

The Effects of Job Strain on Risk Factors for Cardiovascular Disease

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### **The Objectives and Scope of the Review**

In our review, we focus on work-related psychological stress. Within this stress research domain, we exclude from our review event-based types of stress – including acute and critical job events such as being demoted or going on involuntary vacation (Eden, 1982, 1990). We also exclude work-related hassles, such as being caught in a traffic jam while commuting to work (cf. Gajendran & Harrison, 2007). It follows that we focus on chronic stress at work. Researchers may disagree on the conceptual definition of work-related chronic stress (Cooper, 1998; Monroe, 2008). There is basic agreement, however, about the notion that work-related chronic stress, hereafter referred to simply as stress, may be implicated in cardiovascular disease risk factors, specifically physiological ones, such as elevated cholesterol and blood pressure levels, and in certain maladaptive behavioral responses (Aboa-Eboule et al., 2007; Chandola et al., 2008; Chandola, Brunner, & Marmot, 2006; Williams, 2008).

We decided to focus on the effects of work-related stress on risk factors for cardiovascular disease (CVD). We define *cardiovascular disease* (CVD) as a composite of coronary heart disease, stroke, and cardiovascular mortality. This focus is due to the fact that CVD, including myocardial infarction (MI) and stroke, is a principal cause of death in most economically advanced countries; it is associated with multiple physiological, psychological, and socio-demographic risk factors that often interact in complex causal paths (Brotman, Golden, & Wittstein, 2007; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Williams, 2008). Chronic stress could be directly implicated in CVD by causing spasms of coronary blood vessels,

electrical instability in the heart conduction system, and abnormal heart rhythms (cf. Williams, 2008). Chronic stresses are thought to influence the pathogenesis of CVD by causing negative affective states such as burnout (Melamed, Shirom, Toker, Berliner, & Shapira, 2006), and anxiety and depression (Suls & Bunde, 2005), which in turn exert direct effects on maladaptive behavioral and physiological responses. There are two major physiological mechanisms that are considered as the most likely mediators of the effects of chronic stress on CVD: the hypothalamic-pituitary-adrenocortical axis (HPA) and the sympathetic-adrenal-medullary (SAM) system (Miller, Chen, & Zhou, 2007). Prolonged or repeated activation of the HPA and SAM axes can interfere with their control of other physiological systems, and could result in increased risk for a variety of physical and psychiatric disorders (McEwen, 2007). Cortisol, the primary endocrine response of the HPA axis, regulates a broad range of physiological processes, including the metabolism of fats and proteins represented in our review by blood lipids, and anti-inflammatory responses, represented in our review by biomarkers of micro-inflammation in the serum. SAM axis activation is associated with the secretion of catecholamines, which – interacting with the autonomic nervous system – exert regulatory effects on many organ systems in the body, including the cardiovascular system (Cohen, Janicki-Deverts, & Miller, 2007). SAM axis activation is represented in our review by both blood pressure and sleep disturbances.

The chapter begins by describing a general theoretical perspective within which our review is embedded. The general theoretical perspective provides a comprehensive, system-based view of the antecedents of stress- CVD risk factors linkages. We then briefly review three leading theoretical models that have been used to investigate stress-CVD risk factors associations: the Person-Environment Fit model, the Effort-Reward model, and the Job Demand-

Control-Support model (JDC-S) model, also referred to as the Job Strain model (hence the term *job strain* in the title). We explain why we chose to focus, in the following sections of our review, on the JDC-S model. We then use the JDC-S model to present what is known about the effects of work-related stress on four major risk factors for CVD: elevated levels of blood lipids, blood pressure, and micro-inflammation biomarkers, and sleep disturbances. These risk factors represent only a subset of possible physiological and behavioral strains that may be impacted by work-related stress. For example, the effects of stress may include alterations in neuroendocrine factors, the autonomic nervous system, and immune functions. The concluding section discusses the limitations of this review and highlights promising avenues for future research in this field.

Above we explained several physiological considerations that guided the choice of the CVD risk factors covered here. There were additional considerations, related to the availability of empirical studies and the contents of other chapters in this volume. This review focuses on empirical studies; therefore, a body of such studies should exist. Additionally, an explicit attempt was made to avoid duplication with other chapters of this volume, including chapters that specifically cover the maladaptive health responses of psychological distress, drug abuse, and alcoholism. The broad scope of this review necessarily limits the depth of the presentation. Readers should note that the range of the literature covered probably reflects the author's personal viewpoints on several key issues.

### **A General Framework for the Study of the Health Consequences of Stress at Work**

The theoretical model guiding this chapter is represented in Figure 1. Within the model, an individual's state of health is viewed as being determined by multiple factors, including heredity, environment, early background, and socioeconomic influences. This theoretical model

draws on earlier conceptualizations (Macik-Frey, Quick, & Nelson, 2007; Quick, Quick, Nelson, & Hurrell, 1997, pp. 65-89). Among the multiple causal chains leading to maladaptive health responses is the effect of work-related stress. This effect is depicted as being moderated by individuals' coping resources and personality factors. To simplify the presentation of the main effects, several arrows indicating moderating effects were omitted from Figure 1. We focus on the hypothesized arrow that leads from work-related chronic stress to maladaptive physiological and behavioral responses, primarily because of the considerations detailed above. Stress is posited in Figure 1 as precipitating the development of maladaptive health responses, like when it is implicated in raising a person's blood pressure from normal to borderline. The following is a brief discussion of the theoretical model presented in Figure 1. It is introduced by a description of the conceptual approach followed by the definitions of stress and maladaptive health responses.

