NOTE TO USERS

Examining the Relationship between Stress and Absenteeism: A Research Synthesis

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ABSTRACT

Examining the Relationship between Stress and Absenteeism: A Research Synthesis

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Work stress and stress-related illness have been touted as significant causes of absenteeism in the popular press. Yet, solid research evidence for a stress-absence connection is currently lacking. Methodological variation across primary studies and multiple theoretical viewpoints preclude a comprehensive understanding of the association between these two variables. The purpose of this research was to synthesize and meta-analyze accumulated findings on the topic. The theory of causation was used to explore and build hypotheses about the nature of the stress-absence relationship, including mediating processes and moderating influences. Findings from 137 studies and 275 effects provide support for a positive causal relationship between stress and absenteeism, and confirm the operation of illness mediating processes. In addition, there is evidence suggesting that absenteeism might have the potential to play a maintenance role in regulating subsequent levels of stress and illness. Little support was obtained for the underlying voluntary-involuntary distinction between frequency and time lost measures of absenteeism. Among the individual-level moderators, the influence of attribution and disposition were confirmed, while the macro social context was the only contextual moderator to receive support. Findings shed light on many theoretical viewpoints, and provide a comprehensive understanding of work stress and absenteeism.

ACKNOWLEDGEMENTS

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CHAPTER ONE

1. Problem Statement and Objectives

Work stress has received tremendous attention over the years. The rise in stress-related publications, which began almost 25 years ago (Barley & Knight, 1992), is even more noticeable as a recent Internet search using the term "work stress" yielded approximately 16 million hits. The buzz surrounding this topic is largely due to associated direct costs to organizations in the form of lost wages due to absenteeism, and indirect ones such as employee health insurance claims, reduced productivity, and overtime wages. Work stress and stress-related illness, regarded as causes of absenteeism in the popular press, have been estimated to account for about 60 to 75 percent of all work time lost (Adams, 1987; Cartwright, 2000, Kearns, 1986). In a national survey of U.S. organizations, human resources executives reported that 47 percent of all unscheduled absence in their organization was due to stress or personal illness (CCH Inc., 2002). While work stress is estimated to cost Canadian businesses $16 billion per year, American industry is said to lose $300 billion (Tangri, 2003). According to the Health and Safety Executive board in the United Kingdom, stress-related costs to U.K. employers are estimated between £353 and £381 million per year (Mackay, Cousins Kelly, Lee, & McCaig, 2004). While these figures include both direct and indirect costs, the Canadian Policy Research Network estimates that stress-related absenteeism alone costs employers $3.5 billion annually (Williams & Normand, 2003).

These estimated organizational costs of stress-related absenteeism suffice to show that the issue is worth some attention. However, there is a greater need to integrate accumulated research on work stress and absenteeism, because methodological variation

across primary studies and multiple theoretical viewpoints preclude a comprehensive understanding of the association between these two variables. Although Farrell and Stamm (1988) provided a meta-analytical estimate of the association between psychological stress and absenteeism, the analysis was based on four samples only, and does not provide much insight on the topic. The common argument in this research arena is that stress causes employees to temporarily withdraw from the workplace in the form of absenteeism (Hill & Trist, 1955). Illness mediating processes are assumed to underlie this relationship such that stress weakens an employee's physical ability to attend work, which in turn causes one to be absent. Although previous research syntheses have demonstrated a link between stress and underlying physiology or immune functioning (Cohen & Williamson, 1991; O'Leary, 1990; Segerstrom & Miller, 2004), connections between stress, psychological illness, and somatic symptoms are less clear. Stress researchers have tended to use these terms interchangeably. Consequently, little can be said about the assumed medical mediation model of absenteeism.

The purpose of this research is to meta-analyze accumulated findings on work stress and absenteeism, in a manner that enables a test for various theoretical models of absenteeism. As the primary objective is to determine the status of stress as a causal explanation for absenteeism, the literature review is organized around three questions pertaining to Campbell's (1967) theory of causation: does stress influence absence?, does absence influence stress?, and is a third factor responsible for the stress-absence association? Within each of these three general questions, some of the specific queries explored pertain to the mediating connection between stress and absenteeism, underlying voluntary-involuntary dimensionality of frequency and time lost absence measures, the

restorative function of absenteeism, and moderating influences of individual (e.g.,

disposition) and contextual (e.g., occupational status) factors on the stress-absence effect.

Rather than provide an exhaustive coverage of the area, this research focuses on key

issues, building related hypotheses that are to be empirically tested against accumulated

research. For exhaustive narrative reviews on absenteeism, readers are directed to

Harrison and Martocchio (1998), and Johns (1997, 2001a, 2002).

CHAPTER TWO

2. Constructs, Concepts, and Variables

Before delving into questions about the relationship between stress and absence, it is necessary to briefly understand the domain of interest in this paper, as a variety of variables and terminology have been used to reflect absenteeism (Gaudet, 1963) and stress (Beehr & Franz, 1987).

2.1. *Absenteeism*

As an indicator of the broad work withdrawal construct, which includes lateness and turnover among other behaviors (Hanisch & Hulin, 1990), absenteeism is defined as the failure to report for scheduled work (Johns, 2002). Despite some debate over the treatment of these various work withdrawal behaviors as separate (e.g., Hanish & Hulin), this research focuses on absenteeism, because the objective is to obtain an in-depth understanding of this particular behavior's relationship with stress (c.f. Johns, 1998). This focus also allows for an examination of various theoretical frameworks around which a portion of absenteeism research has accumulated.

As Hackett and Guion (1985) discussed, the measurement of absenteeism has been a problem in the field, because a variety of different operationalizations have been used. The two most common individual-level measures of absenteeism are frequency and time lost indices (Chadwick-Jones, Nicholson, & Brown, 1982; Hackett & Guion, 1985). The attitudinal index, which measures single day absences in a given period is also used to some extent, and is included in this examination. Frequency measures are obtained by calculating the number of times a person is absent, where as time lost measures are calculated as the total number of days that a person is absent in a given

period. As an example, consider a person who was absent twice in the last month. The first absence episode was only a day long, while the second absence episode was three days long. In this example, a time lost measure of absenteeism would calculate the person to be absent four days in the last month, the frequency measure would indicate the person to be absent twice in the last month, while an attitudinal measure of absenteeism would calculate the person to be absent only once in the last month.

Measures of absenteeism in terms of hours (e.g., total hours lost) or rate of absence (e.g., days lost/total days worked in a given period) are also regarded as time lost indices. In addition to measuring the number of times or days of absence, some researchers also examine the reason or purpose for an absence. For example, absences supposedly due to illness are generally studied as sickness absence in the epidemiological literature (e.g., North, Syme, Feeney, Head, Shipley & Marmot, 1993). Non-medical reasons for absence such as personal, transportation, and family have also been examined, but less frequently (e.g., Kohler & Mathieu, 1993). Some researchers further distinguish between short-term sickness absences (also referred to as self-certified absences) and long-term or medically-certified absences (e.g. Vahtera, Kivimaki & Pentti, 1997). Absenteeism data has largely been gathered through employee self-reports (e.g., Landerweerd & Boumans, 1994) and company records (e.g., Martocchio, 1994). Obtained absence measures are often collected over varying periods of time, and are aggregated to form a composite measure of absenteeism. For example, Mowday and Spencer (1981) aggregated absences over a one-year period. In the discussions to follow, the term 'absence' is used in a general sense. Reference to a particular aspect of absenteeism is made whenever theoretically relevant, or when hypotheses are built

around a specific measure of absence.

2.2. Stress

A number of definitions and conceptualizations of stress exist in the literature. Ganster and Schaubroeck's (1991) overview illustrates how varied conceptualizations of stress evolved as different theoretical models came be to recognized and used by stress researchers. Selye's (1974) general adaptation model led researchers to focus on objective characteristics of the environment. For example, Kahn, Wolfe, Quinn, Snoek, and Rosenthal (1964) and Beehr (1976) define work stress in terms of its sources. Organizational and personal factors such role ambiguity, role conflict, job characteristics, job security, family situation, and socio-economic status are, therefore, proposed to adversely affect individuals. The P-E fit theory (Caplan, Cobb, French, Van Harrison, & Pinneau, 1975) and Karasek's job demands model (1979), on the other hand, led researchers to describe stress as a state of tension resulting from an imbalance between demands and resources. These models focus on the processes through which work features are experienced as stressful. Finally, Lazarus' (1990) transactional model conceptualizes stress in terms of an individual's subjective appraisal of the environment.

As reflected above, Kahn and Byosiere (1992) noted that there is no agreement among researchers on whether the term 'stress' should be used to refer to objective stimuli in the work setting, the subjective perception of these stimuli, or subsequent individual responses. For the purpose of this research, I adapt Schuler's (1982) definition of stress, defining it as the perceived uncertainty about coping with demands. This conceptualization is consistent with the transactional model of stress (Lazarus, 1990), in which a reciprocal relationship between the individual's environment and his/her

responses is assumed. The model is dynamic, and captures a person's responses over time. The model also helps maintain a distinction between stressors, stress, and stress responses. For example, the model categorizes organizational conditions as antecedents or stressors that give rise to certain cognitive (e.g., perceptions, evaluations) and physiological (e.g., chemical, neurological) processes, which in turn result in physical (e.g., headaches, cancer), psychological (e.g., anxiety, fatigue), and behavioural (e.g., dispensary visits, overeating) consequences in the individual (cf. Beehr & Newman, 1978; Schuler, 1982, 1984). In the present investigation, the focus is on stress and its responses. Despite recent references to positive aspects of stress (e.g., Boswell, Olson-Buchanan, & LePine, 2004), this research regards stress as an adverse condition as does the bulk of research on absenteeism.

Consistent with past researchers' focus on chronic or long-term organizational stressors and stress (Anshel, Robertson, & Caputi, 1997; Beehr & Franz, 1987), the present study is primarily concerned with chronic work stress. However, some stress researchers have also examined stress responses and absenteeism in the context of some short-term traumatic event. For example, Kushnir, Fried, and Malkinson (2001) examined employee emotional reactions to the assassination of a Prime Minister, whereas Byron and Peterson (2002) examined certain outcomes in relation to the 9-11 terrorist attacks. When examined in the context of such events, measures of stress are likely to reflect acute rather than chronic stress (Harvey & Bryant, 2002). Depending on the number of such studies, acute stress-absenteeism associations will be examined post-hoc.

Measures of stress, which most closely correspond to the conceptualization adopted in this study, include Spielberger and Reheiser's (1994) Job Stress Survey, which

asks respondents to rate the amount of stress they associate with a number of job stressors. Davidson and Cooper's (1983) Survey of Work Pressure, which assesses the amount of pressure experienced by an individual on a number of work-related factors listed in the survey, is another example. While the bulk of stress research has used subjective or self-report measures, a few have used objective measures of stress such as heart-rate and blood pressure (Folger & Belew, 1985). These appear to correspond more closely with the physiological responses described in Beehr and Newman's (1978) model, and will be examined separately, depending on their frequency. Conversely, measures assessing work demands such as Hackman and Oldham's (1975) Job Diagnostic Survey, or Rizzo, House and Lirtzman's (1970) measures of role conflict and role ambiguity are regarded as assessing work stressors, and are not examined in the present research.

2.3. Illness

As described earlier, the stress response includes physical, psychological, and behavioural reactions to some stressor. There is, however, some confusion over the categorization of these responses as stress versus health or illness outcomes (Beehr & Franz, 1987). Researchers have interchangeably referred to psychological and physical outcomes as symptoms of stress (e.g., Lee, 1997) and as symptoms of illness (e.g., Spector & Jex, 1998). This confusion is also reflected in Fleming and Baum's (1987) review on the assessment of stress. In building a stress-health connection in their paper, they refer to symptoms such as headaches, depression, and anxiety as being health-related consequences of stress. Yet, these same variables appear in the Symptom Checklist (Derogatis, 1977), often used as a self-report measure of stress. In addition, Fleming and

Baum's section on health consequences of stress includes only studies examining hypertension and coronary heart disease.

Literature reviews on health also reflect the interchangeable use of stress and health concepts. Included in Danna and Griffin's (1999) conceptualization of health are depression, anxiety, and psychosomatic symptoms, in addition to perceived stress itself (p. 363). Similarly, Baba, Jamal, and Tourigny (1998) acknowledge the multiple facets of mental health, and varied manifestations such as stress, anxiety, and depression surface in their discussion on this construct. In Cohen and Williamson's (1991) review of stress and disease, measures of stressful life events, depression, mood states, general health, and some personality indices were collectively referred to as stress, while reports of upper respiratory tract infections (e.g., colds, influenza, flu), herpes virus infections (e.g., cold sores), and bacterial infections (e.g., disease severity) were categorized as illnesses. As evident above, there appears to be no clear distinction between the concepts of stress and illness. To provide some clarity, I continue to refer to the transactional process framework (e.g., Beehr & Newman, 1978; Schuler, 1984).

In this framework, variables such as anxiety, fatigue, and depression among others are referred to as *psychological* health consequences of stress, while cardiovascular disease, headaches, and gastrointestinal problems are regarded as somatic or *physical* health consequences (Beehr & Newman, 1978). This distinction is also maintained in Miller and Ingham's (1976) examination of illness, which regarded backache, palpitations, dizziness, and breathlessness as physical symptoms, and tiredness, anxiety, depression, and irritability as psychological ones. The distinction between psychological and physical illness variables is important, given some suggestion that

stress-triggered physical illnesses follow the development of psychological ones (Frese & Zapf, 1988). Consistent with Cohen and Williamson's (1991) discussion, I acknowledge the distinction between signs and symptoms of illness. While signs of illness include objective measures such as x-ray reports or visible lesions, symptoms are based on patient self-reports and as such are not true reflections of underlying illness pathology. As the bulk of stress research has relied on self-report assessments, measures of psychological outcomes such as anxiety and depression are regarded as psychological *symptoms* of illness, and physical outcomes such as headaches and stomach problems are viewed as *symptoms* of physical or somatic illness. The terms "symptoms" and "illness" are used interchangeably in this paper, but are acknowledged as representing unverified illness (cf. Cohen & Williamson, 1991).

Measures typically used in research to assess psychological and somatic illness include the General Health Questionnaire (Goldberg, 1972) and the Somatic Complaints Index (Caplan et al., 1980). Also examined among the psychological outcomes of stress is burnout, defined as a prolonged response to chronic work-place stressors (Maslach, Schaufeli, & Leiter, 2001). Burnout and its components (emotional exhaustion, depersonalization, and lack of accomplishment) are regarded as outcomes of stress, because these conditions are thought to develop over time and to be triggered by stress (Cherniss, 1980; Leiter, 1993; Maslach & Jackson, 1981). This developmental progression from stressors, to stress, to burnout has also been maintained in empirical research (e.g., Taris, Peeters, Leblanc, Schreurs, & Schaufeli, 2001). Although certain behaviors such as smoking and exercise are regarded as health outcomes of stress, these outcomes are not examined in the present research because they have already been

quantitatively or qualitatively reviewed in relation to absenteeism (e.g., Griffiths, 1996;

Kelloway, Barling, & Weber, 2002; Unckless, Mathieu, & Kelley, 1998).

. CHAPTER THREE

3. Literature Review and Development of Hypotheses

Absenteeism research can be roughly categorized into process and decision

models. Research within the process framework seeks general causal factors of

absenteeism, whereas decision models probe micro cognitive processes and decisions

underlying an individual's absence (Johns, 1997). Research examining stress in relation

to absenteeism falls under the process umbrella, where stress correlates are seen as

general causes of absence. Any theory of causation, however, must simultaneously

consider three basic questions (Campbell, 1967). In determining the existence of a causal

relationship between stress and absenteeism, these questions are: does stress (X)

influence absence (Y), does absence (Y) influence stress (X), and is there a third factor

(Z) that causes X and Y to covary?

To show that stress is related to later absence is insufficient in demonstrating the

existence of a causal relationship between these two variables, because the association

could also be a function of the influence of prior absence on stress and/or the influence of

some third related variable. In this study I consider each of these three questions

separately, and also probe an extension of the first, does stress (X) influence absence (Y)

through illness? Figure 1 presents a visual representation of the organization of the

remainder of this paper, and of the main variables and associations of interest. Portions

A, B, and C represent the main associations of interest in this paper. In building a case

for each association, I first discuss its relevance to prevailing conceptual frameworks in

absenteeism research. I also discuss key methodological and substantive issues in

existing research or thinking that preclude a straightforward examination of this

association. Finally, I present hypotheses for each variable depicted in the figure, which are to be tested against accumulated empirical findings in the literature.

3.1 Stress (X) causes Absence (Y)

The discussion in this section is represented by portion A in Figure 1, and focuses on three areas. I begin with an examination of stress as a predictor of absence, discussing key theoretical models (i.e., withdrawal, escape, and medical) that have been used to study this association in absenteeism research. After building hypotheses about this particular relationship through substantive and methodological considerations, I then examine potential mediating processes underlying the stress-absence association. Integrating the adaptation model of absenteeism, the transactional coping model of stress, and the illness-pathways model, I offer several hypotheses about relevant mediators. Finally, I present a separate discussion on the measurement and dimensionality of absenteeism, exploring the possibility of obtaining differential associations due to type of absence measure. The following discussions are directed at building hypotheses 1 through 5 as shown in Figure 1.

Relevant Theory

The stress-causes-absence proposition is often explored within the withdrawal, escape, or medical model of absenteeism (Johns, 2002). Consistent with the treatment of absence as a behavioral consequence of stress in many stress models, Hill and Trist (1955) were the first to suggest that work-related stressors and stress may prompt individuals to withdraw or flee from the workplace by going absent. In this escape model of absenteeism, employees are thought to stay away from work because they want to avoid negatively perceived aspects of their work environment (March & Simon, 1958).

However, further development of this view by Steers and Rhodes (1978) led to the recognition that perhaps employees stay away from work because they are unable to attend. This concept, the inability to attend, is said to index factors such as transportation, family illness, or personal illness, and is also seen as a regulator of absenteeism. Personal illness, however, has often provided the conceptual link between work stress and absenteeism in research. For example, the stress-causes-absence proposition is often explored within the medical model of absenteeism (Johns, 2002), in which absenteeism is thought to result from the inability to attend work because of a weakened state of well-being.

Existing Research

Previous narrative reviews of the absence literature show some support for the withdrawal or escape function of absence with respect to stress (e.g., Johns, 1997). For example, positive associations between stress and absenteeism have been reported in the literature (e.g., Tang & Hammontree, 1992). However, the causal precedence of stress, a methodological requirement for a true test for causation, has not always been maintained in empirical studies. In their review of the absenteeism literature, Harrison and Martocchio (1998) concluded that more than half the studies aimed at *predicting* absence were postdictive in nature. For example, Mckee, Markham, and Scott (1992) attempted to predict absenteeism from stress using an employee's past absence as the dependent variable. In addition to research design, interchangeable use of stressor and stress measures (e.g., Dwyer & Ganster, 1991) in the literature precludes a straightforward conclusion about the effects of stress as defined on absenteeism.

The existing empirical literature has also failed to address the theorized mediating

processes of the stress-absence association (Harrison & Martocchio, 1998). Johns (1997)

suggested that the medical mediation proposition (i.e., inability to attend due to illness)

has remained largely untested. Most studies have generally assumed this to be the case.

For example, based on their obtained association between job strain and absence due to

illness, Dwyer and Ganster (1991) concluded that there are health-related consequences

of high strain jobs. In addition, Ho (1997) used the illness explanation to support a

positive relationship between stress and absence, but did not measure any health

variables. This taken-for-granted mediation of illness in the stress-absence association is

also reflected in Karasek's (1990) inclusion of absence in a measure of health problems,

and Vahtera, Kivimaki, Uutela, and Pentti's (2000) use of sick leave as an indicator of ill

health.

The Stress-Illness-Absence Connection

As the withdrawal and process models suggest, employees stay away from work

either because they want to escape negatively perceived aspects of their work

environment (March & Simon, 1958), or because they are unable to attend due to illness

(Steers & Rhodes, 1978). These views are not at odds with each other, and can be

reconciled in the context of various frameworks on absenteeism, stress, and illness. In

building an understanding of the stress-illness-absence progression, I draw upon Cohen

and Williamson's (1991) model of stress-illness pathways, Rosse and Miller's (1984)

adaptation process model of absenteeism, and Lazarus and Folkman's (1984)

transactional model of stress coping. For the purposes of this discussion, I focus on the

basic processes highlighted in these models, although the complexities (e.g., moderators

and constraints) identified in each model are acknowledged and addressed to some extent

in later sections of this paper.

Stress and illness. Cohen and Williamson's (1991) stress-illness-pathways model

suggests that stress triggers illness through three main pathways. Briefly, stress produces

physiological (e.g., cathecholamines, cortisol) and behavioral changes (e.g., smoking,

poor eating habits) in an individual, which in turn weaken immune functioning, giving

rise to disease. Physiological changes may also influence one's attention to internal

physical states, increasing the sensation of symptoms associated with the experience of

stress, thereby prompting the labeling of illness or the seeking of medical attention.

Although studies vary in their operationalization of stressors and stress, several reviews

provide some support for the basic propositions of the stress-illness pathways model. For

example, O'Leary (1990) reported that chronic stressful events increased the levels of

antibodies or lowered the number of helper cells in one's immune system. Focusing on

the onset of disease in their review, Cohen and Williamson (1991) found consistent

evidence for the causal role of stress (which included measures of stressful life events,

depression, mood states, general health, and some personality indices) in the onset of

upper respiratory tract infections.

Schnall, Landerbergis, and Baker (1994) reviewed studies on job strain

(operationalized in terms of Karasek's [1979] job demands model) and cardiovascular

disease (CVD), which included outcomes such as myocardial infarction, hypertension,

coronary heart disease (CHD), CHD symptoms, angina pectoris, and ambulatory blood

pressure. They found positive associations between job strain and CVD outcomes across

cross-sectional, matched-control, and longitudinal cohort studies. Focusing specifically

on studies on blood pressure, Schwartz, Pickering, and Landerbergis (1996) cited support

for a positive association between Karasek's job strain and ambulatory blood pressure. A recent meta-analysis on varied stressors and immune functioning confirms the conclusion that chronic stressors have detrimental effects on the immune system (Segerstrom & Miller, 2004), which increases susceptibility to illness.

Stress, psychological illness, and somatic illness. With respect to the progression of illness development, Frese and Zapf (1988) suggested that psychosomatic symptoms of stress might develop earlier in time than actual illness. In other words, stress might trigger psychological ill health or the reporting of related symptoms, which contributes to the development or reporting of physical or somatic illnesses. Evans and Edgerton (1991) demonstrated this progression through their finding that certain stressful life events resulted in negative mood states such as feelings of anger and tension, which preceded the onset of colds in individuals. In another study, similar mood factors were also strongly associated with illnesses such as colds, headaches, and general malaise, suggesting that negative affective states may increase one's susceptibility to illness (Evans & Edgerton, 1992).

Although cross-sectional in nature, van Katwyk, Fox, Spector, and Kelloway (2000) hypothesized a similar progression, in which job stressors were expected to result in negative emotional responses such as frustration, anxiety, and fatigue, which in turn resulted in physical illness outcomes. Spector and Jex (1998) also present indirect evidence for this progression through their quantitative review of findings on stressors, psychological strains (i.e., anxiety, depression, and frustration), and the Physical Symptoms Inventory (e.g., headaches, stomach upsets). Stronger correlations between psychological strain and physical symptoms ($.26 < r < .47$) compared to those between

stressors and physical symptoms (-.11< r < .20) suggest that psychological symptoms might indeed follow the onset of stressors and possibly stress, but precede that of physical symptoms. Based on the above, a stress-psychological illness-physical illness connection is proposed.

Stress, illness, and absence. To understand how absenteeism fits into this sequence of events, I refer to Lazarus and Folkman's (1984) transactional and Rosse and Miller's (1984) adaptation process models, which explicate this progression from a behavioral perspective. The premise underlying the propositions of both models is that individuals respond or interact with their environment when faced with a threatening situation. In both models, some aversive stimulus contributes to an individual's dissatisfying state (in this case, stress). Stress is thought to trigger favorable or unfavorable adaptive coping mechanisms in the individual, which are cognitive or behavioral efforts aimed at managing stress and its sources. Cognitive coping efforts can include the reappraisal of stressful events or the denial of their existence. Behavioral efforts, on the other hand, comprise behaviors such as over eating, smoking, or seeking social support or exercising. These coping efforts have the capacity to reduce one's stress levels; however, when ineffective or lacking, stress levels can escalate, resulting in psychological and physical ill health consequences (Lazarus, 1990; Edwards, 1992). Wiebe and McCallum (1986) empirically demonstrated that poor health practices during a month following stress resulted in an increased severity and number of illnesses a month later.

Aspinwall and Taylor (1997) suggested that individuals choose between specific strategies of coping, depending on the degree or extent of the perceived problem.

Representative criteria are used in choosing a coping strategy such that individuals choose small efforts to address small problems, while greater efforts are used to deal with larger problems. Although these authors are concerned with proactive coping efforts, Edwards (1992) suggests that the same coping effort can be reactive when one tries to address a recent source of stress after well-being has been damaged, or proactive when these efforts are thought to prevent further damage to well-being. In the following discussion, it is assumed that coping may be undertaken reactively to address stressful experiences that have already occurred, or proactively to prevent future experiences from occurring.

If the stress - psychological illness - physical illness sequence is viewed as reflecting an increasing severity of stress, then based on Rosse and Miller's (1984), Lazarus and Folkman's (1984), and Aspinwall and Taylor's (1997) theoretical views, individuals can be thought to expend progressively greater coping efforts in dealing with the preceding episode or the next immediate event in this sequence. For example, an individual experiencing stress at work may initially engage in cognitive coping such as reappraising the situation or ignoring it. In addition, certain behaviors requiring relatively little effort such as overeating, increased smoking, or relaxation may be employed. If these efforts are unsuccessful in reducing the stressful experience, then according to the stress-illness pathways model, psychological illness symptoms (e.g., fatigue, emotional exhaustion, anxiety, depression) are manifested. At the awareness of a greater problem (i.e., increased stress severity experienced), the individual may expend greater efforts to cope. These efforts might, perhaps, include problem-solving approaches, taking a break from work by being absent, or seeking medical attention.

