

# Work Stress and Employee Health: A Multidisciplinary Review

Daniel C. Ganster  
*Colorado State University*  
Christopher C. Rosen  
*University of Arkansas*

---

*Research examining the relationship between work stress and well-being has flourished over the past 20 years. At the same time, research on physiological stress processes has also advanced significantly. One of the major advances in this literature has been the emergence of the Allostatic Load model as a central organizing theory for understanding the physiology of stress. In this article, the Allostatic Load model is used as an organizing framework for reviewing the vast literature that has considered health outcomes that are associated with exposure to psychosocial stressors at work. This review spans multiple disciplines and includes a critical discussion of management and applied psychology research, epidemiological studies, and recent developments in biology, neuroendocrinology, and physiology that provide insight into how workplace experiences affect well-being. The authors critically review the literature within an Allostatic Load framework, with a focus on primary (e.g., stress hormones, anxiety and tension) and secondary (e.g., resting blood pressure, cholesterol, body mass index) mediators, as well as tertiary disease endpoints (e.g., cardiovascular disease, depression, mortality). Recommendations are provided for how future research can offer deeper insight into primary Allostatic Load processes that explain the effects of workplace experiences on mental and physical well-being.*

**Keywords:** stress; strain; occupational health; well-being; Allostatic Load

---

More than 20 years ago, Ganster and Schaubroeck (1991) began their review of the literature on work stress and health by noting that, in the 10 years prior to then, there were hundreds of studies of work stress, as well as many volumes that compiled and summarized this literature. They noted that work stress studies appeared in a broad array of journals in

---

*Corresponding author: Daniel C. Ganster, Department of Management, Colorado State University, 134 Rockwell Hall, Fort Collins, CO 80523, USA.*

*E-mail: [dan.ganster@business.colostate.edu](mailto:dan.ganster@business.colostate.edu)*

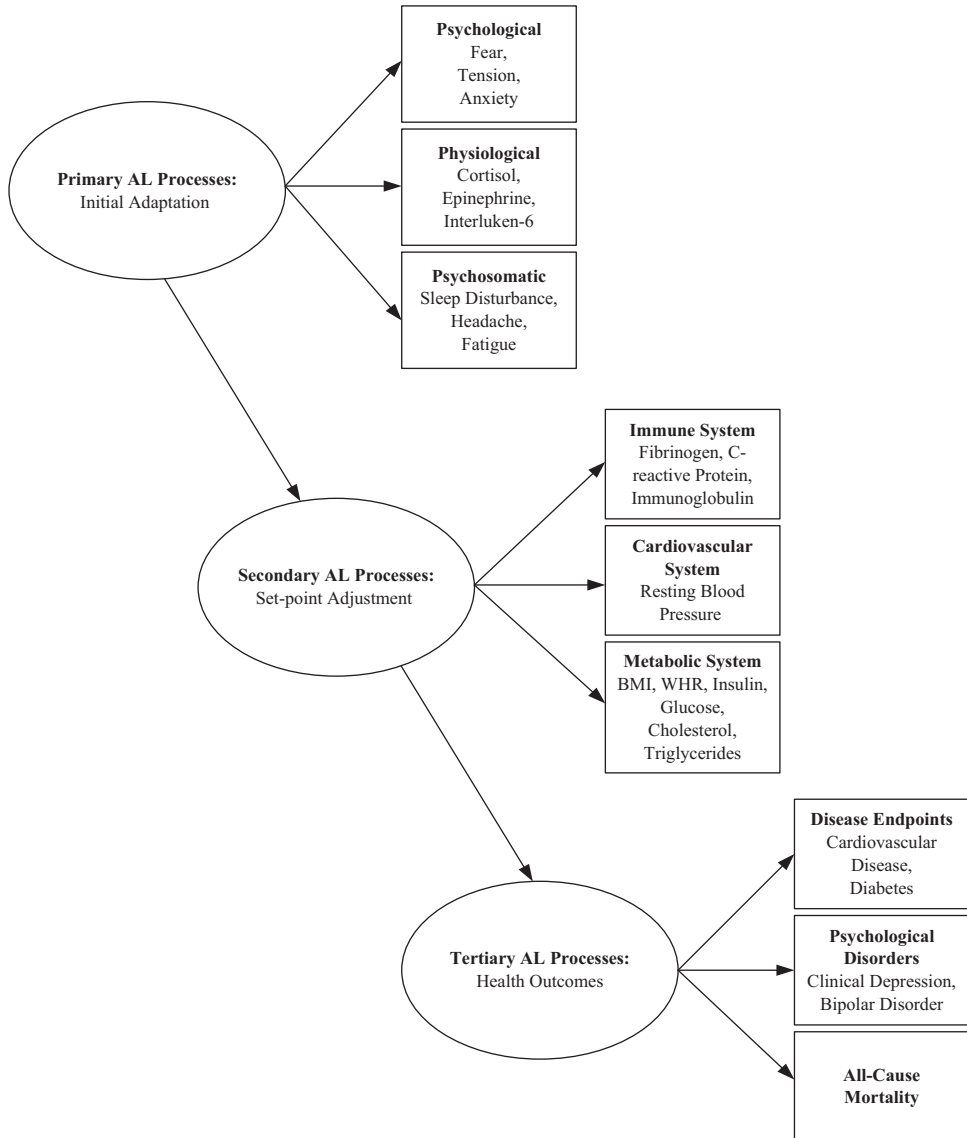
many disciplines, making this literature especially difficult to compile and integrate. Since that time, work stress research has continued to grow and at an even faster pace, making any effort to summarize it even more challenging. During this same time period, research on physiological stress processes has also advanced significantly. Unfortunately, this research has not been summarized and related to work stress research in any systematic way.

Inspired by Selye's (1955) description of the General Adaptation Syndrome, management researchers have recognized the usefulness of incorporating a physiological perspective into their research on work stress for a long time (Caplan, Cobb, French, Harrison, & Pinneau, 1975; Fried, Rowland, & Ferris, 1984; Ganster, Mayes, Sime, & Tharp, 1982), and this interest continues today. Moreover, Heaphy and Dutton (2008: 138) recently suggested that researchers should pay more attention to human physiology across a broad array of management phenomena, as such research has the potential to affirm "the fundamentally important effect of work contexts." Similarly, numerous organizational scholars have argued that organizational research could benefit from the study of human physiology because physiological reactivity may explain the underlying processes that link exposure to workplace stressors to impaired functioning at work, absenteeism, and health care costs incurred by employers (e.g., Ganster, 2005; Greenberg, 2010; Halpern, 2005; Zellars, Meurs, Perrewé, Kacmar, & Rossi, 2009). Consistent with this perspective, we believe it is important for researchers to be aware of the progress that has occurred in this area and how it can inform the course of work stress research in the management literature.

The physiological literature is especially important for understanding the effects of work experiences on mental and physical health, which is the particular focus of our review. Thus, the primary goal of this article is to provide an integrative review of the work stress literature that incorporates the diverse fields that have contributed to it. We believe that management scholars, to the extent that they influence the design of jobs, pay systems, leadership training, and other aspects of organizational functioning, can play a significant role in improving the health of workers. But to effectively serve this role, we need to know what the basic research tells us about the factors that underlie health and well-being, and the processes through which work experiences might affect them. We thus view the work stress literature through the lens of the Allostatic Load (AL) model, which has emerged as the dominant theoretical perspective in stress physiology (Juster, McEwen, & Lupien, 2010; Lupien et al., 2006; McEwen & Stellar, 1993). Specifically, we (a) present a review of the work stress literature that is structured around categories of outcomes (i.e., primary, secondary, and tertiary outcomes) specified by the AL model (see Figure 1), (b) consider the extent to which findings in the work stress literature are consistent with AL specifications, and (c) use the AL framework to provide guidance to researchers interested in investigating relationships between work stress and well-being.

We begin by providing basic definitions of stress and well-being and an overview of prominent theories of work stress. We then proceed to a discussion of the physiological basis of stress, which includes a review of the AL model and supporting evidence from biology, neuroendocrinology, and physiology. Next, we present a critical review of the empirical literature that links work characteristics to mental and physical health outcomes. When reviewing this literature, we explicitly consider evidence for a *causal* relationship between work experiences and well-being in general and with specific diseases. We are especially

**Figure 1**  
**Allostatic Load Model of the Stress Process**



*Note:* AL = Allostatic Load; BMI = body mass index; WHR = waist-to-hip ratio.

interested in discussing what this literature tells us about the physiology of stress and disease and how it can help guide research in the work sphere. We conclude with an integrative summary and a discussion of strategies for how future research can provide deeper insight into primary AL processes that explain the effects of workplace experiences on mental and physical well-being.

## Definitions

Stress can be thought of as (a) a feature of the external environment that acts on an individual, (b) the individual's responses (psychological, physiological, and behavioral) to environmental demands, threats, and challenges, or (c) the interaction of the two (Ganster & Perrewé, 2011; Kahn & Byosiere, 1992). The last conceptualization views stress more as the process by which environmental events initiate a series of cognitive and physiological reactions that ultimately affect well-being. Environmental events that trigger these processes are commonly referred to as *stressors*, while the individual's responses are generally called *strains* (Griffin & Clarke, 2011). Furthermore, in the work stress literature when we refer to environmental stressors, our focus is on what are generally referred to as *psychosocial stressors*. These are events and work characteristics that affect individuals through a psychological stress process, as opposed to a directly physical one. Thus, in this review we ignore a broad array of risk factors and exposures such as chemical toxins, noise, temperature, and other physical or safety-related hazards. Some physical complaints, such as lower back pain, might be caused by both physical ergonomic factors and psychosocial stressors, but our interest here is on the psychosocial ones. We thus define work stress as the *process by which workplace psychological experiences and demands (stressors) produce both short-term (strains) and long-term changes in mental and physical health*. Next we provide an overview of the theoretical models of stress that have had the greatest impact on research in the work domain. Our discussion of these theories is brief, however, and recent reviews provide more detailed and critical discussions of these models (Cox & Griffiths, 2010; Ganster & Perrewé, 2011; Meurs & Perrewé, 2011).

## Theories of Work Stress

The most influential model of the psychosocial stress process is Lazarus's transactional model (Lazarus, 1966). It is labeled "transactional" because it asserts that stress resides neither in the person nor the environment, but rather in the interaction between the two. The transactional model describes primary and secondary appraisal processes, whereby individuals cognitively process information about potential stressors in conjunction with their ability to cope with them. It is these cognitions that play the critical role in initiating physiological processes. The assumption that all environmental stressors operate through cognitive appraisals has been contested in the past. For example, Zajonc (1984) argued that affective and physiological responses can be evoked without cognitive mediation. More recently, the transactional model has come under attack by Hobfoll (1998), who argues that too much emphasis has been placed on cognitive

appraisals and not enough on the objective environment. Even Hobfoll, however, seems to vacillate about the importance of cognitive appraisals, noting that they “are the best proximal indicators in the stress process” (Hobfoll, 2001: 359).

There seems to be little disagreement that psychosocial stressors exert their effects primarily through how the individual perceives and evaluates them. Neither Lazarus nor Hobfoll would likely argue with the contention, moreover, that individuals appraise potential stressors in different ways, and such differences in appraisals depend on many factors, including prior experiences and genetic and cultural differences. Interestingly, even the physiological theorists place cognitive appraisals (especially anticipatory appraisals) in the primary position of the stress process, with the sequence of physiological responses beginning in the central nervous system where potential threats are encoded (Gaab, Rohleder, Nater, & Ehlert, 2005). More recently, Meurs and Perrewé (2011) presented the Cognitive Activation Theory of Stress (CATS) as an extension of cognitive appraisal models, arguing that (a) previous experiences lead individuals to develop stimulus and outcome expectancies to which they adapt over time and (b) CATS has the potential to integrate both positive and negative aspects of the stress experience.

The cognitive appraisal model addresses the question of *how* psychosocial stressors affect well-being, but it does not tell us *what* specific features of the work environment matter most. Yet work stress researchers must choose a limited number of workplace characteristics to address in any given study. This choice of stressors has been guided by several models in the past, such as role stress theory (Kahn, Wolfe, Quinn, Snoek, & Rosenthal, 1964) and the P-E fit theory (Harrison, 1985). Although some of the concepts discussed in role stress and P-E fit models continue to be included in contemporary work stress theories (e.g., the general notion that strain is associated with lack of fit between a person and his or her environment is part of many work stress theories), these models began losing their influence on the empirical literature at least two decades ago (Ganster & Schaubroeck, 1991). In the past two decades, the work stress literature has been most strongly guided by the job demands-control model (JDC; Karasek, 1979) and its derivatives, such as the job demands-resources (JDR) model (Demerouti, Bakker, Nachreiner, & Schaufeli, 2001). The JDC model has shown great heuristic power, stimulating many large-scale studies in epidemiology and many smaller-scale studies in the psychology and management literature, and has established the construct of job control as a central one in the work stress literature (Ganster & Perrewé, 2011). More recently, job control has come to be seen as just one of several resources that can either buffer the effects of demands (e.g., high workload, conflict) on well-being or have its own direct salutary effects. The JDR model (Demerouti et al., 2001), for example, defines control as a resource, but suggests that other resources can be found in physical, psychological, social, or organizational spheres.

An even more extensive set of resources is proposed in Conservation of Resources (COR) theory (Hobfoll, 2001), which describes four main categories of resources (object, condition, personal characteristic, and energy). Hobfoll (2001: 341) lists 74 specific resources that he describes as “a comprehensive set that appears to have validity in many Western contexts.” As other critics have noted, however, the definition of a resource is so broad that it is difficult to think of something that might not fit the definition (Ganster & Perrewé, 2011; Thompson & Cooper, 2001), which makes this a relatively generic theory.

Another model that has become prominent in the epidemiological literature is the Effort-Reward Imbalance Model (ERI), which posits that stress results from imbalances in social exchange and reciprocity in the workplace (Siegrist, 2002). In this model, failed reciprocity is defined as occurring when the individual expends high efforts that are insufficiently matched by rewards from the organization. The imbalance (too much effort in relation to too few rewards) is thought to trigger negative emotions and physiological stress responses. The positive emotions evoked by a balanced social exchange, conversely, are theorized to promote general growth and well-being.

Together, these psychological models of stress are useful for describing how events in the environment generate stressful appraisals, yet they are all based on the premise that psychosocial stressors exert their effects on mental and physical well-being through intervening physiological processes. Unfortunately, such processes are typically not explicitly described by work stress theorists. Thus, prior to reviewing the work stress literature, we provide an overview of the AL model (McEwen & Seeman, 1999; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997), a theoretical model that identifies intervening physiological processes that link stress exposure to health outcomes.