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Insert Fig.1 about here

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Early reviews of the vast area of work-related stress and physical health (Danna & Griffin, 1999; Ganster & Schaubroeck, 1991; Mackay & Cooper, 1987) mostly followed the theoretical framework depicted in Figure 1, defining health and well-being broadly to include psychological and physical health. By *maladaptive health responses*, we refer to a subclass of what has been labeled *strain* in the Michigan model, namely any deviation from the normal state of responses of the person (French, Caplan, & Harrison, 1982). This definition of strain included psychological strain, such as job dissatisfaction and anxiety, physiological strain like high blood pressure, and behavioral symptoms of strain such as sleep disturbances. Continuing high levels

of strain were postulated to affect morbidity and mortality levels (French et al., 1982). In this review, we refer only to the latter two types of strain.

There are several reasons for introducing the model depicted in Figure 1 in this chapter. First, as argued by several researchers (Kasl, 1996; Marmot, Theorell, & Siegrist, 2002), studies of the relationships between stress and maladaptive health responses need to maintain a broad conceptual perspective of the etiology of these responses. Specific etiological factors leading from the work environment to health responses are embedded in a complex matrix of additional psychosocial influences. There are several classes of variables that were included in Figure 1, but were not discussed or reviewed here because of space limitations. The potential usefulness of each of those panels needs to be considered by future researchers. Salient examples are socioeconomic indicators (cf. Gallo & Matthews, 2003), stable individual differences (cf. Smith, Glazer, Ruiz, & Gallo, 2004; Smith & MacKenzie, 2006), and work role and work environment characteristics that represent individuals' exposures to earlier work and job experiences (Theorell, 1998).

Figure 1 depicts several bi-directional arrows. These double-headed arrows represent interactions or non-recursive processes between panels of variables. To illustrate, the bi-directional arrow between psychological and physiological maladaptive responses represents reciprocal feedback loops that can occur, as when distress - such as depression or burnout - affects the immune system (cf. Melamed, Shirom, Toker, Berliner, & Shapira, 2006). Again, given the confines of this review, it was not possible to discuss each double-headed arrow in detail.

The role of panels not discussed in our review could be illustrated by taking as an example the role of socioeconomic disadvantage, considered to have direct and indirect influences on maladaptive health responses. Decades of research have shown that socioeconomic status is a significant predictor of stress, strain, and state of health (Banks, Marmot, Oldfield, & Smith, 2006; Hemingway & Marmot, 1999; Marmot, 2006). Socioeconomic status differences are found for rates of morbidity and mortality for almost every disease and health condition (Adler et al., 1994). Components of socioeconomic status, income, education and occupation shape individuals' early life experiences, including early-age health habits like diet and exercise, and significantly influence their work experiences, including access to coping resources such as social support at work (cf. Danna & Griffin, 1999).

Researchers have often posited a strong relationship between perceived stress - an individual's coping resources and coping mechanisms - and the etiology of stress-related maladaptive health responses (Lazarus, 1999; Taylor & Stanton, 2007). How an individual handles stress plays an important role in determining the health outcomes of the individual's encounter with stress. Coping may be loosely defined as things we think and actions we take to ameliorate or remove the negative aspects of stressful situations, including indirect coping like avoidance (Taylor & Stanton, 2007). The ability to cope with stress is represented in Figure 1 by the panel of work-related coping resources. These resources interact with individuals' subjective appraisal to determine their experienced stress. If a situation is not appraised as taxing or exceeding one's coping resources, it is not likely to be experienced as stress (Lazarus, 1999). Personality factors like hardiness represent additional coping resources. Because of space limitations, this chapter does not cover the issue of effective coping mechanisms, which may

prevent psychosocial and physiological disequilibria that may in turn lead to stress-related illnesses.

Adaptive and maladaptive responses to stress represent a complex set of an organism's reactions intended to reestablish psychosocial and physiological equilibriums. As indicated, we focus only on a specific set of risk factors for CVD. The hypothesized effects of stress may appear in any combination of the physiological, behavioral, and psychological domains of strain. To illustrate, high blood pressure, sleep disturbances and high levels of "bad" serum cholesterol and obesity often co-occur. The synergic relationships among the panels of Figure 1 indicate that there is not any single consistent maladaptive health response applicable to most people in all work situations. This basic premise of inter-individual variability in stress response is related to the direct and indirect effects of coping resources and coping effectiveness considered above, and in addition to other individual difference variables depicted in other panels of Figure 1.

### **Models Explaining the Effects of Work Stress on Physiological Risk Factors**

In recent years, occupational health researchers have devoted considerable attention to possible paths of influence linking work and job characteristics with employees' physical health. They have been guided in their attempts to gain additional understanding of the pathways linking the world of work and employee physical health by several important and often used models. Each of these models represents a distinct way of reducing the complex reality into a comprehensive yet parsimonious model. Each of these models focuses on specific core elements in order to explain work-related physical health. One of the most important models is referred to as the Job Demands-Control-Support (JDC-S) model; for reasons explained below, we focus on this model in our review. However, we also briefly review in this section two additional models



that have been used to explain the effects of chronic work-related stress on the risk factors for CVD considered in our review. We describe the two other models largely because they are based on similar theoretical principles and therefore could be combined with the JDC-S model in future research explaining the pathways linking work-related stress with risk factors for CVD.