Although absence is not typically cited as a coping strategy, it may be regarded as such because time away from work affords the opportunity to offset or minimize the cumulative effects of stress (cf. Hackett & Bycio, 1996). Alternatively, staying in a chronic stress environment can impede the effective utilization of coping resources, contributing to further stress (Aspinwall & Taylor, 1997). Failure to successfully address the psychological outcomes of stress results in the onset of physical illness, at which point absence from work becomes an inevitable behavior. Therefore, absence is viewed as a response to increased severity of stress, undertaken to address psychological or physical manifestations of stress.

There is some evidence for the above-described progression. For example, Baba, Galerpin, and Lituchy (1999) found support for burnout and depression as pathways through which stress influenced absenteeism intentions. Hendrix, Spencer, and Gibson (1994) also found that stress influenced absence via emotional exhaustion and somatic complaints. Although stress was not measured, health complaints mediated the association between procedural unfairness (a stressor) and absence twelve months later in De Boer, Bakker, Syroit, and Shaufeli's (2002) study. Similarly, Hendrix and Spencer (1989) provide partial support for this progression, in which cold and flu episodes were found to be stronger predictors of absenteeism than emotional exhaustion. In addition, slightly stronger associations were obtained for absenteeism and physical health complaints than for absenteeism and emotional exhaustion or depressed mood (de Jonge, Reuvers, Houtman, & Kompier, 2000).

In light of the above discussion, the following hypotheses are offered.

Hypothesis 1:   The association between stress and absence will be positive

Hypothesis 2:   The association between psychological illness and absence will be

positive.

Hypothesis 3:   The association between physical illness and absence will be

positive.

Hypothesis 4:   The association between stress and absence will be partially

mediated by illness such that psychological illness precedes the

development of physical illness.

3.1.1. Types of Absence

Despite the use of varied measures of absenteeism, the primary objective has been

to capture two main types of absenteeism, voluntary and involuntary (Hackett & Guion,

1985). Voluntary absences are defined as being under the direct control of the individual,

while involuntary absences are thought to result from factors beyond an individual's

immediate control. Time lost measures of absenteeism are regarded as involuntary,

because longer absences are assumed to result from factors beyond the control of an

individual (Steers & Rhodes, 1978). Such factors could include illness, transportation,

and family problems. Frequency and attitudinal measures, which do not capture the

duration of an absence, are assumed to result from the employee's decision to be absent

for whatever reason (Hammer & Landau, 1981). They have been imputed the voluntary

label because of Fox and Scott's (1943) reasoning that voluntary absences tend to be

short in duration. Whether or not the stress-absence association varies with the type of

absence measure depends on the degree of correspondence between the absence measure

and its antecedent (cf. Ajzen & Fishbein, 1977). Based on the above discussion and

proposed mediation through illness, one would expect stronger stress/illness-absence

associations for time lost measures in comparison to frequency measures of absenteeism. However, for several reasons discussed below, I anticipate little difference in the magnitude of these associations.

Although there is some evidence for the bi-dimensional nature of absenteeism (e.g., Hackett & Guion, 1985), there is much scepticism about the precise nature of the processes underlying these dimensions. For example, Johns and Nicholson (1982) discussed the inappropriateness of attributing voluntary causes to frequency measures and involuntary ones to time lost absence, because absenteeism means different things to different people. Martocchio and Harrison (1993) believe that in the absence of solid empirical evidence for a connection between an absence measure and its voluntary or involuntary correlate, it may be premature to distinguish these measures on this basis. There has also been some speculation in the literature that sick leave might have non-medical or voluntary determinants. In an interview with one employee who received medical permission to take two weeks off, Dodier (1985) found that the employee decided to save his sick leave for a time when he was feeling better. Youngblood (1984) also found that total time lost due to illness was significantly associated with the value that an individual placed on non-work time. Burton, Lee, and Holtom (2002) specifically measured sickness absence and the ability to attend, but found the two variables to be poorly correlated, suggesting that absence due to illness might not always reflect an inability to attend, and may comprise a volitional component.

Nicholson (1977), on the other hand, has argued that absenteeism might be better categorized in terms of its required justifiability. In his view, absences should be placed along a justifiability continuum, ranging from absences that require no justification to

those requiring high justification. The resulting number of absence dimensions would, therefore, vary with the number of valid and discriminate justifiability categories. Therefore, for a variety of factors discussed above, distinction between frequency and time lost absence indices in terms of underlying voluntary or involuntary processes is likely to be weak. Existing findings tend to support this suggestion. For example, Nielsen, Kristensen, and Smith-Hansen (2002) reported similar associations for absence days and absence spells with a variety of stress and health variables. There were also no observed differences in correlations between absence days and spells for job-related anxiety, depression, and general health in Hardy, Woods, and Wall's (2003) study. Note that in both these studies, frequency and time lost measures of absenteeism were highly correlated. The moderating influence, if any, of the strength of association between frequency and time lost absence measures in each study will be examined post hoc. The following hypothesis is offered in relation to short-term absence, because most studies in management research tend to focus on this type of absenteeism.

Hypothesis 5:   The magnitude of stress-absence and illness-absence associations will not differ across frequency and time lost measures of absence.

3.2 Absence (Y) causes Stress (X)

Referring to Figure 1, this section focuses on portion B. Stress is examined as a potential outcome of absenteeism. In hypothesizing the direction of this association, absenteeism is examined within the restorative model, and findings from respite research and stress coping are integrated to understand the restorative function of absenteeism.

Relevant theory

Following Staw and Oldham's (1978) suggestion that absence might provide

employees with an opportunity to recharge, the absence-causes-stress proposition has often been explored within the restorative model of absence. This view is consistent with Caplan and Jones's (1975) treatment of respite as a proxy for decreased exposure to workplace stressors. Respite research, which is largely based on Hobfoll's (1989) conservation of resources theory, provides further insight into the expected absence-stress association. Defined as a day off, a weekend, a vacation, or some other form of absence from the workplace, respite was examined in the form of a vacation by Westman and Eden (1997). They hypothesized and found that in comparison to pre-vacation periods, employees reported less burnout during and following their vacation. Of course, vacations are very different from sick day absences, but Etzion, Eden, and Lapidot's (1998) study suggests that it might be the mere change of venue that is important in reducing stress. They found that military personnel who were scheduled for reserve service perceived less stressors and felt less burned out a week after returning to work from reserve service, as compared to their levels before leaving and to levels of burnout reported by employees in a matched control group. The benefits of being away from work have also been demonstrated for periods as short as an evening, which allow employees to recover from the day's work (Sonnentag, 2003). Therefore, the restorative model focuses on the potential consequences of absenteeism, and suggests a negative association between prior absence and stress.

Existing Research

The restorative function of absence has rarely been directly examined in absenteeism research (Johns, 2002). Methodologically, one would require the measurement of absence to precede that of stress. Many postdictive studies appear to

fulfill this requirement, but have failed to interpret their results in accordance with this proposition, focusing instead on the prediction of absenteeism. For example, Parker and Kulik (1995) obtained measures of nurse absence during the preceding six months from hospital records, and measured current levels of burnout, but reported that "higher levels of emotional exhaustion significantly and uniquely predicted...more days off for mental health reasons" (p. 591). Although there is some indirect support for the restorative function of absenteeism (e.g., George, 1989; Hackett & Bycio, 1996; Williamson, Gower, & Clarke, 1994), a closer perusal of postdictive findings points to a relationship that is counter to the propositions of the restorative model. For example, in Manning and Osland's (1989) comparison of prior stress-subsequent absence and prior absence-subsequent stress associations, the latter were found to be positive. Even in Hardy, Woods, and Wall's (2003) study, absenteeism did not moderate the relationship between psychological stress at time 1 and at time 2, suggesting that taking absence in response to stress at time 1 was not useful in alleviating stress at time 2. In a sample of police officers, Tang and Hammontree (1992) found absence to have non-significant, but positive associations with stress six months and 12 months later. These findings lead one to question the adequacy of absence in performing a restorative function.

The Restorative Function of Absence

I draw primarily upon the coping literature in proposing that absenteeism performs an ineffective restorative function. The coping literature distinguishes between emotion-focused and problem-focused coping. Briefly, emotion-focused coping attends to an individual's emotional response to the problem, and includes thoughts or behaviors such as avoidance, denial, isolation, and venting (Lazarus & Folkman, 1984; Tamres,

Janicki, & Helgeson, 2002). On the other hand, problem-focused coping is more directed at managing or solving the problem or source of distress. It includes active efforts to remove the stressor, planning, and seeking social support directed at resolving the problem (e.g., seeking advice and information on the problem).

Problem-focused coping is said to be more effective in reducing distress than emotion-focused coping, especially when the source of the problem is controllable (Lazarus, 1990). Consistent with this view, Tamres et al. (2002) suggested that action-oriented or problem-focused coping approaches may generally be more effective in dealing with work stressors such as role overload, role conflict, or challenging work. Indeed, problem-focused strategies such as creating plans to deal with a problem has been associated with improved health (Parkes, 1990) and decreased depression and anxiety (Greenglass & Burke, 1991), while emotion-focused strategies such as mental disengagement (e.g., Griffith, Steptoe, & Cropley, 1999), and denying or avoiding the problem (e.g., Tyler & Cushway; 1995) were associated with increased perceived stress and psychological distress.

Absence as non problem-focused coping. Although absenteeism research has not specifically categorized absence as problem- or emotion- focused, Haccoun and Dupont's (1987) examination of the kinds of activities that people engage in, on their unscheduled day off, suggests that absence might not be problem-focused. These activities related mainly to entertainment (e.g, sports, hobbies, reading), family (e.g., housework, car care, garden care), trips (e.g., appointments, short meetings), resting (e.g., sleep), work (e.g., another paid job, courses), maintenance (e.g., preparing meals, eating), and relationships (e.g., writing, discussion). It may be premature to label absence as an emotion-focused

coping strategy without any confirmation of a stress-absence association, or further fine-grained probes into the purpose for engaging in certain absence day activities. However, evidence for absence as being non-problem focused is suggested by Brebner's (2001) finding that individuals high in conscientiousness tend to adopt task- or problem- focused coping strategies in dealing with stressful situations. Conscientiousness has also been consistently found to have a negative association with absenteeism (e.g., Ashton, 1998; Judge, Martocchio, & Thoresen, 1997). The low absence level of conscientious individuals is, perhaps, a result of their better management of stress using problem-focused approaches. Therefore, if absence is regarded as a non problem-focused coping behavior, it is expected to be ineffective in alleviating stress.

Accompanying adverse consequences of absence. The effectiveness of absenteeism in alleviating stress is further hampered because of its accompanying accrual of negative work-related consequences. Jackson and Schuler (1985) discussed increased job responsibilities upon return to work as a reason for employees' low inclination to go absent when faced with work stressors. Tharenou (1993) also found that absenteeism at time 1 lowered job satisfaction and job performance ratings by a supervisor at time 2. While lowered job satisfaction could by itself exacerbate an individual's initial stressful experience, Bycio's (1992) explanation for the negative meta-analytic association between absenteeism and job performance points to additional sources of stress for the employee. Employees could potentially get into trouble with their superiors for their frequent absence, which could lower their next performance evaluation, partly a product of the supervisor's annoyance with frequent absenteeism.

Absence could also provoke further stress because it disrupts coworker

relationships. Goodman and Leyden (1991) found that absenteeism reduced familiarity among coal miners, which in turn contributed to lowered group productivity. Therefore, absence may be ineffective in alleviating felt stress because it could potentially contribute to the development of additional sources of stress for the absent employee. On average, a positive association between prior absence and subsequent stress is expected. However, an employee's reported stress level may remain unchanged, resulting in a null association. Indeed, Hackett and Bycio (1996) found that stress reported on shifts following absence were not much different on shifts preceding absence. They interpreted their findings in terms of the stress maintenance function of absence. If absence performs a maintenance function, one might expect only a weak positive association between prior absence and subsequent stress.

Limited effectiveness of restorative absence. Moving away from viewing absence as problem- or emotion-focused, there is another reason for expecting a positive association between prior absence and subsequent stress. Evidence from respite research suggests that the potential benefits of staying away from work tend to be short lived. Westman and Eden (1997) found that the positive effects of a vacation disappeared three weeks after returning to the job, such that burnout progressively returned to pre-vacation levels as post-vacation time passed. Eden (1990) similarly reported that relief from respite had faded one month later. Even Hackett and Bycio (1996) express skepticism over the long-term contributions of absence in addressing employee stress in the work place. These findings suggest that even if absence does have a restorative effect on stress, this effect is likely to be short-lived, and detected only when short time frames between measures of absence and post-absence stress are used (cf., Harrison &

Martocchio, 1998). Respite research suggests that these time frames vary from one day
to three weeks. With exceptions of studies employing an idiographic approach (e.g.,
Hackett & Bycio, 1996; Hackett, Bycio & Guion, 1989), absenteeism research has rarely
used such short time intervals between predictor and outcome measurement, making it
difficult to detect any potential beneficial effects of absence even if they did exist.
Therefore, on average,

Hypothesis 6:   The association between prior absence and subsequent stress and
                    illness will be positive.

3.3. Stress-absence (X-Y) relationship as a function of some third variable (Z)

Central to the discussion in this section is the role of confounding or extraneous
influences on the stress-absence association. In Figure 1, this section is represented by
portion C. Several individual and social influences on the stress-absence association are
examined. To situate the following discussion within some theoretical context, I refer to
Nicholson's (1977) conceptualization of absence as a two-stage process in which
proximal or immediate reasons for absence (stress or illness in this case) are filtered by
second stage processes. Second stage influences include individual motives or attitudes
and social or normative factors. Nicholson's model suggests that absence may not be a
direct result of stress or illness, but rather a reflection of other factors such as micro-level
decisions of the individual or macro-level influences on this decision. Indeed, previous
references to non-medical determinants of sickness absence (Smulders, 1980;
Youngblood, 1984) allude to this possibility.

In this paper, I examine a few individual and social influences on the relationship
between stress and absence. Substantive and methodological criteria guided the

identification of the following factors. In other words, I chose to focus on a particular

factor not only because prior literature suggested its relevance to stress and absenteeism,

but also because the factor could be adequately measured and tested at the study level.

As extraneous influences, these factors can render the stress-absence association spurious

when they are not specifically controlled in a study. Alternatively, they can influence the

magnitude or direction of the stress-absence relationship when they are specifically

examined as moderators or deliberately excluded from a study (Pedhazur & Schmelkin,

1991). The discussions to follow probe these possibilities in the context of the primary

relationship of interest in this paper, stress as a cause of absence. The following sections

are grouped under individual and social contextual influences to improve clarity of the

presentation, but this does not suggest that social factors are not at play when individual

influences are being discussed or vice versa.

3.3.1 *Individual Influences*

Potential individual confounds in the stress-absence relationship include the role

of attribution, disposition, and demographic factors. Acknowledgement of these factors

is reflected in Marmot, Feeney, Shipley, North, and Syme's (1995) concluding paragraph

cautioning readers that their obtained health status-sickness absence association may be a

result of reporting tendencies of the individual or predisposition towards illness. The

former is a methodological issue concerned primarily with the role of attribution, while

the latter is a substantive one involving the role of individual differences in relation to

stress and absence.

Role of Attribution

Heider (1958) was the first to discuss the influence of attribution biases in the

examination of causal relationships. People tend to provide acceptable explanations for their behavior such that the reasons are either plausible or consistent with their wishes. Clegg (1983), for example, suggested that motivational causes of withdrawal behavior might reflect an individual's perception and attribution of withdrawal to the concerned cause (i.e., low motivation). The degree of attributional influences is partly dependent on methodological issues. For example, Miller and Ross's (1975) early review of experimental studies exploring causal attribution revealed that self-serving attribution processes were only evident in experimental tasks that were designed in a way to elicit such reactions. Folger and Belew (1985) also explained how the growing use of self-reports and retrospective designs in both absenteeism and stress research increases the likelihood that the stress-absence association may be a function of response bias.

Illusory correlations between stress or illness and absenteeism can result from several sources of common method bias (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). Particularly relevant to this research is the influence of the consistency motif and implicit theories. Rater tendencies to appear rational and to respond in accordance with certain assumptions they have about a phenomenon can contribute to artifactual covariance between two variables. Merely asking participants to report on work-related stress may make them more aware of stressors in their work environment at that particular point in time. As Harrison and Martocchio (1998) suggested, questionnaire items asking employees to report the days off due to sickness or illness are highly loaded with attributional content, increasing the probability that individuals may be cued to respond in a way that strengthens the illness-absence association.

The search for causal explanations or justifications for behavior is even more

likely for absenteeism, because the behavior is generally regarded as deviant (Martocchio & Harrison, 1993; Johns, 1994). Consistent with attribution theory, Johns (1994, 1999) discussed how the negative connotations of this behavior might prompt individuals to disassociate themselves from it through underreporting. However, the unfavorableness of this behavior may also prompt individuals to seek external, justifiable explanations for their absence. In interviewing individuals about their past absences, Haccoun and Desgent (1993) found that those who were absent more frequently tended to provide more reasons to justify their absence. There is also evidence suggesting that stress and illness are often used as acceptable reasons for being absent. Parsons (1975) conceptualized the "sick role" as a function that society regards as legitimate, because the capacity of sick individuals to function normally in their daily roles (worker role, for example) is diminished. This legitimacy of the sick role was also evident in Segall's (1976) study, in which the majority of his sample agreed that a sick person has an obligation to get well and must be exempt from other social roles.

Even though people underestimate their propensity to go absent because of minor illness, minor ailments are often provided as reasons for employee absence events (Nicholson & Payne, 1987). Minor illness and "mental health day" (p. 431) were also the two most frequently cited reasons for past absences in Hackett et al.'s (1989) study of nurses. Despite some variation across a number of illnesses, Harvey and Nicholson (1999) found that employees are prepared to endorse illness as being a legitimate or acceptable reason for being absent. Indeed, Haccoun and Dupont (1987) demonstrated that individuals provided sickness as a reason for being absent on a certain day even though 72% of their sample admitted to engaging in other activities on that sick day off.

Medical reasons for absence were also regarded as being more legitimate than domestic ones across two culturally different samples (Johns & Xie, 1998), pointing to the universality of illness as a legitimate reason for absenteeism.

Given the above, stronger stress-absence associations may be due to respondents' heightened awareness of stress/illness issues when questioned about such states, their need to find acceptable reasons to justify absence days, or the need to respond in accordance with perceived research goals and requirements. Evidence for attribution bias in the stress-absence association is likely to be provided by a comparison of effects obtained in studies using retrospective self-report measures of absence against those using records-based absence measures.

Hypothesis 7a: The average stress-absence association in retrospective studies using self-report measures of absence will be stronger than in those using records-based absence.

Hypothesis 7b: The average illness-absence association in retrospective studies using self-report measures of absence will be stronger than in those using records-based absence.

Dispositional Influences

While the preceding section dealt with confounding cognitive influences resulting primarily from methodological approaches in examining the stress-absence association, this section is concerned with substantive dispositional confounds. Is the stress-absence association a result of individual differences in perceiving stress or in taking absence? In building a hypothesis about dispositional influences, I first discuss the relevance of disposition to stress and absenteeism, followed by a discussion on the approach to be

used in testing this particular hypothesis in the meta-analysis.

Disposition and stress. Referring to the stress-illness-absence sequence,

personality has been shown to affect each element in this sequence. Motivational and

cognitive aspects of personality can increase perceptions of stress through the appraisal of

stressors as threatening (Wiebe & Smith, 1997). One trait in particular that has received

tremendous attention in the stress and health literature is neuroticism or negative

affectivity (NA). While some regard these two personality traits as distinct, others (e.g.,

Watson & Clark, 1984) suggest that they are interchangeable. Collectively, the terms are

used to refer to individual differences in the tendency to experience negative emotions

(Costa & McCrae, 1985; Watson & Clark, 1984). Watson and Clark explain that high

NA individuals experience more distress, have less favorable views of themselves, are

introspective, and tend to focus on the negative side of events. Similarly, those high on

neuroticism are described as having a tendency to be anxious, hostile, depressed, and

somatically over-concerned (Stone & Costa, 1990). The potential influence of this

disposition on stress appraisal is so strong that Brief, Burke, George, Robinson, and

Webster (1988) urge researchers to control for this variable in their examinations of job

stress.

Other personality factors such as conscientiousness and the Type A Behavior

Pattern (TABP) have also been linked to stress. While conscientious individuals tend to

persevere in the face of challenging situations (e.g. Hollenbeck, Klein, O'Leary &

Wright, 1989), individuals with TABP tend to experience higher levels of emotional

distress in similar situations (Suls & Wan, 1989). Type A Behavior Pattern (TABP)

individuals tend to be aggressively involved in accomplishing more and more in lesser

time despite opposing efforts and constraints (Friedman & Rosenman, 1974). For example, Jamal and Baba (1991) found that nurses with TABP reported higher levels of job stress than their Type B counterparts. Type A and/or its components (e.g., time pressure, competitiveness, achievement striving, impatience/ irritability) were also found to be positively and significantly associated with burnout (Jamal & Baba, 2001) and health problems (Jamal & Baba, 2003).

Disposition and absenteeism. Temporal and cross-situational stability of absence behaviors (Johns, 2002) has led to the conclusion that some individuals might be pre-disposed or prone to be absent. For example, Rentsch and Steel (1998) found absence frequency in year 1 to be highly correlated with absenteeism in year 4 ($r = .59$) and year 6 ($r = .53$). The average corrected correlations between absence history and time lost ($r = .71$) and frequency ($r = .65$) measures, presented in Farrell and Stamm's (1988) meta-analysis, offer further support for the temporal consistency of this behavior. Absenteeism also exhibits with a high degree of consistency across situations. In addition to Brenner's (1968) finding that high school absence is positively associated with work absence, individual absenteeism was found to be fairly consistent across 11 different settings which included work, class, medical appointments, religious services, social functions, weddings, and parties (Harrison & Price, 2003).

Although there is presently no consensus on the precise disposition responsible for absenteeism (Harrison & Price, 2003), many personality traits have received attention in the literature. For example, neuroticism and negative affectivity have often been theorized to have a positive association with absence, because individuals with such traits find it difficult to psychologically adjust to challenging situations, and are more likely to

withdraw from the workplace (George, 1989; Iverson & Deery, 2001; Judge, Martocchio,

& Thoresen, 1997). With respect to TABP, Evans Palsane, and Carrere (1987) found that

in comparison to Type B bus drivers, those exhibiting TABP had a higher number of

absences per month. Significant associations between absenteeism and other

dispositional variables such as hostility (e.g., Vahtera, Kivimaki, Uutela, & Pentti, 2000),

anxiety (e.g., Bernadin, 1977; Ferris, Youngblood & Yates, 1985), conscientiousness

(Ashton, 1998), and extraversion (Judge et al., 1997) provide further evidence for

dispositional influences on absenteeism.

Testing for the moderating role of disposition. In the present meta-analysis,

testing the moderating role of disposition on the stress-absence association requires an

understanding of the stability of this source of variance. Evidence for stable dispositional

sources of variance in any behavior is based on the assumption of generality across time

and situations (Mischel, 1968). As an example, Mischel explained how an individual's

behavioral difficulties with siblings, peers, parents and several authority figures across

one's life span signals the presence of an underlying individual difference characteristic

(e.g., generalized attitude towards authority or peers). The demonstration of consistency

in behavior across situations and time is generally discussed in terms of reliability. For

example, Epstein and O'Brien (1985) advocated the aggregation of behavioral items

across time or situations to improve reliability. Aggregation allows for a sufficiently

wide range of generality of the behavior, and is more likely to correspond with an

underlying stability of a person's disposition.

Consistent with this logic, Harrison and Martocchio (1998) suggested that the

detectability of an absenteeism predictor is likely to depend on the time frame used to

aggregate absence episodes. According to their time-based approach, stable causes like personality and attitudes are likely to be detected when longer absence aggregation periods are used. Work place stress and more acute sources (e.g., one time events), on the other hand, are regarded as mid- and short-term sources of variance, respectively. Note that even though work stress is regarded as chronic, its sources (i.e., stressors) fluctuate temporally, being mid- to short-term in nature (LaCroix & Haynes, 1987). For example, a typical disagreement with one's supervisor, coupled with an approaching project deadline or a customer complaint, may heighten an employee's stressful experience at one particular point in time. However, in the absence of any deadline, these additional stressors might not be perceived as stressful. Therefore, the effects of stress on absenteeism are likely to be more pronounced when mid- to short-term absence aggregation time periods are used. Martocchio, Harrison, and Berkson's (2000) meta-analysis provides some empirical support for this time-based examination of absenteeism. They found that the association between chronic low back pain and absence was stronger when longer absence aggregation time periods were used, reflecting the stability of that source of variance (i.e., long standing back pain condition).

To the extent that the stress-absence association is largely attributable to a stable source of variance, its magnitude should increase with increasing absence aggregation times. On the other hand, if absence is purely a result of workplace stress, then a negative relationship between aggregation time and this association is expected. However, it is likely that the observed association could result from a joint influence of long-term (i.e., personality) and mid-term (i.e., stress) sources of variance. In this case, the hypothesized positive and negative associations are likely to cancel each other,

yielding a near-zero moderating effect. Because illness conditions can range from being short-term, such as colds and flu, to long term ones, such as heart disease and depression (Harrison & Martocchio, 1998), it is difficult to attribute any obtained positive association between absence aggregation periods and illness-absence effects to disposition, as the same could reflect the stable nature of a particular illness. Therefore, the following two competing hypotheses are restricted to the stress-absence effect only.