### **The Physiological Basis of Stress: The Allostatic Load Model**

The AL model evolved from the stress-response work of Selye (1955), who argued that stress is the nonspecific response of the body to a demand, regardless of whether the demand resulted in pleasant or unpleasant conditions. Selye's model of responses to stress, which suggests that the body adapts to stress via single-point tuning (e.g., changing blood oxygen levels), was challenged by subsequent research that demonstrated that there was a great variety in physiological responses depending on the different cues and situations facing individuals (Goldstein & Eisenhofer, 2000). Selye also relied on the concept of homeostasis, which refers to the body's attempt to maintain a stable internal environment through a complex system of feedback mechanisms. Subsequent research demonstrated that organisms exhibit a broad range of behavioral and physiological responses to stressors, often responding to demands by adopting new set points of physiological systems. Thus, the homeostasis model is no longer seen as adequate for explaining the stress response and has been replaced by the concept of allostasis (Sterling & Eyer, 1988).

Frequently referred to as *stability through change*, allostasis is the process of adjustment of various effector systems (cardiovascular, neuroendocrine, and others) that serve to cope with real, imagined, or anticipated challenges to homeostatic systems. Allostatic systems, such as the hypothalamic-pituitary-adrenal (HPA) axis, operate around certain set points, some of which are subject to diurnal or even seasonal rhythms; these set points can also be reset after exposures to chronic demands that continually push them beyond their normal ranges. Another important feature that distinguishes the allostasis model from Selye's (1955) stress-response model is the critical role played by the central nervous system, which controls physiological reactions, often directly, by using prior knowledge and experience in conjunction with environmental events, to anticipate the need for adaptation. In this respect, AL theorists are very much in the cognitive appraisal camp, and they see organisms as active perceivers and anticipators of demands from the environment.

One of the central features of the AL model is that various adaptation systems interact with each other in complex and nonlinear ways, and there is a temporal sequence to these processes. The first phase involves the stimulation of *primary mediators*. These consist of stress hormones (e.g., epinephrine, norepinephrine, and cortisol) and pro- and anti-inflammatory cytokines (e.g., interleukin-6). These initial responses are triggered in the central nervous system, and they serve an adaptive role by preparing the organism to cope with demands that threaten to disrupt homeostatic systems. When primary mediators are activated repeatedly or chronically, however, their effects on cellular activities can adversely affect the integrity of physiological systems. This chronic activation of primary mediators leads to a set of *secondary mediators* in which other biological systems adjust their normal operating ranges (set points) in response to over- and underproduction of primary mediators. It is at this stage that various secondary indicators show subclinical perturbations. These secondary mediators involve the metabolic (e.g., insulin, glucose, cholesterol, and triglycerides), cardiovascular (e.g., blood pressure), and immune systems (e.g., fibrinogen, C-reactive protein). These mediators, when consistently out of normal ranges (dysregulations), are key risk factors for mental and physical diseases. Although permanent damage may not have been inflicted at this stage, the continuation of these secondary dysregulations over time leads to the *tertiary phase of allostatic overload*, characterized by disease endpoints (e.g., cardiovascular disease [CVD], depression, and death).

In many ways, the stages of the AL model resemble Selye's General Adaptation Syndrome. The major difference, however, is that researchers have taken a more comprehensive and multivariate approach to exploring the mediating processes at the different stages. Thus, a major thrust in AL research has been to examine composite indexes of primary and secondary AL mediators for their ability to predict disease endpoints, cognitive and physical functioning, and mortality. Among the first such efforts were the MacArthur Studies of Successful Aging (Seeman et al., 1997), in which an index was developed based on 10 primary (e.g., cortisol, epinephrine, norepinephrine) and secondary (e.g., cholesterol, blood pressure, waist-to-hip ratio) AL mediators. Early validation studies (Berkman et al., 1993) indicated that the AL composite successfully correlated with both cognitive and physical functioning measures at baseline, predicted declines in cognitive and physical functioning and incident CVD after 3 and 7 years (Seeman et al., 1997), and predicted all-cause mortality independently of demographics and baseline health status (Seeman, McEwen, Rowe, & Singer, 2001).

These early AL investigations were followed by dozens of new studies and continuing analyses of earlier cohorts. A recent review of 58 of these studies concluded that the AL model "has received compelling empirical support in its ability to predict a plethora of health outcomes using a multi-systemic approach" (Juster et al., 2010: 14). However, researchers have used a great variety of different AL algorithmic formulations ranging from the index approach noted above to difference scores, dynamic scores, and bootstrapping methods; Juster et al. (2010) described 12 different such formulations. Moreover, given inconsistencies in scoring and statistical analyses with these different formulations, researchers have not reached a consensus about which approach to constructing an AL composite is most useful for predicting disease.

This literature also illustrates the complexity of the three-stage, cascading sequence illustrated in Figure 1. Specifically, the AL model proposes that continued overstimulation of

primary mediators leads to dysregulation in secondary mediators and then to tertiary outcomes, but this sequence has proven difficult to validate empirically (Goldman, Turra, Glei, Seplaki, Lin, & Weinstein, 2006; Seeman et al., 2001). Thus, although the evidence does seem compelling that combinations of primary and secondary AL mediators predict well-being, existing evidence cannot yet delineate the pathways between primary and secondary mediators, nor the time span needed for overstimulation of primary mediators to produce dysregulation of secondary mediators. In fact, little attention has been paid to investigating the processes linking continued overstimulation of primary AL systems to the development of dysregulations in secondary systems. AL composites, however, when operationalized as some combination of primary and/or secondary mediators, do seem to be implicated in the progression of disease, declines in cognitive functioning in older age, and mortality (Juster et al., 2010). As such, it is of interest to explore psychosocial predictors of such composites. In this regard, various formulations of AL composites have been found to be associated with socioeconomic status (SES), age, racial/ethnic differences, and some lifestyle and workplace factors, although results again depend on the AL formulation used (Juster et al., 2010).

What lessons can organizational stress researchers learn from research on the AL model? First, this body of research points to a set of biological (and anthropometric) indicators that are implicated in the development of tertiary well-being, which includes both mental and physical outcomes, including longevity. However, it is not clear which combinations are most important for predicting well-being. Primary (e.g., cortisol) and secondary mediators (e.g., blood pressure) seem important and independently predict tertiary outcomes, suggesting that researchers should include an array of both types in work stress studies. Many of the secondary mediators are easy to sample and are fairly stable from day to day if appropriate collection protocols are used (e.g., fasting for blood lipids). Many of the mediators from the metabolic and cardiovascular systems are now routinely collected in regular medical examinations; and with increasing attention being paid to C-reactive protein, the immune system is often represented as well.

The primary mediators are more challenging, as they tend to be highly variable, making their measurement more complicated. At the same time, the primary mediators' ability to change quickly makes them effective markers for work stressors that might show similar variability over time. Moreover, if work stress researchers are interested in detecting changes in allostatic responses as outcomes of stress interventions, they will likely need to focus on primary AL mediators, as dysregulation of the secondary mediators occurs over a time course that exceeds what organizational investigators can accommodate, especially if they are implementing an experimental design. The question is, which primary mediators are promising and how should they be measured? Primary mediators from the endocrine system (e.g., epinephrine, norepinephrine, and cortisol) have been the subject of much research in the work stress literature (see Sonnentag & Fritz, 2006). Cortisol, in particular, is one of the most researched primary AL mediators and cortisol research serves as a useful exemplar of both the complexity of investigating AL mediators, as well as its potential for work stress researchers. Thus, we provide an overview of cortisol research that serves to identify several concepts and issues (e.g., diurnal rhythms, the time-course of dysregulation, and reactivity and recovery) that are relevant to the study of how individuals respond to work stress.



### *Cortisol and the HPA Axis*

The HPA axis is one of the primary AL systems and among the first to respond to social stressors (Juster et al., 2010). Beginning with cognitive appraisals of stressors, the HPA axis initiates the release of cortisol throughout the body. Cortisol affects most major organ systems and helps provide energetic resources to prepare the individual to cope with a stressor. As one of the primary AL mediators, cortisol has been linked to a variety of clinical and subclinical conditions, including metabolic syndrome (Brunner et al., 2002), depression (McEwen, 2007), and risk for CVD (Smith, Ben-Shlomo, Beswick, Yarnell, Lightman, & Elwood, 2005).

*Diurnal rhythms.* Early studies incorporating cortisol into AL formulations assessed cortisol output across 12 or 24 hours (Seeman et al., 1997). Cortisol excretion, however, follows a diurnal rhythm which shows a high level upon awakening, an increase in the ensuing 30 to 45 minutes, and a gradual decline throughout the day (Kirschbaum & Hellhammer, 1989). Instead of assessing cumulative daily cortisol output, researchers now generally focus on the diurnal pattern, with the assumption that deviations from this pattern represent dysregulations of the system (Stone et al., 2001). Several aspects of the diurnal cycle have been investigated including waking cortisol; the Cortisol Awakening Response (CAR), which occurs in the first hour after awakening; diurnal cortisol slope (i.e., the rate of decline from awakening until night); area under the curve (AUC), which is the estimated total output throughout the day; bedtime levels; and levels at specific points during the day (e.g., before and after work). There is no consensus as to which indicator is most relevant to secondary AL composites and tertiary outcomes, although most researchers now focus on the CAR and slope indicators (Chida & Steptoe, 2009).

*Time course of dysregulation.* An issue that has received scant attention concerns the time-course of cortisol dysregulation. There are several questions for investigators to pursue in this area. Do changes in the CAR, AUC, or evening levels of cortisol reflect stable perturbations of the HPA axis that lead to secondary AL indicators? If so, how long must one's exposure be to environmental demands before cortisol dysregulations become apparent? How long must diurnal disruptions persist before secondary AL mediators change? Finally, can changes in work stressor exposures reverse cortisol dysregulation, and will such reversals lead to improvements in secondary mediators? The time-course issue is especially vexing, as there is evidence that cortisol responses are hyper-reactive in early phases of stressor exposure, but become hypo-reactive after prolonged exposures (Heim, Ehlert, & Hellhammer, 2000). This places a burden on investigators to assess whether (a) exposures were to novel stressors, (b) exposures were chronic, or (c) the individuals had already developed stress-related disorders (e.g., fibromyalgia, burnout, and depression) associated with hypocortisolism (Juster et al., 2010). Research exploring change in the diurnal patterns of cortisol has barely begun to address these questions. At the same time, the field has progressed to the point that obtaining one or two cortisol measures during a work day is not likely to produce significant advances in the understanding of work stress.

*Reactivity and recovery.* A different approach is to relate chronic occupational stressors to how individuals react to and recover from exposure to acute psychological stressors. With this

approach, individuals are brought into a laboratory where their physiological responses to stressful tasks can be measured in a controlled setting. Dienstbier (1989) theorized that repeated exposures to challenges that were under the individual's control (e.g., aerobic exercise) would result in physiological *toughening* characterized by a vigorous yet quickly recovering Sympathetic-Adrenal-Medullary (SAM) response (indicated by epinephrine, heart rate, blood pressure, etc.) and a low HPA (cortisol) response. Schaubroeck and Ganster (1993) theorized an analogous *untoughening* process when individuals were chronically exposed to uncontrollable stressors, such that chronically stressed individuals were expected to become hyper-reactive and show slow recovery in the HPA axis and become hypo-reactive in the SAM axis. In recent years, research has provided evidence that such acute reactivity and recovery responses are predictors of secondary (e.g., waist-to-hip ratios, blood pressure recovery) and tertiary AL (e.g., carotid atherosclerosis) outcomes (Epel et al., 1999; Epel et al., 2000; Heopniemi, Elovainio, Pulkki, Puttonen, Raitakarai, & Keltigangas-Jarvinen, 2007; Schuler & O'Brien, 1997; Steptoe & Wardle, 2005). Thus, reactivity and recovery measures are promising indicators of changes in the SAM and HPA axes that are potentially related to chronic stress exposures.

Recent meta-analyses underscore the importance of considering how different types of work stressors relate to reactivity and recovery. Specifically, Chida and Hamer (2008) found a significant (i.e., nonsampling error) amount of interstudy variability in effect sizes across studies that considered how chronic work stressors related to SAM and HPA reactivity and recovery. Similarly, in a review of laboratory reactivity studies, Dickerson and Kemeny (2004) found large differences in the ability of different types of acute stressors to elicit cortisol responses, with social-evaluative tasks and those that were uncontrollable eliciting the largest cortisol responses. Thus, given the significant associations of acute physiological reactivity and recovery to AL markers and specific disease states (Chida & Steptoe, 2010), this approach has potential to provide new insights into the effects of work stress. In the following section, we review the work stress literature to determine whether there is evidence that chronic work stress is associated with similar patterns.

## Literature Review

To facilitate our integration of the psychological, management, physiological, and epidemiological literatures our review is structured around the three broad categories of outcomes suggested by AL theory. Specifically, we first consider the most commonly investigated outcomes of work stress (i.e., affective outcomes and health complaints), which reflect subjective indicators of primary AL mediators. The remainder of the review follows the sequential order suggested by the AL model, with a focus on physiological indicators that can be classified as primary or secondary mediators and tertiary outcomes in the AL stress process.

### *Primary Indicators*

Evidence linking work stress to primary outcomes has, by and large, been provided by the vast work stress literature that has considered affective outcomes (e.g., anxiety, psychological

distress, emotional exhaustion) or acute stress-related health complaints (e.g., headache, fatigue, gastrointestinal problems). In the great majority of these studies, outcomes are self-reported and represent subclinical measures of well-being. These outcomes reflect proximal, relatively immediate reactions to stress exposure and are, therefore, most likely to operate during the acute phase. Subsets of this literature have been summarized in several meta-analytic reviews, and we begin by discussing patterns that have emerged across these meta-analyses. Ganster and Schaubroeck (1991) noted that a strong limitation of this literature was the reliance on nonexperimental research designs and perceptual measures. Therefore, in addition to reviewing trends across meta-analyses, we also discuss individual primary studies that have made unique contributions to the literature by employing relatively novel or more sophisticated research methodology (e.g., event and time sampling procedures, objective assessments of work stressors, quasi-experimental and experimental research designs). Finally, we review the smaller body of research that has explicitly considered primary physiological AL mediators, including stress hormone levels and daily fluctuations in blood pressure.