#### *The Person-Environment Fit Model*

One of the earliest (French & Caplan, 1973) models focusing on stress and health outcomes is the Person-Environment Fit model (P-E Fit). It has been widely applied to predict a variety of risk factors for disease (Edwards, Cable, Williamson, Lambert, & Shipp, 2006; Edwards, Caplan, & Van Harrison, 1998), including those that we focus on (cf. French, Caplan, & Harrison, 1982). *Fit*, in this model, includes the relationships between environmental supplies and individuals' values and needs, referred to as the S-V (supplies-values) Fit, and the relationships among environmental demands and individuals' abilities, skills and knowledge, referred to as the D-A (demands-abilities) Fit. The model postulates that the more pronounced a misfit, either S-V or D-A, the higher the level of the resulting strains will be. An additional postulate of the model is that the major components of S-V, and in turn also D-A, interact to influence one's level of strain (Edwards et al., 1998). Many additional types of Person-Environment Fit that could conceptually exist were described in a major conceptual review of the area (Kristof, 1996). Kristof (1996) pointed out that the type of fit mainly investigated up till now has been the D-A rather than the S-V. Several other approaches to the study of stress-strain relationships have also incorporated elements of this model, such as Cybernetic Stress theory (Edwards, 1998).

A recent meta-analysis of the P-E Fit literature at large, including studies that followed

the specific P-E Fit model described above (Kristof-Brown, Zimmerman, & Johnson, 2005), concluded that fit was strongly associated with several attitudinal and behavioral outcomes. For example, Person-Job Fit was found to be strongly linked with job satisfaction, job performance, and turnover, while Person-Organization Fit was closely associated with organizational commitment. However, the number of studies linking the model with physical health related outcomes was found to be small (Schnall, Landsbergis, & Baker, 1994), and therefore this was not the model chosen to guide our review.

#### *Effort-Reward Imbalance at Work Model*

The Effort-Reward Imbalance (ERI) model (Siegrist, 1995) builds upon the notion of social reciprocity, a fundamental principle of interpersonal behavior which lies at the core of employment relationships. In the context of the ERI, social reciprocity is interpreted as representing the norm of return expectancy. *Return expectancy* refers to employees' expectations that the effort they invest at work would be equal to the rewards they receive. *Efforts*, in the context of ERI, represent job demands and requirements that are imposed on the employee. *Rewards*, in turn, refer to money, job security, self-esteem, and career opportunities, mostly distributed by the employer (but also by society at large). Reward in the ERI model is probably closely related to the notion of supplies in the P-E Fit model, while efforts resemble the notion of demands in the P-E Fit model. Therefore, it could be argued that the ERI model is embedded in the PE-Fit model. A job situation characterized by high efforts and low rewards represents a reciprocity deficit. Perceived lack of reciprocity is hypothesized to lead to strong negative emotions. These negative emotions, in turn, lead to sustained autonomic and endocrine activation and to negative health outcomes (Ursin & Eriksen, 2007). The wider the discrepancy between the

costs incurred by employees, in terms of their efforts invested at work to face work-related demands; and their gains, in terms of the rewards they receive, the stronger the psychological strain reaction and the higher the likelihood that the employees concerned will develop maladaptive physiological responses.

The reciprocity norm is almost never fully explicated in employment contracts. Therefore, formal employment contracts are supplemented by mutual trust and informal understandings and commitments. Lack of trust reinforces and augments the effort-reward imbalance. Certain personality characteristics aggravate the imbalance once it exists. For example, intolerance of ambiguity may lead to exaggerated appraisals of uncertainties associated with rewards. Besides efforts and rewards, the model includes a third factor, referred to as *over-commitment* or *intrinsically-motivated investment of efforts at work*. The model predicts that over-committed employees are at high risk to experience efforts-rewards imbalance, relative to their under-committed colleagues. Additionally, highly overcommitted employees experiencing imbalance will respond with more strain reactions to a reciprocity deficit, in comparison with less overcommitted employees. This interactional hypothesis is often referred to as the "intrinsic" ERI hypothesis (van Vegchel, de Jonge, Bosma, & Schaufeli, 2005).

In recent years, several qualitative reviews integrated and evaluated the many studies that had applied the ERI model to explain physical health outcomes (Tsutsumi & Kawakami, 2004; van Vegchel, de Jonge, Bosma, & Schaufeli, 2005). Generally, these qualitative reviews found that the "extrinsic" ERI hypothesis - that is, the hypothesis that high efforts in combination with low rewards increase the risk of poor health - has gained considerable empirical support. However, support for the "intrinsic" ERI remained inconclusive. The most recent review of the

ERI model (van Gegechel et al., 2005) found that 13 out of 17 studies supported the model in that employees reporting a high effort-low reward imbalance had higher levels of CVD risk factors, including blood lipids and blood pressure. However, less than half of the 17 studies used a prospective design. As argued by Tsutsumi and Kawakami (2004), the ERI model and the Job-Demand-Control-Support (JDC-S) model, reviewed in the next section, are complementary. We decided to focus on the latter, rather than the former, primarily because of the fact that the preponderance of studies linking CVD risk factors with job characteristics used the JDC-S model (for an early review, see: Schnall, Landsbergis, & Baker, 1994).

#### *The Job Demand-Control-Support Model*

Work-related stressors have been increasingly studied for their potential adverse effects on cardiovascular risk factors (Brotman, Golden, & Wittstein, 2007). A leading theoretical model in studying the effects of job characteristics on physical health is the Job-Demand-Control-Support (JDC-S) model, developed by Karasek and Theorell (1990). In the initial formulation of the model, Karasek (1979) identified two crucial job aspects in the work situation which are expected to be associated with a number of health outcomes: job demands and job control. *Job demands* usually refers to psychological job demands, primarily defined as referring to perceived workload, while *job control* refers to the freedom permitted the worker in deciding how to meet demands or how to perform tasks (Karasek & Theorell, 1990). Based on empirical research, Johnson and his colleagues (Johnson & Hall, 1988; Johnson, Stewart, Hall, Fredlund, & Theorell, 1996) extended the initial model to include the dimension of "workplace social support"; this extended model was termed the Demand-Control-Support (JDC-S) model (Karasek & Theorell, 1990). *Workplace social support* refers to "overall levels of helpful social

interaction available on the job from both co-workers and supervisors" (Karasek & Theorell, 1990, p. 69). Two major hypotheses were derived from the JDC-S model. The first, often referred to as the additive hypothesis, postulates that the model's components have additive effects on strain. The second hypothesis, dubbed as the interactional hypothesis, maintains that the most unfavorable and potentially stressful working environment, and the highest risk of poor psychological well-being and ill-health, occurs in the high '*iso-strain*' condition characterized by high job demands, low control, and low social support (Karasek & Theorell, 1990). The first hypothesis is the simplest and has been supported by most longitudinal studies testing the effects of the model's components on strain reactions (de Lange, Taris, Houtman, & Bongers, 2003). The second hypothesis received considerably less support and was often tested using inappropriate statistical procedures (Kasl, 1996; Sargent & Terry, 2000). Therefore, we focus below on the model's first hypothesis.