Hypothesis 8a: Reflecting stable dispositional sources of variance, the stress-absence effect will be positively associated with absence aggregation periods.

Hypothesis 8b: Reflecting combined mid-term and stable sources of variance, the stress-absence effect and absence aggregation association will be near zero..

Role of Gender

Another potential individual-level confound in the stress-absence association is gender. That women tend to be more absent from work than men has been a pervasive finding in absenteeism research (Côté & Haccoun, 1991). However, reasons underlying this finding have yet to be determined, and a variety of social and psychological explanations have been advanced.

Relevant theoretical perspectives. Explanations of the gender differential in stress can be roughly categorized as pertaining to a *differential exposure* or *differential vulnerability* argument. The *differential exposure* view supports a structuralist perspective and suggests that occupational variations created by a traditionally male-dominated organizational system might expose women to less favorable factors than men,

causing women to have higher stress levels than men (Jick & Mitz, 1985; Kanter, 1977; Nelson & Quick, 1985; Scott & McClellan, 1990). For example, employed women have been found to work fewer hours, have less complex jobs and lesser job control than men (Roxburgh, 1996). In addition, they tend to be disadvantaged in terms of career mobility (Lyness & Thompson, 2000) and are paid less in comparison to their male counterparts (Goldin, 1990). However, the *differential exposure* view is not consistent with Martocchio and O'Leary's (1989) meta-analytic finding that men and women tend to experience similar levels of stress.

The *differential vulnerability* explanation might be more viable, and appears to have received consistent support in the literature. Within this view, some researchers argue that genetic factors predispose women to react differently to threatening situations, while others advance a socio-psychological explanation that women may be socialized to seek help and to admit difficulty in the face of stress (Jick & Mitz, 1985; Kandrack, Grant, & Segall, 1991; Nadelson, 1983). Still others explain that women might be more likely to experience the adverse effects of stress because of the "double burden" introduced by their working role, coupled with childrearing and household responsibilities (e.g., Schwartz, Pickering & Landsbergis, 1996). Therefore, a combination of factors might increase women's vulnerability to stress. I argue that although men and women experience similar levels of stress, they are likely to *react* differently to it.

Gender and stress reactions. There is evidence to suggest that women may be more responsive to stress than men are. In comparing various responses to stress across genders, Wofford (2002) found women exhibited more anger and irritability than men.

Women are also more likely than men to report psychosomatic complaints such as headaches, fatigue, sleeping problems, and so on (Steffy & Jones, 1988). In a meta-analysis on gender differences in coping strategies, Tamres, Janicki, and Helgeson (2002) also found that women tended to use a larger number and variety of coping strategies than men, partly because they perceived higher threat from stressors than men did. In comparison to men, women have been found to be more concerned about their health (Hibbard & Pope, 1983), to readily recognize psychological or emotional problems in themselves (Horowitz, 1977), and to seek medical attention (Corney, 1990).

Absence as stress reaction. It is likely that workplace stress may heighten women's awareness of their psychological states, which further encourages their escape from the work environment. Some evidence is provided by Hackett's (1989) meta-analysis, in which the relationship between job satisfaction and absenteeism became more strongly negative as the proportion of women in the sample increased. He explained that women might have lower thresholds for dissatisfying work conditions than men, which increases their likelihood to escape the workplace. Stronger positive associations between adverse job conditions and absence have also been reported for women than for men (e.g., Stansfeld, Fuhrer, Head, Ferrie, & Shipley, 1997). Leigh (1983) found women were more likely than men to be absent because of a cold and flu episode. Similarly, minor illness was cited as a reason for taking absence by a significantly larger portion of women than men (Scott & McClellan, 1990).

Depression, a condition more prevalent among women (Culbertson, 1997), has also been found to be responsible for absenteeism in the workforce (Broadhead, Blazer, George, & Tse, 1990; Kessler & Frank, 1997; Moncrieff & Pomerleau, 2000). In

examining a sample of women, Bratberg, Dahl, and Risa (2002) used income and number of children as proxies for work and non-work demands, to find that women with more children and higher incomes had higher levels of sickness absence. It is likely that non-work demands placed on women may either contribute to their stress levels, or prevent them from effectively dealing with stress experienced at work. For example, Frankenhaeuser et al. (1989) found than the stress levels of male managers decreased more quickly at home after work than for women managers, suggesting that family demands may contribute to the maintenance of unpleasant work stress experiences for women. Williams and Alliger (1994) also found that spillover from family to work and vice versa strongly contributed to general distress in women, but not in men.

I expect the stress-absence association to be stronger for women because of their increased vulnerability to stress, resulting from a variety of factors such as a predisposed diminished capacity to respond effectively to work stress, the socialized tendency to adopt the sick role, and/or the burden of non-work responsibilities. In building a stress-absence link for women, I have relied on the substantive understanding that women's *responses* to stress may differ from men's, increasing their propensity to be absent. As these responses include the manifestation of ill health or the reporting of illness symptoms, one would expect illness to be more strongly associated with absenteeism than stress. However, absenteeism, a behavior also manifested as part of the sick role (Parsons, 1975), may be directly related to measures of job stress when women choose to respond to early signs of work stress by staying away from work. The moderating role of gender is likely to be demonstrated by increasing effect sizes as more women are represented in a study's sample (cf. Hackett, 1989). In other words, in samples

comprising mostly male employees, weaker stress/illness-absence associations are expected.

Hypothesis 9:   The positive stress/illness-absence effect will increase with the percentage of women in the sample.

3.3.2 Social Contextual Influences

While social influences on the stress-absence association have been alluded to in preceding sections, these factors are discussed more explicitly in the following paragraphs. The impact of occupational status and macro contextual changes are highlighted as potential extraneous influences on the stress-absence effect.

Role of Occupational Status

A consistent finding in absenteeism research is the negative association between absenteeism and occupational grade levels. For example, Feeney, North, Head, Canner, and Marmot (1998) found that absenteeism rates were higher for individuals in clerical employment groups than for those in administrative and executive groups. Adjusting for age, Hemingway, Shipley, Stansfeld, and Marmot (1997) also reported higher absence rates for those in lower employment grades. In addition, Eyal, Carel and Goldsmith (1994) found that high absence workers (i.e., those absent more than 20 days per year) comprised a larger number of blue-collar employees such as skilled workers and foremen than white-collar employees such as academics.

Occupational status and stress reactions. Stress as an explanation for occupational differences in absenteeism is highly plausible because individuals of low socioeconomic status have often been described as having a diminished reserve capacity such that they lack certain structural, interpersonal and intrapersonal resources for coping

(Gallo & Matthews, 2003). In addition to having low self-efficacy, self-esteem, and perceived control, which are personality traits that enable one to cope better (Twenge & Campbell, 2002), low occupational status individuals tend to have fewer coping resources (e.g., health care and residential facilities, well-developed social networks and support) at their disposal (Gallo & Matthews). The diminished reserve capacity of such individuals, coupled with the typically greater physical and environmental demands their low status occupations place on them, is likely to prompt them to react adversely to stress. Yet, many counterintuitive findings have been reported in the literature. For example, in a study designed to provide a homogeneous sample of blue-collar workers (i.e., low occupational status employees) with stress-management techniques, Peters and Carlson (1999) found that the intervention succeeded in increasing employees' self-efficacy and management of stress, but absenteeism levels remained unchanged. Stansfeld, Head, and Ferrie (1999) also found that sickness absenteeism rates for low grade workers did not increase with increasing job demands during a period of high job insecurity. These findings warrant alternative explanations, involving factors not directly linked to the diminished reserve capacity explanation.

Occupational status and cultural factors. Nicholson and Johns (1985) explained how expectations and beliefs about acceptable levels of absence originate out of social and organizational contextual factors such as absence control policies, attendance incentive programs, and attendance-oriented norms (Johns & Nicholson, 1982; Martocchio, 1994). Systematic differences across these elements are reflected in the basic psychological contract between the organization and employees belonging to varying occupational status groups. For example, employees in low occupational

prestige groups, which operate under low discretion or low trust contracts, are likely to have diminished control over their use of work time due to stringent absence control recording systems and policies. This lack of control may prompt such individuals to see absence as legitimate, as something that they are entitled to (Nicholson & Johns, 1985). Knuttson and Goine (1998) also speculated that the higher absence rates of individuals in low occupation status groups could result from group norms prompting strong anti-firm sentiment, and consequently higher absenteeism. Employees in such groups are also likely to search for acceptable external reasons such as illness to justify their absence taking. Indeed, Harvey and Nicholson (1999) found that those in lower grade levels were more likely to legitimize illness as a reason for absence than those in higher grades.

Low absenteeism levels among high occupational status groups, on the other hand, is explained through the high discretion or high trust nature of the psychological contract of these occupational groups. Nicholson and Johns proposed that these elements of the contract contribute to managers' and professionals' view of absenteeism as illegitimate, resulting in guilt over the use of absence even for legitimate physical ailments. Employees belonging to high status occupations may also find less need for time away from work, because the generally flexible nature of their work schedules affords them greater control over the use of their time. Addae and Johns (2003), for example, explained the meta-analytic finding that flextime work schedules reduce absenteeism (Baltes, Briggs, Huff, Wright, & Newman, 1999) in terms of the lowered perceived legitimacy of absence as a work behavior by employees who work such schedules.

Adopting a socio-cultural view of stress, Barley and Knight (1992) suggested that

management levels and certain occupations tend to portray stress as an individual's responsibility, such that lack of resilience may be viewed as a failure to cope. Such occupational groups may refrain from taking absences when stressed because of the socialized view of their role as being resilient. In other words, norms about stress resilience may preclude such groups from taking time away from work when experiencing stress. Such resilience is often demonstrated in the nursing occupation as in Buchan and Seccombe's (1995) study where 70% of nurses admitted that they often came to work when they should have been off sick. In addition, Jamal and Baba (1991) found that job stress did not affect nurses' involvement in their jobs nor the amount of effort they put in their jobs. These findings suggest that occupation might moderate the stress-absence association, such that it is stronger for certain occupations than for others.

If occupational status increases with managerial and professional occupations, then the stress-absence association is expected to decrease. However, evidence for a moderator effect may also be provided by a change in the direction of the stress-absence association, such that highly stressed professionals or managers exhibit lower absenteeism. Corresponding somewhat with the occupational groupings used by Caplan et al. (1980), I use codes to distinguish between blue-collar (e.g., assembly line worker), low-level white collar (e.g., technicians, secretaries, clerks, supervisors), and high-level white-collar (e.g., managers, accountants, engineers, professors) occupational samples.

Occupational status is used as a proxy for social-cultural factors; however, I recognize that any observed effects may also result from underlying correlates, some of which were mentioned earlier (e.g., attitudes, beliefs, and job characteristics like physical work conditions, hours and schedules of work). Additional information about the sample

(i.e., health care, part-time, or shift workers) will be coded to enable additional comparisons whenever appropriate. I also recognize that any decreasing effect for occupational status might result in part from restricted variance in absenteeism at higher occupational levels (i.e., a floor effect). In other words, selection on occupational status (a third variable) restricts the range of absenteeism estimates. Although corrections for range restriction are possible, these might not be appropriate in this case for several reasons discussed by Sackett and Yang (2000). First, lack of knowledge of the population variances for stress, occupational status, or absenteeism precludes such a correction. Second, many unmeasured variables (e.g., attitudes, beliefs, absenteeism recording systems) and less-understood processes as theorized above might produce biased corrections. Terborg, Lee, Smith, Davis, and Turbin (1982) also suggested that corrections to associations involving variables that are correlated with features of the work environment could produce type II errors or falsely concluding that there are no situational effects.

Hypothesis 10:   The stress/illness-absence effect will decrease as occupational
                    status increases.

Role of Macro Contextual Changes

Pfeffer and Salancik (1978) were among the first to emphasize the influence of external environmental factors on individual experiences and behaviour. The influence of context has since then been recognized as an important source of variation in organizational behavior (Johns, 2001b; Johns, in press; Rousseau & Fried, 2001). Contextual factors can exist at various levels, some of them more distal than others (Mowday & Sutton, 1993). However, I discuss context in a very general sense, focusing

on changes in organizational and societal environments over the years. A noticeable and relevant trend is the increased attention to individual well-being and mental health by society in general. For example, the United States media labeled the 1980s as the stress decade, with titles like "Stress: Can we cope?" adorning the cover of popular magazines such as *Time* in 1983 (Best, 1990; Hatfield, 1990; Ramsay, 1999).

In the 1990s, national efforts to further understand and prevent work-related stress disorders were put in place by the United States government (Sauter, Murphy, & Hurrell, 1990). A special issue on the topic also appeared in the American Psychologist (1990). This increased awareness and recognition of stress in the workplace has not been restricted to the United States. A recent report by the World Health Organization suggests that the issue is global, and the United Kingdom, the European Union, and Canada have also taken steps to further understand and improve mental health in the workplace (World Health Organization, 2000). The increased societal-level recognition of stress is consequently witnessed at the organizational level. In a 1992 survey of organizations in the U.S., the implementation of work site health promotion programs was found to have increased dramatically over a period of only seven years (U.S. Public Health Service, 1993). Workplace health programs were also on the rise in Europe (Griffiths, 1996). Meta-analytic efforts to understand the effects of such programs on employee outcomes (e.g., Bauer, Amelio, LaGanek & Baltes, 2002; Unckless et al.,1988) also reflect the increased adoption of such programs in recent years, as the bulk of meta-analyzed studies date between late 1980 and late 1990. Have these contextual changes affected the basic stress-absence association over the years? Below, I explore alternate possibilities.

Influence of intervention effectiveness. On one hand, one might expect the

stress-absence effect be become increasingly constrained as stress-related organizational

interventions and policies succeed in reducing stress and absence in the workplace (note

that absence is often examined as an outcome in such interventions because of an

assumed stress-illness-absence progression). However, there is weak evidence for the

effectiveness of such programs. Even though Griffith's (1996) literature review on

employee fitness programs suggests some reported reductions in stress and absence, the

methodological limitations of such studies preclude any solid conclusions about the

beneficial effects of such programs.

Bauer et al.'s (2002) quantitative analysis of health promotion and fitness

programs also yielded a small effect on absenteeism, where as Unckless et al.'s (1998)

meta-analytic findings indicates that work wellness interventions were the least effective

in reducing absenteeism. In addition, wide variations in the effectiveness of a particular

type of stress intervention program have also been reported. For example, van der Klink,

Blonk, Schene, and van Dijk (2001) found cognitive-behavioral approaches to be better

than relaxation and job design efforts in reducing stress-related symptoms. However,

none of these approaches was effective in reducing absenteeism. These reported

variations and weak effectiveness of such interventions work against the constraint

hypothesis. Consequently, a noticeable decline in the stress-absence association over the

years is unlikely.

Influence of social information processing. The increasing attention to and

coverage of workplace stress and health issues over the years could, however, strengthen

the stress-absence association because of social information processing effects. Social

information processing theory suggests that formally and informally conveyed information influences individuals' attitudes and behaviors by directing their attention to certain issues (Salancik & Pfeffer, 1978). The proliferation of stress-related information in society and in organizations might have increased the salience of stress in the lives of employees. For example, the increasing frequency of stress-related publications in the popular and academic press over the years was explained from a social-cultural perspective as being a contributing factor to the rise in stress claims in North America (Barley & Knight, 1992). A U.S. survey mentioned in Spielberger and Reheiser (1994) found that the number of employees feeling highly stressed doubled over 1985-1990, the same period during which stress-related academic publications also increased. While the increase in employee stress levels over the years is plausible, Barley and Knight (1992) believe that people might have become more comfortable with stating that they're experiencing stress, an expression that is purely symbolic, having little to do with underlying stress-related psycho-physiological changes.

Their discussion implies that stress might have gained increased salience or legitimacy in the workplace because of increasing societal and organizational attention to related issues. Barley and Knight (1992) further explained how stress is often used as rhetoric by nursing unions to build collective support for some action. Similarly, employees might be more inclined to use stress as a reason for being absent because of its increased acceptance as a legitimate concern in organizations. This is likely given that the mere labelling of individuals as hypertensive resulted in them being more absent following awareness, even when no objective medical reasons to do so existed (Gafni & Peled, 1984; Haynes, Sackett, Taylor, Gibson, & Johnson, 1978). The increased salience

of this piece of information (i.e., hypertension) to employees was explained as having increased their propensity to view themselves as fragile and to adopt the sick role.

In a review of early findings, Levine and Kozloff (1978) suggested that physical ailments tended to enjoy higher legitimacy than mental or psychological illnesses. However, stress and other psychological conditions might have gained similar legitimacy over the years. For example, although physical symptoms such as throat infections, viral illness, diarrhoea, tonsillitis, and severe backache were rated as being the most legitimate reasons for being absent, depression was not far behind in its rating, perceived as being moderately legitimate on average (Harvey & Nicholson, 1999). The increased legitimacy of psychological symptoms is also reflected in Hackett et al's (1989) study, in which nurses created the term "mental health day," and cited it as the second most frequent reason for being absent on past occasions. Johns and Xie's (1998) Canadian sample of employees also rated minor illness and stress as being nearly equivalent in legitimacy. However, this finding was not evident in their Chinese sample, and was attributed to strong cultural values of self-restraint and self-control. While variations such as this one are likely to be observed in the literature, on average, I speculate that increased attention to workplace well-being issues might have raised the legitimacy of psychological illness and stress as acceptable reasons for staying away from work. In other words, individuals might feel less constrained in taking absence in response to stress or its psychological consequences. Consequently, the connection between stress/illness and absence is likely to be have become elevated over the years.

Any observed changes in effect size over the years are attributed to underlying substantive factors; however, artifactual confounds such as changing absence base-rates

over the years cannot be ruled out. However, it is unlikely for these rates to change so

dramatically as to alter the nature of the absenteeism distribution from year to year,

reducing its influence on the stress-absence association (cf., Harrison & Hulin, 1989).

Using a study's publication year as a proxy for some of the macro contextual changes

described above, the following is expected.

Hypothesis 11:   Reflecting an increasing legitimacy of stress in the workplace, the

stress/illness-absence effect will increase as chronological time

increases.

CHAPTER FOUR

4. Summary of Objectives and Hypotheses

This study investigates the causal influence of stress on absenteeism using a focussed theory-guided examination of the literature and a meta-analysis of accumulated findings. The general theory of causation is used to obtain a comprehensive understanding and tentative test for a causal association. Three main questions, depicted in Figure 1 by portions A, B, and C, are probed. As depicted in the figure, the following 11 hypotheses are to be tested against accumulated empirical findings using meta-analytic techniques.

Hypothesis 1:   The association between stress and absence will be positive

Hypothesis 2:   The association between psychological illness and absence will be positive.

Hypothesis 3:   The association between physical illness and absence will be positive.

Hypothesis 4:   The association between stress and absence will be partially mediated by illness such that psychological illness precedes the development of physical illness.

Hypothesis 5:   The magnitude of stress-absence and illness-absence associations will not differ across frequency and time lost measures of absence.

Hypothesis 6:   The associations between prior absence and subsequent stress, physical illness, and psychological illness will be positive.

Hypothesis 7a: The average stress-absence association in retrospective studies

using self-report measures of absence will be stronger than in those

using records-based absence.

Hypothesis 7b: The average illness-absence association in retrospective studies

using self-report measures of absence will be stronger than in those

using records-based absence.

Hypothesis 8a: Reflecting stable dispositional sources of variance, the stress-

absence effect will be positively associated with absence

aggregation periods.

Hypothesis 8b: Reflecting combined mid-term and stable sources of variance, the

stress-absence effect and absence aggregation association will be

near zero.

Hypothesis 9:   The positive stress/illness-absence effect will increase with the

percentage of women in the sample.

Hypothesis 10:   The stress/illness-absence effect will decrease as occupational

status increases.

Hypothesis 11:   Reflecting an increasing legitimacy of stress in the workplace, the

stress/illness-absence effect will increase as chronological time

increases.

CHAPTER FIVE

5. Methodology

5.1. *Literature Search*

An electronic and manual search was used to identify relevant articles. The search rule, absenteeism AND stress OR strain OR illness OR health, was inputted in electronic databases representing disciplines such as business/management (ABI Inform), psychology (PsycInfo), sociology (SocioFile), and epidemiology/ medicine (MedLine). The search was restricted to studies published between 1975 and 2003. Year 1975 was used as the start year not only because it is the earliest year covered by most of the databases to be searched (cf. Konrad, Ritchie, Lieb, & Corrigall, 2000), but also because relatively little research on stress *and* absenteeism occurred before this year. For example, in their qualitative review of the stress and health literature Beehr and Newman (1978) identified only one article examining stress and absenteeism prior to 1975. A later empirical review of predictors and consequences of absenteeism by Farrell and Stamm (1988) also confirms the paucity of research prior to this year; only two studies examining psychological stress were published before 1975.

A manual search followed with a scan of previous reviews covering the topic (e.g, Beehr & Newman, 1978; Farrell & Stamm, 1988; Johns, 1997; Shaufeli & Peeters, 2000). The aim was to identify articles that were potentially missed in the electronic search. In addition, popular journals focusing on stress research (i.e., *Journal of Advanced Nursing, Work & Stress, International Journal of Stress Management, and Stress Medicine)* were perused depending on their availability in local libraries and electronic full-text resources.

Unpublished doctoral dissertations, which appeared in the electronic search results, were obtained through *Digital Dissertations* or inter-library loans. An email (see Appendix B) requesting unpublished research or conference presentations was also sent to a list of members belonging to the Canadian Society for Industrial and Organizational Psychologists (CSIOP) in April-May 2004.

5.2. Data Compilation

The above search processes yielded approximately 3600 articles/abstracts, which included about 93 dissertations and 630 non-English articles in a variety of languages. For cost and quality control reasons, only studies published in the English language were probed further, because determining the relevance of each non-English article required first translating it into English, a rather expensive process, which did not guarantee an article's inclusion in the meta-analysis. In addition, approximately 95 percent of the non-English articles were published in the epidemiology discipline, in which stress research methodology tends to be less rigorous than that in the management discipline (Xie & Schaubroeck, 2001). Therefore, in addition to cost, quality control issues influenced the decision to focus on English articles.

Study titles and abstracts from the search results were reviewed for relevance. Studies excluded at this stage were those that were either qualitative in nature, examined absenteeism from medical intervention programs, examined school absenteeism, or tested the effects of certain drugs on post-surgery recovery. This screening process yielded approximately 900 remaining published studies and 58 dissertations, which were obtained in full-text format to further discern their relevance. Studies excluded during this second screening process included non-empirical studies, those that did not report the

required statistical information, and those that did not measure the variables of interest. For example, studies examining partial absence (e.g., leaving work early or spending time on the phone), absenteeism intentions, absenteeism not specific to work (i.e., absence from usually performed activities), presenteeism, or costs associated with being absent were excluded from further examination.

Consistent with previous meta-analyses on absenteeism, only studies with individual-level data and analysis were retained. As discussed earlier, studies examining stressors or personal stress (i.e., domestic, financial, health) were excluded. For the illness variables, care was taken to ensure that the measure was not contaminated by its inclusion of an item assessing how many times a person was absent due to illness. Studies examining student samples or those examining the same sample and measures in multiple publications were also excluded from further examination. An attempt was made to contact authors of studies that measured the desired variables but that did not report zero-order correlations. Of the 16 authors who were contacted via email, three supplied the requested information. Most of the others indicated that they did not have the data anymore; a few did not respond. For one unpublished dissertation (Knowles, 1980), which reported tables of raw data I was able to calculate the required zero-order correlation myself. The above process identified 137 empirical studies (112 published and 22 dissertations) containing sufficient information required for a meta-analysis of correlations.

Although the focus of this synthesis involved an analysis of correlations (r), an effort was made to provide an analysis of the odds ratio (OR), a statistic used to estimate the association between two dichotomous variables. Previous meta-analysts in the field

of organizational behavior have tended to limit their focus to studies that report r or information to enable the calculation of d estimates. For example, Martocchio et al. (2000) attributed the low number of studies in their meta-analysis on low back pain and absenteeism to the lack of correlation matrices in medical articles. As a substantial proportion of epidemiology studies tended to report information surrounding the calculation of OR rather than r, a separate meta-analysis of odds ratios was performed for studies that reported cell frequencies.

While it is possible to calculate the phi coefficient between dichotomous variables, and then convert it to r (Glass, McGaw, & Smith, 1981; Rosenthal,1994), such conversions are inappropriate because they use effect sizes developed for continuous variables to estimate effects based on dichotomous data (Fleiss, 1994; Haddock, Rindskopf, & Shadish, 1998). Haddock et al. strongly recommend the use of the odds ratio in estimating the association between dichotomous variables. Out of a total of 96 empirical studies meeting the basic requirements for inclusion (i.e., measured absenteeism from work and stress or illness), only 16 provided sufficient information that enabled the calculation of the odds ratio (OR).

Data coding. Following Stock (1994), a coding sheet was designed for the purposes of this project (see Appendix A). For each study, substantive items such as the type of absence measure, stress, illness variables, gender, occupational status, research design, and additional variables (e.g., acute stress, long-term absence measures) were coded. Coded methodological items included effect size estimates, scale reliabilities, and sample size. A list of all the studies included in this meta-analysis is presented in Table 15, and shows some of the key coded characteristics. Given the wide variation in

measures used to assess stress and illness in the literature, care was taken to avoid

subjective interpretation in coding a measure as stress, psychological illness, or physical

illness. Whenever a study reported insufficient information about any of these measures,

additional information was sought by either locating the measure itself, consulting

publications containing details about the items in the scale, or obtaining reviews of the

measure published in the Mental Measurements Yearbook.