*Affective outcomes and health complaints.* Three patterns have emerged across meta-analyses of the work stress literature. First, there is consistent evidence that work stressors demonstrate modest to strong correlations with well-being. This pattern of results is best illustrated by Lee and Ashforth (1996) and Nixon, Mazzola, Bauer, Krueger, and Spector's (2011) meta-analyses, both of which summarized how various psychosocial stressors relate to subjective reports of well-being. Lee and Ashforth's (1996) study indicated that work stressors (e.g., role clarity, role conflict, role stress, stressful events, workload, and work pressure) demonstrate relatively strong correlations with emotional exhaustion, a dimension of job burnout (Maslach, 1982) that is a commonly used indicator of psychological well-being. Similarly, Nixon et al. (2011) focused on relationships between various work stressors and self-reported physical complaints (i.e., backache, headache, eye strain, sleep disturbance, dizziness, fatigue, appetite loss, and gastrointestinal problems). All seven work stressors included in Nixon et al.'s (2011) meta-analysis (i.e., interpersonal conflict, lack of control, organizational constraints, role ambiguity, role conflict, work hours, and work load) demonstrated statistically significant, but modest, correlations with a composite measure of physical symptoms.

Thus, Lee and Ashforth (1996) and Nixon et al.'s (2011) meta-analyses clearly demonstrate that there is an association between workplace stressors and primary indicators. At the same time, a second pattern that emerged during our review is that work stressors are most strongly associated with affective outcomes (e.g., emotional exhaustion) relative to physical symptoms. This pattern is corroborated by two meta-analyses that considered the effect of job insecurity (i.e., Cheng & Chan, 2008; Sverke, Hellgren, & Naswell, 2002), both of which indicated that job insecurity demonstrates a stronger association with psychological well-being than to physical health. Similarly, Herscovis and Barling's (2010) meta-analysis indicated that supervisor, coworker, and outsider aggression are generally more strongly related to indicators of psychological (e.g., emotional exhaustion) than physical well-being. These findings suggest that the threshold for experiencing affective reactions to stress exposure may be lower than that for experiencing physical health symptoms.

A third pattern that we observed across meta-analyses is that stressors that prevent employees from achieving work goals were more strongly related to well-being than stressors that promote personal growth. For example, LePine and colleagues (Crawford, LePine, & Rich, 2010; LePine, LePine, & Jackson, 2004; Podsakoff, LePine, & LePine, 2007) have considered the differential effects of challenge (i.e., job/role demands, pressure, time urgency, and workload) and hindrance (e.g., constraints, hassles, resource inadequacy, role stressors, and organizational politics) stressors on composite measures of strain (e.g., anxiety, depression, emotional exhaustion, frustration, health complaints, illness, physical symptoms) and burnout. Corrected correlation coefficients from these meta-analyses indicate that both stressors are correlated with strain and burnout, but the associations between hindrance stressors and both strain and burnout are stronger relative to challenge stressors.

Overall, these meta-analytic reviews provide evidence for an association between psychosocial work stressors and various primary AL indicators associated with psychological and physical well-being. However, several methodological issues (e.g., temporal spacing of predictors and criteria, reliance on nonexperimental research designs, lack of consistency of operationalizations across studies) may place limits on the inferences that can be drawn from this research. Moreover, by and large, the studies on which these conclusions are based have relied on self-reported measures of stressors and well-being, which limits the strength of the causal inferences that can be drawn from this research. Perceptual measures, while likely related to objective stressors (though the degree of association between objective and subjective measures of stressors remains to be determined), are often contaminated by other factors, such as the predispositions (e.g., cognitive appraisal styles, prior health status, personality, coping resources) that people bring with them to work. This creates a threat to the validity of research findings, as these predispositions may account for observed health outcomes (Ganster & Schaubroeck, 1991), not the stressors perceptual measures are designed to assess. Therefore, we consider research that has addressed some of these concerns, and subsequently advanced knowledge of how work stress relates to primary AL mediators by utilizing more rigorous research methodology.

- Correlational evidence suggests that there is a strong link between psychosocial work stressors and primary AL indicators associated with psychological and physical well-being, but this evidence should be considered in the context of the methodological issues that may place limits on the inferences that can be drawn from it.

*Event and time sampling procedures.* Event and time sampling procedures, which typically ask employees to record ongoing events and their reactions (psychological or physiological) to those events, are an increasingly popular alternative to traditional cross-sectional self-report measures. Though these measurement techniques generally rely on self-reports, and are thus subject to many of the biases that affect other self-reports (Semmer, Grebner, & Elfering, 2004), they also rely less on memory and are thought to be less subject to retrospective biases. Event and time sampling techniques, such as experience sampling methodology (ESM) and diaries are particularly relevant to the study of primary outcomes associated with work stress exposure because these measures allow researchers to integrate experiences with many situations over time to assess cumulative effects on transient, affect-laden within-person outcomes, such as emotions and anxiety.

In general, this group of studies has provided additional support for a stress effect, indicating that psychological well-being fluctuates with stress exposure. For example, Teuchmann, Totterdell, and Parker (1999) conducted a study in which accountants were asked to complete a survey three times a day for four weeks. Results indicated that mood and emotional exhaustion changed in parallel with perceived time pressure. Similarly, Totterdell, Wood, and Wall (2006) conducted a diary study aimed at testing whether Karasek's (1979) JDC model could account for weekly variations in strain. Totterdell et al. (2006) asked portfolio workers to record their perceptions of work demands, job control, social support, and psychological strain (e.g., anxiety and depression) every week for 26 weeks. Results indicated that participants experienced greater psychological strain during weeks that involved high work demand and low control. There was also evidence for an interaction between demands, control, and optimism, suggesting that individual differences are important to understanding how demands and control jointly explain weekly variation in strain.

Sonnentag and Zijlstra (2006) conducted a diary study aimed at understanding relationships between poor job characteristics, need for recovery, and well-being over a period of five working days. Their results indicated that high job demands, low job control, and unfavorable off-job activities (e.g., domestic demands) predicted need for recovery. In turn, need for recovery was negatively related to well-being at bedtime. More recently, Ilies, Johnson, Judge, and Keeney (2011) used ESM to assess the effects of interpersonal conflict on negative emotions. Ilies et al. (2011) analyzed data from university employees who completed a personality measure, as well as Internet-based surveys, three times a day over a two-week period. Participants reported interpersonal conflict episodes experienced during the three hours prior to the survey as well as social support and momentary negative affect. Results indicated that interpersonal conflict at work was more strongly associated with negative affect for agreeable employees and for those reporting lower social support at work.

Though there is substantial overlap between event and time sampling procedures and traditional self-reports, it should not be assumed that these measures assess the same thing (Semmer et al., 2004). Rather, within-person measures assess something (e.g., discrete events, more acute forms of stress) that can be distinguished from chronic, background characteristics of the environment. As such, event and time sampling measures are likely to contain unique variance over and above what is measured by traditional self-reports. Moreover, these measures seem most appropriate for assessing short-term reactions to stressors, such as momentary well-being, mood, coping behaviors, and reactivity and recovery responses. Consistent with this perspective, a primary finding from these studies is that affective responses fluctuate with stressful work conditions over time, which indicates that individuals demonstrate relatively immediate responses to acute demands and events. Nonetheless, these studies do not completely address concerns surrounding the perceptual nature of the stress measures employed and design limitations also preclude this group of studies from conclusively showing causal effects of work stress exposure on well-being (e.g., reverse causal explanations could not be ruled out for relationships between stressors and the self-reported outcomes). As such, it is important to consider the extent to which studies that have utilized more objective measures of work stress and experimental (or quasi-experimental) designs provide corroborating evidence for an effect of work stress on primary indicators.

- Research using event and time sampling procedures indicates that affective responses fluctuate with stressful work conditions over time, such that individuals demonstrate relatively immediate reactions to acute demands at work.

*Objective assessments of job stressors.* In comparison to the vast literature that has considered relationships between perceived stressors and well-being, relatively few studies have investigated how employees respond to *actual* job exposures. The studies that have considered actual job exposures do, however, provide evidence for modest convergence between self and other-reported measures of work stressors. For example, Grebner, Semmer, and Elfering (2005) measured work conditions (e.g., time pressure, concentration demands, uncertainty, work interruptions) through self-reports and ratings provided by trained observers. Data were collected at two points in time and correlations between self- and observer-rated stressors ranged from .50 to .70 across different time periods, providing some evidence for correspondence between measures. Self-reported stressors generally demonstrated stronger zero-order correlations with indicators of well-being (e.g., psychosomatic complaints, irritated reactions, exhaustion) relative to observer-rated stressors. Unfortunately, we could not determine how much unique variance in the dependent variable was explained by ratings from different sources.

Further evidence for convergence has been provided by studies (Schaubroeck, Ganster, & Kemmerer, 1994; Shaw & Gupta, 2004: Study 2; Xie & Johns, 1995) that have utilized self and other measures of job complexity to assess interactive effects of job complexity on well-being. In all three of these studies, researchers collected ratings of job complexity from incumbents and also acquired occupation-level data by matching incumbent job titles to occupational database ratings (e.g., Occupational Network [O\*Net] ratings). Though correlations between self-rated complexity and occupation-level measures were generally lower than those reported in research that has assessed self- and other-rated job characteristics (Grebner et al., 2005), results of all three studies provided evidence for hypothesized moderating effects across different operationalizations of job complexity and multiple primary indicators of psychological well-being (e.g., exhaustion and anxiety).

- Though there is evidence for convergence among self-reported and more objective measures of stressors, self-reported measures tend to demonstrate stronger relationships with well-being relative to more objective assessments of workplace stressors.

*Intervention studies.* Intervention studies have also provided evidence for a stress effect that is associated with actual exposure to stress, with the most consistent evidence coming from quasi-experimental research that has studied the effects of job redesign interventions. A trend in this literature has been to consider the role of perceived job characteristics in mediating the effects of job redesign interventions. For example, Bond and Bunce (2001) and Bond, Flaxman, and Bunce (2008) conducted job redesign interventions aimed at increasing employee job control. Results of both studies indicated that perceived job control mediated the effects of the intervention on work-related mental ill health as well as absenteeism. Similarly, a quasi-experimental study conducted by Holman, Axtell, Sprigg, Totterdell, and Wall (2010) indicated that perceived job characteristics mediated the effects of participative

job redesign interventions on job-related well-being. As a group, these studies provide a fairly believable picture that (a) participative interventions influence employee perceptions of job characteristics and (b) the effects of job characteristic interventions on primary outcomes are mediated by enhanced perceptions of job characteristics.

A small number of fully randomized field experiments have also considered the effects of work stress on primary indicators. Full randomization allows researchers to rule out most threats to internal validity that are present in field studies (Shadish, Cook, & Campbell, 2002). Despite this strength, the randomized experiments that have evaluated the effectiveness of stress interventions have provided only mixed evidence for a stress effect. For example, Schaubroeck, Ganster, Sime, and Dittman (1993) performed a randomized field study aimed at reducing role stress. In this study, participants were randomly assigned to either a role clarification treatment condition or a control group. A follow-up questionnaire indicated that the intervention reduced role ambiguity and supervisor dissatisfaction. However, the intervention was not related to changes in indicators of strain (e.g., subjective strain, physical symptoms). Logan and Ganster (2005) manipulated the job control of project managers in a trucking company by randomly assigning managers to either an intervention or no intervention condition. In the intervention condition, project managers participated in training designed to give them more control over various aspects of their jobs. The intervention increased perceptions of control after four months, but only for participants who also had supportive supervisors. For participants who had supportive supervisors, the intervention was linked to changes in job attitudes and these effects were fully mediated by control perceptions. Similar to Schaubroeck et al.'s (1993) findings, Logan and Ganster's (2005) results failed to demonstrate that the intervention affected nonwork indicators of well-being (e.g., anxiety, depression, somatic complaints). Mikkelsen, Saksvik, and Landsbergis (2000), who used a randomized experimental design to assess the effects of an intervention in health care institutions, reported similar results, such that the participatory intervention in their study had an effect on perceived job characteristics and subjective reports of work-related stress, but there was no evidence for an effect on subjective health reports.

Overall, therefore, evidence from studies that utilized fully randomized experimental designs indicates that work interventions have an effect on indicators of work-related well-being, but not more general, off-the-job indicators of well-being, which may be more relevant to the primary AL response. There are three potential explanations for these somewhat discrepant findings. First, the randomized field experiments that we located reported findings pertaining to a particular intervention (e.g., Logan & Ganster, 2005; Schaubroeck et al., 1993), suggesting that intervention studies show either (a) the theory underlying these interventions is inadequate or (b) some combination of intervention approaches may be needed to have a measureable impact on well-being. The Logan and Ganster (2005) experiment, for example, suggests that supervisor support may need to be addressed in an intervention in conjunction with control.

Second, little is known about the time lags involved in interventions. In general, the literature has shown that perceived stressors demonstrate stronger relationships with subjective measures of well-being when measures are taken in close proximity (Nixon et al., 2011). However, not much is known about how the stress appraisal process unfolds over time or how long it takes for stressors to influence more general well-being outcomes. Experimental studies are best for

capturing short-term effects, as job (i.e., diffusion of interventions) and non-job (e.g., life changes) factors are likely to intervene to reduce observed effects of interventions on well-being over time. Thus, it may be challenging for scholars to link stressors to broader secondary and tertiary indicators of well-being using randomized field experiments.

Finally, the relationship between stress and well-being may be much more complex than what has been tested in these studies. Specifically, individual differences and situational characteristics are likely to influence how interventions are experienced by different employees. Failing to measure and model such variables may explain null findings observed in previous studies. For example, negative affect may enhance sensitivity to negative information, such that individuals high in negative affect are more responsive to changes in job characteristics (Spector, Zapf, Chen, & Frese, 2000). Though random assignment reduces the likelihood that there will be between-group differences in negative affect, within groups there will still be substantial variation in this individual difference. If only people high in negative affect are influenced by the intervention, then this effect would be diluted within the intervention group unless negative affect was measured and modeled as a moderating variable. Similarly, contextual conditions, such as factors related to supervisor-subordinate relationship quality, may influence the effectiveness of stress interventions (Logan & Ganster, 2005). Therefore, when possible, future intervention studies should measure and model theoretically relevant individual differences and contextual variables that may influence the effectiveness of stress interventions.

- There is evidence that work interventions have an influence on work-related well-being, but more research is necessary to determine whether such interventions have an effect on broader (i.e., nonwork) AL indicators of well-being.

*Physiological indicators.* Compared to the literature that has investigated the association between work stress and primary psychological outcomes, relatively few studies have considered more objective, physiological indicators of well-being. These studies have generally relied on nonexperimental methodology and much of the focus has been on outcomes of perceived stressors, with cortisol being one of the most commonly studied physiological outcomes. A clear challenge to measuring physiological indicators is that biological cycles must be taken into consideration. As noted earlier, cortisol shows pronounced diurnal patterns. Although work stress researchers have assessed cortisol for quite some time (Rose, Jenkins, Hurst, Herds, & Hall, 1982), rarely have they taken the kinds of measures that allow for exploration of diurnal patterns. For example, Fox, Dwyer, and Ganster (1993) measured cortisol with saliva samples taken at awakening, at the midpoint of the workday, and again at 2 to 3 hours after work on a sample of nurses on two different work days. Although this sampling plan would have allowed for some assessments of diurnal pattern, such calculations were not yet routinely done, so Fox et al. focused on at-work and after-work cortisol.

