EARLY LIFE INFLUENCES ON ADULT SLEEP AND HEALTH RISK INDICATORS:

EXAMINING MEDIATIONAL RELATIONSHIPS

by

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Abstract

Evidence suggests that early life stress increases risk for cardiovascular disease in adulthood. However, less is known about how early life experiences propagate risk into adulthood. The current study examines a potentially critical mediator of the relationship between early life stress and disease outcomes, namely poor sleep. Early life stress was hypothesized to contribute to poor sleep, as well as to other physiological health risk indicators (i.e., BP and BMI), by affecting adult psychosocial functioning. Further, poor sleep was examined as a mediator between adult psychosocial functioning and BP and BMI. An online sample and a university population sample completed measures of early life stress (i.e., a measure of childhood socioeconomic status and the risky family questionnaire), along with measures of adult psychosocial functioning and sleep quality. The university sample also participated in a 6-night actigraphy assessment to obtain objective sleep measurements. Results indicated that adult psychosocial functioning mediated the relationship between family risk in childhood and poorer subjective sleep quality for both sample populations. However, in the university sample, family risk also demonstrated a direct effect with respect to poorer subjective sleep quality, as well with respect to a latent factor reflecting physical restlessness and wakefulness during the sleep period. Family risk also demonstrated an indirect relationship with sleep length, largely via relationships with social support. Results did not suggest that family risk was associated with adult BP or BMI. However, in the university sample, social support, perceived stress, and negative affect were found to be significant predictors of BMI, and social support was found to be predictive of BP indices. Hypotheses examining sleep as a mediator of the relationship between psychosocial variables and BP and BMI were not well supported. Overall, results suggest that an adverse early childhood environment is a
potentially important determinant of adult psychosocial and sleep functioning, which in turn may increase disease risk.
Preface

This dissertation is an original intellectual product of the author, A. Talbot Ellis. No part of the research presented in this document has been published. The study presented in the dissertation was approved by the UBC Behavioural Research Ethics Board, certificate number H11-01981.
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Introduction

Cardiovascular diseases (CVD) remain the leading cause of death in Canada, accounting for approximately 29% of all deaths (Statistics Canada, 2012). Less than 50% of the variance of new cases of CVD can be explained by classic risk factors such as obesity, smoking and family history (Roig et al., 1987). As such, considerable research effort has focused on identifying psychosocial factors that may promote CVD. In particular, the role of psychological stress in the pathogenesis of CVD has received a great deal of attention (for review, see Schwartz et al., 2003). Prospective studies have indicated that chronic maladaptive responding in the face of stress, including heightened and prolonged physiological and affective responses to stress, plays a role in the development of risk factors for CVD including hypertension (Brosschot, Pieper, & Thayer, 2005; Treiber et al., 2001).

Therefore, identifying individual difference factors that increase the likelihood of maladaptive responding is of critical importance. An emerging body of research suggests that early life stress, such as growing up in an adverse childhood family environment or in a low socioeconomic (SES) household, may promote maladaptive alterations in stress response systems, thereby increasing vulnerability to the deleterious influences of stress over the lifespan (McEwen, 2003; Taylor, Lerner, Sage, Lehman, & Seeman, 2004). Indeed, early life stress has been linked to CVD in both cross-sectional and prospective studies (Galobardes, Smith, & Lynch, 2006).

Studies linking early life stress with CVD highlight several potential explanatory mechanisms with respect to this relationship, including maladaptive physiological, affective, and behavioural responses to stress (Luecken & Lemery, 2004; Taylor, Lerner, et al., 2004). However, current models largely fail to account for a potentially critical mediator of the
relationship between early life stress and health outcomes, namely, the role of physiological recovery via adequate sleep. Statistics Canada data estimate that approximately 3.3 million Canadians over the age of 15 years sleep 6.5 hours or less on an average night (Tjepkema, 2005). This chronic sleep deprivation may have important health implications as less than 7 hours of sleep a night has been associated with increased rates of mortality and decreased cardiovascular health (Heslop, Smith, Metcalfe, Macleod, & Hart, 2002). Therefore, it is imperative to include processes occurring during the sleep period in models of CVD risk.

Poor sleep has been directly associated with several indices of early life stress, including a risky family environment and low household SES (Chapman et al., 2011; Tomfohr, Ancoli-Israel, & Dimsdale, 2010), and is also associated with proposed mediational pathways between early life stress and health outcomes, such as negative affect and perceived stress (Hanson & Chen, 2010; Mezick et al., 2009). Therefore, poor sleep may be a more proximal risk factor for deleterious health outcomes. This study seeks to test this hypothesis by examining the indirect influence of early life stress on risk factors for CVD (i.e., poor sleep, resting blood pressure (BP) and body mass index (BMI)) via relationships with negative affect, perceived stress and social support, and also explores poor sleep as a proximal mediator of the relationship between early life stress and BP and BMI.

Defining Early Life Stress

During childhood an individual’s survival and well-being rests in the hands of their caregivers. Warm and responsive caregiving creates an internal representation of the world as a safe and predictable place, setting the stage for the development of adaptive coping and resilience to stressful life events (Lopez & Brennan, 2000). However, not all children receive adequate caregiving, making these children more vulnerable to deleterious psychological and
physical health outcomes. At the extreme end, children may be subjected to environments in which they are the victims of outright physical, emotional, or sexual abuse. Child maltreatment is quite prevalent in Canadian society. For instance, the Canadian Incidence Study of Reported Child Abuse and Neglect (2008) revealed 85,440 new cases of substantiated child abuse in Canada in 2008 alone, of which 34% involved exposure to intimate partner violence, 34% involved neglect, 20% involved physical abuse, 9% involved emotional maltreatment, and 3% involved sexual abuse. Notably, these statistics refer only to substantiated claims of child maltreatment, meaning many more cases have likely not been identified. Further, many households may establish negative environments which limit adaptive child development but which are not characterized by outright abuse.

Data from the Adverse Early Childhood Experiences (ACE) study involving over 17000 Kaiser Permanente Health Plan members, suggest that several factors indicative of an adverse family environment, though not necessarily classified as abuse, are also common. For example, over 25% of subjects reported having been exposed to drug and alcohol abuse, approximately 19% reported parental psychopathology, and approximately 23% reported parental separation or divorce. Altogether, over half of all participants (64%) reported at least one adverse childhood experience (e.g., including emotional, physical, or sexual abuse, but aspects of household dysfunction), and 12.5% reported having experienced four or more adverse childhood experiences (Anda et al., 2006).

At an even broader level, childhood environments may be characterized by more insidious factors such as family conflict or cold and hostile or neglectful parenting. In the current study, we focus on risky family environments as the primary indicator of early life stress. Risky family environments are those that are characterized by family conflict,
aggression, household dysfunction, and lack of parental warmth and affection. They are termed “risky” due to the fact that they are associated with increased vulnerability to mental and physical health outcomes (Repetti, Taylor, & Seeman, 2002; Taylor, Lerner, et al., 2004). We chose to focus on risky families because while this type of environment may indeed capture families exhibiting more extreme forms of child maltreatment, not all risky families are characterized by abuse. Thus, this conceptualization also captures family dysfunction that falls within “normal” bounds, thereby allowing us to capture a broader spectrum of early life stress (Taylor, Lerner, et al., 2004).

In addition to risky family environment, childhood socioeconomic status (SES) is considered an indicator of early life stress. Low SES may be associated with chronic stressors such as financial hardship, lack of access to resources, and increased risk of exposure to violence or criminal behaviour (Bradley & Corwyn, 2002). Low SES is also considered a broader environmental factor contributing to a risky family environment as it may correspond to increased family conflict due to chronic stress, low parental warmth, and inconsistent parenting and instability within the household (Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Dodge, Pettit, & Bates, 1994).

Mental and Physical Health Outcomes of Early Life Stress

**Childhood consequences of early life stress.** Life stress experienced in childhood places children at risk for impairment across behavioural, psychosocial, and even cognitive domains (see Cicchetti & Toth, 2005; Crouch & Milner, 1993; Trickett & McBride-Chang, 1995). For example, research on the sequelae of child maltreatment has shown that children who have suffered abuse or neglect are more likely to develop internalizing and externalizing behavioural problems (Bolger & Patterson, 2001; Pears, Kim, & Fisher, 2008), experience
greater difficulty with emotion regulation and aggression (Chang, Schwartz, Dodge, & McBride-Chang, 2003; Shields & Cicchetti, 1998), are more often rejected by their peers (Bolger, Patterson, & Kupersmidt, 1998), and tend to be less socially competent (Manly, Cicchetti, & Barnett, 1994; Shields, Cicchetti, & Ryan, 1994) than children who have not suffered maltreatment. Further, experiencing abuse, neglect, or household dysfunction (e.g., parental substance use or depression, intimate partner violence) in early childhood has been associated with poorer health as rated by caregivers and with greater rates of illness requiring medical attention in childhood and early teenage years (Flaherty et al., 2009; Flaherty, Thompson, Litrownik, & et al., 2006). Similar developmental outcomes have been found for children in homes with overt family or marital conflict (Cummings & Davies, 2002; Davies & Cummings, 1994; Grych & Fincham, 1990; Katz & Gottman, 1993) and lack of parental responsiveness and warmth (Davidov & Grusec, 2006). As well, children from lower socioeconomic (SES) groups are more likely to experience these various forms of maladjustment (for review see Bradley & Corwyn, 2002).

Adult consequences of early life stress. Childhood maladjustment associated with early life stress also persists into adulthood. Meta-analytic studies have provided support for the supposition that childhood adversity translates to poorer adult mental health outcomes including depression, anxiety, and increased risk of suicidality (Paolucci, Genuis, & Violato, 2001). This association has been shown to hold when assessed internationally. For example, Kessler et al. (2010) found that retrospective recall of childhood adversity was associated with higher rates of mental health issues such as mood and anxiety disorders, behavioural disorders and substance abuse, particularly when the adversity was specific to maladaptive family functioning (e.g., child abuse, neglect, family violence, parental mental illness).
Further, results from the ACE Study have revealed graded associations between retrospective accounts of childhood adversity and adult mental health (e.g., depression, anxiety, anger, suicide attempt), as well as physical health (e.g., ischemic heart disease, cancer, chronic lung disease, liver disease and increase risk of premature death) (Anda et al., 2006; Brown et al., 2009; Chapman et al., 2004, 2007; Dong et al., 2004; Dube et al., 2001; Felitti et al., 1998).

In a Canadian sample, child abuse and adversity was similarly associated with adult health, including report of multiple health conditions, poor self-rated health, pain, and medical services utilization (Chartier, Walker, & Naimark, 2010; Chartier, Walker, & Naimark, 2007). Early life stress in the form of low SES has also been linked to adult health.

Systematic reviews have found near uniform evidence for an association between low childhood SES and all-cause mortality, including increased risk of mortality from CVD (e.g., coronary heart disease, stroke), cancer, and respiratory disease, irrespective of gender and adult SES (Galobardes, Lynch, & Smith, 2004; Galobardes et al., 2006; Pollitt, Rose, & Kaufman, 2005).

**Early life stress and cardiovascular disease risk factors.** Specific to this study, early life stress has also been associated with risk factors for CVD, including with poor sleep both in childhood (El-Sheikh et al., 2013; El-Sheikh, Buckhalt, Mark Cummings, & Keller, 2007; El-Sheikh, Kelly, Bagley, & Wetter, 2012; Rhoades et al., 2012) and adulthood (Bader, Schäfer, Schenkel, Nissen, & Schwander, 2007; Chapman et al., 2011; Koskenvuo, Hublin, Partinen, Paunio, & Koskenvuo, 2010), with higher rates of BP in adulthood (Blane et al., 1996; Janicki-Deverts, Cohen, Matthews, & Jacobs, 2012; Kivimäki et al., 2006; Kivimäki et al., 2006; Lehman, Taylor, Kiefe, & Seeman, 2009), as well as higher BMI (Al-Emrani, Stafström, & Östergren, 2013; Mattsson, Rönner, Juonala, Viikari, & Raitakari, 2008). As
such, these factors are explored as intermediary links between psychosocial sequelae of early life stress and ultimate disease outcomes.

**Physiological and Psychosocial Pathways to Disease Outcomes**

Mounting evidence reliably links adverse early life experiences to adult health outcomes; however, how this early risk propagates into adulthood is still under investigation. Converging empirical evidence suggests that differential development of neurobiological systems that guide behavioural, affective, and physiological responses to stress are likely at the root (Heim & Nemeroff, 1999; Meaney, 2001; Karlamangla, Friedman, & Seeman, 2011; Taylor, Lerner, et al., 2004). In turn, these maladaptive responses to stress are thought to contribute to the progression of disease states.

**Physiological responses to stress.** Allostatic load models (see Linden, Earle, Gerin, & Christenfeld, 1997; McEwen, 1998) help to explain why alterations in stress responsivity stemming from early life stress may confer to risk of disease over time. While the term stress is generally used to denote a negative physical and psychological state, the stress response is in fact adaptive as it alerts the body to recognize and cope with challenges within the environment and is therefore necessary for survival. The stress response involves physiological activation of stress response systems, such as the sympatho–adrenomedullary (SAM) and hypothalamic-pituitary-adrenal axis (HPA). Physiological activation of these systems releases catecholamine from the nerves and adrenal medulla, stimulating the release of corticotrophin from the pituitary, which acts to mediate the release of cortisol from the adrenal cortex (McEwen, 1998). Physiological activation also mobilizes energy stores, increases cardiovascular/pulmonary tone to facilitate oxygen delivery to tissue, and reduces digestion, inflammatory responses, and immune system functioning (Van Reeth et al., 2000).
Following activation, negative feedback systems return the body to baseline states. If negative feedback mechanisms are successful, the stress response is adaptive in alerting an individual to a challenge or threat in the environment, thus priming the body to physically deal with the stressor. Once the stressor is terminated, typically, so too is the stress response. However, stress responding can become extremely pathogenic if the stress response is frequent and prolonged. Stress response systems that are over- or under-active create pathogenic ‘wear and tear’ on the body. McEwen (1998) describes three types of physiological activation that make up allostatic load: frequent stress, failed shut down (chronic activity and failure to shut off the stress response, i.e., poor recovery), and inadequate response (failure to respond to a challenging stressor, causing other systems to become active).

In-keeping with allostatic load models, prospective studies have demonstrated that elevated or prolonged cardiovascular activation in response to a stressor (i.e., stress reactivity and recovery) predict increased tonic BP and the subsequent development of essential hypertension, known risk factors for CVD development (Menkes et al., 1989; Moseley & Linden, 2006; Stewart, Janicki, & Kamarck, 2006; Wood, Sheps, Elveback, & Schirger, 1984). Reactivity refers to the magnitude of elevation in an individual’s physiological response (for example, increased BP, HR and elevated levels of the stress hormone cortisol) to an aversive, challenging, or engaging stressor (Linden, Gerin, & Davidson, 2003; Schwartz et al., 2003). Over time, chronic stress induced elevations in HR, and especially BP, are thought to create lasting detrimental changes in cardiovascular functioning eventually leading to the development of CVD (Schwartz et al., 2003). In the past decade, attention has also turned to the role of delayed recovery in predicting CVD, wherein the
stress response system fails to shut down shortly after removal of a stressor, prolonging the 
chronicity of the stress response. Longitudinal studies have implicated delayed recovery in 
risk for CVD. For example, borderline hypertension was predicted by delayed recovery when 
assessed after a laboratory stress task (Stewart & France, 2001), and familial hypertension 
was predicted by slow recovery following a submaximal exercise task when assessed 10-
years later (Tanji et al., 1989). These results have been supported by meta-analytic data 
(Hocking Schuler & O’Brien, 1997).

Data stemming from animal and human models have implicated that family 
characteristics may be important determinants of physiological responses to stress early in 
life. For example, in rats and nonhuman primates maternal separation and quality of 
caregiving have been associated with increases in behavioural and physiological responses to 
stress such as increased HR and cortisol reactivity, both acutely and over the lifespan 
(Plotsky et al., 2005; Reite, Kaemingk, & Boccia, 1989). Additionally, toddlers provided 
with sensitive and responsive caregiving have shown attenuated cortisol elevations in 
response to stress compared to those with poorer quality caregiving (Nachmias, Gunnar, 
Mangelsdorf, Parritz, & Buss, 1996). Maternal caregiving has also been associated with 
delayed cortisol recovery (i.e., slowed time to return to baseline) in infants following mild 
daily stress (Albers, Riksen-Walraven, Sweep, & Weerth, 2008). Evidence suggests that 
these early alterations in stress reactivity and recovery may persist over time. For example, 
adults who had experienced parental loss or poor quality family relationships as children 
exhibited higher blood pressure reactivity and recovery following stress tasks than those with 
no such history, as well as higher cortisol levels in response to stress (Luecken, 1998; 
Luecken & Appelhans, 2006; Luecken, Rodriguez, & Appelhans, 2005). As such, early
family environment factors such as lower levels of paternal sensitivity and warmth, may promote risk of disease development by increasing allostatic load across the lifespan.

**Affective Pathway**

Given that emotional and physiological responses are inexorably linked, it is not surprising that the tendency to repeatedly experience negative affect, including depression, anxiety, and anger, has been associated with disease outcomes, including CVD (Gallo & Matthews, 2003; Rozanski, Blumenthal, & Kaplan, 1999; Rugulies, 2002). A substantial literature base has suggested this association may be in large part due to the relationship between paired negative affective and maladaptive physiological responses to stress.

Negative emotions can increase physiological reactivity to stress, as well as increase the chronicity of the stress response by delaying immediate recovery from stress. For example, perseverative cognitive factors (e.g., worry and rumination) associated with negative affect (e.g., with anxiety and depression, respectively) may serve to reactivate a stress response in periods outside of the actual stressor, or contribute to anticipatory stress (Brosschot et al., 2005). Accordingly, a recent meta-analysis by Chida and Hamer (2008) found that the tendency to experience anger/hostility was reliably associated with increased cardiovascular responses (i.e., increased BP and heart rate) to stress, particularly in men, and that anxiety and general negative affect were associated with poor cardiovascular recovery. Similarly, individuals who score higher on measures of depression have shown increased vulnerability to cardiovascular reactivity when confronted with a stressor (Carroll, Phillips, Hunt, & Der, 2007; Light, Kothandapani, & Allen, 1998). Therefore, the affective difficulties associated with early life stress may serve to exacerbate maladaptive physiological responses to stress.

**Parental contributions to emotion regulation.** Evidence suggests that children who
have experienced early life stress often fail to develop adaptive emotion regulation and coping strategies, increasing their vulnerability to elevated physiological and emotional responses to stress (Repetti et al., 2002), and subsequently, to negative health outcomes. Emotion regulation skills pertain to the ability to modulate emotional experience to attain desired affective states and adaptive outcomes and are considered a core requirement for adaptive psychological functioning (Lopes, Salovey, Côté, Beers, & Petty, 2005). Through modeling, sensitive responding, and open communication about emotions, as well as by scaffolding appropriate responses to emotional arousal, parents and caregivers are intrinsically involved in the development of emotion regulation skills (Morris, Silk, Steinberg, Myers, & Robinson, 2007). Failure to develop adaptive emotion regulation abilities is in turn associated with various forms of psychopathology, again implicating the role of early caregiving in adult mental and physical health (Aldao, Nolen-Hoeksema, & Schweizer, 2010).

**Early life stress and affective stress responses.** While early childhood experiences may shape the development of negative emotional characteristics in adulthood, they may also influence emotional responses to stress experienced as an adult. Life stress often precedes the onset of an episode of heightened depression or anxiety (Faravelli et al., 2012; Mazure, 1998). However, not everyone who experiences life stress develops a depressive or anxious reaction (Monroe & Reid, 2009). As noted above, several authors posit that early life stress, in combination with genetic vulnerability, serves to sensitize one to negative emotional reactions to stress (Espejo et al., 2007; Hammen, Henry, & Daley, 2000; Heim & Nemeroff, 2001), likely due to common underlying neurobiological adaptations. For example, hyperactivity of corticotrophin-releasing factor (CFS) is thought to mediate the relationship
between stress and the development of depression and anxiety, and has also been associated with early life stress (Heim & Nemeroff, 1999; Heim, Plotsky, and Nemeroff, 2004).

Several studies have found that current stressful life events mediate the relationship between early life stress and adult depression (Hankin, 2005; Turner & Butler, 2003; Uhrlass & Gibb, 2007). For example, Hazel, Hammen, Brennan, & Najman (2008) found that when assessed longitudinally, early life stress (such as financial hardship and parental discord) was predictive of life stress in adolescents, which in turn mediated the relationship between early life stress and current levels of depression. Further, women with exposure to one or more childhood adversities (e.g., parental marital problems, experiencing or witnessing violence in the family, parental psychopathology) were found to be more likely to experience a depression reaction following lower levels of total life stress than women who had not experienced any form of assessed childhood adversity (Hammen et al., 2000). This was independent of previous depressive episodes or current chronic stressors. Similar results were found with an adolescent sample (Espejo et al., 2007). Specifically, the study demonstrated that depressive symptoms were predicted not only by childhood adversity but also by a history of an anxiety disorder. The researchers suggest that this finding is likely reflective of underlying dysregulated stress response systems described above, potentially accounting for the high rate of comorbidity amongst these disorders. Evidence has also supported the stress sensitization hypothesis with respect to pathogenesis of PTSD and other anxiety disorders (McLaughlin, Conron, Koenen, & Gilman, 2010).

While McLaughlin et al. (2010) frame their study in terms of stress sensitization, they recognize that an alternative explanation also exists. Namely, consistent with a stress generation hypothesis, individuals who experience childhood adversities may be at increased
risk for experiencing negative life events in adulthood due to the individual’s personality and behavioural characteristics which affect interpersonal interactions (Hammen, 2005, 2006). Presumably, these models are not in competition; early life stress likely increases the probability of experiencing emotional reactions to lower levels of stress, and also increases the risk of experiencing stressful life events due to maladaptive interpersonal interactions.

