

DAILY STRESS AND HEALTH SYMPTOMS: THE MEDIATING AND
MODERATING ROLES OF BIOPSYCHOSOCIAL FACTORS

by

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Abstract

Psychological stress has been cited as a risk factor for poor health across the lifespan, in many cultures, and in both the human and animal literatures. In this program of research we tested potential psychological and biological mediators linking daily stressful life experiences to daily health symptoms. Namely, we tested whether the psychological factors of coping or emotional responses to stress, as well as biological responses of daily cortisol or sleep, mediated the daily stress-health association. A second goal of this research was to test whether stress-biology associations varied according to the childhood psychosocial environment in which an individual was raised. 87 healthy undergraduate students ($M_{\text{age}}=21.51$, 66.7% female, 27.6% Caucasian) participated in the study. Participants provided information on characteristics of their childhood family environment (conflict, parental warmth). For one week they completed a daily stress checklist via electronic diary, and noted their coping and emotional responses to the most serious stressor of their day. As well, they provided salivary cortisol samples 4 times/day, and wore an Actiwatch to measure sleep (minutes, efficiency). Using hierarchical linear modeling techniques, we found that, on days when individuals reported more severe stressors, they also reported more health symptoms ($\beta = .24$, $p = .04$). Emotional responses to stress significantly mediated the relation between daily stress and daily health symptoms, such that when emotional responses to stress were controlled, the relationship between daily stress and health symptoms became non-significant ($\beta = -.001$, $p = .99$). In contrast, coping and biological profiles did not mediate the daily stress-health relationship. Furthermore, some evidence emerged that the childhood psychosocial environment moderated the relationship between daily stress and biological outcomes; among individuals from risky childhood environments, on days when they experienced a greater number of stressors, they slept for fewer minutes ($\beta = -12.10$, $p = .02$). As well, among

individuals from childhoods characterized as low in parental warmth, on days when they experienced more severe daily stressors, they had greater daily cortisol output ($\beta = -0.16$, $p = .04$). Overall, findings from this study suggest that individuals may be more susceptible to the negative effects of stress on health if they are from difficult childhood environments and have negative emotional responses to stressors.

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Co-authorship Statement

Chapters 2 and 3 of the dissertation are manuscripts that are either under submission or to be submitted shortly, in which I am the first author, and my supervisor, Dr. Edith Chen, is a co-author. I designed the research project and collected all of the data. As well, I performed all of the data analyses. In addition, I drafted both manuscripts, while feedback from Dr. Chen was incorporated into the final versions.

General Introduction

Psychological stress is a fairly universal experience. It refers to “a process in which environmental demands tax or exceed the adaptive capacity of the organism, resulting in psychological and biological changes that may place persons at risk for disease,” (Cohen, Kessler & Gordon, 1995), “any event in which environmental demands, internal demands, or both tax or exceed the adaptive resources of an individual, social system, or tissue system,” (Monat & Lazarus, 1977), or “the nonspecific response of the body to any demand made upon it,” (Selye, 1955). Hence, the term stress is sometimes used to refer to external environmental stimuli or demands that tax an organism’s systems. These demands, or stressors, can vary in number, duration, and severity, and can be evoked within the laboratory or occur within the natural environment (Seegerstrom & Miller, 2004). Psychological stress can also refer to the subjective perception of events as threatening or negative, the idea being that the impact of stress depends on how an individual appraises that event (Baum, Cohen, & Hall, 1993). Finally, the term stress is sometimes used to refer to physiological perturbations from homeostasis, such as hormonal changes, disturbed sleep, fatigue, and infertility that indicate effects of a stressor on an organism (Greil, 1997; Kales, Soldatos, & Kales, 1987; Maier & Watkin, 1998; Selye, 1955).

While stress has long been linked to psychological well-being, adaptation, and distress (Folkman, 1997; Lazarus, DeLongis, Folkman, & Gruen, 1985), more recently, intriguing evidence has emerged that psychological stress also predicts physical health. Stress has been associated with poor health in individuals across the lifespan and in many cultures (Lin & Ensel, 1989; Marmot & Wilkinson, 1999). This relationship is so robust and universal that it has been observed in infants, children, adolescents, young adults, adults, and the elderly (Graham, Christian, & Kiecolt-Glaser, 2006). As well, stress is a risk factor for a wide

variety of diseases, including cardiovascular disease, stroke, cancer, diabetes, asthma, ulcers, and rheumatoid arthritis (Cohen, Janicki-Deverts, & Miller, 2007; McEwen, 1998; Sandberg, et al., 2000). Taken together, previous clinical evidence indicates that psychological stress is associated with poor health.

However, this clinical evidence raises the question of what pathways can explain how psychological stress manifests into physical illness. In this general Introduction, we will provide an overview of previous research relevant to the constructs in this dissertation. First we will review different types of stress and how they relate to health outcomes. Second we will review the existing evidence on both psychological and biological pathways that may serve as pathways linking stress to health. Third, we will review research suggesting that there are moderators of the biological responses to stress, with a focus on psychosocial factors such as one's childhood environment. Finally, we will describe the rationale and aims and for the present study, which involves a daily diary study of the biological and psychological mediators of the relationship between daily stress and daily health symptoms, as well as tests of the moderating characteristics of these relationships. Following this general introduction, chapter 2 will report on an empirical study testing psychological and biological mediators of the relationship between daily stress and health. Chapter 3 will report on an empirical study testing the childhood psychosocial environment as a moderator of the relationship between stress and biology. Finally, we will conclude with a general discussion in chapter 4.

Conceptualizing Stress

Stressful events, termed 'stressors' (Selye, 1955), can be categorized according to at least two important dimensions, as described by Elliot and Eisdorfer (Elliot & Eisdorfer, 1982):

duration and course. Acute or 'episodic' stressors are short in duration and are time-limited, and include laboratory based stress tasks as well as brief stressful events that occur within the natural environment. Laboratory stress tasks include manipulations such as public speaking and mental arithmetic (i.e., the 'Trier Social Stress Task'; Kirschbaum, Pirke, & Hellhammer, 1993). Examples of naturally occurring acute stressors include a fight with a friend, being fired from a job, and getting into a car accident. In contrast, stressors that are long in duration and lack a known endpoint are termed chronic stressors. Examples of chronic stress include ongoing financial difficulty and caring for a terminally ill loved one.

To measure acute stressful events, stressful life event checklists have been developed in order to capture the number of events that occur within a certain amount of time (e.g., Holmes & Rahe, 1967). Many of these instruments are also designed to capture the occurrence of daily hassles, which are stressors that tend to occur frequently but are not considered as severe as a major life event, such as being stuck in traffic. Chronic stress is often measured by defining an ongoing role that places demands on an individual with no foreseeable ending, such as caring for a loved one with Alzheimer's disease, or a stable, pervasive stress that permeates an area of a person's life, such as chronic work strain.

Apart from measuring acute or chronic stress, stress can also be measured in terms of its subjective severity, referred to as 'perceived stress.' Perceived stress is a subjective account of how overloaded or overwhelmed a person feels in response to the stressors in their life. Some researchers have argued that the impact of stress will vary depending on an individual's subjective assessment of the experience (Ebrecht, Hextall, Kirtley, Taylor, Dyson, & Weinman, 2004). In the next section, we will provide an overview of evidence linking these different types of stressors to disease.

Acute/Episodic Stress

Acute, or 'episodic,' life events have been identified in previous studies as a risk factor for diseases including breast cancer (Lillberg, Verkasalo, Kaprio, Teppo, Helenius, & Koskenvuo, 2003), type 2 diabetes (Mooy, de Vries, Grootenhuus, Bouter, & Heine, 2000) and Grave's disease (Yoshiuchi, et al., 1998). For example, Lillberg and colleagues (2003) reported that, in a prospective cohort study of Finnish Twins, the number of life events over a five year period predicted an increased risk of breast cancer diagnosis 15 years later. As well, Mooy and colleagues (2000) reported that the number of stressful life events over a period of five years predicted diagnoses of diabetes at the end of the five year period in a sample of Caucasian adults, and Yoshiuchi and colleagues (1998) reported that stressful life events in the previous 12 months predicted increased risk of Grave's disease in women, but not in men. Acute stressful life events have also been cited to increase risk for overall mortality (Rosengren, Orth-Gomér, Wedel, & Wilhelmsen, 1993). In addition, episodic stressors have been reported to trigger asthma exacerbations (Sandberg, et al., 2000), as well as greater incidence of fatal and nonfatal myocardial infarction (Chi & Kloner, 2003; Rosengren, et al., 2004; Rozanski, Blumenthal, & Kaplan, 1999).

Some animal research has reported that experimental manipulations of acute stress are associated with increased risk of stroke in rats (Caso, Moro, Lorenzo, Lizasoain, & Leza, 2007; DeVries, et al., 1999; Sugo, Hurn, Morahan, Hattori, Traystman, & DeVries, 2002). Acute restraint stress has also been shown to increase risk for cardiac events in Apolipoprotein (Apo) E knockout mice (Huang, Pang, Letourneau, Boucher, & Theoharides, 2002). As well, acute stress in the form of repeated tail shocks was shown to increase the risk for the development of experimental allergic encephalomyelitis (EAE), an animal model of multiple sclerosis (Campbell, Meagher, Sieve, Scott, Storts, & Welsh, 2001).

Chronic Stress

Chronic stress also has been identified as a risk factor for a variety of diseases, including cardiovascular disease, asthma, infectious illness, and neoplastic diseases (Andersen, Kiecolt-Glaser, & Glaser, 1994; Chandola, et al., 2008; Cohen & Williamson, 1991; Karasek, Baker, Marxer, Ahlbom, & Theorell, 1981; Sandberg, et al., 2000). For example, in a prospective study that included participants from the Whitehall II study, Chandola and colleagues (2008) reported that individuals who experienced chronic work stress had a 68% greater risk of heart disease at the 14 year follow up than individuals who were not enduring stress. Chronic stress also increased children's risk for asthma exacerbations within 4 weeks of the stress assessment, according to results from a prospective study by Sandberg and colleagues (2000). In a prospective study of Swedish men by Karasek and colleagues (1981), chronic work stress, or 'job strain,' was associated with a 4-fold increase in risk of death related to cardiovascular disease during a six year follow up.

Animal research has employed experimental designs that are able to show that chronic stress is related to disease outcomes in a causal fashion. For example, mice that were placed under chronic stress and then exposed to ultraviolet light developed skin cancer twice as quickly as those mice not under chronic stress (Tausk & Nousari, 2001). As well, chronic stress, as induced by restraint stress or disruption of social hierarchies, has been related to 27% slower wound healing as compared to control mice (Padgett, Marucha, & Sheridan, 1998). Studies have also shown that social isolation in mice is related to elevated risk for liver disease (Hilakivi-Clarke & Dickson, 1995).

Daily Hassles

Daily hassles may also place individuals at risk for poor health. More specifically, daily hassles have been cited in the literature as a risk factor for a number of poor health outcomes,

including hypertension (Junlapeeya, Cy, & Junlapeeya, 2004), migraine headaches (Spierings, Sorbi, Haimowitz, & Tellegen, 1996), cardiovascular disease (Twisk, Boreham, Cran, Savage, Strain, & van Mechelen, 1999), declines in lung functioning in patients with asthma (Kullowatz, Rosenfield, Dahme, Magnussen, Kanniess, & Ritz, 2008), cervical cancer (Fang, et al., 2008) and blood pressure ‘non-dipping’ (less than 10% decrease in pressure from awake to asleep) (Sica, Wilson, & Ampey-Thornhill, 2007). Daily diary studies have indicated that daily hassles predict greater somatic symptoms, including the flu, sore throat, headaches, and backaches in a sample of healthy adults (DeLongis, Folkman, & Lazarus, 1988). Importantly, Weinberger and colleagues (Weinberger, Hiner, & Tierney, 1987) found that daily hassles predicted health status in a sample of adults with osteoarthritis over and above the effects of major life events.

Perceived Stress

In addition to the duration, course, and number of stressful events, perceptions of stress have also been identified as a risk factor for poor health. According to Ebrecht and colleagues (Ebrecht, Hextall, Kirtley, Taylor, Dyson, & Weinman, 2004), individuals with higher scores on the Perceived Stress Scale (PSS) two weeks prior to the lab visit exhibited slower wound healing after a 4mm-punch biopsy, indicating that high perceptions of stress may interfere with the body’s ability to heal itself. As well, according to Truelsen and colleagues, greater perceived stress almost doubled the risk of having a fatal stroke during the 13 year follow-up (Truelsen, Nielsen, Boysen, M, & Study, 2003). In addition, recent evidence has suggested that greater perceived stress within the past month at baseline was related to increased risk of cervical cancer diagnosis (Fang, et al., 2008) and breast cancer diagnosis (Helgesson, Cabrera, Lapidus, Bengtsson, & Lissner, 2003). As well, Jacobson and others (Jacobson, Aldana, Goetzel, Vardell, Adams, & Pietras, 1996) reported that higher

perceptions of stress were related to more work absenteeism due to illness across the year following the stress assessment.

In summary, previous studies have consistently reported psychological stress to be a risk factor for poor health. More specifically, stressors may trigger life threatening events, including asthma and heart attacks. Stress increases an individuals' risk for the onset and progression of a variety of chronic conditions, including hypertension, cardiovascular disease, and rheumatoid arthritis. Finally, perceived stress also places individuals at heightened risk for poor health outcomes. Given these robust associations, researchers have wondered why stress would be associated with disease. Next, we will review evidence from the literature on several psychological (coping and emotional responses to stress) and biological (cortisol secretion and nightly sleep) pathways that may link psychological stress to physical illness.

Psychological and Biological Pathways from Stress to Disease

In order to assess how psychological stress affects health, it is necessary to first understand the *psychological* consequences of stress. For example, identical stressors can have different effects on different individuals, so what is it that makes stress 'stressful' for one person but not another?

Psychological Pathways

While the stress response systems may have initially evolved to deal with physical threats to survival, such as escaping from a predator or wound healing post-attack, sources of stress have evolved over time to also include more psychological experiences. Such psychological experiences include emotional responses to stress, as well as coping response to stress.

Below, we review evidence for both emotional and coping responses as pathways linking stress and health.

Emotions

The impact of a stressor may be best understood by considering the effect it has on a person's emotions. For example, the experience of being stuck in a traffic jam may be very different for a person depending on whether or not they are or late for a meeting. Similarly, playing a piece on the piano may be soothing in private but stressful in public. Hence, a person's emotional response to an event can change the nature and severity of the stressor. Currently, emotion theorists define emotion as "complex coping-motivational-relational systems of evaluation and intention," (Lazarus, 1991). In other words, emotional responses to stress are borne out of an evaluation of the personal relevance of the situation and whether or not it affects one's motivations, or goals.

Stress and Emotion. Do all stressors elicit general negative emotional responses, or are certain types of stressors linked to specific emotional responses? Previous research has addressed this question, and while many different types of stressors seem to evoke negative emotions, particular characteristics of stress elicit stronger emotional responses.

Previous research has found that many types of stress induce a general array of negative emotions. For example, in a daily diary study, daily hassles explained a significant portion of the variance in negative mood, and negative mood spilled over into the next day particularly if the stressor was interpersonal in nature (Bolger, DeLongis, Kessler, & Schilling, 1989). As well, in a study by Zautra and colleagues (Zautra, Reich, Davis, Nicolson, & Potter, 2000), they found that, regardless of the source of stress (daily hassles, acute events, or experimentally induced stressors), stress led to greater experiences of negative emotions and

fewer reports of positive emotions. In addition, within a sample of adolescents, multiple stressful life events was a stronger predictor of negative affect than the experience of a single stressful life event (Larson & Ham, 1993).

However, some researchers have identified particular characteristics of stressors that appear to be most likely to evoke negative emotions. For example, stressors that are repeated, unpredictable, and novel appear to elicit the greatest increases in negative emotions. For example, in a daily diary study by van Eck and colleagues (van Eck, Nicolson, Berkhol, & Sulon, 1996), the experience of unpredictable, novel events over which individuals had little control were related to the greatest increases in negative emotion, whereas predictable, controllable events were related to smaller changes in negative emotion.

In addition, repeated exposure to uncontrollable events increases risk for negative emotions such as depression. Previous studies have shown that persistent and unmanageable stressors increase an individual's risk for depression (Miller & Blackwell, 2006). Within the animal literature, Seligman (Seligman & Beagley, 1975) found that repeatedly and unpredictably shocking rats led to behavioral helplessness, paralleling symptoms characteristic of depression. In another animal study, Willner (1997) exposed rats to a variety of chronic mild stressors over a period of three weeks. The animals lost interest in pleasurable things (another symptom of depression in humans) such as drinking sugar water.

Emotion and Health. In turn, mounting evidence suggests that negative emotional states are associated with disease onset and progression. In particular, in a review by Carney and Freedland (Carney & Freedland, 2003), they concluded that negative emotional states increase risk for cardiac death following a recent myocardial infarction. A review by Leserman (Leserman, 2003) also indicated that negative emotional states may be related to

faster HIV progression. As well, some evidence suggests that negative emotions elevate an individuals' cancer risk (Spiegel & Giese-Davis, 2003), and previous studies have indicated that a relationship exists between negative emotions and type 2 diabetes (Eaton, Armenian, Gallo, Pratt, & Ford, 1996).

Specific emotional responses to stress have also been linked to specific illnesses in past research. For example, it has been shown that depression doubles the risk of having a heart attack (Musselman, Evans, & Nemeroff, 1998) and increases reports of pain in women with rheumatoid arthritis (Zautra, Hamilton, Potter, & Smith, 1999). As well, feelings of hopelessness increase an individual's risk for developing hypertension (Everson, Kaplan, Goldberg, & Salonen, 2000). Previous studies have found that feelings of anger and hostility increase a person's risk of developing cardiovascular disease (Friedman, 1992; Kamarck & Jennings, 1991; Smith & Brown, 1991).

Both depression and anxiety have been shown to predict more severe health symptoms in patients with pre-existing conditions of hyper or hypothyroidism (Gulseren, Gulseren, Hekimsoy, Cetinay, Ozen, & Tokatlioglu, 2006). As well, both depression and anxiety were independent predictors of the development of hypertension in a community sample (Jonas, Franks, & Ingram, 1997). Symptoms of anxiety significantly predicted the incidence of cardiac events, as well as greater health care utilization post-myocardial infarction (Strik, Denollet, Lousberg, & Honig, 2003). In a sample of older individuals, anxiety predicted greater functional disability (de Beurs, Beekma, van Balkom, Deeg, van Dyck, & van Tilburg, 1999).

While negative emotions appear to be damaging for health, positive emotions may serve to buffer individuals from detrimental health outcomes. For example, Cohen and colleagues

(Cohen, Tyrrell, & Smith, 1991) exposed their participants to the rhinovirus and then tracked who contracted a cold. Participants who reported more everyday positive emotions were less likely to get the flu after exposure to the virus. Positive emotions may also buffer individuals from poor cardiovascular health. In a study by Affleck and colleagues (Affleck, Tennen, Croog, & Levine, 1987), they interviewed individuals post-myocardial infarction and then tracked these individuals for 8 years following the cardiac event. Those who were able to experience some positive emotion or identify a 'silver lining' as a result of their initial heart attack were less likely to experience another one as compared to individuals who did not express any positive emotion.

Positive emotions may also be related to longevity. More specifically, in a study by Danner and colleagues (Danner, Snowdon, & Friesen, 2001) they coded the autobiographies of nuns for positive and negative emotional content. They found that greater positive emotional content was associated with longevity, such that survival was 2.5 times greater for nuns whose autobiographies included the greatest percentage of positive emotions.

Overall, the experience of stress has been shown in previous literature to elicit negative emotional responses, with the greatest increases in negative affect occurring when the stressor is uncontrollable and unpredictable. In addition, past research has demonstrated that emotional states are related to health outcomes. More specifically, negative emotions, including depression and anxiety, predict poor health status. Finally, positive emotional states may be protective for health. Taken together, findings from previous studies indicate that negative emotions may represent an important pathway between stress and poor health.

Coping

The impact of stress on health may also depend on how an individual manages or ‘copes’ with the stressor. For example, if an individual is having academic problems but chooses to ignore them, the consequence is likely to be greater than if they were to recognize the problem and get extra tutoring earlier on. Conversely, if an individual attempts to manage a problem that is out of their control, as is the case with some terminal illnesses, they may experience greater stress than if they tried to avoid thinking about the illness and focused on other things, such as a hobby. Therefore, the ways in which an individual copes with stressors may modify the severity of the stress, and in turn, modify the impact that the stressor has on health.