More recent studies have, however, considered specific components of the diurnal pattern. Lundberg and Hellstrom (2002) obtained cortisol samples at awakening and at 15-minute intervals until 45 minutes after awakening for more than 200 working women. They found that those who worked more than 10 hours of overtime showed increased cortisol during these morning periods. In a study that specifically examined the CAR, Schlotz,



Hellhammer, Schulz, and Stone (2004) found a relationship between the increase in cortisol during the first 30 minutes after awakening and high workload. Similar results were reported by Kunz-Ebrecht, Kirschbaum, Marmot, and Steptoe (2004) who found that high morning cortisol was predicted by a combination of high job demands and low status, consistent with the JDC model. More recently, Karlson, Eek, Hansen, Garde, and Orbaek (2011) assessed the CAR at 45 minutes after awakening and found that men, but not women, in the high CAR quartile had *lower* mean job demands than those in the lower 3 quartiles (combined). However, both men and women in the high CAR quartile reported lower levels of control and work mastery, and higher levels of subjective health complaints.

In addition to the CAR, there have been a few attempts to link work stressors to flattening of the diurnal slope. The study by Karlson et al. (2011) calculated the slope as the difference between cortisol levels at 2100 hours and the highest of their two morning readings, and again dichotomized the sample by comparing the high quartile to the rest. After a series of univariate analyses conducted for each stressor, Karlson et al. found differences in reward, effort-reward imbalance, and overcommitment, although these effects differed by gender. Ganster, Fox, and Dwyer (2001) obtained cortisol samples at awakening, midwork shift, and evening, but did not calculate a diurnal slope. However, they did find that evening cortisol was predicted by an interaction of low control and high job demands. High evening cortisol levels would generally be associated with a flatter diurnal curve, although this was not tested explicitly in this study. Similarly, Rystedt, Cropley, Devereux, and Michalianou (2008) collected morning and evening measures of cortisol, but did not calculate a diurnal slope. Interestingly, their research indicated that chronic isostrain (i.e., an aggregate measure of work demands, control, and social support) was related to evening, but not morning, cortisol levels. They also observed that morning cortisol exhibited a sharp drop from working days to weekend days, suggesting that morning cortisol levels may be more sensitive to specific daily changes in job strain (i.e., acute stressors), whereas evening cortisol levels may be more sensitive to chronic exposure (Rystedt et al., 2008).

A relatively large-scale study that examined diurnal cortisol patterns and work stressors was reported by Holleman, Vreeburg, Dekker, and Penninx (2012). Holleman et al. collected seven salivary cortisol samples from 1,995 participants, starting with awakening and at 30, 45, and 60 minutes later and then again at 2200 and 2300 hours. They calculated the CAR response and an AUC over the course of the day. In addition, they collected another morning sample the next day after respondents ingested 0.5 mg of dexamethasone. This procedure, referred to as a dexamethasone suppression test (DST), measures the extent to which an individual's cortisol response is suppressed by dexamethasone, which reduces cortisol responses by acting on the pituitary gland. These cortisol values were related to self-reports of demands and control (analyzed as job strain by combining high and low scores) as well as important life events and childhood trauma. The only significant associations they discovered with work variables involved the DST measure. This test correlated negatively with social support at work and positively with job strain, which would be consistent with an interpretation that high work stress and low social support enhance the suppression of cortisol and might be a mechanism behind hypocortisolism. However, because of the large number of statistical tests conducted, Holleman et al. concluded that these significant findings may have arisen from chance and thus should be discounted until replicated.

Earlier reviews of the work stress and cortisol literature have found little consistent evidence relating work stress to cortisol from serum and urine (Hansen, Larsen, Rugulies, Garde, & Knudsen, 2009), but a review by Chida and Steptoe (2009) concluded that work stress was positively related to cortisol CAR (assayed mostly from saliva). A later review by Chandola, Heraclides, and Kumari (2010) examined studies of the postmorning diurnal pattern of cortisol, and concluded that there was no clear and consistent relationship between work stressors and postmorning (generally afternoon and evening) levels of cortisol. They noted, however, that most of these studies were cross-sectional and had small sample sizes.

In addition to a growing focus on the diurnal cortisol pattern and whether disruptions in it are related to work stressors, two other trends have emerged in this literature. First, though relatively little empirical research has provided evidence for a main effect of work stress on physiological health, several studies have supported JDC-based hypotheses. Specifically, various forms of control appear to moderate the effects of work demands on physiological well-being. Second, recent studies have provided evidence for correspondence between psychological and physiological indicators of primary AL outcomes, suggesting that work stress may have near-simultaneous effects on these primary indicators of well-being. Fox et al.'s (1993) results illustrate both trends. Specifically, Fox et al. utilized both psychological and physiological measures of well-being to test JDC-derived hypotheses. Results indicated that perceived work load interacted with control to predict a variety of primary outcomes, including job satisfaction, blood pressure at work, and cortisol level at work. These findings provide evidence that primary psychological and physiological outcomes may occur simultaneously.

More recently, Perrewé, Zellars, Ferris, Rossi, Kacmar, and Ralston's (2004) cross-sectional study demonstrated that self-reported work stressors converge in terms of predicting psychological, somatic, and physiological strain criteria. Perrewé et al. surveyed oil company employees about their perceptions of role conflict, political skill, psychological anxiety, and somatic complaints. Physiological measures included heart rate and blood pressure, both of which may reflect secondary AL mediators in this study because these measures were taken off the job at a professional biofeedback clinic. Perrewé et al. proposed that political skill would attenuate the effects of workplace stressors because political skill acts as a coping resource that provides individuals with a sense of control over stressful aspects of the work environment. Consistent with Perrewé et al.'s hypothesis, results indicated that the relationship between role conflict and psychological anxiety, somatic complaints, and blood pressure (systolic and diastolic) was weaker for more politically skilled employees.

Research that has utilized event and time sampling procedures (i.e., ESM) has also provided evidence supporting JDC-based interactions across different measures of well-being. For example, Ilies, Dimotakis, and de Pater (2010) examined within-individual effects of perceived workload on affective (e.g., affective distress) and physiological (e.g., blood pressure) responses over a two-week period. Workload demonstrated significant between-individual correlations with self-reported measures of affective distress and burnout, but not with blood pressure (at work or at the end of the day). However, within-individual correlations indicated that workload was associated with affective distress and blood pressure (either throughout or at the end of the workday). Beyond main effects, job control and organizational support demonstrated moderating effects on the relationships between

workload and both affective distress and blood pressure, such that workload was more strongly related to indicators of primary AL responses (i.e., blood pressure and affective distress at work) for employees reporting less support and control.

- There are three trends in the literature that has looked at primary physiological indicators: (a) Researchers have started to focus on understanding how work stressors relate to disruptions in biological cycles (e.g., the diurnal pattern of cortisol production), (b) empirical studies have provided evidence that control buffers the effects of work demands on well-being, and (c) multiple studies have indicated that physiological and psychological outcomes may occur simultaneously in response to work stressors.

### *Secondary Mediators*

After repeated chronic activation, primary mediators can adversely affect functioning of allostatic mechanisms, which may lead to a set of secondary outcomes in which other biological systems change in response to over- and underproduction of primary mediators. These secondary mediators include indicators of metabolic, cardiovascular, and immune system functioning. Some responses (e.g., blood pressure) can be classified as primary when they are measured as momentary reactions, but are seen as secondary when assessed at resting levels. The major trend in this literature has been to move away from single, independent indicators to considering multidimensional measures that either tap into specific physiological pathways (i.e., individual biological systems) or provide an overall assessment of the functioning of multiple systems. Our review is organized around these different approaches to examining secondary AL indicators.

*System-focused measures.* Empirical studies have provided somewhat mixed evidence linking work stress to the functioning of specific regulatory systems. The most consistent evidence has come from research that has examined CVD risk factors. For example, average daily blood pressure (assessed by aggregating ambulatory readings) has been frequently correlated with job strain (or its components), ERI, threat-avoidant vigilant work, and long work hours (see Landsbergis, Schnall, Belkic, Baker, Schwartz, & Pickering, 2011; Light, Turner, & Hinderliter, 1992; Schnall, Schwartz, Landsbergis, Warren, & Pickering, 1992, 1998). There is also evidence that job demands and control interact to predict on and off the job blood pressure (Fox et al., 1993; Perrewé et al., 2004; Schaubroeck & Merritt, 1997). More recently, Ferris, Sinclair, and Kline (2005) considered the effects of biopsychosocial strain (e.g., sleep, emotional health, energy, concentration, perceived stress) on a broader measure of CVD risk, which included blood pressure, body composition, fasting cholesterol, and fasting glucose. Results indicated that job resilience and biopsychosocial strain mediated the effects of job demands on CVD risk indicators. This finding could be interpreted as supporting a link between primary (e.g., energy, perceived stress level) and secondary AL mediators.

A few studies have also provided evidence for a stress effect on metabolic functioning. For example, Li, Zhang, Sun, Ke, Dong, and Wang (2007) found significant associations

between job strain groups (based on values of demands and control) and an 11-parameter (e.g., body mass index; waist-to-hip ratio; high-density lipoprotein; low-density lipoprotein; total cholesterol; triglyceride; glycosylated hemoglobin) index of glyco-lipid allostatic load, after controlling for age, smoking, marital status, and educational differences. Though Li et al. noted a few associations between job strain (i.e., high demands and low control) and individual physiological indicators, the largest association was with the AL composite score. Similarly, using a sample from the Whitehall II cohort of British civil servants, Chandola, Brunner, and Marmot (2006) examined the relationship between chronic stress at work and metabolic syndrome, which is a cluster of risk factors (e.g., abdominal obesity, high blood pressure, insulin resistance, cholesterol) that increase the risk of heart disease and type 2 diabetes. Results indicated that those who reported greater exposure to work stressors (i.e., high demands and low control) over the 14-year period of the study were more at risk for metabolic syndrome.

Studies examining immune system indicators have provided more mixed results. For example, Xie, Schaubroeck, and Lam (2008) examined the effects of job demands, job control, traditionality (i.e., adherence to traditional cultural values), and distributive justice on immune system functioning in manufacturing employees. Their immune functioning indicators included immunoglobulin A (IgA) and immunoglobulin M (IgM) taken from blood samples and self-reported sickness (i.e., upper respiratory infections). Xie et al. also included blood pressure and psychological well-being (i.e., emotional exhaustion) as dependent variables. Only a small proportion of the JDC-based interactions tested were statistically significant, and the predictive power of these interactions was small. Moreover, main effects and interactions showed little consistency across self-reported and physiological measures of well-being. Nonetheless, results indicated that a three-way interaction between traditionality, work load, and job control predicted chronic upper respiratory infections as well as IgA.

Shirom, Toker, Berliner, and Shapira (2008) also considered the effects of workload and job control on immune system indicators (i.e., C-reactive protein, fibrinogen, and white blood cell count) that reflect stress-induced inflammation in the body. Similar to Xie et al. (2008), Shirom et al.'s results provided support for only a small proportion of JDC-based relationships such that main and interactive effects of workload and perceived control generally failed to predict physiological outcomes. Shirom et al. interpreted these findings as suggesting that the physiological mechanisms that link the demands-control-support model to cardiovascular morbidity probably do not include inflammatory processes in the body. Interestingly, Shirom et al. and Xie et al. both considered the effects of stress exposure on individual indicators independently. Thus, an alternative explanation for the weak findings across these two studies is that their relatively narrow measures of immune functioning were not sensitive to the effects of work stressors, a notion supported by the significant results generally reported in studies that have incorporated broader measures of cardiovascular (Ferris et al., 2005) and metabolic (Chandola et al., 2006; Li et al., 2007) functioning.

- Research that has focused on specific individual regulatory systems has failed to provide consistent evidence supporting a stress effect, but studies have shown that work stress relates to a variety of cardiovascular disease risk factors as well as indicators of metabolic functioning.

*Broad AL indices.* Drawing from Seeman et al. (1997), researchers have started to assess AL system functioning with indices that include physiological indicators that tap into multiple biological systems. For example, Sun, Wang, Zhang, and Li (2007) obtained questionnaire assessments of psychosocial job characteristics (e.g., decision latitude, job demands, and social support) from hospital employees. These employees also underwent a medical exam, during which 13 physiological measures were collected (e.g., body mass index, waist-to-hip ratio, blood pressure, high-density lipoprotein, cholesterol, cortisol, and adrenaline) and combined to create an AL index that summarized physiological activity across regulatory systems. Participants were classified as experiencing either high or low job strain based on their scores on decision latitude and job demand scales. Cross-sectional analyses indicated that AL in the high strain group was significantly greater than in the low group, before and after adjusting for other risk factors (e.g., smoking status and exercise). Analysis of individual indicators indicated that BMI, systolic blood pressure, triglycerides, the ratio of total cholesterol to high-density lipoprotein, and cortisol were the most sensitive to stress exposure, a finding that further suggests that multiple biological systems (e.g., cardiovascular and metabolic) are involved in the stress regulation process. Similarly, Bellingrath, Weigl, and Kudielka (2009) utilized an AL index comprising the 10 indicators studied by Seeman et al. (1997), as well as an extended index that also included metabolic, immunological, and blood coagulation markers. Results indicated that ERI and exhaustion were related to both AL composite formulations, but not to their individual components. These findings led Bellingrath et al. to conclude that chronic work stress and exhaustion are associated with multisystem summary indicators of physiological risk.

These studies provide support for the utility of considering AL composite formulations, but provide limited insights regarding which specific workplace factors are most related to these composites. This limitation stems from the limited variety of stressors that have been examined, as well as the measurement and analytical approaches these researchers used for work stressors. Bellingrath et al. (2009) calculated a ratio variable from self-reports of effort and reward, and Li et al. (2007) used the JCQ measure (Karasek, Brisson, Kawakami, Houtman, Bongers, & Amick, 1998) and created high and low stress groups by combining job demands and control scores split at the quartiles. This practice confounds the effects of demands and control constructs, and fails to test the interaction proposed by the theory. Another limitation is that these studies assessed AL mediators with one-time samples on a given day. Some of these AL mediators (e.g., primary ones such as epinephrine, norepinephrine, and cortisol) show significant within-person variability during the day and between days, making a single-day sampling strategy subject to much error variance (Hellhammer, Fries, Schweisthal, Schlotz, Stone, & Hagemann, 2007). This absence of within-person stability for primary mediators might account for the generally poor level of empirical support for the association between primary and secondary AL clusters.