Irrespective of actual number of stressful life events, the appraisal of events as stressful is likely an important contributing factor to emotional reactions to stress. Consistent with this hypothesis, findings from the study conducted by McLaughlin et al. (2010) also revealed that participants who endorsed higher levels of early life stress also tended to endorse higher levels of perceived stress at a given level of stressful life events. The authors suggest this finding may reflect poorer coping and greater emotional reactivity to stressful life events. Thus, in assessing the impact of early life stress on adult physical or psychological health, research should not ignore the impact of stress appraisal. This line of inquiry would be consistent with early work by Lazarus (1966) who introduced the cognitive mediation model of stress, which highlights the importance of appraisal in initiating a stress response. Specifically, an individual must first interpret a stimulus as a threat. This model helps to explain why a relatively benign life situation for one individual may be highly stressful for another and highlights the importance of subjective measures of stress when conceptualizing the impact of psychological stress on physical and psychological health.

Overall, the current literature would suggest that emotional responses to stress are likely heightened in individuals who experienced more adversity in childhood. Furthermore, both early life stress and negative emotionality are closely related to other pathways thought to contribute to poor physiological health, including effective use and attraction of social
support and sleep processes.

**Social Pathway**

Beginning with a seminal study by Berkman and Syme (1979), considerable research attention has been devoted to the role of social support in physical health outcomes. A number of epidemiological studies have found that individuals with low levels of social support suffer more deleterious health consequences and have higher rates of mortality, particularly from CVD (Berkman, Leo-Summers, & Horwitz, 1992; Brummett et al., 2001; Rutledge et al., 2004). A substantial portion of the literature has suggested that social support may mitigate the negative physiological impact of stress, in part by attenuating cardiovascular and neuroendocrine responses to stress (for review see Uchino, 2006), by facilitating physiological recovery (Cohen & Wills, 1985), as well as by attenuating psychological distress under stressful conditions (Cohen, 1988). In addition, social support has been shown to be protective against mental health difficulties, including depression, which also has direct implications for health (Aneshensel & Frerichs, 1982).

**Contribution of family environment to social support.** Just as the family context is thought to shape emotion regulation skills in children, socialization experiences within the family are thought to shape social competence. While child characteristics such as temperament or other personality factors contribute to the quality of children’s relationships, caregivers also play a role in the development of social skills by modeling appropriate social interaction, scaffolding their children’s early interactions, and helping to navigate conflicts (Luecken, Roubinov, & Tanaka, 2013). Additionally, as noted above, caregivers play a role in the development of adaptive emotion regulation abilities, which in turn have been found to influence social competence and quality of peer relationships as children develop (Eisenberg
Children from homes with high levels of conflict and aggression or that are characterized by cold, unsupportive or neglectful caregiving, tend to have fewer positive social skills, are more likely to engage in an aggressive or antisocial manner, and are more likely to be rejected or victimized by peers (Luecken et al., 2013; Repetti et al., 2002). Further, studies have demonstrated that parental warmth and emotional expression serve to shape children’s empathic responding, which also bears on social functioning (Zhou et al., 2002). Early psychosocial environment also appears to be related to several aspects of impaired social information processing, which impedes the ability to interact with others in an adaptive manner (Luecken et al., 2013; Repetti et al., 2002). For instance, children from chaotic, unpredictable, or threatening environments tend to be more easily alerted to potential danger in their environment and demonstrate greater vigilance to social threat cues and anger in others. This tendency has been found to impair their ability to attend to socially relevant cues and to think of effective behavioural responses to difficult social situations, this in turn increases the likelihood of ineffective responding (e.g., hostile or angry responding).

Importantly, childhood deficits in social competence are thought to contribute to difficulty establishing social support networks in adulthood, impairing the ability to benefit from the mental and physical health buffering effects of social support. A recent study found that adversity assessed in childhood was associated with smaller social network size, as well as higher subjective ratings of negative aspects of a select relationship (e.g., how often a selected social support unit, namely, the person closest to the rater, gave the rater worries, problems, or stress) when assessed in adulthood (Ford, Clark, & Stansfeld, 2011). In addition, these aspects of social support were associated with mood and anxiety symptoms in
adulthood. Despite these relationships, the study did not find support for their prediction that social support would mediate the relationship between early life stress and mental health indices. Notably, restricting the subjective perception of support to a single individual may not account for the cumulative perception of support amongst the entire social network. For example, while experiencing a stressful relationship with a spouse, one might turn to a close friend for support. Therefore, utilizing a more global measure of perceived social support may better capture relationships amongst these variables.

Sleep Pathway

Sleep is seen as playing a critical role in health maintenance. It is posited to be our main restorative period that has evolved to allow the body time to heal and consolidate information at the level of the central nervous system (Zeman & Reading, 2005), meaning that chronic sleep deprivation may have important health implications. Indeed, short and long sleep duration has been associated with increased rates of mortality and decreased cardiovascular health in a series of meta-analyses (Cappuccio, Cooper, D’Elia, Strazzullo, & Miller, 2011; Gallicchio & Kalesan, 2009). Therefore, research has been aimed at better understanding sleep disturbance.

A substantial body of literature has linked sleep disturbance with acute stress (though some evidence has been mixed) (Van Reeth et al., 2000). For example, sleep disturbances have been documented following acute stress such as trauma and marital separation (Cartwright & Wood, 1991; Lazarus, 1993), as well as following daily stress (Hanson & Chen, 2010). Evidence linking stress and sleep has also derived from studies showing changes in sympathetic activation involving HPA axis hormones following stress. Typically, cortisol and corticotrophin levels are at a minimum within the first period of the sleep cycle.
and increase with lighter sleep, becoming maximal shortly after sleep wakening (Van Reeth et al., 2000). However, higher levels of nighttime corticotrophin and cortisol have been associated with daily stress, and in turn have been associated with greater sleep latency (i.e., the time it takes to fall asleep), disturbed sleep, and enhanced vigilance (Steiger, 2002).

Sleep has also been closely linked to psychological functioning. One of the primary symptoms of major depressive disorder is change in sleep duration, manifesting as either insomnia or hypersomnia. However, even individuals with subclinical levels of depression have been shown to suffer from sleep disturbance (Hall et al., 2000). Similarly, anxiety has been linked to sleep disturbances in both cross-sectional and longitudinal studies (LeBlanc et al., 2009; Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). This relationship was found with respect to trait and state levels of anxiety, as well as to specific anxiety disorders (Spira et al., 2008). For example, Spira et al. (2008) found that after adjusting for depression, higher rates of trait anxiety were associated with greater wake after sleep onset in older adults with primary insomnia. Further, a population-based study found that trait anxiety was a risk factor for the development of insomnia when individuals classified as good sleepers were followed longitudinally over a one-year period (LeBlanc et al., 2009). Therefore, anxiety may not only be a correlate of sleep disturbance, but may be causally related to development of sleep disorders. While depression and anxiety are more commonly examined with respect to their effects on sleep, anger has also been implicated in this relationship. For example, trait anger was found to be predictive of self-reported sleep quality in middle-aged men and women (Shin et al., 2005), as well as in juvenile offenders (Ireland & Culpin, 2006). While negative affective states or dispositions may promote sleep disturbance, social support may correspond to better sleep. For example, Troxel, Buysse, Monk, Begley, & Hall (2010)
demonstrated that higher levels of social support were associated with less actigraphy assessed wake after sleep onset. Results were consistent across both insomnia and control groups suggesting that the effect of social support on sleep may hold in the general population.

Evidence also suggests that individuals who are more susceptible to negative emotional reactions to stress are more likely to experience sleep disturbances. For instance, Mezick et al. (2009) found that individuals reporting higher negative affect (calculated by the standardized average of depression and anxiety measures) had greater actigraphy assessed sleep disturbances when also experiencing greater perceived stress. Further, Brummett et al. (2006) found that negative affect mediated the relationship between caregiving stress and self-reported sleep quality. The study also found an inverse relationship between social support and negative affect, suggesting that this may be an additional mechanism by which negative affect influences sleep quality.

**Early life stress and sleep.** Taken together, the findings described above suggest that several of the proposed pathways linking early life stress and health are also important for sleep. Yet, current models explaining relationships between early life stress and health fail to adequately consider sleep as a pathway to health outcomes (e.g., Taylor, Lerner, et al., 2004; Luecken et al., 2004). Despite this lack of recognition, several studies have indeed described a relationship between early life stress and sleep disruption. In children, insecure attachment, marital conflict, family stress (including low SES), paternal depressive symptoms, hostile parenting, as well as child abuse have all been associated with sleep disturbances (Benoit et al., 1992; El-Sheikh et al., 2013, 2007, 2012; Glod et al., 1997; Rhoades et al., 2012). Furthermore, early life stress has been shown to be associated with poorer adult sleep, with
evidence converging from polysomnography, actigraphy, and self-report sleep studies. For example, Tomfohr and colleagues (2010) found that adults from low SES backgrounds demonstrated less time in slow wave sleep, indicating less restorative sleep, and that women from low SES households had longer sleep onset latency when assessed with polysomnography. Further, childhood adverse experiences have been found to predict actigraphy assessed sleep onset latency, sleep efficiency, number of body movements, and moving time during sleep (Bader et al., 2007). Additionally, a graded relationship was found between early childhood adversity and self-reported sleep quality in a population based study of approximately 26000 Finns (Koskenvuo et al., 2010). This finding was replicated with data from the ACE study (Chapman et al., 2011). Lastly, higher family conflict at ages 7 to 15 was shown to predict self-reported insomnia at 18 years after controlling for childhood SES when assessed longitudinally, suggesting a causal relationship between family environment and adult sleep disturbance (Gregory, Caspi, Moffitt, & Poulton, 2006).

While several studies have provided support for an association between early life stress and adult sleep, few studies have examined potential mechanistic pathways involved in this relationship. In an attempt to explore whether individuals who have experienced adverse childhood events are indeed more vulnerable to stress related sleep disturbances, Bader and colleagues (2007) experimentally manipulated stress (by recall of negative autobiographical events) directly before sleep with a group of subjects suffering from insomnia. A significant association was found between adverse childhood events and sleep disturbance across groups (e.g., greater number of wakenings and more movement arousals when assessed with polysomnography and actigraphy), however, no differences were found between experimental and control groups following stress induction. The authors suggest this result
may have been due to small manipulation effects, namely, minimal shifts into negative mood states. Such a finding therefore, may speak to the importance of assessing the effect of more ecologically valid stressors on sleep.

Hanson and Chen (2010) also examined the hypothesis that early life stress increases vulnerability to stress related sleep disturbances. Using a week-long actigraphy and daily diary protocol the researchers found that individuals who more strongly endorsed a risky family environment experienced greater sleep disruption in terms of sleep duration on days they experienced greater stress. Another study found that neuroticism, or the general tendency to experience negative emotions, partially mediated the relationship between childhood adversity and adult sleep (Ramsawh, Ancoli-Israel, Sullivan, Hitchcock, & Stein, 2011). Neuroticism is highly related to negative affect and the two terms are often used synonymously (Watson & Clark, 1984). Overall, these combined efforts suggest there is some evidence to support the hypothesized mediational pathways between early life stress and poor adult sleep in this study, though there is much to be explored in this regard.

**Indicators of Disease Risk**

Several of the pathways described above are considered risk factors for CVD in their own right (Everson-Rose & Lewis, 2005). However, this study also sought to examine whether pathways of risk between early life stress and CVD had manifested in more tangible physical ways in otherwise healthy young adults. As such, resting BP and BMI were examined as further indicators of disease risk.

Previous work has demonstrated an association between early life stress and BP and BMI. Early life stress in the form of childhood SES has been the predominant line of inquiry in this regard thus far. For example, in a population based study recruiting a large sample of
almost 26000 Finns, childhood SES was associated with higher blood pressure and waist circumference in adulthood (Kivimäki et al., 2006). Further, in a secondary analysis published that same year, the research group found that childhood SES was associated with higher systolic blood pressure (SBP) across several time points spanning from childhood to adulthood, suggesting that SES is associated with long-lasting cardiovascular changes beginning early in life (Kivimäki et al., 2006). However, evidence has also shown that risky family environment plays a role. For example, several specific indices of childhood adversity, including indicators of household dysfunction, were associated with increased BMI in adulthood (Thomas et al., 2008). Further, risky family environment partially mediated the relationship between childhood SES and change in BP when assessed longitudinally, through negative affect and health behaviour pathways (Lehman et al., 2009). A similar result was found when the research group examined metabolic functioning as an outcome (which included measures of abdominal obesity); namely, childhood SES was associated with a risky family environment, which was in turn associated with poorer psychosocial functioning (i.e., depression, hostility, poor social support) and both childhood SES and adult psychosocial functioning were related to adult metabolic functioning (Lehman, Taylor, Kiefe, & Seeman, 2005).

As the findings uncovered by Lehman and colleagues (2005, 2009) highlight, several of the potential mediational pathways described so far are also thought to account for the relationships between early life stress and BP and BMI. With respect to BP, stress, negative affect, and social support have all been implicated in cardiovascular reactivity and recovery, which are thought to lead to lasting changes in BP as described above. Further, sleep processes are also related to cardiovascular functioning. Generally, sleep is associated with
decreased cardiovascular activity. During slow-wave or non-REM sleep, reductions in BP and HR associated with parasympathetic activity are typical. Conversely, REM sleep is associated with increases in BP and HR and sympathetic activity (Parish & Shepard, 1990). In humans, REM sleep accounts for a very small amount of total sleep time, with more REM sleep experienced after sleep deprivation (termed ‘rebound sleep’; Beersma, Dijk, Blok, & Everhardus, 1990). Alterations in sleep patterns or duration may therefore increase cardiovascular burden, which, if chronic, may alter cardiovascular structure. Cross-sectional and longitudinal studies provide support for this hypothesis by describing increases in BP following periods of poor sleep, even after controlling for other cardiovascular risk factors (Gangwisch et al., 2010; Javaheri, Storfer-Isser, Rosen, & Redline, 2008; Knutson, Van Cauter, Rathouz, & et al., 2009; Kotani, Saiga, Sakane, Mu, & Kurozawa, 2008). Importantly, these alterations were found even in healthy, normotensive populations, including adolescents and young adults, suggesting that poor sleep promotes cardiovascular alterations relatively early in life.

With regard to BMI, cross-sectional data have revealed a relationship between psychological factors, including anxiety, depression, and anger, and BMI, though results have sometimes been inconsistent (Anderson, Cohen, Naumova, & Must, 2006; Carpenter, Hasin, Allison, & Faith, 2000; Onyike, Crum, Lee, Lyketsos, & Eaton, 2003; Pasco, Williams, Jacka, Brennan, & Berk, 2013; Tsenkova, Carr, Coe, & Ryff, 2014; Zhao et al., 2009). Further, a series of prospective studies have provided support for causal relationships between negative affect variables and BMI, with some identifying specific gender effects (Anderson et al., 2006; Goodman & Whitaker, 2002; Hasler et al., 2005; Richardson, Davis, Poulton, & et al., 2003; Rofey et al., 2009). For example, depressive and anxious symptoms
in childhood and adolescence have been found to be associated with higher BMI in women in early adulthood after controlling for initial BMI (Anderson et al., 2006; Richardson et al., 2003), while other studies have found this relationship to hold across genders (Pine, Goldstein, Wolk, & Weissman, 2001). Notably, relationships in these studies were evident over and above the relationship between childhood SES and BMI in adulthood, as well as relevant health behaviours (e.g., physical inactivity, poor diet, drug and alcohol use). Given that anxiety and subclinical levels of depression (i.e., a depressogenic disposition rather than acute depressive episodes) have been shown to be relatively stable across the lifespan (Lovibond, 1998; Suls & Bunde, 2005; Woodall & Matthews, 1993), these findings suggest that mental health symptoms associated with early life stress have lasting effects into adulthood and are in keeping with existing mediational analyses identifying psychosocial factors as mediators between early life stress and BMI (Lehman et al., 2005, 2009).

Evidence also suggests that neuroendocrine responses to stress may account for some of the relationship between early life stress and BMI. For example, acute and daily stressors have been shown to elicit increases in cortisol levels and in turn these stress-induced cortisol responses have been consistently related to increased abdominal obesity and to the development of metabolic syndrome (Björntorp, 1991; Epel et al., 1999; Epel et al., 2000; Rosmond, 2005). Given that negative affective variables are associated with heightened neuroendocrine stress responses, this finding may also help explain why negative affective states are associated with BMI.

Cortisol is also implicated in the relationship between sleep and BMI. Sleep disturbances are associated with several alterations in metabolic and endocrine functions that are thought to be causally related to weight gain, including decreased glucose tolerance,
increased insulin sensitivity, increased levels of ghrelin, decreased levels of leptin, and increased evening concentrations of cortisol (Van Cauter & Knutson, 2008). Accordingly, poor sleep has been linked to BMI in several cross-sectional and prospective studies with both adult and child populations, though the relationship for adults is more tentative when assessed longitudinally (see Cappuccio et al., 2008; Marshall, Glozier, & Grunstein, 2008; Van Cauter & Knutson, 2008). For example, with a sample consisting of 612 adults, the CARDIA sleep study identified a relationship between sleep duration and fragmentation and BMI when assessed with actigraphy, however they did not find support for a longitudinal association between sleep parameters and BMI after a 5-year follow up (Lauderdale et al., 2009). In contrast, Gangwisch, Malaspina, Boden-Albala, and Heymsfield (2005) did find support for this relationship in a population based sample of approximately 9000 adults. Specifically, they found that self-reported sleep duration of under 7 hours predicted higher BMI at 10-year follow up. This finding suggests that factors associated with self-reported sleep quality (e.g., sleep satisfaction, daytime sleepiness) may be associated with BMI outcomes (Magee, Iverson, Huang, & Caputi, 2008).

The Current Study

Given that CVD development is multifactorial in nature, elucidating the various factors contributing to disease pathogenesis is critical in informing intervention efforts. Emerging research efforts converge on the concept that early life experiences create differences in the development of emotional, social, and biological mechanisms that underlie the ability to regulate stress, which in turn contribute to the progression of disease states. In particular, a constellation of family characteristics thought to capture a broad realm of dysfunction, namely families characterized by cold, conflict-ridden, or neglectful parenting
(i.e., risky families), have been hypothesized to produce offspring that are poorly equipped to manage stress, creating sequelae of psychological and physiological health difficulties which extend into adulthood (Taylor, Lerner, et al., 2004).

While a number of mediational pathways have been proposed to explain the relationship between early life stress and adult health outcomes, relatively little research has been directed toward sleep disturbance as a potentially potent mediating risk factor. Poor sleep has been identified as an outcome of early life stress in several studies, however, how early life stress translates to sleep disturbance still needs clarifying. A small number of studies have identified psychological stress and negative affect (assessed by the related concept of neuroticism) as potential mediating factors in this relationship. Further, these psychosocial variables, along with lack of social support, have been implicated in the relationship between early life stress and other health outcomes, including BP and metabolic syndrome, bolstering the hypothesis that these variables may play an important role in conferring the effects of early life stress into adulthood. The current study seeks to further explore potential factors mediating the relationship between early life stress and health risk outcomes in otherwise healthy young adults using structural equation modeling.

**Hypothesized Model**

Primary analyses will focus on describing mediational relationships between early life stress and individual indicators of health risk (i.e., sleep parameters, BP and BMI) as depicted in Figure 1. Previous research has demonstrated that childhood SES is associated with family dysfunction including harsh discipline, lack of maternal warmth, exposure to aggressive adult models and family life stress (Dodge et al., 1994). Accordingly, childhood SES was found to predict Taylor, Lerner, et al.'s (2004) conceptualization of risky family
environment, which incorporates related factors of family risk (Lehman et al., 2005, 2009). As such, it is hypothesized that childhood SES will confer risk for poor health outcomes through its relationship with risky family environment. Risky family environment in turn is expected to be associated with social support, perceived stress, and negative affect, consistent with previous research describing relationships amongst these variables. Further, social support is expected to be inversely related to both negative affect and perceived stress. Additionally, perceived stress is modeled as predictive of negative affect given previous literature suggesting that early childhood environment serves to sensitize one to negative emotional responses to stress. Finally, it is hypothesized that both negative affect and perceived stress will contribute to sleep parameters, BP, and BMI.

While depression, anxiety and anger have singularly been associated with early life stress, Suls and Bunde (2005) argue that a general disposition for negative affect may be more important for disease risk than any one negative affect indicator, particularly when thought to arise from a common underlying etiology, in this case early alteration of stress response systems. The researchers, as well as others, suggest that a single-factor approach prevents examination of the clustering of psychosocial factors on disease development (which may behave synergistically) (Raynor, Pogue-Geile, Kamarck, McCaffery, & Manuck, 2002; Suls & Bunde, 2005). Anxiety and depression in particular are known to highly correlate, and factor analytic studies have shown that anxiety, depression, and anger all load on a single factor reflecting the broad band dimension of negative affect, namely the tendency to experience negative feelings such as anger, sadness, anxiety, and guilt (Costa & McCrae, 1992; Suls & Bunde, 2005). Therefore, a negative affect factor is expected to be most relevant for predicting indicators of disease risk in this study. Consistent with this
supposition, Stewart, Rand, Hawkins, & Stines (2011) found that a latent negative affective factor better predicted self-reported sleep quality than unique effects of depression, anxiety, and anger.