Examples of measures coded as assessing work-related stress included Parker and

DeCotiis' (1983) measure of job stress, the Stress Diagnostic Survey (Ivancevich &

Matteson, 1985), the Occupational Stress Indicator (OSI, Cooper, Sloan, & Williams,

1987), job-related tension (Kahn et al., 1964), job-induced anxiety (House & Rizzo,

1972), job frustration scale (Peters, O'Connor, & Rudolf, 1980), Teacher Stress Scale

(Pettegrew & Wolf, 1982), and the Nursing Stress Scale (Gray-Toft & Anderson, 1981).

Many studies created their own measure of perceived stress, and these were coded as

such if respondents were asked to rate the amount of stress, pressure, or anxiety

experienced from their job in general or from various aspects of their work. Job-induced

anxiety and job-related tension were included as measures of stress because their items

are often found in typical job stress scales. In fact, Parker and Decotiis' job stress

measure contains two dimensions of stress, time pressure and job-related feelings of

anxiety. In studies that did not report details on their stress measure, some discretion was

used in determining its inclusion.

Measures coded as assessing psychological illness included the General Health

Questionnaire (GHQ, Goldberg, 1972), negative mood states (PANAS, Watson, Clark &

Tellegen, 1988), mental illhealth subscale of the OSI (Cooper et al., 1987), stress

reactions subscale (Setterlind & Larsson, 1995), state anxiety subscale (Spielberger,

1979), the Symptom Checklist (Derogatis, 1977), the Maslach Burnout Inventory (MBI,

Maslach & Jackson, 1981), the Zung Self-rating Depression Scale (SDS, Zung, Richards,

& Short, 1965), and the Center for Epidemiologic Studies - Depression scale (CES-D,

Radloff, 1977). Note that only studies using *state* measures for some constructs (e.g.,

mood, anxiety) were retained in this analysis.

Examples of physical illness measures include the Somatic Complaints Index

(Caplan et al., 1980), somatic symptoms subscale of the GHQ (Goldberg, 1972), physical

illhealth subscale of the OSI (Cooper et al., 1987), and the Cornell Medical Index

(Brodman, Erdmann, & Wolf, 1960). Many studies used measures containing items of

both psychological and physical symptoms; these were coded as measures of

psychosomatic illness. Other studies assessed a particular illness by asking respondents

to report on the number of asthma attacks, migraines, or headaches. Note that many of

the above measures were administered in their extant translated version (e.g., German or

Dutch translation of the MBI) or were translated to another language prior to use (e.g.,

Chinese translation of the OSI). Studies examining low back pain were excluded,

because an existing meta-analysis on the association between this variable and

absenteeism already exists (e.g., Martocchio et al., 2000).

Coding Accuracy. To ensure consistency in coding, I was responsible for coding

all the studies. However, to ensure the accuracy of my coding, a random sample of 20

studies was selected for recoding by a second coder. A fellow PhD student, who was

given an orientation session explaining the requirements of the task, along with practice

time to code three samples, performed this task for a small fee. Given that each article

can take between 10 and 40 minutes to code, only items directly pertinent to the

hypotheses were recoded. These items included the percent of women in a sample, the

occupational status of a study's sample, research design, absence measure, number of

months over which absence was aggregated, reason for absence, type of absence, the

scoring direction of the independent variable, sample size, and health-care nature of the

sample.

Given much disagreement over the appropriateness of a particular inter-rater

agreement statistic (Orwin, 1994), two indices, agreement rate (AR) and kappa (κ), were

calculated for categorical items. For continuous items such as sample size, percent of

women, absence aggregation period, and year of publication, the simple correlation (r)

between each coder's entry was calculated. Consistent with Orwin's discussion, inter-

rater agreement estimates are presented on an item-by-item basis: percent of women in a

sample ($r = 0.99$), occupational status of a study's sample (AR = 0.85, $\kappa = .811$),

research design (AR = .50, $\kappa = .50$), type of absence measure (AR = 1, $\kappa = 1$), number of

months over which absence was aggregated ($r = .98$), reason for absence (AR = .80, $\kappa =$

.762), type of absence (AR = .95, $\kappa = .92$), scoring direction of the independent variable

(AR = .90, $\kappa = 0$), sample size ($r = .99$), and health-care nature of the sample (AR = 1, κ

= 1).

As seen above, AR and kappa estimates are quite similar, with one exception

(scoring direction of the independent variable). For this particular item, there were no

cases in the sample of studies where the scoring direction was opposite. Therefore, one

wrongly coded item resulted in the observed agreement being equal to that expected by

chance. This is not surprising, because even though kappa overcomes the problems of

AR by taking into account chance agreement, it is not suitable when observations are concentrated in only a few cells (Orwin, 1994). Therefore, the skewed distribution of observations might have contributed to the obtained zero kappa value for this item. In determining acceptable agreement, values greater than .75 (for kappa) and greater than .80 (for correlations) are regarded as being excellent (Orwin, 1994). With the exception of the research design item, there appears to be acceptable agreement across all items. Nevertheless, both coders exchanged viewpoints to discuss items that had less than perfect agreement. For all items except research design, the main source for a wrong code was the inability of the second coder to locate relevant information about the item within the article. In such cases, making the coder aware of the information resolved most of the discrepancies in coding.

For research design, most disagreements were due to differences in interpretation of the description of a measure provided in each study. For example, a study's research design could be coded as predictive, postdictive, and cross-sectional. Coding a study as predictive tended to be more straightforward, as there was perfect agreement between coders on this category. The key factor used to determine whether a study was postdictive or cross-sectional was the wording of the instructions on the independent variable measure (i.e., stress or illness). If respondents were asked to rate their stress or symptoms over some past time period, and if this time period overlapped with the absence aggregation period by 25 percent or more, the study was coded as cross-sectional. If respondents were asked to rate their current state of health/stress and past absence, then the study was coded as postdictive. Most of the studies receiving discrepant codes used the Maslach Burnout Inventory, which asked respondents to rate

the frequency with which they experienced burnout (scale anchors: never occurs to everyday), and the General Health Questionnaire, which asked respondents to indicate if they recently experienced any symptoms. These studies were coded as cross-sectional by the second coder, but as postdictive by the first. Upon further discussion, it was concluded that some reference to the past is generally likely when determining a respondent's current health/wellbeing condition. Therefore, such studies retained their postdictive codes.

5.3. Meta-analytic Procedure

The Comprehensive Meta-Analysis software (Borenstein & Rothstein, 1999) was used to synthesize the compiled effect sizes. Whenever a study reported mean differences between two or more groups, the d effect size was first computed and then converted to r. Whenever a study compared two independent groups, procedures outlined in Hunter and Schmidt (1990a, 2004) were used for this conversion. For studies comparing more than 2 independent groups or those with factorial, matched- or repeated-measures designs, other methods and decisions had to be utilized. Based on discussions surrounding the inappropriateness of combining d estimates obtained from independent groups designs with those from repeated- or matched-designs (Gillett, 2003; Morris & DeShon, 2002; Ray & Shadish, 1996), studies with matched- or repeated-measures designs were excluded from the analysis. In addition, difficulties associated with converting estimates of d obtained from one design to another (i.e., requires correlation between the repeated-measures or matched factors) precluded the inclusion of such effects. The focus on effects from independent groups designs is consistent with the correlational nature of the bulk of absenteeism research, where between-person

differences have been of interest (Johns, 2003).

For calculations of d in studies with factorial designs, procedures outlined in Cortina and Nouri (2000) were used. For studies comparing more than two independent groups (e.g., very mild, mild, moderate, severe symptoms), the sample size-weighted mean and pooled standard deviation of the very mild and mild groups were calculated and compared against the sample size-weighted mean and pooled standard deviation of the moderate and severe symptoms group. Combining the groups in this way corresponds somewhat to continuous stress/illness measures used in surveys, in which rating scales range from low stress/illness to high stress/illness. In some studies, absenteeism among those without any illness was compared to those with some form of an illness. Given that continuous survey measures of illness sometimes ask respondents to check the number of illnesses they experience, where a zero score suggests no illnesses, comparisons of "no illness" to "some illness" were deemed appropriate for inclusion with the remainder of the studies in this sample.

Care was taken to avoid comparing extreme groups. For example, in one study, two different effect size computations were possible (one comparing past absenteeism of individuals who were HIV-negative against those who were HIV-positive, and the other comparing HIV-negative individuals against those with AIDS). A comparison of extreme groups (i.e., HIV-negative and AIDS) resulted in a higher effect size ($r_{pb} = .60$) than one obtained from a comparison of HIV-negative and HIV-positive individuals ($r_{pb} = .24$). To enable a fairer comparison, the sample size-weighted average of the HIV-positive and AIDS group was compared with the HIV-negative group in this particular study. Whenever possible, studies involving conversions from d to r were calculated in a

way that enabled a comparison of their effects with those obtained from r.

Artifact corrections. Each effect size was corrected individually for sampling

error, while corrections for measurement error were made using artifact distributions (i.e.,

aggregate estimates of the reliabilities). The average reliability for each variable, which

was calculated from studies included in the present meta-analysis, was used to correct the

distribution of effects for attenuation. These estimates are: stress ($r_{xx}= .791$, $k = 36$),

psychological illness ($r_{xx} = .832$, $k = 58$), somatic illness ($r_{xx} = .799$, $k = 27$), frequency/

attitudinal absence ($r_{yy} = .530$, $k = 3$), and time lost absence ($r_{yy} = .532$, $k = 8$). In

correcting for attenuation in the dependent variable, the average reliability ($r_{yy} = .532$)

across time lost and frequency absence was used. For the sake of comparison, the

average reliabilities of absenteeism measures obtained in Hackett and Guion's (1985)

meta-analysis, which are often used in other meta-analyses to correct for attenuation, are

.66 (time lost) and .51 (frequency).

Consistent with previous meta-analyses on absenteeism (e.g., Bycio, 1992; Farrell

& Stamm, 1988, Hackett, 1989, Hackett & Guion, 1985; Martocchio, 1989), effect sizes

were not corrected for range restriction, because of problems in first defining the

population to which the effect size is to be generalized, and then obtaining estimates of

the variance of a particular variable in this population (Sackett & Yang, 2000). The

conversion of d to r introduces another artifact (i.e., dichotomization), because estimates

of r obtained from the conversion of d are really point biserial rather than bivariate in

nature, and biserial correlations tend to be smaller than bivariate ones (Hunter &

Schmidt, 1990a; MacCallum, Zhang, Preacher, & Rucker, 2002). Dichotomization issues

are also relevant when studies report correlations between a dichotomized variable (e.g.,

high/low absence, high/low stress, presence/absence of stress or illness) and a continuous one.

Corrections for dichotomization are available such that the point biserial correlation is corrected by a factor based on the proportion of individuals in each category (cf., Wanous, Poland, Premack, & Davis, 1992). However, such corrections are based on the assumption that the distribution of the underlying continuous variable is normal (Hunter & Schmidt, 1990b). Absence is a low base-rate behavior, and its distribution is generally positively skewed, while the distributions of some other categorical variables included in this meta-analysis (e.g, headaches, hypertension) are unknown. Therefore, available corrections for dichotomization were not appropriate in this synthesis, yielding potentially biased estimates because of violated normality assumptions. In addition, even when biserial correlations are corrected for dichotomization, they are likely to deflate the estimated variance due to artifacts, which inflates the estimated standard deviation of the distribution of observed correlations. Hunter and Schmidt (1990a, 2004) recommend that it is best to delete non-Pearson correlations from the meta-analysis; however, this is a feasible option only when the sample of compiled effects is large. In an effort to retain as many effect size estimates as possible, effects based on point biserial correlations were coded as such, to determine the extent to which such correlations influenced the main results.

Independence assumption. In meta-analyzing findings using the Hunter and Schmidt (1990a, 2004) approach, there is a requirement that the accumulated estimates be statistically independent. Studies having fully replicated designs (i.e., measures and associations are reported for different organizations), conceptually similar measures, and

sub-sample analyses often report more than one estimate. Based on Hunter and Schmidt, estimates from studies with fully replicated designs (i.e., correlations are reported for each organization separately) were treated as being independent and were all included in the meta-analysis. For studies whose measures were replicated over time (e.g., stress-absence associations were examined before and after some intervention), only estimates at time 1 (when reported) were included, to obtain an effect devoid of the impact of the intervention. However, when a study reported only time 2 or post-event estimates, these were included but coded in a way to enable posthoc comparisons.

For studies using conceptually similar multiple measures (i.e., measure the same construct), Hunter and Schmidt recommend the calculation of a composite correlation (based on the intercorrelation of the conceptually similar measures and their associations with the target variable), or the average correlation whenever intercorrelations are not reported. Composite correlations were calculated using procedures outlined in Nunnally (1978) and Hunter and Schmidt (2004). For example, for studies assessing physical illness using measures of headaches and sleep problems, or psychological illness using measures of anxiety and depression, a single composite correlation between the conceptually similar measures and absenteeism was calculated and entered in the meta-analysis. To enable post hoc comparisons across each burnout dimension, studies reporting associations for absenteeism and emotional exhaustion, depersonalization, and lack of personal accomplishment were entered as is. Other exceptions included cases where multiple estimates enabled a test for any of the hypotheses (e.g., frequency and time lost absence measures, or for predictive and postdictive measures). While this introduces some dependence among effects, an attempt was made to determine the

impact of non-independence in the full sample of effects.

With respect to correlations based on multiple sub-samples, Hunter and Schmidt

caution against using sub-sample estimates, especially when there is no solid evidence for

a moderator effect based on the variable dividing the sample. As sub-sample estimates

are based on smaller sample sizes and a restricted range of scores, they are likely to

increase the risk of type II error. The use of total group correlations is recommended,

unless the meta-analysis is specifically aimed at explaining sub-group differences. As

gender and occupational status (e.g., blue/white collar) were hypothesized as potential

moderators in the present study, estimates for males and females and for blue- and white-

collar samples reported in a study were each entered in the meta-analysis. Sub-sample

estimates based on variables not theorized in this examination (e.g., Anglophone versus

Francophone employees), were averaged across the two samples.

CHAPTER SIX

6. Results

A total of 275 effects from 115 published studies and 22 dissertations were available for analysis. Each study, therefore, contributed an average of two effects sizes. This sample of effects represented varied occupations and settings. These included the medical profession (nurses, doctors, technologists), manufacturing/production (engineers, technicians), education (teachers, counselors), social work (social workers, personal care workers), blue collar occupations (factory assembly line workers, miners, welders, cleaners, bakers, sewing machine operators), administration (clerical, secretaries, managers, directors), government (civilian and military employees), retail (sales persons, customer service representatives), and security-related fields (e.g., security guards, firefighters, police). Aggregating over samples, the mean age of a sample was approximately 37.54 years ($SD = 4.54$), and comprised an equal percentage of male and female employees ($M_{percent\ female} = 51.90$, $SD = 30.63$).

Consistent with the Hunter and Schmidt (1990a) approach, the following analyses assume a random effects model. In other words, between-study variation is considered to reflect true rather than random differences across studies (Borenstein & Rothstein, 1999). Credibility and confidence intervals were calculated in accordance with Whitener (1990). Credibility interval computations utilized the posterior distribution of effects (i.e., those corrected for sampling and measurement error), and were calculated before the calculation of confidence intervals. Confidence interval computations depended on the width of the credibility interval (homogeneous or heterogeneous), and were based upon

observed or sample size-weighted effects. Non-zero confidence intervals provided a test for the significance of a particular effect size, while the Z-test (Hunter & Schmidt 1990a, p. 437) was used to compare two effect sizes.

In determining the presence of moderators in this synthesis, a conjunctive combination of the 75 percent rule and credibility intervals was used (Cortina, 2003). In other words, the operation of substantial moderators was deemed likely whenever the amount of variance explained by artifacts was less than 75 percent and the credibility intervals were wide or contained zero values. There is presently no established criterion for determining how wide a credibility interval should be to signal the presence of moderators. A credibility interval ranging from .04 to .48 was interpreted by Whitener (1990) as being positive and thus the search for additional moderators was deemed unnecessary. Yet, Kuncel, Hezlett, and Ones (2004) simply examined the lower value of the credibility interval to determine whether it was positive or negative. In their meta-analysis, a positive lower credibility value with substantial unexplained variance was interpreted as signaling some fluctuation in the effect's magnitude across settings. In the present study, the CI formula for heterogeneous samples was always used when the lower credibility value was negative. But, when the lower credibility value was positive, the percent of variance explained by artifacts dictated the use of a particular CI formula. When this percent exceeded 75, CI computations were based on a homogeneous sample.

6.1. *Absence as Withdrawal from the Workplace*

Table 1 presents the sample size-weighted and corrected mean effects for each theorized predictor and type of absenteeism. The overall associations between absenteeism and stress ($r_{obs} = .09$, $r_{corr} = .15$, $k = 56$), psychological illness ($r_{obs} = .13$, r_{corr}

$= .20, k = 128$), and somatic illness ($r_{obs} = .14$, $r_{corr} = .22$, $k = 65$) are positive and

significant, but small (Cohen, 1988). As indicated by the lower negative credibility value

and the small percent of variance accounted for by artifacts, the effects are likely to vary

across persons and situations. However, some of this unaccounted variance may also be

due to uncorrected artifacts. The observed effect for stress is significantly smaller than

that for psychological illness ($Z = 2.50, p < .01$) and somatic illness ($Z = 2.67, p < .01$),

providing some evidence for discriminant validity between measures of stress and

measures of illness. There is no significant difference between effects for psychological

and somatic illness. (Note that the Z-test for significance here and in all other

comparisons is based on the distribution of observed effects). The results reported in

Table 1 are based on all absence measures (i.e., those reported to be due to illness or

stress-related causes, those due to reasons like family or personal needs, and those in

which the reason for absence was not specified in a study). When effects are aggregated

using only those absence measures said to be due to illness, the results are not

significantly different from those reported above (for stress: $r_{obs} = .119$, $r_{corr} = .186$, $k = 28$; for psychological illness: $r_{obs} = .135$, $r_{corr} = .205$, $k = 79$, for somatic illness: $r_{obs} = .129$, $r_{corr} = .199$, $k = 45$).

In an effort to infer causality, Hypotheses 1- 3 were offered in the context of

predictive studies. Table 3 presents these effects broken down by research design.

Although based on fewer samples, the overall effects for stress, psychological illness, and

somatic illness in predictive studies continue to remain positive, and are even larger for

stress ($r_{obs} = .26$, $r_{corr} = .40$, $k = 6$) and somatic illness ($r_{obs} = .17$, $r_{corr} = .25$, $k = 5$) in

comparison to their full sample counterparts. The non-zero positive confidence intervals

for each overall effect provides a test for Hypotheses 1-3, confirming that individuals experiencing increased stress or illness are more likely to escape the workplace in the form of absenteeism. When effects are examined for each type of absence, the stress-absence effect appears to be larger for frequency measures (r_{obs} = .28, r_{corr} = .431, k = 4), but it is not significantly different from the effect for time lost absence measures (r_{obs} = .16, r_{corr} = .247, k = 2, Z = 1.23, p > .05). However, given the small number of effects (2 ≤ k ≤ 4) upon which this comparison is based, one cannot rule out the risk of committing a type II error or concluding that there is no significant difference when there could be one (Hunter & Schmidt, 2004). With the exception of somatic illness, substantial unexplained variance (45 to 50 percent) in the effects for psychological illness and stress suggests some variation across settings. Overall, the results in Table 1 provide general support for the withdrawal model of absenteeism.

Levels of analysis and interpretation of results. Even though this meta-analysis is based on effects collected and analyzed at the individual level, interpretation of the aggregated population estimate is dependent on the extent to which primary studies sampled a single organization or multiple organizations. Ostroff and Harrison (1999) refer to the population estimate based on single sample effects as the *correlation within*, which is interpreted as the correlation that can be expected within any single organization. Effects based on multiple samples, on the other hand, are said to reflect the *individual correlation* between any two variables. When a meta-analysis is based on studies that contain single organization samples, the resulting meta-analytic effects are accurately interpreted as being generalizable across other settings or organizations. In other words, validity generalization statements about the results are appropriate.

However, when a meta-analysis includes studies with single and multiple organization samples, such an interpretation becomes ambiguous. In addition, as Ostroff and Harrison (1999) illustrate, it is also possible for the *correlation within* and *the individual correlation* to be different.

To ensure accurate interpretations about the obtained stress/illness-absence effects, effects based on single- and multiple-organization samples were meta-analyzed separately. This was possible whenever studies clearly reported such information. As seen in the upper portion of Table 2, an almost equal number of effects for stress, psychological illness, and somatic illness are represented by single- and multiple-organization samples. When compared across the two types of samples, there are no significant differences in the overall observed effect for stress ($r_{single} = .09$, $k = 29$; $r_{multiple} = .11$, $k = 20$; $Z = .784$, $p > .05$), psychological illness ($r_{single} = .11$, $k = 69$; $r_{multiple} = .14$, $k = 51$; $Z = 1.81$, $p > .05$), or somatic illness ($r_{single} = .16$, $k = 30$; $r_{multiple} = .14$, $k = 31$; $Z = .78$, $p > .05$). These findings suggest that validity generalization interpretations of the overall effects reported in Table 1 are feasible. In addition, the stress/illness-absence correlation expected within any single organizations is not likely to differ from the individual correlation between stress or illness and absenteeism.

Impact of non-independence. The results reported in Table 1 are based on non-independent samples, which may underestimate sampling error and overestimate the population variance (Hunter & Schmidt, 1990a). In an effort to determine the impact of non-independence, effects compiled from less-dependent samples are reported in the upper portion of Table 2. Multiple effects reported within a single study were averaged either across research design, type of absence measure, gender sub-samples, or

conceptually similar variables (e.g., burnout dimensions). As observed, the results

reported for overall effects in a less dependent sample are not significantly different (.32

$\leq Z \leq .48$, $p > .05$) from those reported in Table 1, suggesting that non-independence is

not likely to be a major problem in this synthesis (for stress: $r_{obs} = .10$, $r_{corr} = .16$, $k = 49$;

for psychological illness: $r_{obs} = .14$, $r_{corr} = .21$, $k = 75$; for somatic illness: $r_{obs} = .15$, r_{corr}

$= .23$, $k = 47$).

Availability/publication bias. One of the criticisms against meta-analyses is that

published studies contain larger effects than those that are not published (Hunter &

Schmidt, 2004). To determine the extent of this bias, effects for stress, psychological

illness, and somatic illness reported in unpublished dissertations were compared to the

overall effects reported in Table 1. The overall effects in unpublished studies (see Table

2) for stress ($r_{obs} = .12$, $k = 10$), psychological illness ($r_{obs} = .16$, $k = 15$), and somatic

illness ($r_{obs} = .17$, $k = 4$) are not significantly different from those for the full sample of

effects ($r_{stress} = .09$, $r_{psych} = .13$, $r_{somatic} = .14$, $.87 \leq Z \leq 1.05$, $p > .05$). Therefore, there

appears to be little evidence for publication bias.

Type of effect size. Effects obtained from statistics other than bivariate

correlations (i.e., point biserial correlations) might be smaller in size, because of

restricted variance in the dichotomized variable. Alternatively, such effects can also be

large, depending on whether the comparison groups were based on extreme scores. The

influence of dichotomization can be minimized through corrections, but as explained

earlier, lack of certain requirements (e.g., normal distribution, knowledge of population)

precluded the correction for dichotomization. The impact of including point biserial

correlations was assessed by comparing such effects (reported in lower portion of Table

2) against those reported for the full sample of studies in Table 1. The point biserial

effects for stress (r_{obs} = .13, r_{corr} = .20, k = 4) and somatic illness (r_{obs} = .12, r_{corr} = .12, k =

10) did not differ significantly from their full sample counterparts, while the effect for

psychological illness (r_{obs} = .23, r_{corr} = .34, k = 9, Z = 2.08, p < .05) was significantly

larger than that obtained for the full sample. However, its impact on the overall full

sample effect for psychological illness was negligible, as a re-analysis without the point

biserial subset of effects for psychological illness yielded a similar overall effect for this

predictor (r_{obs} = .13, r_{corr} = .19, k =110, Z = .618, p > .05). The effect for psychological

illness in predictive studies also remained similar when point biserial correlations were

removed from the set (r_{obs} = .11, r_{corr} = .16, k = 27, Z = .14, p > .05). All subsequent

analyses involving psychological illness were examined with and without the set of point

biserial correlations. The outcomes of this analysis are reported only when notable

differences across the two sets are observed.

In some studies, reported correlations were based on absence measures that were

reportedly transformed (mostly log transformations). To determine the extent to which

effects based on transformed absence measures influenced the obtained results, a sub-set

of such effects was compared with those reported in Table 1. This was possible only for

psychological illness, because a sufficient number of effects were available for this

comparison. The obtained mean based on transformed absence measures (r_{obs} = .12, r_{corr}

= .18, k = 8) was not significantly different from the overall full sample psychological

illness effect (r_{obs} = .13, r_{corr} = .20, Z = .463, p > .05), suggesting that statistical

transformations of the absence measure are not likely to influence the obtained average.

Bidirectionality of effects. Tett, Jackson, Rothstein, and Reddon (1999) discuss

how the presence of positive and negative effects in a sample can underestimate the

aggregated magnitude of an effect when standard meta-analytic procedures are used (e.g.,

Hunter & Schmidt, 1990a). While knowledge of key characteristics can be used to

account for opposing signs by conducting moderator sub-group analysis, Tett et al.

recommend alternate approaches to estimating an effect when potential moderators

cannot be identified in advance. In the present meta-analysis, negative effects were

expected in studies examining the influence of some stress management intervention (i.e.,

post-intervention effects). This expectation was confirmed as a significant negative

association ($r = -.20$, $p < .01$) between such effects (coded 1= post-intervention, 0 = no

intervention) and the sign of an effect (coded 1 = positive, 0 = negative) was observed.