- There is evidence that multiple systems are simultaneously involved in the work stress regulation process, but there is little consensus regarding which AL composite formulations are best for assessing how the body responds to work stress.

### *Tertiary Outcomes*

The continuation of secondary dysregulations over time leads to the tertiary phase of allostatic overload, which is characterized by disease endpoints. Much of the research on these longer-term outcomes has been published in the epidemiological literature, rather than in mainstream management and psychology journals, which tend to focus more on subclinical indicators (i.e., nondiagnostic) of well-being. Epidemiological studies tend to be population-based (i.e., based on representative sampling strategies) or have very large sample sizes, even if they are not strictly representative of specific populations. In addition, epidemiological studies tend to rely on both case-control designs, which generally involve cross-sectional analyses and retrospective measures of exposures, and cohort studies, which are prospective designs that generally predict the different risks of onset of well-being outcomes between cohorts who experienced different exposures and were equivalent in terms of the well-being indicators at the beginning of the study. The calculation of odds ratios in the former designs, and relative risk ratios in the latter, is standard practice in this literature, but sometimes leads researchers to dichotomize data that otherwise could have been more thoroughly analyzed. The following review is grouped around specific disease endpoints that have been examined in this literature.

*Cardiovascular disease.* Belkic, Landsbergis, Schnall, and Baker (2004) conducted a comprehensive review of studies examining various CVD indicators. Though earlier reviews (Hemingway & Marmot, 1999; Kuper, Marmot, & Hemingway, 2002; Schnall, Landsbergis, & Baker, 1994) covered much of this literature, Belkic et al.'s review (also summarized in Landsbergis et al., 2011) is distinctive because it is limited to studies that were based exclusively on the job strain model (Karasek & Theorell, 1990) and it examined specific manifestations of CVD, including death. This review was so narrowly focused that it even excluded studies that examined only the main effects of the job strain model (i.e., demands and control) rather than their combination. In addition, Belkic et al. developed multidimensional methodological quality ratings for each study based on 15 criteria (e.g., avoidance of selection bias, adjustment for medical and behavioral confounders). They reviewed 38 studies that consisted mostly of population-based samples, and their overall conclusion was that there was strong and consistent evidence among men for a significant relationship between job strain (i.e., high demands and low control) and CVD, although the results were not as consistent for women. From their analysis of methodological artifacts, they concluded that methodological limitations led to *underestimation* of effect sizes. Interestingly, Belkic et al. argued that meta-analysis of these studies was not appropriate because of the wide variety of methods used. As a consequence, however, we do not know what the true variability in effect sizes was across studies or whether their carefully scored methodological ratings for each study might have explained this variability.

Kivimaki, Virtanen, Elovainio, Kouvonen, Vaananen, and Vahtera (2006) meta-analyzed studies that examined the link between work stress and CVD, in this case focusing only on prospective cohort studies that tested JDC, ERI, or organizational-justice-based models. They computed average effect sizes in terms of risk ratios of incident CVD, which reflect the increased risk of showing some hard measure of CVD, or death from CVD (4 studies),

in a cohort that was free of CVD at the initial time of measurement. The prospective periods ranged from 4 to 26 years. For tests of the JDC model, the overall age- and gender-adjusted risk ratio of being in the high strain cohort (i.e., high demand and low control quadrant) was 1.42 ( $p < .05$ ), which was reduced to a nonsignificant level when adjusted for a longer list of risk factors and potential mediators. There was a significant level of heterogeneity across studies in terms of age- and gender-adjusted risk ratios. For the ERI model tests, the overall age- and gender-adjusted risk ratio was 1.58 ( $p < .05$ ), and, in this case, adjustments for a more extensive list of covariates did not reduce the risk ratio to an insignificant level. There was also a significant amount of heterogeneity across studies in both the age- and gender-adjusted models and models with more extensive adjustments. Finally, there was a significant overall age- and gender-adjusted risk ratio for organizational injustice of 1.62, and this remained high and significant even after adjusting for covariates that included job strain and ERI. Because there were only two injustice studies, no test for effect size heterogeneity was conducted.

Several characteristics of this meta-analysis are worth noting. First, by including only prospective cohort studies with sound CVD measures, Kivimaki et al. (2006) attempted to derive conservative estimates. Despite this conservatism, their conclusions about the risks associated with work stress reinforce those from Belkic et al.'s (2004) broader narrative review. Second, covariates controlled in these studies included physiological variables that, from an AL theory perspective, represent secondary mediators (e.g., blood glucose, cholesterol, blood pressure, MBI, triglycerides) that should lead to the CVD outcomes that were assessed. Controlling for them, then, is essentially partially testing a mediating model. If risk ratios were reduced as a result of these controls, this would suggest support for an AL theory explanation. Using this test, the potential mediating effect was only evident in the case of the JDC model.

Finally, estimates of risk from exposure to work stress were almost always assessed based on a single measurement of exposure (JDC, ERI, or injustice). Assuming it takes a relatively long time for such work experiences to produce CVD outcomes, a single measurement of exposure presumes a high level of stability in working conditions over the time frame of the prospective study. There is not a strong basis for this assumption, however. A prospective study by Bosma, Marmot, Hemingway, Nicholson, Brunner, and Stansfeld (1997), and not covered in the Belkic et al. (2004) review, measured job control at two different times (about 3.7 years apart) and used it to predict incident CVD measures at a third time about 4 years later. Subjects who had stable and high measures of control showed the lowest risk for CVD compared to those with uniformly low levels of control or those whose control levels changed during the first two periods. These results suggest that exposures can change over the course of just a few years and that the chronicity of exposure likely makes a difference in developing CVD.

An important trend in this literature is that many secondary outcomes (e.g., blood lipids, blood pressure, and anthropometric measurements such as waist-to-hip ratio) have been treated as confounders of the relationship between work stress and CVD, and reviews of the CVD outcome literature often compare risk ratios (or odds ratios) that are minimally adjusted (usually for age and gender) to ones that are more fully adjusted with a wider selection of biological markers. For example, a recent review (Backé, Seidler, Latza, Rossmagel,

& Schumann, 2012) of studies using prospective designs that examined the association between work stress and CVD indicators (myocardial infarction, stroke, angina pectoris, high blood pressure) reported a measure of methodological quality based on the Scottish Intercollegiate Guidelines Network (Harbour & Miller, 2001), and related this quality measure to study findings. Studies with the highest quality rating (Kuper & Marmot, 2003; Kuper, Singh-Manoux, Siegrist, & Marmot, 2002) were from the Whitehall II cohort, and reported associations between demands and control (and combinations) to risk of CVD. Both of the studies with the highest quality ratings adjusted for an extensive list of “confounders,” including smoking, cholesterol, hypertension, exercise, alcohol consumption, and BMI. Controlling for such factors had only small downward impacts on risk ratios.

Another recent study that examined job stress as a predictor of CVD and that controlled for an extensive list of CVD risk factors was reported by Kivimaki et al. (2011), who used the Whitehall II study of British civil servants and tracked deaths from coronary disease or nonfatal myocardial infarctions in a cohort of 5,533 adults who were free of CVD at baseline. Kivimaki et al. measured job strain and its components (demands and control and support) at three time periods and tracked new cases of CVD for over a decade. As is generally done in epidemiological studies, Kivimaki et al. classified respondents into the high strain quadrant if they had scores on control below the median, scores on demands above the median, and scores on support in the lowest third of the distribution. This classification was done at each of three time periods, and thus they could track the cumulative exposure by adding the number of occasions when respondents were classified into the high strain condition. They found that being classified at two or three phases compared to none or one phase was significantly related to incident CVD both before and after controlling for the Framingham risk score. The Framingham risk score is computed from scores on age, total cholesterol, systolic blood pressure, diabetes mellitus, and current smoking and is the most common tool for classifying for CVD risk. They found that chronic job strain predicted incident CVD after controlling for the Framingham score.

- There is fairly consistent evidence that job stressors—especially those from the JDC and ERI models—are predictive of several indicators of CVD, even after controlling for an array of CVD risk factors representing secondary AL mediators.

*Mental health outcomes.* In the epidemiological literature, most of the emphasis has been on depression (including bipolar disorders) as the main outcome representing mental well-being. This attention to depression seems well-founded in that recent data on the U.S. population (Substance Abuse and Mental Health Services Administration, 2007) indicates that about 7% of full-time workers aged 18 to 64 experienced a major depressive episode in the past year. There are also significant variations across occupations, with rates in occupations such as personal care and service and health care practice being twice as high as in life, physical, and social science occupations and those in engineering, architecture, and surveying. Moreover, across almost all occupations, rates of depression for women are about twice the rate of men. Depression is also a leading cause of disability (World Health Organization, 2001) and sickness absence (Bultman, Rugulies, Lund, Christensen, Labriola, & Burr, 2006). Although occupational differences are likely confounded by SES differences, such



occupational disparities and their association with absence and disability have motivated researchers to explore specific occupational characteristics as possible explanations for occupational variability in depression. In recent years there has been an active epidemiological literature relating depression to such factors as long working hours (Kleppa, Sanne, & Tell, 2008), job demands and control (Blackmore, Stansfeld, Weller, Munce, Zagorski, & Stewart, 2007; Paterniti, Niedhammer, Lang, & Consoli, 2002; Stansfeld, North, White, & Marmot, 1995; Wang, 2004), ERI (Godin, Kittel, Coppieters, & Siegrist, 2005), and organizational injustice (Stansfeld & Candy, 2006).

As with CVD outcomes, the dominant theoretical model tested in epidemiological studies of depression has been the JDC model (e.g., Mausner-Dorsch & Eaton, 2000; Neidhammer, Goldberg, Leclerc, Bugel, & David, 1998; Stansfeld, Fuhrer, Head, & Ferrie, 1997; Stansfeld, Fuhrer, Shipley, & Marmot, 1999). An especially large-sample test of this model was reported by Blackmore et al. (2007), who examined data from the Canadian Community Health Survey. Survey data included multi-item scales for demands, control (decision latitude), social support, job insecurity, and physical exertion. As is customary in this literature, the authors created categorical variables for predictors (4 categories ranging from high to low), and a job strain variable was created by combining high and low categories of demands and control. In addition to examining differences in depression across job strain categories, Blackmore et al. (2007) compared groups based on the individual component variables (also categorized into 4 groups). Depression was measured using a version of the Composite International Diagnostic Interview (based on the *DSM-IV*) and respondents were classified as having (or not) a major depressive episode over the past 12 months (6.0% for women and 3.4% for men). Their results generally replicated those from prior studies, except they found more significant gender differences than reported before; job strain, job demands, and job insecurity were associated with a significantly greater risk of depression for men but not women. Control, however, was a significant predictor for women but not for men.

The JDC model has also been assessed in longitudinal epidemiological studies in which stability and change in job demands and control could be assessed as predictors of depression (deLange, Taris, Kompier, Houtman, & Bongers, 2002; Stansfeld et al., 1999; Wang, Schmitz, Dewa, & Stansfeld, 2009). For example, Wang et al. (2009) used several waves of the Canadian Community Health Survey. Specifically, they used data from the first cohort surveyed in 1994-1995 (Cycle 1) who were then resurveyed in 1996-1997 (Cycle 2), 1998-1999 (Cycle 3), 2000-2001 (Cycle 4), 2002-2003 (Cycle 5), and 2004-2005 (Cycle 6). The initial cohort had a sample size of 17,276, but Wang et al. used data only from those who were interviewed in both Cycle 1 and Cycle 4 ( $N = 4,866$ ). They had access to the same job demands, control, support, and insecurity data as Blackmore et al. (2007), and classified respondents into one of four groups: (a) high job strain in Cycles 1 and 4, (b) low job strain in Cycles 1 and 4, (c) high job strain in Cycle 1 but low job strain in Cycle 4, and (d) low job strain in Cycle 1 but high job strain in Cycle 4. Job strain groups at each time period were formed by calculating the ratio of demands and control scores, such that high strain was when the ratio was greater than 1 and low strain when the ratio was less than 1. Wang et al. computed logistic regression estimates of odds ratios for incident depression in either Cycle 5 or Cycle 6 while controlling for gender, age, education, status of major depression from 1994-95 to 2000-01, perceived health status, and childhood traumatic events at baseline.

Odds of depression were higher than the no-change low strain group for the consistent high strain group ( $OR = 1.52$ ) and the group that changed from low to high job strain, while those who changed from high to low strain did not differ from the consistent low strain group. However, they found a significant interaction between group status and baseline self-rated health status. The consistent high strain group had a significant odds ratio ( $OR = 1.77$ ) if they had reported good or excellent health at baseline but not if they had reported fair or poor health at baseline, suggesting that those in good health are more susceptible to stressful work conditions.

- The most well-researched tertiary mental health indicator in epidemiological research is depression, and research suggests that employees who experience higher levels of job stress (e.g., high job demands and low job control) are more at risk for depression than those who experience lower levels of job stress.

*Other tertiary outcomes.* Although CVD and depression are the most commonly studied disease endpoints associated with work stress, researchers have also examined other relevant outcomes. For example, several studies have considered type 2 diabetes. Although the results seem mixed from cross-sectional studies, there is relatively strong prospective evidence that job strain predicts the onset of diabetes after a 15-year follow-up period and after controlling for a range of confounding factors (Heraclides, Witte, Chandola, & Brunner, 2009). There is also evidence that exposure to work stress is associated with employee health care costs, which could reflect a variety of underlying physical conditions. For example, Ganster et al. (2001) found that job demands interacted with control to predict health insurance claims. The data were averaged over a five-year period, so this measure likely taps into more chronic health conditions that may reflect disease-related endpoints. Interestingly, Ganster et al.'s (2001) results also indicated that cortisol levels after work mediated the effects of the demands-control interaction on health care costs, which is consistent with an AL model framework.

- In addition to cardiovascular disease and depression, longitudinal research indicates that job stress predicts type 2 diabetes and elevated employee health care costs.

## Summary

Our review of the work stress literature indicates that there is consistent evidence supporting a link between work stressors and primary AL indicators for self-reported affective outcomes such as anxiety, job attitudes, and job-related tension, but the evidence is much weaker for short-term physical complaints and physiological responses. Support for the former comes from multiple meta-analyses attesting to the robustness of cross-sectional relationships, but also from convincing within-person correlations generated by repeated measures designs. There is also now a small set of randomized field experiments that have tested interventions that manipulated major stressors such as lack of control and role ambiguity. These experiments have, however, failed to support the large nonexperimental literature regarding both the short-term psychological and

physical health effects of these stressors, despite demonstrating that the interventions had significant impacts on participants' assessments of the targeted stressors. Thus, although there is much correlational evidence suggestive of a causal effect of work stressors on well-being, there is a distinct paucity of evidence that demonstrates that real-world changes in these stressors actually produce changes in well-being.