Secondary analyses will focus on exploring the role of sleep as a proximal mediator of the relationship between factors associated with early life stress, namely negative affect and perceived stress, and other physical health indicators including BP and BMI. The conceptualization of sleep as a mediator in these relationships results from evidence demonstrating associations between early life stress and sleep quality, as well between proposed mediational pathways between early life stress and health outcomes, namely, ongoing vulnerability to stress and negative affect. In terms of sleep parameters, both subjective (via the Pittsburgh Quality Sleep Index) and objective (via actigraphy) sleep measures will be examined due to previous research showing differential effects for the two types of sleep measures. While actigraphy data may more accurately capture changes in sleep architecture (e.g., nightly awakenings), subjective sleep assessment captures the individual’s experience of sleep, such as feelings of restfulness or daytime fatigue that may also be important for disease risk (e.g., daytime fatigue may be associated with other important health behaviours such as reduced physical activity). Notably, the two types of sleep measurements are typically shown to only moderately correlate. For example, data from the CARDIA sleep study found that self-reported sleep duration was only moderately correlated ($r = .45$) with actigraphy assessed sleep duration (Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008).

**Study Objectives**

1) To test mediational relationships between early life stress and individual
indicators of health risk, using both an online study sample and a university sample (primary analyses).

2) To explore whether or not sleep parameters mediate the relationship between psychosocial factors and indicators of CVD risk (secondary analyses).
Methods: Study 1 Online Sample

Participants

An online study was conducted using the Mechanical Turk (MTURK) research database. The sample included 298 participants (63.1% female; age range, 18 – 45 years; mean age 29.55, \(SD = 7.20\); 77.7% Caucasian, 8.1% African American 6.7% Asian, 5.7% Hispanic, and 1.7% ‘other’). Subjects received a small honorarium in exchange for their participation. Inclusion criteria for this study included being between the ages of 18 and 45 and possessing a working knowledge of the English language. Exclusion criteria included self-reported diagnosis of a severe psychiatric disorder (e.g., bipolar disorder, schizophrenia, posttraumatic stress disorder) or chronic disease (e.g., cancer, diabetes), as the target population was healthy young adults.

Measures

**Demographic information.** Participants completed a demographic questionnaire indicating their race, age, gender, chronic illness profile, and any sleep disorders they may have, including insomnia or sleep apnea as diagnosed by a physician (see Appendix A).

**Childhood family environment.** The Risky Families Questionnaire (Taylor, Lerner, et al., 2004) is an 11-item scale assessing overt family conflict, family violence, and family relationships that are cold, unsupportive, or neglectful. The scale was adapted from an original measure created by Felitti et al. (1998) in their Adverse Childhood Experiences study to include items pertaining to family conflict (e.g., “How often would you say there was quarreling, arguing, or shouting between a parent and one of your siblings?”). Participants are asked to respond to items on a 4-point Likert scale ranging from 1 (“none of..."
the time”) to 4 (“most or all of the time”) with higher scores denoting greater family risk/conflict in childhood. The scale has demonstrated good internal consistency (α = .86; Taylor, Eisenberger, Saxbe, Lehman, & Lieberman, 2006) and has been validated against clinical interviews conducted by trained interviewers, demonstrating high agreement and reliability (Taylor, Lerner, et al., 2004).

Childhood SES was also assessed using the Four Factor Index of Social Status (Hollingshead, 1975). This measure is based on parental educational and occupational attainment, applying more weight to occupation using a ratio of five to three. While this measure is based on data from the 1970 U.S. Census, it has been shown to be comparable to more recently developed measures of U.S. SES and to a Canadian-based SES measure (Cirino et al., 2002).

Social support. Participants completed the Enrichd Social Support Instrument (ESSI; Mitchell et al., 2003). The ESSI is a 7-item scale intended to assess emotional and instrumental aspects of social support. Subjects respond to items such as “Is there someone available to you whom you can count on to listen to you when you need to talk?” on a 5-point scale, with 1 corresponding to “none of the time” and 5 corresponding to “all of the time.” When analyzed within a sample of cardiac patients, the ESSI has shown strong psychometric properties including good internal consistency (α = .88) and 5- and 6-month test-retest reliability (r = .94). Due to lack of a “gold standard” social support measure, construct validity of the ESSI was determined by assessing depressed and non-depressed individuals, with the view that ESSI scores would be lower in depressed individuals, and data support this hypothesis (Vaglio et al., 2004). In addition, lower scores on the ESSI were correlated with cardiac symptoms and social functioning after 6-months, providing evidence
for predictive validity (Vaglio et al., 2004).

**Perceived stress.** The Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) is a 10-item scale designed to measure the degree to which individuals view their life as stressful. Respondents are asked to indicate on a 5-point Likert scale, ranging from 0 ("never") to 5 ("very often"), how often in the last month they experienced specific feelings related to coping and the perception that life is unpredictable, uncontrollable and overwhelming (e.g., “How often have you felt difficulties were piling up so high that you could not overcome them?”), as well as direct questions pertaining to current levels of experienced stress (e.g., “How often have you felt nervous and “stressed”?). The scale has demonstrated good internal consistency with three samples (α ranging .84-.86) and good test-retest reliability after two days (r = .85). Evidence for convergent validity was demonstrated by moderate correlations with scores on the life-events scale. In addition, scores on the PSS were more predictive of depressive and physical symptomology at follow-up than scores on the life-events scale indicating predictive validity (Cohen et al., 1983).

**Depression.** The Centre for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) is a 20-item scale developed to measure depressive symptomology in the general population, with an emphasis on affective symptoms. Subjects respond to the frequency of symptoms over the past week, such as “I did not feel like eating; my appetite was poor” and “I felt depressed”, on a 4-point scale with 0 corresponding to “rarely or none of the time (less than 1 day)” to 3 corresponding to “most or all of the time (5-7 days).” The CES-D has demonstrated good internal consistency (α = .85) when assessed within the general population, and adequate test-retest reliability over 2-8 weeks. In terms of validity, CES-D scores discriminated between general population and patient samples, in addition to
level of severity within each sample when compared with nurse-clinician ratings on
depression rating scales. The measure also demonstrated convergent and discriminant
validity when compared to other self-report scales, and was positively associated with
negative life events. Further, scores on the CES-D were attenuated following treatment
(Radloff, 1977). Notably, item 11, which corresponds to sleep disruption, was omitted from
the total CES-D calculation in order to avoid confound with sleep parameters assessed in the
study.

**Anxiety.** Trait-type anxiety was assessed using the State-Trait Anxiety Inventory,
Form X2 (STAI-X2; Spielberger, Gorsuch, & Lushene, 1970). The STAI-X2 is the trait
form of the STAI and includes 20 items on which participants rate themselves in terms of
their level of anxiety. They are asked to respond to items by circling the number that
corresponds to how they generally feel, from 1 (*almost never*) to 4 (*almost always*). Items
include, “I am ‘calm, cool and collected’” and “I get in a state of tension or turmoil as I think
over my concerns and interests.” Spielberger and colleagues (1970) report good test-retest
reliability for the STAI-X2 trait anxiety scale (*r* = .81), with internal consistency coefficients
ranging from .83–.92. As well, various studies have provided evidence for convergent
validity when correlated with other self-report measures (Endler & Magnusson, 1976;
Knight, Waal-Manning, & Spears, 1983; Spielberger et al., 1970).

**Anger.** The 15-item version of the Trait Anger Scale (TAS; Spielberger, 1988) was
used to assess trait anger, namely, the proneness for one to experience anger. Using a 4-point
Likert Scale (1 = "*almost never*" to 5 = "*almost always*”) participants are asked to rate how
angry they generally feel across items (e.g., “I am quick tempered”; “It makes my blood boil
when I am pressured”). The TAS has demonstrated good internal consistency with college
students ($\alpha = .91$) and correlates positively with measures of anger and hostility such as the Buss-Durkee Hostility Inventory (Spielberger, 1988). Further, it has been shown to discriminate between high and low anger groups and the tendency toward angry responding in anger provocation studies (Deffenbacher, Oetting, Lynch, & Morris, 1996; Lopez & Thurman, 1986; Spielberger, 1988).

**Body mass index.** The online sample reported their height and weight for calculation of BMI using the following formula: $\text{BMI} = \frac{\text{weight(kg)}}{\text{height(m)}^2}$.

**Sleep measure.** The online sample completed a *subjective measure* of sleep quality. The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) assesses participants’ retrospective and subjective recall of sleep patterns during the last month (i.e., month prior to study commencement). The PSQI is a 19-item self-report measure that assesses overall sleep quality and dysfunction over a 1-month period. Participants report, for example, on the time of day they wake and sleep, how long it takes to fall asleep (i.e., sleep latency), the number of hours of sleep per night, and the number of times in the past month they had trouble sleeping for reasons such as ‘being too hot’, or ‘having trouble breathing.’ The 19 items compile to create seven composite scores, weighted on a 0-3 scale. These composite scores include sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction. Summing of the composite scores provides a global score, which ranges from 0-21, with higher scores indicating poorer sleep quality. The PSQI has demonstrated good internal consistency ($\alpha = .83$) and good test-retest reliability for the global score ($r = .85$) and for each of the seven composite scores (ranging from .65 for medication use to .84 for sleep latency) for each of the seven composite scores (Buysse et al., 1989). Internal consistency
was $\alpha = .80$ for the global score (composite scores ranged from $\alpha = .70-.78$). PSQI global scores were highly correlated with single or multi-item measures of sleep quality, indicating convergent validity, but did not correlate with unrelated constructs, indicating adequate discriminant validity (Buysse et al., 1989).

**Procedure**

Online participants completed the questionnaire battery through the MTURK research database where they were able to participate in the online survey in exchange for a nominal honorarium ($0.50 per half hour of their participation deposited directly into their MTURK account) which was assigned after approval of completion of the questionnaire in order to encourage accurate responding and completion of the battery. Subjects were informed the study was designed to better understand how personality and psychological factors predict sleep activity, therefore no deception was used. The MTURK database links users to questionnaires stored on the Survey Monkey online survey platform. Questionnaires were presented in randomized order, as per the Survey Monkey software. Subjects were asked not to participate in the study if they met exclusion criteria noted above and were asked to complete the survey in one sitting. Consent was obtained before completion of the survey.

**Statistical Analysis**

The online MTURK sample allowed us to explore whether the theoretical model depicted in Figure 1 was supported with self-reported sleep quality data before initiating the more time intensive actigraphy protocol. The primary goal for subsequent analyses was to determine whether the hypothesized risky family model contributed to the variance of health risk indicators (i.e., self-reported sleep quality and BMI). While childhood SES has been
shown to have a direct relationship with several health outcomes, we tested a more restrictive model initially, with childhood SES modeled to contribute to risky family environment.

Structural equation modeling was completed using EQS 6.2 software. Data were inspected for normality using Yuan, Lambert, and Fouladi’s (2004) normalized estimate (estimates < 3.0 were considered to meet the assumption of multivariate normality). If assumptions of normality were not met, robust methodology was utilized. In addition, EQS output specifying cases with the largest contribution to nonnormality were inspected to identify outliers that may influence results even in light of robust corrections (e.g., data points several standard deviations from the mean). Missing values, if present, were treated using Full Information Maximum Likelihood estimates (Yuan & Bentler, 2000).

Prior to inclusion in the overall theoretical model, a latent negative affect factor was tested to determine whether depression, anger, and anxiety loaded on a single factor as hypothesized. Given that the model was just-identified (i.e., had 0 degrees of freedom) \( \chi^2 \) statistics were not produced; therefore, model fit was estimated by examining reliability estimates and ensuring that each indicator was significantly predicted by the latent factor. Both Cronbach’s Alpha and the reliability coefficient Rho are reported, though more weight was given to Rho as it is typically considered a more accurate estimate of reliability in cases where the measurement model is based on unequal unstandardized loadings (Raykov, 1997).

Following confirmatory factor analysis for the latent factor, hypothesized models predicting our health risk indicators were then tested. Consistent with Hu and Bentler’s (1999) recommendations, we examined model \( \chi^2 \) statistics, comparative fit (CFI > .95) and parsimony-adjusted fit indices (RMSEA < .06) to assess model fit. Models were modified based on examination of Lagrange Multiplier (LM) tests, as well as standardized residuals
(i.e., the difference between predicted and observed covariance, standardized) to achieve better fit of the underlying covariance structure. Mediational hypotheses were tested via examination of indirect effects provided in the EQS output, which are based on Sobel’s work (1982, 1986, 1987; see Bentler, 2006). To control for potential confounds in the model, sensitivity analyses were then conducted which included health behaviours and demographic variables that were significantly correlated with our health risk indicator variables as covariates in the model. While this type of post-hoc analysis is not always desirable (Babyak, 2004), given the number of potential covariates that could be associated with variables in the model, we thought it essential in order to maintain power for our main variables of interest. Multiple groups analyses of final models were then conducted to determine whether the model differed across gender.

Secondary analyses focused on examining self-reported sleep as a mediator of the relationships between psychosocial model variables and BMI. To avoid fitting overly complex models, psychosocial variables were included as independent variables in mediational analyses if they demonstrated a significant relationship with our outcome variable (BMI) and our hypothesized mediator (self-reported sleep quality), based on the criteria for mediation put forth by Baron and Kenny (1986).
Results: Study 1 Online Sample

Data Screening

The data did not meet the assumption of multivariate normality (Yuan, Lambert & Fouladi’s normalized estimate > 3.0; Yuan et al., 2004). One female subject reported a BMI that was substantially above the mean (8.27 standard deviations from the mean) contributing to the deviation from normality; as such results were inspected with this outlier included as well as excluded. Additionally, 16 participants (5.4%) had missing questionnaire data. An additional 11 participants (3.7%) were missing childhood SES data due to vague or incomplete responses that did not lend themselves to reliable coding of parent occupation. Given missing and non-normal data, Full Information Maximum Likelihood estimates with robust standard errors were used for all SEM analyses and Yuan-Bentler Scaled $\chi^2$ statistics are reported for tests of model fit (Yuan & Bentler, 2000).

Descriptives

Descriptives for the online sample are presented in Table 1 and the correlation matrix is presented in Table 2. Age demonstrated a moderate positive correlation with body mass index. Women reported poorer sleep, higher social support, and lower SES than men. Smokers (coded ‘yes’ or ‘no’) reported poorer sleep and greater levels of perceived stress. Psychotropic medication use (coded ‘yes’ or ‘no’) was associated with greater endorsement of perceived stress, anxiety, and anger. Lack of exercise (coded ‘yes’ or ‘no’) was significantly correlated with poorer sleep, greater perceived stress, and higher levels of depression, anger, and anxiety, while more habitual exercise (minutes of exercise per week) was associated with lower perceived stress, and lower levels of anger and anxiety. A one-way
ANOVA did not reveal any significant relationships between ethnicity and psychosocial variables, sleep quality, or BMI.

In the MTURK sample, 21.5% reported they had the following sleep related difficulties: 11.4% indicated they were diagnosed with insomnia by a physician, 2.4% reported sleep apnea, 2.4% reported restless leg syndrome, 3.0% reported night terrors, 4.4% reported sleep talking, and 10.1% reported bruxism (i.e., tooth grinding). Further, 4.4% of the sample indicated they suffered from anxiety and/or depression, and 62.7% reported they were married or in a long-term relationship. Only 23.9% of participants identified themselves as students, 48.8% were employed full-time, 20.2% were employed part-time, and 31.0% were unemployed.

In terms of family risk, 65% of the sample endorsed some degree of verbal abuse (i.e., rated questions pertaining to being sworn at, insulted, put down or made to feel threatened above a 1 “not at all” denoting no verbal abuse), 48% of the sample reported physical abuse (e.g., pushed, shoved or slapped), 35% endorsed domestic violence, 46% reported neglect, and 36% endorsed having being exposed to substance abuse above a rating of “not at all”.

**Confirmatory Factor Analysis for Negative Affect**

A confirmatory factor analysis was conducted to determine whether depression, anxiety, and anger were appropriately modeled as indicators of a single latent negative affect factor. The model demonstrated strong reliability estimates (Cronbach’s Alpha = .82; Reliability Coefficient Rho = .87) and each indicator was significantly predicted by the latent factor (see Figure 2), supporting the assumption of a common underlying factor.
Theoretical Model Testing: Self-Reported Sleep

The theoretical model depicted in Figure 1 was fit to the data using self-reported sleep quality as the outcome of interest. Initial analysis of the risky family model for the MTURK sample indicated model fit [Yuan-Bentler $\chi^2\ (16) = 23.11, p > .10, CFI = .99, RMSEA = .04, 90\% CI = 0.00, 0.71$] (see Figure 3). Similar results were found when the identified outlier was excluded from the model, both in terms of model fit [Yuan-Bentler $\chi^2\ (16) = 23.29, p > .10, CFI = .99, RMSEA = .04, 90\% CI = 0.00, 0.71$] and regression coefficients, therefore results for the entire sample are described (see Table 3 for unstandardized regression coefficients and robust standard errors for direct effects). As depicted in Figure 3, most of the relationships in the hypothesized model were supported. Subjects who reported lower SES as children were more likely to report growing up in a risky family environment, though childhood SES predicted only 5% of the variance in risky family environment. In turn, those reporting a higher family risk reported lower levels of current perceived social support (explaining 11% of the variance in social support) and higher levels of perceived stress and report of negative affect. As predicted, social support was associated with lower levels of perceived stress and negative affect. Further, higher perceived stress was strongly associated with greater negative affect, as would be anticipated given the close theoretical association of these constructs. Risky family environment and social support explained 18% of the variance in perceived stress, while the combined influence of risky family environment, social support, and perceived stress explained 79% of the variance in the latent negative affect factor. Negative affect was strongly related to sleep quality, with greater negative affect associated with poorer sleep quality (a higher score on the PSQI denotes poorer sleep quality). Inconsistent with the hypothesized model, a near zero
and non-significant relationship was found between perceived stress and self-reported sleep quality ($\beta = -.01, p > .05$), suggesting that the impact of stress on self-reported sleep quality may be due to its influence on negative affect. The indirect effect between perceived stress and self-reported sleep quality was quite large, providing evidence of this mediational relationship (see Table 4). Overall, negative affect and perceived stress explained 22% of the variance of self-reported sleep quality.

Significant indirect effects were also found between risky family environment and sleep quality, and social support and sleep quality. This suggests that, as hypothesized, greater endorsement of a risky family environment may be associated with poorer sleep quality through its contribution to adult psychosocial functioning, and that social support may promote sleep quality by attenuating perceived stress and negative affect. Further, a large indirect effect was found between risky family environment and negative affect, suggesting that with the model specified as it is here, social support and perceived stress partially mediated this relationship. In addition, a small but significant indirect effect was found between risky family environment and perceived stress, indicating that social support may partially mediate this relationship to a degree; namely, that for individuals reporting greater family risk, higher levels of perceived social support may attenuate perceived stress.

With regard to childhood SES, very small, yet significant indirect relationships were found between childhood SES and endogenous variables in the model (e.g., the indirect effect between childhood SES and sleep quality, while significant due to a small robust standard error, was negligible, $\beta = -.04, Z = -2.69, p < .05$; see Table 4). This suggests that childhood SES may not be as significant a contributor to the overall model as risky family environment. Overall, the original conceptual model was supported, though perceived stress did not
demonstrate a direct relationship with self-reported sleep quality, instead the relationship was mediated by negative affect.

**Covariate Testing**

To control for potential confounds in the model, sensitivity analyses were conducted with health behaviour and demographic variables shown to have a significant relationship with the outcome variable of interest, namely self-reported sleep quality. Zero-order correlations between potential continuous and dichotomous covariates and our sleep outcome variable are presented in Table 2. As noted above, gender, smoking, and exercise (coded ‘yes’ or ‘no’) were all significantly correlated with self-reported sleep quality, while self-reported habitual exercise (minutes/week), overall medication use (coded ‘yes’ or ‘no’) and psychotropic medication use (coded ‘yes’ or ‘no’) were not (drug use to facilitate sleep was not examined as a covariate as this question is already incorporated into the PSQI composite variable). Specifically, women reported poorer sleep quality \((M = 6.65, SD = 3.40)\) than men \((M = 5.85, SD = 2.83)\), as did smokers (smokers, \(M = 7.59, SD = 3.23\); nonsmokers; \(M = 6.10, SD = 3.17\)). Further, lack of exercise was associated with poorer self-reported sleep quality (exercise, \(M = 6.02, SD = 3.06\); no exercise, \(M = 7.05, SD = 3.44\)). Therefore, smoking and exercise were included as covariates in subsequent sensitivity testing, while the effect of gender on the model was assessed via multiple groups analysis. A one-way ANOVA did not reveal a significant relationship between ethnicity and sleep quality \([F (5, 290) = .60, p = .70]\). Therefore, ethnicity was not included in the sensitivity analyses.

Covariates were included in the initial model depicted in Figure 3 with smoking and exercise correlated with one another and with paths added between each covariate and each observed and latent variable in the model. The overall model fit the data \(\chi^2\)
(20) = 24.47, \( p > .10 \), CFI = .99, RMSEA= .03, 90\% CI = 0.00, 0.06. Importantly, the relationships observed in the initial unadjusted model remained significant and relatively unaffected. For ease of presentation Table 5 describes model statistics while Figure 4 presents the adjusted model without paths from covariates shown. After inclusion in the model, only smoking was significantly associated with self-reported sleep quality. Smoking was also associated with greater perceived stress, while exercise was associated with less perceived stress. Exercise also demonstrated a small but significant association with negative affect, with endorsement of exercise corresponding to lower levels of negative affect.

Amongst covariates, the correlation between errors for smoking and exercise was not significant (\( r = -.12, p > .05 \)). In sum, the risky family model for sleep quality was supported even after controlling for relevant health behaviours.