Post-intervention effects were taken into account in subsequent analyses whenever

relevant, and are reported only when significant differences with and without their

inclusion are observed. Nevertheless, the influence of bidirectionality on the obtained

results is likely to be minimal, as 90.2 percent of the entire sample of effects had positive

signs. In addition, the removal of effects with negative signs from the sample yielded

results that are no different from those reported in Table 1 (for stress, $r_{obs} = .12$, $r_{corr} =$

$.18$, $k = 48$, $Z = 1.17$, $p>.05$; for psychological illness, $r_{obs} = .14$, $r_{corr} = .21$, $k = 112$, $Z =$

0.55, $p>.05$; for somatic illness, $r_{obs} = .14$, $r_{corr} = .22$, $k = 63$, $Z = 0.02$, $p>.05$).

Effects by Coded Illness Categories

Table 4 presents the average effect for each coded illness category. Effects

grouped under the "other" category represent varied illnesses such as colds/flu, sleep

problems, stomach problems, and headaches. To enable causal inferences, effects within

each illness category were further broken down by predictive and postdictive research

design. This was done whenever there were at least three effects within each design.

Although no hypotheses were offered in relation to specific illnesses, several

observations are notable. For example, as reported near the top of Table 4, a small

positive association ($r_{corr} = .09$) is evident for acute stress or illness. These effects were

obtained in the context of short-term distressing events such as the 9-11 terrorist attacks,

organizational restructuring, or a prime minister's assassination. As five of the six effects

for acute stress/illness were obtained from cross-sectional studies, which utilized self-

report measures of absence, the influence of common method bias cannot be ruled out.

In addition, causality cannot be inferred.

The effects obtained for burnout are also interesting. Burnout is often theorized as

an extreme stress response, occurring after repeated and long-term exposure to chronic

stressors (Maslach et al., 2001). It is conceptualized as having a three-factor structure

(emotional exhaustion, depersonalization, and lack of personal accomplishment),

although support for three dimensions has not always been received (e.g., Kalliath,

O'Driscoll, Gillespie, & Bluedorn, 2000). To infer causality for each burnout dimension,

consider the effects for predictive studies (see Table 4). Although each effect is based on

a small number of samples ($4 \leq k \leq 6$), the confidence interval for lack of personal

accomplishment (95% CI = .12 to .27) is much larger than that obtained for emotional

exhaustion (95% CI = .01 to .13) and depersonalization (95% CI = -.03 to .13). These

findings suggest that employees are more likely to be absent in response to feelings of

lack of personal accomplishment than in response to feeling emotionally exhausted, while

the effect for depersonalization is not significant. The pattern of findings is somewhat

aligned with Maslach's (1993) suggestion that the burnout process unfolds such that

feelings of lack of personal accomplishment follow those of emotional exhaustion and depersonalization. Feeling a lack of personal accomplishment reflects, perhaps, more severe burnout, making employees realize the toll that work stress exerts on their capabilities and prompting them to escape the workplace.

If burnout is theorized as a response to stress, it should have stronger associations with absenteeism because it occurs closer to absence in the nexus of theorized events. However, an examination of the effects for each dimension in comparison to that obtained for stress in predictive studies provides little evidence for this progression. The predictive effect for stress ($r_{obs} = .25$, $r_{corr} = .43$, $k = 6$) is not significantly different from that for lack of personal accomplishment ($r_{obs} = .19$, $r_{corr} = .29$, $k = 4$, $Z = 1.09$, $p > .05$), but it is significantly larger than for emotional exhaustion ($r_{obs} = .07$, $r_{corr} = .10$, $k = 6$, $Z = 3.52$, $p < .01$, one-tailed) and depersonalization ($r_{obs} = .05$, $r_{corr} = .08$, $k = 4$, $Z = 3.26$, $p < .01$, one-tailed). Although these findings suggest that emotional exhaustion and depersonalization might precede the onset of stress, these effects are based on four to six samples only, and must be interpreted with caution. Clearly, more research into the distinction between these varied dimensions and stress is required.

Examining the effects for some of the other illnesses in Table 4, it can be seen that all the effects (overall, predictive, postdictive) for depression and absenteeism are positive, significant, and homogeneous, suggesting that the obtained positive associations ($.18 \leq r_{corr} \leq .29$) are likely to remain stable across persons and situations. As reflected by their zero confidence intervals, the effects for anxiety and negative mood are not significant, but this finding may due to the small number of samples ($k = 3$) upon which the effects are based. In comparison, the effect for fatigue is strongly positive ($r_{corr} =$

.49), but likely to be moderated by substantial or other uncorrected artifacts, because of its wide credibility value and low percent of the artifactual variance. As for the composite measures of psychological, somatic, and psychosomatic illnesses (i.e., those measuring a number of psychological or somatic symptoms within the same scale), the obtained pattern of effects is consistent with that reported in Table 1. For example, the overall effects for composite measures of psychological ($r_{corr} = .22$, $k = 50$) and somatic illness ($r_{corr} = .25$, $k = 49$) are comparable to their counterpart effects in Table 1.

6.2. Mediating Influences of Illness

Following procedures outlined in Viswesvaran and Ones (1995), Structural Equation Modeling (SEM) was used to test the sequential influence of psychological and somatic illness as mediators of the stress-absence association (Hypothesis 4). This approach required an inter-correlation matrix of effects among stress, psychological illness, somatic illness and absenteeism. Therefore, additional data had to be compiled to enable the estimation of the stress-psychological illness, stress-somatic illness, and psychological-somatic illness effect through separate meta-analyses. Whenever studies included in the present synthesis reported any such correlations, they were compiled along with their reported reliabilities. The stress-psychological illness effect was estimated from 14 studies, the stress-somatic illness effect from 12 studies, and the psychological-somatic illness effect from 21 studies. While it would have been more appropriate to include only studies with predictive designs in this analysis, doing so would have greatly reduced the number of studies contributing inter-predictor associations (e.g., only 2 predictive studies provided stress-somatic illness correlations, while no predictive studies reported stress-psychological illness associations).

The compiled correlation matrix of true or corrected effect sizes (see Table 5), using variable reliabilities along its diagonal and a harmonic mean sample size ($N =$ 478.74), was analyzed using EQS (version 6). Although some statisticians recommend the input of a covariance rather than a correlation matrix in SEM, the former requires information on the standard deviation and mean for each variable as well. This is a difficult task especially when varied measures are used to assess a construct. For this reason, previous meta-analyses have tended to input the correlation matrix (Shadish, 1996). Despite considerable unexplained variation for each effect reported in Table 5, which suggests the presence of moderators or artifactual variance, the following analyses test basic processes underlying the stress-absence association. The following analyses focus on estimating path coefficients between observed variables. Although it would have been ideal to test the measurement model, this analysis required the compilation of additional information (e.g., intercorrelations between conceptually similar measures such as anxiety and frustration). Based on the rationale that the use of corrected correlations takes into account measurement error, previous meta-analyses testing theoretical models have focused on estimating path coefficients among observed variables (e.g., Podsakoff, Mackenzie & Bommer, 1996).

The theoretical model proposed in Hypothesis 4 was tested using the maximum likelihood method of estimation. The obtained statistics ($\chi^2 = 28.94$, df = 3, NFI = .95, NNFI = .91, CFI = .96, RMR = .04) for the theoretical model suggest that it fits the data well. The fit indices are high with low standardized residuals, and the path coefficients (see Figure 2a) are all significant. When compared against several other models, the proposed theoretical model remained superior. For example, model 1 (see Figure 2b)

which tested the direct effects of stress, psychological illness and somatic illness on absenteeism (χ^2 = 211.88, df = 2, NFI = .65, NNFI = -.05, CFI = .65, RMR = .20), and model 2 (see Figure 2c) examining psychological and somatic illness as simultaneous rather than sequential mediators of the stress-absence association (χ^2 = 196.72, df = 2, NFI = .68, NNFI = .03, CFI = .68, RMR = .11) yielded larger chi-square values and lower fit indices. (Note that correlating all the predictor variables in model 2 would have resulted in a just- identified model, which cannot be statistically compared because it has no degrees of freedom). Because psychological illness and somatic illness are highly related, the mediating sequence of these illnesses was reversed in model 3 (not shown), to determine whether the hypothesized progression from psychological illness-to-somatic illness really mattered. The comparison model testing this reversed sequence of mediators (χ^2 = 69.59, df = 3, NFI = .86, NNFI = 78, CFI = .89, RMR = .06) also had lower fit indices compared to the theoretical model, suggesting that the latter is superior. Therefore, Hypothesis 4 is supported.

6.3. Type of Absenteeism

Hypothesis 5 predicted no difference in the magnitude of effects for frequency and time lost measures of absenteeism. This hypothesis was confirmed by non-significant Z-tests comparing overall frequency and time lost effects for each variable in Table 1 [for stress (r_{frq} = .11, k = 14; r_{tlost} = .09, k = 40, Z = .456, p > .05), for psychological illness (r_{frq} = .14, k = 37; r_{tlost} = .12, k = 86, Z = 1.08, p > .05), for somatic illness (r_{frq} = .15, k = 24, r_{tlost} = .14, k = 40; Z = 0.18, p > .05)]. Even when comparisons included only measures of absenteeism aimed at capturing sickness absence or stress-related absence, the same non-significant results were obtained for stress (r_{frq} = .06, k = 2;

$r_{\text{tlost}} = .12$, $k = 26$; $Z = 1.45$, $p > .05$), psychological illness ($r_{\text{frq}} = .16$, $k = 21$; $r_{\text{tlost}} = .12$, $k = 56$; $Z = 1.64$, $p > .05$), and somatic illness ($r_{\text{frq}} = .14$, $k = 15$; $r_{\text{tlost}} = .12$, $k = 28$; $Z = 0.73$, $p > .05$). Given the theorized mediating role of illness, it is more appropriate to test this hypothesis in predictive or cross-sectional studies. However, this was not possible because some effects (e.g., stress and somatic illness) were based on one or two samples only.

To rule out the possibility that high intercorrelations (typically reported in the literature) between frequency and time lost absence measures might have contributed to the lack of difference between these effects, a sub-set of effects from studies reporting low to moderate correlations between frequency and time lost measures ($.18 \leq r \leq .45$) were compared. This comparison was possible only for psychological illness, for which a sufficient number of effects were available. Findings from this analysis confirm that frequency and time lost measures are not significantly different from each other ($k = 8$, $N = 4098$, $r_{\text{frq}} = .11$, $r_{\text{tlost}} = .14$, $Z = 1.16$, $p > .05$). Taken together, these results provide little support for the voluntary-involuntary distinction between frequency and time lost measures of absenteeism.

6.4. Restorative Model of Absence

Hypothesis 6 predicted little support for the restorative model of absenteeism. Prior absence was hypothesized to increase rather than decrease subsequent stress. Table 3b reports the results for postdictive studies, which are broken down not only by type of absence but also by the approach used to measure absence. This was done to rule out the possible operation of common method variance (theorized as a possible moderator) due to the retrospective nature of this design. Examining the confidence intervals for the

stress effects reported in the upper portion of Table 3b, it appears that regardless of the approach used to assess absenteeism (i.e., self-report or records), the stress effect for frequency measures is not significantly different from zero (r_{srep} = .02, k = 5, r_{rec} = .03, k = 4). For time lost measures, the effect based on self-reports is significantly positive (r_{obs} = .12, r_{corr} = .18, k = 20), while that for records-based absence is non-significant (r_{obs} = - .00, r_{corr} = .00, k =7). Assuming that common method variance is largely responsible for the obtained positive self-report time lost effect, there is little support for the influence of prior absence on subsequent stress. With respect to prior absence and subsequent psychological and somatic illness, all effects are significantly positive. For psychological illness, the effects for frequency absence (r_{srep} = .25, k = 5, r_{rec} = .11, k = 14) and time lost measures (r_{srep} = .13, k = 37, r_{rec} = .11, k = 20) remain significantly positive across both measurement approaches. The same is evident for somatic illness (for frequency absence: r_{srep} = .19, k = 4, r_{rec} = .21, k = 7; for time lost absence: r_{srep} = .17, k = 17, r_{rec} = .10, k = 7).

Taken together, these findings provide some support for Hypothesis 6, and work against the restorative model of absenteeism. Absence is likely to maintain subsequent stress levels, but increase subsequent illness levels. The coping literature suggests that the beneficial effects of respite from work tend to be short-lived. In other words, absence might have the potential to maintain or reduce subsequent illness levels when shorter time periods between measurements are used. To test this suggestion, illness effects from postdictive studies in which the time interval between absence and illness measurement ranged from one to three months were examined separately. To rule out the influence of common method variance and the influence of stress management programs, only

records-based measures from studies without any reported interventions were included.

Since the overall effects for psychological and somatic illness in postdictive studies were

no different from each other, they were combined in this analysis. The postdictive

absence-illness effect based on studies with shorter measurement intervals ($r_{obs} = .07$, r_{corr}

$= .11$, $k = 6$, CI = -.04 to .19) is non-significant while that aggregated over all postdictive

studies is positive ($r_{obs} = .15$, $r_{corr} = .22$, $k = 117$, CI = .14 to .16). This finding supports

the view that absenteeism might have the potential to prevent further damage to well-

being, albeit for a short period of time. According to respite research, these periods may

range from a week to three weeks. As there were no postdictive studies with such short

absence aggregation periods, this suggestion could not be explicitly tested.

6.5. Moderating Influences on the Stress/Illness-Absence Association

As evident in Tables 1 and 3, considerable variance remains unexplained even

after accounting for type of absence, research design, and type of absence measure,

suggesting the presence of additional moderators or uncorrected artifactual variance.

Before proceeding with tests for the hypothesized moderators, two issues deserve

discussion. First, Hunter and Schmidt (1990a, 2004) advise that it is more appropriate to

conduct a hierarchical breakdown of moderator analyses, where the effects of moderators

of lesser importance are examined sequentially within subsets of more important ones.

However, they acknowledge that this type of analysis requires a large sample of effects,

with a sufficient number representing each moderator variable. Most meta-analysts tend

to conduct partially hierarchical moderator analyses. The results discussed earlier are

somewhat presented in this manner, especially when there is theoretical reason for doing

so. For example, following presentation of the overall effects for each predictor in Table

1, effects are then broken down by research design, followed by type of absence in Table

3. For postdictive studies, effects were also broken down by method of absence

measurement.

The second issue concerns the interpretation of moderator analyses. Hunter and

Schmidt suggest that clear interpretations are possible only when moderator variables are

unrelated to each other. In addition, Russell and Gilliland (1995) discuss how the

influence of moderating processes can be misinterpreted as moderator effects. In other

words, convenience sampling can invariably result in the selection of certain moderating

characteristics. For example, in choosing to study absenteeism in blue-collar workers,

one might end up using records-based measures of absence, because absence-recording

systems are usually maintained for such employee groups. In addition, blue-collar

samples might be larger than higher white-collar samples within a single organization

because of the typically larger proportion of front-line employees in comparison to

managerial positions. In such situations, moderator variables can be sources of

nonrandom sample selection error and tend to be confounded with other variables

(Russell & Gilliland, 1995). The operation of moderating processes is typically indicated

by significant intercorrelations between sample size and a moderator variable or between

sample size and effect size. Russell and Gilliland argue that under such circumstances,

moderator analyses are un-interpretable. However, for continuous moderator variables, it

might be possible to examine their effects while statistically controlling for confounding

influences. While most of the hypothesized moderator variables were continuous,

categorical moderators were coded in a meaningful way to aid interpretation of the

following analyses.

An intercorrelation matrix of all moderators, effect size, sample size, and other study characteristics was examined (Table 7). As with the analyses reported earlier, the following results include only effects for stress, psychological illness, and somatic illness. In other words, effects for acute stress/illness and psychosomatic illnesses were removed from the data set. As observed in Table 7, there is evidence for the operation of non-random sample selection error as the size of a study's sample is significantly related to absence aggregation period and occupational status, while several hypothesized moderator variables are correlated with certain study characteristics in somewhat expected directions. For example, cross-sectional studies tended to use self-report absence measures, while health care samples were more likely to comprise females. Therefore, to examine the unique influence of a particular moderator variable, it was necessary to control for correlated confounds. Whenever a test for a moderator was based on a sub-sample of effects (e.g., the influence of personality was restricted to stress effects only), confounds were identified by examining the intercorrelation matrix based on the sub-sample of effects. Whenever the mean age of the sample was significantly correlated with a moderator, two separate regression analyses were conducted (with and without age as a control). This was necessary because of a large number of missing cases in the data (i.e., many studies did not report the average age of their sample), which lowered the degrees of freedom and consequently the power of the regression analysis to detect an effect.

Role of attribution. Hypothesis 7 predicted that attribution processes would inflate the stress/illness- absence association. This influence would be more likely to manifest in self-report measures, especially those asking respondents about absence due

to stress or illness. To test for the role of attribution, effects from postdictive and cross-sectional studies were grouped by type of absence measure (i.e., self-report versus records-absence), and then compared. As the overall effects obtained for psychological and somatic illness were no different from each other, these two variables were combined in this analysis. The results of these analyses are reported in Table 6.

For stress, the average observed effect for self-report measures of absenteeism is significantly greater than that obtained for records-based absence ($r_{srep} = .09$, $k = 33$, $r_{rec} = .03$, $k = 16$, $Z = 1.73$, $p < .05$ one-tailed). The extent of this attributional influence is further depicted by the confidence interval for records-based effects, which includes a zero value. The obtained stress-absence effect is positive when self-report measures of stress and absence are used, but does not differ significantly from zero when different measures are used to assess stress (i.e., self-report) and absence (i.e., records). These findings suggest that any obtained positive stress-absence connection in retrospective studies is a likely product of common method variance. The illness-absence association is also dependent on the type of method used to measure absence. As seen in Table 6, self-report measures of absence yield significantly stronger effects as compared to records-based absence ($r_{srep} = .16$, $k = 84$, $r_{rec} = .11$, $k = 74$, $Z = 3.09$, $p < .01$, one-tailed).

Considerable noted variation in the effects reported in Table 6 (i.e., wide credibility intervals and low percent of variance explained by artifacts) led to a comparison of each of these effects by reason for absence. This analysis determined whether asking respondents to report specifically on stress-related or sickness absence yielded greater differences between the illness effects for self-report and records-based absence. The pattern of results reported in Table 6 remained unchanged. Effects based

on self-report measures of absence, regardless of the specified reason for absence ($r_{illness}$

= .15, k = 60, Z =2.60, p <.01, one-tailed; $r_{not\ specified}$ = .21, k = 20, Z = 2.75, p <.01, one-

tailed), were both stronger than the average for all retrospective records-based absence

effects (r = .11, k = 74).

Given significant intercorrelations of this moderator variable (i.e., absence

measure) with a number of other variables (see Table 7), a regression analysis was

conducted to determine whether the above interpretation holds after controlling for

confounds. An examination of the correlation matrix of effects for retrospective studies

only revealed additional confounds (absence aggregation period and health care sample),

while the design of a study was no longer significant. Therefore, six interrelated

variables (excluding age) were simultaneously entered as controls on step 1, followed by

the main variable of interest on step 2. As seen in Table 8, a significant negative

association between absence measure (coded 0 = self-report, 1 = records) and

stress/illness-absence effect is observed (β = -.30, p < .01). This finding is consistent

with those reported above; larger effects are more likely when self-report measures are

used. The measure of absence accounts for seven percent of the variance after

controlling for confounds. When the analysis was repeated by including age as a control,

the effect remained essentially the same but was not significant (β = -.33, p = .05),

reflecting the lowered power of this test to detect an effect. Taken together, there is

support for the moderating role of attributional processes.

Dispositional influences. Hypothesis 8a proposed that dispositional influences

on the stress-absence effect would be evident if the effect increased with increasing

absence aggregation periods. This test for the influence of disposition was based on

inferences about the detection of stable, long-term sources of variance (e.g., Harrison & Martocchio, 1998; Epstein & O'Brien, 1985). If disposition is primarily responsible for the stress-absence effect then a significant positive association should be evident between this effect and longer absence aggregation periods. On the other hand, a negative association would indicate that the stress-absence effect is more likely a result of less stable, mid-term sources of variance (i.e., workplace stressors). Consequently, Hypothesis 8b predicted that both sources of variance (disposition and workplace stressors) could have combined influences on this association, cancelling out potential positive and negative directions of either influence, and yielding a near-zero association instead. This hypothesis was proposed for stress effects only, because any obtained positive association between absence aggregation and illness-absence effects could not rule out the long-term chronic nature of some illnesses. For the sake of comparison, however, an analysis of illness effects was also conducted.

An intercorrelation matrix for a sub-sample of stress effects revealed no significant correlations of other variables with absence aggregation periods. Therefore, no controls were included in this analysis. Given the wide range in absence aggregation periods (1 month to 12 months) for this sub-sample of effects, a test for curvilinear effects (i.e., inverted-U shaped relationship) was conducted. As reported in the upper portion of Table 9, a hierarchical regression analysis testing the linear and curvilinear influences of absence aggregation periods failed to provide support for Hypothesis 8a. A non-significant effect for absence aggregation period ($\beta = .05$, $p > .05$) suggests that the effect is not significantly different than zero, providing some support for Hypothesis 8b instead.

The above analysis was repeated for illness effects, and an examination of the intercorrelations between moderators revealed several control variables, including age. As seen in Table 9, the linear influence of absence aggregation periods on illness-absence effects (without controlling for age) is positive but non-significant ($\beta = .18$, $p > .05$). Instead, there is evidence for a curvilinear relationship (3.6 percent additional variance is explained by the quadratic equation), suggesting that the illness-absence effect is strongest when absence is aggregated over approximately 6-12 months. Note that the range of absence aggregation periods was wider for this sub-sample (2 weeks to 36 months). When the analysis was repeated by including age as a control, the curvilinear influence remained strong, though not significant ($\beta = .73$, $p = .08$; $\beta_{squared\ term} = -.80$, $p = .08$, $\Delta R^2 = .048$). Taken together, the results suggest that while stable sources might contribute to the stress/illness-absence connection, the influence of mid-term sources (i.e., work stressors) cannot be ruled out.

Role of gender. To test the prediction that women are more likely to respond to stress or illness by going absence, the percentage of women in a study's sample was regressed on stress/illness-absence effects, controlling for variables related to gender (see Table 10). Although no significant effect for gender was noted ($\beta = -.16$, $p > .05$), the direction of this effect is opposite to that predicted. Samples containing a larger proportion of men (i.e., lower percent of women) were more likely to yield larger effects than those containing more women. A sub-set comparison of effects obtained from samples with less than 30 percent women (i.e., mostly males) and those with more that 70 percent women, however, suggested that the stress/illness-absence effect for predominantly male- versus female- samples was not significantly different from each

other (for illness, r_{male} = .134, k = 34, r_{female} = .119, k = 55, Z = .68, p >.05; for stress, r_{male} = .110, k = 17, r_{female} = .064, k = 14, Z = 1.23, p >.05). In either case, Hypotheses 9a and 9b were not supported.

Role of occupational status. Hypothesis 10 proposed that blue-collar samples would yield stronger stress/illness-absence effects than white-collar (i.e., managers) samples. As a considerable number of samples comprised both blue- and white- collar employees, codes such as "mostly lower white collar" or "mostly higher white collar" were used to identify samples with approximately 70 percent or more of the respective occupational categories (whenever such percentages were reported). To form a meaningful continuous variable for occupational status, numerical codes were assigned in this manner (mostly blue- collar = .5, blue-collar = 1, mostly lower-white = 1.5, all lower-white = 2, mostly higher-white = 2.5, and all higher-white = 3). This coding scheme was used to retain as many data points as possible, and to facilitate easier coding as codes 1, 2, and 3 already existed in the data set. Studies that did not specify the occupational breakdown of their sample or those that contained an equal mix of occupational categories were left as missing cases. The results testing for Hypothesis 10 (without controlling for age) are presented in Table 11. Although the direction of the obtained effect is as hypothesized, its magnitude is small and non-significant (β = -.06, p > .05). When age is included as a control, the size of the effect remains non-significant but its direction changes (β = .02, p = .88). There is no change in the percent of unique variance explained. Therefore, Hypothesis 10 is not supported.

Although attempts were made to code other occupational variables (e.g., part-time status, shift work), a majority of the studies failed to report such information, precluding

robust comparisons based on these variables. Effects were, however, compared across

health care (e.g., nurses) and non health care samples. While a significant difference was

observed for somatic illness, there were only two such effects representing health care

samples (r_{health} = .205, k = 2; $r_{nonhealth}$ = .140, k = 52, Z = 3.99, p < .01). The effects for

stress (r_{health} = .146, k = 8; $r_{nonhealth}$ = .087, k = 45; Z = 1.15, p > .05) and psychological

illness (r_{health} = .141, k = 18; $r_{nonhealth}$ = .133, k = 90; Z = 0.35, p > .05) did not differ

significantly across these two occupational settings. Note that point biserial correlations,

because they were found to be larger for psychological illness, were not included in this

particular analysis.

Effects of macro social context. Hypothesis 11 proposed a positive association

between the stress/illness-absence effect and the year of publication. It was argued that

social information processing effects due to increasing media attention to stress-related

issues over the years was likely to have correspondingly inflated the basic stress/illness

absence effect. As reported in Table 12, the effect for publication year (without

controlling for age) is positive and significant (β = .12, p = .03 one-tailed, ΔR^2 = .013).

Controlling for age yields essentially the same result (β = .15, ΔR^2 = .019). Therefore,

there is support for the influence of the larger social context on the stress/illness-absence

effect.