The picture for physiological indicators of primary AL responses is even more mixed. Laboratory reactivity studies from the basic physiology literature clearly demonstrate the ability of psychosocial stressors that embody a social-evaluative component and lack of personal control to elicit a strong response from the HPA axis, which is a major part of the primary AL response profile. But how such effects might persist over time is not well understood. Relationships in the field between work stressors and cortisol, the most frequently studied primary AL indicator, for example, are inconsistent, in part owing to the complexities of operationalizing this response system. But there are enough positive findings to encourage the further exploration of this important response system.

Our review also indicated that a large body of research has considered relationships between job stressors and a variety of secondary and tertiary AL outcomes. Both categories of outcomes have been investigated with large-sample epidemiological studies that embody some significant methodological strengths. For example, these studies tend to have large samples that are sometimes representative of specific populations, affording plenty of statistical power and generalizability. In addition, some studies have used multiple wave designs in which the consistency of exposure to job stressors can be assessed and related to the outcomes. This is an important feature as it has revealed that chronic exposures over the course of several years show greater risk than consistently low or inconsistent exposures. Such designs are still relatively rare but offer much potential, especially when trying to determine the etiology of secondary and tertiary outcomes that are believed to develop gradually over moderately long (secondary) or very long (tertiary) periods of time. More recent studies also rely less on entirely self-report measurements of stressors. Finally, in general, outcome measurement is acceptably reliable and valid in this set of studies.

Even the stronger designs in this group, however, tend to share some common limitations. For one, the great majority of epidemiological studies have focused on a relatively narrow set of work stressors and measures emanating from a small set of theories. The JDC model's dominance of this literature is beginning to be rivaled by the ERI model and related justice conceptualizations. But the value of this newer research from the ERI model has been strongly criticized based on the very questionable measurement practices used for the theory's constructs (see Ganster & Perrewé, 2011). In contrast, research in the psychology and management literatures, which focuses much more on primary AL indicators, explores a far broader set of stressors and more complex models that incorporate various moderators and mediators. Researchers in the latter camp also use more appropriate analytical approaches when testing some key propositions of the JDC model. This model predicts an interactive (buffering) effect between job demands and control and epidemiologists still mostly rely on additive formulations of job strain (e.g., comparing groups high on job demands and low on control to others) that do not specifically model the theory's core hypothesis. Consequently, then, there is strong evidence that high demands and low control each are associated with

secondary and tertiary AL outcomes, but it is less clear whether control actually shows a buffering effect for high demands.

The strong evidence for an association between demands and control on secondary and tertiary AL outcomes also supports a fairly convincing case for a causal interpretation, despite its nonexperimental nature. The two dominant alternative explanations for a causal interpretation stem from individual differences and other work characteristics that are potentially confounded with demands and control. While some critics have discussed the possible confounding effects of traits such as negative affectivity (Moyle, 1995; Watson & Pennebaker, 1989), we think the most challenging is SES. Because most of the strong evidence comes from large multioccupation studies, SES factors such as income, education, and occupational prestige, which are known to vary by occupation and consistently predict secondary and tertiary AL outcomes, need to be carefully controlled. More studies are currently controlling one or two of these factors, but they would benefit from more thorough coverage. For example, not only personal income, but family income and even neighborhood SES have been associated with individual well-being (Adler et al., 1994; Bradley & Corwyn, 2002; Ferrer-i-Carbonell, 2005), and it is plausible that job demand and especially control constructs are confounded with these factors. The other source of confounding comes from other work and organizational characteristics that tend to covary with job demands and control factors and could explain some of their association with well-being outcomes. A broader coverage of other potential work stressors such as job insecurity, injustice, and abusive supervision would be a significant step forward for the epidemiological literature.

In sum, there is evidence that work stress relates to a variety of outcomes that represent a diverse array of AL indicators. In fact, indicators from all three AL categories are represented in the work stress literature. In the following section, we present recommendations for how the field might proceed, with a focus on identifying specific ways that organizational scientists can contribute to the advancement of our understanding of how exposure to work stress affects employee health and well-being.

## Directions for Future Research

There are many research questions worthy of pursuit in the work stress field and many methodological approaches that are likely to be productive. In the brief space we have here, however, we describe one focus that we believe has much potential; it is one that cuts across multiple disciplines and yet is one in which organizational scholars can have a significant impact. As noted above, research on the tertiary AL outcomes is fairly well advanced. The research needs in that area for better measurement of stressors, a broader conceptual focus, and more extensive control of potential confounders are ones that organizational scholars can help address, but the most productive research strategy is likely to remain that of the large-scale epidemiological investigation. A similar case can be made for the secondary AL mediators because they also reflect longer-term exposures and etiologies. Other writers have provided detailed methodological recommendations about how to best conduct such studies (e.g., Kasl & Jones, 2011).

Research questions about the primary AL processes and indicators are arguably the most conceptually and methodologically complex, but they are critically important for understanding

the basic processes of psychosocial stress, how these processes are enacted in the organizational sphere, and how one can design interventions that will make a positive difference in the well-being of organizational members. Referring to Figure 1 again, the AL model posits a sequential process from acute stress-related responses (primary AL) to longer-term processes (secondary and tertiary AL). Although many research questions remain, the linkage between secondary and tertiary AL outcomes is fairly well established, to the point where secondary outcomes are routinely referred to as risk factors for diseases at the tertiary level. The linkage between primary and secondary AL processes is far less developed, although it is here where the concept of stress really emerges and where stressors from the environment are thought to trigger the physiological processes that drive the subsequent stages. As noted earlier, studies in physiology have yet to show strong relationships between primary and secondary AL indicators, although all are related to tertiary outcomes. In large part we believe that this is because the role of time of exposure to stressors is poorly understood. Whereas laboratory studies demonstrate that psychosocial stressors reliably elicit primary AL responses such as cortisol, we as yet do not know what happens with chronic exposures.

A central proposition of the AL model is that sustained activation of primary AL responses at levels beyond their normal ranges produces dysregulations in those systems themselves as well as secondary AL responses. It is not clear what form such dysregulations take, however. In the case of cortisol, the most studied of the primary processes, such dysregulations can be observed either in changes to aspects of the diurnal pattern (CAR, AUC, flatness of curve, etc.) or in patterns of acute reactivity and recovery to stressors, or both. In either case, how long does exposure need to be before such dysregulations become evident? How long must such dysregulations persist before they affect changes in the more stable secondary AL indicators? Are dysregulations in primary indicators reversible by altering the stressor exposures? Will such reversals lead to changes in the secondary indicators? We believe these are among the most pressing questions concerning the physiology of stress. We also believe that organizational scholars can make significant contributions to this basic literature by conducting work stress studies because work settings are characterized by an abundance of highly salient stressors that naturally occur at levels far beyond what a researcher could practically or ethically manipulate in the laboratory. And unlike laboratory reactivity studies, work settings allow the study of the time factor in physiological processes. Consequently, they provide an appropriate setting for studying AL processes. We thus recommend that work stress researchers focus on primary AL response systems and their association with short-term and longer-term exposures to work stressors. Because primary responses are believed to change much more quickly than those in later stages, they also serve as useful outcome variables in intervention studies and other longitudinal studies in which stressor exposures vary within individuals. Based on our review of the literature, we identified four strategies that would serve to enhance the contributions of future research aimed at studying primary AL responses.

### *Dysregulations of Diurnal Patterns*

Cortisol is the most studied in terms of diurnal patterns, and as noted earlier, there is no consensus yet on which aspects of the diurnal pattern are most important as indicators of

primary AL processes (e.g., CAR, AUC, morning or evening levels). To obtain estimates of these parameters one needs at least three samples, and preferably more. The first sample must be obtained immediately upon awakening and subsequent ones within 30 to 60 minutes. This suggests at least two samples to measure CAR plus an evening sample before bedtime to compute a diurnal slope and the AUC. Some research suggests that the evening measure itself might also be revealing as a measure of lack of recovery from work (Ganster et al., 2001). In addition to obtaining multiple samples throughout the day, one should sample more than one day as well. There is little data about interday stability, but Fox et al. (1993) found that sampling two days produced reliabilities of .60 for midday cortisol and .71 for evening cortisol. Based on these figures, three days would likely yield an adequately reliable assessment of “typical” diurnal patterns. However, Hellhammer et al. (2007) suggest that six days may be necessary to assess CAR in order to adequately separate state from trait effects. The assumption here is that the investigator is interested in the association of chronic work stressors (assessed with a suitable methodology) with the “typical” diurnal pattern, and this represents essentially a trait-trait association. Of course, additional days would need to be sampled if one were interested in testing the effects of chronic work stress on both work day and rest day diurnal patterns.

### *Changes in Acute Reactivity and Recovery*

We also recommend that, in addition (or as an alternative) to *in situ* sampling, researchers should assess reactivity to and recovery from a laboratory stressor as a marker of the impact of chronic work stress on physiological stress systems. Schaubroeck and Ganster (1993) and Hamer, Williams, Vuonovirta, Giacobazzi, Gibson, and Steptoe (2006) are examples of studies that used this combined lab and field strategy to relate work stressor exposures from the field to laboratory reactivity and recovery patterns. For such studies it is advisable to measure multiple parameters (e.g., blood pressure and salivary cortisol). Blood pressure could be monitored throughout the protocol, whereas saliva samples would be obtained at the beginning (prestress), during the stress task, and after the stress task. For cortisol it takes about 20 to 30 minutes for increases in unbound cortisol to show up in saliva, and about 60 to 90 minutes for it to decline to pretask levels after task cessation (Lovallo & Thomas, 2000).

### *Robust Stress Tasks*

Research indicates that stressors vary in their ability to elicit physiological responses and that some stressors are likely to demonstrate only small or negligible effects on reactivity and recovery (Chida & Hamer, 2008; Dickerson & Kemeny, 2004). Therefore, in order to better inform our understanding of the reaction and recovery process, it is critical in laboratory studies that researchers use a stress task that reliably elicits a strong physiological response, as this will provide researchers with the best opportunity for observing and testing the physiological responses specified by the AL model. Dickerson and Kemeny's (2004) review indicates that motivated tasks that incorporate a low level of outcome controllability

and a social-evaluative component are likely to elicit the greatest responses. The Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), in which participants assume the role of a job applicant and perform tasks (i.e., giving a speech and performing oral arithmetic) in front of an audience, combines both (i.e., lack of control and social evaluation) of these elements. As such, this task is likely to elicit a strong physiological response. Thus, it may be useful to organizational scientists to incorporate such tasks into research aimed at understanding various physiological responses to work stressors.

### *Experimental Studies*

To better understand the effects of acute versus chronic exposures to stressors, researchers must study changes in primary AL responses from the beginning of stressor exposure and over a more extended period. One strategy is to study new employees and track them over time, making repeated measurements of both stressors and primary AL indicators. It might be difficult to encounter such an opportunity, however. In such a design the researcher is also not ensured that there will be adequate within-person variation in stressor exposures to detect a measurable effect. Thus, we recommend actively manipulating such exposures, and a good way to do that is through stress intervention studies using either randomized or rigorous quasi-experimental designs. Such a strategy can track changes in primary AL responses from a baseline and through early and later stages of experience with organizational changes. Experimental designs, of course, provide the strongest data for making causal inferences about the effects of work stressors, and they also have the advantage of allowing the study of acute and chronic effects on primary AL systems that other designs would have difficulty doing. Finally, such designs allow investigators to readily study more complex models involving mediators and moderators, including how contextual factors affect the efficacy of stress interventions (e.g., Logan & Ganster, 2005).

### *Field Studies*

Much progress has been made in relating emotional reactions to employee outcomes, including decision making, performance, and counterproductive work behaviors (Estrada, Isen, & Young, 1997; Fox, Spector, & Miles, 2001; Ilies, Scott, & Judge, 2006). It is less clear, however, how dysregulation of primary AL physiological systems affects employee attitudes and behaviors. While there is some evidence that physiological reactivity (e.g., elevations in cortisol levels) predicts health-related outcomes in the longer term, such as health care costs (Ganster et al., 2001), there is relatively little research investigating linkages between cortisol dysregulation and nonhealth outcomes (especially in work contexts). Such research, which has the potential to link dysregulation of primary AL systems to employee and group performance, would be of great interest to management scholars and those interested in developing interventions aimed at enhancing employee functioning and organizational effectiveness. Thus, given this gap in the literature, there is a real opportunity here for organizational scientists to conduct field studies that investigate these linkages in the work context.

## Conclusions

Much progress has been made in the work stress literature over the past two decades, and the basic physiological literature has made parallel empirical and theoretical advances. The AL model has emerged as the central organizing theory for understanding the physiology of stress, and thus we chose it as the organizing mechanism for our review. Yet many questions remain about the physiological processes underlying the experience of all kinds of psychosocial stressors, including those prevalent in the workplace. We believe that the work setting is one that is well suited to exploring the core processes whereby events in the environment initiate a cascade of physiological responses and how they progress over time from acute reactions through stable perturbations in bodily systems to mental and physical well-being. Because work settings provide such high potential to study these processes over time, and even with some experimental control, organizational scholars have an opportunity to contribute not only to our own literature but to an understanding of the underlying physiological processes as well.

## References

- Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. 1994. Socioeconomic status and health: The challenge of the gradient. *American Psychologist*, 49: 15-24.
- Backé, E.-M., Seidler, A., Latza, U., Rossmagel, K., & Schumann, B. 2012. The role of psychosocial stress at work for the development of cardiovascular diseases: A systematic review. *International Archives of Occupational and Environmental Health*, 85: 67-79.
- Belkic, K. L., Landsbergis, P. A., Schnall, P. L., & Baker, M. D. 2004. Is job strain a major source of cardiovascular disease risk? *Scandinavian Journal of Work, Environment, and Health*, 30: 85-128.
- Bellingrath, S., Weigl, T., & Kudielka, B. M. 2009. Chronic work stress and exhaustion is associated with higher allostatic load in female school teachers. *Stress*, 12: 37-48.
- Berkman, L. F., Seeman, T. E., Albert, M., Blazer, D., Kahn, R., Mohs, R., Finch, C., Schneider, E., Cotman, C., McClearn, G., Nesselroade, J., Featherman, D., Garnezy, N., McKhann, G., Brim, G., Prager, D., & Rowe, J. 1993. High, usual, and impaired functioning in community-dwelling older men and women: Findings from the MacArthur Foundation Research Network on Successful Aging. *Journal of Clinical Epidemiology*, 46: 1129-1140.
- Blackmore, E. R., Stansfeld, S. A., Weller, I., Munce, S., Zagorski, B. M., & Stewart, D. E. 2007. Major depressive episodes and work stress: Results from a national population survey. *American Journal of Public Health*, 97: 2088-2093.
- Bond, F. W., & Bunce, D. 2001. Job control mediates change in a work reorganization intervention for stress reduction. *Journal of Occupational Health Psychology*, 6: 290-302.
- Bond, F. W., Flaxman, P. E., & Bunce, D. 2008. The influence of psychological flexibility on work redesign: Mediated moderation of a work reorganization intervention. *Journal of Applied Psychology*, 93: 645-654.
- Bosma, H., Marmot, M., Hemingway, H., Nicholson, A. C., Brunner, E., & Stansfeld, S. A. 1997. Low job control and risk of coronary heart disease in Whitehall II (prospective cohort) study. *British Medical Journal*, 314: 558-565.
- Bradley, R. H., & Corwyn, R. F. 2002. Socioeconomic status and child development. *Annual Review of Psychology*, 53: 371-399.
- Brunner, E. J., Hemingway, H., Walker, B. R., Page, M., Clarke, P., Juneja, M., Shipley, M. J., Kumari, M., Andrew, R., Seckli, J. R., Papadopoulos, A., Checkley, S., Rumley, A., Lowe, G. D. O., Stansfeld, S. A., & Marmot, M. G. 2002. Adrenocortical, autonomic, and inflammatory causes of the metabolic syndrome. *Circulation*, 106: 2659-2665.