**Multiple Groups Analyses**

Multiple sequential steps were taken to assess whether the final model depicted in Figure 4 differed significantly across gender (see Table 6). Given that covariates did not affect the relationships amongst variables of interest, they were not included in this analysis. When no constraints were imposed the model fit the data for both males and females. The model also fit when factor loadings were constrained (i.e., forced to be equal across groups), as well as when regression coefficients were constrained to be equal across groups. However, when residual variances for observed variables were constrained across groups the chi-square difference test was significant, indicating differences across gender.
Theoretical Model Testing: Body Mass Index

The risky family model was also tested with BMI as the health risk indicator of interest, consistent with the view that risky family environment corresponds to poor health outcomes in adulthood. In the overall sample, with the outlier included, the model did not fit the data [Yuan-Bentler $\chi^2 (16) = 39.90, p < .01; \text{CFI} = .96; \text{RMSEA} = .07, 90\% \text{ CI } = .05, .10]$ and neither perceived stress ($\beta = -.07, p > .05$) nor negative affect (despite the moderately large regression coefficient, $\beta = .24, p > .05$, suggesting a large standard error estimate) were significantly associated with BMI. LM tests suggested a path between childhood SES and BMI. This modification would be in keeping with previous work by Lehman et al. (2005) who found that childhood SES had a unique relationship with adult metabolic functioning (which included waist circumference in the latent factor), over and above mediation via childhood family environment and current psychosocial functioning. As such, these modifications were made. Subsequently, the model demonstrated approximate fit [Yuan-Bentler $\chi^2 (15) = 28.50, p = .02; \text{CFI} = .98; \text{RMSEA} = .06, 90\% \text{ CI } = .02, .09]$ and both childhood SES ($\beta = -.17, p < .05$) and negative affect ($\beta = .24, p < .05$) were significantly associated with BMI. However, when this final model was examined with the outlier excluded, the model once again demonstrated lack of fit [Yuan-Bentler $\chi^2 (15) = 37.01, p < .001; \text{CFI} = .97; \text{RMSEA} = .07, 90\% \text{ CI } = .04, .10$] and relationships between BMI and childhood SES ($\beta = -.13, p < .05$) and negative affect ($\beta = .19, p > .05$) were attenuated suggesting that results were likely driven by the outlier. Given lack of model fit further analyses were not conducted.
Sleep as a Mediator between Psychosocial Variables and BMI

Given that adult psychosocial functioning was not significantly associated with BMI in previous analyses, planned mediational analyses exploring self-reported sleep quality as a mediator of the relationship between psychosocial variables and BMI were not conducted.
Methods: Study 2 University Sample

Participants

The university sample included 158 participants (55.7% female; age range, 18 – 45 years; mean age 23.41, \(SD = 5.08\); 30.4% Caucasian, 60.1% Asian, and 9.5% ‘other’) who were recruited from the University of British Columbia via the UBC Psychology paid participants study list and advertisements posted around campus. Subjects received an honorarium in exchange for their participation. Inclusion and exclusion criteria were the same as for the online sample.

Measures

The university sample completed the same measures as the online sample, including the demographic questionnaire, a measure of childhood family environment (RFQ), social support (ESSI), perceived stress (PSS), depression (CES-D), anxiety (STAI-X2), anger (TAS), and subjective sleep quality (PSQI). In addition, the university sample completed a daily sleep diary (see Appendix B), and actigraphy and BP data were collected as described below. Height and weight were also directly measured in order to calculate BMI.

Objective sleep assessment. Actigraph monitors, worn on the wrist, provide an objective index of sleep quality by measuring sleep/wake times as indicated by motor activity (Mini-Mitter, Respironics, Bend, Oregon; Canadian supplier, Biolynx/Montreal). The monitor includes an accelerometer that provides data on sleep architecture (e.g., sleep length, motor activity during sleep, sleep onset latency, and wake times) by translating motor activity into numeric representation. Movement is sampled frequently and is then aggregated to provide a 30-second recording (epoch), which is then stored in the devices’ internal
memory until the data are downloaded for use (Sadeh, Hauri, Kripke, & Lavie, 1995). Epochs are scored either as wake or sleep by comparing activity counts for the epoch and those immediately surrounding it, to a threshold wake value (a medium threshold value was used in this study). Actigraph measurements when compared with “gold standard” Polysomnography (PSG) show minute-by-minute sleep/wake scoring agreement rates of 75-95% for normal subjects. Further, they show strong correlations ($r = .83$ to $.97$) for measures of sleep/wake times, sleep efficiency and total sleep time (for review see, Sadeh et al., 1995). PSG is unfortunately an expensive and cumbersome measurement process that must be employed in a laboratory, and involves the attachment of electrodes to an individual while sleeping in order to monitor brain activity (EEG), eye movement (EOG), muscle activity (EMG), and breathing and respiratory rates (Kushida et al., 2001). In contrast, the actigraph is inexpensive, non-cumbersome, and noninvasive, and can be employed in the home, and is therefore more ecologically valid. Nevertheless, studies have found that actigraphy can overestimate sleep duration due to lengthy periods of inactivity (as when an individual may lay in bed for a period of time before actually falling asleep), or underestimate sleep duration for those who sleep restlessly. Consequently, it is recommended that actigraphy data be combined with subjective sleep data to enhance accuracy of acquired data (Kushida et al., 2001). Using this coupled data collection procedure, Kushida and colleagues (2001) found no significant difference between PSG and combined actigraphy and subjective recordings of total sleep time and sleep efficiency. Therefore, participants were asked to complete a sleep diary for the nights they wore the actigraph. The sleep diary asks for a detailed report of that night’s activities and sleep patterns, including the time they went to bed, the estimated time they fell asleep, and the estimated time it took to fall asleep. Time to bed and wake times
from the subjective sleep diary were used to set the sleep interval required for calculation of sleep variables using the actiwatch software (see Appendix B).

Given that several of the variables calculated by the actigraphy software are overlapping, select variables that demonstrated strong but non-redundant correlations (supporting the creation of a latent variable) were chosen to include in subsequent analyses (see Table 8). Specifically, percent of the sleep period spent awake (total wake time in minutes/sleep duration), percent of the time spent mobile (an indicator of physical restlessness during sleep; percentage of total mobile time in minutes/sleep duration), and number of wake bouts (number of wake bouts/sleep duration) were chosen as indicators of a latent sleep activity variable. Variables were standardized by sleep duration to control for the increased likelihood of having a greater number of wake bouts and minutes of wake and physical restlessness the longer one sleeps. Total sleep time (time in minutes between actigraphy assessed sleep and wake times – minutes coded as wake) was also of interest, given the associations between sleep length and health outcomes found in previous studies (Cappuccio et al., 2011; Gallicchio & Kalesan, 2009). Total sleep time demonstrated weaker correlations with the sleep activity variables, consistent with a previous confirmatory factor analysis demonstrating that sleep length and activity during sleep are distinct yet related factors (Slaven, Andrew, Violanti, Burchfiel, and Vila, 2008). A confirmatory factor analysis was conducted to support the decision to analyze duration and activity separately.

Data collected across the six-day sleep assessment period were averaged to increase reliability before inclusion as observed variables in subsequent SEM models. Previous research has indicated that at least five nights of actigraphy measurements are required to obtain stable estimates of individual sleep patterns (Acebo et al., 1999). In the current study,
night-to-night reliability estimates revealed high stability for most variables (percent wake, \( \alpha = .85 \); percent mobile, \( \alpha = .86 \); number of wake bouts, \( \alpha = .86 \)) supporting averaging data across nights. Total sleep time demonstrated less stability overall (\( \alpha = .63 \)). Similar results (i.e., reliability estimate \( \alpha = .60 \) for sleep duration) were found to be acceptable prior to averaging across nights in previous actigraphy studies (El-Sheikh et al., 2007). Given the expectation of some loss of data, sleep data were coded as missing if more than two nights of actigraphy measurements were missing.

**Blood pressure.** BP Monitors were attached to participants’ nondominant arm to assess resting BP at the completion of the study week. The VSM-100 BpTRU automatic blood pressure device has been found to be a reliable non-invasive measurement within pediatric and adult populations (ages 3-83 years) (Mattu, Heran, & Wright, 2004). Specifically, when compared to standard auscultatory mercury sphygmomanometer measurements, 89.2% of the BpTRU measurements were within 5 mmHg, with 96.4% and 99.3% of these measures being within 10-and 15 mmHg, respectively (Mattu et al., 2004). Furthermore, in a sample of hypertensive patients, the BpTRU clinic blood pressure monitor was found to correlate significantly better with daytime ambulatory BP (\( r = 0.57 \)) than clinic averages (\( r = 0.15 \)) (Beckett & Godwin, 2005).

Resting BP values were collected every 2 minutes for 10 minutes. The average resting BP value was calculated by averaging the last two baseline readings of a 10-minute period. These two values are the most representative of resting BP and averaging them provides additional reliability. Consistent with previous research assessing similar models, systolic (SBP) and diastolic blood pressure (DBP) were modeled separately (Lehman et al., 2009).
**Procedure**

University participants contacted the lab via telephone or email and were then screened for eligibility. If they met eligibility requirements they attended the lab portion of the study protocol which included consenting to participate in the study and completing the questionnaire battery outlined above using the Survey Monkey online survey platform to promote accurate data collection and scoring. Questionnaires included in the battery were presented to each participant in a randomized order, as per the Survey Monkey software. Subject’s height and weight measurements were then obtained. Participants were provided with detailed instructions about use of the actigraph watches and the required diary completion, and advised to return to the lab following the six-day sleep assessment period. During the sleep assessment period, motor movement and sleep quality were assessed via joint use of actigraphy and self-report diary completed each morning. Participants accessed and completed the daily diary using the Survey Monkey online survey platform first thing in the morning so that data could be collected and downloaded into relevant software programs. Participants were provided the option of completing the daily diary by hand if they did not have access to a computer (only four subjects chose this option). An email reminder was sent daily to ensure completion of the diary data that same day. If participants failed to complete the diary for an allotted day, a telephone reminder was provided. Subjects received $5.00 for each diary completion, in addition to $15.00 for completion of demographic and psychosocial questionnaires and actigraph return. Upon returning the actigraph after the six-day period, five BP readings across a 10-minute period were taken to assess resting BP, and subjects were fully debriefed both verbally and in writing.
Statistical Analyses

Structural equation modeling was completed using EQS 6.2 software. Data were inspected for normality using Yuan, Lambert & Fouladi’s normalized estimate (estimates < 3.0 were considered to meet the assumption of multivariate normality; Yuan et al., 2004). If assumptions of normality were not met, robust methodology was utilized. In addition, EQS output specifying cases with the largest contribution to nonnormality was inspected to identify outliers that may influence results even in light of robust corrections (e.g., data points several standard deviations from the mean). Missing values, if present, were treated using Full Information Maximum Likelihood estimates (Yuan & Bentler, 2000).

Prior to inclusion in hypothesized models, a latent negative affect and a sleep activity factor were tested to determine whether proposed indicators loaded on a single factor. Given that the models were just-identified (i.e., had 0 degrees of freedom) $\chi^2$ statistics were not produced; therefore, model fit was estimated by examining reliability estimates and ensuring that each indicator was significantly predicted by the latent factor. Risky family models predicting health indicator variables (i.e., sleep parameters, BMI and SBP and DBP) were subsequently tested. Due to small sample size each outcome variable was analyzed in a separate model. Consistent with Hu and Bentler's (1999) recommendations, we examined model $\chi^2$ statistics, comparative fit (CFI > .95) and parsimony-adjusted fit indices (RMSEA < .06) to assess model fit. Models were modified based on examination of LM tests, as well as standardized residuals to achieve better fit of the underlying covariance structure. Mediational hypotheses were tested via examination of indirect effects provided in EQS output. Multiple groups analyses of final models were not conducted with the university sample due to smaller sample size than the online sample. Further, due to small sample size
and given the fact that covariates did not substantially impact the relationships between model variables in the MTURK sample, sensitivity analyses controlling for health behaviour and demographic variables were also not conducted.

Secondary analyses focused on examining sleep parameters as mediators of the relationships between psychosocial model variables and our health indicator variables (i.e., BMI and BP indices). To avoid fitting overly complex models psychosocial variables were included as independent variables in mediational analyses if they demonstrated a significant relationship with an outcome variable and a specific sleep parameter, based on the criteria for mediation put forth by Baron and Kenny (1986).
Results: Study 2 University Sample

Data Screening

The data for the university sample did not meet the assumption of multivariate normality (Yuan, Lambert & Fouladi’s normalized estimate > 3.0; (Yuan et al., 2004). However, multivariate kurtosis analyses provided in EQS output did not identify that any one subject was substantially contributing to multivariate nonnormality, therefore all participant data was analyzed. In the university sample, four subjects (1.3%) were missing SES data due to vague or incomplete responses and nine participants (5.8%) had missing or incomplete daily diary entries for the sleep assessment period. Further, seven participants (4.5%) had no actigraphy data due to actiwatch malfunction (six participants) or a misplaced actiwatch (one participant). Nineteen participants (12.3%) had one night of missing actigraphy data and two participants (1.3%) had two nights of missing data. Only one subject had more than two nights of missing actigraphy data not explained by malfunction. Given missing and non-normal data, Full Information Maximum Likelihood estimates with robust standard errors were used for all SEM analyses and Yuan-Bentler Scaled $\chi^2$ statistics are reported for tests of model fit (Yuan & Bentler, 2000).

Descriptives

Descriptives for the student sample are presented in Table 1. The correlation matrices describing relationships between model variables, outcome variables and demographic and health behaviour variables, and between psychosocial variables and demographic and health behaviour variables are presented in Tables 7, 8, and 9, respectively. Age demonstrated positive correlations body mass index and BP indices. In addition, age was inversely
correlated with self-reported sleep quality and depression. Women had a lower body mass index than men, longer total sleep time, lower number of wake bouts, and lower SBP and DBP. As well, women reported higher levels of perceived stress and social support.

Psychotropic medication use in two subjects (coded ‘yes’ or ‘no’) was associated with longer total sleep time. Lack of exercise (coded ‘yes’ or ‘no’) was significantly correlated with lower childhood SES. A one-way ANOVA revealed differences in total sleep time amongst ethnic groups, \(F(2, 148) = 3.33, p < .05\). A Tukey post-hoc test revealed marginally significant differences in sleep time across Caucasian \(M = 407.82, SD = 48.36\) and Asian \(M = 385.53, SD = 56.95\) participants \(p = .06\). No other significant relationships between ethnicity and psychosocial or health outcome variables were demonstrated.

In the overall student sample, 12.9% reported they had the following sleep related difficulties: 1.3% indicated they were diagnosed with insomnia by a physician, 0% reported sleep apnea, 0% restless leg syndrome, 0% night terrors, 4.5% sleep talking, and 9% bruxism (i.e., tooth grinding). Further, 5.2% indicated they suffered from anxiety and/or depression, 1.3% reported they had an eating disorder (two subjects) and one subject indicted they had been diagnosed with attention-deficit/hyperactivity disorder. The sample largely consisted of university students (85.2%), 9.7% were employed full-time, 41.9% were employed part-time, and 48.4% were unemployed, and 33.9% reported they were married or in a long-term relationship.

In terms of family risk, 60% of the sample endorsed some degree of verbal abuse (i.e., endorsed questions pertaining to being sworn at, insulted, put down or made to feel threatened above a 1 denoting no verbal abuse), 40% of the sample reported physical abuse
(e.g., pushed, shoved or slapped), 37% endorsed domestic violence, 41% reported neglect, and 9% endorsed having being exposed to substance abuse above “not at all”.

**Confirmatory Factor Analyses for Negative Affect and Sleep Activity**

A confirmatory factor analysis was conducted to determine whether depression, anxiety, and anger were appropriately modeled as indicators of a single latent negative affect factor in the university sample. The model demonstrated strong reliability estimates (Cronbach’s Alpha = .74; Reliability Coefficient Rho = .84). However, the factor loading for the anxiety indicator was equal to 1, meaning the latent factor was fully explained by this indicator alone. This likely reflects the fact that anxiety is the indicator most related to the latent negative affect construct. Notably, when included in the context of a larger model, this effect was no longer apparent (though the anxiety indicator still demonstrated the strongest factor loading) and therefore the latent factor was deemed acceptable for inclusion in our overall hypothesized SEM model. Anxiety has also been shown to be the indicator most related to a latent negative affect factor in previous studies (e.g., see Stewart et al., 2011, who demonstrate similar factor loading coefficients as those found in the larger SEM models examined here).

A confirmatory factor analysis was also conducted to determine whether select actigraphy variables were appropriately modeled as indicators of a single latent sleep activity factor in the university sample. As anticipated, total sleep time did not load strongly onto the latent factor ($\beta = .02$, $p > .05$) and the latent factor did not explain any of the variance of sleep time ($R^2 = .000$). Therefore, total sleep time was modeled separately. After excluding sleep time, the model demonstrated good reliability estimates (Cronbach’s Alpha = .60;
Reliability Coefficient Rho = .86) (see Figure 6) and each indicator was significantly predicted by the model. Therefore, both latent factors were retained for further analyses.

**Theoretical Model Testing: Self-Reported Sleep Quality**

Data from the university sample were fit to the hypothesized risky family model depicted in Figure 1 with self-reported sleep quality as the health risk outcome variable of interest. Initial analysis of the theoretical model indicated lack of model fit \[\text{Yuan-Bentler } \chi^2 (16) = 51.51, p < .001, \text{CFI} = .92, \text{RMSEA} = .12, 90\% \text{CI} = .09, .16\] (see Figure 7). LM tests suggested adding a path between childhood SES and the negative affect factor would improve model fit. Even after adding the path between childhood SES and negative affect, inspection of residuals revealed that large residuals between childhood SES and depression (standardized residual = .16) and childhood SES and anger (standardized residual = .12) remained, creating misfit in the model. Given that we were primarily interested in childhood SES insofar as it contributed to risky family environment, the variable was omitted from subsequent models so as to focus on risky family environment. Results from the MTURK data analysis also suggested that childhood SES may not be as important a contributor to model as it is specified, supporting our decision to omit it from the model.

Once childhood SES was omitted from the model, inspection of residuals suggested that the model did not fully explain the relationship between risky family environment and self-reported sleep (standardized residual = .15), as such a direct path was added between the two variables. Following this modification the model fit the data according to overall and comparative fit indices \[\text{Yuan-Bentler } \chi^2 (9) = 16.75, p > .05, \text{CFI} = .99, \text{RMSEA} = .07, 90\% \text{CI} = .00, .13\] (see Figure 8 and Table 10), though the RMSEA was slightly higher than desired. Results indicated that consistent with findings from the MTURK sample, greater
endorsement of a risky family environment was significantly associated with lower levels of social support (explaining 8% of the variance in social support) and higher levels of perceived stress. Higher perceived stress in turn was strongly associated with greater negative affect, and greater negative affect was significantly related to poorer self-reported sleep quality. Also consistent with previous analyses, perceived stress did not show a significant association with sleep quality. Inspection of indirect effects (see Table 11) suggested that the relationship between perceived stress and sleep quality was mediated through its relationship with negative affect. Further, the indirect path between social support and self-reported sleep quality was small and just barely significant, similar to results found in the MTURK sample. Indirect effects also suggested that the relationship between risky family environment and negative affect was mediated by social support and perceived stress, and that although a direct path was added between risky family environment and sleep quality, the relationship between the two variables was still partially mediated by psychosocial variables in the model. Altogether, perceived stress, negative affect and risky family environment explained 36% of the variance in self-reported sleep quality.

While many of the hypothesized relationships were supported in the model, some of the paths that were weaker in the MTURK model analyses failed to reach significance (i.e., the path between risky family environment and negative affect). This is likely in large part due to differences in sample size, as the student sample was almost half that of the online sample. The model revealed similar rates of explained variance for perceived stress (15%) and negative affect (71%). Contrary to previous findings, social support was not significantly associated with perceived stress ($\beta = -0.10, p > .05$ in the university sample, compared to $\beta = -0.31, p < .05$ in the online sample). In sum, childhood SES was not a significant contributor to
risky family environment. After excluding SES from the model, a risky family model was largely supported, though the relationship between family risk and self-reported sleep was only partly mediated by psychosocial variables in the model.

**Theoretical Model Testing: Latent Sleep Activity**

The risky family model depicted in Figure 1 was examined with respect to a latent sleep activity variable, characterizing increased motor movement and motor movement coded as wake during the sleep period. Childhood SES was similarly excluded from this model, as it was for all subsequent models. Initial analysis revealed that the model did not fit the data \[ \chi^2 (22) = 37.84, p < .05, \text{CFI} = .95, \text{RMSEA} = .07, 90\% \text{ CI} = .03, .11 \] and neither perceived stress nor negative affect significantly predicted the latent sleep quality variable (see Figure 9). LM tests and inspection of standardized residuals suggested adding a path between risky family environment and the latent sleep quality factor, similar to self-reported sleep quality. Following this modification, the model fit the data \[ \chi^2 (21) = 31.57, p > .05, \text{CFI} = .97, \text{RMSEA} = .06, 90\% \text{ CI} = .00, .10 \] and the path between risky family environment and the latent sleep factor was significant, suggesting the model failed to fully mediate this relationship (see Figure 10 and Table 12). Relationships amongst the variables common to the previously assessed model remained unchanged. Indirect effects suggested there was no partial mediation between risky family environment and the latent factor, and that there was no indirect relationship between perceived stress and the latent sleep activity factor (though with a larger sample size the regression coefficient of .15 may have reached significance; see Table 13). Contrary to our hypothesis and to results found with subjective sleep data, negative affect demonstrated a negative, though non-significant, relationship with sleep activity, suggesting that individuals with greater
negative affect tended to demonstrate less physical restlessness and actigraphy assessed wake activity during sleep. However, overall, risky family environment was the only statistically significant contributor to the latent sleep activity factor and only a small proportion of the variance in this factor was explained (5%). Therefore, while the overall risky family model was not supported, family risk was still associated with disturbed sleep.