The JDC-S model, much like the ERI model, could be regarded as embedded in the PE-Fit model. Previous reviews (Van Der Doef & Maes, 1998, 1999) found the JDC-S model to have predictive powers relative to both psychological and physiological strain. More recent qualitative reviews of the research on the JDC-S model (Belkic, Landsbergis, Schnall, & Baker, 2004; Steenland et al., 2000) found that it predicted the prevalence and incidence of cardiovascular disease (CVD). A recent meta-analytic study summarized the results of 14 prospective cohort studies that quantitatively estimated the prediction of CVD by the JDC-S model (Kivimaki et al., 2006). As reported by Kivimaki et al. (2006), they found that the highest incidence of cardiovascular morbidity and mortality in these studies occurred when individuals'

jobs were characterized by high job demands, low amounts of employee control with which to cope with these demands, and low levels of social support.

Researchers have suggested several specific pathways to explain the association between the JDC-S model and CVD. These mediating physiological mechanisms include excessive activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis (Miller, Chen, & Zhou, 2007), altered autonomic regulation of the heart (Belkic et al., 2004), and damaging health behaviors, including smoking, lack of physical activity, and high calorie intake (Van der Doef & Maes, 1999). The precise biological mechanisms underlying the associations of the JDC-S model with CVD remain unclear (Belkic et al., 2004). Therefore, in the following sections we will present evidence linking the model with several CVD risk factors.

### **The JDC-S Model and Blood Lipids**

We will first investigate the extent to which the model's components predict subsequent levels of blood lipids. We shall use the term *blood lipids* to refer to both lipids and lipoproteins. The physiological pathways linking exposure to chronic stress and elevated blood lipids have not yet been established, but they probably involve the mediation of the sympathetic nervous system and the HPA axis. Increased HPA activity during stress typically results in the secretion of catecholamines, cortisol, and glucagon, which in turn cause lipolysis and the subsequent release of fatty acids into the circulation (Stoney, Bausserman, Niaura, Marcus, & Flynn, 1999; Stoney, Niaura, Bausserman, & Matacin, 1999). There is strong evidence linking work-related chronic stressors with atherogenic lipids, but there is less evidence that lipids are immediately responsive to elevations of chronic stressors (Niaura, Stoney, & Herbert, 1992). The blood lipids investigated in this area of research were high-density lipoprotein cholesterol (HDL-C), high

values of which are considered to be protective against CVD, and also low-density lipoprotein cholesterol (LDL-C) and triglycerides (TRI) - high values of which are causally implicated in the etiology of CVD (Tirosh et al., 2007).

We conducted a search of English-language articles (i.e., excluding conference papers and doctoral dissertations) published between 1980 and 2007 that link the JDC-S model or its earlier variant, the JD-C model, with blood lipids at the individual level of analysis. We found 14 such studies (a table summarizing these studies is available from the first author upon request), all of which used a cross-sectional design, and therefore could not rule out the possibility that subclinical CVD - as indicated by high levels of LDL-C and TRI and low levels of HDL-C - influenced the components of the JDC-S model, rather than the reverse. The authors of nine studies (64%) reported that they found little or no support for the model's predictions. The remaining five studies provided only mixed and inconsistent support for the model's predictions.

Niaura et al. (1992), in their qualitative reviews of the literature on this subject, concluded that there was some evidence, albeit inconsistent, implicating objective or perceived stress as a source of elevated blood concentrations of lipids, particularly those lipid fractions that are most atherogenic. The qualitative review of 14 cross-sectional studies that we conducted provided mixed and inconsistent support for the expected link between the JDC-S model and blood lipids. Past research has been primarily concerned with episodic, or event-based, stress (Niaura et al., 1992). Event-based and ongoing, chronic exposure-based conceptualizations of stress derive from differing theoretical approaches (Derogatis & Coons, 1993), and have often been found to be differently related to physiological risk factors in coronary heart disease (Kahn,

Wolfe, Quinn, Snoek, & Rosenthal, 1964). In their research report, Stoney et al. (1999) provided a summary of an unpublished meta-analytic study of the literature on stress and blood lipid concentrations. A total of 101 studies were included in this meta-analysis, and each study was separately analyzed according to whether the stress was chronic (lasted more than 30 days), episodic (demands addressed during a period of one to thirty days) or acute (lasting no more than 24 hours). Acute and episodic stress relationships with lipids were both found to have positive effect sizes on several lipids' parameters. In comparison, chronic stress and total cholesterol associations resulted in a small positive effect size, but none of the other lipid parameters provided a significant effect size with chronic stress. Stoney et al. (1999) concluded that the evidence for a connection between acute and episodic stress on the one hand, and lipid reactivity on the other, is generally more consistent than the evidence for a connection between chronic stress and lipid reactivity. It should be noted, however, that these meta-analytic results might stem in part from the arbitrariness of the stress classification criteria, and from the small number of studies available on chronic stress and lipid parameters.