- Bultman, U., Rugulies, R., Lund, T., Christensen, K. B., Labriola, M., & Burr, H. 2006. Depressive symptoms and the risk of long-term sickness absence: A prospective study among 4747 employees in Denmark. *Social Psychiatry and Psychiatric Epidemiology*, 41: 875-880.
- Caplan, R. D., Cobb, S., French, J. R. P., Jr., Harrison, R. V., & Pinneau, S. R. 1975. *Job demands and worker health* (Publication No. (NIOSH) 75-160). Washington, DC: U.S. Department of Health, Education, and Welfare.
- Chandola, T., Brunner, E., & Marmot, M. 2006. Chronic stress at work and the metabolic syndrome: Prospective study. *British Medical Journal*, 332: 521-525.
- Chandola, T., Heraclides, A., & Kumari, M. 2010. Psychophysiological biomarkers of workplace stressors. *Neuroscience and Biobehavioral Reviews*, 35: 51-57.
- Cheng, H.-L. G., & Chan, K.-S. D. 2008. Who suffers more from job insecurity? A meta-analytic review. *Applied Psychology: An International Review*, 57: 272-303.
- Chida, Y., & Hamer, M. 2008. Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: A quantitative review of 30 years of investigations. *Psychological Bulletin*, 134: 829-885.
- Chida, Y., & Steptoe, A. 2009. Cortisol awakening response and psychosocial factors: A systematic review and meta-analysis. *Biological Psychology*, 80: 265-278.
- Chida, Y., & Steptoe, A. 2010. Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension*, 55: 1026-1032.
- Cox, T., & Griffiths, A. 2010. Work-related stress: A theoretical perspective. In S. Leka & J. Houdmont (Eds.), *Occupational health psychology*: 57-87. Chichester, UK: Wiley-Blackwell.
- Crawford, E. R., LePine, J. A., & Rich, B. L. 2010. Linking job demands and resources to employee engagement and burnout: A theoretical extension and meta-analytic test. *Journal of Applied Psychology*, 95: 834-848.
- deLange, A. H., Taris, T. W., Kompier, M., Houtman, I., & Bongers, P. 2002. Effects of stable and changing demand-control histories on worker health. *Scandinavian Journal of Work, Environment, and Health*, 28: 94-108.
- Demerouti, E., Bakker, A. B., Nachreiner, F., & Schaufeli, W. B. 2001. The job demands-resources model of burnout. *Journal of Applied Psychology*, 86: 499-512.
- Dickerson, S. S., & Kemeny, M. E. 2004. Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130: 355-391.
- Dienstbier, R. A. 1989. Arousal and physiological toughness: Implications for mental and physical health. *Psychological Review*, 96: 84-100.
- Epel, E. E., McEwen, B. S., Seeman, T., Matthews, K., Castellazzo, G., Brownell, K. D., Bell, J., & Ickovics, J. R. 2000. Stress and body shape: Stress-induced cortisol secretion is consistently greater among women with central fat. *Psychosomatic Medicine*, 62: 623-632.
- Epel, E. E., Moyer, A. E., Martin, C. D., Macary, S., Cummings, N., Rodin, J., & Ebufe-Scrive, M. 1999. Stress-induced cortisol, mood, and fat distribution in men. *Obesity Research*, 7: 9-15.
- Estrada, C. A., Isen, A. M., & Young, M. J. 1997. Positive affect facilitates integration of information and decreases anchoring in reasoning among physicians. *Organizational Behavior and Human Decision Processes*, 72: 117-135.
- Ferrer-i-Carbonell, A. 2005. Income and well-being: An empirical analysis of the comparison income effect. *Journal of Public Economics*, 89: 997-1019.
- Ferris, P. A., Sinclair, C., & Kline, T. J. 2005. It takes two to tango: Personal and organizational resilience as predictors of strain and cardiovascular disease risk in a work sample. *Journal of Occupational Health Psychology*, 10: 225-238.
- Fox, M. L., Dwyer, D. J., & Ganster, D. C. 1993. Effects of stressful job demands and control on physiological and attitudinal outcomes in a hospital setting. *Academy of Management Journal*, 36: 289-318.
- Fox, S., Spector, P. E., & Miles, D. 2001. Counterproductive work behavior (CWB) in response to job stressors and organizational justice: Some mediator and moderator tests for autonomy and emotions. *Journal of Vocational Behavior*, 59: 291-309.
- Fried, Y., Rowland, K., & Ferris, G. 1984. The physiological measurement of work stress: A critique. *Personnel Psychology*, 37: 583-615.

- Gaab, J., Rohleder, N., Nater, U. M., & Ehlert, U. 2005. Psychological determinants of the cortisol stress response: The role of anticipatory cognitive appraisal. *Psychoneuroendocrinology*, 30: 599-610.
- Ganster, D. C. 2005. Executive job demands: Suggestions from a stress and decision-making perspective. *Academy of Management Review*, 30: 492-502.
- Ganster, D. C., Fox, M. L., & Dwyer, D. J. 2001. Explaining employees' health care costs: A prospective examination of stressful job demands, personal control, and physiological reactivity. *Journal of Applied Psychology*, 86: 954-964.
- Ganster, D. C., Mayes, B. T., Sime, W. E., & Tharp, G. D. 1982. Managing organizational stress: A field experiment. *Journal of Applied Psychology*, 67: 533-542.
- Ganster, D. C., & Perrewé, P. L. 2011. Theories of occupational stress. In J. C. Quick & L. E. Tetrick (Eds.), *Handbook of occupational health psychology* (2nd ed.): 37-53. Washington, DC: American Psychological Association.
- Ganster, D. C., & Schaubroeck, J. 1991. Work stress and employee health. *Journal of Management*, 17: 235-271.
- Godin, I., Kittel, F., Coppieters, Y., & Siegrist, J. 2005. A prospective study of cumulative job stress in relation to mental health. *BMC Public Health*, 5: 67. <http://www.biomedcentral.com/1471-2458/5/67>.
- Goldman, N., Turra, C. M., Gleib, D. A., Seplaki, C. L., Lin, Y. H., & Weinstein, M. 2006. Predicting mortality from clinical and nonclinical biomarkers. *Journal of Gerontology: Medical Sciences*, 61A: 1070-1074.
- Goldstein, D. S., & Eisenhofer, G. 2000. Sympathetic nervous system physiology and pathophysiology in coping with the environment. In B. S. McEwen (Ed.), *Handbook of physiology, Coping with the environment*: 21-43. New York: Oxford University Press.
- Grebner, S., Semmer, N. K., & Elfering, A. 2005. Working conditions and three types of well-being: A longitudinal study with self-report and rating data. *Journal of Occupational Health Psychology*, 10: 31-43.
- Greenberg, J. 2010. Organizational injustice as an occupational health risk. *Academy of Management Annals*, 4: 205-243.
- Griffin, M. A., & Clarke, S. 2011. Stress and well-being at work. In S. Zedeck (Ed.), *APA handbook of industrial and organizational psychology*, vol. 3: 359-397. Washington, DC: American Psychological Association.
- Halpern, D. F. 2005. How time-flexible work policies can reduce stress, improve health, and save money. *Stress and Health*, 21: 157-168.
- Hamer, M., Williams, E., Vuonovirta, R., Giacobazzi, P., Gibson, E. L., & Steptoe, A. 2006. The effects of effort-reward imbalance on inflammatory and cardiovascular responses to mental stress. *Psychosomatic Medicine*, 68: 408-413.
- Hansen, A., Larsen, A., Rugulies, R., Garde, A., & Knudsen, L. 2009. A review of the effect of the psychosocial working environment on physiological changes in blood and urine. *Basic & Clinical Pharmacology & Toxicology*, 105: 73-83.
- Harbour, R., & Miller, J. 2001. A new system for grading recommendations in evidence based guidelines. *British Medical Journal*, 323: 334-336.
- Harrison, R. V. 1985. The person-environment fit model and the study of job stress. In T. A. Beehr & R. S. Bhagat (Eds.), *Human stress and cognition in organizations*: 23-55. New York: John Wiley.
- Heaphy, E., & Dutton, J. 2008. Positive social interactions and the human body at work: Linking organizations and physiology. *Academy of Management Review*, 33: 137-162.
- Heim, C., Ehlert, U., & Hellhammer, D. H. 2000. The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, 25: 1-35.
- Hellhammer, J., Fries, E., Schweisthal, O. W., Schlotz, W., Stone, A. A., & Hagemann, D. 2007. Several daily measurements are necessary to reliably assess the cortisol rise after awakening: State and trait components. *Psychoneuroendocrinology*, 32: 80-86.
- Hemingway, H., & Marmot, M. 1999. Psychosocial factors in the aetiology and prognosis of coronary heart disease: A systematic review of prospective cohort studies. *British Medical Journal*, 318: 1460-1467.
- Heopniemi, T., Elovainio, M., Pulkki, L., Puttonen, S., Raitakarai, O., & Keltigangas-Jarvinen, L. 2007. Cardiac autonomic reactivity and recovery in predicting carotid atherosclerosis: The cardiovascular risk in young Finns study. *Health Psychology*, 26: 13-21.
- Heraclides, A., Witte, D. R., Chandola, T., & Brunner, E. J. 2009. Psychosocial stress at work doubles the risk for type 2 diabetes in middle-aged women. *Diabetes Care*, 32: 2230-2235.

- Herschcovis, M. S., & Barling, J. 2010. Towards a multi-foci approach to workplace aggression: A meta-analytic review of outcomes from different perpetrators. *Journal of Organizational Behavior*, 31: 24-44.
- Hobfoll, S. E. 1998. *Stress, culture, and community: The psychology and philosophy of stress*. New York: Plenum.
- Hobfoll, S. E. 2001. The influence of culture, community, and the nested-self in the stress process: Advancing Conservation of Resources theory. *Applied Psychology: An International Review*, 50: 337-421.
- Holleman, M., Vreeburg, S. A., Dekker, J., & Penninx, B. 2012. The relationships of working conditions, recent stressors and childhood trauma with salivary cortisol levels. *Psychoneuroendocrinology*, 37: 801-809.
- Holman, D. J., Axtell, C. M., Sprigg, C. A., Totterdell, P., & Wall, T. D. 2010. The mediating role of job characteristics in job redesign interventions: A serendipitous quasi-experiment. *Journal of Organizational Behavior*, 31: 84-105.
- Ilies, R., Dimotakis, N., & de Pater, I. 2010. Psychological and physiological reactions to high workloads: Implications for well-being. *Personnel Psychology*, 63: 407-436.
- Ilies, R., Johnson, M. D., Judge, T. A., & Keeney, J. 2011. A within-individual study of interpersonal conflict as a work stressor: Dispositional and situational moderators. *Journal of Organizational Behavior*, 32: 44-64.
- Ilies, R., Scott, B. A., & Judge, T. A. 2006. The interactive effects of personal traits and experienced states on intraindividual patterns of citizenship behavior. *Academy of Management Journal*, 49: 561-575.
- Juster, R.-P., McEwen, B. S., & Lupien, S. J. 2010. Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews*, 35: 2-16.
- Kahn, R. L., & Byosiere, P. 1992. Stress in organizations. In M. D. Dunnette & L. M. Hough (Eds.), *Handbook of industrial and organizational psychology*, vol. 3, no. 2: 571-650. Palo Alto, CA: Consulting Psychologists Press.
- Kahn, R. L., Wolfe, D. M., Quinn, R. P., Snoek, J. D., & Rosenthal, R. A. 1964. *Organizational stress: Studies in role conflict and ambiguity*. New York: John Wiley.
- Karasek, R. 1979. Job demands, job decision latitude, and mental strain: Implications for job redesign. *Administrative Science Quarterly*, 24: 285-306.
- Karasek, R., Brisson, C., Kawakami, N., Houtman, I., Bongers, P., & Amick, B. 1998. The Job Content Questionnaire (JCQ): An instrument for internationally comparative assessments of psychosocial job characteristics. *Journal of Occupational Health Psychology*, 3: 322-355.
- Karasek, R., & Theorell, T. 1990. *Healthy work: Stress, productivity, and the reconstruction of working life*. New York: Basic Books.
- Karlsen, B., Eek, F., Hansen, A. M., Garde, A. H., & Orbaek, P. 2011. Cortisol variability and self-reports in the measurement of work-related stress. *Stress and Health*, 27, e11-e24.
- Kasl, S. V., & Jones, B. A. 2011. An epidemiological perspective on research design, measurement, and surveillance strategies. In J. C. Quick & L. E. Tetrick (Eds.), *Handbook of occupational health psychology* (2nd ed.): 375-394. Washington, DC: American Psychological Association.
- Kirschbaum, C., & Hellhammer, D. H. 1989. Salivary cortisol in psychobiological research: An overview. *Neuropsychobiology*, 22: 150-169.
- Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. 1993. The "Trier Social Stress Test"—A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28: 76-81.
- Kivimaki, M., Nyberg, S. T., Batty, G. D., Shipley, M. J., Ferrie, J. E., Virtanen, M., Marmot, M. G., Vahtera, J., Singh-Manoux, A., & Hamer, M. 2011. Does adding information on job strain improve risk prediction for coronary heart disease beyond the standard Framingham risk score? The Whitehall II study. *International Journal of Epidemiology*, 40: 1577-1584.
- Kivimaki, M., Virtanen, M., Elovainio, M., Kouvonen, A., Vaananen, A., & Vahtera, J. 2006. Work stress in the etiology of coronary heart disease: Systematic review and meta-analysis of prospective cohort studies. *Scandinavian Journal of Work, Environment, and Health*, 32: 431-442.
- Kleppa, E., Sanne, B., & Tell, G. S. 2008. Working overtime is associated with anxiety and depression: The Hordaland Health Study. *Journal of Occupational and Environmental Medicine*, 50: 658-666.
- Kunz-Ebrecht, S. R., Kirschbaum, C., Marmot, M., & Steptoe, A. 2004. Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoendocrinology*, 29: 516-528.
- Kuper, H., & Marmot, M. 2003. Job strain, job demands, and decision latitude, and risk of coronary heart disease within the Whitehall II study. *Journal of Epidemiology and Community Health*, 57: 147-153.