**Theoretical Model Testing: Total Sleep Time**

The risky family model was also tested with actigraphy assessed total sleep time as the outcome of interest. Initial analysis revealed that model did not fit the data \([Yuan-Bentler \chi^2 (10) = 25.04, p < .05, CFI = .97, RMSEA= .10, 90\% CI = .05, .15]\) and neither perceived stress nor negative affect were significant predictors of sleep time (see Figure 11). A large standardized residual (.20) was observed between social support and total sleep time, suggesting that stress and negative affect did not completely mediate this relationship. Social support has been shown direct relationships with sleep indices in previous research (Friedman et al., 2005; Troxel et al., 2010), therefore the model was modified to include this path. Following modification the model fit the data according to model and comparative fit indices \([Yuan-Bentler \chi^2 (9) = 16.29, p > .05, CFI = .98, RMSEA= .07, 90\% CI = .00, .13]\) (see Figure 12 and Table 14), and the path between social support and total sleep time was significant. Overall, the model explained 7% of the variance in total sleep time (only 2% of the variance of total sleep time was explained prior to the addition of a path from social support). A small but significant inverse indirect path was found between risky family environment and total sleep time following the addition of the path from social support, suggesting that social support in particular may mediate some of the effect between these two variables (see Table 15). Therefore, while the hypothesized risky family model was largely
not supported, once again family risk was associated with sleep time, through a social support pathway.

**Theoretical Model Testing: Body Mass Index**

The hypothesized model depicted in Figure 1 was also assessed with BMI as the health risk indicator outcome of interest. Initial analysis revealed that model did not fit the data [$\chi^2 (10) = 30.63, p < .05, CFI = .94, RMSEA = .12, 90\% CI = .07, .16$] (see Figure 13). Inspection of residuals revealed a very large standardized residual between social support and BMI (-.27) suggesting that perceived stress and negative affect did not fully mediate the relationship between social support and BMI. As such, this path was added. Following this modification the model fit the data [$\chi^2 (9) = 15.17, p > .05, CFI = .98, RMSEA = .07, CI = .00, .12$] (see Figure 14 and Table 16), though once again the RMSEA was slightly higher than desired. Results suggested that relationships amongst psychosocial variables remained unchanged from previous analyses and that higher levels of social support were associated with a lower BMI, while greater perceived stress was associated with a higher BMI. Contrary to what was anticipated, negative affect once again demonstrated a negative relationship with this health risk outcome with greater negative affect was associated with a lower BMI. Overall, social support, perceived stress and negative affect explained only 13\% of the variance in BMI. Examination of indirect effects revealed that the relationship between perceived stress and BMI was partially mediated by the model. The indirect relationship between risky family environment and BMI was not significant, again failing to support mediational hypotheses with respect to the relationship between risky family environment and BMI (see Table 17).
Previous research has indicated that underweight and overweight individuals are more prone to mental health issues than those with a normal BMI (Carpenter et al., 2000; Zhao et al., 2009). To explore whether under- or overweight individuals demonstrated greater negative affect, a one-way ANOVA was conducted following with BMI categorized as underweight (< 18.5), normal (18.5-25), or overweight (>25). Results did not indicate any differences in anxiety ($F(2, 155) = 1.27, p = .29$), depression ($F(2, 155) = 2.00, p = .14$), or anger ($F(2, 155) = .05, p = .96$) amongst categories.

**Theoretical Model Testing: Systolic Blood Pressure**

Systolic BP was examined as another potential disease risk outcome of a risky family environment. Initial analysis revealed that model did not fit the data [$\chi^2(10) = 28.90, p < .05$, CFI = .95, RMSEA= .11, 90% CI = .06, .16] and neither perceived stress nor negative affect were significant predictors of SBP (see Figure 15). Similar to BMI analyses, inspection of standardized residuals revealed a very large residual between social support and SBP (-.26) suggesting that perceived stress and negative affect did not fully mediate the relationship between social support and SBP. Once again, this is consistent with a number of studies demonstrated the importance of social support in explaining BP (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). As such, this path was added. Following this modification the model fit the data [$\chi^2(9) = 14.78, p > .05$, CFI = .99, RMSEA= .06, 90% CI = .00, .12] (see Figure 16 and Table 18). Results indicated that lower social support was significantly related to higher SBP. Direct paths between perceived stress and negative affect were not significant, nor was the indirect path from risky family environment (see Table 19). Therefore, mediational relationships were once again not supported. The model explained 10% of the variance in SBP.
Theoretical Model Testing: Diastolic Blood Pressure

With respect to DBP as the outcome variable of interest, initial analysis revealed model fit [Yuan-Bentler $\chi^2 (10) = 17.33, p > .05$, CFI = .98, RMSEA = .07, 90% CI = .00, .12], though the RMSEA was slightly higher than desired. However, neither perceived stress nor negative affect were significant predictors of DBP (see Figure 17). Similar to SBP and BMI analyses, inspection of standardized residuals revealed a large residual between social support and SBP (-.16). As such, this path was added. Following this modification the model also fit the data [Yuan-Bentler $\chi^2 (9) = 12.81, p > .10$, CFI = .99, RMSEA = .05, 95% CI = .00, .11] (see Figure 18 and Table 20). Higher levels of perceived social support was associated with lower DBP. The model explained only 4% of the variance in DBP. Similar to SBP, evidence for mediational relationships between risky family environment and DBP were not supported when indirect effects were examined (see Table 21).

Sleep as a Mediator between Psychosocial Variables and Body Mass Index

Given that poor sleep has been associated with a variety of negative health outcomes, secondary analyses in the current study focused on examining sleep as a mediator of the relationships between adult psychosocial variables and health indicator variables, namely BMI and BP. Selection of variables to include in mediational analyses were based on Baron and Kenny (1986) as described in the statistical analyses section.

Self-reported sleep quality. Given that previous analyses demonstrated that negative affect was associated with self-reported sleep quality in the university sample, as well as with BMI (though in the opposite direction than anticipated), self-reported sleep quality was assessed as a mediator between negative affect and BMI. The overall model demonstrated model fit [Yuan-Bentler $\chi^2 (4) = 7.68, p > .05$, CFI = .99, RMSEA = .08, 95% CI = .00, .16]
and negative affect remained significantly associated with self-reported sleep quality (see Figure 19 and Table 22). However, neither negative affect nor sleep quality were significantly associated with BMI, and correspondingly, the indirect effect between negative affect and BMI was not significant ($\beta = .07$, $p > .05$). Therefore, self-reported sleep quality did not mediate the relationship between negative affect and BMI.

**Sleep activity.** Previous SEM analyses revealed that none of the psychosocial variables included in the model were significantly associated with the latent sleep activity variable. As such, mediational analyses assessing sleep activity as a mediator between psychosocial model variables and BMI were not conducted.

**Sleep time.** Social support was significantly associated with total sleep time and with BMI in previous SEM analyses. Therefore, total sleep time was assessed as a mediator of the relationship between social support and BMI. Given that the model was just-identified (i.e., had 0 degrees of freedom) $\chi^2$ statistics were not produced. However, inspection of regression coefficients revealed anticipated relationships. Specifically, high social support was associated with greater sleep time and with lower BMI, and higher sleep time was also associated with lower BMI (see Figure 20 and Table 23). Despite a significant relationship between social support and BMI, a very small but significant indirect effect between social support and BMI was also found ($\beta = -.04$, $p < .05$), suggesting that sleep time may partially mediate the relationship between social support and BMI to a small degree.

**Sleep as a Mediator between Psychosocial Variables and Blood Pressure Indices**

Similar to BMI, we hypothesized that sleep parameters would mediate the relationship between psychosocial model variables and BP indices. Given that only social support was found to be significantly associated with SBP and DBP of all the model
psychosocial variables, only social support was examined. Further, social support was found to be directly predictive of total sleep time only amongst sleep parameters. Therefore, total sleep time was examined as a mediator between social support and SBP and social support and DBP. Given that the models were just-identified (i.e., had 0 degrees of freedom) \( \chi^2 \) statistics were not produced. With respect to SBP, results indicated that higher social support was significantly associated with greater total sleep time, as well as with lower SBP (see Figure 21 and Table 24). Total sleep time was not significantly associated with SBP and the indirect effect between social support and SBP was also not significant (\( \beta = -.03, p > .05 \)). Therefore, sleep time did not mediate the relationship between social support and SBP. With regard to DBP, while higher social support was associated with greater total sleep time, neither total sleep time, nor social support were significantly associated with DBP (see Figure 22 and Table 25). Overall, sleep time did not mediate the relationship between social support and BP indices.

**Study 1 and Study 2 Sample Comparisons**

In light of some discrepant findings between SEM model analyses in Study 1 as compared to Study 2, differences in variances and means of observed model variables were explored. A series of independent t-tests revealed unequal variances for all variables, with the exception of perceived stress. Overall, the student sample was a more homogenous group. Further, the online sample was older, reported lower childhood SES, poorer sleep quality, and a higher BMI, and endorsed a riskier childhood environment (see Table 26).
Discussion

The primary aim of the current studies was to describe mediational relationships between early life stress and individual indicators of CVD risk (i.e., sleep parameters, BP and BMI). Consistent with previous research, results indicate that early life stress is likely an important determinant of adult psychosocial functioning. Evidence from the two studies outlined above indicated that retrospective report of a risky family environment (i.e., a family environment characterized by various adverse factors, including family conflict or violence, household dysfunction, neglect, and lack of parental warmth) in childhood was associated with poorer adult psychosocial functioning, including social support, perceived stress, and negative affect, through both direct and indirect pathways depending on psychosocial construct. Analyses examining adult psychosocial factors as mediational pathways between early life stress and indicators of CVD risk were less consistent, as discussed below. Further, secondary hypotheses pertaining to sleep as a mediator between psychosocial factors and other health risk indicators were largely unsupported. Notably, both studies were cross-sectional designs, and therefore do not allow for causal or directional conclusions with respect to examined relationships.

Early Life Stress and Self-Report Sleep Quality

Online study. The online study was conducted to determine whether the proposed models predicting sleep outcomes were supported prior to engaging in the more costly and time consuming actigraphy study. This also allowed for examination of these relationships in a non-student sample. Overall, results obtained from structural equation analyses with the online data were highly consistent with our hypothesized model.

In the first step of the model, retrospective report of low childhood SES was
associated with greater endorsement of a risky childhood family environment, though SES explained only a small proportion of the variance in family risk. This finding is consistent with the hypothesis that chronic situational stress may contribute to higher levels of family dysfunction, including increased marital and family conflict, leading to inconsistent, neglectful, or abusive parenting. On the other hand, this result may also reflect individual differences in the personal characteristics of family members that affect both their SES standing, and their family relationships (Conger, Conger, & Martin, 2010). For example, low intelligence or emotional dysregulation may be associated with difficulties maintaining financial stability, and may also be associated with increased risk of child maltreatment.

While childhood SES has shown associations with several of the psychosocial and health outcome variables examined in this study in previous research (e.g., Kivimäki et al., 2006; Tomfohr et al., 2010), results support a more restrictive model, with SES contributing indirectly to model variables through risky family environment. Very small, though significant, indirect relationships were found between childhood SES and endogenous variables in the model, suggesting that childhood SES may not be a significant contributor to adult psychosocial or sleep outcomes once family environment is considered. These findings may also reflect generally weak associations between childhood SES and psychosocial constructs in the model overall, irrespective of family environment. For example, in correlational analyses between childhood SES and psychosocial variables, only a small significant correlation between childhood SES and adult perceived stress was observed. Overall, results support childhood SES and risky family environment as distinct constructs and suggest that risky family environment may be a more meaningful predictor of health.
outcomes. Given that both constructs were assessed via retrospective self-report, risky family environment may therefore be a preferable measure for inclusion in future research.

Results from both the online and university sample, suggested that on average participants had experienced a moderate level of family risk, consonant with previous research demonstrating that family environments with at least some level of dysfunction are quite prevalent (Anda et al., 2006). Retrospective recall of a risky family environment in childhood was associated with lower levels of perceived social support and higher levels of perceived stress and negative affect in adulthood, consistent with the supposition that early life stress translates to poorer psychosocial outcomes in adulthood. As reviewed earlier, such a result may stem from early alterations in neurobiological systems leading to increased responsivity to stress and negative emotionality. Additionally, these findings may reflect family level influences on social and emotional development and coping. Results indicated that low social support and higher perceived stress may account for some of the observed relationship between risky family environment and negative affect. Furthermore, for individuals reporting greater family risk, higher levels of perceived social support in adulthood may attenuate perceived stress.

Take as a whole, individuals from households with higher levels of family dysfunction may not adequately develop the interpersonal capabilities required to attain and maintain social support networks, or may not be able to benefit from support from others, thereby reporting less perception of support. Risky family environment alone explained 11% of the variance in social support. In turn, this lack of perceived support appears to translate to higher levels of perceived stress and greater negative emotional states such as depression, anger, or anxiety. This study did not allow for conclusions pertaining to whether childhood
family risk is associated with the actual experience of more objective life stress, or whether individuals from riskier families are more sensitive and reactive to stress, thereby reporting more perceived stress than others at similar levels of objective life stress. Regardless, recall of family dysfunction in childhood does seem related to adult levels of perceived stress, both through direct and indirect pathways. The combined influence of family risk and social support demonstrated a moderate effect on perceived stress, explaining 18% of the variance in perceived stress.

Contrary to our hypothesized model, perceived stress did not show a significant direct effect with respect to self-reported sleep quality. Instead, higher levels of perceived stress appeared to negatively impact sleep quality through increases in negative affect. This is consistent with previous research demonstrating that individuals experiencing childhood adversity are more likely to develop depressive or anxious symptoms following increased psychological stress (Espejo et al., 2007; Hammen et al., 2000). However, given the cross-sectional nature of the current study we cannot presume directional effects.

The findings were consistent with the conceptualization that a latent, overarching negative affect construct, thought to underlie the tendency to experience negative affective states, may be more related to sleep quality than unique indicators of the latent factor (i.e., depression, anger, anxiety). Overall, the model explained 22% of the variance in self-reported sleep quality, representing a moderate effect size (Cohen, 1992). Multiple-groups analyses exploring invariance across gender did not reveal differences in the structural or measurement model in terms of path coefficients, suggesting the model applies to both men and women.
Health behaviour variables. While we theorize that early life stress contributes to sleep disturbance through psychosocial pathways, an alternative explanation also exists. Namely, early life stress may lead to negative health behaviours that may then impact sleep (see Repetti et al., 2002). As such, the risky family model described above was adjusted by controlling for reported health behaviours that demonstrated a significant relationship with self-reported sleep (i.e., dichotomous coding of smoking and exercise). Inclusion of these health behaviour variables did not meaningfully impact modeled relationships. However, there are several other health behaviour variables (e.g., poor diet, alcohol use) that may contribute to poor sleep but were not assessed in this study. As such, future research may wish to include a more comprehensive analysis of health behaviours. Importantly, several studies have demonstrated that associations between early life stress and mental and physical health outcomes hold even after controlling for health behaviours (e.g., Lehman et al., 2005; Tomfohr et al., 2010).

University study. A risky family model examining self-reported sleep as a health risk outcome was also analyzed with data from the university sample in Study 2. Overall, results support the hypothesis that a risky family environment is associated with poorer adult psychosocial functioning, which in turn may lead to poor sleep. However, results were not as consistent with the hypothesized model compared to the online sample. First, inconsistent with our conceptual model, childhood SES was not significantly associated with report of a risky family environment. Instead, childhood SES was most strongly related to unique indicators of negative affect, namely depression and anger. The model, along with correlational analyses, did not suggest that childhood SES was closely associated with any other psychosocial construct in the model, or to sleep quality. It is unclear why childhood
SES did not show the expected relationship with family risk. One potential factor may simply be sample characteristics. The university sample was somewhat more homogenous than the online sample with respect to childhood SES and also reported a somewhat higher average and lower end range of SES than did the online sample. This may suggest that overall, university participants came from relatively affluent backgrounds. In addition, relationships, or lack thereof, between childhood SES and model variables may also be spurious. Coding of parental occupation responses in both samples was often difficult due to vague or indecipherable responses. Therefore, measurement error is likely higher for this scale than for other study measures. However, there was enough variability in childhood SES in the student sample to demonstrate relationships with depression and anger. Such a result is consistent with previous research describing such relationships (Gallo & Matthews, 2003; Gilman, Kawachi, Fitzmaurice, & Buka, 2002). Therefore, these findings may reflect that factors other than childhood SES contribute to a risky family environment in this specific sample.

Second, after omitting childhood SES from the model so as to focus on risky family environment, model analyses revealed that the relationship between risky family environment and self-reported sleep quality was not fully explained by adult psychosocial functioning. As such, a direct relationship was modeled between family risk and sleep quality to obtain model fit. We did not find this modification overly problematic because the indirect relationship between this index of early life stress and our sleep quality outcome was still significant, suggesting that the relationship was partially mediated by psychosocial constructs in the model.

Third, two paths that were significant in the online analysis were not significant with the university sample. This may in part be due to a substantially smaller sample size (almost
half that of the online sample), particularly when considering the weaker direct path between risky family environment and negative affect (the indirect effect was still significant suggesting that social support and perceived stress mediated this relationship). However, a significant deviation was found with respect to the impact of social support on perceived stress. This relationship appeared fairly robust with data from the online sample, while it was small and non-significant in the student sample, though the path still demonstrated the expected inverse relationship. Once again, this result may be due to differences in sample characteristics. For university students, academic stress may be a particularly poignant (Abouserie, 1994), while other sources of stress may be predominant for community populations. The online sample was, on average, older than the university sample, and almost twice as likely to be married or in a long-term relationship. Thus, social support may derive more from spousal or family relationships for these individuals, and sources of stress may also be more related to family functioning. For example, experiencing financial insecurity as an adult may be associated with greater perceived stress when caring for a family, especially if one does not perceive their spouse to be supportive. Meanwhile, academic stress may be less related to interpersonal functioning or support. While peer or family support may buffer against exam stress, other internal, goal or mastery related motivations might still induce stress, mitigating some of the buffering effect of social support. However, research has found that social support may be inversely related to academic stress (Felsten & Wilcox, 1992; Solberg & Viliarreal, 1997) and that students are vulnerable to interpersonal stress (Ross, Neibling, & Heckert, 1999). As such, further research is required to further explore differences in relationships between social support and perceived stress in student vs. nonstudent samples.
Another notable difference between sample populations pertains to ethnicity. In the online sample, participants were predominantly Caucasian (approximately 78%), while in the student sample the majority of participants (approximately 60%) endorsed an Asian heritage. Accumulating evidence has consistently suggested that individuals from Asian backgrounds are less likely to seek social support for coping with stressful life events (across a range of stressors including social, academic, and health stressors) when compared to individuals from Western backgrounds (Kim, Sherman, Ko, & Taylor, 2006; Wang, Shih, Hu, Louie, & Lau, 2010). This may pertain particularly to emotional support. For example, in a series of studies Taylor, Sherman, et al. (2004) found that individuals from Asian backgrounds (including Asian Americans) were less likely to endorse seeking emotional support to cope with stress than individuals of European descent. Further, acculturation mitigated this finding to a degree. Specifically, second-generation Asian Americans were more likely than first-generation Asians to seek emotional support. Differences in support seeking across cultural groups are presumed to be due to divergent views of the self and relationships. Asian cultures tend to emphasize interdependence and social harmony, meaning individuals may be more reluctant to impose their personal needs on the social group, as opposed to Western cultures that tend to emphasize independence. Indeed, Taylor, Sherman, and colleagues (2004) found that Asian participants endorsed disturbing the harmony of the group, losing face, and receiving criticism as reasons to avoid seeking social support to cope with stress.

Taken as a whole, this line of research suggests that in the current study differences in the magnitude of the pathway between perceived social support and perceived stress across study samples may be due to cultural differences in support seeking. However, due to sample size more detailed analyses in this respect were not conducted. Further, analyses controlling
for other potentially relevant demographic or health related behaviours were also not conducted. Age and gender did show associations with several of the outcome variables, including BMI, BP indices, and several of the sleep parameters. As such, studies that are more adequately powered should be conducted to examine differences across ethnicity, age, and gender. Notably, results stemming from the online study suggest that relationships between conceptual model constructs may not be overly impacted by inclusion of these factors.

**Early Life Stress and Objective Sleep Measurements**

While the conceptual model was relatively well supported in the online and university study with respect to self-reported sleep quality, results are limited in that they rely on self-report rather than objective measurement of sleep parameters. As such, study findings may reflect response style rather than accurate reflections of sleep architecture. To address this limitation, objective sleep measurements were obtained from the university sample, which provided data on motor movement during sleep, as well as on sleep length.

**Early life stress and motor activity during sleep.** Actigraphy derived indices of physical restlessness and wakefulness were used as indicators of an underlying factor related to activity during sleep, presumed to reflect sleep disturbance. Overall, evidence did not support the hypothesis that psychosocial factors would mediate the effect of risky family environment on the latent activity factor. Instead, risky family environment was directly associated with the factor. This finding is consistent with previous work that also demonstrated a relationship between adverse childhood experiences and increased motor movement during sleep, independent of a stress induction manipulation (Bader et al., 2007). On the one hand, this result is positive and in support of the prediction that family
dysfunction is important in explaining adult sleep processes. On the other hand, it leads to questions as to how this dysfunction carries into adulthood to affect sleep if not through stress and affective pathways. Once again, given small sample size we did not address health behaviours that may account for some of this relationship. However, correlational analyses did not suggest any meaningful relationships between motor activity indicators and health behaviours reported on in this study. A second possibility is that we did not account for specific cognitive factors that may more specifically relate to sleep disturbance. Depression, anxiety, and even anger are associated with cognitive factors such as worry and rumination that are presumed to be the mechanism by which these mental health constructs influence sleep, at least in part (Brosschot et al., 2005; Guastella & Moulds, 2007). As such, effects may be more robust if studies account for these factors directly. Further, increased physiological hyperarousal or hypervigilance often associated with early childhood adversity may also account for the observed direct relationship between family risk and sleep activity (Bader & Schäfer, 2007; see below). Notably, the relationship between family risk and sleep activity was based on model modification, increasing the likelihood that this is a spurious finding. As such, replication is needed.

