with labor–management committees and other health professionals to develop and evaluate the impact of job redesign programs, help convince employers of the long-term benefits of such programs, involve employees in such programs, and help develop appropriate legislation and regulations. In short, the occupational health psychologist can potentially play a pivotal, multifaceted role in helping to create and promote a "heart healthy" work environment.

## References


Alfredsson, L., Spetz, C., & Theorell, T. (1985). Type of occupation and near-future hospitalization for myocardial infarction and some other diagnoses. *International Journal of Epidemiology, 14,* 378–388.

Anda, R., Willianson, 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*(4), 285–293.

Appels, A., & Otten, F. (1992). Exhaustion as precursor of cardiac death. *British Journal of Clinical Psychology, 31,* 351–356.

Aromaa, A., Raitasalo, R., Reunanen, A., Impivaara, O., Heliovaara, M., Knekt, P., Lehtinen, V., Joukamaa, M., & Maatela, J. (1994). Depression and cardiovascular disease. *Acta Psychiatrica Scandinavica* (Suppl. 377), 77–82.

Belkić, K. (2000a). Cardiac electrical stability and environmental stress. *Occupational Medicine: State of the Art Reviews, 15*(1), 117–120.

Belkić, K. (2000b). Myocardial oxygen supply and demand: Environmental triggers of imbalance. *Occupational Medicine: State of the Art Reviews, 15*(1), 132–136.

Belkić, K., Emdad, R., & Theorell, T. (1998). Occupational profile and cardiac risk: Possible mechanisms and implications for professional drivers. *International Journal of Occupational Medicine and Environmental Health, 11,* 37–57.

Belkić, K., Emdad, R., Theorell, T., Cizinsky, S., Wennberg, A., Hagman, M., Johnasson, L., Savic, C., & Olsson, K. (1996). *Neurocardiac mechanisms of heart disease risk among professional drivers.* Final report. Stockholm: Swedish Fund for Working Life.

Belkić, K., Landsbergis, P. A., Schnall, P., Baker, D., Theorell, T., Siegrist, J., Peter, R., & Karasek, R. (2000). Psychosocial factors: Review of the empirical data among men. *Occupational Medicine: State of the Art Reviews, 15*(1), 24–46.

Belkić, K., Savic, C., Theorell, T., & Cizinsky, S. (1995). *Work Stressors and Cardiovascular Risk: Assessment for Clinical Practice.* Part I (256). Stockholm: National Institute for Psychosocial Factors and Health. Section for Stress Research, Karolinska Institute, WHO Psychosocial Center.

Belkić, K., Schnall, P., Landsbergis, P., & Baker, D. (2000). The workplace and CV health: Conclusions and thoughts for a future agenda. *Occupational Medicine: State of the Art Reviews, 15*(1), 307–322.

Belkić, K. B., Schnall, P. L., Landsbergis, P. A., Schwartz, J. E., Gerber, L., Baker, D., & Pickering, T. G. (2001). Hypertension at the workplace—An occult disease? The need for work site surveillance. *Advances in Psychosomatic Medicine, 22,* 116–138.

Belkić, K., Schnall, P., Savic, C., & Landsbergis, P. A. (2000). Multiple exposures: Toward a model of total occupational burden. *Occupational Medicine: State-of-the-Art Reviews, 15*(1), 94–98.

Belkić, K., Schnall, P., & Ugljesic, M. (2000a). Cardiovascular evaluation of the worker and workplace: A practical guide for clinicians. *Occupational Medicine: State of the Art Reviews, 15*(1), 213–222.

Bond, J. T., Galinsky, E., & Swanberg, J. E. (1998). *The 1997 National Study of the Changing Workforce.* New York: Families and Work Institute.

Bosma, H., Peter, R., Siegrist, J., & Marmot, M. (1998). Two alternative job stress models and the risk of coronary heart disease. *American Journal of Public Health, 88,* 68–74.

Bosma, H., Stansfeld, S. A., & Marmot, M. G. (1998). Job control, personal characteristics, and heart disease. *Journal of Occupational Health Psychology, 3*(4), 402–409.

Bourbonnais, R., Brisson, C., Moisan, J., & Vezina, M. (1996). Job strain and psychological distress in white-collar workers. *Scandinavian Journal of Work, Environment and Health, 22,* 139–145.