- Kuper, H., Marmot, M., & Hemingway, H. 2002. Systematic review of prospective cohort studies of psychosocial factors in the etiology and prognosis of coronary heart disease. *Seminars in Vascular Medicine*, 2: 267-314.
- Kuper, H., Singh-Manoux, A., Siegrist, J., & Marmot, M. 2002. When reciprocity fails: Effort-reward imbalance in relation to coronary heart disease and health functioning within the Whitehall II study. *Occupational and Environmental Medicine*, 59: 777-784.
- Landsbergis, P. A., Schnall, P. L., Belkic, K. L., Baker, D., Schwartz, J. E., & Pickering, T. G. 2011. Workplace and cardiovascular disease: Relevance and potential role for occupational health psychology. In J. C. Quick & L. E. Tetrick (Eds.), *Handbook of occupational health psychology* (2nd ed.): 243-264. Washington, DC: American Psychological Association.
- Lazarus, R. S. 1966. *Psychological stress and the coping process*. New York: McGraw-Hill.
- Lee, R. T., & Ashforth, B. A. 1996. A meta-analytic examination of the correlates of the three dimensions of job burnout. *Journal of Applied Psychology*, 81: 123-133.
- LePine, J. A., LePine, M. A., & Jackson, C. L. 2004. Challenge and hindrance stress: Relationships with exhaustion, motivation to learn, and learning performance. *Journal of Applied Psychology*, 89: 883-891.
- Li, W., Zhang, J. Q., Sun, J., Ke, J. H., Dong, Z. Y., & Wang, S. 2007. Job stress related to glycol-lipid allostatic load, adiponectin and vistafin. *Stress and Health*, 23: 257-266.
- Light, K. C., Turner, J. R., & Hinderliter, A. L. 1992. Job strain and ambulatory work blood pressure in healthy young men and women. *Hypertension*, 20: 214-218.
- Logan, M. S., & Ganster, D. C. 2005. An experimental evaluation of a control intervention to alleviate job-related stress. *Journal of Management*, 31: 90-107.
- Lovallo, W., & Thomas, T. 2000. Stress hormones in psychophysiological research. In J. Cacioppo, L. Tassinary & G. Bernston (Eds.), *Handbook of psychophysiology* (2nd ed.): 342-367. Cambridge, UK: Cambridge University Press.
- Lundberg, U., & Hellstrom, B. 2002. Workload and morning salivary cortisol in women. *Work & Stress*, 16: 356-363.
- Lupien, S. J., Ouellet-Morin, I., Hupbach, A., Tu, M. T., Buss, C., Walker, D., Pruessner, J., & McEwen, B. S. 2006. Beyond the stress concept: Allostatic load—a developmental biological and cognitive perspective. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, volume 2: Developmental neuroscience*: 578-628. Hoboken, NJ: John Wiley.
- Maslach, C. 1982. *Burnout: The cost of caring*. New York: Prentice-Hall.
- Mausner-Dorsch, H., & Eaton, W. W. 2000. Psychosocial work environment and depression: Epidemiological assessment of the demand-control model. *American Journal of Public Health*, 90: 1765-1770.
- McEwen, B. S. 2007. Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, 87: 873-904.
- McEwen, B. S., & Seeman, T. E. 1999. Protective and damaging effects of mediators of stress. In N. E. Adler, M. Marmot & B. S. McEwen (Eds.), *Socioeconomic status and health in industrial nations: Social, psychological and biological pathways*: 30-47. New York: Academic Sciences.
- McEwen, B. S., & Stellar, E. 1993. Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine*, 153: 2093-2101.
- Meurs, J. A., & Perrewé, P. L. 2011. Cognitive activation theory of stress: An integrative theoretical approach to work stress. *Journal of Management*, 37: 1043-1068.
- Mikkelsen, A., Saksvik, P. O., & Landsbergis, P. 2000. The impact of participatory organizational intervention on job stress in community health care institutions. *Work & Stress*, 14: 156-170.
- Moyle, P. 1995. The role of negative affectivity in the stress process: Tests of alternative models. *Journal of Organizational Behavior*, 16: 647-668.
- Neidhammer, I., Goldberg, M., Leclerc, A., Bugel, I., & David, S. 1998. Psychosocial factors at work and subsequent depressive symptoms in the GAZEL cohort. *Scandinavian Journal of Work, Environment, and Health*, 24: 197-205.
- Nixon, A. E., Mazzola, J. J., Bauer, J., Krueger, J. R., & Spector, P. E. 2011. Can work make you sick? A meta-analysis of the relationships between job stressors and physical symptoms. *Work & Stress*, 25: 1-22.
- Paterniti, S., Niedhammer, I., Lang, T., & Consoli, S. M. 2002. Psychosocial factors at work, personality traits and depressive symptoms: Longitudinal results from the GAZEL study. *British Journal of Psychiatry*, 181: 111-117.

- Perrewé, P. L., Zellars, K. L., Ferris, G. R., Rossi, A. M., Kacmar, C. J., & Ralston, D. A. 2004. Neutralizing job stressors: Political skill as an antidote to the dysfunctional consequences of role conflict. *Academy of Management Journal*, 47: 141-152.
- Podsakoff, N. P., LePine, J. A., & LePine, M. A. 2007. Differential challenge stressor-hindrance stressor relationships with job attitudes, turnover intentions, turnover, and withdrawal behavior: A meta-analysis. *Journal of Applied Psychology*, 92: 438-454.
- Rose, R. M., Jenkins, C. D., Hurst, M., Herds, J. A., & Hall, R. P. 1982. Endocrine activity in air traffic controllers at work: II. Biological, psychological and work correlates. *Psychoendocrinology*, 7: 113-123.
- Rystedt, L. W., Cropley, M., Devereux, J. J., & Michalianou, G. 2008. The relationship between long-term job strain and morning and evening saliva cortisol secretion among white-collar workers. *Journal of Occupational Health Psychology*, 13: 105-113.
- Schaubroeck, J., & Ganster, D. C. 1993. Chronic demands and responsivity to challenge. *Journal of Applied Psychology*, 78: 73-85.
- Schaubroeck, J., Ganster, D. C., & Kemmerer, B. E. 1994. Job complexity, type A behavior, and cardiovascular disorder: A prospective study. *Academy of Management Journal*, 37: 426-439.
- Schaubroeck, J., Ganster, D. C., Sime, W., & Dittman, D. 1993. A field experiment testing supervisory role clarification. *Personnel Psychology*, 46: 1-25.
- Schaubroeck, J., & Merritt, D. E. 1997. Divergent effects of job control on coping with work stressors: The key role of self-efficacy. *Academy of Management Journal*, 40: 738-754.
- Schlottz, W., Hellhammer, J., Schulz, P., & Stone, P. 2004. Perceived workload and chronic worrying predict weekend-weekday differences in the cortisol awakening response. *Psychosomatic Medicine*, 66: 207-214.
- Schnall, P., Landsbergis, P., & Baker, D. 1994. Job strain and cardiovascular disease. *Annual Review of Public Health*, 15: 381-411.
- 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.
- Schnall, P. L., Schwartz, J. E., Landsbergis, P. A., 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.
- Schuler, J. L. H., & O'Brien, W. H. 1997. Cardiovascular recovery and hypertension risk factors: A meta-analytic review. *Psychophysiology*, 34: 649-659.
- Seeman, E., McEwen, B. S., Rowe, J. W., & Singer, B. H. 2001. Allostatic load as a marker of cumulative biological risk: MacArthur Studies of Successful Aging. *Proceedings of the National Academy of Sciences of the United States of America*, 98: 4770-4775.
- Seeman, E., Singer, B. H., Rowe, J., Horwitz, R. I., & McEwen, B. 1997. Price of adaptation—allostatic load and its health consequences: MacArthur Studies of Successful Aging. *Archives of Internal Medicine*, 157: 2259-2268.
- Selye, H. 1955. Stress and disease. *Science*, 122: 625-631.
- Semmer, N. K., Grebner, S., & Elfering, A. 2004. Beyond self-report: Using observational, physiological, and situation-based measures in research on occupational stress. *Research in Occupational Stress and Well Being*, 3: 205-263.
- Shadish, W. R., Cook, T. D., & Campbell, D. T. 2002. *Experimental and quasi-experimental designs for generalized causal inference*. Boston: Houghton-Mifflin.
- Shaw, J. D., & Gupta, N. 2004. Job complexity, performance, and well-being: When does supplies value fit matter? *Personnel Psychology*, 57: 847-879.
- Shirom, A., Toker, S., Berliner, S., & Shapira, I. 2008. The job demand-control-support model and stress-related low-grade inflammatory responses among healthy employees: A longitudinal study. *Work & Stress*, 22: 138-152.
- Siegrist, J. 2002. Effort-reward imbalance at work and health. In P. L. Perrewé & D. C. Ganster (Eds.), *Research in occupational stress and well-being. Volume 2: Historical and current perspectives on stress and health*: 261-291. Amsterdam: Elsevier/JAI.
- Smith, G. D., Ben-Shlomo, Y., Beswick, A., Yarnell, J., Lightman, S., & Elwood, P. 2005. Cortisol, testosterone, and coronary heart disease: Prospective evidence from the Caerphilly study. *Circulation*, 112: 332-340.
- Sonnentag, S., & Fritz, C. 2006. Endocrinological processes associated with job stress: Catecholamine and cortisol responses to acute and chronic stressors. In P. Perrewé & D. C. Ganster (Eds.), *Research in occupational stress and well-being*, vol. 5: 1-59. Amsterdam: Elsevier/JAI.

- Sonnentag, S., & Zijlstra, F. R. H. 2006. Job characteristics and off-job activities as predictors of need for recovery, well-being, and fatigue. *Journal of Applied Psychology*, 91: 330-350.
- Spector, P. E., Zapf, D., Chen, P. Y., & Frese, M. 2000. Why negative affectivity should not be controlled in job stress research: Don't throw the baby out with the bath water. *Journal of Organizational Behavior*, 21: 79-95.
- Stansfeld, S., & Candy, B. 2006. Psychosocial work environment and mental health—A meta-analytic review. *Scandinavian Journal of Work, Environment and Health*, 32: 443-462.
- Stansfeld, S., Fuhrer, R., Head, J., & Ferrie, J. 1997. Work and psychiatric disorder in the Whitehall II study. *Journal of Psychosomatic Research*, 43: 73-81.
- Stansfeld, S., Fuhrer, R., Shipley, M. J., & Marmot, M. G. 1999. Work characteristics predict psychiatric disorder: Prospective results from the Whitehall II study. *Occupational and Environmental Medicine*, 56: 302-307.
- Stansfeld, S., 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.
- Steptoe, A., & Wardle, J. 2005. Cardiovascular stress responsivity, body mass and abdominal adiposity. *International Journal of Obesity*, 29: 1329-1337.
- Sterling, P., & Eyer, J. 1988. Allostasis: A new paradigm to explain arousal pathology. In S. Fisher & J. Reason (Eds.), *Handbook of life stress, cognition, and health*: 629-649. New York: John Wiley.
- Stone, A. A., Schwartz, J. E., Smyth, J., Kirschbaum, C., Cohen, S., Hellhammer, D., & Grossman, S. 2001. Individual differences in the diurnal cycle of salivary free cortisol: A replication of flattened cycles for some individuals. *Psychoneuroendocrinology*, 26: 295-306.
- Substance Abuse and Mental Health Services Administration, Office of Applied Studies. 2007, October 11. *The NSDUH report: Depression among adults employed full time, by occupational category*. Rockville, MD. <http://oas.samhsa.gov/2k7/depression/occupation.htm>. Accessed January 14, 2012.
- Sun, J., Wang, S., Zhang, J.-Q., & Li, W. 2007. Assessing the cumulative effects of stress: The association between job stress and allostatic load in a large sample of Chinese employees. *Work & Stress*, 21: 333-347.
- Sverke, M., Hellgren, J., & Naswell, K. 2002. No security: A meta-analysis and review of job insecurity and its consequences. *Journal of Occupational Health Psychology*, 7: 242-264.
- Teuchmann, K., Totterdell, P., & Parker, S. 1999. Rushed, unhappy, and drained: An experience sampling study of relations between time pressure, perceived control, mood, and emotional exhaustion in a group of accountants. *Journal of Occupational Health Psychology*, 4: 37-54.
- Thompson, M. S., & Cooper, C. L. 2001. A rose by any other name . . . : A commentary on Hobfoll's conservation of resources theory. *Applied Psychology*, 50: 408-418.
- Totterdell, P., Wood, S., & Wall, T. 2006. An intra-individual test of the demands-control model: A weekly diary study of psychological strain in portfolio workers. *Journal of Occupational and Organizational Psychology*, 79: 63-84.
- Wang, J. 2004. Perceived work stress and major depressive episodes in a population of employed Canadians over 18 years old. *Journal of Nervous and Mental Disorders*, 19: 160-163.
- Wang, J., Schmitz, N., Dewa, C., & Stansfeld, S. 2009. Changes in perceived job strain and the risk of major depression: Results from a population-based longitudinal study. *American Journal of Epidemiology*, 169: 1085-1091.
- Watson, D., & Pennebaker, J. W. 1989. Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review*, 96: 234-254.
- World Health Organization (WHO). 2001. *Mental health: New understanding, new hope*. World health report 2001. Geneva: WHO.
- Xie, J. L., & Johns, G. 1995. Job scope and stress: Can job scope be too high? *Academy of Management Journal*, 38: 1288-1309.
- Xie, J. L., Schaubroeck, J., & Lam, S. S. K. 2008. Theories of job stress and the role of traditional values: A longitudinal study in China. *Journal of Applied Psychology*, 93: 831-848.
- Zajonc, R. B. 1984. On the primacy affect. *American Psychologist*, 39: 117-123.
- Zellars, K. L., Meurs, J. A., Perrewé, P. L., Kacmar, C. J., & Rossi, A. M. 2009. Reacting and recovering from a stressful situation: The negative affectivity-physiological arousal relationship. *Journal of Occupational Health Psychology*, 14: 11-22.