6.6. Additional Analyses

Cross-cultural differences. Whenever reported, the country of each sample was

coded, to determine evidence for cross-cultural differences in effects. The full sample of

effects was grouped into five regional categories: Asia, Australia/New Zealand, Europe,

Middle East, and North America. As only a handful of effects were obtained from

Middle Eastern samples, Table 13 reports the effects for the remaining four regions only. While the bulk of the effects are from North American samples, no notable differences in average effect sizes across these regions are evident. The general pattern of progressive increase in effects for stress, psychological illness, and somatic illness tends to be similar across the continents, suggesting that the basic theoretical processes underlying the stress-absence association is likely to hold across cultures.

Effects from dichotomous data. As described in the methodology section, effects based on dichotomous measures (i.e., fourfold tables) were not included in the above-reported analyses. As an analysis of odds ratios (OR) is more appropriate for such data (Haddock et al., 1998), a separate meta-analysis of odds ratios was conducted. As one study contributed two independent effects, a total of 17 effects from 16 studies published in epidemiological/medical journals were available for this analysis. These included eight effects for psychological illness, eight for somatic illness, and one for psychosomatic illness. The psychological illness variables included measures of mental illhealth, anxiety, and depression, while the somatic illness variables included sleep difficulties, diabetes, haemophilia, migraine, hypertension, and colds/flu. The dichotomous categories varied across studies. For example, while some distinguished between major depression and mild depression, others distinguished between people with migraines and no migraines. For the absence variable, some studies examined those with and without recent absence, while others categorized individuals according to some amount of absence (e.g., 0-1 spells versus more than 2 spells). The following effects can, therefore, be interpreted as the probability of being ill/having a more severe form of illness and being absent/having higher absence in comparison to those without an illness

or less severe forms of an illness. Because of the small number of effects, only overall effects are reported in Table 14.

In analyzing the compiled odds ratios, the Mantel-Haenszel summary estimate was used because it is valid under both conditions (small number of effects with large sample sizes or vice versa) (Shadish & Haddock, 1994). As reported in the Table 14, the odds ratios are positive and non-zero for psychological illness ($OR = 2.90$, $k = 8$) and somatic illness ($OR = 1.72$, $k = 8$), suggesting that those in the illness/severe illness group are more likely to be absent/have higher absence than those in the no illness/less severe illness groups. The non-zero confidence intervals provide a test for the significance of each effect. Employees with some form of psychological illness are 2.9 times more likely than those without such illness or less severe forms of the illness to be absent or have higher absences. In comparison, the odds of having somatic illness and being absent are slightly lower. As there is considerable variation within effects for psychological illness ($Q = 55.48$, $p < .001$), the reported OR is based on a random effects model. On the other hand, the fixed effects OR is reported for somatic illness, because no significant variation is evident within this sub-sample of effects ($Q = 11.55$, $p = .11$).

To compare these effects with those obtained from the main meta-analysis of correlations, the log odds ratio was converted to d by dividing it by 1.81 (Haddock et al., 1998; Hasselblad & Hedges, 1995). The obtained estimates ($d = .588$ for psychological illness; $d = .299$ for somatic illness) were then converted to point biserial correlations using the same Hunter and Schmidt procedures as before. As this calculation requires the proportion of individuals in each category of the dichotomized variable, the average proportion in each category of the independent variable (i.e., illness) across the sample of

OR studies ($p = .311$) was used. While the resulting average effect for somatic illness ($r_{pb} = .139$) is comparable to the overall somatic effect reported in Table 1, the psychological illness ($r_{pb} = .263$) is somewhat larger than its counterpart in Table 1. However, this larger effect simply mirrors the results of the main meta-analysis, where the average psychological illness effect based on point biserial associations ($r_{pb} = 0.23$) tended to be larger than those based on the full sample of effects. Taken together, these findings consolidate the main findings of this synthesis, providing some support for the generalizability of the illness-absence effect across management and epidemiology disciplines.

CHAPTER SEVEN

7. Discussion

This empirical synthesis sought not only to estimate the magnitude of the stress-absence effect, but also to determine causality or the predictive role of stress. Being theory-focused, 11 hypotheses were offered and tested around key questions pertaining to the theory of causation. Seven hypotheses were confirmed, while two received partial support. The resulting findings are discussed in relation to each question posed by this research.

7.1. Does Stress cause Absenteeism?

In answering this question, one would typically interpret findings from predictive studies in consideration of answers to the other two questions posed by the theory of causation (i.e., reverse causation and moderators) (Clegg, 1983). However, the small number of predictive effects ($k = 6$, $N = 1214$) precludes confident validity generalization statements about stress as a cause of absenteeism. In estimating the true stress-absence effect, however, one might consider the effect aggregated over all studies ($r_{obs} = .09$, $r_{corr} = .15$, $k = 56$, $N = 18,630$) as representing a lower bound estimate of the population effect. The effect aggregated over predictive studies, on the other hand, can be seen as an upper bound or best-case estimate ($r_{obs} = .26$, $r_{corr} = .40$, $k = 6$, $N = 1214$), because its is somewhat methodologically superior to the former. The predictive effect is based on records-based absence measures, which rules out possible inflation due to common method bias. In addition, findings from postdictive studies provide little evidence for

reverse causation, increasing the likely causal influence of stress on absenteeism.

Second-order sampling error (due to the small k), however, cannot be ruled out, but this

form of error is likely to influence the *variation* in effects rather than the obtained

magnitude (Hunter & Schmidt, 1990a).

The 95 percent confidence intervals for both effects (overall and predictive) are

positive, and provide a test for the escape model of absenteeism. Employees

experiencing stress at work are likely to escape or withdraw from their workplace by

going absent (Hill & Trist, 1955). The magnitude of the stress effect aggregated over all

studies is comparable to related meta-analytic findings examining other correlates

(particularly job satisfaction) within the escape or withdrawal model of absenteeism. For

example, the obtained corrected associations between stress and absenteeism in the

present meta-analysis ($r_{overall} = .145$, $k = 56$; $r_{frq} = .165$, $k = 14$; $r_{tlost} = .136$, $k = 40$) can

be said to be within the range of effects reported for overall job satisfaction and

absenteeism ($r_{frq} = -.134$, $k = 33$; $r_{tlost} = -.096$, $k = 22$) in Hackett and Guion's (1985)

meta-analysis and in Hackett's (1989) examination ($r_{frq} = -.15$, $k = 17$; $r_{tlost} = -.23$, $k = 8$).

In both these meta-analyses, effects for the facet of work satisfaction (Hackett & Guion,

1985: $r_{frq} = -.162$, $k = 40$; $r_{tlost} = -.068$, $k = 42$; Hackett, 1989: $r_{frq} = -.210$, $k = 28$; $r_{tlost} = -$

$.140$, $k = 28$) tended to be somewhat larger than the effects for some facets like pay,

promotion, coworkers, and supervision. Perhaps, negative feelings stemming from one's

actual work activities (i.e., the job itself) are more likely to prompt temporary withdrawal

from the work place than those related to contextual elements such as pay or coworkers.

Of course, an important question concerns the distinctiveness between measures of work

stress and job satisfaction, and their unique influence on absenteeism.

To provide some insight into this question, I compiled correlations between overall job satisfaction, stress, and absenteeism from studies included in the main meta-analysis (whenever such information was provided). All studies reporting such information, regardless of research design, were included in this additional meta-analysis of job satisfaction correlates. In computing the corrected effects between job satisfaction and these variables, I used the job satisfaction reliability estimate ($r_{xx} = .74$) reported in Judge, Thoresen, Bono, and Patton's (2001) meta-analysis. The obtained corrected association between measures of job satisfaction and stress ($r_{obs} = 0.343$, $r_{corr} = .448$, $k = 19$, $N = 7541$) is moderate, suggesting some discriminant validity. Calculating the partial associations between absenteeism and each of these two variables revealed that the corrected job satisfaction-absence effect (controlling for stress) is stronger ($r_{corr} = .128$) than the effect for stress and absenteeism ($r_{corr} = .064$) when job satisfaction is controlled. This finding suggests that job satisfaction may be more directly linked to absenteeism than stress is, which is feasible given support for the transactional process model of stress which dictates that individuals stay and cope with stress before escaping the workplace.

While the findings do suggest that stressed employees are likely to escape the workplace in the form of absenteeism, the effect is not as large as some sources have previously reported (e.g., 60 percent in Cartwright, 2000). Constructing confidence intervals around the corrected correlation for the lower bound estimate (i.e., effect aggregated over all studies) and the upper bound estimate (i.e., effect aggregated over predictive studies) provides two possible ranges likely to contain the unattenuated population estimate. In the best-case scenario, the true stress-absence estimate is likely to vary between .25 and .55, while lower-bound estimates are likely to range between .09

and .19. Even when upper boundary values are considered, stress accounts for only between six and 29 percent of the variance in absenteeism. Ranges for the population estimate of the psychological illness-absence effect ($.11 \leq r_{corr} \leq .20$) and somatic illness-absence effect in predictive studies ($.18 \leq r_{corr} \leq .32$) also suggest that the maximum variance explained by illness is only about 10 percent.

Although these percentages are much lower than those suggested in the popular press, they have several implications for organizations. Based on financial estimates reported by a U.S. tax and business law firm, unscheduled absenteeism can cost small companies up to $60,000 annually, while larger companies can lose up to $ 3.9 million (CCH Inc., 2002). Using the higher bound stress-absence effect obtained in this meta-analysis, it is estimated that small companies can lose up to $17,400 (i.e., $60,000 x 0.29) annually, while it can cost larger companies up to $1.13 million. These figures are not trivial, and are likely to be even higher when indirect costs such as health insurance claims, legal claims, lost productivity, and overtime wages are taken into consideration. Therefore, organizations have reason to be concerned about stress in the workplace. However, the findings also suggest that using stress-intervention programs to solve absenteeism problems may not be an effective solution. Indeed, Unckless et al.'s (1998) meta-analysis on absence control programs revealed that wellness interventions were the least effective in reducing absenteeism. Managers are, therefore, encouraged to determine the exact cause of absenteeism in their organization prior to considering a particular strategy for dealing with the issue.

Conservatively speaking, the present findings suggest that almost 70 percent of absenteeism remains unexplained by stress or illness. Some of this unaccounted variance

might be due to uncorrected artifacts, other identifiable factors, or unpredictable random ones. Residual variance is not unexpected in process models, for which antecedents are considered to be necessary, but not sufficient conditions for the outcome (Fichman, 1999; Mohr, 1982). In other words, whether or not stress causes absenteeism depends on a sequence of events that follow (psychological and somatic illness), each of which is likely to be influenced by random events (e.g., approaching deadlines, weather conditions, flu virus), hence the term unpredictable factors. As to what some of the other identifiable factors might be, human resources executives surveyed in the U.S. cited personal needs and family issues as reasons for unscheduled absenteeism in their organizations (CCH Inc., 2002). If these non-work factors are indeed predictive of absenteeism, employers might consider programs that allow employees more flexibility over the use of their work time. In fact, the effects for flextime schedules on absenteeism reported in Baltes et al.'s (1999) meta-analysis are rather strong (r_{obs} = .42, reverse-coded), suggesting that non-work factors requiring flexibility in work arrangements might have stronger influences on absenteeism. Although it is possible to argue that flextime reduces absenteeism, because it offers an opportunity to reduce negative reactions towards one's work, the much lower average effect for flextime schedules on job satisfaction (r_{obs} = .07), a correlate of stress and its reactions, does not strongly support this argument.

With respect to implications for future research, a lot more predictive studies examining stress (as conceptualized in this study) are needed. Consistent with Ganster and Schaubroeck's (1991) assessment, cross-sectional studies using self-report measures are least likely to provide information that will advance knowledge in this area. The

influence of attribution bias on the stress/illness-absence effect is strong enough to merit

attention, and primary researchers are encouraged to devise approaches (e.g., multiple or

objective sources, time delays between measures) for reducing such bias when faced with

using this type of research design. Researchers must also clearly specify the construct of

interest to them (i.e., stress versus illness), because there is evidence for their

distinctiveness. For example, the stress effect was significantly smaller than the illness

effect when aggregated over all studies, but was significantly larger than the illness effect

when only predictive studies were examined. Primary research designed explicitly to

evaluate discriminant validity among these measures might also be valuable, providing

more insight into the interconnections among these variables. With respect to

absenteeism, the use of both frequency and time lost measures in predictive studies might

more solidly determine the type of absence that employees are most likely to take when

stressed. Given the possible influence of individual and contextual factors (discussed

later), controlling for these factors will enable more accurate conclusions about causality

in individual studies.

7.1.1. *Does stress cause absence through illness?* As for the basic theoretical

processes thought to underlie the stress-absence connection, the results are consistent

with predictions. Illness does mediate the relationship between stress and absenteeism.

As theorized by Lazarus and Folkman's (1984) transactional model and Rosse and

Miller's (1984) adaptation process, employees experiencing stress tend to stay at work

and "fight" the stress before fleeing the workplace. However, this general process is

likely to be moderated by person or situation factors, because the effects used to test this

theory were based on heterogeneous samples. Both theoretical models also depict this

complexity, in which relationships between variables are constrained or enhanced by moderating influences. Nevertheless, there is support for the basic stress-illness-absence progression. There is also support for the view that absenteeism might be used a last resort coping mechanism. Consistent with Aspinwall and Taylor's (1997) argument that individuals exert greater coping efforts in response to more severe problems, there is strong support for the stress-psychological illness-somatic illness-absence progression. In other words, ineffective coping by stressed employees who stay at work and attempt to cope with stress is likely to be reflected in further damage to their psychological state, which in turn weakens their physical well-being, making the temporary withdrawal from work an inevitable coping response.

One limitation of the above test for the medical mediation model is that it included effects from all designs, a large proportion of which were postdictive and cross-sectional in nature. One might therefore question whether a stronger connection between somatic illness and absence is largely due to sick role legitimacy or people's attributions of their absence to physical illness (Harvey & Nicholson 1999; Levine & Kozloff, 1978; Parsons, 1970). However, the overall somatic illness-absence effect in cross-sectional (r_{obs} = .12) and postdictive studies (r_{obs} = .17) is not significantly different from that in predictive designs (r_{obs} = .17), suggesting that the obtained mediation pattern is not likely due to attribution influences alone. These estimates are also comparable to the overall effect obtained in Martocchio et al.'s (2000) meta-analytic examination of another physical ailment (low back pain) and absenteeism (r = .18).

Another issue worth examining is whether the above pattern of results is due in part to the moderately high intercorrelations among stress, psychological illness, and

somatic illness. Although stress is related to both forms of illness, its association with these variables tends to be moderate ($r_{corr} = .42$ and $r_{corr} = .46$). As mentioned earlier, the stress-absence effect is significantly different from the illness-absence effect, providing some evidence for the distinction between stress and illness. With respect to the distinction between psychological illness and somatic illness, the stronger association between the two ($r_{corr} = .58$) suggests some overlapping variance. However, when the partial correlation between each variable and absenteeism, controlling for the other, is calculated there is still support for the theoretical model. The partial corrected somatic illness-absence effect ($r = .127$) appears larger than the partial corrected psychological illness-absence effect ($r = .091$). Both effects, however, are smaller than the overall corrected effects for psychological illness ($r = .20$) and somatic illness ($r = .22$), suggesting some influence of psychological states on self-reports of physical illness and vice-versa. Therefore, one cannot rule out the possibility that self-reports of physical illness are amplified by psychological states, and might not have any bearing on underlying physiological pathology. However, this is a problematic issue even in the medical profession, in which diagnosticians must carefully weigh patient symptom self-reports against objective signs of an illness (Cohen & Williamson, 1991; Wiebe & Smith, 1997). As discussed earlier, the above results best represent the association between absenteeism and perceived illness symptoms rather than verifiable illness.

7.1.2. Type of absence. Evidence for medical mediation presents a good backdrop for assessing the underlying basis for frequency and time lost measures of absenteeism. Despite claims that time lost measures capture involuntary factors such as illness, while frequency measures reflect voluntary decision processes, the voluntary-

involuntary basis for the distinction was not supported. Time lost measures were not more strongly related to measures of illness or stress than frequency measures, and this remained true even when these measures were specifically directed at assessing sickness or stress-related absenteeism. Accounting for high correlations between frequency and time lost absence did not change this finding. Several explanations are plausible. The first explanation is related to the predicted view that voluntary and involuntary influences are likely to be reflected in both types of absence measures. For example, a sick employee (involuntary factor) might decide (voluntary factor) that a single absence day is sufficient for recovery. Or, employees feeling somewhat stressed might execute some discretion in choosing the day to be absent (perhaps, one that precedes a weekend).

Second, factors pertaining to Nicholson's (1977) justifiability explanation may also underlie the two types of absence measures, blurring their distinction. For example, epidemiological researchers argue that certified absences, which are greater than 3 days in duration and which require medical justification, are more likely to be predicted by illness, while those not requiring medical certification which are typically less than 3 days in duration are likely to reflect voluntary or decision influences. Because frequency and time lost measures could both include absence days of longer duration, distinguishing them on underlying illness might be difficult. In the present study, it is assumed that most studies assessed short-duration absences (i.e., one to three-day absences), although two studies asked respondents to report on absences that were up to 2 weeks and 20 days long, respectively.

A third explanation for the lack of a voluntary-involuntary distinction concerns the construct validity of self-report measures of psychological and somatic illness. As

alluded to earlier, self-report measures of illness are highly influenced by an individual's sensitivity to physical sensations, which influences one's reporting of symptoms and labeling of these as some disease (Cohen & Williamson, 1991). In the majority of studies included in this meta-analysis, the measures of illness symptoms were not verified by objective assessments of underlying pathology by a trained professional. Perhaps self-report illness measures, which are highly influenced by circumstantial factors and short-term cognitive or affective states, are not valid measures of illness theorized as an involuntary factor (i.e., reflecting underlying pathological damage). If this is true, then time lost absence measures (posited as reflecting underlying illness) should have had relatively poor associations with such measures, which was not the case. This reasoning actually reinforces the explanation advanced in the beginning of this section that both frequency and time lost measures are likely to represent factors within and beyond an employee's control.

The above discussion is based on a comparison of frequency and time lost effects for all studies combined. Ideally, one would compare these effects for predictive studies only, because of the theorized view that illness *causes* one to be absent. However, the low number of available effects for stress and somatic illness in such designs precluded such a comparison. More studies with predictive designs are clearly required. In addition, attempts to verify self-reports of illness might provide more insight into the processes underlying frequency and time lost absence. Probes into such underlying processes may also be accomplished through semi-qualitative approaches such as those used in Haccoun and Desgent's (1993) study, in which employees were directly asked about the reasons prompting their absence. Until the accumulation of further research of

this nature, the voluntary-involuntary distinction between frequency and time lost measures remains unresolved.

7.2. Does Absenteeism cause Stress?

Effects obtained in postdictive studies suggest that the influence of prior absence on subsequent stress is not different from zero, while that obtained for psychological or somatic illness is significantly positive. This is true even after accounting for attributional influences. When these findings are interpreted within the restorative model of absenteeism, the overall trend in the data provides little support for the view that respite from work lowers subsequent levels of stress or illness. Absenteeism appears to play more of a maintenance function by having little or no impact on an employee's subsequent stress levels, thereby preventing the further escalation of work stress. This finding is consistent with Hackett and Bycio's (1996) idiographic examination of absenteeism and psychological states in nurses over two-to-three day comparisons. However, absence in the face of psychological or somatic illness is less likely to be beneficial to an employee because it slightly increases these states upon return to work. But, findings from a sub-set of studies using shorter time intervals between measures suggest that although absence might have the potential to play a maintenance function, its beneficial effects are likely to be short lived.

The above pattern of results is consistent with the theorized explanations put forth earlier. In this research, stress is viewed as an early stage of subsequent damage to well-being. At this stage, taking a break from work might actually benefit employees, helping them recharge and feel better equipped at dealing with the source of their stress upon return to work. However, escaping the workplace when one is experiencing

psychological and somatic illness, both of which reflect a failure to adequately cope with the source of initial stress, is likely to exacerbate these states. In addition, the accrual of other stressors accompanying an employee's absence such as increased workload, coworker conflict or supervisor annoyance is likely to add to the burden of an already incapacitated employee. These explanations are consistent with related findings that prior absence decreases subsequent job satisfaction (Tharenou, 1993) and job performance (Bycio, 1992).

The above findings provide further insight into the predictive effects obtained in this examination. The stress-absence effect was found to be stronger than the psychological-illness and somatic-illness effect in predictive studies, suggesting that employees might be less inclined to be absent in response to illness, because they are aware that staying away might only make them feel worse upon returning to work. In such circumstances employees might benefit from focusing directly on resolving the source of their stress (i.e., use problem-focused coping strategies) instead of withdrawing from the workplace, a theorized emotion-focused coping response. Nevertheless, in the absence of comparisons between employees who decide to stay at work and those who remain absent in response to weakened states, this suggestion is tentative. Further insight into the restorative function of absenteeism is likely to be obtained from studies that integrate findings from respite research and use within-subjects approaches to examine absence as a coping behavior.

7.3. Does some Third Factor cause the Stress-Absence Relationship to Covary?

Even after controlling for confounds and sample selection error, there is evidence for individual and contextual moderating influences on the stress/illness-absence effect.

However, the largest amount of uniquely explained variance by a moderator tends to be small (seven percent), suggesting that the obtained stress/illness-absence effect is not likely to change substantially.

Individual influences. Of the hypothesized individual moderators (attribution, disposition, and gender), the influence of attribution processes is the strongest, because it explains more unique variance in comparison to the others. Although the use of self-report absence measures in retrospective studies was not problematic for illness variables, the large gap in the average effect for stress and self-report absence ($r = .12$) versus records-based absence ($r = .00$) deserves attention. It reinforces Ganster and Schaubroeck (1991) conclusion that future stress research using self-report measures is unlikely to improve our understanding of the phenomenon in question. Again, stress researchers examining using retrospective self-report measures must be mindful of the strong influence perceptual biases or common method variance can have on their findings.

Findings with respect to disposition, although non-significant, raise confidence in saying that workplace stressors are more or less responsible for withdrawal from work. While mid- and long-term sources (i.e., disposition) might jointly influence a person's absence due to stress, it appears that mid-term sources (i.e., work stressors) are largely responsible for prompting absence in an employee experiencing weakened psychological or somatic states. This finding actually reinforces the discussion on mediating processes, suggesting that the employment of problem-focussed approaches directed at the work stressor is likely to be more beneficial than staying away from the workplace. Finally, the prediction that women are more likely than men to escape the workplace when

stressed or ill was not supported. Although not significant, the direction of this effect was opposite to that predicted; the stress/illness-absence effect was stronger in samples containing a larger proportion of men. Several confounding influences are likely to have played a role in these findings. For example, gender correlated significantly with occupational status and health care work. Even though these variables were controlled for in this analysis, it is acknowledged that statistical controls are imperfect in removing all variance associated with these variables.

Samples with more women were more likely to comprise white-collar employees and health care providers (e.g., nurses), characteristics that have been linked to the phenomenon of sickness presenteeism. Described as a trend in which employees come in to work even when experiencing ill health, sickness presenteeism has been theorized to be more likely in employees responsible for providing care or assistance to other people, and less likely in employees who can be easily replaced when they are absent or delay work deadlines (Aronsson, Gustafsson & Dallner, 2000). Blumberg (1980) provides some insight into the replaceability of blue-collar workers. In blue-collar employment, the costs associated with an absent employee to an operation are quite great, and advance replacement arrangements are often in place to cover for the absent employee. Therefore, blue-collar workers or those in low occupational status groups are probably less likely to exhibit sickness presenteeism in comparison to white-collar employees, which might explain why gender (which was positively related to occupational status) varied inversely with stress/illness-absence effects. However, the above explanation is not supported by the data because the direction of the zero-order association between occupational status and effect size is positive, while that for the health care sample is near zero. Clearly,

gender as a potential moderator deserves further exploration. Primary research, carefully designed to control for some of the potential confounds discussed above, is likely to yield more insight into whether women are more likely than men to go absent when stressed or ill.

Contextual influences. Of the two contextual moderators (occupational status and macro social change), macro social change (operationalized as year of publication) was the only factor found to covary with the stress/illness absence effect. The non-significant influence of a sample's occupational status on the stress/illness absence-effect is again attributable to confounding influences, especially gender. Contrary to the proposition that samples of lower occupational status will have stronger stress/illness-absence effects, the zero-order correlation suggests a positive association between these two variables. Even an explanation suggesting that perhaps the greater proportion of women in the sample might have contributed to stronger positive effects is not supported because the zero-order correlation between gender and effect size is negative.

An alternate explanation may have to do with range restriction in values of the predictor variable. Occupational codes were assigned to distinguish between blue-collar, lower white-collar, and higher white-collar samples. However, the higher white-collar category was represented by only three percent of the full sample of effects. In addition, 40 percent of the effects were not included in this analysis because they represented samples of either mixed occupational categories or those that could not be identified. With only three percent of higher white-collar employees represented in the sample, the comparison appears to be between blue- collar and lower white-collar employees. This distribution provides a rather weak operationalization of occupational status, a variable

that was meant to capture differences in absence policies, norms, and attendance systems between blue-collar and professional/ managerial employee groups. Perhaps, minor differences across blue and lower white-collar employment groups in these systemic features might have rendered the influence of occupational status on stress/illness-absence effects non-significant. In other words, the absence cultures of blue-collar and lower white-collar employees are likely to be similar, prompting employees in either group to respond alike to workplace stress/illness.

With respect to the influence of macro social change, it was hypothesized that social information processing effects, resulting from increased media attention to stress-related issues over the years, might have correspondingly inflated the stress/illness-absence association. A significant positive effect for year of publication and effect size provides some support for this suggestion. However, this influence is small, and doesn't rule out the effective operation of stress and absence interventions that have been increasingly adopted by organizations over the years. In building Hypothesis 11, I suggested that the effectiveness of such programs could have lowered the levels of stress and absence in organizations, resulting in a restricted range of scores in both variables and causing reported stress/illness-absence associations to become lower over the years. A small set of post-intervention illness-absence effects obtained from studies that implemented some stress intervention was significantly smaller ($r = .03$, $k = 5$, $Z = 1.90$, $p < .05$) than those obtained in studies without such interventions ($r = .144$, $k = 116$). However, the removal of post-intervention effects from the regression analysis did not significantly strengthen the impact of publication year on effect size.