Bourbonnais, R., Comeau, M., & Vezina, M. (1999). Job strain and evolution of mental health among nurses. *Journal of Occupational Health Psychology, 4,* 95–107.

Braunwald, E. (1997). Cardiovascular medicine at the turn of the millennium: Triumphs, concerns and opportunities. *New England Journal of Medicine, 337,* 1360–1369.

Brisson, C. (2000). Women, work and cardiovascular disease. *Occupational Medicine: State of the Art Reviews, 15*(1), 49–57.

Brisson, C., Laflamme, N., Moisan, J., Milot, A., Masse, B., & Vezina, M. (1999). Effect of family responsibilities and job strain on ambulatory blood pressure among white-collar women. *Psychosomatic Medicine, 61*(2), 205–213.

Brunner, E. J., Smith, G. D., Marmot, M. G., Canner, R., Beksinska, M., & O'Brien, J. (1996). Childhood social circumstances and psychosocial and behavioral factors as determinants of plasma fibrinogen. *Lancet, 347,* 1008–1013.

Cahill, J., & Feldman, L. H. (1993). Computers in child welfare: Planning for a more serviceable work environment. *Child Welfare, 72,* 3–12.

Carvalho, J. J. M., Baruzzi, R. G., Howard, P. F., Poulter, N., Alpers, M. P., Franco, L. J., Marcopito, L. F., Spooner, V. J., Dyer, A. R., Elliott, P., Stamler, J., & Stamler, R. (1989). Blood pressure in four remote populations in the INTERSALT study. *Hypertension, 14,* 238–246.

Cooper, C. (1996). Working hours and health. *Work & Stress, 10,* 1–4.

Cooper, R. S., Rotimi, C. N., & Ward, R. (1999, Feb.). The puzzle of hypertension in African-Americans. *Scientific American,* 56–63.

Costa, P. T., & McCrae, R. R. (1985). Hypochondriasis, neuroticism, and aging: When are somatic complaints unfounded? *American Psychologist, 40,* 19–28.

Cottington, E. M., Matthews, K. A., Talbott, E., & Kuller, L. H. (1986). Occupational stress, suppressed anger, and hypertension. *Psychosomatic Medicine, 48,* 249–260.

deGaudemaris, R. (2000). Clinical issues: Return to work and public safety. *Occupational Medicine: State of the Art Reviews, 15*(1), 223–230.

Dejoy, D. M., & Southern, D. J. (1993). An integrative perspective on work site health promotion. *Journal of Occupational Medicine, 35*(12), 1221–1230.

Eaker, E. D. (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*(8), 854–864.

Eaker, E. D., Packard, B., & Thom, T. H. (1989). Epidemiology and risk factors for coronary heart disease in women. In P. S. Douglas (Ed.), *Heart disease in women* (pp. 129–145). Philadelphia: FA Davis.

Emdad, R., Belkić, K., Theorell, T., Cizinsky, S., Savic, C., & Olsson, K. (1997). Work environment, neurophysiologic and psychophysiologic models among professional drivers with and without cardiovascular disease: Seeking an integrative neurocariologic approach. *Stress Medicine, 13,* 7–21.

European Foundation. (1997). *Time constraints and autonomy at work in the European Union.* Dublin: European Foundation for the Improvement of Living and Working Conditions.

Falger, P. R. J., & Schouten, E. G. W. (1992). Exhaustion, psychologic stress in the work environment and acute myocardial infarction in adult men. *Journal of Psychosomatic Research, 36,* 777–786.

Fisher, J., & Belkić, K. (2000). A public health approach in clinical practice. In P. Schnall, K. Belkić, P. A. Landsbergis, & D. Baker (Eds.), *The workplace and cardiovascular disease. Occupational medicine: State of the art reviews* (Vol. 15, pp. 245–256). Philadelphia: Hanley and Belfus.

Fossum, E., Hoieggen, A., Moan, A., Rostrup, M., & Kjeldsen, S. E. (2000). The cardiovascular metabolic syndrome. *Occupational Medicine: State of the Art Reviews, 15*(1), 146–150.