### **The JDC-S Model and Blood Pressure**

Blood pressure and other biological parameters, such as catecholamines, continuously fluctuate in response to changes in the external or internal environment, to facilitate the adaptation of individuals to their environments (James & Brown, 1997). Researchers and clinicians label the maximal pressure of the pulse of blood expelled by the heart's left ventricle during contraction into the aorta as *systolic blood pressure*; the minimal pressure, exerted when the heart is at rest just before the next heartbeat, is labeled *diastolic blood pressure*. Arterial blood pressure may change substantially within seconds, in response to the physiological state



and environmental conditions of the individual. Therefore, researchers tend to use non-invasive ambulatory monitors that can measure blood pressure response many times during daily life while the subjects go about their normal activities (Pickering, Shimbo, & Haas, 2006); we refer to the results of these types of blood pressure measurements as *ambulatory blood pressure*. The other type of blood pressure measurement is referred to as *causal blood pressure*; it is usually undertaken while the examinee is sitting in the clinic and represents the average of several consecutive measurements of systolic and diastolic blood pressures.

The etiology of elevated blood pressure remains unknown, but it is well accepted that multiple factors are responsible for this CVD risk factor (Pickering et al. 2006). Acute diastolic blood pressure reactivity to various stresses has been prospectively linked to increased incidence of cardiovascular disorders, including coronary heart disease, stroke and renal disease (e.g., Fredrikson & Matthews, 1990); however, the conceptual and empirical differences between acute and chronic stresses have already been discussed in the section on blood lipids. The growing interest in the effects of work-related stress on blood pressure is explained by the consistent finding that blood pressure measured at work is higher than all other measures of blood pressure taken during the day, independent of the time of day (James & Brown, 1997).

Chronic exposure to job-related demands may be associated with increased physical activity and changes in posture. These changes in activity level may in turn give rise to elevated levels of blood pressure. In a series of early longitudinal studies designed to test the effects of the JDC-S model on ambulatory blood pressure, the combination of low control (low decision latitude) and high demand (high workload) predicted elevated levels of blood pressure at work, at home, and during sleep (for a review of the early studies, see: Schwartz, Pickering, &

Landsbergis, 1996). This consistent finding has focused researchers' attention on the pivotal role of perceived job control as a powerful moderator of the effects of work-related stress on elevated blood pressure levels. Subsequent attempts to replicate this finding in longitudinal studies using ambulatory blood pressure yielded mixed results, with one study supporting the JDC-S model (Landsbergis, Schnall, Pickering, Warren, & Schwartz, 2003), and another study failing to find support for the model's expectations (Fauvel et al., 2003). Considering only longitudinal studies that used the JDC-S model to predict future causal blood pressure, there is a relative preponderance of studies that found some support for the model's major hypotheses (Guimont et al., 2006; Markovitz, Matthews, Whooley, Lewis, & Greenlund, 2004; Ohlin, Berglund, Rosvall, & Nilsson, 2007). The carefully conducted study of the effects of task (job) strain on ambulatory blood pressure (T.W. Kamarack et al., 1998) did report a main effect of decisional control: Situations rated in this study as high on control were associated with lower levels of diastolic blood pressure activity, suggesting that control may protect against acute sympathetic activation. In yet another carefully crafted study using ecological momentary assessment, Kamarack et al. (2002), found that the JDC-S model has predictive value with ambulatory blood pressure, again adding to the body of evidence that psychological demands are – independently of possible confounders – associated with ambulatory blood pressure fluctuations during daily work life.

### **The JDC-S Model and Micro-Inflammation Biomarkers**

Several recent reviews of the literature have concluded that repeated episodes of acute psychological stress or chronic psychological stress can lead to a chronic inflammatory process (Black, 2002, 2003; Black & Garbutt, 2002). Accumulated evidence indicates that the inflammatory proteins fibrinogen and C-reactive protein, as well as the white blood cell count

(WBC) were found to be independent predictors of CVD incidence (Madjid, Awan, Willerson, & Casscells, 2004; Mora, Rifai, Buring, & Ridker, 2006). Studies in healthy individuals exposed to chronic psychological stress have shown that they exhibited increased circulating concentrations of fibrinogen, CRP and WBC (Clays et al., 2005). Based on past research, we evaluated the possibility that CRP, WBC and fibrinogen plasma concentrations could provide the mechanism linking the JDC-S model with CVD.

We were unable to find any past study that has related the JDC-S model to WBC. Three cross-sectional studies related the JDC-S model to CRP, reporting no association (Clays et al., 2005; Hemingway et al., 2003) and a significant positive association (Schnorpfeil et al., 2003). The JDC-S model has been used to predict fibrinogen in several past studies. With one exception (Riese, Van Doornen, Houtman, & De Geus, 2000), all other past studies relating the JDC-S model to fibrinogen were cross-sectional and most of them did not support the model (for a summary of the studies and references, see: Shirom, Toker, Berliner, & Shapira, 2008) In our recent study (Shirom et al., 2008) assessing the impact of the JDC-S model on the above inflammation biomarkers over a period of 18 months, we also failed to find support for the JDC-S model.