Coping refers to attempts to adapt to a stressful situation, either by directly changing the situation or by minimizing one’s distress in response to the situation (Lazarus & Folkman, 1984; Rothbaum, Weisz, & Snyder, 1982). Two overarching categories of coping strategies have been cited in the literature to describe how individuals respond to stress: approach and avoidance coping strategies (Roth & Cohen, 1986). Approach coping methods, also termed problem-focused coping (Lazarus & Folkman, 1984) or primary coping (Rothbaum, Weisz, & Snyder, 1982), include efforts to change the stressor, while avoidance strategies, also termed emotion-focused coping (Lazarus & Folkman, 1984) or secondary coping (Rothbaum, Weisz, & Snyder, 1982), include efforts to ignore or withdraw from the stressor. Both are aimed at reducing the impact of the stressor, but these aims are achieved using different means.

Coping and Stress. Previous research has demonstrated that individuals are quite consistent in their selection of coping strategies in response to stressful events. In other

words, certain individuals tend to prefer approach or avoidance coping strategies across a variety of stressful situations (Cohen & Roth, 1984). However, evidence suggests that individuals are not exclusively ‘approachers’ or ‘avoiders’ and will often utilize a mix of both types of coping, depending on the demands of the stressor (Cohen & Roth, 1984; Manuel & Roth, 1984).

Some evidence exists to suggest that different types of stressors elicit different types of coping responses. For example, when stressors are uncontrollable or short-lived, people are more apt to utilize avoidance coping strategies (Lazarus, 1983; Mullen & Suls, 1982). In contrast, when stressors are longer in duration or perceived as controllable, people are more likely to employ approach coping strategies (Heckman et al., 2004; Keay & Bandler, 2001; Suls & Fletcher, 1985). Both types of coping have potential benefits and costs for health.

Coping and Health. In two recent meta-analyses, Penley and colleagues (Penley, Tomaka, & Wiebe, 2002), and Roesch and colleagues (Roesch, et al., 2005), found that the use of approach coping strategies was linked to better health than the use of avoidance coping strategies. For example, the use of approach coping strategies was related to improved mood and less pain in a sample of rheumatoid arthritis patients (Keefe, Affleck, Lefebvre, Starr, Caldwell, & Tennen, 1997), and better immune responses to pathogen exposure (Stowell, Kiecolt-Glaser, & Glaser, 2001).

In contrast, avoidance strategies can have detrimental effects on health, including greater viral load in HIV patients (Weaver, Llabre, Duran, Antoni, Ironson, & Penedo, 2005), greater reports of pain (Rosenberger, Ickovics, Epel, D'Entremont, & Jokl, 2004), and poorer recovery post-surgery (Stephens, Druley, & Zautra, 2002). As well, avoidance coping has been related to faster disease progression in cancer patients (Epping-Jordan, Compas, &

Howell, 1994), HIV patients (Leserman, 2003), and rheumatoid arthritis patients (Evers, Krassimaat, Geenen, Jacobs, & Bijlsma, 2003). In patients with rheumatoid arthritis, greater use of avoidance coping in response to stress has been found to increase functional disability (Morgan & Spiegel, 1987). Cross-sectional (Billings & Moos, 1981), longitudinal (Nowack, 1988), and daily diary (Freeman & Gil, 2004; Park, Armeli, & Tennen, 2004) studies have found that the use of avoidance coping strategies in response to daily life stressors predicted greater health symptoms.

In summary, despite the selection of consistent coping strategies across individuals, it appears that avoidance coping is more readily utilized when stressors are short-term and uncontrollable, whereas approach strategies are more likely to be utilized when stressors are longer in duration and are perceived as controllable. As well, previous findings indicate that avoidance coping is associated with poorer health outcomes, whereas the use of approach coping strategies may buffer an individual from the negative effects of stress on health. In other words, when individuals use avoidance coping strategies in response to a stressor, they may be more likely to experience health symptoms, disease progression, and disability than if they utilize approach coping strategies. Therefore, coping responses to stress may represent an important pathway linking stress to poor health.

Biological Pathways

Another approach to understanding stress-health relationships is to explore the *biological* sequelae of stressful experiences. In order for psychological stress to manifest into physical illness, there must be some biological processes that become dysregulated under conditions of stress. Over time, dysregulated biological processes may create vulnerability to disease. Below we review the literature on the relation between stress and two biological markers: cortisol and sleep. We focused on cortisol and sleep because dysregulations in both cortisol

secretion and sleep have been noted as markers of allostatic load (McEwen, 2007) and are risk factors for poor health (Cohen, Janicki-Deverts, & Miller, 2007; Dickerson & Kemeny, 2004; Meerlo, Koehl, van der Borght, & Turek, 2002). As well, both cortisol and sleep follow daily circadian patterns (Van Cauter, Polonsky, & Scheen, 1997). Therefore, stress may influence these daily biological patterns that, over time, could lead to poor health.

Cortisol

Cortisol is a glucocorticoid released by the hypothalamic-pituitary-adrenal (HPA) axis in a diurnal pattern, with highest cortisol levels secreted soon after wake-up, and lowest levels secreted after sleep onset (Dickerson & Kemeny, 2004). The HPA axis is a major part of the neuroendocrine system and is involved in the regulation of stress, digestion, immune functioning, mood, and energy. In response to stress, the hypothalamus, a gland in the brain located just above the brain stem, secretes the peptides vasopressin and corticotrophin-releasing hormone (CRH). Vasopressin and CRH stimulate the secretion of adrenocorticotrophic hormone (ACTH) by the pituitary gland, located below the hypothalamus. ACTH then activates the adrenal cortex to produce cortisol.

Greater amounts of cortisol are released by the HPA axis under conditions of stress in order to restore homeostasis to the body (Phillips, 2007). The role of cortisol is to increase blood pressure and blood sugar and to inhibit the functioning of the immune system in order to preserve energy for the ‘fight or flight’ response (Sapolsky, Romero, & Munch, 2000). While adaptive in response to short-term challenges, frequent or prolonged activation of the HPA axis can create vulnerability to illness.

Cortisol and Stress. Previous research has reported that a variety of different types of stressful experiences alter cortisol secretion. For example, acute psychological stressors that

involve social evaluation are known to increase cortisol levels (Dickerson & Kemeny, 2004), whereas chronic stressors have been found to initially increase cortisol secretion, but over time to result in blunted cortisol secretion (Heim, Ehlert, & Hellhammer, 2000; Miller, Chen, & Zhou, 2007; Sternberg, Chrousos, Wilder, & Gold, 1992; Yehuda, 2000). As well, some studies have demonstrated that cortisol levels increase only if the acute stressor occurs within the context of chronic stress (Marin, Martin, Blackwell, Stetler, & Miller, 2007).

Studies that have included daily measures of stressors have primarily reported greater daily cortisol secretion on high-stress days (Peeters, Nicholson, & Berkhof, 2003; Schlotz, Schulz, Hellhammer, Stone, & Hellhammer, 2006; Smyth, Ockenfels, Porter, Kirschbaum, Hellhammer, & Stone, 1998; van Eck, Nicolson, Berkhof, & Sulon, 1996). For an exception, see Hanson and colleagues (Hanson, Maas, Meijman, & Godaert, 2000). As well, some studies have reported that the experience of daily hassles is predictive of heightened cortisol secretion. For example, van Eck and colleagues (1996) found that, in a sample of healthy volunteers, reports of daily hassles were related to greater cortisol secretion, especially if the event was ongoing or novel. As well, Sondergaard and colleagues (Sondergaard & Theorell, 2003) reported that, in a sample of recent refugees, the perception of excessive demands in everyday life was related to greater cortisol secretion. According to these findings, the duration of stress, the frequency of stressful experiences, and the perceived impact of the stressor are all important predictors of the magnitude and direction of cortisol secretion.

Certain characteristics of stress may be most likely to increase cortisol. For example, in their meta-analysis, Miller and colleagues (Miller, Chen, & Zhou, 2007) reported that chronic stressors perceived as threatening, traumatic, or uncontrollable yielded the highest levels of cortisol secretion. Similarly, in their meta-analysis, Dickerson and Kemeny (Dickerson & Kemeny, 2004) found that uncontrollable acute stressors elicited an increase in cortisol that

was on average three times larger than that from controllable stressors. As well, if stressors were evaluated as threatening to an individual's 'social self,' or their core abilities/attributes, cortisol responses were higher than if a stressor was not a threat to a person's social self (Dickerson & Kemeny, 2004). Finally, past research has shown that stressors that are new and unfamiliar predict greater increases in cortisol than stressors that are familiar. For example, in a study by Schommer and colleagues (Schommer, Hellhammer, & Kirschbaum, 2003), participants displayed elevations in cortisol in response to an initial psychosocial stress task, but cortisol responses declined with repeated exposure, despite the trials being spaced 4 weeks apart.

Cortisol and Health. Higher cortisol output has been cited in previous research as a risk factor for a number of diseases, including cardiovascular disease and the metabolic syndrome (Bjorntorp & Rosmond, 1999; Smith, Ben-Shlomo, Beswick, Yarnell, Lightman, & Elwood, 2005). As well, dysregulated cortisol secretion may make individuals more susceptible to colds (Cohen, Frank, Doyle, Skoner, Rabin, & Gwaltney, 1998), have lower rates of cancer survival due to a reduced number of natural killer cells (Sephton, Sapolsky, Kraemer, & Spiegel, 2000), and have slower wound healing (Kiecolt-Glaser, Marucha, & Malarkey, 1995). Overall, previous findings demonstrate that the persistent activation of the HPA axis has negative consequences for health.

Sleep

Sleep has been observed in all mammals and is necessary for survival, as it provides a time for healing and growth. In particular, during sleep, growth hormone levels increase and cortisol levels decrease (Dahl & Lewin, 2002). Previous researchers have posited that sleep evolved to help animals avoid harm during dangerous periods of darkness, to allow the body to repair itself, and to allow time for the brain to consolidate information (Zeman & Reading,

2005). Therefore, sleep is an important factor that contributes to normal daily functioning and good health.

Sleep is a biological process that is regulated by the brain stem, thalamus, hypothalamic hormones, and external stimuli (i.e., light) (Dahl & Lewin, 2002; Hall, 1998). When the 'circadian clock' (located in the suprachiasmatic nucleus in the hypothalamus) releases the hormone melatonin, sleepiness ensues. Melatonin, therefore, determines the timing of restorative sleep episodes (Wyatt, Ritz-De Cecco, Czeisler, & Dijk, 1999). Sleep follows a cyclical pattern of non-rapid eye movement (NREM) and rapid eye movement (REM), with each stage lasting between 90 and 110 minutes (Wyatt, Ritz-De Cecco, Czeisler, & Dijk, 1999). NREM sleep is characterized by parasympathetic dominance and a lack of dreaming, whereas REM sleep is characterized by rapid eye movement, greater dreaming, and lower muscle tone (Rechtschaffen & Kales, 1968).

Sleep and Stress. Previous research has demonstrated that sleep disorders, including insomnia, hypersomnia, narcolepsy, and sleep apnea, are often a symptom of psychological stress (Kales, Soldatos, & Kales, 1987; Nixon & Pearn, 1977; Vgontzas & Kales, 1999). In addition, past research in both the human and animal literatures has reported that stress is related to changes in sleep architecture and duration. In humans, naturally occurring stressors, such as periods of marital separation, were reported to be associated with less slow wave, indicative of less restorative, sleep (as measured by polysomnography which enables the recording of brain wave activity, respiration, blood saturation, and muscle activity during sleep via electrodes placed along the scalp (Zeman & Reading, 2005)), in men whose marriages ended in divorce as compared to men who reconciled with their spouses (Cartwright & Wood, 1991). In contrast, Paulsen and Shaver (Paulsen & Shaver, 1991) reported that stressful life events were unrelated to sleep, as measured by polysomnography.

Other studies have reported that the effects of stress on sleep is apparent only in certain subgroups, such as in depressed individuals (Hall, Buysse, Dew, Prigerson, Kupfer, & Reynolds, 1997).

Chronic stress has been shown to be associated with poorer sleep in both humans and animals. More specifically, in healthy college students, chronic stress (operationalized as the 4 weeks of exam stress) was associated with a 20% reduction in sleep duration as compared to sleep during non-exam periods (Takase, et al., 2006). In contrast, Machado and colleagues (Machado, Hipolide, Benedito-Silva, & Tufik, 2004) reported that chronically stressed rats (who underwent foot shocks multiple times a day after a 96 hour period of sleep deprivation) displayed sleep “rebound,” or sleep that lasted 327.3% above baseline in the 72 hours following the foot shocks, as compared to the sleep debt only group.

Within the animal literature, Cheeta and colleagues (Cheeta, Ruigt, Proosdij, & Willner, 1997) found that chronic mild stressors in rats was related to greater REM onset latency, consistent with sleep patterns in depressed individuals. In a study using an intermittent foot shock on rats across 14 days, Kant and colleagues (Kant, et al., 1995) reported that, on the first day of stress, sleep duration and REM sleep decreased. However, after 7 days, both sleep duration and REM sleep returned to baseline, indicating that rats habituated to the foot shock stressor.

Daily diary studies have also demonstrated that daily stress is associated with poorer sleep. For example, several studies have reported that daily stress (Ancoli-Israel & Roth, 1999) or reports of daily stress related to financial strain (Hall et al., 2000) were associated with greater incidences of insomnia, sleep disruptions, sleep onset latency, and poorer sleep efficiency in elderly samples.

As well, previous studies have reported daily stress to be related to poorer *subjective* sleep quality. Tworoger and colleagues (Tworoger, Davis, Vitiello, Lentz, & McTiernan, 2005) found that perceived daily stress was related to poorer subjective sleep quality, but not actigraphic sleep. (Actigraphy is a method by which the individual wears an accelerometer on their wrist to measure gross motor activity.) In addition, Urponen and colleagues (Urponen, Vuori, Hasan, & Partinen, 1988) reported that, in an epidemiological study conducted on Finish adults, self-reported stress was associated with greater self-reported sleep problems. Knudsen and colleagues (Knudsen, Ducharme, & Roman, 2007) indicated that job stress was related to reports of difficulty falling asleep, maintaining sleep, and trouble waking up. Finally, in some studies, the relationship between stress and sleep was apparent only in certain subgroups, such as in individuals diagnosed with post traumatic stress disorder (Dagan, Zinger, & Lavie, 1997; Pillar, Malhotra, & Lavie, 2000), or among those who use certain types of coping strategies (Sadeh, Keinan, & Daon, 2004).

Sleep and Health. Past research has reported that sleep is associated with health symptoms, diseases, and overall mortality. According to the National Sleep Foundation, adults need between 7 and 9 hours of sleep per night for optimal overall health. Long-term sleep deprivation has been linked to changes in immune functioning, medical, and psychiatric disorders (Zeman & Reading, 2005). Sleep problems have been identified as risk factors for diseases including cardiovascular diseases and psychiatric difficulties (Breslau, Roth, Rosenthal, & Andreski, 1996; Newman, et al., 2001). As well, research has shown that poor sleep is a risk factor for compromised immune functioning and disease in animals (Ironson, et al., 1997; Krueger, et al., 1994).

Sleep duration has been related to health in past research, and the majority of studies have reported that shorter time asleep is detrimental to health. In a study by Taske and colleagues

(Takase, et al., 2006), they found sleep deprivation in healthy college students was related to decreased endothelial function and intra-cellular magnesium levels, indicative of impaired cardiovascular functioning. As well, shorter sleep duration was related to higher body mass index scores in healthy adolescents (Horne, 2008; Young, 2008).

Conversely, *longer* sleep duration doubled the risk from dying from cardiovascular disease, according to Ferrie and colleagues (Ferrie, et al., 2007), but the authors suggested that these findings may have been confounded by the longer sleep intervals in depressed individuals who are also at greater risk of cardiovascular death. As well, within the animal literature, shorter sleep duration (e.g., sleep deprivation by repeated immersion in cold water) in rats was related to *longer* survival to high doses of amphetamine as compared to non-sleep deprived rats (Stern & Hartmann, 1972). Hence, these studies indicate that longer sleep times may not always be beneficial for good health.

In addition to sleep quantity, previous research has examined the relation between the quality of sleep and health. Sleep quality can be measured using self-report scales, such as the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), or by using objective tools, including actigraphy or polysomnography. Studies using self-report measures have found that poorer sleep quality was related to poorer health measures, including self-reported health status in a sample of healthy college students (Pilcher, Ginter, & Sadowsky, 1997), and health-related quality of life in haemodialysis patients (Iliescu, et al., 2003). As well, Cohen and colleagues (Cohen, Doyle, Alper, Janicki-Deverts, & Turner, 2009) found that self-reported sleep efficiency was related to the production of antibodies after experimentally exposing participants to the rhinovirus vaccine. More specifically, when participants' sleep was less than 92% efficient, they were 5.5 times more likely to get a cold than participants whose sleep was 98% efficient. Using actigraphy, Gupta and colleagues

(Gupta, Mueller, Chan, & Meininger, 2002) found that poorer sleep efficiency was related to higher BMI scores in healthy adolescents. They concluded that poor sleep quality represents a risk factor for the development of obesity. Poorer sleep efficiency (sleeping < 85% during the sleep interval), as measured by polysomnography, predicted pre-hypertension in a sample of healthy adolescents (Javaheri, Storfer-Isser, Rosen, & Redline, 2008).

In summary, previous research has shown that psychological stress induces changes in biological processes including cortisol secretion and sleep. However, there are some inconsistencies in the patterns documented in this research, and some evidence suggests that the stress-biology association may depend on moderating factors such as the duration of the stress, the characteristics of the stressor, or other background factors. In particular, some previous research has suggested that one important background moderating factor in the relation between stress and the biological processes of cortisol and sleep may be the psychosocial characteristics of one's childhood environment. In other words, the social context in which biological systems develop may shape future biological responses to stress. Next we will review the literature that has examined the moderating influence of the childhood environment on the stress-biology association.

Biological Pathways: The moderating role of the childhood environment

Biological responses to stress may not be uniform across individuals, and individual differences in biological stress responses may provide one explanation as to why stress can lead to disease in some people but not others (Miller, Chen, & Cole, 2009). Previous research suggests that characteristics of the childhood social environment may partially explain differences in biological responses to stress in adults (Miller & Chen, 2007; Repetti, Taylor, & Seeman, 2002).

In attempting to understand how it is that a social experience early in life could come to affect physical health decades later, some researchers have proposed that early life represents a critical period during which a person's biological response tendencies develop, and that once formed, they persist across the lifespan. These systems are still developing in the first few years of life, and it is believed that during this period these systems are particularly vulnerable to the effects of stress (Davidson, Jackson, & Kalin, 2000). More specifically, psychosocial challenges during the first few years of life have been argued to 'program,' or calibrate, the biological systems that respond to stress, and to do so in a way that persists throughout the lifetime (Boyce & Ellis, 2005). Both the patterns of cortisol secretion and sleep are established early in life (Phillips & Jones, 2006; Rivkees, 2003), and hence form plausible targets that may be influenced by childhood family environments.

Cortisol and Stress: The Moderating Role of Childhood Environments

The quality of family relationships has been proposed to be an important psychosocial factor that impacts the magnitude of cortisol change under conditions of stress. Previous studies have found that difficult childhood environments predict increased cortisol response post-task in adults (Luecken, 1998; Repetti, Taylor, & Seeman, 2002), although some studies report hypo-responsiveness to a lab stressor (Carpenter, et al., 2007). According to Taylor and colleagues (Taylor, Lerner, Sage, Lehman, & Seeman, 2004), children who experience insecure attachments to caregivers, harsh parenting, or lack of social support exhibited greater cortisol secretion in response to an acute laboratory stressor in adulthood. In addition, Taylor and Seeman (Taylor et al., 1999) reported that young adults (aged 18-25) who characterized their early family environments as conflictual or cold had higher cortisol responses to stress tasks than adults who characterized their childhood environments as warm and nurturing. Early experiences of social deprivation, such as when a child experiences

distant or cold parental relationships (Heim, et al., 2000), or when a child spends their first years of life in a Romanian orphanage (Rutter & O'Connor, 2004), have also been shown to result in heightened cortisol reactivity to stress in adulthood.