Frankenhaeuser, M. (1989). A biopsychosocial approach to work life issues. *International Journal of Health Services, 19*(4), 747–758.

Frankenhaeuser, M., & Johansson, G. (1986). Stress at work: Psychobiological and psychosocial aspects. *International Review of Applied Psychology, 35,* 287–299.

Fredriksson, M., Sundin, O., & Frankenhaeuser, M. (1985). Cortisol excretion during the defence reaction in humans. *Psychosomatic Medicine, 47,* 313–319.

Friedman, R., Landsbergis, P. A., Schnall, P. L., Pieper, C., Gerin, W., Pickering, T. G., & Schwartz, J. E. (2001). Psychological variables in hypertension: Relationship to casual or ambulatory blood pressure in men. *Psychosomatic Medicine, 63,* 19–31.

Georges, E., Wear, M. L., & Mueller, W. H. (1992). Body fat distribution and job stress in Mexican-American men of the Hispanic Health and Nutrition Examination Survey. *American Journal of Human Biology, 4,* 657–667.

Gertler, M. M., & White, P. D. (1976). *Coagulation factors and coronary heart disease: a 25-year study in retrospect.* Oradell, NJ: Medical Economics.

Gonzalez, M. A., Artalejo, F. R., & Calero, J. R. (1998). Relationship between socioeconomic status and ischaemic heart disease in cohort and case-control studies: 1960–1993. *International Journal of Epidemiology, 27*(3), 350–358.

Green, K. L., & Johnson, J. V. (1990). The effects of psychosocial work organization on patterns of cigarette smoking among male chemical plant employees. *American Journal of Public Health, 80,* 1368–1371.

Haft, J. J. (1974). Cardiovascular injury induced by sympathetic catecholamines. *Progress in Cardiovascular Disease, 17,* 73–86.

Hall, E. M. (1992). Double exposure: The combined impact of the home and work environments on psychosomatic strain in Swedish women and men. *International Journal of Health Services, 22*(2), 239–260.

Hallqvist, J., Diderichsen, F., Theorell, T., Reuterwall, C., Ahlbom, A., & the SHEEP Study Group. (1998). Is the effect of job strain on myocardial infarction due to interaction between high psychological demands and low decision latitude? Results from Stockholm Heart Epidemiology Program (SHEEP). *Social Science and Medicine, 46*(11), 1405–1415.

Hallqvist, J., Lundberg, M., Diderichsen, F., & Ahlbom, A. (1998). Socioeconomic differences in risk of myocardial infarction 1971–1994 in Sweden: Time trends, relative risks and population attributable risks. *International Journal of Epidemiology, 27,* 410–415.

Haynes, S. G., & Feinleib, M. (1980). Women, work and coronary heart disease: Prospective findings from the Framingham Heart Study. *American Journal of Public Health, 70*(2), 133–141.

Heaney, C. A., & Goetzel, R. Z. (1997). A review of health-related outcomes of multi-component worksite health promotion programs. *American Journal of Health Promotion, 11,* 290–308.

Hellerstedt, W. L., & Jeffery, R. W. (1997). The association of job strain and health behaviors in men and women. *International Journal of Epidemiology,* 26(3), 575–583.

Henry, J. P. (1992). Biological basis of the stress response. *Integrative Physiological and Behavioral Science, 27,* 66–83.

Herbert, R., Plattus, B., Kellogg, L., Luo, J., Marcus, M., Mascolo, A., & Landrigan, P. J. (1997). The union health center: A working model of clinical care linked to preventive occupational health services. *American Journal of Industrial Medicine, 31,* 263–273.

International Labour Office. (1992). *Conditions of Work Digest: Preventing Stress at Work.* Geneva: International Labour Office.

International Labour Office. (1999). *Key Indicators of the Labour Market 1999.* Geneva: International Labour Office.

Israel, B. A., Schurman, S. J., & House, J. S. (1989). Action research on occupational stress: Involving workers as researchers. *International Journal of Health Services, 19,* 135–155.

Johansson, G., Johnson, J. V., & Hall, E. M. (1991). Smoking and sedentary behavior as related to work organization. *Social Science and Medicine, 32,* 837–846.