### **The JDC-S Model and Sleep Problems**

The modern era of sleep research began in the 1950s with the discovery that sleep is a highly active state, rather than a passive condition of non-response. The most prevalent type of sleep disturbance, insomnia, may occur in a transient, short-term or chronic form. Stress is probably the most frequent cause of transient insomnia (Gillin & Byerley, 1990). Chronic

insomnia could result from an underlying medical or psychiatric disorder (cf. Gillin & Byerley, 1990)

*Insomnia* is defined as difficulties in initiating sleep or maintaining sleep, prolonged awakenings during the night, or waking up too early in the morning for more than a one-month period (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006; Gillin & Byerley, 1990). Increasing evidence indicates that insomnia leads to fundamental impairments in quality of life and functional capacity, and represents a substantial economic burden (T. Roth & Ancoli-Israel, 1999; Walsh & Engelhardt, 1999; Zammit, Weiner, Damato, & al., 1999). Insomnia has been linked to daytime fatigue, greater medical service utilization, self-medication with alcohol or over-the-counter medication, greater functional impairment, greater work absenteeism, impaired concentration and memory, decreased enjoyment of interpersonal relationships, and increased risk of serious medical illness and traffic and work accidents (Roth & Roehrs, 2003; Thomas Roth, Roehrs, & Pies, 2007). Insomnia can be viewed as an inability to recover and replenish depleted resources after exposure to stress. This may result in a state of physiological and cognitive hyperarousal (Thomas Roth, Roehrs, & Pies, 2007).

The association between the JDC-S model and insomnia may be maintained through a vicious circle where stress at work evokes physical and cognitive hyperarousal; this disturbs sleep, which in turn reduces the ability to renew coping resources (represented by perceived control and social support), and in turn increases the feeling of stress. Additionally, high levels of physiological tension, such as heart rate and muscle activity, may make it more difficult to relax. Psychosocial factors at work may also be a fundamental source for cognitive arousal, manifested by disturbing thoughts that become intrusive when a person attempts to sleep. Thus, while

attempting to relax and fall asleep, thoughts about stressful situations at work may be a source of rumination, disrupt relaxation, and create arousal which induces difficulties in falling asleep.

Akerstedt (2006) recently reviewed cross-sectional studies on the relationship between the JDC -S model and insomnia. This review (Akerstedt, 2006) concluded that most of these studies tend to support the model's predictions. Several cross-sectional studies that followed the above review (Akerstedt, Kecklund, & Gillberg, in press; Knudsen, Ducharme, & Roman, 2007) were also supportive of the JDC-S model's predictions. However, as noted above, in our review we focus on longitudinal studies. We found several studies that prospectively predicted insomnia by the JDC-S model. Linton (2004) found that only lack of social support predicted the development of new cases of insomnia one year later. Jansson and Linton (2006) found that only high work demand predicted the development of new cases of insomnia in a follow-up conducted approximately one year later. Subsequently, in a study based on a considerably larger national sample (Jansson-Frojmark, Lundqvist, Lundqvist, & Linton, 2007), it was found that among individuals with no insomnia at baseline, high work demands increased the risk of developing insomnia one year later. In the three prospective studies referred to above, the JDC-S components were found to reinforce the continuation of insomnia among individuals with insomnia at baseline. In summary, the evidence coming out of the three longitudinal studies provides support for the JDC-S model's prediction relative to sleep disturbances.

The study of the behavioral outcomes of the JDC-S model is complicated because these outcomes frequently appear in pairs or triads, analogous to the co-morbidity patterns of chronic illnesses. Different combinations of outcomes are the rule, rather than the exception. A well-known example is the very close associations of the JDC-S model's components with insomnia,

smoking, and burnout (Armon, Shirom, Shapira, & Melamed, 2008). This has led to the construction of dual-diagnosis and triple-diagnosis schemes and to the development of comprehensive, multi-faceted treatment approaches. The pattern of appearance of several outcomes in an individual may vary, depending on background characteristics and genetic and environmental factors. Stress and sleep disturbances may reciprocally influence each other: Stress may promote transient insomnia, which in turn causes stress and increases risk for episodes of depression and anxiety (Partinen, 1994).

### **Summary and Conclusions**

In this concluding section, we discuss some of the theoretical and methodological issues reviewed in this chapter. Additionally, we present some of the limitations of the approach that we adopted and suggest promising avenues for future research on the model and CVD risk factors.

### **Major Conclusions**

The maladaptive health responses covered in this chapter were all characterized as having multifactor etiology. For each of the maladaptive responses considered here, stress at work is but one of the possible culprits. For the great majority of the studies cited in this review, different additive and interactive combinations of the components of the JDC-S model were found to be the most powerful predictors. Still, one of the major conclusions that we could reach is that the JDC-S model was not found, in studies based upon longitudinal designs, to account for a significant proportion of the variance of blood lipids and of inflammation biomarkers. Therefore, we would like to suggest that blood lipids and inflammation biomarkers probably do not represent linking pins between the model's components and CVD.

The evidence on the effects of the JDC-S model on blood pressure tends to be equivocal. A recent review of the relationships between job-related stress and causal blood pressure that covered 48 studies, including studies that used the JDC-S model (Mann, 2006), concluded that the evidence for a relationship between chronic job stress and blood pressure is weak. However, there is a major difference between the three physiological risk factors for CVD reviewed in our chapter. While we could positively assert that the pathway leading from the components of the JDC-S model to blood lipids and inflammation biomarkers appears unpromising and dubitable, this is not the case for blood pressure. While the evidence is weak, some of the longitudinal studies that we reviewed above could be interpreted to mean that job demands, control and support - either additively or synergistically - could move individuals from the relatively benign category of 'borderline' to that of 'inflicted with hypertension and in need of medication'.

The JDC-S model is definitely a useful tool to investigate the etiology of insomnia, the only maladaptive behavioral outcome covered in our chapter. As we noted, insomnia is a prevalent condition in the general population worldwide, with conservative estimates ranging from 9% to 12% of all adults inflicted with this disorder (Ancoli-Israel & Roth, 1999). Therefore, the conclusion that the JDC-S model could profitably be used to predict future cases of insomnia is very important. Like all other risk factors included in our chapter, the pathogenesis of insomnia is both complex and multidimensional, with several mechanisms influencing the course of this condition's development (cf Jansson & Linton, 2006). The JDC-S model is but one among many possible mechanisms.