Past studies have also examined the impact of early life maternal bonding on the stress response. In particular, studies have shown early life maternal-infant separation to be associated with increased activation of the HPA response to stress (Hennessy, 1997). As well, Lupien and others (Lupien, King, Meaney, & McEwen, 2000) reported that adults who were raised by depressed mothers had higher morning cortisol secretion. Conversely, Evans and colleagues (Evans, Kim, Ting, Teshler, & Shannis, 2007) found that youths' perception of maternal responsiveness (i.e., supportive, nurturing maternal behavior) buffered the effects of stress on daily HPA functioning.

Work in the animal literature has found that non-human primates raised in stressful childhood environments (e.g, intermittent food availability) displayed amplified cortisol responses to laboratory stressors as adults (Gorman, Mathew, & Coplan, 2002; Hennessy, 1997; Rosenblum, Forger, Noland, Trost, & Coplan, 2001). Conversely, rats raised in nurturing environments (e.g., with mothers who spend more time licking) display more modest HPA responses to restraint stress as adults (Caldji, Diorio, & Meaney, 2000; Meaney, 2001).

Stress and Sleep: The Moderating Role of Childhood Environments

Patterns of sleep are established early in life (Phillips & Jones, 2006; Rivkees, 2003) and some evidence indicates that the development of sleep patterns, including sleep disruptions under conditions of stress, may be susceptible to the effects of the psychosocial environment during childhood. Studies investigating childhood adversity have found associations with

sleep, using both human and animal models. For example, in a longitudinal study by Gregory and colleagues (Gregory, Caspi, Moffitt, & Poulton, 2006), they found that greater family conflict in childhood predicted symptoms of insomnia at age 18, over and above the current psychosocial environment. In contrast to childhood adversity, childhood environments characterized by parental warmth have been shown to be predictive of earlier bedtimes and longer sleep times in a nationally representative sample of children and adolescents by Adam and colleagues (Adam, Snell, & Pendry, 2007).

Similarly, animal studies have shown that primates separated from their mothers displayed sleep disturbances including a greater number of arousals and decreased REM sleep (Reite & Snyder, 1982). As well, Tiba and colleagues (Tiba, Tufik, & Suchecki, 2004) found that rats who experienced maternal separation have permanent changes in sleep patterns, such that they exhibited less rapid eye movement (REM) sleep than rats who were not separated from their mothers. Taken together, findings from previous research suggest that early life experiences may influence the regulation of the sleep-wake cycle during childhood, in turn impacting sleep in adulthood.

The Present Study

The present study had two aims: 1) to assess the psychological and biological pathways linking daily stress to health; and 2) to assess the moderating influence of childhood psychosocial environments on stress-biology relationships. As we reviewed earlier, stress is a risk factor for poor health, and clear links exist between stress and psychological and biological changes. As well, changes in psychological and biological patterns have been linked to poor health. While previous work has provided preliminary evidence for these links, this work has been limited in its assessment of how stress, psychological and biological mediators, and health fluctuate on a day-to-day basis, as well as how these relationships

operate within healthy populations. Therefore, we aimed to address these limitations by conducting a daily diary study within a sample of healthy undergraduate students.

In addition, because previous research has indicated that biological changes under conditions of stress are not uniform across individuals, we explored one possible psychosocial moderator of this relationship. Variability in biological responses may be partially explained by the childhood environment in which the individual was raised. The HPA axis and sleep patterns develop early in life, therefore, in this study, we aimed to determine whether psychosocial characteristics of the childhood environment moderated the day-to-day associations between stress and cortisol secretion or nightly sleep.

Daily diary methodologies offer several advantages that complement experimental designs. First, the ecological validity of measuring stress within the context of individuals' lives enhances our understanding of how stress, health, and the biological and psychological pathways under examination relate within natural settings. In addition, an investigation of day-to-day experiences of stress and health symptoms allows researchers to better understand how, within the same individual, different types of experiences from one day to the next may lead to different types of psychological and biological responses with implications for health.

As well, daily stressors, or 'hassles', may create more vulnerability for poor health than less frequent episodic events. While acute events and daily hassles both appear to increase an individuals' risk for disease, some findings suggest that daily hassles are more potent predictors of poor health (Weinberger, Hiner, & Tierney, 1987). More specifically, Weinberger and colleagues found that, in a sample of adults with osteoarthritis, hassles were better predictors of health status than major life events. As well, they found that major events impacted health via changes in daily hassles. Similarly, Twisk and colleagues found that, in a

sample of healthy adults, daily hassles were a stronger predictor of lipoprotein levels, a precursor to cardiovascular disease, than major life events (Twisk, Boreham, Cran, Savage, Strain, & van Mechelen, 1999). Therefore, in this study we will measure stress in terms of daily hassles.

Hypotheses

In this study, we hypothesize that psychological responses to stressful events will be a significant pathway linking daily hassles to daily health symptoms. More specifically, we hypothesize that on days when individuals experience more severe stressors, they will also report greater health symptoms, and that this relationship will be partially accounted for by greater cortisol secretion, poorer sleep, greater use of avoidance coping, less approach coping, and more negative emotional responses.

In addition, we hypothesize that changes in biological patterns will serve as a significant pathway linking stress to health, but only in those individuals who are vulnerable to the effects of stress. More specifically, based on previous research, we hypothesize that among individuals who experienced difficult early life environments, on days when they experience greater stress, they will also display greater cortisol output and poorer sleep. In contrast, among those who experienced nurturing early life environments, cortisol output and sleep patterns will not vary as a function of daily stress experiences.

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Daily Stress and Health Symptoms in Healthy Young Adults: The Role of Biological and Psychological Mediators¹

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Daily Stress and Health Symptoms in Healthy Young Adults: The Role of Biological and Psychological Mediators

The relationship between stress and health has been well-established for many years (Cohen & Herbert, 1996, Segerstrom & Miller, 2004; McEwen, 2007). In particular, greater psychological stress has been linked to increased risk for cardiovascular disease, cervical cancer, autoimmune disorders, infectious diseases, and mental illnesses (Fang, et al., 2008; McEwen, 1998). Studies have also shown that stress is related to health symptoms, including pain (Affleck, Tennen, Urrows, & Higgins, 1994), fatigue (Schanberg, et al., 2000), and other somatic symptoms (Walker, Garber, Smith, Van Slyke, & Claar, 2001).

Given the robust link between stress and health, researchers have sought to determine the pathways through which stress exerts its affect on health. In particular, previous research has reported that both biological (McEwen, 1998; Miller, Chen, & Cole, 2009) and psychological responses to stress (DeLongis, Folkman, & Lazarus, 1988) are important intermediary factors in the stress-health association. For example, researchers have studied how stress disrupts the normal functioning of physiological systems, and how these disruptions impact disease states (Cole, Kemeny, Fahey, Zack, & Naliboff, 2003; Krantz & McCeney, 2002; Manuck, 1995; McEwen, 1998; Porges, 1995; Rozanski, Blumenthal, & Kaplan, 1999). As well, researchers have investigated how psychological resources such as coping can account for the relation between stress and health (Mahat, 1997; Manne & Zautra, 1992).

In the present study, we explored mediators of the relationship between daily stress and daily health symptoms. We chose to focus on daily processes in part because there is some evidence that daily stressors are better predictors of health than major life events (Weinberger, Hiner, & Tierney, 1987). In addition, an investigation of day-to-day

experiences of stress and health symptoms allows researchers to better understand how, within the same individual, different types of experiences from one day to the next may lead to different types of psychological and biological responses with implications for health.

Biological mediators in the stress-health link

During times of acute stress, individuals exhibit a heightened activation of neural, neuroendocrine, and immune mechanisms in order to prepare the body to overcome or to avoid danger. While activation of these mechanisms are adaptive in the short run, over time, it is thought that repeated activation of these systems can cause wear and tear on the body, referred to as allostatic load (McEwen, 1998). Allostatic load has been reported as a risk factor for numerous health problems, including obesity and cardiovascular disease, due to dysregulated stress hormone reactivity (McEwen, 2007).

In this study, we focused on cortisol as one potential biological intermediary of the daily stress and health relationship. Cortisol is a hormone controlled by the hypothalamic-pituitary adrenal (HPA) axis and released under conditions of stress (Phillips & Jones, 2006). In addition, cortisol is secreted in a diurnal pattern across the day (Van Cauter, Polonsky, & Scheen, 1997) enabling us to assess whether events during the day are related to altered cortisol patterns. Over time, alterations in daily circadian patterns may result in permanent changes in biological stress response systems, which may then increase an individuals' risk for the development of illness (Meerlo, Koehl, van der Borght, & Turek, 2002).

Cortisol has been implicated in a number of health problems. High levels of cortisol increase risk for cardiovascular disease and the metabolic syndrome (Bjorntorp & Rosmond, 1999; Smith, Ben-Shlomo, Beswick, Yarnell, Lightman, & Elwood, 2005). As well, individuals are more susceptible to colds (Cohen, Frank, Doyle, Skoner, Rabin, & Gwaltney,

1998), less likely to survive cancer (Sephton, Sapolsky, Kraemer, & Spiegel, 2000), and to have slower wound healing (Kiecolt-Glaser, Marucha, Malarkey, Mercado, & Glaser, 1995).

Cortisol has been proposed as one likely mediator in the pathway from stress to disease (Miller, Chen, & Cole, 2009). However, the empirical evidence linking cortisol to stress and health in daily diary studies has been mixed. For example, one study demonstrated that flatter cortisol rhythms mediated the association between daily stress and daily reports of health symptoms and fatigue in a sample of older adults (Adam, Hawkley, Kudielka, & Cacioppo, 2006). In addition, studies have shown that cortisol responses to perceived work stressors predicted anthropometric, endocrine, and metabolic markers associated with increased risk of cardiovascular disease in a sample of older men (Rosmond, Dallman, & Björntorp, 1998). However, other studies did not find cortisol to mediate the relationship between daily stress and immune responses to the influenza vaccine (Miller, Cohen, Pressman, Barkin, Rabin, & Treanor, 2004).

Laboratory based studies have also found evidence that cortisol reactivity to stress is different in patient populations. For example, in a study by Crofford and colleagues (Crofford, et al., 1994), individuals with fibromyalgia displayed cortisol hyporesponsiveness to stress as compared to normal controls. Similarly, Buske-Kirshbaum and colleagues (Buske-Kirschbaum, et al., 1997) found attenuated cortisol responses to a psychosocial stress task in children with atopic dermatitis as compared to healthy controls.

Hence previous research has found mixed support for the role of cortisol in the stress-health relationship. As well, previous studies of daily stress and cortisol have primarily focused on older adults, leaving the role of cortisol as a mediator in the stress-health association within young, healthy samples unclear. In addition, previous studies measured

stress within specific life domains, such as work-related stress, and it is unclear whether cortisol serves to mediate the stress-health association across all categories of life stress. To address these limitations, in this study we assessed daily cortisol secretion as a mediator in the daily stress-health symptom relation within a sample of healthy young adults. As well, participants were queried about their stress experiences across a variety of domains.

Psychological mediators in the stress-health link

Stress may also be related to health because of the psychological consequences of experiencing stress, which in turn exacerbate health problems. Two constructs that may be important intermediaries in the association between stress and health include the emotional and coping responses to stress (Taylor, 1999; Taylor, Lerner, Sage, Lehman, & Seeman, 2004).

Coping. Coping refers to attempts to adapt to a stressful situation, either by directly changing the situation or by minimizing one's distress in response to the situation (Lazarus & Folkman, 1984; Rothbaum, Weisz, & Snyder, 1982). Two overarching categories of coping strategies have been cited in the literature to describe how individuals respond to stress: approach and avoidance coping strategies (Roth & Cohen, 1986). Approach coping methods include efforts to change the stressor, while avoidance strategies include efforts to ignore or withdraw from the stressor. Effective coping will serve to mitigate the negative effects of stress, whereas ineffective coping strategies will exacerbate or prolong the effects. Therefore, how a person copes with stress may affect the impact of the stressor, and in turn, health consequences.

In research that has considered the role of coping as a psychological mediator in the association between stress and health, most studies have concluded that the use of avoidance

coping in response to stress leads to poor health outcomes. Cross-sectional (Billings & Moos, 1981), longitudinal (Nowack, 1988), and daily diary (Freeman & Gil, 2004; Park, Armeli, & Tennen, 2004) studies found that the use of avoidance coping strategies in response to daily life stressors predicted greater health symptoms and more health-compromising behaviors. More specifically, daily diary studies by Park and colleagues (2004) and Freeman and Gil (2004) reported that the use of avoidance coping in response to stress predicted greater alcohol consumption and binge eating behaviors, respectively. In addition, studies that considered retrospective recall of negative life events (Billings & Moos, 1981) and daily irritants (Nowack, 1988) found that the use of avoidance coping mediated the relation between stress and self-reported health symptoms. Finally, in patients with rheumatoid arthritis, greater use of avoidance coping in response to stress has been found to increase functional disability (Morgan & Spiegel, 1987).

The use of approach coping strategies may also mediate the stress-health association. For example, Billings and Moos (1981) found that the use of approach coping strategies attenuated the impact of stress on self-reported health symptoms. As well, Morgan and Spiegel found that the use of approach strategies decreased reports of pain in rheumatoid arthritis patients (Morgan & Spiegel, 1987). However, there are some studies that have not found approach or avoidance coping to be a mediator of the stress-health relation. For example, coping did not mediate the relation between daily stressors and health symptoms in individuals with rheumatoid arthritis (Folkman, Lazarus, Gruen, & DeLongis, 1986). Similarly, Nowack (1989) reported that coping did not moderate the relation between work-stress and health status in a sample of professional employees.

In summary, coping is one potential intermediary process linking psychological stress to physical health, and the choice of approach or avoidance coping strategy may attenuate or

exacerbate the negative impact of stress. Findings from the literature indicate that avoidance coping is associated with poorer health outcomes, and when individuals use avoidance coping strategies in response to a stressor, they are more likely to experience health symptoms, disease progression, and disability.

However, of the studies that have considered how coping mediates the stress-health association, most asked participants to retrospectively recall coping responses to stressors that occurred weeks or months prior to the study, or coping was assessed solely within patient populations. These findings, therefore, may not be valid or generalizable to healthy populations. More research is needed to assess how day-to-day coping mediates the relation between *daily* stress and health symptoms in individuals who are healthy in order to determine whether coping responses to stress can be linked to health symptoms more generally.

Emotion. Emotions refer to subjective experiences related to reactions to stimuli, cognitive appraisals of bodily sensations, and motivations and intentions, and which can motivate behaviors (James, 1922; Tompkins, 1984). Previous studies have documented links between negative emotions and health. For example, negative emotional responses to stress increase individuals' risk for cardiac death following a myocardial infarction (Carney & Freedland, 2003), cancer (Spiegel & Giese-Davis, 2003), HIV progression (Leserman, 2003), and type 2 diabetes (Eaton, Armenian, Gallo, Pratt, & Ford, 1996).

A limited number of previous studies have tested whether emotional responses to stress are important mediators in the relation between stress and health. Wiebe (Wiebe, 1991) reported that participants who responded to an evaluative threat task with more positive and less negative affect displayed lower heart rate reactivity to the task than individuals who

responded with less positive and more negative affect. In a series of daily diary studies by Stone and colleagues (Stone, Cox, Valdimarsdottir, Jandorf, & Neale, 1987; Stone, Neale, Cox, Napoli, Valdimarsdottir, & Kennedy-Moore, 1994; Stone, 1984), they found that emotion mediated the relation between undesirable daily events and immunoglobulin A, an antibody that defends the body from the common cold. As well, Cohen and colleagues (Cohen, Frank, Doyle, Skoner, Rabin, & Gwaltney, 1998) exposed their participants to the rhinovirus and then tracked who contracted the cold. Participants who reported more positive emotions were less likely to get the flu after exposure to the virus. In other words, positive emotional responses may enhance the immune response, while negative emotional responses may hinder the immune response. As well, when mood was manipulated by a laboratory task (Labott, Ahleman, Wolever, & Martin, 1990), positive emotional states were related to activation of immune responses (beneficial to health), whereas negative emotional states were associated with suppressed immune activity. Therefore, previous research has shown that negative emotional responses to stress have detrimental effects on health, and that emotional responses to stress may serve to mediate the stress-health association. However, studies are still needed that assess whether emotional responses mediate the relation between daily life stress and health symptoms.

In summary, while previous research has provided evidence on the importance of biological and psychological mediators in the stress-health association, our understanding is limited by studies that rely on retrospective recall of stress or coping over long periods of time, that have focused only on patient populations, or that have assessed biomarkers but not broader health effects, such as symptoms. This study attempts to address limitations of the existing literature by utilizing a daily diary methodology, by studying daily stress and health within a healthy sample, and by focusing on health symptoms.

Hypotheses

In this study, we hypothesize that more severe daily stress will predict greater daily health symptoms. In addition, we expect that, on days when participants report greater stress severity, they will also display greater daily cortisol secretion, more negative emotions, and more avoidance coping. As well, we hypothesize that, on days when individuals display greater daily cortisol secretion, greater use of avoidance coping, and more negative emotions, they will also exhibit greater daily health symptoms. Finally, we hypothesize that the relation between daily stress severity and daily health symptoms will be mediated biologically by greater cortisol secretion and psychologically by greater use of avoidance coping, and more negative emotional responses.

Method

Participants

87 healthy college students participated in the current study. They were recruited from the University of British Columbia in Vancouver, BC Canada through campus postings. Students were eligible to participate in the study if they 1) were between the ages of 19 and 25, 2) were medically healthy, and 3) were fluent in English. Eligible participants were scheduled for a laboratory visit. The study sample was about 67% female, and was 28% Caucasian, 57% Asian, and 15% 'other.' Sample descriptive statistics are reported in Table 2.1.

Measures

Daily Stressors

Participants reported the severity of the most bothersome stressor of the day using a web-based diary format. Once an evening throughout the seven day monitoring period, participants were asked to check off which of a list of sixteen items they had experienced within the last 24 hours. Items included achievement stressors, interpersonal stressors, and daily hassles. Participants were then asked to select the most severe stressor experienced during the day and respond to the question, “How serious was this for you?” Responses ranged from 1 to 5, with 1 equaling ‘none’ and 5 equaling ‘a lot.’ This measure was a modified version of the Hassles Scale (from the Hassles and Uplifts Scales; DeLongis, Folkman, & Lazarus, 1988) and was developed by Lee-Baggley and DeLongis for use in a university sample (2007, unpublished data). Previous research has demonstrated significant relations between the Hassles Scale and other life event scales (Holmes & Rahe, 1967), psychological symptoms scales (Derogatis, Lipman, Covi, Rickels, & Uhlenhuth, 1970; Derogatis, Lipman, Covi, & Rickels, 1971; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974), and health symptom checklists (DeLongis, Folkman, & Lazarus, 1988). Although one can also calculate the total number of daily stressors that occurred, in the present study, only the severity rating for the worst stressor of the day was used due to the fact that the coping and emotion questions were asked only with respect to the most severe stressor of the day.

Coping Response

Within the web-based diary, participants were also asked to respond on a 3-point scale (1=not at all, 2=some, 3=a lot) the degree to which they used several coping strategies in response to the most bothersome daily stressor within the past 24 hours. Coping was assessed

via 24 items from the Brief Ways of Coping scale, modified for use in daily diary studies (Lee-Baggley, Preece, & DeLongis, 2005). This scale has been shown to display adequate internal consistency, construct, and concurrent validity (Vitaliano, Russo, & Carr, 1985). Coping responses clustered into two over-arching coping strategies: approach and avoidance coping. An example of an approach item is, “concentrated on what I had to do next to solve the problem,” while an example an avoidance coping item is, “wished the situation would go away or somehow be over with.” Higher scores indicate greater use of each coping strategy.

Emotional Response

Also within the daily web-based diary, participants were asked to report their emotional response to the most bothersome daily stressor using a measure adapted from Larsen’s emotion circumplex (Larsen & Diener, 1992). Participants indicated the degree that they felt each of eight negative emotions in response to the stressor on a 5-point likert scale, ranging from 1=not at all, to 5=extremely. Emotions were also categorized into two groups based on arousal: tense, nervous, stressed, upset were classified as activated negative emotions; sad, depressed, lethargic, fatigued were classified as deactivated negative emotions (Larsen & Diener, 1992). This measure has been used previously in daily diary studies of university students (Park, Armeli, & Tennen, 2004), and has displayed adequate convergent validity with peer ratings, as well as acceptable discriminant validity (Watson & Clark, 1991).