Johnson, J. V., & Hall, E. M. (1988). Job strain, workplace social support, and cardiovascular disease: A cross-sectional study of a random sample of the Swedish working population. *American Journal of Public Health, 78*(10), 1336–1342.

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,* 43–51.

Kannel, W. B. (1992). The Framingham experience. In M. Marmot & P. Elliott (Eds.), *Coronary heart disease epidemiology* (pp. 67–82). Oxford: Oxford University Press.

Kaplan, G. A., & Keil, J. E. (1993). Socioeconomic factors and cardiovascular disease: A review of the literature. *Circulation, 88*(4 Pt. 1), 1973–1998.

Karasek, R. A. (1979). Job demands, job decision latitude and mental strain: Implications for job redesign. *Administrative Science Quarterly, 24,* 285–308.

Karasek, R. A., Gordon, G., Pietroskovsky, C., Frese, M., Pieper, C., Schwartz, J., Fry, L., & Schirer, D. (1985). *Job content instrument: Questionnaire and user's guide.* Los Angeles/ Lowell, MA: University of Southern California/University of Massachusetts, Lowell.

Karasek, R., & Theorell, T. (1990). *Healthy work: Stress, productivity, and the reconstruction of working life.* New York: Basic Books.

Kawachi, I., & Marmot, M. (1998). What can we learn from studies of occupational class and cardiovascular disease? *American Journal of Epidemiology, 148,* 160–163.

Kawakami, N., Haratani, T., & Araki, S. (1998). Job strain and arterial blood pressure, serum cholesterol, and smoking as risk factors for coronary heart disease in Japan. *International Archives of Occupational and Environmental Health, 71*(6), 429–432.

Knox, S., Svensson, J., Waller, D., & Theorell, T. (1988). Emotional coping and the psychophysiological substrates of elevated blood pressure. *Behavioral Medicine, 2,* 52–58.

Kohn, M. L., & Schooler, C. (1982). Job conditions and personality: A longitudinal assessment of their reciprocal effects. *American Journal of Sociology, 87,* 1257–1286.

Kristensen, T. S., Kronitzer, M., & Alfedsson, L. (1998). *Social factors, work, stress and cardiovascular disease prevention.* Brussels: European Heart Network.

Kubzansky, L. D., Kawachi, I., Weiss, S., & Sparrow, D. (1998). Anxiety and coronary heart disease: A synthesis of epdemiological, psychological, and experimental evidence. *Annals of Behavioral Medicine, 20*(2), 47–58.

LaCroix, A. Z. (1984). *High demands/low control work and the incidence of CHD in the Framingham cohort.* Unpublished doctoral dissertation, University of North Carolina, Chapel Hill.

Landsbergis, P. (2000). Collective bargaining to reduce CVD risk factors in the work environment. *Occupational Medicine: State-of-the-Art Reviews, 15*(1), 287–292.

Landsbergis, P. A., Cahill, J., & Schnall, P. (1999). The impact of lean production and related new systems of work organization on worker health. *Journal of Occupational Health Psychology, 4*(2), 108–130.

Landsbergis, P. A., Schnall, P. L., Deitz, D., Friedman, R., & Pickering, T. (1992). The patterning of psychological attributes and distress by "job strain" and social support in a sample of working men. *Journal of Behavioral Medicine, 15*(4), 379–405.

Landsbergis, P. A., Schnall, P. L., Deitz, D. K., Warren, K., Pickering, T. G., & Schwartz, J. E. (1998). Job strain and health behaviors: Results of a prospective study. *American Journal of Health Promotion, 12*(4), 237–245.

Landsbergis, P. A., Schnall, P. L., Warren, K., Pickering, T. G., & Schwartz, J. E. (1994). Association between ambulatory blood pressure and alternative formulations of job strain. *Scandinavian Journal of Work, Environment and Health, 20*(5), 349–363.

Landsbergis, P. A., Schnall, P. L., Warren, K., Pickering, T. G., & Schwartz, J. E. (1999). The effect of job strain on ambulatory blood pressure in men: Does it vary by socioeconomic status? In N. E. Adler, M. Marmot, B. S. McEwen, J. Stewart (Eds.), *Socioeconomic status and health in industrial nations* (pp. 414–416). New York: Academy of Sciences.