**Early life stress and total sleep time.** Sleep length has been consistently linked with adult health outcomes (Cappuccio et al., 2011), and has also been associated with early life stress (Hanson & Chen, 2010). As such, total sleep time was also examined as an independent outcome of a risky family environment model. Results supported the indirect association between risky family environment and sleep time, however, this appeared to be through relationships with social support, rather than via perceived stress and negative affect as anticipated. Specifically, consistent with the latent sleep activity analyses, perceived stress
and negative affect did not show significant associations with total sleep time as was hypothesized. Meanwhile, social support demonstrated a direct relationship with sleep time, independent of the relationship between social support and stress and negative affect. We hypothesized that social support would mitigate the impact of perceived stress and negative affect on sleep outcomes. However, social support demonstrated only weak associations with these adult psychosocial outcomes, which may account for the observed direct relationship between social support and sleep time. Once again, questions remain as to how social support may work to affect sleep length if not through stress and affective pathways.

Social support is thought to promote better physical health outcomes through several pathways, including by buffering negative psychological states as we hypothesized. However, social support may also promote healthy behaviour (Cohen & Wills, 1985; Uchino, 2006) and in this way positively impact sleep length (e.g., may be associated with less alcohol use or smoking which may affect sleep), or more specifically, promote good sleep hygiene (e.g., consistent bedtimes and earlier time to bed). Alternatively, social support may also be associated with greater feelings of safety and security, which are also posited to promote better sleep (Dahl & Lewin, 2002).

**Physiological Indicators of Health Risk**

While sleep indices were the primary outcome variables of interest in this study, we also sought to examine whether early life stress had manifested in more tangible physical ways in otherwise healthy young adults, as such, we looked at health indices often associated with CVD development, including BMI and BP.
Body mass index.

**Online sample.** The overall risky model assessing BMI as the health risk indicator of interest did not fit the underlying data structure in the online sample. When bivariate correlations between BMI and model variables were examined with the outlier excluded from the analyses, BMI demonstrated significant correlations with childhood SES, social support, and anger. Specifically, low childhood SES and perceived social support were associated with a higher BMI, and higher ratings of anger were also associated with a higher BMI. These findings are consistent with the supposition that early life stress (in this case low SES) is associated with adult health outcomes and adult psychosocial functioning (i.e., social support and anger). However, our model failed to capture overall relationships in this regard. Notably, in the online study BMI was calculated based on self-reported height and weight which may have increased measurement error.

**University sample.** In the university sample, despite the fact that risky family environment was associated with adult psychosocial functioning, which was in turn associated with BMI, results did not support mediational hypotheses because the indirect effect between risky family environment and BMI was not significant. Furthermore, similar to sleep time analyses, the relationship between social support and BMI was not mediated by perceived stress and negative affect. Instead, social support demonstrated a direct inverse relationship with BMI, which was added to the model subsequent to the preliminary analysis. Once again, this may be due to the relationship between social support and health behaviour promotion. Also not anticipated was the finding that negative affect demonstrated an inverse relationship with BMI; namely, higher negative affect was associated with a lower BMI. Previous work assessing the relationship between negative emotional dispositions such as
anxiety and depression and BMI has also been inconsistent, at times showing a lack of relationship (John, Meyer, Rumpf, & Hapke, 2005) or an inverse or u-shaped relationship, with risk of poorer mental health associated with both over- and underweight individuals (Carpenter et al., 2000; Zhao et al., 2009). Therefore, this finding should be considered in this context.

**Blood pressure.** Similar to BMI, a risky family model was not supported in terms of either SBP or DBP. While models fit the data, the indirect relationships between risky family environment and BP indices were not significant, nor were paths from perceived stress and negative affect to BP indices. Social support was once again found to be an important predictor of both SBP and DBP. Null findings with respect to relationships between perceived stress and negative affect were unexpected. A substantial portion of the literature has demonstrated that psychological functioning is closely connected to cardiovascular reactivity and delayed recovery, which are thought to increase tonic blood pressure (see Chida & Hamer, 2008). It is possible that using such a young, healthy population meant that detrimental effects from stress and negative affect had not yet manifested. As such, future studies should examine these relationships in a community-based population comprised of an older, nonstudent sample. Further, directly examining blood pressure reactivity and recovery, rather than the hypothesized outcomes of such constructs may be more fruitful.

**The Relationship between Sleep and Health Risk Outcomes**

Contrary to our hypotheses, the current studies found little evidence to support the supposition that sleep is an intermediary mechanism by which psychosocial model variables translate to health risk. A very small but significant indirect effect was uncovered between social support and BMI when total sleep time was examined as a mediator of this
relationship. However, the magnitude of the relationship was not practically meaningful and all other analyses were not in support of original hypotheses. Notably, causal directions with regard to the relationship between sleep time and BMI cannot be accounted for in this study. While short sleep duration has been associated with BMI cross-sectionally, longitudinal results have been mixed and it is possible that BMI may contribute to shorted sleep duration due to other health-related factors such as sleep apnea, lack of physical exercise or poor nutrition (see Cappuccio et al., 2008; Marshall et al., 2008). As such, additional longitudinal studies are needed. Additionally, examining sleep as a mediator between adult psychosocial health and health risk outcomes with a more heterogeneous sample may be more beneficial in uncovering some of these relationships.

**Strengths and Limitations**

The line of research described above has several strengths. It is the first known research to examine a comprehensive risky family model predicting sleep outcomes. While associations between early life stress and adult sleep outcomes have been supported, very few studies have described mechanisms by which this relationship is formed. Overall, results were highly consistent with the supposition that risky family environment in childhood affects adult sleep parameters. However, support for the proposed mediational pathways were mixed. An additional strength of this research was the use of an online sample and a university-based sample which allowed us to replicate and cross validate the model with respect to subjective report of sleep quality, with largely corresponding findings. Further, the university study examined both subjective and objective measures of sleep, rather than solely relying on subjective sleep measurement, as is predominant in the existing literature base. Results suggested that risky family environment is associated with both the subject
experience of sleep, as well as objective actigraphy derived sleep parameters, through indirect or direct pathways.

Examination of both subjective and objective measures of sleep is important when considering health risk. Objective sleep assessment allows for data collection that is not coloured by subjective experience and allows for greater accuracy with respect to capturing nocturnal activity such as restlessness and wake periods. However, subjective sleep measurement is also important because it goes beyond sleep architecture and is related to factors such as daytime fatigue, which may be associated with health behaviours such as physical exercise, or with increased irritability or negative mood. Objective sleep assessment may also be impractical when conducting large prospective studies. Indeed, the majority of the literature linking sleep duration to CVD risk stems from subject sleep reports (Cappuccio et al., 2011).

Across the two studies, several of proposed pathways that were hypothesized to link early family risk and sleep parameters were supported with respect to subjective sleep quality, but were not supported by studying objective sleep indices. This suggests that other pathways may exist that are not yet included in the model, offering a jumping off point for further research. It may also reveal a limitation of relying on subject report of sleep disturbance. Namely, report of poor sleep may reflect an overall tendency to endorse measures in a way that communicates distress or reflects an underlying propensity for negative mood. For example, depressed individuals have been shown to present themselves more negatively on self-report measures due to cognitive bias (Coyne & Gotlib, 1983). Therefore, paths between negative affect, perceived stress, social support and poor subjective sleep may all reflect this type of response bias. A similar limitation may be noted with
respect to our use of retrospective recall of risky family environment and childhood SES. Depressed individuals are more likely to remember negative events consistent with their negative self-concepts, and therefore may remember and report more negative childhood events (Zuroff, Colussy, & Wielgus, 1983). In turn, associations between risky family environment and adult psychosocial outcomes may be due to this type of response bias. As such, further prospective studies are needed to better examine causal relationships early life stress and adult psychosocial and sleep functioning.

The primary limitation of these studies is also applicable to cross-sectional research in general. Namely, our findings do not allow us to make causal inferences about the identified relationships. For example, while we found that negative affect demonstrated a direct relationship with poor sleep quality and also mediated the relationship between perceived stress and sleep quality, studies have also shown that sleep deficits can affect mood and the subjective experience of stress (for review see Meerlo, Sgoifo, & Suchecki, 2008), as well as symptoms of depression and state anxiety (Babson, Trainor, Feldner, & Blumenthal, 2010; Pilcher, Ginter, & Sadowsky, 1997; Selvi, Gulec, Agargun, & Besiroglu, 2007). Further, while the model was specified in a way that would support a stress sensitization hypothesis with regard to adult psychological functioning (i.e., individuals with greater family risk have greater negative emotional responses to stress), it is possible that higher negative affect may also be causally related greater objective life stress, or may be associated with report of greater perceived stress due to feeling overwhelmed and limited with regard to the capacity to meet daily expectations (Hammen, 2006). Experimental studies would better tease apart these directional relationships, though it is likely that bidirectional relationships exist.
Given that the studies did not allow for causal claims about relationships, alternative models that also fit the underlying covariance structure may exist. Other statistical limitations of the current research include small sample size with respect to the university study, limiting our ability to detect potentially meaningful relationships amongst study variables. Further, models were modified from the original conceptual model based on LM tests or standardized residuals that indicated misfit. While no modifications were deemed theoretically inconsistent, they do increase the likelihood of capitalizing on chance. In addition, multiple comparisons were made with the same data set, increasing the likelihood of Type I error. As such, further replication is needed in order to replicate and cross-validate the research findings outlined here.

Another limitation of the current research is that while we presume heightened stress response systems are the common underlying factor linking early life stress to adult psychological and sleep outcomes, we did not actually assess whether these individuals were more physiologically reactive to a distinct stressor. The existing literature does provide evidence for this association (e.g., Luecken, 1998; Luecken & Appelhans, 2006), however, research linking early life stress to sleep may be bolstered by examining physiological responsivity to stress, in addition to exploring whether these individuals experience heightened physiological activity accompanying sleep disruption.

Further limitations alluded to above include the use of a student sample comprised largely of first and second year undergraduate students. Students have been found to have highly variable sleep patterns, and increased sleep difficulties as compared to non-student populations (Buboltz, Brown, & Soper, 2001; Lack, 1986). Therefore, our study findings may not replicate to other non-student populations, and similarly, the hypotheses not
supported here may still hold in an older or more heterogeneous sample. However, cross-validation with the online sample suggests that, at least in terms of self-reported sleep, the model did generalize. We were also not able to control for health behaviours in the university sample due to lack of power. However, few significant correlations were observed between model variables and health behaviours assessed via self-report. There were however, significant correlations amongst age and gender and model variables, as such, future studies should examine whether models similarly hold across gender and age ranges. Despite these limitations, aspects of the study design may be considered strengths, including use of a 6-day actigraphy assessment period, which meets a high standard for a sleep study, and the use of psychometrically sound measurement scales, including the risky family environment questionnaire. Therefore, null findings should not be attributable to insensitive measures.

**Future Directions**

One of the primary questions that emanated from the university study is how risky family environment becomes associated with objective measures of adult sleep, if not through stress and affective pathways. From an evolutionary psychology perspective, feeling safe is a prerequisite to falling asleep. Specifically, sleep and flight from a predator or other threat require mutually inconsistent actions and physiological states (Cosmides & Tooby, 2000). As a result, we have great difficulty sleeping when our bodies are physiologically primed for flight, or fight for that matter. Growing up in unstable family household is thought to increase hypervigilance to threat which may then carry into the sleep period through cognitive or physiological pathways (Dahl & Lewin, 2002). Accumulated literature on anxiety disorders, which are common amongst children who have experienced early life stress, may help to elucidate these cognitive or physiological pathways.
Cognitive information processing theory suggests that anxious individuals tend to selectively process threat cues, both physical and psychological, from their environment and overestimate their vulnerability to these threats (Clark & Steer, 1996). A number of studies have demonstrated that individuals with anxiety have increased threat-related attentional biases, even to stimuli outside awareness, than those low in anxiety (e.g., Dalgleish & Watts, 1990; MacLeod, Mathews, & Tata, 1986). For example, MacLeod et al. (1986) studied individuals high on self-reported trait depression or anxiety who were presented with two words on a screen, one a neutral word and one a threat word pertaining to either physical threat (e.g., harm, violence) or social threat (ashamed, scorned). Participants were asked to respond as quickly as possible by pressing a button when a dot probe appeared on the screen. The dot probe would appear in the location of either the neutral or the threat word in a counterbalanced order. Individuals scoring high on measures of anxiety demonstrated a faster response time when the dot probe was preceded by a threatening word than those scoring high on depression or control participants. A recent meta-analysis revealed no difference in threat-related attentional bias between individuals with a clinical diagnosis of anxiety compared those high on self-reported anxiety, nor did they differ between varying anxiety disorders (e.g., GAD, PTSD) or state compared to trait anxiety, suggesting that this bias is a critical component of anxiety in general (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007). This attentional bias is then presumably followed by subsequent physiological, cognitive and behavioural activation aimed at coping with the threat. It follows that increased vigilance to threat at bedtime may be one mechanism by which anxiety is associated with disturbed sleep. Indeed, an insomnia group was significantly more likely to listen to noises in the house or outside compared with good sleepers, perhaps
indicating more hypervigilance to potential threat stimuli (A. G. Harvey, 2000).

Maltreated adults and children have also shown similar attentional biases with regard to threat (e.g., increased vigilance and slower disengagement when confronted with negative emotional cues such as angry faces; Pollak & Tolley-Schell, 2003), though research in this area is more limited. Therefore, this may be a potentially fruitful research direction with regard to sleep. While the current study accounted for anxiety in the context of a more global negative affect factor, future research may want to examine threat vigilance as a causal contributor to poor sleep more specifically. For example, adults who have experienced early life stress may be more vulnerable to sleep disturbances following experimental manipulation of threat salience.

Increased cognitive arousal through worry and/or rumination is also thought to promote sleep disturbance (Bélanger, Morin, Langlois, & Ladouceur, 2004; Thomsen, Yung MehlSEN, Christensen, & Zachariae, 2003). According to Brosschot, Gerin, and Thayer (2006) worry in part reflects an attempt to cope with anticipatory threat, making the individual physiologically primed to deal with the threat. Thus, if worry processes are engaged prior to sleep, this increased cognitive arousal and heightened physiological arousal would be incompatible with sleep. In addition, the research group posit that “unconscious” perseverative cognitions (i.e., worry and rumination) may lead to greater sleep disturbance as they remain active over this period (Brosschot, Verkuil, & Thayer, 2010). Given that brain processes are indeed still active during the sleep period, this hypothesis, while difficult to test, may hold merit. While this study assessed anxiety and depression, which are closely linked with worry and rumination, future studies may wish to examine these specific cognitive processes, particularly occurring prior to sleep, as a mediational pathway between
risky family environment and poor sleep.

Future studies may also want to explore how early life stress affects daily relationships between stress and sleep in adult populations. As noted above, the relationships between stress and sleep are likely complex and bidirectional. Daily stress may contribute to poor sleep, which may then impact mood and how one tolerates stress the following day, again contributing to poor sleep the following night. However, the directional relationship between stress and sleep disturbances is not well understood. Only one known study has examined the temporal relationship between stress and sleep (Morin, Rodrigue, & Ivers, 2003). Using time-series analysis the study found that stress had a small effect on self-reported sleep disturbance but this was mediated by cognitive and somatic arousal at bedtime. Importantly, this research examined these processes in individuals already diagnosed with insomnia. It remains unclear whether these same processes occur in “normal sleepers” and whether these relationships will hold when using objectively measured sleep data. Further, because sleep deprivation is often followed by compensatory increases in sleep duration the following night it may be more revealing to examine within person variability in stress-related sleep disturbances rather than averages across nights or a single night of assessment (Mezick et al., 2009). Using hierarchical linear modeling, Hanson and Chen (2010) found that individuals who had experienced family risk demonstrated more sleep disturbance on nights when they experienced greater stress. Studies using a similar design may better tease apart daily processes, and may lend themselves to the examination of specific cognitive or physiological (e.g., through ambulatory BP monitoring) processes occurring just prior to or during the sleep period that may affect sleep.
As noted above, prospective studies are also needed. In addition to shedding more light on mechanistic pathways hypothesized in this line of research, such studies may also wish to assess how attachment processes contribute to the observed relationships between family risk and adult psychosocial and health outcomes. Early caregiving and family environment is thought to be directly linked to attachment formation, and insecure attachment has been associated with adult psychosocial and sleep outcomes (Carmichael & Reis, 2005; Luecken, 2000; Maunder & Hunter, 2001). As such, attachment style may be an intermediary mechanism by which family risk translates to poorer adult outcomes.

Finally, it should be noted that observed associations between family risk and psychosocial and sleep parameters revealed in these studies may also reflect genetic influences. Approximately 30-50% of the variation in habitual sleep patterns is thought to be due to genetic influences (Barclay, Eley, Rijsdijk, & Gregory, 2011). Additionally, at the root of the risky family model is the supposition that early life stress promotes alterations in neurobiological stress-response systems (e.g., HPA-axis) during critical periods of brain development, increasing vulnerability to a cascade of psychological and health problems over the life span (Heim & Nemeroff, 1999). However, accumulated evidence suggests that risk may be partially dependent on genetic predisposition to stress-reactivity (Harvey, Gehrman, & Espie, 2014). Diathesis stress models, where genetic expression is dependent on the presence of environmental stressors, have been well supported in terms of the development of anxiety and depression (see Heim & Nemeroff, 1999) and there is preliminary evidence to suggest the same may hold for sleep disruption. For example, Brummett et al. (2007) found that a variation in 5-HTTLPR genotype (related to serotonin) interacted with caregiving stress to predict self-reported sleep disturbance (PSQI). Specifically, only individuals with a
specific variation on this allele were susceptible to stress related sleep disturbances and there was no direct relationship between the allele variation and sleep quality independent of stress. This suggests that early life stress may propagate risk only in individuals who are genetically vulnerable. Barclay et al. (2011) also found evidence for a gene-environment correlation with respect to sleep disturbance, whereby genes influencing one trait also influence exposure to specific environments. The research group found that dependent negative life events (those that an individual has a role in bringing about) were more strongly associated with disturbed sleep quality as compared to independent life events (those not influenced by an individual’s behaviour), and that there was substantial overlap in the genes influencing sleep quality and those influencing negative life events. They concluded that individuals more prone to stress-related sleep disturbances may also be more likely to create high-risk environments. This is particularly interesting with respect to risky family environments, because it means that genetic influences may in part account for stressful family environments, as well for sleep disturbances. Notably, the aforementioned studies examined self-reported sleep indices rather than objective measures. Therefore, further examination of genetic influence on early life stress and sleep using objective sleep measurements may be warranted.

Contributions to the Literature

Despite noted limitations, the line of research described above contributes substantially to the existing literature base with respect to early life stress and adult mental and physical health outcomes. Many of the contributions have been alluded to in this discussion, but a few findings deserve additional attention. As noted above, the current research was a concerted effort to begin building a comprehensive model to explain how early family risk propagates
into adulthood to effect sleep in particular, and other health outcomes secondarily. The focus on sleep is timely, as research over the past decade has increasingly highlighted the importance of sleep for good health, especially cardiovascular health. While the model was quite well supported with regard to self-reported sleep, it was less so in terms of objectively measured aspects of sleep. Nevertheless, across two sample populations, a risky family environment was closely linked through direct and indirect pathways to adult psychosocial and sleep functioning. Specifically, in both studies, family risky was associated with lower social support and higher perceived stress, and these processes in turn largely mediated the relationship between risky family environment and negative affect. These findings are particularly important because they suggest areas for potential intervention. Consistent with cognitive-behavioural theory, negative affective states are difficult to change, however, cognitive and behavioural interventions targeted toward increasing social support and improving stress management may be more effective in indirectly improving negative affective states for these individuals. In turn, this improvement in negative mood may contribute to the subjective experience of restful sleep. Furthermore, this research supported previous work demonstrating a link between a risky family environment and both subjective and objective aspects of sleep, including increased motor activity, suggesting increased physical restlessness and wakefulness, and shorted sleep duration, once again through either direct or indirect pathways. Given that adequate sleep is seen as critical for health maintenance and disease prevention, these research findings once again highlight the import of primary prevention programs targeted toward curtailing family dysfunction in families with young children, and also highlight the need for continued research to elucidate pathways in the relationship.
While results of the current research did not find strong support for the hypothesis that poor sleep mediates the relationship between adult psychosocial functioning and physiological health risk factors, the results did highlight the association between social support and health risk indicators. Social support has been reliably linked to healthy cardiovascular functioning, including tonic BP, in cross-sectional, intervention, and laboratory studies (for review see Uchino et al., 1996). However, why limited social support is detrimental to health is less known. Many theorists focus on the stress-buffering hypothesis, whereby social support is thought to mitigate the effect of psychological distress, thereby impacting health. However, this research has been criticized for being largely cross-sectional and retrospective, and several studies have demonstrated main effects of social support on health (House, Landis, & Umberson, 1988). Main effects models posit that social support impacts health irrespective of stress, and that social integration promotes health behaviour, and provides stability, a sense of belonging, and security, which may impact neuroendocrine or immune functioning (Cohen, 2004). The current findings would be more in keeping with a main effects model, though specific mechanisms linking social support to BMI and BP were not considered.