Cortisol

Participants collected salivary cortisol samples using Salivettes (Sarstedt, Nuembrecht, Germany). Samples were collected four times per day at one, four, nine, and eleven hours after awakening over five consecutive days following the lab visit to capture the total daily output of cortisol secretion. To determine whether participants were compliant with the

sampling schedule, Salivettes were stored in a bottle sealed by a MEMS 6 TrackCap Monitor (Medication Event Monitoring System, Aardex Ltd., Switzerland). Caps record the date and time of each opening. 86.6% of samples were completed, and 88.1% of completed saliva samples were completed within 1 hour of the scheduled collection time. Saliva samples were returned to the lab and then centrifuged at 1000g for 5 min, transferred to deep-well plates, and stored at -30°C until assayed. Free cortisol levels in saliva were measured in duplicates using a commercially available chemiluminescence assay (IBL, Hamburg, Germany).

In order to assess total cortisol secretion throughout the day, data were first log transformed to reduce substantial skewness. Next, daily cortisol output was calculated via an area-under-the-curve (AUC) statistic using the trapezoidal rule.

Health Symptoms

Participants reported all symptoms they had or had not experienced (in a 'yes/no' format) over the past 24 hours within the web-based daily diary. The list of symptoms is comprised of 15 items and is based on the PILL (Pennebaker Inventory of Liquid Languidness; Pennebaker, 1982). Items included symptoms such as headache, muscle soreness, poor appetite, sore throat, and upset stomach. This measure has demonstrated adequate internal consistency (Cronbach α range from .88 to .91) and has been used previously in college samples. As well, this measure correlates with health center visits, ibuprofen use, and work absences (Spruijt-Metz, 1999). The total number of symptoms reported on each day was calculated.

Procedure

Participants came to the lab and signed consent forms. During their initial lab visit, participants provided demographic information. As well, participants were given instructions

on how to complete web-based surveys and how to collect salivary cortisol samples.

Schedules for saliva sampling were set for the five days following the lab visit.

For the 7 days following the lab visit, subjects completed a web-based diary entry at the end of each day. Specifically, subjects were asked about the stressful events they experienced in the past 24 hours, their coping responses, and their emotional responses. As well, participants indicated whether they experienced any health symptoms via a symptom checklist (Larsen & Kasimatis, 1991).

To increase compliance, study participants received a daily reminder email with a link to the web-based survey, and those who did not complete the entry the previous day were phoned by the study coordinator. Participants completed an average of 93.9% of daily diary entries, out of 7 possible days. Three participants completed diaries on paper because they did not have daily access to computers. If participants completed an entry within 24 hours of the intended time, their data were included. If diaries were completed more than one day late, data were excluded from analyses. Participants received \$1.00 per diary entry completed on the appropriate day. Finally, participants received \$10 dollars at the initial lab visit, and \$10 for returning their equipment at the end of the study.

Statistical Analyses

Data were analyzed using hierarchical linear modeling techniques (HLM). This method of statistical analysis enabled us to test the within-person relationships between daily stress, biological and psychological responses to stress, and health symptoms. Stress, possible psychological mediators (coping, emotion), biological mediator (cortisol), and health symptoms were each modeled as within-person factors because they were collected daily.

First, we conducted a series of within-person (level-1) models to determine whether there was a main effect of daily stress predicting daily health symptoms. Level-1 models generate a set of slopes for each individual that reflect variations in health symptoms as a function of daily stress.

Second, in order to determine whether daily biological or psychological factors mediated the relation between daily stress and health symptoms, it was necessary to test the direct pathways between daily stress and the biological/psychological mediators, as well as the direct path between biological/psychological mediators and daily health symptoms. In order to do this, we conducted a series of level-1 models in order to test whether daily stress significantly predicted daily biological and psychological responses to stress. Next, we conducted a series of level-1 models to assess whether the biological/psychological variables predicted daily health symptoms.

Third, for those pathways from daily stress to biological/psychological responses, and from biological/psychological responses to health symptoms that were significant, we ran a series of level-1 models to determine whether the relation between daily stress and health symptoms was mediated by biological or psychological pathways. To do this we simultaneously entered mediator variables together with daily stress to predict daily health symptoms. We used full maximum likelihood and robust standard errors to estimate all models. For all analyses, level-1 predictors were centered around the mean of each individuals' average score across the study. This allowed us to test whether health symptoms differed, for example, when an individual experienced more or less daily stress than their average. As well, random intercepts were modeled in all analyses.

Results

Descriptive Data and Preliminary Analyses

Means, standard deviations, and ranges for all predictor and outcome variables are presented in Table 2.2. Participants reported their most severe stressor to be ‘somewhat’ serious. Participants also reported about 2.4 out of 15 possible health symptoms per day. Across the monitoring period, participants reported experiencing no health symptoms between 12 and 26% of the time.

Before testing our hypotheses, we ran a series of preliminary analyses in order to determine whether protocol compliance or demographic information was associated with any of our predictor or outcome variables, including stress, negative emotions, coping, cortisol, or health symptoms. Below we present results from these preliminary analyses.

Compliance. Overall, we found that compliance was not significantly related to any of our daily diary variables. Out of a possible 609 entries, a total of 572 daily diary entries were completed (93.9% compliance), or an average of 6.6 out of 7 days of daily diary entries per person. As well, out of a possible 1740 salivary cortisol samples, a total of 1507 samples were completed (86.6% compliance), or an average of 17.3 out of 20 samples per person.

However, the number of completed diary entries were not significantly related to the severity of daily stressors ($\beta = .13$, $SE = .12$, $p = .30$), coping strategy (approach: $\beta = .18$, $SE = .14$, $p = .20$; avoidance: $\beta = -.03$, $SE = .10$, $p = .79$), emotional response to stress (activated: $\beta = .10$, $SE = .14$, $p = .46$; deactivated: $\beta = .08$, $SE = .12$, $p = .52$), or reports of daily health symptoms ($\beta = .37$, $SE = .23$, $p = .11$). Finally, compliance to salivary cortisol sampling was not significantly related to daily cortisol output ($\beta = .03$, $SE = .42$, $p = .94$).

Demographics. We then assessed whether any demographic variables (age, gender, or ethnicity) were related to any of the predictors or outcome variables, and found that only ethnicity was related to study variables. More specifically, age did not significantly predict daily stress severity ($\beta = -.08$, $SE = .05$, $p = .11$). Age did not significantly predict approach ($\beta = -.01$, $SE = .05$, $p = .93$) or avoidance coping strategies ($\beta = -.04$, $SE = .04$, $p = .26$), nor did age significantly predict activated emotional responses to stress ($\beta = -.05$, $SE = .05$, $p = .35$) or deactivated emotional responses ($\beta = -.07$, $SE = .05$, $p = .17$). As well, age was not significantly related to reports of daily health symptoms ($\beta = -.09$, $SE = .09$, $p = .28$). Finally, age did not predict daily cortisol output ($\beta = -.07$, $SE = .15$, $p = .64$).

Gender was not significantly related to reports of severity of daily stressors ($\beta = -.22$, $SE = .18$, $p = .22$). Gender was also not significantly related to the use of approach ($\beta = -.20$, $SE = .22$, $p = .37$) or avoidance coping strategies ($\beta = -.07$, $SE = .16$, $p = .64$), nor was it significantly related to emotionally activated ($\beta = -.23$, $SE = .22$, $p = .29$) or deactivated ($\beta = -.03$, $SE = .18$, $p = .85$) responses to stress. As well, gender did not predict reports of daily health symptoms ($\beta = .02$, $SE = .36$, $p = .95$). Finally, gender marginally predicted daily cortisol output ($\beta = -1.41$, $SE = .76$, $p = .07$).

Ethnicity significantly predicted reports of stress severity ($\beta = .38$, $SE = .14$, $p = .01$), such that non-Caucasian participants reported more severe stress than Caucasian participants. Ethnicity did not significantly predict participants' use of approach ($\beta = .07$, $SE = .16$, $p = .69$) or avoidance coping strategies ($\beta = .09$, $SE = .11$, $p = .45$), nor did ethnicity predict emotional responses to stress (activated emotions: $\beta = .14$, $SE = .15$, $p = .37$; deactivated emotions: $\beta = .15$, $SE = .14$, $p = .29$). Ethnicity was not significantly related to cortisol output ($\beta = .67$, $SE = .49$, $p = .18$) or health symptoms ($\beta = .23$, $SE = .31$, $p = .46$). Because of the significant association with stress, ethnicity was controlled for in subsequent analyses.

Variance. We next tested whether there was sufficient variance in each of our daily variables in order to justify examining relationships among daily variables. Overall, we found significant variability in all daily variables. To test this, we first ran a series of unconditional level-1 models in order to determine the proportion of within-person variability that existed for each variable. Tau and sigma-squared coefficients represent between and within-person variance components, respectively. Coefficients, variance components, and the percent of variation occurring at the within-person level, are presented in Table 2.4. There was a significant amount of variability in the severity of stressors (variance = 0.46, $p < .001$), daily health symptoms (variance = 3.26, $p < .001$), as well as coping (approach variance = 0.59, $p < .001$; avoidance variance = 0.29, $p < .001$) and emotional responses to stress (activated emotion variance = 0.71, $p < .001$; deactivated emotion variance = 0.55, $p < .001$). Finally, cortisol output varied significantly across the week within individuals (variance = 1.99, $p < .001$). About 70% of variability in daily stress severity occurs within individuals, 61% of variability in daily health symptoms occurs within individuals, 20% of the variability in coping strategy occurs within individuals, 15% of variability in emotional responses to stress occurs within individuals, and finally, 82% of variability in cortisol output occurs within individuals. These results suggest that there was significant variability in study variables from day to day within a person, and hence that it was appropriate to model predictors of within person variance in study variables.

Finally, in preliminary analyses, we tested relationships among the study variables averaged across the monitoring period with bivariate correlations (hence describing overall relationships, but not day-to-day variability in relationships). Across the entire study period, greater average severity of stressors was significantly correlated with more health symptoms ($r = .38$, $p < .001$). Average stress severity was also significantly related to the use of coping

strategies (approach: $r = .43, p < .001$, avoidance: $r = .34, p < .01$) and emotional responses to stress (activated: $r = .72, p < .001$, deactivated: $r = .51, p < .001$), indicating that reports of more severe stress were related to greater use of both approach and avoidance coping strategies. As well, experiencing more severe stress was related to reports of both activated and deactivated emotional responses to stress across the monitoring period. Stress severity was not significantly related to average cortisol output. Results are presented in Table 2.3.

Daily Stress and Health Symptoms

We next tested relationships between stress and health symptoms on a day-to-day basis (in contrast to the bivariate correlations above which show associations averaged across the study period). Results showed a significant positive association such that, on days when individuals reported more severe stressors, they also reported greater health symptoms as compared to days when the same individuals reported less severe stressors ($\beta = .24, SE = .11, p = .04$). In contrast, stress did not predict next day health symptoms after controlling for previous health symptoms ($\beta = .09, SE = .07, p = .19$). (Because there were no lagged effects of stress on health symptoms the following day, sleep at night was not included in this paper as it could not mediate associations between stress and health measured on the same day.)

Relations among Daily Stress and Psychological Outcomes

Coping responses to stress. We then generated a series of level-1 models in order to determine whether daily perceived stress or the severity was related to daily use of coping strategies. Results showed that the severity of the worst daily stressor significantly predicted the utilization of both approach ($\beta = .16, SE = .08, p = .05$) and avoidance ($\beta = .12, SE = .06, p = .03$) strategies, indicating that, on days when participants reported experiencing more severe daily stressors, they reported a greater use of both approach and avoidance coping

strategies in response to stress compared to days in which they experienced less severe stressors.

Emotional responses to stress. We also generated a series of level-1 models in order to determine whether daily perceived stress predicted daily emotional responses. Results showed that reports of daily stress severity significantly predicted higher degrees of activated negative emotions ($\beta = .51$, $SE = .04$, $p < .01$) as well as deactivated negative emotions ($\beta = .32$, $SE = .04$, $p < .01$). These findings indicate that, on days when participants experience more severe stressors, they also report greater activated and deactivated emotional responses to stress compared to days in which they experienced less severe stressors. Results are presented in Table 2.5.

Relations among Daily Stress and Daily Cortisol Secretion

In order to test whether the experiences of daily stress were related to daily cortisol secretion, we generated a series of level-1 models assessing whether the severity of the worst stressor impacted cortisol output. Results showed that daily stress severity did not significantly predict daily cortisol output ($\beta = .21$, $SE = .41$, $p = .61$). Results are presented in Table 2.5.

Given the lack of associations above, we were unable to meet the conditions necessary to test whether cortisol mediated the relation between daily stress severity and health symptoms. Therefore, in subsequent analyses, we will focus on psychological pathways only.

Main effects of psychological variables on health symptoms

Coping response to stress. We tested whether daily coping responses predicted reports of daily health symptoms. Results showed that neither approach ($\beta = .09$, $SE = .07$, $p = .18$) nor

avoidance coping strategies ($\beta = .14$, $SE = .11$, $p = .22$) predicted daily health symptoms, indicating that on days in which participants utilized more coping strategies, they were not more likely to report health symptoms.

Emotional response to stress. We also tested whether daily emotional responses predicted reports of daily health symptoms. Results showed that, on days when participants reported more activated emotional responses to stress, they also reported a significantly greater number of health symptoms compared to days in which they reported fewer activated emotional responses ($\beta = .45$, $SE = .12$, $p < .01$). As well, on days when participants reported more deactivated emotional responses to stress, they also reported a significantly greater number of health symptoms ($\beta = .60$, $SE = .16$, $p < .01$). Results are presented in Table 2.5.

Psychological Mediation of the Stress-Health Symptom Association

Coping responses to stress. Given that neither the use of approach nor avoidance coping strategies were significant predictors of daily health symptoms, we did not test whether participants' coping responses to stress mediated the relation between daily stress and daily health symptoms.

Emotional responses to stress. Given that the severity of daily stress significantly predicted both activated and deactivated negative emotional responses to stress, and that both activated and deactivated negative emotional responses to stress significantly predicted reports of daily health symptoms, we next tested whether emotional responses to stress mediated the stress-health symptom association. To perform these analyses we entered both daily stress severity and daily emotional response to stress as predictors of daily health symptoms. Results showed that, when activated emotional responses were added to the model, daily stress severity was no longer a significant predictor of daily health symptoms (β

= -.001, SE = .10, $p = .99$), indicating that activated emotional responses to daily stress mediated the relation between daily stress and health symptoms. After comparing the variance components between the unconditional model and variance of the model with activated emotional responses included, we found that activated emotional responses to daily stress accounted for 7% of the variance in the relation between daily stress and health symptoms.

As well, results showed that when deactivated emotional responses were included as a predictor, daily stress severity no longer significantly predicted reports of daily health symptoms ($\beta = .04$, SE = .09, $p = .64$), indicating that deactivated emotional responses to stress also mediated the relation between daily stress and health symptoms. After comparing the difference in variance components between the unconditional model and variance of the model with deactivated emotional responses included, results showed that deactivated emotional responses to stress accounted for 21% of the variance in the relation between daily stress severity and health symptoms. Overall, these results indicate that the experience of more severe stressors on one day may be associated with a greater number of health symptoms that same day, in part because of the ways in which individuals emotionally respond to the stressor that day.

Although all variables (stress, negative emotions, health) were measured on the same day, and hence we were not able to definitively determine the directionality of these associations, we conducted additional analyses to assess whether there was evidence to support alternative directional pathways. Our working model was that stress would elicit negative emotions, which in turn would lead to health symptoms. However, it is possible that stress might first affect health symptoms, and that the experience of health symptoms would in turn lead to negative emotions. To test this possibility, we conducted level-1

analyses in which we tested whether the relationship between stress and negative emotions could be explained by health symptoms. As reported above, on days when participants experience more severe stressors, they also report greater activated and deactivated emotional responses to stress compared to days in which they experienced less severe stressors (activated: $\beta = .51$, $SE = .04$, $p < .001$; deactivated: $\beta = .32$, $SE = .04$, $p < .001$). When we included health symptoms in the model as a control, the relation between stress severity and emotions remained significant (activated: $\beta = .49$, $SE = .04$, $p < .001$; deactivated: $\beta = .27$, $SE = .04$, $p < .001$), suggesting that health symptoms do not serve as a pathway linking daily stress to daily negative emotions.

It is also possible that negative emotional states elicit reports of more severe daily stressors, which in turn predict reports of greater daily health symptoms. Again, to test this possibility, we conducted level-1 analyses in which we tested whether the relationship between negative emotions and health symptoms could be explained by the severity of daily stressors. As reported above, on days when participants report more activated and deactivated negative emotions, they also report greater health symptoms compared to days in which they experienced less severe stressors (activated: $\beta = .45$, $SE = .12$, $p < .001$; deactivated: $\beta = .60$, $SE = .15$, $p < .001$). When we included stress severity in the model as a control, the relation between activated and deactivated negative emotions and health symptoms remained significant (activated: $\beta = .44$, $SE = .11$, $p < .05$; deactivated: $\beta = .53$, $SE = .15$, $p < .001$), which indicates that the severity of daily stressors does not serve as a pathway linking negative emotions to daily health symptoms. Results are presented in Table 2.6.²

² Given that previous research has indicated that stressors that are uncontrollable and socially evaluative illicit the greatest stress response (see Dickerson & Kemeny, 2004), we ran additional analyses to test whether stressors that were perceived as uncontrollable or interpersonal in nature were associated with daily health symptoms. Results

Discussion

In summary, we found that daily stress severity predicted reports of daily health symptoms, such that on days in which individuals experienced more severe stress they also experienced a greater number of health symptoms compared to days in which those same individuals experienced less severe stress. Emotional responses to stress, but not coping responses or cortisol, mediated this relationship, such that on days in which individuals reported more severe stress, they also experienced more negative emotional responses, and in turn, on days in which individuals experienced more negative emotional responses, they also reported more health symptoms. In addition, when emotional responses were controlled, the relationship between daily stress severity and daily health problems became non-significant. In contrast, we did not find evidence for alternative directional pathways, that is, that stress operates via health symptoms to affect negative emotions, or that negative emotions operate via severity of stressors to affect health symptoms.

Overall, the study findings are consistent with previous research that has shown negative emotions to mediate the association between stress and markers of health (Stone et al., 1987; Stone et al., 1994; Stone, 1984), as well as between stress and disease progression in rheumatoid arthritis patients (Crosby, 1988). However, this study builds upon previous work by documenting that emotional responses mediated the relation between daily stress and daily health *symptoms* within a healthy population. Therefore, these findings suggest that negative emotional responses may serve as an important pathway by which daily stress creates vulnerability for poor health.

showed that neither uncontrollable ($\beta = -0.10$, $SE = 0.15$, $p = .52$) nor interpersonal ($\beta = -0.11$, $SE = 0.09$, $p = .26$) predicted daily health symptoms.

How might daily experiences of negative emotions affect the relationship between daily stress and daily health? One explanation is that negative emotional responses to stress trigger changes in biological processes, which could in turn affect health. However, we did not find support for cortisol as a mediator in the stress-health association. It may be that different biological systems are altered due to emotional responses to stress, such as the sympathetic-adrenal-medullary system. For example, Jacob and colleagues (Jacob, Thayer, Manuck, Muldoon, Tamres, & Williams, 1999) found that the negative emotional responses to a lab stressor predicted heightened ambulatory blood pressure.

Alternatively, negative emotional responses to stress may predicted more daily health symptoms because of the negative health behaviors individuals engage in to off-set the negative emotions. More specifically, previous studies have reported negative affect to be a risk factor for negative health behaviors including drinking alcohol (Cooper, Frone, Russell, & Mudar, 1995), smoking cigarettes (Carmody, Vieten, & Astin, 2007), and binge eating (Telch, Agras, & Linehan, 2000). Consistent with this explanation, Park and colleagues (2004) reported that college students consumed more alcohol on days when they reported more severe stressful events. In turn, engaging in these negative health behaviors may have increased health symptoms, such as headache, upset stomach, and nausea.