Landsbergis, P. A., Schurman, S. J., Israel, B. A., Schnall, P. L., Hugentobler, M. K., Cahill, J., & Baker, D. (1993, Summer). Job stress and heart disease: Evidence and strategies for prevention. *New Solutions,* 42–58.

Langosch, W., Brodner, B., & Borcherding, M. (1983). Psychosocial and vocational longterm outcomes of cardiac rehabilitation with postinfarction patients under the age of forty. *Psychosomatic Medicine, 40,* 115–128.

Lennon, M. C. (1987). Sex differences in distress: The impact of gender and work roles. *Journal of Health and Social Behavior, 28,* 290–305.

Levi, L. (2000). Legislation to protect worker CV health in Europe. *Occupational Medicine: State-of-the-Art Reviews, 15*(1), 269–273.

Lewis, R. J., Huebner, W. H., & Yarborough, C. M. (1996). Characteristics of participants and nonparticipants in worksite health promotion. *American Journal of Health Promotion, 11,* 99–106.

Liao, Y., & Cooper, R. S. (1995). Continued adverse trends in coronary heart disease mortality among blacks, 1980–91. *Public Health Reports, 110,* 572–579.

Lynch, J., Krause, N., Kaplan, G. A., Salonen, R., & Salonen, J. T. (1997). Workplace demands, economic reward and progression of carotid atherosclerosis. *Circulation, 96*(1), 302–307.

Lynch, J., Krause, N., Kaplan, G. A., Tuomilehto, J., & Salonen, J. T. (1997). Work place 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.

Markovitz, J., Matthews, K. A., Kannel, W. B., Cobb, J. L., & D'Agostino, J. B. (1993). Psychological predictors of hypertension in the Framingham Study: Is there tension in hypertension? *Journal of the American Medical Association, 270*(20), 2439–2443.

Markowe, H. L., Marmot, M. G., Shipley, M. J., Bulpitt, C. H., Meade, T. W., Stirling, Y., Vickers, M. V., & Semmence, A. (1985). Fibrinogen: A possible link between social class and coronary heart disease. *British Medical Journal, 291,* 1312–1314.

Marmot, M. G., Bosma, H., Hemingway, H., Brunner, E., & Stansfeld, S. (1997). Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet, 350,* 235–239.

Marmot, M., & Theorell, T. (1991). Social class and cardiovascular disease: The contribution of work. In J. V. Johnson & G. Johansson (Eds.), *The psychosocial work environment: Work organization, democratization and health* (pp. 33–48). Amityville, NY: Baywood.

Matthews, K. A., & Haynes, S. G. (1986). Type A behavior pattern and coronary disease risk: Update and critical evaluation. *American Journal of Epidemiology, 123*(6), 923–960.

Mausner-Dorsch, H., & Eaton, W. (2000). Psychosocial work environment and depression: Epidemiologic assessment of the demand-control model. *American Journal of Public Health, 90,* 1765–1770.

McCrae, R. R. (1990). Controlling neuroticism in the measurement of stress. *Stress Medicine, 6,* 237–241.

McKinlay, J. B., & Marceau, L. D. (1999). A tale of three tails. *American Journal of Public Health, 89,* 295–298.

Melin, B., Lundberg, U., Soderlund, J., & Granqvist, M. (1999). Psychophysiological stress reactions of male and female assembly workers: A comparison between two different forms of work organization. *Journal of Organizational Behavior, 20,* 47–61.

Menotti, A., & Seccareccia, F. (1985). Physical activity at work and job responsibility as risk factors for fatal coronary heart disease and other causes of death. *Journal of Epidemiology and Community Health, 39,* 325–329.

Murphy, L. R. (1991). Job dimensions associated with severe disability due to cardiovascular disease. *Journal of Clinical Epidemiology, 44*(2), 155–166.

Murphy, L., Hurrell, J., Sauter, S., & Keita, G. E. (1995). *Job stress interventions.* Washington, DC: American Psychological Association.

Netterstrom, B., Kristensen, T. S., Damsgaard, M. T., Olsen, O., & Sjol, A. (1991). Job strain and cardiovascular risk factors: A cross-sectional study of employed Danish men and women. *British Journal of Industrial Medicine, 48*(10), 684–689.