Alternate constraints on the stress/illness-absence effect over the years could also

include other macro changes such as regional employment and absenteeism rates. For example, increasing unemployment rates over the years could deter people from taking absence when stressed or ill, thus lowering the effect over the years. The obtained positive year-effect size association could also reflect tougher publication criteria in the face of increasing submissions. In other words, only studies reporting strong effects are selected for publication. However, this explanation is ruled out as the zero-order association between year and effect size reported in unpublished dissertations continues to remain positive ($r = .29$, $p = .06$).

In concluding this section on moderators, future researchers are reminded about the significant influence that study characteristics can have on the obtained associations. In testing for the moderating role of attribution, for example, approximately 30 percent of the variance in effects was explained by confounds alone (e.g., type of absence, absence aggregation period, occupational status of sample, post-intervention study). To enable confident conclusions about moderators, future primary research must consider ways to minimize the influence of other potential factors.

CHAPTER EIGHT

8. Conclusion

One of the criticisms surrounding an empirical synthesis or meta-analysis is its

limited capacity to contribute to theory development in a field, partly due to its focus on

the estimation of an effect's magnitude (Shadish, 1996) and a lack of pre-planned

objectives (Cooper, 2003). The present synthesis addresses both issues by employing a

theory-focused review in answering three key questions pertaining to the causal influence

of stress on absenteeism. In its exploration of predictors, mediating processes, reverse

causation, and moderators, this synthesis yields a comprehensive understanding of

absenteeism in relation to stress and its correlates. In addition, it provides an overall

assessment of the primary research arena by pointing out methodological and substantive

gaps requiring future empirical attention. For example, while the basic propositions of

some absenteeism models received general support (e.g., escape model and adaptation

process models), future predictive research is likely to enable solid conclusions about the

causal role of stress. In addition, the restorative model of absenteeism deserves some

rethinking, especially in the context of related literature (e.g., respite, coping) and other

methodological approaches (e.g., within-subjects designs, qualitative research). The

accumulation of primary research over the last three decades has greatly enabled the

capacity to develop and test theory. The present synthesis attests progress in this field by

providing a snapshot of the past and pointing ways for continued development in the

future.

Table 1. Associations between main variables of interest and absenteeism (all studies)

	k (# effects)	N	Observed r	Corrected r	90% Cred Int	% artifacts	95% CI
Stress	56	18630	.093	.145	-.08 to .37	29.52	.06 to .12
Frequency	14	3373	.106	.165	-.15 to .48	23.06	.03 to .18
Time lost	40	14743	.087	.136	-.05 to .33	35.36	.06 to .12
Psych Illness	128	66679	.133	.201	.04 to .36	39.14	.12 to .15
Frequency	37	16319	.144	.219	-.00 to .44	28.46	.11 to .18
Time lost	86	46890	.124	.188	.06 to .32	47.23	.11 to .14
Somatic illness	65	50632	.144	.220	-.01 to .45	20.68	.12 to .17
Frequency	24	18570	.147	.225	-.01 to .46	20.25	.10 to .19
Time lost	40	31929	.142	.217	-.01 to .45	20.65	.11 to .17

Table 2. Associations between stress/illness and absenteeism by type of sample, unpublished studies, and type of effect

	k (# effects)	N	Observed r	Corrected r	95% CI
Organizational sample					
Single sample					
Stress	29	6953	.085	.134	.05 to .12
Psych Illness	69	20885	.113	.172	.09 to .13
Somatic Illness	30	20770	.155	.238	.13 to .18
Multiple samples					
Stress	20	8048	.111	.173	.06 to .16
Psych Illness	51	43803	.141	.214	.12 to .16
Somatic Illness	31	29001	.135	.207	.09 to .18
Less dependent samples					
Stress	49	16103	.099	.155	.07 to .13
Psych Illness	75	45055	.138	.209	.12 to .16
Somatic Illness	47	36943	.150	.230	.12 to .18
Unpublished studies					
Stress	10	4772	.121	.190	.06 to .18
Psych Illness	15	2885	.163	.247	.11 to .22
Somatic Illness	4	861	.170	.260	.10 to .23
Effects based on point biserial correlations					
Stress	4	990	.125	.197	.06 to .19
Psych Illness	9	3398	.225	.340	.14 to .31
Somatic Illness	10	12293	.121	.185	.04 to .21

Table 3a. Associations between stress/illness and absenteeism in predictive studies

	k	N	Observed r	Corrected r	90% Cred Int	% artifacts	95% CI
Stress	6	1214	.255	.398	.20 to .60	55.70	.16 to .35
Frequency	4	994	.275	.431	.27 to .59	63.85	.17 to .38
Time Lost	2	220	.158	.247	.12 to .37	80.31	.03 to .29
Psych Illness	29	7848	.100	.152	-.01 to .31	50.10	.07 to .13
Frequency	12	3520	.091	.138	-.02 to .30	46.8	.04 to .14
Time Lost	17	4328	.108	.163	.01 to .32	53.49	.06 to .15
Somatic Illness	5	2133	.166	.254	.19 to .32	84.60	.12 to .21
Frequency	4	1861	.155	.238	.17 to .31	82.50	.11 to .20
Time Lost	(1 sample)						

Table 3b. Associations between stress/illness and absenteeism in postdictive studies

	k	N	Observed r	Corrected r	90% Cred Int	% artifacts	95% CI
Stress	37	11182	.080	.126	-.08 to .33	34.58	.05 to .11
Frequency	9	2320	.026	.041	.01 to .08	95.87	-.01 to .07
Self-report	5	1156	.022	.035	-.04 to .11	83.59	-.04 to .08
Records	4	1164	.031	.048	-	100.00	-.03 to .09
Time Lost	27	8740	.089	.139	-.05 to .33	37.65	.05 to .13
Self-report	20	6743	.116	.181	.01 to .35	43.93	.08 to .16
Records	7	1997	-.000	-.001	-	100.00	-.04 to .04
Psych Illness	81	47389	.138	.209	.06 to .36	40.065	.12 to .15
Frequency	19	9300	.159	.242	.01 to .47	29.94	.11 to .21
Self-report	5	3272	.246	.372	.14 to .60	33.07	.15 to .34
Records	14	6028	.112	.170	.06 to .28	59.43	.08 to .15
Time Lost	57	34619	.126	.191	.07 to .31	49.59	.11 to .14
Self-report	37	30204	.127	.192	.07 to .31	28.09	.11 to .15
Records	20	4415	.115	.174	.04 to .31	63.31	.08 to .15
Somatic Illness	36	25581	.165	.253	.06 to .44	31.86	.14 to .19
Frequency	11	8371	.201	.308	.16 to .46	47.15	.15 to .25
Self-report	4	3230	.194	.297	.05 to .55	22.53	.08 to .31
Records	7	5141	.205	.315	-	100.00	.18 to .23
Time Lost	23	15527	.145	.228	.03 to .43	27.15	.11 to .18
Self-report	17	12718	.165	.254	.09 to .42	36.71	.13 to .21
Records	7	4359	.096	.146	-.04 to .34	25.25	.03 to .16

Table 3c. Associations between stress/illness and absenteeism in cross-sectional studies

	k	*N*	Observed *r*	Corrected *r*	90% Cred Int	% artifacts	95% CI
Stress	12	5535	.079	.124	-.08 to .32	28.76	.02 to .13
Frequency	1 sample						
Time Lost	10	5084	.078	.121	-.08 to .32	26.71	.02 to .13
Psych Illness	18	11442	.134	.203	.03 to .37	32.67	.09 to .17
Frequency	6	3499	.158	.239	.04 to .44	30.82	.08 to .23
Time Lost	12	7943	.123	.187	.03 to .34	36.09	.08 to .17
Somatic Illness	24	22918	.118	.181	-.08 to .44	13.10	.07 to .16
Frequency	9	8338	.091	.139	-.11 to .38	12.94	.02 to .16
Time Lost	15	14580	.133	.204	-.06 to .47	14.07	.08 to .19

Table 4. Associations between each coded illness and absenteeism

	k	N	Observed r	Corrected r	90% Cred Int	% artifacts	95% CI
Anxiety	3	452	.063	.095	-.08 to .26	58.84	-.06 to .18
Acute stress/illness	6	13993	.059	.090	-.06 to .23	14.53	.04 to .08
Burnout	6	1277	.250	.377	-	100.00	.20 to .30
Emotional Exhaustion	24	11080	.120	.181	-.02 to .38	29.45	.08 to .16
Predictive	6	1040	.068	.102	-	100.00	.01 to .13
Postdictive	15	7005	.133	.200	-.04 to .45	22.76	.08 to .19
Depersonalization	11	1970	.077	.117	-.07 to .30	51.14	.01 to .14
Predictive	4	612	.053	.081	-	100.00	-.03 to .13
Postdictive	7	1358	.088	.133	-.09 to .35	40.93	.00 to .17
Lack of Pers Accomp	11	1970	.111	.168	.04 to .29	69.66	.06 to .17
Predictive	4	612	.192	.290	-	100.00	.12 to .27
Postdictive	7	1358	.075	.113	.03 to .19	83.63	.02 to .13
Depression	13	20437	.130	.197	-	100.00	.12 to .14
Predictive	3	386	.189	.286	-	100.00	.09 to .29
Postdictive	8	17227	.121	.183	-	100.00	.11 to .14
Fatigue	3	1063	.324	.489	.12 to .86	23.78	.13 to .52
Negative Mood	3	471	-.015	-.023	-.18 to .13	61.72	-.13 to .10
Somatic composite	49	25795	.165	.253	.12 to .39	52.37	.14 to .19
Predictive	5	2133	.166	.255	.24 to .27	84.60	.13 to .21
Postdictive	30	20790	.163	.249	.13 to .37	52.23	.14 to .19
Psyc composite	50	24747	.143	.216	.08 to .35	47.76	.12 to .16
Predictive	8	4713	.094	.141	-.03 to .32	29.20	.03 to .15
Postdictive	33	18099	.152	.230	.21 to .25	97.79	.14 to .17
Psychosomatic/illhealth	20	9601	.205	.310	.06 to .56	27.66	.15 to .26
Predictive	4	735	.152	.229	.05 to .41	54.19	.04 to .26
Postdictive	6	2126	.170	.257	-	100.00	.13 to .21
Other	19	27535	.113	.172	-.09 to .44	10.08	.06 to .16

Table 5. Correlation matrix input used in Structural Equation Modelling to test mediation model

	1	2	3	4
1. Stress	**.791**			
2. Psyc. Illness	.462	**.832**		
3. Somatic Illness	.423	.576	**.799**	
4. Absence	.145	.201	.220	**.531**

Harmonic N = 473.623
Boldface entries are reliabilities

Table 6. Associations between stress/illness and absenteeism in retrospective studies by absence measurement approach

	k (# effects)	N	Observed r	Corrected r	90% Cred Int	% artifacts	95% CI
Stress							
Self-report	33	13021	.093	.145	-.05 to .34	33.73	.06 to .12
Records	16	3696	.034	.054	-.15 to .26	40.75	-.02 to .09
Psych & Somatic Illness							
Self-report	84	64196	.157	.238	.06 to .42	30.79	.14 to .18
Records	74	41584	.112	.170	-.03 to .37	26.73	.09 to .13

Table 7. Correlation matrix for moderator variables and study characteristics

	1	2	3	4	5	6	7	8	9	10	11	12
1. Effect size												
2. Abs. measure	-.265**											
3. Abs. type	-.070	.354**										
4. Research design	.009	-.200**	-.117									
5. Aggregation period	.080	.111	.009	.089								
6. Gender	-.167*	-.009	-.001	-.102	-.100							
7. Health care	-.015	-.109	.018	-.181**	-.075	.286**						
8. Occupation status	.112	-.248**	-.234**	-.076	.177*	.245**	.135					
9. Sample size	-.025	-.083	-.021	.113	.254**	-.098	-.062	-.240**				
10. Year	.233**	-.236**	-.043	-.012	.196**	-.029	.035	.069	.112			
11. Type of effect	.086	-.075	-.009	.119	.375**	-.134*	.146*	.061	.067	-.013		
12. Post-intervention	-.217**	.241**	.101	.034	-.022	.052	.024	-.110	-.088	-.021	-.097	
13. Age	.050	-.297**	-.121	.117	.188*	-.033	-.019	.214*	.095	.234	.132	-.255**

$86 \leq N \leq 249$

$*p < .05$

$**p < .01$

abs. measure (1= records, 0 = self-report), abs.type (1 = frequency, 0 = time lost), research design (1 = predictive, 2 = postdictive, 3 = cross-sectional), health care (1 = yes, 0 = no), Occupational status (1= blue collar, 2 = lower white collar, 3 = higher white collar), Type of effect (1= point biserial, 0 = bivariate), post-intervention (1 = yes, 0 = no)

Table 8. Regression analysis testing the influence of type of absence measure on effect
size

	Variables	β	ΔR^2
Step 1	Controls		.296**
	Abs type	.06	
	Abs aggregation	.11	
	Health care sample	-.09	
	Occ status	.11	
	Year	.29**	
	Post-intervention	-.36**	
Step 2	Predictor		
	Abs. measure	-.30**	.070**

N = 105
**p <.01
Abs. measure (0=self-reports, 1= records)

Table 9. Regression analysis testing the impact of absence aggregation period on effect size

Variables	β	ΔR^2
Outcome: stress-absence effect (N = 48)		
Step 1 Predictor (linear)		.003
Abs aggregation	.05	
Step 2 Predictor (non-linear)		.000
Abs aggregation	.05	
Abs aggregation^2	.01	
Outcome: illness-absence effect (N = 103)		
Step 1 Controls		.134
Occ status	.04	
Sample size	-.08	
Year	.29*	
Type of effect	.08	
Step 2 Predictor (linear)		.016
Abs aggregation	.18	
Step 3 Predictor (non-linear)		.036
Abs aggregation	.76*	
Abs aggregation^2	-.69*	

*$p <.05$

Table 10. Regression analysis testing the impact of sample female proportion on effect size

	Variables	β	ΔR^2
Step 1	Controls		.006
	Type of effect	.01	
	Occ status	.08	
	Health care	.01	
Step 2	Predictor		.020
	Gender	-.16	

$N = 123$

Table 11. Regression analysis testing the impact of sample occupation status on effect size

	Variables	β	ΔR^2
Step 1	Controls		.139**
	Abs measure	-.32**	
	Abs type	.14	
	Gender	-.14	
	Sample size	-.12	
	Abs aggregation	.16	
Step 2	Predictor		.002
	Occ status	-.06	

$N = 114$
**$p < .01$
Occ status (1= blue collar, 2 = lower white, 3 = higher white)

Table 12. Regression analysis testing the influence of publication year on effect size

	Variables	β	ΔR^2
Step 1	Controls		.077**
	Abs measure	-.27**	
	Abs aggregation	.11	
Step 2	Predictor		.014
	Year	.12*	

$N = 234$

* $p < ..05$, one-tailed

**$p < .01$

Table 13. Effects aggregated by geographic region of sample

	k (# effects)	N	Observed r	Corrected r	95% CI
Asia					
Stress	7	2465	.102	.156	.03 to .19
Psych Illness	4	1790	.116	.175	.01 to .16
Somatic Illness	2	1401	.127	.195	.08 to .18
Australia & New Zealand					
Stress	2	416	.080	.125	-.02 to .18
Psych Illness	7	2577	.123	.186	.05 to .20
Somatic Illness	1 sample only				
Europe					
Stress	6	1904	.092	.144	.05 to .14
Psych Illness	53	45805	.140	.211	.12 to .16
Somatic Illness	32	28085	.170	.261	.14 to .20
North America					
Stress	37	12963	.093	.146	.06 to .13
Psych Illness	60	15738	.117	.177	.09 to .15
Somatic Illness	25	10647	.177	.272	.12 to .23

Table 14. Effects based on a meta-analysis of odds ratios (OR)

	k	N	OR	95% CI	Log OR
Psych Illness	7	58166	2.761	1.83 to 4.17	1.103
Somatic Illness	8	11118	1.720	1.55 to 1.90	0.542

Table 15. List of meta-analyzed studies showing key coded characteristics

Study	Effect (r)	Independent Variable	N	Design	Percent Female	Occupational Status	Absence Type	Months Absence
Abebe, 1987	0.25	stress	337	postdictive	69	lower white	time lost	9
Abramis, 1994	0.10	psychological	281	postdictive	46	not specified	time lost	1
Anderson et al., 2002	0.07	psychosomatic	2240	crosssectional	50	mixed	time lost	3
Anderson, 1985	0.14	psychological	77	postdictive	100	lower white	time lost	6
Baba, 1990	0.09	psychological	175	postdictive	0	white collar	frequency	9
	0.19	psychological	175	postdictive	0	white collar	time lost	9
Bakker et al., 2003	0.16	psychological	214	predictive	31	not specified	time lost	12
	0.06	psychological	214	predictive	31	not specified	frequency	12
Bass et al., 1996	0.04	stress	143	postdictive	25	mostly white collar	time lost	12
	0.12	somatic	143	postdictive	25	mostly white collar	time lost	12
	0.19	psychological	143	postdictive	25	mostly white collar	time lost	12
Bass, 2003	0.05	psychological	124	postdictive	56	blue collar	frequency	3
Bhagat & Allie, 1989	-0.08	stress	276	postdictive	82	lower white	time lost	12
	0.10	psychological	276	postdictive	82	lower white	time lost	12
	0.14	psychological	276	postdictive	82	lower white	time lost	12
	0.02	psychological	276	postdictive	82	lower white	time lost	12
Bhagat et al., 1985	0.02	stress	132	crosssectional	58	white collar	time lost	12
Biggam et al., 1997	0.29	psychological	699	crosssectional	12	mostly lower white	time lost	12
	0.11	stress	699	-	12	mostly lower white	time lost	12
Blumberg, 1980	0.00	stress	54	crosssectional	0	blue collar	time lost	-
Boise, 1989	0.05	stress	1355	crosssectional	100	mostly higher white	time lost	1
	0.00	stress	854	crosssectional	0	mostly higher white	time lost	1
Bond & Bunce, 2001	0.11	psychological	97	postdictive	37	lower white	time lost	12
	0.21	somatic	97	postdictive	37	lower white	time lost	12
Burggraf, 1998	0.12	psychological	62	postdictive	83	not specified	time lost	6
Byron & Peterson, 2002	0.22	acute (stress)	108	crosssectional	40	mixed	attitudinal	2.5
Carson et al., 1999	0.06	psychological	103	postdictive	53	lower white	time lost	12
Caughey, 1996	0.27	psychological	36	postdictive	78	lower white	time lost	6
Chen & Spector, 1992	0.06	stress	392	crosssectional	65	white collar	other	-
Christie & Schultz, 1998	0.08	psychosomatic	181	postdictive	69	mostly lower white	frequency	6

Study							
Clegg et al., 1987	0.07	stress	181	postdictive	69	mostly lower white	frequency	6
	0.15	psychological	209	predictive	100	blue collar	frequency	6
Coffey & Coleman, 2001	0.22	psychological	80	postdictive	46	lower white	time lost	12
	0.14	psychological	80	postdictive	46	lower white	time lost	12
	0.17	psychological	80	postdictive	46	lower white	time lost	12
	0.07	psychological	80	postdictive	46	lower white	time lost	12
	0.14	stress	80	postdictive	46	lower white	time lost	12
Cohen, 1998	0.23	stress	238	postdictive	95	lower white	time lost	12
Cooper & Bramwell, 1992	0.19	psychological	236	postdictive	-	mostly higher white	frequency	6
	0.10	somatic	377	postdictive	-	blue collar	time lost	6
	0.09	psychological	236	postdictive	-	mostly higher white	time lost	6
	0.10	psychological	377	postdictive	-	blue collar	time lost	6
	0.03	somatic	236	postdictive	-	mostly higher white	time lost	6
	0.10	somatic	377	postdictive	-	blue collar	time lost	6
	0.02	psychological	377	postdictive	-	blue collar	frequency	6
	0.25	somatic	236	postdictive	-	mostly higher white	frequency	6
Dartigues et al., 1998	0.07	somatic	2586	postdictive	-	not specified	time lost	36
De Boer et al., 2002	0.24	somatic	514	predictive	0	lower white	frequency	12
	0.33	somatic	514	crosssectional	0	lower white	frequency	12
De Croon et al., 2001	0.40	somatic	735	postdictive	2	blue collar	frequency	12
	0.43	psychological	735	postdictive	2	blue collar	frequency	12
Deery et al., 2002	0.07	psychological	480	postdictive	69	lower white	frequency	12
	0.26	somatic	480	postdictive	69	lower white	frequency	12
deJonge et al., 2000	0.06	psychological	1412	crosssectional	30	not specified	time lost	12
	0.20	psychological	1412	crosssectional	30	not specified	frequency	12
	0.14	psychological	1412	crosssectional	30	not specified	frequency	12
	0.16	psychological	1412	crosssectional	30	not specified	time lost	12
Donaldson & Blanchard, 1995	0.20	psychological	345	postdictive	66	mixed	other	6
Donaldson et al., 1999	0.01	stress	146	postdictive	2	blue collar	time lost	6
Edwards, 1982	0.16	psychological	395	postdictive	49	lower white	time lost	12
	0.13	somatic	395	postdictive	49	lower white	time lost	12
	0.19	stress	395	postdictive	49	lower white	time lost	12
Engel et al., 1999	0.05	acute (illness)	13161	postdictive	12	lower white	time lost	3
Erickson et al., 2000	0.23	psychological	211	crosssectional	49	not specified	time lost	3

Study	r	Symptom	N	Design	%	Occupation	Measure	Months
Fenwick & Tausig, 2001	0.09	psychological	2905	crosssectional	50	mixed	time lost	3
	0.04	psychological	2905	postdictive	50	mixed	time lost	3
	0.08	somatic	2905	postdictive	50	mixed	time lost	3
Folkedal et al., 2000	0.29	stress	70	crosssectional	-	blue collar	time lost	2.5
	0.23	somatic	70	crosssectional	-	blue collar	time lost	2.5
Freeborn et al., 1977	0.17	somatic	1550	postdictive	54	mixed	time lost	12
	0.18	psychosomatic	1550	postdictive	54	mixed	time lost	12
Fried et al., 2002	0.14	somatic	802	crosssectional	55	white collar	frequency	24
Galloway et al., 1984	0.17	psychological	296	postdictive	-	lower white	time lost	6
	0.10	stress	296	postdictive	-	lower white	attitudinal	6
George, 1989	-0.03	psychological	210	predictive	83	lower white	attitudinal	3
Geurts et al., 1994	0.15	somatic	453	predictive	0	blue collar	frequency	12
Gray-Toft & Anderson, 1985	0.03	stress	159	postdictive	-	lower white	frequency	9
Hackett, 1982	0.06	stress	168	crosssectional	54	lower white	time lost	9
Hardy et al., 2003	0.31	psychological	310	predictive	72	higher white	time lost	12
Hemingway & Smith, 1999	-0.02	stress	252	postdictive	98	lower white	frequency	6
Hendrix & Spencer, 1989	0.37	somatic	443	crosssectional	55	mostly white collar	time lost	6
	0.28	psychological	443	postdictive	55	mostly white collar	time lost	6
Hendrix & Taylor, 1987	0.26	stress	463	postdictive	-	white collar	time lost	6
Hensing & Spak, 1998	0.06	psychological	292	crosssectional	100	not specified	frequency	24
	0.05	psychological	292	crosssectional	100	not specified	time lost	24
Ho, 1997	0.29	stress	188	postdictive	59	white collar	time lost	12
Hornquist et al., 1993*	0.22	psychological	88	predictive	50	not specified	time lost	12
	0.25	psychological	88	predictive	50	not specified	frequency	12
Ivancevich, 1986	0.41	psychological	185	crosssectional	10	blue collar	frequency	6
Iverson et al., 1998	0.03	psychological	487	predictive	74	mostly lower white	frequency	6
	0.19	psychological	487	predictive	74	mostly lower white	frequency	6
	0.04	psychological	487	predictive	74	mostly lower white	frequency	6
Jackson, 1983*	-0.07	psychological	77	postdictive	-	mostly lower white	time lost	3
	-0.15	psychological	77	postdictive	-	mostly lower white	frequency	3
Jamal, 1981	0.24	psychological	431	predictive	97	lower white	frequency	4
Jamal, 1984	0.39	stress	435	predictive	97	lower white	frequency	4
Jamal, 1986	0.05	somatic	252	postdictive	0	lower white	frequency	4
	0.21	stress	285	predictive	18	blue collar	frequency	4