Another explanation could be that individuals are more likely to report health symptoms on days when they experienced more negative emotional responses to stress. Barsky and colleagues (Barsky, Peekna, & Borus, 2001) reported in their review that individuals with affective disorders were more likely to report bodily symptoms than individuals not suffering from mood disorders. In addition, Piccinelli and Simon (Piccinelli & Simon, 1997) found that emotional distress predicted greater reports of somatic symptoms in the absence of an organic illness. Taken together, these studies raise the possibility that individuals who have

more negative emotional responses to stress may also have a tendency to report greater health symptoms.

In contrast to our hypotheses, daily coping responses did not significantly mediate the relation between daily stress and health. Results from our studies showed that stress severity predicted greater use of all types of coping approaches; however coping did not predict health symptoms. It is possible that simply engaging in coping strategies is not necessarily beneficial for health, but rather, only when one engages in coping strategies that effectively manage stress are there health benefits (Zautra, 2003). Hence future studies may need to include assessments of the effectiveness of coping strategies in testing relationships with health.

In addition, daily cortisol did not mediate the relation between daily stress and health. This is in contrast to previous studies that have reported daily stressful experiences to predict increases in cortisol secretion (Peeters, Nicholson, & Berkhof, 2003; Schlotz, Schulz, Hellhammer, Stone, & Hellhammer, 2006; Smyth, Ockenfels, Porter, Kirschbaum, Hellhammer, & Stone, 1998; van Eck, Nicolson, Berkhol, & Sulon, 1996). However, recent research suggests that stress may lead to changes in cortisol secretion only in individuals who are predisposed to be biologically sensitive to stressors. For example, researchers have found that individuals from childhoods characterized by family conflict or non-nurturing relationships have heightened cortisol responses to stress, whereas individuals from more positive environments are buffered from the effects of stress (Carpenter, et al., 2007; Luecken, 1998; Repetti, Taylor, & Seeman, 2002). Therefore, the non-significant relation between daily stress and cortisol found in this study may have been due to the fact that we did not take into account the moderating role of certain psychosocial characteristics.

We note that daily stress severity was associated with same-day, but not next day, health symptoms. This suggests that, in our sample, stress severity significantly predicted transient or short-term health symptoms, rather than having more enduring effects. It may have been that greater stress severity was associated with health symptoms that were short in duration, including fatigue and stomach upset, due to short-term elevations in stress-induced nervousness, or stress-induced headaches. As well, after experiencing negative health symptoms, individuals may have engaged in restorative activities, such as exercise, seeking social support, or getting a good night's sleep, enabling them to 're-set' for the following day.

Results from this study provide an important step forward in our understanding of the intermediary factors that may mediate the relation between daily stress and daily health symptoms within a sample of healthy individuals. However, there are several noteworthy limitations of this study. First, health symptoms were collected via self-report and therefore may not be an accurate reflection of their health status. Future studies are needed that gather more objective daily measures of health, such as school or work absenteeism, aspirin use, or doctors visits, in order to determine whether negative emotional responses to stress are important mediators in the association between stress and health outcomes that interfere with daily functioning and have broader social consequences. In addition, the fact that stress, emotional responses, and health symptoms were all assessed via self-report raises the possibility of shared method variance accounting for the study findings. As well, we did not record individuals' health behaviors in this study. Future studies should also include a measure of health behaviors in order to assess whether these behaviors serve as another important intermediary factor in the relation between stress and health. Finally, while the daily diary design allowed us to assess stress as it occurred in a natural setting, our design

was limited in that we only considered the most severe stressor of the day. The use of other study designs to measure daily events, such as event-based responding, would allow for the assessment of emotion and coping responses to a number of daily stressors, rather than just the most severe daily stressor.

Modern society is brimming with daily challenges, and stress-related illnesses including cardiovascular disease, cancer, and stroke, are among the deadliest that plague our society (“World Health Report: Mental Health,” 2001). It is essential to understand the psychological and biological sequelae of stress in order to formulate effective interventions. Findings from this study suggest the possibility that interventions aimed at reducing negative emotional responses to stress might help to buffer individuals from the acute negative health consequences of stressful daily experiences.

Table 2.1

Sample Descriptive Statistics

| <u>Variable</u> | <u>Mean (SD)</u> | <u>Range</u> |
|-----------------|------------------|--------------|
| Age | 21.51(1.84) | 19-25 |
| Gender | | |
| Male | 33.3% | |
| Female | 66.7% | |
| Ethnicity | | |
| Caucasian | 27.6% | |
| Asian | 57.5% | |
| Other | 14.9% | |

Table 2.2

Descriptive Statistics of Study Variables

| Variable | Mean (SD) | Range |
|-----------------------|-------------|------------|
| Daily Stress | | |
| Severity of stressor | 3.17 (0.81) | 1.80-4.71 |
| Coping Strategy | | |
| Approach | 4.68 (1.55) | 1-6 |
| Avoidance | 3.24(1.08) | 1-5 |
| Emotional Response | | |
| Activated emotions | 2.24(1.25) | 0 - 5 |
| Deactivated emotions | 1.52(1.09) | 0 - 5 |
| Cortisol | | |
| Total output | 9.07(4.29) | 1.31-36.13 |
| Daily Health Symptoms | | |
| Number of Symptoms | 2.42(1.58) | 0.00-6.57 |

Note. Variables were aggregated across all days of data collection. The stress severity response rating options ranged from 1-5, indicating that on average participants reported moderately severe stressors. Coping approach strategy scores had a possible range of 0-12. Coping avoidance strategy scores had a possible range of 0-7. Activated and deactivated

emotional response scores had a possible range of 0-5. Health symptoms were selected from a list of 15.

Table 2.3

Between-person Correlations among Study Variables Averaged Across Days

| Variable | 1. | 2. | 3. | 4. | 5. | 6. | 7. |
|--------------------------|--------|--------|--------|--------|--------|-----|----|
| 1. Stressor Severity | — | | | | | | |
| 2. Coping – Approach | .43*** | — | | | | | |
| 3. Coping – Avoidance | .34** | .61*** | — | | | | |
| 4. Emotion – Activated | .72*** | .58*** | .53*** | — | | | |
| 5. Emotion – Deactivated | .51*** | .40*** | .43*** | .80*** | — | | |
| 6. Cortisol Output (AUC) | -.07 | -.02 | .03 | -.05 | -.02 | — | |
| 7. Health Symptoms | .38*** | .23* | .18 | .44*** | .55*** | .04 | — |

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 2.4

Multilevel Variance Components for Daily Stress, Health, and Biological Variables

| | Between-person variance (Tau) | Within-person variance (sigma ²) | Variance Coefficient | Percent within-person variance |
|-------------------|----------------------------------|---|-------------------------|--------------------------------------|
| Stressor severity | 0.46 | 1.09 | .46*** | 70.32% |
| Coping – Appr. | .59 | .15 | .59*** | 20.30% |
| Coping – Avoid. | .29 | .07 | .29*** | 19.44% |
| Emotion – Act. | .71 | .13 | .71*** | 15.48% |
| Emotion – Deact | .55 | .10 | .55*** | 15.38% |
| Cortisol output | 1.99 | 3.10 | 1.99*** | 60.90% |
| Health Symptoms | 3.26 | 15.16 | 3.26*** | 82.30% |

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 2.5

Direct Associations between Stress Severity and Mediators, and Mediators and Health Symptoms

| <u>Stress Severity Predicting Psychological and Biological Mediators</u> | | | |
|--|----------|-----|----------|
| | <i>b</i> | SE | <i>p</i> |
| Stress Severity | | | |
| Coping-Approach | .16 | .08 | .05 |
| Coping-Avoidance | .12 | .06 | .03 |
| Emotion-Activated | .51 | .04 | <.01 |
| Emotion-Deactivated | .32 | .04 | <.01 |
| Cortisol (AUC) | .21 | .41 | .61 |
| <u>Psychological Mediators Predicting Daily Health Symptoms</u> | | | |
| | <i>b</i> | SE | <i>p</i> |
| Daily Health Symptoms | | | |
| Coping-Approach | .09 | .07 | .18 |
| Coping-Avoidance | .14 | .11 | .22 |
| Emotion-Activated | .45 | .12 | <.01 |
| Emotion-Deactivated | .60 | .16 | <.01 |

Note. Each direct association represents a separate model. All predictor variables were entered separately into the models.

Table 2.6

Activated and Deactivated Emotional Responses to Stress as Mediators in the Stress-Health Symptom Relation

| | <u>Health Symptoms</u> | | |
|-------------------------|------------------------|------|----------|
| | <i>b</i> | SE | <i>p</i> |
| <u>Model</u> | | | |
| 1. Stress Severity | 0.25 | 0.11 | .03 |
| 2. Emotion-Activation | 0.44 | 0.11 | <.001 |
| Stress Severity | -0.001 | 0.10 | .999 |
| 3. Emotion-Deactivation | 0.53 | 0.15 | .001 |
| Stress Severity | 0.04 | 0.09 | .64 |

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Daily Stress, Cortisol, and Sleep: The Moderating Role of Childhood Psychosocial
Environments³

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Daily Stress, Cortisol, and Sleep: The Moderating Role of Childhood Psychosocial Environments

Psychological stress has been associated with poor health in individuals across the lifespan and in many countries (Lin & Ensel, 1989; Marmot & Wilkinson, 1999). For example, greater psychological stress has been linked to increased risk for cardiovascular disease, autoimmune disorders, infectious disease, and mental illness (McEwen, 1998). This relationship is so robust that it has been observed across the lifespan, from infants to the elderly (Graham, Christian, & Kiecolt-Glaser, 2006).

One mechanism linking psychological stress to disease is the biological responses that are associated with stress. Under high levels of acute stress, individuals exhibit a heightened activation of certain biological systems, termed the fight-or-flight response (Canon, 1932). The biological 'fight-or-flight' response involves the activation of neural, neuroendocrine, and immune mechanisms that prepare the body to overcome or to avoid danger. Over time, it is possible that repeated activation of these systems can cause wear and tear on the body, referred to as allostatic load (McEwen, 1998), eventually leading to poor health.

Biological responses to stress, however, are not uniform across individuals, and individual differences in biological stress responses may provide one explanation as to why stress can lead to disease in some people but not others (Miller, et al., 2009). While much research has been conducted on the direct relationship between stress and biological risk for disease, less is known about factors that might moderate the stress-biology relationship across individuals.

Previous research suggests that characteristics of the childhood social environment may partially explain differences in biological responses to stress in adults (Miller & Chen, 2007;

Repetti, Taylor, & Seeman, 2002). For example, according to Taylor and colleagues (Taylor, Lerner, Sage, Lehman, & Seeman, 2004), children who experience insecure attachments to caregivers, harsh parenting, or lack of social support exhibited greater secretion of stress hormones, higher heart rates, and higher blood pressure in response to an acute laboratory stressor in adulthood than individuals raised in more nurturing environments. In addition, early experiences of social deprivation, such as a child experiencing distant or cold parental relationships (Heim, et al., 2000), or a child spending their first years of life in a Romanian orphanage (Rutter & O'Connor, 2004), have also been shown to result in heightened biological reactivity to stress in adulthood.

These early life experiences also have implications for later-life health problems. For example, a recent review of the literature reported that children raised in “risky families” (i.e., families characterized by harsh or cold parenting) were at greater risk for heart disease, cancer, chronic lung disease, and skeletal fractures in adulthood than children whose early life parental relationships were warm and nurturing (Repetti et al., 2002). Taken together, previous findings suggest that family relationships in childhood may continue to impact both biological stress responses and health in adulthood.

In the present study, we investigate whether childhood family environments affect the relationship between daily stress and daily cortisol and sleep patterns. The majority of previous research has been laboratory based or cross-sectional, and it is important to understand how childhood environments may affect the relationship between naturally occurring stress and daily biological rhythms (van Eck, Nicolson, Berkhof, & Sulon, 1996). We focused on cortisol and sleep because dysregulations in both cortisol secretion and sleep have been noted as markers of allostatic load (McEwen, 2007) and are risk factors for poor health (Cohen, Janicki-Deverts, & Miller, 2007; Dickerson & Kemeny, 2004; Meerlo,

Koehl, van der Borgh, & Turek, 2002). As well, both follow daily circadian patterns (Van Cauter, Polonsky, & Scheen, 1997), enabling us to assess whether daily stress was related to altered daily biological patterns. In addition, both the patterns of cortisol secretion and sleep are established early in life (Phillips & Jones, 2006; Rivkees, 2003), and hence form plausible targets that may be influenced by childhood family environments.

Stress and Cortisol

Cortisol is a hormone released by the hypothalamic-pituitary-adrenal (HPA) axis under conditions of stress in order to restore homeostasis to the body (Phillips & Jones, 2006). Previous research has reported that a variety of different types of stressful experiences alter cortisol secretion. For example, acute psychological stressors that involve social evaluation are known to increase cortisol levels (Dickerson & Kemeny, 2004). Chronic stressors have been found to initially increase cortisol secretion, but over time to result in blunted cortisol secretion (Miller et al., 2007). The majority of daily diary studies have reported that naturally occurring stressors elicited greater daily cortisol secretion (Peeters, Nicholson, & Berkhof, 2003; Schlotz, Schulz, Hellhammer, Stone, & Hellhammer, 2006; Smyth, Ockenfels, Porter, Kirschbaum, Hellhammer, & Stone, 1998; van Eck et al., 1996; for an exception, see Hanson and colleagues, Hanson, Maas, Meijman, & Godaert, 2000).

Furthermore, previous research has demonstrated that the early life environment also affects daily cortisol secretion. Nicholson (2004) found that parental loss during childhood was related to elevated daily cortisol in adulthood. As well, Heim and colleagues reported that adult men with childhood histories of trauma showed hyperactive HPA responses to the administration of an exogenous steroid test (indicative of dysregulated cortisol secretion), as

compared to men with no history of childhood trauma (Heim, Mletzko, Purselle, Musselman, & Nemeroff, 2008).

However, the above research focuses on main effects – that is, whether stress or early life environments have direct effects on cortisol. The question remains as to whether childhood environments *moderate* the relationship between stress and cortisol. A small number of studies have addressed this question. One approach in the human literature has been to utilize acute laboratory stressors and test whether childhood environments affect how adults respond to experimentally manipulated stress. Using this paradigm, studies have found that difficult childhood environments predict increased cortisol response post-task in adults (Luecken, 1998; Repetti et al., 2002), although some studies report hypo-responsiveness to a lab stressor (Carpenter, et al., 2007). Conversely, perceptions of maternal responsiveness have been reported to mitigate the effects of an acute lab stressor on cortisol (Evans, Kim, Ting, Teshler, & Shannis, 2007). Work in the animal literature has found that non-human primates raised in stressful childhood environments (e.g., intermittent food availability) displayed amplified cortisol responses to laboratory stressors as adults (Gorman, Mathew, & Coplan, 2002; Hennessy, 1997; Rosenblum, Forger, Noland, Trost, & Coplan, 2001). Conversely, rats raised in nurturing environments (e.g., with mothers who spend more time licking) display more modest HPA responses to restraint stress as adults (Caldji, Diorio, & Meaney, 2000; Meaney, 2001).

Hence, these studies have focused on laboratory responses to acute stress. In the present study, we focus on naturalistically occurring daily life stressors, and test whether childhood family environments would moderate the relationship between naturally occurring daily stress and daily secretion of cortisol in young adulthood. We hypothesize that the relation between daily stress and increased cortisol secretion will be stronger among individuals from

difficult childhood environments, whereas the relation will be attenuated in individuals from less difficult childhood environments.

Stress and Sleep

Sleep is another process that is both important for health and impacted by stress. Although it is also a behavior, sleep can be considered a biological process in that it is regulated by the brain stem, thalamus, hypothalamic hormones, and external stimuli (i.e., light) (Dahl & Lewin, 2002; Hall, 1998). Sleep disorders, including insomnia, hypersomnia, narcolepsy, and sleep apnea, are often a symptom of psychological distress (Kales, Soldatos, & Kales, 1987; Nixon & Pearn, 1977; Vgontzas & Kales, 1999). Experimental studies in both humans and animals have documented that acute stressors experienced during the day result in disruptions in sleep architecture, including longer transitions into REM sleep, at night (Cheeta, Ruigt, Proosdij, & Willner, 1997; Meerlo, Koehl, van der Borght, & Turek, 2002). Naturally occurring stressors such as periods of marital separation are associated with less delta sleep (Cartwright & Wood, 1991). Effects of stress on sleep are sometimes apparent only in certain subgroups (Hall, Buysse, Dew, Prigerson, Kupfer, & Reynolds, 1997), and occasionally, studies have reported no significant relationship between stressful life events and sleep (Paulsen & Shaver, 1991).

Daily diary studies have also demonstrated that daily stress is associated with poorer sleep (Åkerstedt, 2007; Ancoli-Israel & Roth, 1999; Hall et al., 2000; Tworoger, Davis, Vitiello, Lentz, & McTiernan, 2005; Urponen, Vuori, Hasan, & Partinen, 1988). However, in some cases, the relationship between stress and sleep is apparent only in certain subgroups (Dagan, Zinger, & Lavie, 1997; Sadeh, Keinan, & Daon, 2004; Pillar, Malhotra, & Lavie,

2000), or among those who use certain types of coping strategies (Sadeh, Keinan, & Daon, 2004).

Studies investigating childhood adversity have also found associations with sleep, using both human and animal models. For example, in a longitudinal study by Gregory and colleagues (Gregory, Caspi, Moffitt, & Poulton, 2006), they found that greater family conflict in childhood predicted symptoms of insomnia at age 18, over and above the current psychosocial environment. In contrast to childhood adversity, childhood environments characterized by parental warmth have been shown to be predictive of earlier bedtimes and longer sleep times in a nationally representative sample of children and adolescents by Adam and colleagues (Adam, Snell, & Pendry, 2007). Similarly, animal studies have shown that primates separated from their mothers displayed sleep disturbances including a greater number of arousals and decreased REM sleep (Reite & Snyder, 1982).

Hence findings from the literature indicate that there is some support for the notion that stress is related to sleep, and that childhood environments can predict sleep in adulthood. However, it remains unclear whether childhood environments are capable of moderating the relation between daily stress and sleep. In the present study, we tested this relationship, hypothesizing that, among individuals from difficult childhood family environments (e.g., high conflict, low warmth), experiences of current stress would be associated with poorer sleep (i.e., shorter duration, lower efficiency) at night. In contrast, we hypothesized that the relation between stress and sleep would be attenuated in individuals from less difficult childhood environments.

Method

Participants

87 healthy college undergraduate students participated in the current study. They were recruited from the University of British Columbia in Vancouver, BC Canada through campus postings. Students were eligible to participate in the study if they 1) were between the ages of 19 and 25, 2) were medically healthy, and 3) were fluent in English. Eligible participants were scheduled for a laboratory visit. The study sample was about 67% female, and was 28% Caucasian, 57% Asian, and 15% ‘other.’ Sample descriptive statistics are reported in Table 3.1.

Measures

Childhood Family Psychosocial Environment

Risky Families Questionnaire. In order to assess the implications of negative characteristics of participants’ childhood family environments, participants completed the Risky Families Questionnaire during their initial lab visit (Taylor, Way, Welch, Hilmert, Lehman, & Eisenberger, 2006). This questionnaire measures the level of family conflict as well as parental coldness/lack of affection in the family environment during childhood. Response options are on a 4-point Likert scale (1= none of the time; 4=most or all of the time). Sample items include, “how often would you say there was quarreling, arguing, or shouting between a parent and you?” and, “how often would you say you were neglected while you were growing up, that is, left on your own to fend for yourself?” Higher scores indicate greater family conflict/risk in childhood. Previous research has demonstrated that this measure has high reliability ($\alpha = .77$). As well, responses to the risky family questionnaire were highly related to responses to interviews regarding early life family

environments, demonstrating adequate validity (Taylor, Lerner, Sage, Lehman, & Seeman, 2004).

Parental Warmth. We also considered the effects of positive childhood family characteristics on responses to daily stress. Participants reported the degree of warmth in their parental relationships by completing the Parent Bonding Inventory (PBI) (Parker, 1979). This inventory consists of 25 items regarding participants' recollection of the quality of their relationship with their mother and father during age 0 through 16. Participants reported on a 4-point Likert scale how true the statement is to their own experiences. Sample items include, "my mother spoke to me in a warm and friendly voice," and, "my father seemed emotionally cold to me (reverse scored)." The original PBI is scored along two dimensions: parental warmth/care and parental overprotection/restrictiveness. In this study, we only considered scores on the warmth scale for this study because of our interest conceptually in the components of risky families (high conflict and low warmth). The inventory has demonstrated adequate test-retest reliability, ranging from .60 to .79, and has demonstrated adequate validity when scores were compared between MZ and DZ twins, as well as with interview-based ratings (Parker, 1990). Scores were averaged across parents, when applicable, with higher scores indicating greater warmth. Scores from this sample were comparable or slightly higher than those from other samples of healthy young men and women (Martin et al., 1994; Russell et al., 1992).