Nowack, K. (2000). Screening and management of the workplace in relation to cardiovascular disease risk. *Occupational Medicine: State-of-the-Art Reviews, 15*(1), 231–233.

Orth-Gomer, K., Eriksson, I., Moser, V., Theorell, T., & Fredlund, P. (1994). Lipid lowering through work stress reduction. *International Journal of Behavioral Medicine, 1*(3), 204–214.

Orth-Gomer, K., Wamala, S. P., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittleman, M. A. (2000). Marital stress worsens prognosis in women with coronary heart disease. *Journal of the American Medical Association, 284*(23), 3008–3014.

Perini, C., Muller, F. B., Rauchfleisch, U., Battegay, R., Hobi, V., & Buhler, F. R. (1990). Psychosomatic factors in borderline hypertensive subjects and offspring of hypertensive parents. *Hypertension, 16,* 627–634.

Peter, R. (1997). Comparative analysis of the effort-reward imbalance model and the job strain model: Preliminary results from a Swedish case-control study. In *Socio-economic variations in cardiovascular disease in Europe: The impact of the work environment and lifestyle* (The Heart at Work Network; pp. 102–104). London: University College London, Department of Epidemiology and Public Health.

Peter, R., Alfredsson, L., Hammar, N., Siegrist, J., Theorell, T., & Westerholm, P. (1998). High effort, low reward and cardiovascular risk factors in employed Swedish men and

women: baseline results from the WOLF study. *Journal of Epidemiology and Community Health, 52,* 540–547.

Peter, R., Hallqvist, J., Reuterwall, C., Siegrist, J., Theorell, T., & the SHEEP Study Group. (1999). Psychosocial work environment and myocardial infarction: improving risk prediction by combining two complementary job stress models. (Submitted).

Peter, R., & Siegrist, J. (1997). Chronic work stress, sickness absence, and hypertension in middle managers: General or specific sociological explanations? *Social Science & Medicine, 45,* 1111–1120.

Pickering, T. G. (1991). *Ambulatory monitoring and blood pressure variability.* London: Science Press.

Rosamond, W. D., Chanbless, L. E., Folsom, A. R., Cooper, L. S., Conwill, D. E., Clegg, L., Wang, C.-H., & Heiss, G. (1998). Trends in the incidence of myocardial infarction and in mortality due to coronary heart disease, 1987 to 1994 [abstract]. *The New England Journal of Medicine, 339,* 863.

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.

Rydstedt, L. W., Johansson, G., & Evans, G. W. (1998). The human side of the road: Improving the working conditions of urban bus drivers. *Journal of Occupational Health Psychology, 3,* 161–171.

Schnall, P., & Belkić, K. (2000). Point estimates of blood pressure at the worksite. *Occupational Medicine: State-of-the-Art Reviews, 15*(1), 203–208.

Schnall, P., Belkić, K., Landsbergis, P., & Baker, D. (2001). *Occupational health psychology: Work organization and health.* http://www.workhealth.org/UCLA%20OHP%20class/UCLA%20OHP%20home%20page.html

Schnall, P., Belkić, K., Landsbergis, P. A., & Baker, D. e. (2000a). The workplace and cardiovascular disease. *Occupational Medicine: State-of-the-Art Reviews, 15*(1).

Schnall, P., Belkić, K., Landsbergis, P. A., & Baker, D. (2000b). Why the workplace and cardiovascular disease? *Occupational Medicine: State-of-the-Art Reviews, 15*(1), 1–5.

Schnall, P. L., & Kern, R. (1981). Hypertension in American society: An introduction to historical materialist epidemiology. In P. Conrad & R. Kern (Eds.), *The sociology of health and illness: Critical perspectives* (pp. 97–122). New York: St. Martin's Press.

Schnall, P. L., Landsbergis, P. A., & Baker, D. (1994). Job strain and cardiovascular disease. *Annual Review of Public Health, 15,* 381–411.

Schnall, P. L., Landsbergis, P. A., Schwartz, J., Warren, K., & Pickering, T. G. (1998). A longitudinal study of job strain and ambulatory blood pressure: Results from a three-year follow-up. *Psychosomatic Medicine, 60,* 697–706.