**Theoretical Issues in the Reviewed Field**

A major theoretical postulate of this chapter has been that job demands have a positive influence, and that control and support have a negative influence on maladaptive health responses. Whenever possible, support for this underlying assumption was provided in the review of each of the specific maladaptive health responses using studies based on longitudinal designs because they provide more robust support for the unidirectional effects posited by the JDC-S model. However, the relationships between the JDC-S model and maladaptive health responses may be reciprocally related; the direction of influence may flow from the health response, such as sleep disturbance, to the appraisal of the job demands included in the model. In all the studies that we co-authored, referred to above, we also tested the reverse-causation hypothesis: only for insomnia did we find some support for it.

The reverse-causation hypothesis was investigated in a few recent studies; however, the criteria used in these studies were indicators of mental health. For example, a study conducted on a large sample of soldiers (Tucker et al., 2008) used multilevel modeling to test the directionality of the relationships between the JDC model and mental health based on six waves of survey data collected at 3 months time lags. Tucker et al. (2008) found strong support for the reverse causal effects such that higher initial strain was associated with higher subsequent work overload and lower control. Another recent study (de Lange, Taris, Kompier, Houtman, & Bongers, 2004) investigated a large sample of Dutch employees, assessing psychological strain and the JDC-S model over four waves of data collection. The results provided support to the expectation that there are reciprocal effects between the model's components and strain over time. In the same vein, reciprocal relationships between the model's components and indicators of mental health



were identified in another study (de Lange, Taris, Kompier, Houtman, & Bongers, 2005).

Therefore, we suggested that possible reciprocal relationships among the JDC-S model and risk factors for CVD should be systematically examined in future research, as well as the causal effects of physiological and behavioral strain on the JDC-S model.

An additional theoretical path of influence may stem from a third variable. That is, the empirical link between the JDC-S model and maladaptive health response might be spurious, arising from the relationship of both the model and maladaptive response to a third variable, such as a certain personality trait that may be genetically determined. For example, negative affectivity, or a person's predisposition to experience negative mood states such as depressive symptomatology, anger, guilt and fearfulness, may affect both stress appraisals and maladaptive health responses (Watson & Clark, 1984). Negative affectivity may lead to high job demands appraisals and low control and support appraisals because it is reflected in individuals being extremely vigilant in scanning their environment for stimuli that may threaten their well being (Watson, Clark, & Carey, 1988). Negative affectivity may be associated with reduced physical activity, obesity and elevated blood pressure (Burke, Brief, & George, 1993), and acting through its influence on depressive symptomatology could influence most of the maladaptive health outcomes under consideration here (Suls & Bunde, 2005).

### **Methodological Issues in the Reviewed Field**

A methodological limitation of the research literature covered in the review concerns the two approaches toward the conceptualization of stress referred to earlier, the one that focuses on chronic stress and the one that emphasizes critical or minor job events. These two approaches to stress measurement have seldom been combined in a single study designed to predict an outcome

considered here (Frese & Zapf, 1988). For example, it is well-established that episodic stressors cause transient elevations of blood pressure, but the relationships between transient elevations and persistent elevations of high blood pressure remain unclear (Mann, 2006). The same generalization is relevant to the combined use, in the same study, of family-related and work-related stress to predict either of the above outcomes. Seldom have antecedent variables, such as objectively measured job demands, been included in the research designs of the studies reviewed here. Finally, the longitudinal studies that we covered used diverse methodologies to assess the components of the JDC-S model.

Research on the JDC-S model has often been criticized for assessing the job characteristics included in the model subjectively, primarily by means of self-reports on questionnaires (Jones, Bright, Searle, & Cooper, 1998; Kristensen, 1996). It was argued by these critics that self-reports do not necessarily represent "true" job conditions due to distorted perceptions and other self-report biases. These claims were empirically examined in multi-methods studies that evaluated the JDC-S model using expert-ratings and aggregated group evaluations (Griffin, Greiner, Stansfeld, & Marmot, 2007; Theorell & Hasselhorn, 2005; Waldenstrom et al., 2008) and in general did not provide any support for the subjectivity argument.

### **Limitations of the Current Review**

Some limitations of the current review are common to any narrative review of a phenomenon. We have made an effort to cover meta-analytic studies for each of the maladaptive health responses discussed, but they were seldom available for the health outcomes that we focused on. When meta-analytic studies were not available, the most recent qualitative reviews

were consulted. However, it was beyond the scope of our chapter to identify and discuss the major inconsistencies in the findings of the relevant longitudinal studies. Researcher used different follow-up times, different operationalizations of the JDC-S model's components, and different sets of control variables. Resolving such inconsistencies constitutes a major challenge for future meta-analytic investigations of the pertinent JDC-S model-maladaptive health response associations.

### **Suggestions for Future Research**

We reviewed above three major theoretical perspectives that guided most of the research currently being carried out in the field of stress and physical health. After reviewing the P-E Fit perspective, the ERI approach, and the JDC-S model, we decided to focus on the latter. Empirically, several studies systematically compared the predictive power of the ERI and the JDC-S models relative to different types of psychological strain (for references, see: Dragano et al., 2008; Griffin, Greiner, Stansfeld, & Marmot, 2007; Rydstedt, Devereux, & Sverke, 2007). These studies yielded inconsistent results, but generally provided support for the argument that each model probably explains a unique variance in strains and therefore combining them is the optimal strategy for increasing our understanding of the influence of work characteristics on people's health. These theoretical perspectives on stress-health relationships differ in their conceptualization of stress, strain and health, and place different emphases on some of the antecedent mediating and moderating variables depicted in Figure 1. However, as we noted, the three theoretical perspectives appear largely complementary. Therefore, we would like to suggest that one of the more promising avenues for research in this area is to systematically compare the predictive validity of these theoretical perspectives with regard to risk factors for CVD. .