Daily Diary Variables

Daily Stressors. Participants reported both the number of stressors that occurred during their day, as well as the negative impact that the most bothersome stressor of the day had on them using a web-based diary format. Once an evening throughout the seven day monitoring

period, participants were asked to check off which of a list of sixteen items they had experienced within the last 24 hours. Items included achievement stressors, interpersonal stressors, and daily hassles. The number of items that participants endorsed was summed to indicate the number of stressors they experienced that day. Participants were then asked to select the most severe stressor experienced during the day and respond to the question, “How serious was this for you?” Responses ranged from 1 to 5, with 1 equaling ‘not at all’ and 5 equaling ‘very serious.’ This measure was a modified version of the Hassles Scale (from the Hassles and Uplifts Scales; DeLongis, Folkman, & Lazarus, 1988) and was developed by Lee-Baggley and DeLongis for use in a university sample (2007, unpublished data). Previous research has demonstrated significant relations between the Hassles Scale and other life event scales (Holmes & Rahe, 1967), psychological symptoms scales (Derogatis, Lipman, Covi, Rickels, & Uhlenhuth, 1970; Derogatis, Lipman, Covi, & Rickels, 1971; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974), and health symptom checklists (DeLongis, Folkman, & Lazarus, 1988).

Sleep. Participants were instructed to wear an ambulatory wristwatch monitor called an Actiwatch (MiniMitter Co., Inc., Boulder, CO) for 7 nights following their lab visit. Subjects were instructed to wear the watch at all times. The Actiwatch measures gross motor movement via a sensor which generates a voltage when the Actiwatch senses acceleration. Watches are worn on the non-dominant arm and can be worn in the shower. Sleep quantity and efficiency (e.g., percent of the sleep interval in which the person is motionless) for each night was calculated using the Actiwatch software.

Cortisol. Participants collected salivary cortisol samples using Salivettes (Sarstedt, Nuembrecht, Germany). Samples were collected four times per day at one, four, nine, and eleven hours after awakening over five consecutive days following the lab visit to capture

total cortisol secretion across the day. To determine whether participants were compliant with the sampling schedule, Salivettes were stored in a bottle sealed by a MEMS 6 TrackCap Monitor (Medication Event Monitoring System, Aardex Ltd., Switzerland). Caps record the date and time of each opening. 86.6% of samples were completed, and 88.1% of completed saliva samples within 1 hour of the scheduled collection time. Saliva samples were returned to the lab and then centrifuged at 1000g for 5 min, transferred to deep-well plates, and stored at -30°C until assayed. Free cortisol levels in saliva were measured in duplicates using a commercially available chemiluminescence assay (IBL, Hamburg, Germany).

In order to assess total cortisol secretion throughout the day, data were first log transformed to reduce substantial skewness. Next, daily cortisol output was calculated via an area-under-the-curve (AUC) statistic using the trapezoidal rule.

Procedure

Participants came to the lab and signed consent forms. During their initial lab visit, participants provided background information on childhood family environment and demographic information. As well participants were given instructions on how to complete web-based surveys and how to collect salivary cortisol samples. Schedules for saliva sampling were set for the five days following the lab visit. Finally, Actiwatches, used to measure sleep, were described and distributed to participants.

For the 7 days following the lab visit, subjects completed a web-based diary entry at end of each day. Specifically, subjects were asked to report upon any stressful events they experienced in the past 24 hours.

To increase compliance, study participants received a daily reminder email with a link to the web-based survey, and those who did not complete the entry the previous day were

phoned by the study coordinator. Participants completed an average of 93.9% of daily diary entries over the course of 7 possible days. Three participants completed diaries on paper because they did not have daily access to computers. If participants completed an entry within 24 hours of the intended time, their data were included. If diaries were completed more than one day late, data were excluded from analyses. Participants received \$1.00 per diary entry completed on the appropriate day. Finally, participants received \$10 dollars at the initial lab visit, and \$10 for returning their Actiwatch and MEMS cap at the end of the study.

Statistical Analyses

Data were analyzed using hierarchical linear modeling techniques (HLM). This method of statistical analysis enabled us to test the within-person (level-1) relationships between daily stress and daily biological outcomes. It also allowed us to test whether between-person (level-2) factors moderate these day-to-day stress-health associations. Stress and biological variables (cortisol and sleep) were modeled as within-person factors because they were collected daily. Childhood family environment variables were modeled as between-person factors because they were collected at one time point and reflect a factor thought to vary across people.

First, we conducted a series of within-person (level-1) models to predict how sleep and cortisol output varied as a function of daily stress experiences. Level-1 models generate a set of slopes for each individual that reflect variations in biological markers (sleep and cortisol outcomes) as a function of daily stress. Level-1 predictor variables were centered around each individual's mean, allowing us to examine whether deviations from an individuals' average stress experiences, for example, impacted sleep that night.

Next, we conducted a series of between-person (level 2) models to determine whether between-person factors (i.e., childhood family environment) explained the variance in slopes from the level-1 models. In other words, we tested whether different levels of our between-person factors were associated with different relationships between daily stress and biological markers. Significant interactions were graphed by depicting the relationship between stress and cortisol/sleep at the 25th and 75th percentiles of the family environment distribution. We used full maximum likelihood and robust standard errors to estimate all models.

Results

Descriptive Data and Preliminary Analyses

Means, standard deviations, and ranges for aggregated stress, sleep, and cortisol data, as well as childhood psychosocial characteristics, are presented in Table 3.2. Participants in this study slept an average of about 420 minutes (about 7 hours) per night, and their nightly sleep was about 84% ‘efficient’, or motionless. Participants reported an average of 3.5 stressors per day, with the most severe stressor reported as ‘somewhat’ serious.

Before testing our hypotheses, we ran a series of preliminary analyses in order to determine whether protocol compliance or demographic information was associated with any of our predictor or outcome variables, including stress, sleep, and cortisol. Below we present results from these preliminary analyses.

Compliance. Overall, we found that compliance was not significantly related to any of our daily diary variables. Out of a possible 609 entries, participants completed a total of 572 daily diary entries (93.9% compliance), or an average of 6.6 out of 7 days of daily diary entries per person. Out of a possible 609 nights of sleep, participants also completed 560

days of actigraphy (92.0% compliance), or an average of 6.4 out of 7 nights of sleep data per person. Finally, out of a possible 1740 salivary cortisol samples, participants in this study completed 1507 samples (86.6% compliance), or an average of 17.3 out of 20 samples per person.

We tested whether protocol compliance was related to daily data (number and severity of stressors) or biological markers (minutes asleep, sleep efficiency, cortisol output). The number of completed diary entries were not significantly related to the number of reported stressors ($\beta = -.18$, $SE = .28$, $p = .53$), or severity of daily stressor ($\beta = .13$, $SE = .12$, $p = .30$). Number of completed days of actigraphy was not significantly related to minutes of sleep per night ($\beta = -15.26$, $SE = 10.33$, $p = .14$) or nightly sleep efficiency ($\beta = .28$, $SE = .78$, $p = .72$). Finally, compliance to salivary cortisol sampling was not significantly related to daily cortisol output ($\beta = .03$, $SE = .42$, $p = .94$).

Demographics. We then tested whether demographic information (age, gender, and ethnicity) predicted daily cortisol output or sleep variables, and found that only ethnicity was related to some study variables. Ethnicity significantly predicted participants' reports of stress severity ($\beta = .38$, $SE = .14$, $p = .009$), indicating that minority participants rated daily stressors as more severe than Caucasian participants. Hence ethnicity was included as a covariate in subsequent analyses. Age was not significantly associated with cortisol output or sleep variables (p 's $> .05$). As well, gender was not associated with daily cortisol output or sleep variables (p 's $> .05$).

Variance. Finally, we tested whether there was sufficient variance in each of our daily variables in order to justify examining relationships among daily variables, and found significant variability to be present for all daily variables. We tested this by running a series

of level-1 unconditional models in order to determine the variance in each of the daily variables. Overall, findings show that there was a significant amount of variability in sleep minutes (variance = 2264.18, $p < .001$) and sleep efficiency (variance = 22.60, $p < .001$) across the monitoring period. There was also a significant amount of variability in the number (variance = 3.05, $p < .001$) and severity (variance = 0.46, $p < .001$) of stressors across the week. Cortisol output varied significantly across the week within individuals (variance = 1.99, $p < .001$). Results are presented in Table 3.3.

Main effects of daily stress on biological markers

We then generated a series of level-1 models to assess main effects of daily stressful experience on daily biological markers. More specifically, we tested whether, on days when participants reported greater number or severity of daily stressors than their own average, they experienced changes in cortisol secretion or sleep patterns. Overall, we found no significant main effects of daily stressful experiences on daily biological outcomes. Results are presented below.

Sleep. We ran a series of level-1 models to test whether the number of stressors or the severity of the worst stressor impacted sleep quantity or efficiency that night. Neither the number of daily stressors ($\beta = -1.94$, $SE = 2.82$, $p = .49$), nor the severity of the worst stressor ($\beta = -4.56$, $SE = 4.97$, $p = .36$) was related to the minutes of sleep per night. As well, neither the number of daily stressors ($\beta = 0.25$, $SE = 0.24$, $p = .31$), nor the severity of the worst stressor ($\beta = -0.39$, $SE = 0.41$, $p = .34$) was related to nightly sleep efficiency. These results indicate that there was no significant direct effect of stress during the day on sleep that night (see Tables 3.4 and 3.5).

Cortisol. We next conducted a similar set of analyses of the main effects between stress and cortisol. Results showed that neither the daily number of stressors ($\beta = 0.16$, $SE = 0.19$, $p = .40$), nor the severity of the worst stressor ($\beta = 0.23$, $SE = 0.40$, $p = .57$) significantly predicted total cortisol secretion. Results are reported in Tables 3.4 and 3.5.

Do childhood psychosocial factors moderate the daily stress-biology relation?

Due to the lack of a main effect of daily stressful experience on daily biological patterns, we next tested whether the level-2 between-person factors, family risk and parental warmth, moderated the relationship between daily stress and cortisol/sleep. In other words, we aimed to determine whether the relationships from daily stress to cortisol output, or daily stress to nightly sleep, were different for individuals from different childhood family environment backgrounds. To do this, we tested whether childhood family environment variables explained variance in the slopes from the level-1 models reported above. The multi-level model was specified as follows:

$$\text{Level 1: } \text{Sleep minutes}_{ij} = b_{0i} + b_{1i}(\text{severity of stressor}) + r_{ij}$$

$$\text{Level 2: } b_{0j} = \beta_{00} + \beta_{01}(\text{risky families}) + \beta_{02}(\text{ethnicity}) + \mu_{0i}$$

$$b_{1j} = \beta_{10} + \beta_{11}(\text{risky families}) + \beta_{12}(\text{ethnicity}) + \mu_{1i}$$

In other words, at level-1, minutes of sleep on any given night [Sleep minutes_{ij}] is a function of their minutes of sleep on an average stressor severity day (b_{0i}), the severity of the worst stressor that day (b_{1i}), and random error (r_{ij}). At level-2, b_{0j} represents a person's intercept (that is, the expected value of sleep minutes on average stressor severity days) as a function of intercepts across all participants (β_{00}), the participant's Risky Families score, ethnic group, and random error (μ_{0i}). As well, b_{1j} represents a person's slope (that is, how

sleep minutes vary in response to deviations from a person's average levels of stressor severity) as a function of the average slope across participants (β_{10}), the participant's Risky Families score, ethnicity, and random error (μ_{1i}). Subsequent models were computed, substituting sleep efficiency and cortisol output as level-1 outcome variables, the number of daily stressors as the level 1 predictor variable, and parental warmth as the between-person level-2 variable.

Risky Families

Sleep. We first tested whether childhood family psychosocial environment, as measured by the Risky Families questionnaire (Taylor et al., 2006), interacted with stress during the day to predict sleep at night. Results showed that family environment significantly moderated the relation between number of stressors during the day and minutes asleep that night ($\beta = -10.66$, $SE = 4.60$, $p = .02$). The significant interaction was graphed in Figure 3.1 by depicting the relationship between stress and sleep at two arbitrary points: at the low (25th percentiles) and high (75th percentiles) ends of the risky family environment distribution. Among subjects from risky family environments, on days when they experienced a greater number of stressors than their average, they spent fewer minutes asleep that night. In contrast, in subjects from less risky families, minutes asleep varies to a lesser degree with the daily number of stressors they experienced. Family risk accounted for 20.3% of the variance in the relation between daily number of stressors and nightly minutes of sleep. Family environment did not significantly moderate the relation between the number of stressors and sleep efficiency ($\beta = .25$, $SE = .50$, $p = .62$), nor did it moderate the relations between stress severity and sleep quantity or efficiency ($\beta = -13.50$, $SE = 10.53$, $p = .20$, and $\beta = .48$, $SE = .71$, $p = .50$, respectively). Results are reported in Table 3.4.

Cortisol. We then tested whether childhood family risk interacted with daily stressors to predict daily cortisol output. Results showed that family risk did not significantly moderate the relation between the daily number of stressors experienced and cortisol output ($\beta = .51$, $SE=.56$, $p=.37$) nor did it moderate the relation between the severity of the worst stressor and cortisol output ($\beta = 1.68$, $SE=1.04$, $p=.11$). These findings indicate that the relation between daily stress and cortisol secretion does not vary as a function of childhood family risk. Results are reported in Table 3.4.

Parental Warmth

Sleep. Next we tested whether parental warmth during childhood interacted with daily stress to predict sleep at night. Results were non-significant (p 's $> .40$) indicating that childhood parental warmth did not moderate the relation between daily stress and nightly sleep. Results are presented in Table 3.5.

Cortisol. Parental warmth significantly moderated the relation between the severity of stress experienced during the day and daily cortisol output ($\beta= -.16$, $SE=.08$, $p=.04$), such that in participants whose parents displayed less warmth during their childhood, on days when they experienced more severe stressors than their average, their cortisol output was higher than on days when they experienced less severe stressors than average. However, in participants whose parents displayed more warmth during childhood, cortisol output is less strongly related to daily stressful experiences. The significant interaction was graphed in Figure 3.2 by depicting the relationship between stress and cortisol output at two arbitrary points: at the low (25th percentiles) and high (75th percentiles) ends of the parental warmth distribution. Parental warmth accounted for 20.9% of the variance in the relation between daily severity of stressor and cortisol output.

Parental warmth did not, however, moderate the relation between the number of stressors experienced in a day and cortisol output ($\beta = -.05$, $SE=.04$, $p=.23$). Results are reported in Table 3.5.⁴

Discussion

The findings from this study provide some evidence that childhood psychosocial environments serve to moderate the relation between stress and biological outcomes. The nature of this interaction was such that among individuals from difficult childhood environments, days on which individuals experienced more stress were associated with less sleep and greater cortisol secretion. In contrast, among individuals from positive childhood environments, biological patterns varied less with stress. The fact that there were no direct effects of daily stress on daily cortisol or sleep suggests that approaches that focus on moderating psychosocial influences may be more useful than analyses of the main effects of day-to-day stress on biological processes.

These results are consistent with previous studies that have reported that difficult childhood environments predict increased cortisol secretion to stress (Gorman, Mathew, & Coplan, 2002; Hennessy, 1997; Luecken, 1998; Repetti, Taylor, & Seeman, 2002; (Rosenblum, Forger, Noland, Trost, & Coplan, 2001) and less sleep (Gregory, Caspi, Moffitt, & Poulton, 2006). As well, results are consistent with findings in the literature that parental warmth buffers the effects of stress on cortisol secretion (Caldji, Diorio, & Meaney, 2000; Evans, Kim, Ting, Tesher, & Shannis, 2007; Meaney, 2001) and is associated with longer

⁴ Despite our previous analyses indicating that gender was not a significant covariate, we chose to run the significant moderation analyses separately by gender, given previous research suggesting that the childhood environment may differentially impact biological programming in males versus females (Weiss, 1999). For both family risk and parental warmth, moderation findings were statistically significant for males (risk: $\beta = -21.72$, $SE = 10.63$, $p = .05$; warmth: $\beta = -0.43$, $SE = 0.16$, $p = .01$) but not females (risk: $\beta = -6.79$, $SE = 5.37$, $p = .21$; warmth: $\beta = -0.04$, $SE = 0.04$, $p = .24$). These results suggest that the childhood psychosocial environment may exert greater effects on biological changes under conditions of stress in adult males as compared to adult females.

sleep (Adam, Snell, & Pendry, 2007). However, our study builds upon previous research in demonstrating that childhood environmental characteristics moderate the relation between daily, naturally occurring stress and biological outcomes.

The findings from this study that individuals from ‘risky’ childhood environments had less sleep after days when they experienced a greater number of stressors suggests that these individuals may have developed a heightened sensitivity to potential threats during their childhood in order to prepare themselves to manage or avoid stressful events (Selye, 1955; Thompson & Calkins, 1996). However, this increased alertness may be incompatible with sleep (Sadeh, Keinan, & Daon, 2004). That is, as the number of daily stressors increases, individuals from risky family environments may become more vigilant or on-guard for upcoming stress, which may then have made it more difficult for them to sleep at night.

We also found that, in individuals whose childhood environments were characterized as low in parental warmth, on days when they experienced more severe stress they also secreted greater cortisol as compared to days when they experienced less severe stress. This is consistent with previous research that has shown that high parental warmth buffers youth from the negative effects of stress on cortisol (Evans, Kim, Ting, Teshler, & Shannis, 2007). As well, studies in the animal literature have reported that nurturing maternal behaviors help calibrate biological responses to stress (Caldji, Diorio, & Meaney, 2000; Meaney, 2001). In the present study, a *lack of nurturing* behavior experienced in childhood may impair children’s abilities to regulate their biological responses to stress. As a result, as adults these individuals may show greater cortisol responses to stressful life situations. Alternatively, parents who displayed low warmth may have modeled maladaptive emotion regulation skills to their children, which could then in turn exacerbate cortisol secretion in response to stress (Repetti, Taylor, & Seeman, 2002). Hence, after experiencing more severe stressful events,

individuals may become more emotionally distressed, leading to greater cortisol secretion across the day.

Non-significant findings from this study also provide important information in understanding the role of the childhood environment as a moderator in the relation between stress and biological processes. First, difficult childhood environments did not moderate the relation between daily stress and sleep efficiency (only sleep minutes). One explanation for these null results is that changes in sleep architecture (a reflection of sleep quality) under conditions of stress are only altered due to extreme childhood conditions, such as abuse (Sadeh, 1996), losing a parent (Luecken, 1998), or growing up in conditions of extreme poverty (Nicolau, Thomson, Steele, & Allison, 2007). Alternatively, it may be that our measure of sleep did not capture changes in sleep stage; Actiwatches detect motor movement, but do not have the ability to measure sleep architecture or sleep stage. Therefore, it is possible that childhood environmental factors moderate the relation between daily stress and nightly REM sleep, for example, but that we were unable to detect this due to the limitations of our equipment.

Findings from this study also indicated that, unlike risky childhood environments, low parental warmth did *not* predispose individuals to have shorter sleep times after days of stress as adults. This may be because a lack of parental warmth in childhood does not necessitate the need for heightened attention or sensitivity to potential environmental threats that would interfere with sleep. In other words, cold parental behaviors may have different effects from conflictual family relationships, with perhaps only the latter triggering heightened sensitivity to threat in response to greater numbers of stressors experienced on a given day.

As well, 'risky' childhood environments did *not* moderate the relation between daily stress and daily cortisol secretion. This may be in part because childhood environments that are characterized by repeated family conflict represent a type of chronic stressor, upon which acute daily stress is superimposed. As noted by Gump and colleagues in a recent review of the literature, when daily stress occurs within the context of background stress, some individuals may have more pronounced physiological reactivity because they have become sensitized to stress, whereas others may display reduced physiological responses because they have habituated to stress (Gump & Matthews, 1999; see also Marin, Martin, Blackwell, Stetler, & Miller, 2007; Murali & Chen, 2005). Therefore, it is possible that risky childhood family environments may have led to sensitivity to stress in some individuals and blunted responses to stress in others, resulting in no moderating effect of risky childhood environments on the relationship between daily stress and cortisol.