Schnall, P. L., Pieper, C., Schwartz, J. E., Karasek, R. A., Schlussel, Y., Devereux, R. B., Ganau, A., Alderman, M., Warren, K., & Pickering, T. G. (1990). The relationship between "job strain," workplace diastolic blood pressure, and left ventricular mass index. Results of a case-control study [published erratum appears in JAMA 1992 Mar 4 267(9):1209]. *Journal of the American Medical Association, 263*(14), 1929–1935.

Schnall, P. L., Schwartz, J. E., Landsbergis, P. A., Warren, K., & Pickering, T. G. (1992). Relation between job strain, alcohol, and ambulatory blood pressure. *Hypertension, 19,* 488–494.

Schwartz, J., Belkić, K., Schnall, P., & Pickering, T. (2000). Mechanisms leading to hypertension and CV morbidity. *Occupational Medicine: State of the Art Reviews, 15*(1), 121–132.

Shapiro, A. P. (1988). Psychological factors in hypertension: An overview. *American Heart Journal, 116,* 632–637.

Shimomitsu, T., & Odagiri, Y. (2000). Working life in Japan. *Occupational Medicine: State of the Art Reviews, 15*(1), 280–281.

Siegrist, J. (1996). Adverse health effects of high-effort/low-reward conditions. *Journal of Occupational Health Psychology, 1,* 27–41.

Siegrist, J., Matschinger, H., Cremer, P., & Seidel, D. (1988). Atherogenic risk in men suffering from occupational stress. *Atherosclerosis, 69,* 211–218.

Siegrist, J., & Peter, R. (1996). *Measuring effort-reward imbalance at work: Guidelines.* Dusseldorf: University of Dusseldorf.

Siegrist, J., Peter, R., Georg, W., Cremer, P., & Seidel, D. (1991). Psychosocial and biobehavioral characteristics of hypertensive men with elevated atherogenic lipids. *Atherosclerosis, 86,* 211–218.

Siegrist, J., Peter, R., Junge, A., Cremer, P., & Seidel, D. (1990). Low status control, high effort at work and ischaemic heart disease: Prospective evidence from blue collar men. *Social Science and Medicine, 31,* 1127–1134.

Smith, M., & Zehel, D. (1992). A stress reduction intervention programme for meat processors emphasizing job design and work organization. *Conditions of Work Digest, 11*(2), 204–213.

Smith, T. W. (1992). Hostility and health: Current status of a psychosomatic hypothesis. *Health Psychology, 11,* 139–150.

Sokejima, S., & Kagamimori, S. (1998). Working hours as a risk factor for acute myocardial infarction in Japan: Case-control study. *British Medical Journal, 317,* 775–780.

Sorensen, G., Himmelstein, J. S., Hunt, M. K., Youngstrom, R., Hebert, J. R., Hammond, S. K., Palombo, R., Stoddard, A., & Ockene, J. K. (1995). A model for worksite cancer prevention: integration of health protection and health promotion in the WellWorks Project. *American Journal of Health Promotion, 10*(1), 55–62.

Sorensen, G., Stoddard, A., Ockene, J. K., Hunt, M. K., & Youngstrom, R. (1996). Worker participation in an integrated health promotion/health protection program: Results from the WellWorks Project. *Health Education Quarterly, 23,* 191–203.

Sparrow, D., Garvey, A. J., Rosner, B., & Thomas, H. E. (1982). Factors in predicting blood pressure change. *Circulation, 65,* 789–794.

Stamler, J. (1991). Blood pressure and high blood pressure: Aspects of risk. *Hypertension, 18*(Suppl. 1), 95–107.

Stansfeld, S. A., North, F. M., White, I., & Marmot, M. G. (1995). Work characteristics and psychiatric disorder in civil servants in London. *Journal of Epidemiology and Community Health, 49,* 48–53.

Steenland, K. (2000). Shift work, long hours, and CVD: A review. *Occupational Medicine: State-of-the-Art Reviews, 15*(1), 7–17.

Steptoe, A. (1981). *Psychological factors in cardiovascular disorders.* London: Academic Press.