In sum, the current research emphasizes the role of family environment in promoting health and well-being across the lifespan. Recall of several aspects of family dysfunction were quite prevalent in the two samples assessed over the course of this research, even in presumably fairly high-functioning university students. In turn, childhood family dysfunction was associated with psychosocial functioning and sleep processes in early adulthood, implying meaningful and deleterious consequences for overall quality of life and risk for future disease development. While further replication of research findings and continued
examination of casual pathways are required, the current findings do stress the need for primary prevention efforts and services focused on improving overall family functioning.
### Table 1

**Descriptive Statistics for Study 1 (MTURK) and Study 2 (University)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>MTURK</th>
<th>University</th>
</tr>
</thead>
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<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Age</td>
<td>29.55</td>
<td>7.20</td>
</tr>
<tr>
<td>% female participants</td>
<td>63.1</td>
<td></td>
</tr>
<tr>
<td>% smoke</td>
<td>17.1</td>
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</tr>
<tr>
<td>% use medication to sleep</td>
<td>7.7</td>
<td></td>
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<tr>
<td>% currently on medication</td>
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<tr>
<td>% psychotropic medication</td>
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<td></td>
</tr>
<tr>
<td>% who exercise</td>
<td>67.1</td>
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<td>Habitual exercise (mins/wk)</td>
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<td>136.44</td>
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<td>Risky Family Environment</td>
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<tr>
<td>Childhood SES</td>
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<td>12.12</td>
</tr>
<tr>
<td>Perceived Stress</td>
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</tr>
<tr>
<td>Social Support</td>
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</tr>
<tr>
<td>Anger</td>
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<td>8.07</td>
</tr>
<tr>
<td>Self-Reported Sleep Quality</td>
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<td>3.22</td>
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<tr>
<td>Body Mass Index</td>
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<tr>
<td>Total Sleep Time</td>
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<tr>
<td>% Wake</td>
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<td>% Mobile</td>
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<td>Number of Wake Bouts</td>
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<tr>
<td>Systolic BP</td>
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<tr>
<td>Diastolic BP</td>
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<td>7.21</td>
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</table>

*Note: Sample size for individual statistics may vary*
### Table 2
**Correlation Matrix: Study 1 MTURK Sample**

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<tr>
<th>Variable</th>
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<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Risky Family Environment</td>
<td>.22**</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
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<td>3. Childhood SES</td>
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<td>-.22**</td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>4. Perceived Stress</td>
<td>.41**</td>
<td>.30**</td>
<td>-.12*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>5. Social Support</td>
<td>-.19**</td>
<td>-.34**</td>
<td>.04</td>
<td>-.37**</td>
<td>1</td>
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<td>6. Depression</td>
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<td>.74**</td>
<td>-.49**</td>
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<td>7. Anxiety</td>
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<td>.35**</td>
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<td>.81**</td>
<td>-.43**</td>
<td>.80**</td>
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<td></td>
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<tr>
<td>8. Anger</td>
<td>.26**</td>
<td>.24**</td>
<td>-.04</td>
<td>.48**</td>
<td>-.19**</td>
<td>.45**</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>9. Body Mass Index</td>
<td>.09</td>
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<td>-.18*</td>
<td>.13*</td>
<td>-.15*</td>
<td>.18*</td>
<td>.14*</td>
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<td>-.01</td>
<td>-.08</td>
<td>-.10</td>
<td>-.03</td>
<td>.22**</td>
<td>1</td>
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<td></td>
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<td>11. Gender</td>
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<td>.01</td>
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<td>12. Smoking</td>
<td>.18*</td>
<td>-.01</td>
<td>.01</td>
<td>.12*</td>
<td>.05</td>
<td>.05</td>
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<td>13. Medication Use</td>
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<td>.02</td>
<td>.10</td>
<td>-.01</td>
<td>.00</td>
<td>.06</td>
<td>-.002</td>
<td>-.03</td>
<td>.16**</td>
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<td>14. Psychotropic Medication</td>
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<td>-.04</td>
<td>-.004</td>
<td>.14*</td>
<td>.05</td>
<td>.10</td>
<td>.16**</td>
<td>.15**</td>
<td>-.003</td>
<td>.05</td>
<td>-.04</td>
<td>-.10</td>
<td>.27**</td>
<td>1</td>
<td></td>
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<td>15. Exercise (yes/no)</td>
<td>-.15*</td>
<td>-.05</td>
<td>-.10</td>
<td>-.26**</td>
<td>.10</td>
<td>-.26**</td>
<td>-.28**</td>
<td>-.17**</td>
<td>-.12</td>
<td>-.07</td>
<td>-.03</td>
<td>-.12*</td>
<td>.02</td>
<td>.01</td>
<td>1</td>
<td></td>
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<tr>
<td>16. Habitual Exercise (min/week)</td>
<td>-.10</td>
<td>-.02</td>
<td>-.06</td>
<td>-.18**</td>
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<td>-.13*</td>
<td>.02</td>
<td>.11</td>
<td>.70**</td>
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</table>

|M| 6.35 | 23.46 | 44.21 | 18.39 | 24.67 | 14.90 | 43.38 | 28.45 | 25.97 | 29.55 | 134.55 |


* p < .05; ** p < .01; *** p < .001

*Note: Sample size for individual statistics may vary. Higher scores on the PSQI denote poorer sleep quality.*
Table 3

*Model Statistics for the Self-Reported Sleep Outcome Model: Study 1 MTURK Sample*

<table>
<thead>
<tr>
<th>Path Description</th>
<th>$\beta$</th>
<th>$B$</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>$R^2$</th>
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<tr>
<td>Childhood SES ➔</td>
<td>- .22*</td>
<td>-.16</td>
<td>.04</td>
<td>[-.24, -.08]</td>
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<tr>
<td>Risky Family Environment ➔ Social Support</td>
<td>- .34*</td>
<td>-.24</td>
<td>.04</td>
<td>[-.32, -.16]</td>
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<td>Risky Family Environment ➔ Perceived Stress</td>
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<td>.14</td>
<td>.05</td>
<td>[.04, .24]</td>
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</tr>
<tr>
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<td>.14</td>
<td>.04</td>
<td>[.06, .22]</td>
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<td>-.32</td>
<td>.06</td>
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<tr>
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<td>-.26</td>
<td>.06</td>
<td>[-.38, -.14]</td>
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<tr>
<td>Perceived Stress ➔ Negative Affect</td>
<td>.77*</td>
<td>1.21</td>
<td>.06</td>
<td>[1.09, 1.33]</td>
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<tr>
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<td>.48*</td>
<td>.15</td>
<td>.04</td>
<td>[.07, .23]</td>
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Latent Variable Indicators

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<th>$R^2$</th>
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<td>.89</td>
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<td>Anxiety</td>
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<td>-</td>
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<td>.04</td>
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$R^2$ for Predicted Variables

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<td>Perceived Stress</td>
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<td>Negative Affect</td>
<td>.79</td>
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<td>Sleep Quality</td>
<td>.22</td>
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*Note:* $\beta$ denotes standardized regression coefficients; $B$ denotes unstandardized regression coefficients.
Table 4

Indirect Paths for the Self-Reported Sleep Outcome Model: Study 1 MTURK Sample

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<td>.01</td>
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<td>-.07</td>
<td>.02</td>
<td>[-.11, -.03]</td>
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<td></td>
<td>➔ Negative Affect</td>
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<tr>
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<td>-.03</td>
<td>.01</td>
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<td>➔ Perceived Stress</td>
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<td>-.01</td>
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<td>Risky Family Environment</td>
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<td>.07</td>
<td>.02</td>
<td>[.03, .11]</td>
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<td>.08</td>
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<td></td>
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Note: β denotes standardized regression coefficients; B denotes unstandardized regression coefficients.
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<td>.13</td>
<td>.04</td>
<td>[.05, .21]</td>
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<td>Social Support ➔ Perceived Stress</td>
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<tr>
<td>Social Support ➔ Negative Affect</td>
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<td>-.30</td>
<td>.06</td>
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<tr>
<td>Social Support ➔ Negative Affect</td>
<td>-.16*</td>
<td>-.26</td>
<td>.06</td>
<td>[-.38, -.14]</td>
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<td>Perceived Stress ➔ Negative Affect</td>
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<td>.06</td>
<td>[1.06, 1.30]</td>
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<td>.14</td>
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<td>-.01</td>
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**Covariates**

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<td>[-.22, 2.66]</td>
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<td>.75</td>
<td>[-4.49, -1.55]</td>
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<td>.77</td>
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<td>Exercise ➔ Sleep Quality</td>
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<td>.01</td>
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<td>[-.75, .77]</td>
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<td>Smoking ➔ Childhood SES</td>
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<td>.93</td>
<td>[-.87, 2.77]</td>
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<td>Smoking ➔ Perceived Stress</td>
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<td>1.92</td>
<td>.91</td>
<td>[.14, 3.7]</td>
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Table 5 cont.

*Self-Reported Sleep Outcome Model Adjusted for Covariates: Study 1 MTURK Sample*

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<td>1.18</td>
<td>.45</td>
<td>[.03, 2.06]</td>
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</table>

**Latent Variable Indicators**

| Depression              | .85*  | .88  | .05       | [.78, .98]   | .73   |
| Anxiety                 | .93*  | -    | -         | -            | .87   |
| Anger                   | .55*  | .42  | .04       | [.34, .50]   | .31   |

$R^2$ for Predicted Variables

<table>
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<td>Perceived Stress</td>
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<td>Negative Affect</td>
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*Note: β denotes standardized regression coefficients; B denotes unstandardized regression coefficients.*
<table>
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<th>df</th>
<th>$\chi^2$ Δ</th>
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<td>Factor Loadings, Regression Coefficients, and Observed Variable Residual Variances</td>
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<td>.04*</td>
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Table 7

Correlations Matrix for Model Variables: Study 2 University Sample

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<td>-.07</td>
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<td>-.08</td>
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<td>-.13</td>
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<td>-.06</td>
<td>.43**</td>
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<td>.09</td>
<td>.17**</td>
<td>.15</td>
<td>.74**</td>
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</table>

|M | 5.51 | 21.25 | 50.51 | 17.77 | 23.61 | 14.12 | 42.01 | 28.16 | 22.73 | 391.5 | 7 | 9.76 | 6.41 | 107.49 | 71.02 |
|SD| 2.53 | 7.53 | 11.00 | 5.71 | 5.80 | 9.13 | 9.32 | 6.41 | 3.82 | 54.28 | 3.86 | .03 | 2.50 | 10.63 | 8.21 |

*p < .05; **p < .01; ***p < .001

Note: Unstandardized number of wake bouts (M = 40.63, SD = 13.10). Sample size for individual statistics may vary. Higher scores on the PSQI denote poorer sleep quality.
# Table 8

Correlations between Covariates and Outcome Variables: Study 2 University Sample

<table>
<thead>
<tr>
<th>Variable</th>
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<tbody>
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<td>3. Total Sleep Time</td>
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<td>4. % Wake</td>
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*p < .05; **p < .01; ***p < .001

Note: Sample size for individual statistics may vary. Higher scores on the PSQI denote poorer sleep quality.
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*p < .05; **p < .01; ***p < .001

Note: Sample size for individual statistics may vary
Table 10

Model Statistics for the Self-Reported Sleep Outcome Model: Study 2 University Sample

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Latent Variable Indicators

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R² for Predicted Variables

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Note: β denotes standardized regression coefficients; B denotes unstandardized regression coefficients.
Table 11

*Indirect Paths for the Self-Reported Sleep Outcome Model: Study 2 University Sample*

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*Note:* \( \beta \) denotes standardized regression coefficients; \( B \) denotes unstandardized regression coefficients.
Table 12

_Model Statistics for the Latent Sleep Activity Outcome Model: Study 2 University Sample_

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_Latent Variable Indicators_

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<td>.88*</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.77</td>
</tr>
<tr>
<td>% Mobile</td>
<td>.83*</td>
<td>.61</td>
<td>.10</td>
<td>[.41, .81]</td>
<td>.69</td>
</tr>
<tr>
<td>Standardized Number of Wake Bouts</td>
<td>.50*</td>
<td>.004</td>
<td>.001</td>
<td>[.002, .006]</td>
<td>.25</td>
</tr>
</tbody>
</table>

_R² for Predicted Variables_

<table>
<thead>
<tr>
<th>Variable</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support</td>
<td>.08</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>.15</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.70</td>
</tr>
<tr>
<td>Sleep Activity</td>
<td>.05</td>
</tr>
</tbody>
</table>

>Note: β denotes standardized regression coefficients; B denotes unstandardized regression coefficients.
Table 13

*Indirect Paths for the Latent Sleep Activity Outcome Model: Study 2 University Sample*

<table>
<thead>
<tr>
<th>Path</th>
<th>β</th>
<th>B</th>
<th>Robust SE</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Full Mediation Paths</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social Support → Sleep Activity</td>
<td>.04</td>
<td>.02</td>
<td>.02</td>
<td>[-.02, .06]</td>
</tr>
<tr>
<td><strong>Partial Mediation Paths</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perceived Stress → Sleep Activity</td>
<td>-.15</td>
<td>-.09</td>
<td>.09</td>
<td>[-.27, .09]</td>
</tr>
<tr>
<td>Risky Family Environment → Sleep Activity</td>
<td>-.08</td>
<td>.04</td>
<td>.02</td>
<td>[.00, .08]</td>
</tr>
<tr>
<td>Risky Family Environment → Perceived Stress</td>
<td>.03</td>
<td>.02</td>
<td>.02</td>
<td>[-.02, .02]</td>
</tr>
<tr>
<td>Risky Family Environment → Negative Affect</td>
<td>.33*</td>
<td>.38</td>
<td>.08</td>
<td>[.22, .54]</td>
</tr>
<tr>
<td>Social Support → Negative Affect</td>
<td>-.08</td>
<td>-.11</td>
<td>.08</td>
<td>[-.27, .05]</td>
</tr>
</tbody>
</table>

*Note: β denotes standardized regression coefficients; B denotes unstandardized regression coefficients.*
Table 14

Model Statistics for the Total Sleep Time Outcome Model: Study 2 University Sample

<table>
<thead>
<tr>
<th>Path Description</th>
<th>β</th>
<th>B</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risky Family Environment ▸ Social Support</td>
<td>-.29*</td>
<td>-.22</td>
<td>.07</td>
<td>[-.36, -.08]</td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment ▸ Perceived Stress</td>
<td>.35*</td>
<td>.27</td>
<td>.06</td>
<td>[.15, .39]</td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment ▸ Negative Affect</td>
<td>.06</td>
<td>.07</td>
<td>.07</td>
<td>[-.07, .21]</td>
<td></td>
</tr>
<tr>
<td>Social Support ▸ Perceived Stress</td>
<td>-.10</td>
<td>-.10</td>
<td>.06</td>
<td>[-.22, .02]</td>
<td></td>
</tr>
<tr>
<td>Social Support ▸ Negative Affect</td>
<td>-.11*</td>
<td>-.17</td>
<td>.08</td>
<td>[-.33, -.01]</td>
<td></td>
</tr>
<tr>
<td>Perceived Stress ▸ Negative Affect</td>
<td>.78*</td>
<td>1.20</td>
<td>.09</td>
<td>[1.02, 1.38]</td>
<td></td>
</tr>
<tr>
<td>Perceived Stress ▸ Total Sleep Time</td>
<td>-.15</td>
<td>-1.42</td>
<td>1.33</td>
<td>[-4.03, 1.19]</td>
<td></td>
</tr>
<tr>
<td>Negative Affect ▸ Total Sleep Time</td>
<td>.05</td>
<td>.32</td>
<td>.96</td>
<td>[-1.56, 2.2]</td>
<td></td>
</tr>
<tr>
<td>Social Support ▸ Total Sleep Time</td>
<td>.22*</td>
<td>2.03</td>
<td>.72</td>
<td>[.62, 3.44]</td>
<td></td>
</tr>
</tbody>
</table>

Latent Variable Indicators

<table>
<thead>
<tr>
<th></th>
<th>β</th>
<th>B</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>.83*</td>
<td>.86</td>
<td>.07</td>
<td>[.72, 1.00]</td>
<td>.68</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.94*</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.89</td>
</tr>
<tr>
<td>Anger</td>
<td>.40*</td>
<td>.29</td>
<td>.06</td>
<td>[.23, .35]</td>
<td>.16</td>
</tr>
</tbody>
</table>

R² for Predicted Variables

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support</td>
<td>.08</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>.15</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.70</td>
</tr>
<tr>
<td>Total Sleep Time</td>
<td>.07</td>
</tr>
</tbody>
</table>

Note: β denotes standardized regression coefficients; B denotes unstandardized regression coefficients.
Table 15

Indirect Paths for the Total Sleep Time Outcome Model: Study 2 University Sample

<table>
<thead>
<tr>
<th>Path</th>
<th>β</th>
<th>B</th>
<th>Robust SE</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full Mediation Paths</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment → Total Sleep Time</td>
<td>-.10*</td>
<td>.71</td>
<td>.30</td>
<td>[.12, .76]</td>
</tr>
<tr>
<td>Partial Mediation Paths</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social Support → Total Sleep Time</td>
<td>.01</td>
<td>.04</td>
<td>.19</td>
<td>[-.33, .37]</td>
</tr>
<tr>
<td>Perceived Stress → Total Sleep Time</td>
<td>.04</td>
<td>.38</td>
<td>1.16</td>
<td>[-1.89, 2.65]</td>
</tr>
<tr>
<td>Risky Family Environment → Perceived Stress</td>
<td>.03</td>
<td>.02</td>
<td>.02</td>
<td>[-.02, .02]</td>
</tr>
<tr>
<td>Risky Family Environment → Negative Affect</td>
<td>.33*</td>
<td>.38</td>
<td>.08</td>
<td>[.22, .54]</td>
</tr>
<tr>
<td>Social Support → Negative Affect</td>
<td>-.08</td>
<td>-.11</td>
<td>.08</td>
<td>[-.27, .05]</td>
</tr>
</tbody>
</table>

Note: β denotes standardized regression coefficients; B denotes unstandardized regression coefficients.
Table 16  
_Model Statistics for the BMI Outcome Model: Study 2 University Sample_

<table>
<thead>
<tr>
<th>Path Description</th>
<th>( \beta )</th>
<th>( B )</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risky Family Environment ➔ Social Support</td>
<td>-.29*</td>
<td>-.22</td>
<td>.07</td>
<td>[-.36, -.08]</td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment ➔ Perceived Stress</td>
<td>.35*</td>
<td>.27</td>
<td>.06</td>
<td>[.15, .39]</td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment ➔ Negative Affect</td>
<td>.06</td>
<td>.07</td>
<td>.07</td>
<td>[-.07, .21]</td>
<td></td>
</tr>
<tr>
<td>Social Support ➔ Perceived Stress</td>
<td>-.10</td>
<td>-.10</td>
<td>.06</td>
<td>[-.22, .02]</td>
<td></td>
</tr>
<tr>
<td>Social Support ➔ Negative Affect</td>
<td>-.11*</td>
<td>-.17</td>
<td>.08</td>
<td>[-.33, -.01]</td>
<td></td>
</tr>
<tr>
<td>Perceived Stress ➔ Negative Affect</td>
<td>.78*</td>
<td>1.20</td>
<td>.09</td>
<td>[1.02, 1.38]</td>
<td></td>
</tr>
<tr>
<td>Perceived Stress ➔ BMI</td>
<td>.41*</td>
<td>.27</td>
<td>.11</td>
<td>[.05, .49]</td>
<td></td>
</tr>
<tr>
<td>Negative Affect ➔ BMI</td>
<td>-.49*</td>
<td>-.22</td>
<td>.08</td>
<td>[-.38, -.06]</td>
<td></td>
</tr>
<tr>
<td>Social Support ➔ BMI</td>
<td>-.31*</td>
<td>-.20</td>
<td>.07</td>
<td>[-.34, -.06]</td>
<td></td>
</tr>
</tbody>
</table>

**Latent Variable Indicators**

<table>
<thead>
<tr>
<th></th>
<th>( \beta )</th>
<th>( B )</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>.83*</td>
<td>.86</td>
<td>.07</td>
<td>[.72, 1.00]</td>
<td>.68</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.94*</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.88</td>
</tr>
<tr>
<td>Anger</td>
<td>.40*</td>
<td>.29</td>
<td>.06</td>
<td>[.23, .35]</td>
<td>.16</td>
</tr>
</tbody>
</table>

**R^2 for Predicted Variables**

<table>
<thead>
<tr>
<th></th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support</td>
<td>.08</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>.15</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.71</td>
</tr>
<tr>
<td>BMI</td>
<td>.13</td>
</tr>
</tbody>
</table>

*Note: \( \beta \) denotes standardized regression coefficients; \( B \) denotes unstandardized regression coefficients.*
Table 17

*Indirect Paths for the BMI Outcome Model: Study 2 University Sample*

<table>
<thead>
<tr>
<th>Partial Mediation Paths</th>
<th>$\beta$</th>
<th>$B$</th>
<th>Robust SE</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support $\rightarrow$ BMI</td>
<td>.06</td>
<td>.04</td>
<td>.02</td>
<td>[.00, .08]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ BMI</td>
<td>.05</td>
<td>.03</td>
<td>.03</td>
<td>[-.03, .09]</td>
</tr>
<tr>
<td>Perceived Stress $\rightarrow$ BMI</td>
<td>-.39*</td>
<td>-.26</td>
<td>.10</td>
<td>[-.46, -.06]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ Perceived Stress</td>
<td>.03</td>
<td>.02</td>
<td>.02</td>
<td>[-.02, .02]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ Negative Affect</td>
<td>.33*</td>
<td>.38</td>
<td>.08</td>
<td>[.22, .54]</td>
</tr>
<tr>
<td>Social Support $\rightarrow$ Negative Affect</td>
<td>-.08</td>
<td>-.11</td>
<td>.08</td>
<td>[-.27, .05]</td>
</tr>
</tbody>
</table>