To illustrate the point made above, we would like to refer to a recent study (Bosma, Peter, Siegrist, & Marmot, 1998) which compared the predictive validity of the effort-reward-imbalance perspective and the demand-control-support model with respect to coronary heart disease. Bosma et al. (1998), in a study of men and women (6,895 and 3,413, respectively) working in British government offices, found that low job control and high cost/low gain work conditions independently influenced the development of heart disease. This research exemplifies the advantages of combining several theoretical perspectives, in a longitudinal design, to predict maladaptive health responses.

Each of the maladaptive health responses may be conceptualized along several dimensions, including its level, variability or consistency, forms of appearance, temporal intensity, and trajectory. For example, we noted above that inflammation biomarkers in the body include a group of indicators, and each could play a different role in the etiology of CVD. Future research on each of the maladaptive health responses covered in this review may consider including in the study design several important dimensions of the response investigated.

Another promising avenue for research concerns the interactive effects of the JDC-S model and socioeconomic disadvantages in predicting maladaptive health responses. Most epidemiological studies that assess the effects of the JDC-S model on the maladaptive health responses considered here statistically control for the effects of the subjects' age, sex, race-ethnicity, obesity, smoking behavior, and a family history of hypertension or hyperlipidemia. Such a model assumes that the JDC-S model influences the maladaptive health response under consideration independently of the confounders that were controlled for. Often, this assumption is unwarranted. It is well known that stress affects obesity and smoking behavior as well as diet.

In addition, it is plausible that some of the antecedent variables tapping socioeconomic disadvantage interact with stress to influence some of the maladaptive health responses. In future research, researchers should consider adopting theoretical models that allow for moderating or mediating influences of the above confounders on the relationships between the JDC-S model and the maladaptive health response under consideration.

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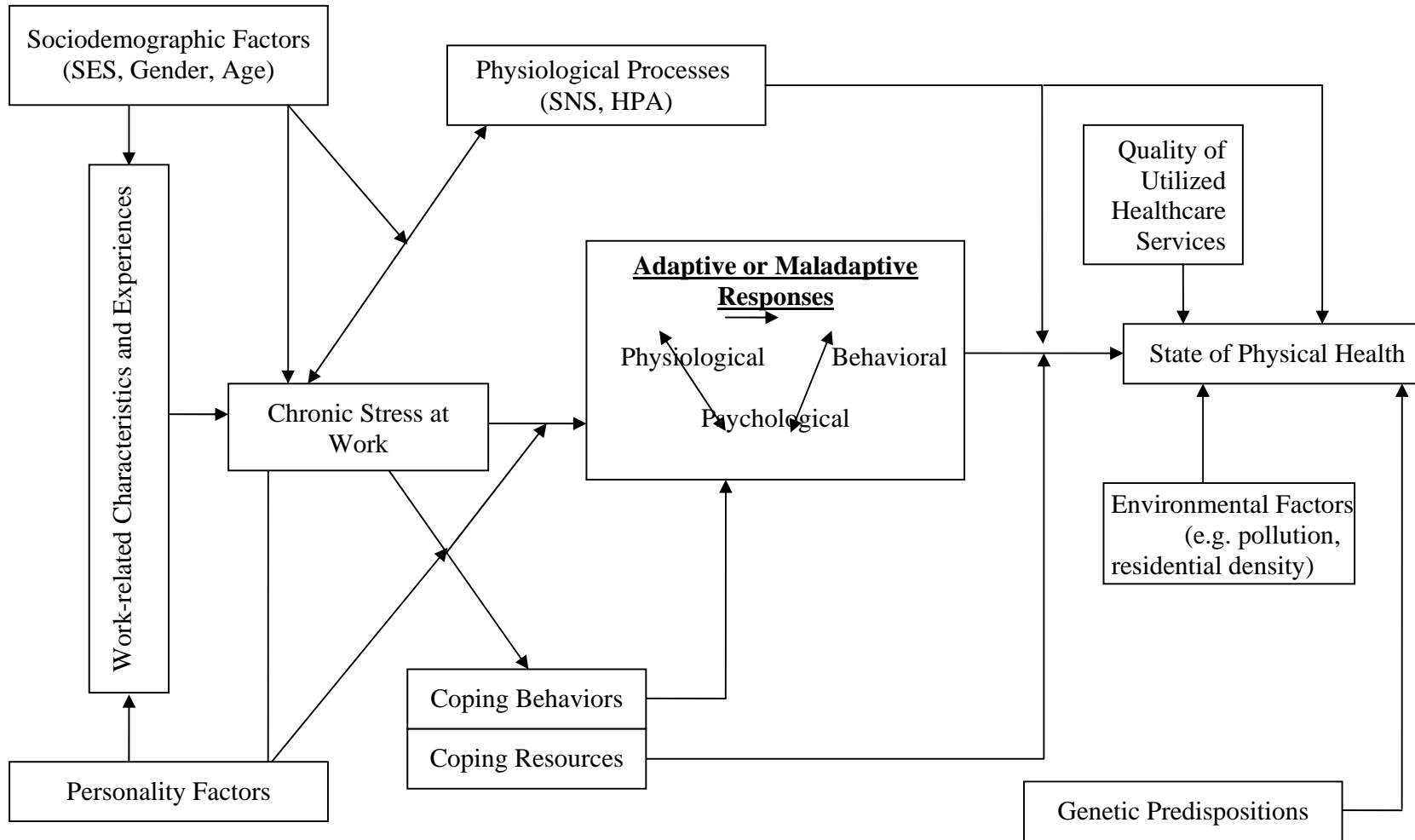


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**Figure 1.** A Theoretical Framework Depicting Possible Pathways for the Effects of Stress at Work on Physical Health