Steptoe, A., & Marmot, M. (2000). Atherogenesis, coagulation and stress mechanisms. *Occupational Medicine: State of the Art Reviews, 15*(1), 136–138.

Stokols, D., Pelletier, K. R., & Fielding, J. E. (1995). Integration of medical care and worksite health promotion. *Journal of the American Medical Association, 273*(14), 1136–1142.

Theorell, T. (1990). Family history of hypertension—An individual trait interacting with spontaneously occurring job stressors. *Scandinavian Journal of Work, Environment and Health, 16*(Suppl. 1), 74–79.

Theorell, T. (2000). Neuroendocrine mechanisms. *Occupational Medicine: State of the Art Reviews, 15*(1), 139–146.

Theorell, T., Perski, A., Akerstedt, T., Sigala, F., Ahlberg-Hulten, G., Svensson, J., & Eneroth, P. (1988). Changes in job strain in relation to changes in physiological states—A longitudinal study. *Scandinavian Journal of Work, Environment and Health, 14,* 189–196.

Theorell, T., Perski, A., Orth-Gomer, K., Hamsten, A., & de Faire, U. (1991). The effects of the strain of returning to work on the risk of cardiac death after a first myocardial infarction before age 45. *International Journal of Cardiology, 30,* 61–67.

Theorell, T., Tsutsumi, A., Hallqvist, J., Reuterwall, C., Hogstedt, C., Fredlund, P., Emlund, N., Johnson, J., & the SHEEP Study Group. (1998). Decision latitude, job strain and myocardical infarction: A study of working men in Stockholm. *American Journal of Public Health, 88,* 382–388.

The Tokyo Declaration. (1998). *Journal of the Tokyo Medical University, 56*(6), 760–767.

Tuchsen, F. (1993). Working hours and ischaemic heart disease in Danish men: A 4-year cohort study of hospitalization. *International Journal of Epidemiology, 22,* 215–221.

Tuchsen, F., & Endahl, L. A. (1999). Increasing inequality in ischaemic heart disease morbidity among employed men in Denmark 1981–1993: The need for a new preventive policy. *International Journal of Epidemiology, 28,* 640–644.

U.S. Departments of Labor and Commerce. (1994). *Fact Finding Report. Commission on the Future of Worker-Management Relations.* Washington, DC: Author.

Verdecchia, P., Clement, D., Fagard, R., Palatini, P., & Parati, G. (1999). Task force III: Target-organ damage, morbidity and mortality. *Blood Pressure Monitoring, 4,* 303–317.

Waldron, I., Nowatarski, M., Freimer, M., Henry, J. P., Post, N., & Witten, C. (1982). Cross-cultural variation in blood pressure: A qualitative analysis of the relationship of blood pressure to cultural characteristics, salt consumption and body weight. *Social Science and Medicine, 16,* 419–430.

Warren, N. (2000). U.S. regulations for work organization. *Occupational Medicine: State-of-the-Art Reviews, 15*(1), 275–280.

Whelton, P. K., He, J., & Klag, M. J. (1994). *Blood pressure in Westernized populations.* In J. D. Swales (Ed.), *Textbook of hypertension* (pp. 11–21). London: Blackwell Scientific.

Willich, S. N., Maclure, M., Mittleman, M., Arntz, H. R., & Muller, J. E. (1993). Sudden cardiac death support for a role of triggering in causation. *Circulation, 87,* 1442–1450.

Wilson, P. W. F., D'Aostino, R. B., Levy, D., Castelli, W. P., Belanger, A. J., Sytkowski, P. A., & Kannel, W. B. (1991, Nov. 11–14). *Trends in coronary heart disease: A comparison of the original (1956–1968) and offspring Framingham study cohorts.* Paper presented at the American Heart Association, Anaheim, CA.

Wing, S., Casper, M., Riggan, W., Hayes, C. G., & Tyroler, H. A. (1988). Socioenvironmental characteristics associated with the onset of decline of IHD mortality in US. *American Journal of Public Health, 78,* 923–926.

Wolf, S. (2000). The environment-brain-heart connection. *Occupational Medicine: State of the Art Reviews, 15*(1), 107–109.

Wolff, E. (1995). *Top heavy: A study of wealth inequality in America.* New York: Twentieth Century Fund Press.

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