At a broader level, there are several explanations for why childhood family environments would moderate the stress-cortisol/sleep relationships in adulthood. First, biological responses to stress may be programmed early in life. Psychosocial challenges during the first few years of life have been shown to 'program,' or calibrate, the biological systems that respond to stress, and to do so in a way that persists throughout the lifetime (Boyce & Ellis, 2005). The most compelling examples of this come from the animal literature, given that childhood environments can be manipulated in these types of studies. For example, studies by Meaney and colleagues have shown that differences in the development of neural systems that control HPA activity in rat pups are mediated by maternal licking behaviors (Caldji, Diorio, & Meaney, 2000; Meaney, 2001), which lead to differences in adult biological responses to stress. Therefore, difficult childhood environments may directly program biological systems responsible for stress responses.

In addition, individuals from difficult childhood environments may have heightened biological responses to stress due to poor emotion regulation or poor coping. Previous studies have shown that individuals raised in homes characterized by conflict or aggression become sensitized to stress and to be more upset after stress (Cummings, Zahn-Waxler, & Radke-Yarrow, 1981; Davies & Cummings, 1994; O'Brien, Margolin, John, & Krueger, 1991). Repetti and colleagues (Repetti, Taylor, & Seeman, 2002) reported that prolonged emotional arousal can then exacerbate biological stress responses, possibly leading to increased cortisol secretion and disrupted sleep as reported in our study.

Another possibility, however, is that difficult childhood environments moderate the relation between stress and biological processes in adulthood due to the fact that many individuals who were socially deprived as children continue to struggle interpersonally into adulthood, and their vulnerability to altered biological processes is, in fact, due to the effects of their current environment. Recent research, however, has shown this not to be the case. Several studies have considered the roles of both childhood and adulthood environments on biological responses to stress, and all have found that the childhood environment predicts biological changes over and above the current psychosocial environment (Gregory, Caspi, Moffitt, & Poulton, 2006; Miller & Chen, 2007; Rutter & O'Connor, 2004). Taken together, this evidence suggests that a critical period may exist early in life during which environmental challenges have strong and long-lasting effects on biological systems involved in the stress response.

Results from this study contribute to the literature by demonstrating that, in order to understand the relation between daily stress and biological processes in young adults, it may be informative to consider the moderating influence of the childhood environment. More specifically, we found some evidence suggesting that difficult childhood environments may

make individuals more susceptible to the detrimental effects of daily stress in adulthood, while positive characteristics of the childhood environment may buffer individuals from stress-related daily biological changes. As well, our findings provide further insight about *daily* life experiences with stress and biological responses, and represent a more ecologically valid approach to measuring stress. However, the research was not without limitations.

First, childhood environmental factors were measured retrospectively, and individuals may not have been able to accurately recall family environments years later. Second, because we only measured childhood environmental characteristics, we cannot rule out the influence of the current psychosocial environment on the relation between daily stress and biological outcomes. Third, our measure of daily stress may not have captured certain aspects of the stress process, including whether the stressor was socially relevant (Dickerson & Kemeny, 2004), or whether the stressor occurred within the context of chronic stress (Miller, Chen, & Zhou, 2007; Marin, Martin, Blackwell, Stetler, & Miller, 2007).

While this research provides an important step forward in understanding potential pathways through which childhood psychosocial factors create vulnerability for disease later in life, future studies are needed that assess these types of patterns across the lifespan. Studies of this kind would provide a better understanding of whether there are critical periods that exist during which the childhood psychosocial environment may be particularly protective or potent, as well as for how long childhood environments continue to affect daily stress and biology relationships. Other research suggests that critical periods may occur during the first two years of life (Cohen, Doyle, Turner, Alper, & Skoner, 2004; Miller & Chen, 2007), or during puberty (Stroud, Papandonatos, Williamson, & Dahl, 2004; Walker, Sabuwalla, & Huot, 2004). If such critical periods are identified, interventions could be designed to minimize negative and maximize positive environmental characteristics, or to

teach coping responses to stress during those periods, that would protect individuals from the harmful effects of stress on health.

The present study provided a first step toward this goal by documenting some evidence for the notion that difficult childhood environments may have long term detrimental effects on how biological processes respond to daily stress in adulthood. Understanding such moderating influences is important for identifying individual differences in biological responses to daily stress and for targeting interventions that will hopefully improve the health of all individuals throughout the lifespan.

Table 3.1

Sample Descriptive Statistics

| <u>Variable</u> | <u>Mean (SD)</u> | <u>Range</u> |
|-----------------|------------------|--------------|
| Age (N=87) | 21.51(1.84) | 19-25 |
| Gender | | |
| Male | 33.3% | |
| Female | 66.7% | |
| Ethnicity | | |
| Caucasian | 27.6% | |
| Asian | 57.5% | |
| Other | 14.9% | |

Table 3.2

Descriptive Statistics of Study Variables

| <u>Variable</u> | <u>Mean (SD)</u> | <u>Range</u> |
|------------------------|------------------|---------------|
| Daily Process Measures | | |
| Stress | | |
| Number of stressors | 3.55 (1.91) | 0.57-8.43 |
| Severity of stressor | 3.17 (0.81) | 1.80-4.71 |
| Sleep | | |
| Sleep minutes | 419.69 (61.18) | 259.50-548.42 |
| Sleep efficiency | 83.68 (6.03) | 62.81-93.38 |
| Cortisol (N=374 days) | | |
| Total AUC | 9.07(4.29) | 1.31-36.13 |
| Background Measures | | |
| Risky Families (N=87) | 1.98 (0.50) | 1.08-3.23 |
| Parental Warmth (N=87) | 34.23 (7.53) | 11.00-48.00 |

Note. Values of daily process variables were aggregated across days. Possible ranges for number of stressors: 0-16; stress severity: 1-5; risky families: 1-5; parental warmth: 0-48.

Table 3.3

Multilevel Variance Components for Daily Stress and Biological Marker Variables

| | Between- person variance (Tau) | Within- person variance (sigma ²) | Percent within- person variance | Percent between- person variance | Variance Coefficient |
|----------------------|---|--|--|---|-------------------------|
| Sleep minutes | 2264.18 | 8650.63 | 79.30% | 20.74% | 2264.18*** |
| Sleep efficiency | 22.61 | 82.95 | 78.60% | 21.42% | 22.61*** |
| Stressor number | 3.05 | 2.83 | 48.13% | 51.87% | 3.05*** |
| Stressor severity | 0.46 | 1.09 | 70.32% | 29.68% | .46*** |
| Cortisol AUC | 3.26 | 15.16 | 82.30% | 17.70% | 3.26*** |

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 3.4

Hierarchical Linear Models Testing the Effects of Daily Stressors, and the Moderating Effects of Childhood Family Risk, on Sleep and Cortisol Outcomes

| | Coefficient (SE) | <i>p</i> |
|--------------------------------|------------------|------------|
| <u>Sleep Minutes</u> | | |
| Predictor variable: | | |
| Stress Number | -1.94 (2.82) | .49 |
| Stress Severity | -4.56 (4.97) | .36 |
| Risky Family | 8.01 (14.70) | .59 |
| Stress Number x Risky Family | -10.66 (6.19) | .02 |
| Stress Severity x Risky Family | -12.10 (10.57) | .26 |
| <u>Sleep Efficiency</u> | | |
| Stress Number | 0.25 (0.24) | .31 |
| Stress Severity | -0.39 (0.41) | .34 |
| Risky Family | 0.02 (1.46) | .99 |
| Stress Number x Risky Family | 0.25 (0.50) | .62 |
| Stress Severity x Risky Family | 0.42 (0.67) | .53 |
| <u>Cortisol Output (AUC)</u> | | |
| Stress Number | 0.16 (0.19) | .40 |
| Stress Severity | 0.23 (0.40) | .57 |
| Risky Family | -0.42 (0.67) | .53 |
| Stress Number x Risky Family | 0.51 (0.56) | .37 |

Cortisol Output (AUC)

| | | |
|--------------------------------|-------------|-----|
| Stress Severity x Risky Family | 1.66 (1.01) | .11 |
|--------------------------------|-------------|-----|

Note: Ethnicity was controlled for in all stress severity analyses. Each predictor variable was modeled separately, such that each line of the table represents a different model. Stress number, stress severity, and risky family models are main effects models, whereas stress number x risky family and stress severity x risky family are moderator models. Stress number and severity predicting biological markers are level-1 models, whereas risky family predicting biological markers are level-2 models.

Table 3.5

Hierarchical Linear Models Testing the Effects of Daily Stressors, and the Moderating Effects of Parental Warmth, on Sleep and Cortisol Outcomes

| | Coefficient (SE) | <i>p</i> |
|-----------------------------------|------------------|----------|
| <u>Sleep Minutes</u> | | |
| <u>Predictor variables :</u> | | |
| Stress Number | -1.94 (2.82) | .49 |
| Stress Severity | -4.56 (4.98) | .36 |
| Parental Warmth | -1.26 (0.89) | .16 |
| Stress Number x Parental Warmth | 0.21 (0.29) | .48 |
| Stress Severity x Parental Warmth | -0.04 (0.67) | .95 |
| <u>Sleep Efficiency</u> | | |
| Stress Number | 0.25 (0.24) | .31 |
| Stress Severity | -0.39 (0.41) | .34 |
| Parental Warmth | -0.11 (0.09) | .24 |
| Stress Number x Parental Warmth | -0.01 (0.04) | .86 |
| Stress Severity x Parental Warmth | -0.02 (0.04) | .59 |
| <u>Cortisol Output (AUC)</u> | | |
| Stress Number | 0.16 (0.19) | .40 |
| Stress Severity | 0.23 (0.40) | .57 |
| Parental Warmth | -0.03 (0.05) | .60 |

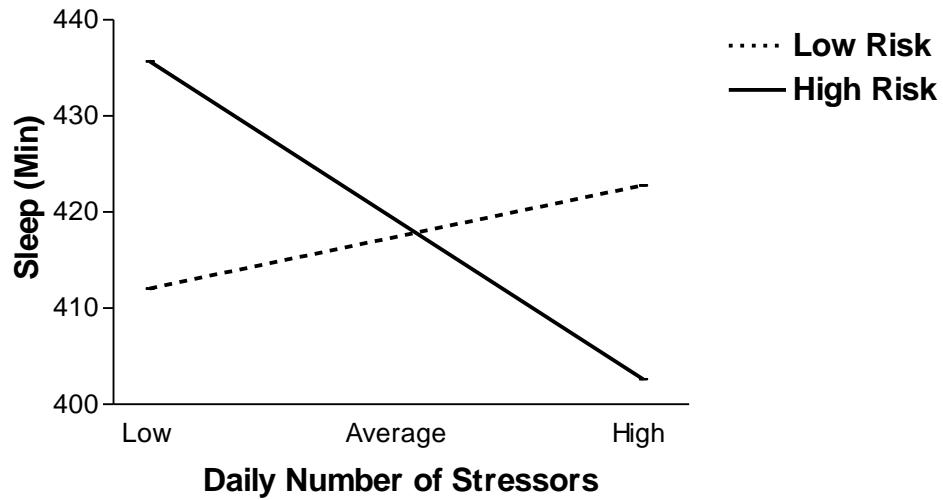
Cortisol Output (AUC)

| | | |
|-----------------------------------|--------------|------------|
| Stress Number x Parental Warmth | -0.05 (0.04) | .23 |
| Stress Severity x Parental Warmth | -0.16 (0.07) | .04 |

Note: Ethnicity was controlled for in all stress severity analyses. Each predictor variable was modeled separately, such that each line of the table represents a different model. Stress number, stress severity, and parental warmth models are main effects models, whereas stress number x parental warmth and stress severity x parental warmth are moderator models. Stress number and severity predicting biological markers are level-1 models, whereas parental warmth predicting biological markers are level-2 models.

Figure 3.1

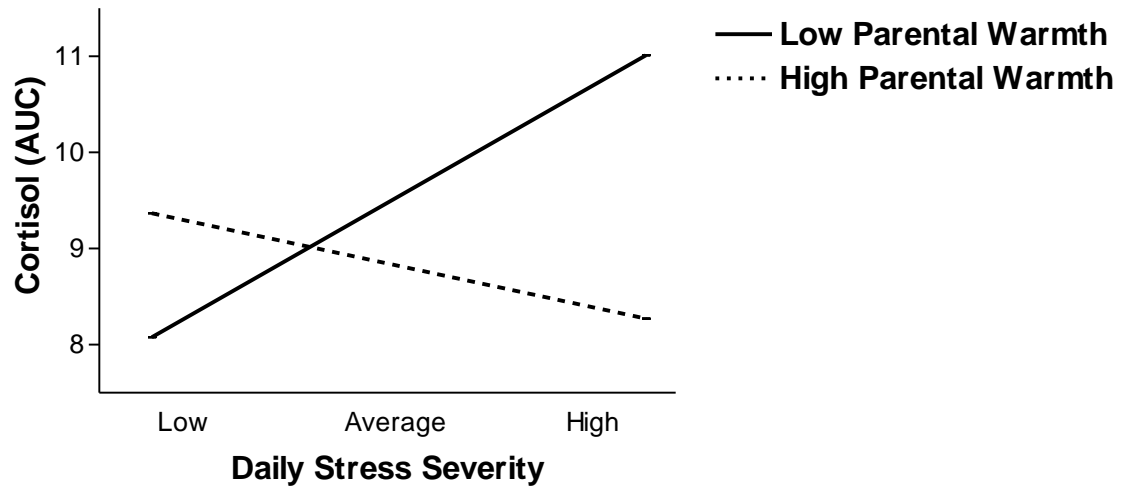
Childhood Family Risk Moderates the relation between Daytime Stress and Nighttime Sleep.



For individuals from high risk families, on days when they experienced more than their average number of stressors they slept for fewer minutes than on days when they experienced fewer than average stressors. However, in individuals from low risk families, minutes asleep did not change significantly as a function of daily stressful experiences.

Figure 3.2

Childhood parental warmth moderates the relation between daily stress and cortisol output.



In individuals from backgrounds low in parental warmth, on days when they experienced more severe stressors than their average they had higher cortisol output than on days when they experienced less severe stressors than average. However, in individuals from backgrounds high in parental warmth, cortisol output did not change significantly as a function of daily stressful experiences.

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General Discussion

Psychological stress is a risk factor for poor health. Previous research has indicated that both psychological and biological responses to stress may modify the impact of stress on health, hence serving as important pathways linking stressful experiences to physical illness. However, it is unclear whether stress is related to health on a daily basis, and if so, what the pathways are through which stress “gets under the skin” to impact daily health symptoms. Therefore, the aims of the present study were to examine whether psychological and biological responses to stress serve as potential pathways linking daily experiences of stressful life events and health symptoms in a sample of healthy, young adults. As well, based on previous research that has indicated heterogeneity in biological responses to stress, we aimed to assess whether childhood psychosocial factors might moderate the relation between daily stress and daily biological rhythms.

The first aim of the study was to determine whether, in a daily diary context, stressful experiences were related to health symptoms. Previous studies have reported that psychological stress increases an individual’s risk for diseases including cardiovascular disease, cervical cancer, autoimmune disorders, infectious diseases, and mental illnesses (Fang, et al., 2008; McEwen, 1998). Studies have also shown that stressful life events are associated with health symptoms, including pain (Affleck, Tennen, Urrows, & Higgins, 1994), fatigue (Schanberg, et al., 2000), and other somatic symptoms (Walker, Garber, Smith, Van Slyke, & Claar, 2001). However, the present study sought to determine whether stress predicted health symptoms *on a daily basis*. We hypothesized that reports of greater daily stress would predict more daily health symptoms.

Results confirmed our hypotheses and were consistent with previous findings. More specifically, we found that, on days when individuals experienced more severe stress, they reported greater health symptoms than on days when those same individuals experienced less severe stress. These findings add to our understanding of the effects of stress on health by indicating that stress is related to changes in health on a day-to-day basis.

Psychological Pathways

Emotion

Next, we aimed to understand the potential psychological pathways linking daily stressful experiences to greater health symptoms. In particular, we considered whether an individual's emotional reaction to a stressful life event explained some of the variance in the stress-health symptom association. Previous research that has tested whether emotional responses to stress mediated the relation between stress and health reported that participants who responded with more negative affect had poorer immune functioning (Labott, Ahleman, Wolever, & Martin, 1990; Stone, Cox, Valdimarsdottir, Jandorf, & Neale, 1987; Stone, Neale, Cox, Napoli, Valdimarsdottir, & Kennedy-Moore, 1994; Stone, Reed, & Neate, 1984), and were more susceptible to catching a cold after being exposed to rhinovirus (Cohen, Frank, Doyle, Skoner, Rabin, & Gwaltney, 1998). Hence, we hypothesized that negative emotional responses would mediate the relation between daily stress and health symptoms.

Indeed, we found that, on days when participants experienced more stress, they reported greater negative emotional responses to stress compared to days in which those same individuals reported less stress. As well, on days when participants reported more negative emotional responses to stress, they also reported greater health symptoms. Finally, we found that the association between daily stress severity and daily health symptoms became non-

significant when emotional responses to stress were included in the model, indicating that the stress-health association was significantly mediated by negative emotions. These results are in line with previous research, and builds upon previous work by documenting that emotional responses to stress mediated the relation between daily stress and daily health *symptoms* within a healthy population. Emotions may serve to reflect the subjective perception of severity of the stressor, how much a stressor interferes with personal goals, and how much a stressor taxes a person's resources. Overall, these findings indicate that negative emotional responses to stressful life events may increase the impact of stress, resulting in more significant daily health consequences as compared to stressful events that do not elicit the same level of emotional reaction. However, we did not find evidence for alternative directional pathways, that is, that stress operates via health symptoms to affect negative emotions, or that negative emotions operate via severity of daily stressors to affect health symptoms.

Coping

We also considered whether another psychological process, coping, explained why participants reported greater health symptoms on days when they experienced more stress. We chose to assess coping in particular because previous research has indicated that the use of coping strategies in response to stressful events has implications for health. More specifically, research has shown that the use of avoidance coping strategies in response to daily life stressors predicted greater health symptoms (Billings & Moos, 1981; Freeman & Gil, 2004; Morgan & Spiegel, 1987; Nowack, 1988; Park, Armeli, & Tennen, 2004). As well, previous work has reported that the use of approach coping strategies in response to stress may attenuate the impact of stress on self-reported health symptoms (Billings & Moos, 1981; Morgan & Spiegel, 1987). Therefore, we predicted that the use of coping strategies in

response to daily stressors would mediate the relation between daily stress and health symptoms.

However, coping responses to stress did not serve to significantly mediate the stress-health relationship in this study. Results showed that greater stress severity predicted greater use of all types of coping approaches; however daily coping was not associated with daily health symptoms, suggesting that the type of coping strategy selected may not buffer individuals from the negative consequences of daily stress on health. These findings indicate that simply engaging in coping strategies is not necessarily beneficial for health, but perhaps only when one engages in coping strategies that *effectively* manage stress are there health benefits (Zautra, 2003).

Biological Pathways

Cortisol

We also considered whether daily cortisol output could significantly explain why, on high-stress days, individuals reported a greater number of health symptoms. Cortisol has been implicated in a number of health problems, including increased risk for cardiovascular disease, the metabolic syndrome, and cancer (Bjorntorp & Rosmond, 1999; Sephton, Sapolsky, Kraemer, & Spiegel, 2000; Smith, Ben-Shlomo, Beswick, Yarnell, Lightman, & Elwood, 2005). Previous studies have identified cortisol as a potential mediator in the stress-health association; however the results have been mixed, with some studies reporting that greater cortisol responses to stress predicted increased risk for poor health (Rosmond, Dallman, & Björntorp, 1998) and others reporting non-significant findings (Miller, Cohen, Pressman, Barkin, Rabin, & Treanor, 2004). We hypothesized that, on days when participants reported greater stress, they would have greater cortisol output, which would in

turn predict greater health symptoms. However, in the present study, daily stress was not significantly associated with daily cortisol output. This is in contrast to previous studies that have reported daily stressful experiences to predict increases in cortisol secretion (Peeters, Nicholson, & Berkhof, 2003; Schlotz, Schulz, Hellhammer, Stone, & Hellhammer, 2006; Smyth, Ockenfels, Porter, Kirschbaum, Hellhammer, & Stone, 1998; van Eck, Nicolson, Berkhol, & Sulon, 1996).