Study								
Jamal & Baba, 1997	0.17	somatic	285	predictive	18	blue collar	frequency	4
Jamal & Baba, 1997	-0.09	stress	252	postdictive	0	lower white	frequency	4
Jamal & Baba, 2003	0.22	psychological	175	postdictive	67	lower white	frequency	2
Jamal & Baba, 2003	0.29	somatic	110	postdictive	20	not specified	frequency	2
Jamal & Baba, 2003	0.19	stress	110	postdictive	20	not specified	frequency	2
Jamal & Baba, 2003	0.07	somatic	175	postdictive	67	not specified	frequency	2
Jamal & Baba, 2003	0.06	stress	175	postdictive	67	not specified	frequency	2
Jenkins, 1985	-0.04	psychological	184	predictive	43	higher white	frequency	12
Jenkins, 1985	0.11	psychological	184	postdictive	43	higher white	time lost	12
Jenkins, 1985	0.10	psychological	184	postdictive	43	higher white	frequency	12
Jenkins, 1985	-0.03	psychological	184	predictive	43	higher white	time lost	12
Jeurissen & Nyklicek, 2001	-0.03	stress	162	postdictive	95	not specified	frequency	12
Jeurissen & Nyklicek, 2001	-0.11	psychological	162	postdictive	95	not specified	frequency	12
Jeurissen & Nyklicek, 2001	0.01	somatic	162	postdictive	95	not specified	frequency	12
Judge et al., 1997	0.15	illhealth	73	predictive	78	mostly lower white	time lost	6
Judge et al., 1997	0.20	illhealth	73	postdictive	78	mostly lower white	time lost	12
Keller, 1984	0.20	stress	190	predictive	40	mixed	frequency	10
Kilfedder et al., 2001	0.10	psychological	510	postdictive	87	lower white	time lost	6
Kilfedder et al., 2001	0.01	psychological	510	postdictive	87	lower white	time lost	6
Kilfedder et al., 2001	0.10	psychological	510	postdictive	87	lower white	time lost	6
Kilfedder et al., 2001	0.10	stress	510	postdictive	87	lower white	time lost	6
Kilfedder et al., 2001	0.22	psychological	510	postdictive	87	lower white	time lost	6
Kirchmeyer & Cohen, 1999	0.22	psychological	200	postdictive	100	mostly lower white	time lost	12
Kivimaki et al., 2002	0.08	psychological	2054	postdictive	100	not specified	time lost	12
Knowles, 1980	0.04	psychological	732	predictive	0	not specified	time lost	12
Knowles, 1980	-0.01	psychological	185	postdictive	85	lower white	time lost	8
Knowles, 1980	-0.03	stress	185	postdictive	85	lower white	time lost	8
Kopp et al., 1995	0.12	psychological	16020	postdictive	50	mixed	time lost	12
Krantz & Ostergen, 2002	0.04	psychosomatic	301	predictive	100	mixed	frequency	12
Krantz & Ostergen, 2002	0.27	psychosomatic	301	predictive	100	mixed	time lost	12
Kumar et al., 2003	0.53	somatic	33	crosssectional	42	not specified	time lost	0.25
Kupperman et al., 1995	0.03	somatic	369	postdictive	47	not specified	time lost	6
Kushmir et al., 2001	0.19	acute (illness)	199	crosssectional	54	mixed	time lost	0.5
Lambert, 1998	0.18	stress	1704	crosssectional	25	lower white	time lost	6

Study	r	Type	N	Design	%	Occupation	Measure	Time
Lawson & O'Brien, 1994	-0.02	psychological	76	postdictive	72	mostly lower white	time lost	3
	-0.06	psychological	76	postdictive	72	mostly lower white	time lost	3
	-0.06	psychological	76	postdictive	72	mostly lower white	time lost	3
	0.25	psychological	76	predictive	72	mostly lower white	time lost	1.5
	0.16	psychological	76	predictive	72	mostly lower white	time lost	1.5
	0.07	psychological	76	predictive	72	mostly lower white	time lost	1.5
Lee, 1997	0.00	psychosomatic	30	crosssectional	76	mixed	time lost	6
Leigh et al., 1995	0.44	somatic	875	crosssectional	3	not specified	time lost	3
Loewenthal et al., 2000	0.02	psychological	105	crosssectional	0	white collar	time lost	12
Long & Gessaroli, 1989	0.09	stress	781	postdictive	79	lower white	time lost	12
Lu et al., 1997	0.10	somatic	1054	postdictive	12	mixed	time lost	12
	0.09	psychological	1054	postdictive	12	mixed	time lost	12
	0.08	stress	1054	postdictive	12	mixed	time lost	12
Lu et al., 1999	0.15	psychological	347	postdictive	44	white collar	time lost	-
	0.21	somatic	347	postdictive	44	white collar	time lost	-
	-0.01	stress	347	postdictive	44	white collar	time lost	-
Maes & Schlosser, 1987	0.36	somatic	397	postdictive	56	not specified	time lost	12
Manning et al., 1989	0.13	somatic	422	postdictive	67	not specified	time lost	12
	0.06	stress	422	postdictive	67	not specified	time lost	12
	0.11	psychological	422	postdictive	67	not specified	time lost	12
Matteson & Ivancevich, 1983	0.29	psychosomatic	388	crosssectional	86	lower white	time lost	6
Mckee et al., 1992	0.02	stress	733	postdictive	98	blue collar	frequency	6
	-0.03	stress	733	postdictive	98	blue collar	time lost	6
Melamed et al., 1995	0.17	psychosomatic	393	crosssectional	100	blue collar	frequency	24
	0.17	psychosomatic	885	crosssectional	0	blue collar	frequency	24
Melamed et al., 1997	0.01	somatic	4326	crosssectional	27	blue collar	frequency	24
	0.02	somatic	4326	crosssectional	27	blue collar	time lost	24
Morken et al., 2002	0.14	somatic	5654	crosssectional	14	mostly blue	time lost	12
Nielsen et al., 2002	−0.19	psychological	2068	postdictive	-	not specified	time lost	12
	0.18	psychological	2068	postdictive	-	not specified	frequency	12
	0.15	somatic	2068	postdictive	-	not specified	frequency	12
	0.18	somatic	2068	postdictive	-	not specified	time lost	12
Ogus, 2004	0.31	stress	59	postdictive	54	higher white	time lost	-
	0.60	psychological	118	postdictive	68	lower white	time lost	-

Study	r	Construct	N	Design	%	Collar	Absence type	
Ostroff, 1993	0.40	psychological	118	postdictive	68	lower white	time lost	-
	0.30	psychological	118	postdictive	68	lower white	time lost	-
	0.34	stress	118	postdictive	68	lower white	time lost	-
Padnick, 1986	0.33	psychological	59	crosssectional	54	higher white	time lost	-
Parasuraman, 1982	-0.07	stress	533	postdictive	66	lower white	time lost	-
	0.21	somatic	219	postdictive	55	mixed	time lost	3
	0.06	stress	160	postdictive	35	blue collar	time lost	9
	0.09	stress	160	predictive	35	blue collar	time lost	3
Park, 2002	0.09	psychological	240	postdictive	76	mostly lower white	time lost	3
Pelled & Xin, 1999	0.17	psychological	99	predictive	27	mostly lower white	time lost	1
Pierce & Molloy, 1990	0.36	psychological	170	postdictive	-	lower white	frequency	12
	0.26	psychological	170	postdictive	-	lower white	time lost	12
Pousette & Hanse, 2002	0.22	somatic	337	crosssectional	97	lower white	time lost	12
	0.32	somatic	209	crosssectional	35	lower white	time lost	12
	0.32	somatic	209	crosssectional	35	lower white	frequency	12
	0.26	somatic	241	crosssectional	39	blue collar	frequency	12
	0.22	somatic	337	crosssectional	97	lower white	frequency	12
	0.33	somatic	241	crosssectional	39	blue collar	time lost	12
Price, 1998	0.08	somatic	609	predictive	86	white collar	attitudinal	3
	0.05	psychological	609	predictive	86	white collar	attitudinal	3
Probst, 2002	0.14	stress	283	postdictive	-	not specified	time lost	12
	0.34	somatic	283	postdictive	-	not specified	time lost	12
	0.16	psychological	283	crosssectional	-	not specified	time lost	12
Rahman, 1989	0.35	stress	59	crosssectional	0	blue collar	frequency	-
Raja, 2004	0.04	stress	366	postdictive	3	mostly lower white	time lost	6
Rascale, 2000	0.26	acute (illness)	145	postdictive	0	mixed	time lost	2
	0.12	acute (stress)	145	crosssectional	0	mixed	time lost	2
Rees & Cooper, 1991	0.16	psychological	1042	postdictive	77	white collar	time lost	6
	0.20	somatic	1042	postdictive	77	white collar	time lost	6
Rees & Cooper, 1990	0.05	stress	383	postdictive	75	white collar	time lost	6
Reisine, 1984	0.35	somatic	571	postdictive	41	mostly white collar	time lost	12
Roberts, 1983	0.03	stress	335	postdictive	56	lower white	time lost	8
Rosse, 1983	0.19	psychosomatic	24	crosssectional	82	lower white	frequency	1
	0.28	psychosomatic	24	crosssectional	82	lower white	time lost	1

Study								
Rousseau, 1978	-0.08	psychological	139	crosssectional	70	mixed	time lost	1
	-0.05	psychological	139	crosssectional	70	mixed	frequency	1
	0.01	somatic	139	crosssectional	70	mixed	frequency	1
	0.00	somatic	139	crosssectional	70	mixed	time lost	1
Saksvik, 1996	0.48	acute (illness)	235	postdictive	-	not specified	other	6
Salyers, 1998	-0.15	psychological	26	predictive	51	lower white	time lost	12
	0.07	psychological	26	predictive	51	lower white	time lost	12
	0.49	psychological	23	predictive	51	lower white	time lost	12
	-0.11	psychological	23	predictive	51	lower white	time lost	12
	0.10	psychological	23	predictive	51	lower white	time lost	12
	0.05	psychological	26	predictive	51	lower white	time lost	12
Schaubroeck et al., 1993*	-0.05	somatic	59	crosssectional	43	mostly lower white	time lost	3
	0.00	psychological	59	crosssectional	43	mostly lower white	time lost	3
	-0.13	somatic	59	crosssectional	43	mostly lower white	frequency	3
	-0.21	psychological	59	crosssectional	43	mostly lower white	frequency	3
Schmieder, 1994	0.06	psychological	563	postdictive	33	blue collar	time lost	12
Seegers & van Elderen, 1996	0.22	psychological	376	postdictive	-	higher white	time lost	12
	0.28	somatic	376	postdictive	-	higher white	time lost	12
Seifert, 1995	0.22	psychological	97	crosssectional	18	lower white	time lost	6
	0.15	somatic	97	crosssectional	18	lower white	time lost	6
Selzer et al, 1978	0.12	psychological	61	predictive	100	blue collar	frequency	1
	0.00	psychological	61	postdictive	100	blue collar	frequency	1
	0.20	psychological	53	postdictive	0	blue collar	frequency	1
	0.01	psychological	53	predictive	0	blue collar	frequency	1
Shaw & Gupta, 2001	0.24	psychological	272	predictive	49	mixed	time lost	6
	0.24	somatic	272	predictive	49	mixed	time lost	6
	0.27	psychological	272	postdictive	49	mixed	time lost	6
	0.42	somatic	272	postdictive	49	mixed	time lost	6
Shelledy, 1990	0.23	psychological	458	crosssectional	58	lower white	time lost	12
Sheridan, 1985	0.08	stress	84	predictive	96	lower white	frequency	4
Singh, 1987	0.11	psychological	60	postdictive	-	not specified	-	-
Siu, 2002	0.20	psychosomatic	129	postdictive	75	lower white	time lost	12
Smulders & Nijhuis, 1999	0.33	illhealth	1348	crosssectional	0	blue collar	frequency	12
	0.40	illhealth	1348	crosssectional	0	blue collar	time lost	12

Study	r	Type	N	Design	%	Collar	Outcome	Months
Spector & Jex, 1991	0.03	stress	232	postdictive	55	mixed	time lost	3
	-0.04	psychological	232	postdictive	55	mixed	time lost	3
	0.06	somatic	232	crosssectional	55	mixed	time lost	3
Spector et al., 1988	0.12	somatic	154	crosssectional	100	lower white	time lost	3
	0.15	psychological	154	crosssectional	100	lower white	time lost	3
	0.04	stress	154	crosssectional	100	lower white	time lost	3
Tang & Hammontree, 1992	0.18	psychosomatic	60	crosssectional	3	lower white	time lost	6
	0.12	psychosomatic	60	predictive	3	lower white	time lost	6
	0.07	psychosomatic	60	postdictive	3	lower white	time lost	6
	0.24	stress	60	crosssectional	3	lower white	time lost	6
	0.15	stress	60	postdictive	3	lower white	time lost	6
	0.34	stress	60	predictive	3	lower white	time lost	6
Taris et al., 2002	0.23	psychological	1297	postdictive	51	not specified	other	12
	0.21	psychological	1297	postdictive	51	not specified	other	12
Taylor, 2000	0.09	somatic	100	postdictive	-	not specified	time lost	6
Thomas, 1983	0.22	psychological	66	postdictive	-	lower white	time lost	12
Trainor, 1994	0.28	psychological	150	postdictive	-	lower white	time lost	6
	0.22	somatic	150	postdictive	-	lower white	time lost	6
Trivedi et al., 1981	0.47	stress	122	postdictive	0	not specified	-	12
Turner, 1988	0.03	stress	117	postdictive	0	blue collar	time lost	12
Turner, 1997	0.30	psychological	236	postdictive	71	lower white	time lost	3
Ulleberg & Rundmo, 1997	0.08	somatic	917	postdictive	9	not specified	time lost	6
Unden, 1996	0.17	psychosomatic	133	postdictive	75	lower white	other	12
	0.18	somatic	133	postdictive	75	lower white	other	12
Vaananen et al., 2003	0.19	psychological	911	postdictive	100	mixed	frequency	18
	0.21	somatic	2865	postdictive	0	mixed	frequency	18
	0.22	somatic	911	postdictive	100	mixed	frequency	18
	0.13	psychological	2865	postdictive	0	mixed	frequency	18
van Dick & Wagner, 2001	0.30	somatic	201	postdictive	55	lower white	time lost	4
	0.31	psychological	201	postdictive	55	lower white	time lost	4
van Dierendonck et al., 1998*	0.01	psychological	149	postdictive	72	white collar	time lost	12
	0.13	psychological	149	postdictive	72	white collar	time lost	12
	0.12	psychological	149	postdictive	72	white collar	time lost	12
	0.03	psychological	149	postdictive	72	white collar	frequency	12

Vasse et al., 1998	0.15	psychological	149	postdictive	72	white collar	frequency	12
	0.02	psychological	149	postdictive	72	white collar	frequency	12
	0.11	psychological	471	postdictive	25	mixed	other	6
Westerling & Jonsson, 1980	0.14	somatic	1711	crosssectional	41	not specified	frequency	24
	0.12	somatic	1711	crosssectional	41	not specified	time lost	24
Woo et al., 1999	0.16	psychological	329	postdictive	59	not specified	time lost	3
	0.08	stress	329	postdictive	59	not specified	time lost	3

*studies with post-intervention statistics

Figure 1. A framework for understanding the stress-absence association
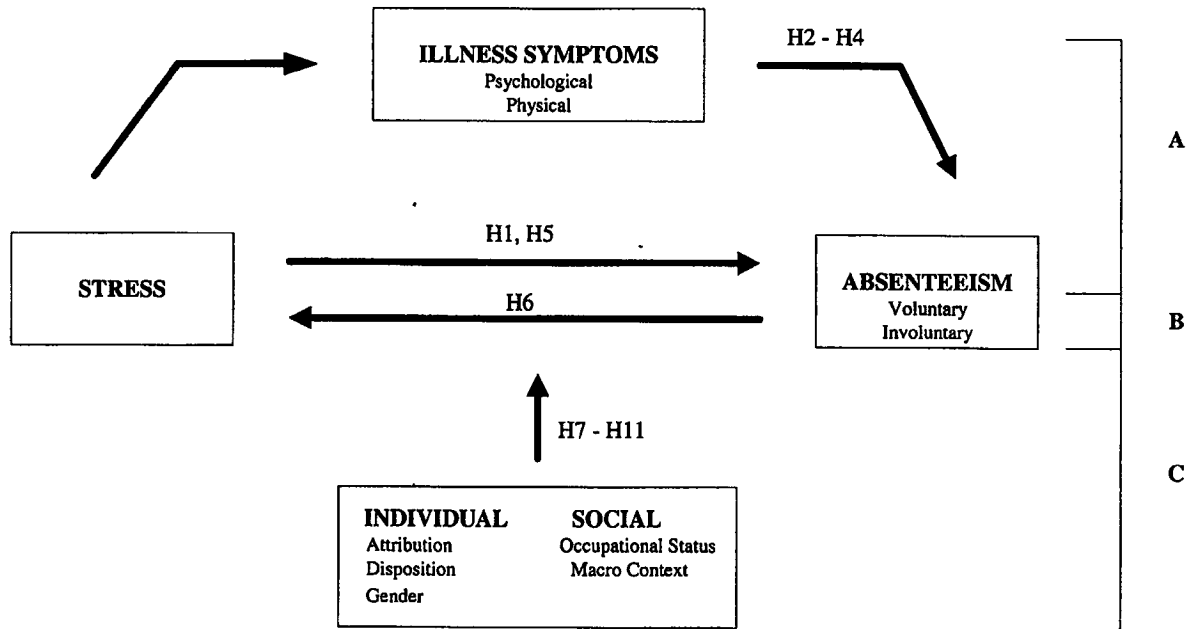
Figure 2. Path diagrams showing models tested using Structural Equation Modeling
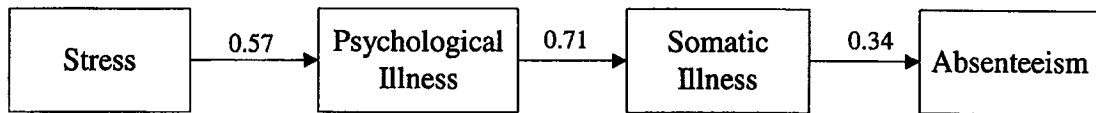
a. Theoretical model showing mediating path coefficients (lambda)

b. Comparison model 1 showing path coefficients (lambda)

c. Comparison model 2 showing path coefficients (lambda)

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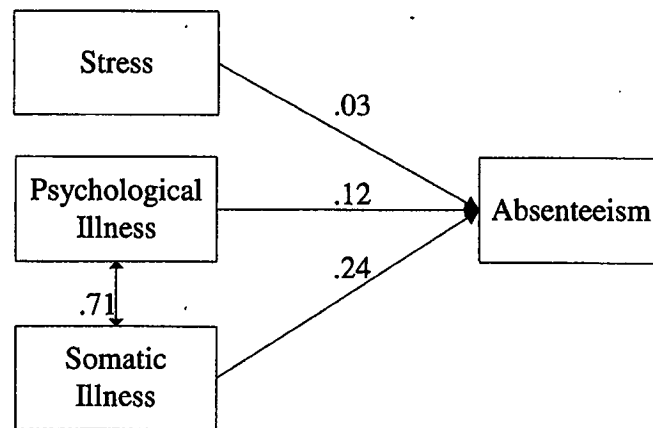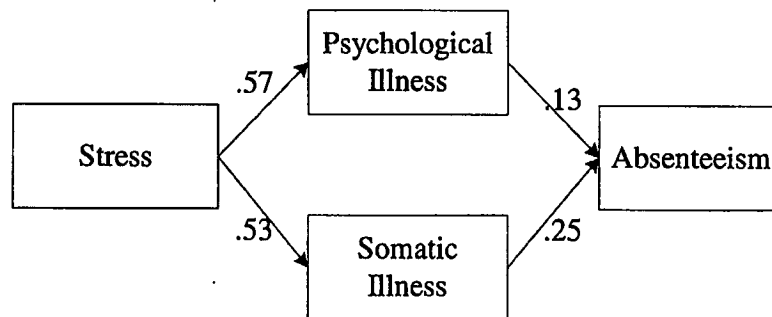References with an asterisk (*) indicate studies included in the main meta-analysis. References with a circumflex (^) indicate studies included in the meta-analysis of odds ratios.

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Appendix A
Coding Sheet

GENERAL:
1. Study ID _____
2. Authors: _____
3. Journal/Book/Presentation Name:

_____

4. Year: _____

SAMPLE/CONTEXT:
5. Sample size (that analysis is based on): _____
6. Aims to examine stress in the context of some traumatic event: no   yes
(event:_____)
If correlations are reported for sub-samples (e.g., by gender or occupation), check here
_____ AND attach sub-sample section)
7. Mean Age (SD) of sample = _____
8. Gender (% women in sample): _____
9. Occupation : ____blue collar or____ mostly blue   ____ mixed: blue and
white

          ____ white collar or ____mostly white   ____not specified
          ____ lower white collar or ____ mostly lower white
          ____ higher white collar or ____mostly higher white
10. Health care workers: yes (describe _____)
                     no (describe_____)
11. ___ mostly part-time ___ mostly full-time ____ mixed ____ not specified
12. Shift workers: ___yes ____ no ____mixed ___not specified
13.   Country: ___North America(_____), ___Europe (_____), ___Other
        (_____)

METHOD:
14.   Type of Survey: 1- questionnaire, 2 - phone interview, 3 - face-to-face interview
15.   Research Design: ____ predictive (stress/illness is measured before
       absence)
   (check all applicable)   ____ postdictive (absence is measured before stress/illness)
                         ____ cross-sectional (period of absence and stress/illness
                         measure
                              overlap; *e.g., absence and stress measures refer to past 3
                         months*)
16. Time lapse b/n IV and DV measures (in months): _____ for (circle) predictive   postdictive
both

MEASURES:
Absenteeism measure (check ALL measures for which associations with stress are

reported)

17. Aggregated over (in months): _____
18. Type of measure :____self report
              ____ records
19. Reason for absence: ____illness

_____other (describe:_____)

_____not specified

20. Type of absence: _____ attitudinal

_____ frequency

_____ time lost

_____ other

(describe:_____)

21. Duration of each absence measured: _____ 1-3 days   _____not specified

_____ > 2-3 days   _____other (

_____)

_____ > 5-8 days

22. Absence reliability: _____ (NR, if not reported); Type of
reliability:_____

23. Association between frequency and time lost (NA, if not applicable) = _____

24. Absence measure transformed (log or other) in correlation analysis (circle):   yes   no

25. Absence measure dichotomized in correlation analysis (circle):   yes   no

Stress & Illness Measures

26. Stress dichotomized (circle):   yes   no

27. Illness dichotomized (circle):   yes   no

Stress Variable	Reliability	Mean (SD)	Name of Measure
_____work stress			
_____job related tension			
Other:			

Illness Variable	Reliability	Mean (SD)	Name of Measure
___Anxiety			
___Burnout ___ emotional exhaustion ___ depersonalization ___ personal accomp.			
_____Cardiovasc/heart disease			
_____Cold & flu			
_____Depression			
_____Fatigue			
_____Gastrointest/stomach probs			
_____General Health			
_____Headaches			
_____Hostility			
_____Negative mood			
_____ Psychological distress			
_____ Sleep problems			
_____ Ulcers			
_____Composite- psych/mental			
_____Composite somatic/physical			
_____ Composite - mixed (psychosomatic)			

Other:			

28. Report **zero-order correlation** for ALL reported measures (**for sub-samples, report in attached**)

****if these are post-event associations, check _____****

Variable	Frq or Att Absence M=___SD=___			Time Lost Absence M=___SD=___			Other Abs_____ M=___SD=___		
	Pre	Post	Cross	Pre	Post	Cross	Pre	Post	Cross
Stress									
Anxiety									
Burnout									
Burnout (exhaustion)									
Burnout (depersonal)									
Burnout (pers accomp)									
Cardiovas/heart disease									
Cold & flu									
Depression									
Fatigue									
Gastrointest/stomach									
General health									
Headaches									
Hostility									
Negative mood									
Psychological distress									
Sleep problems									
Ulcers									
Composite - psychol									
Composite - physical									
Composite – mixed									
Other									

FOR STUDIES REPORTING MEAN DIFFERENCE

Describe the criteria used to split stress, illness, or absence (e.g., hi or low; present or absent):

Group/Category	N	Mean (SD)_____	Mean (SD)_____	t- or F-
effect size (d)				

Group/Category	N	Mean (SD)_____	Mean (SD)_____	t- or F-

effect size (d)				

SUB-SAMPLE Correlations (for: _____)

N = _____

Variable	Frequency or Attitudinal Absence			Time Lost Absence			Other Absence		
	PRE	POST	CROSS	PRE	POST	CROSS	PRE	POST	CROSS
Stress									
Anxiety									
Burnout									
Burnout (emexhaustion)									
Burnout (depersonal)									
Burnout (pers accomp)									
Cardiovasc/heart disease									
Cold & flu									
Depression									
Fatigue									
Gastrointestinal/stomach									
General Health									
Headaches									
Hostility									
Negative mood									
Psychological distress									
Sleep problems									
Ulcers									
Composite – psych									
Composite - physical									
Composite – mixed									
Other									

SUB-SAMPLE Correlations (for: _____)

N = _____

Variable	Frequency or Attitudinal Absence			Time Lost Absence			Other Absence		
	PRE	POST	CROSS	PRE	POST	CROSS	PRE	POST	CROSS
Stress									
Anxiety									
Burnout									
Burnout (emexhaustion)									
Burnout (depersonal)									

Burnout (pers accomp)								
Cardiovasc/heart disease								
Cold & flu								
Depression								
Fatigue								
Gastrointestinal/stomach								
General Health								
Headaches								
Hostility								
Negative mood								
Psychological distress								
Sleep problems								
Ulcers								
Composite - psych								
Composite - physical								
Composite – mixed								
Other								

Appendix B
Email Request for Unpublished Studies

Subject: Seeking Unpublished Studies

Unpublished studies examining the association between absenteeism from work and job stress OR related variables (e.g., anxiety, depression, fatigue, illness, headaches, somatic complaints, general health) are required for inclusion in a research synthesis. For each study, the following information must be available/provided:
- Description of sample (size, gender, occupation)
- Description of measures
- Description of procedures used in collecting measures
- Reliability estimates of measures (if available)
- Measures of association (zero-order correlation, mean difference, or 2x2 contingency table) for each variable and absenteeism.

To ensure proper acknowledgement, please provide the names of all involved researchers, the title of the study, and the year. Also mention whether the findings are part of a Masters thesis, Doctoral dissertation, or conference presentation (provide name). If the requested findings are available in a published conference proceedings, we appreciate if you could send us an email with details on the source.

We look forward to your cooperation, and thank you in advance. The above information may be emailed to   'R mailed to:

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