Recent research suggests that stress may lead to changes in cortisol secretion only in individuals who are predisposed to be biologically sensitive to stressors, such as individuals who come from difficult childhood environments (Luecken, 1998; Repetti, Taylor, & Seeman, 2002). The fact that there were no direct effects of daily stress on daily cortisol suggests that approaches that focus on moderating psychosocial influences may be more useful than analyses of the main effects of daily stress on biological processes. Therefore, the second aim of this program of research was to determine whether the relation between daily stress and cortisol output was moderated by the childhood psychosocial environment.

Stress and Cortisol: Childhood Environment as a Moderator

Past research has reported that difficult childhood environments predict increased cortisol response to stress in adults (Gorman, Mathew, & Coplan, 2002; Hennessy, 1997; Luecken, 1998; Repetti, Taylor, & Seeman, 2002; Rosenblum, Forger, Noland, Trost, & Coplan, 2001), and that nurturing childhood environments mitigate the effects of stress on cortisol output (Caldji, Diorio, & Meaney, 2000; Evans, Kim, Ting, Tesher, & Shannis, 2007; Meaney, 2001). Hence, we hypothesized that individuals from difficult childhood environments would display greater cortisol secretion on high-stress days, whereas

individuals from less difficult childhood environments would display attenuated cortisol secretion on high-stress days.

Consistent with the literature and in line with our hypotheses, results showed that the relation between daily stress and cortisol output was significantly moderated by the childhood psychosocial environment. Individuals from childhood environments characterized by less parental warmth had significantly higher cortisol output on days when they reported more stressors, whereas individuals from more nurturing childhood environments displayed less cortisol secretion on days when they reported more stressors. Hence, findings from the present study suggest that difficult childhood environments may have effects that persist into adulthood in terms of how biological processes respond to daily stress. However, we note that we did not find the childhood parental warmth to moderate the association between daily stress severity and cortisol output, nor did we find that childhood family risk moderated the relations between daily stress and cortisol output.

Sleep

We also considered the impact of daily stress on another biological process: sleep. Sleep problems have been identified as risk factors for diseases including cardiovascular diseases and psychiatric difficulties (Breslau, Roth, Rosenthal, & Andreski, 1996; Newman, et al., 2001). Previous work has also demonstrated that stress is related to disturbed sleep (Åkerstedt, 2007; Ancoli-Israel & Roth, 1999; Hall et al., 2000; Tworoger, Davis, Vitiello, Lentz, & McTiernan, 2005; Urponen, Vuori, Hasan, & Partinen, 1988). However, previous research has indicated that significant associations between stress and sleep are often only apparent in certain groups (Dagan, Zinger, & Lavie, 1997; Sadeh, Keinan, & Daon, 2004; Pillar, Malhotra, & Lavie, 2000). Therefore, we sought to determine whether other factors

might moderate the relation between daily stress and sleep, and in particular, focused on characteristics of the childhood environment.

Stress and Sleep: Childhood Environment as a Moderator

There is some evidence to suggest that the relation between stress and sleep may be moderated by characteristics of the childhood environment. More specifically, past research has reported that adults who were raised in difficult childhood environments displayed greater sleep disturbances after stress (Gregory, Caspi, Moffitt, & Poulton, 2006; Reite & Snyder, 1982), and that nurturing childhood environments predicted longer sleep times during adolescence (Adam, Snell, & Pendry, 2007). Hence, we hypothesized that the childhood psychosocial environment would moderate the relation between daily stress and sleep, such that individuals raised in difficult childhood environments would display poorer sleep (shorter in duration, lower efficiency) on high-stress days, whereas the relation between stress and sleep would be attenuated in individuals from less difficult childhood environments.

Results partially confirmed our hypothesis; individuals who were raised in difficult childhood environments had shorter sleep times on high-stress days, while individuals who were raised in more nurturing childhood environments displayed longer sleep times on days when they reported more stress. However, the childhood psychosocial environment did not serve to moderate the relation between daily stressful experiences and sleep efficiency. Taken together, results indicate that stress is associated with shorter sleep times in individuals from difficult childhood environments. However, in individuals from more positive childhood environments, there may be compensatory biological processes such as ‘rebound sleep,’ following stressful days. Overall, these findings suggest that individuals

from difficult childhood environments may be at greatest risk for heightened cortisol secretion and reductions in sleep on days when they experience stress.

Strengths and Limitations of the Study

There are several noteworthy strengths of the present set of studies. First, we collected objective biological markers that have been shown in previous research to change under conditions of daily stress and are associated with health: cortisol and sleep. By gathering information on daily cortisol and sleep patterns, we were able to assess whether these biological processes served as pathways linking daily stress to health symptoms. In other words, we were able to test whether stress “gets under the skin” to effect health via changes in cortisol or sleep patterns.

A second strength of this set of studies is that we utilized a daily diary methodology in order to examine the pathways linking daily stressors to health outcomes. This study design was beneficial, in part, because there is some evidence that daily stressors are better predictors of health than major life events (Weinberger, Hiner, & Tierney, 1987). As well, an investigation of how individuals respond psychologically and biologically to stressors as they naturally occur provides a better understanding of how stress may, over time, manifest into disease.

In addition to our interest in assessing how psychological and biological processes mediate the relation between daily stress and health, we were also interested in assessing whether characteristics of the childhood environment moderated the daily stress-health association. The majority of previous research addressing this question has been laboratory based or cross-sectional, and it is important to understand how childhood environments may affect the relationship between naturally occurring stress and daily psychological and biological patterns (van Eck, Nicolson, Berkhof, & Sulon, 1996). Therefore, a daily diary

methodology helped to build upon findings from previous research. As well, the daily diary study design enabled us to perform analyses using hierarchical linear models. This allowed us to assess the relationships between stress, health, and the biological and psychological pathways for each individual (i.e., within-person), rather than aggregating variables across the sample and then assessing associations.

Finally, a strength of the present set of studies is that we considered multiple pathways that may explain the relation between stress and health. More specifically, we first considered whether psychological and biological processes mediated the daily stress-health relation, and found that, while certain psychological responses to stress operated as a significant mediator, biological responses did not, as daily stress did not predict cortisol or sleep patterns. However, we then considered whether the childhood psychosocial environment served to *moderate* the relation between stress and biological markers. We found that individuals from more difficult childhood environments had greater cortisol secretion and poorer sleep on high stress days, whereas for individuals from positive childhood environments, the patterns were reversed. The fact that there were no direct effects of daily stress on daily cortisol or sleep suggests that approaches that focus on moderating psychosocial influences may be more useful than analyses of the main effects of day-to-day stress on biological processes.

However, the study is not without limitations. While one of the aims of the study was to determine whether daily stress was related to daily health symptoms, health symptoms were gathered via self-report. Therefore, the conclusions that we can draw are limited. More specifically, it is unclear whether, on high-stress days, individuals are more likely to experience actual changes in health, are more likely to notice health symptoms, or are more likely to endorse health symptoms when completing the daily diary. In future studies, more

objective markers of daily health status should be gathered, such as school or work absenteeism, aspirin use, or doctor visits, in order to determine the nature of the relationship between daily stress and daily health.

As well, this study was limited in that we did not assess health behaviors, and therefore we cannot determine whether health behaviors also serve to mediate the relation between daily stress and daily health. Health behaviors, such as exercise, may help to regulate mood, decrease negative emotional responses to stress, and in turn, buffer an individual from the negative effects of stress on health (Brown & Siegel, 1988). Clearly, exercise also has health-promoting benefits (Rosengren & Wilhelmsen, 1997). Conversely, if an individual chooses to engage in detrimental health behaviors, such as substance use, in order to distract themselves from the stressor, they may also experience greater health symptoms, such as headache, upset stomach, or nausea.

Another limitation was that our sleep measure (actigraphy) was not able to detect sleep stages. Previous work has reported long-term changes in sleep architecture as a result of the childhood environment (Reite & Snyder, 1982), and different sleep stages may serve different health-promoting functions (Wyatt, Ritz-De Cecco, Czeisler, & Dijk, 1999). Therefore, our study was limited in being able to assess only sleep duration and efficiency, but not daily sleep architecture.

In addition, the conclusions that we can draw from the findings regarding the impact of the childhood psychosocial environment on biological responses to stress in adulthood are limited by the fact that we do not have information on the current psychosocial environment. First, childhood environmental factors were measured retrospectively, and individuals may not have been able to accurately recall family environments years later. As well, it may be that individuals from difficult childhood environments may continue to struggle

interpersonally into adulthood. Hence, heightened biological responses to stress in adults from difficult childhood backgrounds may have, in fact, been a product of difficult adult environments. In addition, we were not able to infer from the data whether ‘critical periods’ exist during childhood during which the psychosocial environment has the most potent effects on the long-term development of stress-response systems. In sum, future studies are needed that gather information on the psychosocial environment of individuals at multiple time points throughout childhood and adulthood in order to determine when the environment exerts the greatest influence on biological responses to stress.

There are also limitations of the questions that we asked participants to complete via daily diary. First, our daily stress checklist was not able to distinguish among chronic, acute, or daily hassles. As well, it does not differentiate stressors that are interpersonal in nature from those that do not contain a social component. Therefore, our understanding of stressful experiences lacks some details that may be important in terms of the impact of stress on health. In terms of our measures of the psychological responses to stress, we only asked participants to note their responses to the most severe daily stressor rather than all stressors. The use of other study designs to measure daily stressful events, such as event-based responding, would allow for the assessment of emotion and coping responses to a number of daily stressors, rather than just the most severe daily stressor. Finally, we only queried for *negative* emotional responses to stress, yet positive emotions have also been shown to be important for health in previous research (Affleck, Tennen, Croog, & Levine, 1987; Cohen, Tyrrell, & Smith, 1991; Danner, Snowdon, & Friesen, 2001).

Contributions to the Research Field

Despite these limitations, this study makes several important contributions to the existing literature. First, results from this study provide further insight about how *daily* life

experiences relate to psychological and biological processes. Findings from this study, therefore, represent a more ecologically valid account of how daily stress relates to daily health, as well as the mediating and moderating factors through which stress “gets under the skin” to impact health. It is important to understand the contribution that daily stressful experiences potentially make to disease onset and progression. As well, an understanding of these daily patterns may help to inform interventions.

This study builds upon previous work by documenting that emotional responses mediated the relation between daily stress and daily health *symptoms* (as opposed to disease outcomes) within a healthy population. Therefore, these findings indicate that negative emotional responses to stressful daily events may serve as an important pathway by which daily stress creates vulnerability for poor health. In addition, our mediation results demonstrated that negative emotional responses to stress explained the majority of the variance (around 70%) in the association between daily stress and health symptoms. This suggests that negative emotional reactions to stress may serve as a powerful measure of the subjective impact of stressful life events

Another important contribution that this study makes to the existing literature is the examination of the moderating influence of the childhood psychosocial environment on the relation between daily, naturally-occurring stress and daily cortisol output. The majority of previous research that has considered the effect of the childhood environment has been laboratory-based or cross-sectional, or has examined the *main* effects of childhood environment on biology. Therefore, in order to understand how childhood environments affect stress and daily biological rhythms, it is important to examine these relationships within a natural setting, hence providing a more ecologically valid picture of how stress may lead to poor health. Results from our study indicate that individuals from difficult childhood

environments may be more vulnerable to health problems because, on high-stress days, they display higher cortisol secretion, which over time could result in dysregulated HPA activity, and in turn, poor health.

Finally, this is the first study, to our knowledge, that has considered the moderating influence of the childhood psychosocial environment on the relation between daily stress and nightly sleep. Previous research has assessed how early life environmental characteristics moderate the relation between stress and biological responses, including HPA and immune system functioning, given the implications that these systems have for health outcomes. However, sleep is also a biological process that is affected by stress (Cheeta, Ruigt, Proosdij, & Willner, 1997; Meerlo, Koehl, van der Borght, & Turek, 2002) and is important for health (Zeman & Reading, 2005). As well, sleep patterns are established early in development and may be susceptible to environmental characteristics during this time. Therefore, it is necessary to understand the factors that contribute to sleep problems. Animal research has reported that environmental stress early in life has long-term implications for sleep (Reite & Snyder, 1982). Results from this study extended these findings into the human literature, indicating that individuals from difficult childhood psychosocial environments are more susceptible to sleep difficulties on high-stress days as compared to individuals from less difficult childhood environments.

Applying Study Findings

In addition to the contributions that this study makes to the larger body of research, findings from this study also may be applied to interventions. First, results from this study showed a main effect of daily stress severity on daily health symptoms. Therefore, one target for interventions may be altering the experience of stress itself. For example, interventions

that teach techniques to alter one's appraisal of stress may have beneficial effects for daily health symptoms.

In addition, results from this study showed that the relation between daily stress and health symptoms was partly explained by the negative emotional response to the stressor. This indicates that interventions targeting emotion regulation, such as identifying the positive (rather than only the negative) aspects of a stressor (i.e., seeing a 'silver lining'), may buffer the effects of stress on health. As well, stress management programs that incorporate ways of identifying and potentially modifying emotional responses to stress as a module of therapy may increase the effectiveness of the intervention. Indeed, previous intervention studies suggest that stress management programs may have beneficial effects on health. For example, in a meta-analysis by Miller and Cohen (Miller & Cohen, 2001), they reported a small to medium effect of stress-management interventions on immune functioning in psychologically stressed individuals. As well, in a study by Antoni and colleagues (Antoni, et al., 2000), HIV+ men enrolled in a cognitive behavioral stress management treatment exhibited decreased anger, improved mood, and greater numbers of T-cytotoxic/suppressor lymphocytes (indicative of better health) post-treatment. However it is unclear the degree to which these stress-management interventions focused on emotional responses to stress, or which module of therapy (relaxation, social support, cognitive restructuring, etc.) had the greatest influence on health.

Findings from this study also indicate that the childhood environment serves as a significant period during which biological markers of stress may be programmed. Therefore, at the policy level, educational programs may be designed for parents, teachers, and childcare providers that highlight the impact of family conflict or neglect on long-term health of the child. The aims of these programs would be to reduce early life family conflict and increase

parent-child bonding. Early psychosocial interventions may have long-term benefits for adult health.

Future Directions

Results from this study provide important information on the psychological and biological pathways linking daily stress to daily health, and indicate that childhood environmental characteristics may moderate the stress-biology relationships, in healthy college undergraduate students. These findings add to the larger body literature, while also introducing new questions that could be address in future studies.

First, the study sample was young and medically healthy. Results showed that, on high-stress days, participants reported greater health symptoms; however, there were no lagged effects, indicating that, in individuals without an existing illness, stress predicted short-term changes in health status. A future research direction, therefore, would be to consider whether daily stress triggers an episode of health symptoms spanning longer periods of time within a comparison sample of individuals who have an existing illness, such as cardiovascular disease, chronic pain, asthma, or rheumatoid arthritis. The rationale behind such a study would be that, if an individual has an illness characterized by dysregulated HPA axis functioning, daily stress could cause further dysregulation, greater allostatic load, and as a result, disease symptoms. As well, individuals with medical illnesses often experience poor sleep due to physical discomfort (Shapiro, Devins, & Hussain, 1993). Therefore, it may be that the biological pathways linking stress to health within medical populations are different from those in healthy populations.

Another future research direction would be to conduct a study examining the effects of stress on psychological and biological changes within a comparison group of individuals with a pre-existing psychological disorder, such as depression or post-traumatic stress disorder

(PTSD). Depressed individuals often display dysregulated cortisol profiles (Halbreich, Asnis, Shindlecker, Zumoff, & Nathan, 1985) and sleep disturbances (Breslau, Roth, Rosenthal, & Andreski, 1996). As well, there is some evidence to suggest that sleep disturbances are a central symptom of PTSD. In addition, both depressed and PTSD patients suffer from unpleasant emotions. Therefore, the biological and psychological pathways linking stress to health within samples of individuals with an existing psychological disorder may be different from those in healthy populations.

As well, future prospective studies are needed that track individuals from infancy through adulthood in order to determine whether ‘critical periods’ exist during which the psychosocial environment influences the development of biological responses to stress. Assessments would ideally be gathered every few years, with more frequent assessments occurring during the most likely ‘critical periods.’ More specifically, given that previous research has reported that the first 2 years of life may be a time during which biological systems are particularly susceptible to programming (Cohen, Doyle, Turner, Alper, & Skoner, 2004; Miller & Chen, 2007), a prospective study should include multiple assessments during the first few years of life, perhaps 6 months apart. As well, more frequent assessments may be gathered during puberty, since this may be another critical period during which biological processes are vulnerable to the influence of the psychosocial environment (Stroud, Papandonatos, Williamson, & Dahl, 2004; Walker, Sabuwalla, & Huot, 2004).

Future intervention studies could also be designed in order to determine whether the modification of emotional responses to stressful life experiences would have subsequent health benefits. According to previous research, individuals who are able to identify positive attributes of a stressful situation are at lower risk for health problems as compared to individuals who have more negative emotional responses to stress (Affleck, Tennen, Urrows,

& Higgins, 1994; Cohen, Tyrrell, & Smith, 1991; Danner, Snowdon, & Friesen, 2001).

Therefore, a stress management intervention could be designed that includes an emotion-module that teaches individuals how to monitor their emotional responses and reappraise the stressor in a more positive light, and follow-up health assessments could be conducted.

Overall, results from this study are consistent with the notion that psychological stress may have negative consequences for health on a daily basis. Results also suggest that the impact of daily stress on health may be particularly strong if individuals exhibit stronger negative emotional reactions to the stressor. Although we did not find evidence that biological processes served as a significant pathways linking daily stress to health, results did show that biological changes under conditions of stress varied according the individuals' childhood psychosocial environment, such that, on days when they experienced more stress, individuals from difficult childhood environments had greater cortisol secretion and poorer sleep than individuals from more positive childhood environments. These findings provide important information in terms of our understanding of the pathways linking naturally-occurring daily stressors to health. Results also highlight possible targets for intervention, including the modification of emotional responses to stressful events, as well as interventions that aim to improve the childhood psychosocial environment, in the hopes of minimizing the negative health consequences of stress.

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CERTIFICATE OF APPROVAL - MINIMAL RISK

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|--|---|--------------------------------------|
| PRINCIPAL INVESTIGATOR: Edith Chen | INSTITUTION / DEPARTMENT: UBC/Arts/Psychology, Department of | UBC BREB NUMBER: H07-02790 |
| INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT: | | |
| <small>Institution</small> | <small>Site</small> | |
| UBC Other locations where the research will be conducted: N/A | Vancouver (excludes UBC Hospital) | |
| CO-INVESTIGATOR(S): Margaret Hanson | | |
| SPONSORING AGENCIES: N/A | | |
| PROJECT TITLE: Biological Programming: Do early life psychosocial factors program the relation between stress and sleep in young adults? | | |

CERTIFICATE EXPIRY DATE: January 21, 2009

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|---|---|---------------------|
| DOCUMENTS INCLUDED IN THIS APPROVAL: | DATE APPROVED: January 21, 2008 | |
| <small>Document Name</small> | <small>Version</small> | <small>Date</small> |
| Protocol: Sleep research proposal | 1 | December 15, 2007 |
| Consent Forms: Sleep consent form | 1 | December 15, 2007 |
| Advertisements: Sleep study flyer | 2 | January 14, 2008 |
| Questionnaire, Questionnaire Cover Letter, Tests: Sleep study questionnaires | 1 | December 15, 2007 |
| The application for ethical review and the document(s) listed above have been reviewed and the procedures were found to be acceptable on ethical grounds for research involving human subjects. | | |
| <p>Approval is issued on behalf of the Behavioural Research Ethics Board and signed electronically by one of the following:</p> <p>-----</p> | | |

DAILY SYMPTOMS

In the past 24 hours, did you experience any of the following? (check all that apply):

- | | | |
|------------------------------|-----------------------------|-----------------------|
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Headache |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Backache |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Dizziness |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Nausea/upset stomach |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Heart pounding |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Constipation/diarrhea |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Muscle soreness |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Hot or cold flashes |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Shortness of breath |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Tightness in chest |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Low energy/tired |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Poor appetite |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Congestion |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Sore throat |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Runny nose |