*Note: $\beta$ denotes standardized regression coefficients; B denotes unstandardized regression coefficients.*
### Table 18

**Model Statistics for the SBP Outcome Model: Study 2 University Sample**

<table>
<thead>
<tr>
<th>Path Description</th>
<th>( \beta )</th>
<th>( B )</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risky Family Environment → Social Support</td>
<td>-.29*</td>
<td>-.22</td>
<td>.07</td>
<td>[-.36, -.08]</td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment → Perceived Stress</td>
<td>.35*</td>
<td>.27</td>
<td>.06</td>
<td>[.15, .39]</td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment → Negative Affect</td>
<td>.06</td>
<td>.07</td>
<td>.07</td>
<td>[-.07, .21]</td>
<td></td>
</tr>
<tr>
<td>Social Support → Perceived Stress</td>
<td>-.10</td>
<td>-.10</td>
<td>.06</td>
<td>[-.22, .02]</td>
<td></td>
</tr>
<tr>
<td>Social Support → Negative Affect</td>
<td>-.11*</td>
<td>-.17</td>
<td>.08</td>
<td>[-.33, -.01]</td>
<td></td>
</tr>
<tr>
<td>Perceived Stress → Negative Affect</td>
<td>.78*</td>
<td>1.20</td>
<td>.09</td>
<td>[1.02, 1.38]</td>
<td></td>
</tr>
<tr>
<td>Perceived Stress → SBP</td>
<td>-.05</td>
<td>-.09</td>
<td>.23</td>
<td>[-.45, .36]</td>
<td></td>
</tr>
<tr>
<td>Negative Affect → SBP</td>
<td>-.17</td>
<td>-.21</td>
<td>.16</td>
<td>[-.52, .24]</td>
<td></td>
</tr>
<tr>
<td>Social Support → SBP</td>
<td>-.29*</td>
<td>-.53</td>
<td>.14</td>
<td>[-.80, -.26]</td>
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</tr>
</tbody>
</table>

**Latent Variable Indicators**

<table>
<thead>
<tr>
<th></th>
<th>( \beta )</th>
<th>( B )</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>.82*</td>
<td>.86</td>
<td>.07</td>
<td>[.72, 1.00]</td>
<td>.68</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.94*</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.89</td>
</tr>
<tr>
<td>Anger</td>
<td>.40*</td>
<td>.29</td>
<td>.06</td>
<td>[.23, .35]</td>
<td>.16</td>
</tr>
</tbody>
</table>

**R\(^2\) for Predicted Variables**

<table>
<thead>
<tr>
<th></th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support</td>
<td>.08</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>.15</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.71</td>
</tr>
<tr>
<td>SBP</td>
<td>.10</td>
</tr>
</tbody>
</table>

*Note: \( \beta \) denotes standardized regression coefficients; \( B \) denotes unstandardized regression coefficients.*
Table 19

*Indirect Paths for the SBP Outcome Model: Study 2 University Sample*

<table>
<thead>
<tr>
<th>Partial Mediation Paths</th>
<th>$\beta$</th>
<th>$B$</th>
<th>Robust $SE$</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support $\rightarrow$ SBP</td>
<td>.04</td>
<td>.07</td>
<td>.04</td>
<td>[-0.01, .15]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ SBP</td>
<td>-0.001</td>
<td>-0.002</td>
<td>.06</td>
<td>[-0.12, .12]</td>
</tr>
<tr>
<td>Perceived Stress $\rightarrow$ SBP</td>
<td>-.13</td>
<td>-.25</td>
<td>.20</td>
<td>[-.64, .14]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ Perceived Stress</td>
<td>.03</td>
<td>.02</td>
<td>.02</td>
<td>[-.02, .02]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ Negative Affect</td>
<td>.33*</td>
<td>.38</td>
<td>.08</td>
<td>[.22, .54]</td>
</tr>
<tr>
<td>Social Support $\rightarrow$ Negative Affect</td>
<td>-.08</td>
<td>-.12</td>
<td>.08</td>
<td>[-.28, .04]</td>
</tr>
</tbody>
</table>

*Note:* $\beta$ denotes standardized regression coefficients; $B$ denotes unstandardized regression coefficients.
Table 20

**Model Statistics for the DBP Outcome Model: Study 2 University Sample**

<table>
<thead>
<tr>
<th>Path Description</th>
<th>( \beta )</th>
<th>( B )</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risky Family Environment ( \rightarrow ) Social Support</td>
<td>-.29*</td>
<td>-.22</td>
<td>.07</td>
<td>[-.36, -.08]</td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment ( \rightarrow ) Perceived Stress</td>
<td>.35*</td>
<td>.27</td>
<td>.06</td>
<td>[.15,.39]</td>
<td></td>
</tr>
<tr>
<td>Risky Family Environment ( \rightarrow ) Negative Affect</td>
<td>.06</td>
<td>.07</td>
<td>.07</td>
<td>[-.07,.21]</td>
<td></td>
</tr>
<tr>
<td>Social Support ( \rightarrow ) Perceived Stress</td>
<td>-.10</td>
<td>-.10</td>
<td>.06</td>
<td>[-.22,.02]</td>
<td></td>
</tr>
<tr>
<td>Social Support ( \rightarrow ) Negative Affect</td>
<td>-.11*</td>
<td>-.17</td>
<td>.08</td>
<td>[-.33,-.01]</td>
<td></td>
</tr>
<tr>
<td>Perceived Stress ( \rightarrow ) Negative Affect</td>
<td>.78*</td>
<td>1.20</td>
<td>.09</td>
<td>[1.02,1.38]</td>
<td></td>
</tr>
<tr>
<td>Perceived Stress ( \rightarrow ) DBP</td>
<td>-.001</td>
<td>-.001</td>
<td>.19</td>
<td>[-.37,.37]</td>
<td></td>
</tr>
<tr>
<td>Negative Affect ( \rightarrow ) DBP</td>
<td>-.15</td>
<td>-.14</td>
<td>.13</td>
<td>[-.39,.11]</td>
<td></td>
</tr>
<tr>
<td>Social Support ( \rightarrow ) DBP</td>
<td>-.18*</td>
<td>-.25</td>
<td>.11</td>
<td>[-.47,-.03]</td>
<td></td>
</tr>
</tbody>
</table>

**Latent Variable Indicators**

<table>
<thead>
<tr>
<th></th>
<th>( \beta )</th>
<th>( B )</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>.82*</td>
<td>.86</td>
<td>.07</td>
<td>[.72,1.00]</td>
<td>.68</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.94*</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.89</td>
</tr>
<tr>
<td>Anger</td>
<td>.40*</td>
<td>.29</td>
<td>.06</td>
<td>[.23,.35]</td>
<td>.16</td>
</tr>
</tbody>
</table>

**\( R^2 \) for Predicted Variables**

<table>
<thead>
<tr>
<th></th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support</td>
<td>.08</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>.15</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.71</td>
</tr>
<tr>
<td>DBP</td>
<td>.04</td>
</tr>
</tbody>
</table>

**Note:** \( \beta \) denotes standardized regression coefficients; \( B \) denotes unstandardized regression coefficients.
Table 21

*Indirect Paths for the DBP Outcome Model: Study 2 University Sample*

<table>
<thead>
<tr>
<th>Partial Mediation Paths</th>
<th>$\beta$</th>
<th>$B$</th>
<th>Robust $SE$</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support $\rightarrow$ DBP</td>
<td>.03</td>
<td>.04</td>
<td>.03</td>
<td>[-.02, .10]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ DBP</td>
<td>-.01</td>
<td>-.01</td>
<td>.04</td>
<td>[-.09, .07]</td>
</tr>
<tr>
<td>Perceived Stress $\rightarrow$ DBP</td>
<td>-.12</td>
<td>-.17</td>
<td>.15</td>
<td>[-.46, .12]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ Perceived Stress</td>
<td>.03</td>
<td>.02</td>
<td>.02</td>
<td>[-.02, .02]</td>
</tr>
<tr>
<td>Risky Family Environment $\rightarrow$ Negative Affect</td>
<td>.33*</td>
<td>.38</td>
<td>.08</td>
<td>[.22, .54]</td>
</tr>
<tr>
<td>Social Support $\rightarrow$ Negative Affect</td>
<td>-.08</td>
<td>-.12</td>
<td>.08</td>
<td>[-.28, .04]</td>
</tr>
</tbody>
</table>

*Note:* $\beta$ denotes standardized regression coefficients; $B$ denotes unstandardized regression coefficients.
Table 22

*Model Statistics for Self-Reported Sleep Quality as a Mediator between Negative Affect and BMI:
Study 2 University Sample*

<table>
<thead>
<tr>
<th>Path Description</th>
<th>( \beta )</th>
<th>( B )</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative Affect ➔ Sleep Quality</td>
<td>.59*</td>
<td>1.48</td>
<td>.14</td>
<td>[1.21, 1.75]</td>
<td></td>
</tr>
<tr>
<td>Negative Affect ➔ BMI</td>
<td>-.18*</td>
<td>-.54</td>
<td>.42</td>
<td>[-1.36, .28]</td>
<td></td>
</tr>
<tr>
<td>Sleep Quality ➔ BMI</td>
<td>-.14</td>
<td>.18</td>
<td>.16</td>
<td>[-.14, .50]</td>
<td></td>
</tr>
<tr>
<td><strong>Latent Variable Indicators</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>.86*</td>
<td>7.79</td>
<td>.82</td>
<td>[6.18, 9.40]</td>
<td>.73</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.91*</td>
<td>8.45</td>
<td>.62</td>
<td>[7.23, 9.67]</td>
<td>.83</td>
</tr>
<tr>
<td>Anger</td>
<td>.37*</td>
<td>2.36</td>
<td>.60</td>
<td>[1.18, 3.54]</td>
<td>.14</td>
</tr>
</tbody>
</table>

\( R^2 \) for Predicted Variables

<table>
<thead>
<tr>
<th></th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Quality</td>
<td>.35</td>
</tr>
<tr>
<td>BMI</td>
<td>.01</td>
</tr>
</tbody>
</table>

*Note: \( \beta \) denotes standardized regression coefficients; \( B \) denotes unstandardized regression coefficients.*
Table 23

Model Statistics for Total Sleep Time as a Mediator between Social Support and BMI: Study 2

University Sample

<table>
<thead>
<tr>
<th>Path Description</th>
<th>β</th>
<th>B</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support ➔ Sleep Time</td>
<td>.23*</td>
<td>2.17</td>
<td>.74</td>
<td>[.72, 3.62]</td>
<td></td>
</tr>
<tr>
<td>Social Support ➔ BMI</td>
<td>-.20*</td>
<td>-.13</td>
<td>.065</td>
<td>[-.26, -.003]</td>
<td></td>
</tr>
<tr>
<td>Sleep Time ➔ BMI</td>
<td>-.19*</td>
<td>.01</td>
<td>.005</td>
<td>[.0002, .02]</td>
<td></td>
</tr>
</tbody>
</table>

$R^2$ for Predicted Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Time</td>
<td>.05</td>
</tr>
<tr>
<td>BMI</td>
<td>.09</td>
</tr>
</tbody>
</table>

Note: $\beta$ denotes standardized regression coefficients; B denotes unstandardized regression coefficients.
Table 24

*Model Statistics for Total Sleep Time as a Mediator between Social Support and SBP: Study 2

*University Sample*

<table>
<thead>
<tr>
<th>Path Description</th>
<th>β</th>
<th>B</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support → Sleep Time</td>
<td>.23*</td>
<td>2.17</td>
<td>.74</td>
<td>[.72, 3.62]</td>
<td></td>
</tr>
<tr>
<td>Social Support → SBP</td>
<td>-.20*</td>
<td>-.42</td>
<td>.14</td>
<td>[-.69, -.15]</td>
<td></td>
</tr>
<tr>
<td>Sleep Time → SBP</td>
<td>-.12</td>
<td>.02</td>
<td>.02</td>
<td>[-.02, .06]</td>
<td></td>
</tr>
</tbody>
</table>

$R^2$ for Predicted Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Time</td>
<td>.05</td>
</tr>
<tr>
<td>SBP</td>
<td>.07</td>
</tr>
</tbody>
</table>

*Note:* $\beta$ denotes standardized regression coefficients; $B$ denotes unstandardized regression coefficients.
### Table 25

*Model Statistics for Total Sleep Time as a Mediator between Social Support and DBP: Study 2*

**University Sample**

<table>
<thead>
<tr>
<th>Path Description</th>
<th>β</th>
<th>B</th>
<th>Robust SE</th>
<th>95% CI</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Support → Sleep Time</td>
<td>.23*</td>
<td>2.17</td>
<td>.74</td>
<td>[.72, 3.62]</td>
<td></td>
</tr>
<tr>
<td>Social Support → DBP</td>
<td>-.13</td>
<td>-.19</td>
<td>.11</td>
<td>[-.41, .03]</td>
<td></td>
</tr>
<tr>
<td>Sleep Time → DBP</td>
<td>-.01</td>
<td>.002</td>
<td>.01</td>
<td>[-.02, .02]</td>
<td></td>
</tr>
</tbody>
</table>

\( R^2 \) for Predicted Variables

<table>
<thead>
<tr>
<th></th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Time</td>
<td>.05</td>
</tr>
<tr>
<td>DBP</td>
<td>.02</td>
</tr>
</tbody>
</table>

*Note:* \( \beta \) denotes standardized regression coefficients; \( B \) denotes unstandardized regression coefficients.
Table 2

Sample Comparisons for Study 1 (MTURK, n = 298) and Study 2 (University, n = 158) Samples

<table>
<thead>
<tr>
<th>Variable</th>
<th>MTRUK Mean</th>
<th>MTRUK Variance</th>
<th>University Mean</th>
<th>University Variance</th>
<th>Group Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>29.55</td>
<td>51.84</td>
<td>23.41</td>
<td>25.77</td>
<td>6.14** 26.07***</td>
</tr>
<tr>
<td>Risky Family Environment</td>
<td>23.46</td>
<td>85.35</td>
<td>21.25</td>
<td>56.67</td>
<td>2.21* 28.68**</td>
</tr>
<tr>
<td>Childhood SES</td>
<td>44.21</td>
<td>146.97</td>
<td>50.51</td>
<td>121.06</td>
<td>-6.3* 25.91*</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>18.39</td>
<td>45.78</td>
<td>17.77</td>
<td>32.59</td>
<td>0.62 13.19</td>
</tr>
<tr>
<td>Social Support</td>
<td>24.67</td>
<td>43.51</td>
<td>23.61</td>
<td>33.71</td>
<td>1.06 9.8*</td>
</tr>
<tr>
<td>Depression</td>
<td>14.90</td>
<td>119.39</td>
<td>14.12</td>
<td>83.29</td>
<td>0.78 36.1*</td>
</tr>
<tr>
<td>Anxiety</td>
<td>43.38</td>
<td>129.75</td>
<td>42.01</td>
<td>86.92</td>
<td>1.37 42.83*</td>
</tr>
<tr>
<td>Anger</td>
<td>28.45</td>
<td>65.06</td>
<td>28.16</td>
<td>42.08</td>
<td>0.29 22.98***</td>
</tr>
<tr>
<td>Self-Reported Sleep Quality</td>
<td>6.35</td>
<td>10.39</td>
<td>5.51</td>
<td>6.38</td>
<td>0.84** 4.01**</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>25.97</td>
<td>42.23</td>
<td>22.73</td>
<td>14.63</td>
<td>3.24** 27.6***</td>
</tr>
<tr>
<td>Body Mass Index (no outlier for MTURK sample)</td>
<td>25.78</td>
<td>32.31</td>
<td>22.73</td>
<td>14.63</td>
<td>3.05** 17.68***</td>
</tr>
</tbody>
</table>

*p < .05; **p < .01; ***p < .001

Note: Sample size for individual statistics may vary
Figure 1. Hypothesized risky family model.
Figure 2. Confirmatory factor analysis for negative affect: Study 1 MTURK sample. Note: Path coefficients are standardized; variance of the latent factor fixed to 1 to allow for identification.
Figure 3. Model predicting self-reported sleep quality: Study 1 MTURK sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 4. Model predicting self-reported sleep quality after adjusting for covariates: Study 1 MTURK sample. Note: For ease of presentation covariates are not shown (see Table 5 for covariate path coefficients); path coefficients are standardized; $f$ denotes factor loading is fixed to allow for identification.
Figure 5. Confirmatory factor analysis for negative affect: Study 2 university sample. Note: Path coefficients are standardized; variance of the latent factor fixed to 1 to allow for identification.
Figure 6. Confirmatory factor analysis for latent sleep activity: Study 2 university sample. Note: Path coefficients are standardized; variance of the latent factor fixed to 1 to allow for identification.
Figure 7. Initial model predicting self-reported sleep quality (PSQI): Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 8. Modified model predicting self-reported sleep quality (PSQI): Study 2 university sample. Note: Path coefficients are standardized; "f" denotes factor loading is fixed to allow for identification.
Figure 9. Initial model predicting latent sleep activity: Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 10. Modified model predicting latent sleep activity: Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 11. *Initial model predicting total sleep time: Study 2 university sample. Note: Path coefficients are standardized; * denotes factor loading is fixed to allow for identification.*
Figure 12. Modified model predicting total sleep time: Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 13. Initial model predicting BMI: Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 14. Modified model predicting BMI: Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 15. Initial model predicting SBP: Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 16. Modified model predicting SBP: Study 2 university sample. Note: Path coefficients are standardized; * denotes factor loading is fixed to allow for identification.
Figure 17. Initial model predicting DBP: Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 18. Modified model predicting DBP: Study 2 university sample. Note: Path coefficients are standardized; f denotes factor loading is fixed to allow for identification.
Figure 19. Sleep quality as a mediator between negative affect and BMI. Note: Path coefficients are standardized; variance of the latent factor fixed to 1 to allow for identification.
Figure 20. Total sleep time as a mediator between social support and BMI. Note: Path coefficients are standardized.
Figure 21. Total sleep time as a mediator between social support and SBP. Note: Path coefficients are standardized.
Figure 22. Total sleep time as a mediator between social support and DBP. Note: Path coefficients are standardized.
References


doi:10.1093/eurheartj/ehr007


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from the National Hospital Discharge Survey. *Circulation, 76*(2), 280–288. doi:10.1161/01.CIR.76.2.280


Appendices

Appendix A: Demographics Information Form

**Instructions:** Please provide answers to all of the questions indicated.

<table>
<thead>
<tr>
<th>Background</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age:</td>
</tr>
<tr>
<td>Date of birth: (mm/dd/yy)</td>
</tr>
<tr>
<td>Gender:</td>
</tr>
</tbody>
</table>

Which ethnicity do you identify best with? (Please use 1 check mark)

- Asian
- Caucasian
- Eastern-European
- Hispanic
- Middle-Eastern
- South Asian

Which religion do you identify best with? (Please use 1 check mark)

- Buddhism
- Christianity
- Hinduism
- Islam
- Judaism
- Sikhism
- Non-religious
- Other: (Please specify)

Marital Status: (Please use 1 check mark)

- Divorced
- Married
- Single (stable relationship)
- Single
Separated _______________________
Widowed _______________________
Employment Status: (Please use 1 check mark)
Full-time _______________________
Part-time _______________________
Unemployed _____________________
Your occupation: _______________________
How many hours, if any, do you work in a typical week? _______________________
Are you a student?
Yes ☐
No ☐
Your highest level of education (Please use 1 check mark)
Graduate professional training _______________________
Standard college or university graduate _______________________
Partial college training _______________________
High school graduate _______________________
Partial high school graduate _______________________
Junior high school (7th to 9th grade) _______________________
Less than 7 years of schooling _______________________
Your mother’s highest level of education (Please use 1 check mark)
Graduate professional training _______________________
Standard college or university graduate _______________________
Partial college training _______________________
High school graduate _______________________
Partial high school graduate _______________________
Junior high school (7th to 9th grade) _______________________
Less than 7 years of schooling _______________________

Your father's highest level of education
(Please use 1 check mark)

Graduate professional training
Standard college or university graduate
Partial college training
High school graduate
Partial high school graduate
Junior high school (7th to 9th grade)
Less than 7 years of schooling

Your mother's occupation:

Your father's occupation:

Please indicate whether you experience any of the following: (diagnosed by a physician)

- Insomnia
- Sleep Apnea
- Restless Legs Syndrome (RLS)
- Narcolepsy
- Night terror
- Sleep talking
- Tooth-grinding (bruxism)
- Other

If other, please specify:

Please indicate whether you suffer from a chronic disease (e.g., diabetes, cancer) or have been diagnosed with a psychiatric disorder (e.g., anxiety, depression, posttraumatic stress disorder):
### Sleep Patterns

**What best describes you:**
- [ ] Early bird
- [ ] Night owl

**On a usual weeknight, how many hours of sleep do you get per night?**
- [ ] <4
- [ ] 4-6
- [ ] 6-8
- [ ] >8

**On weeknights what time do you:**
- [ ] Go to bed: ____________
- [ ] Awake in the morning: ____________

**On a usual weekend evening, how many hours of sleep do you get per night?**
- [ ] <4
- [ ] 4-6
- [ ] 6-8
- [ ] >8

**On weekends what time do you:**
- [ ] Go to bed: ____________
- [ ] Awake in the morning: ____________

**How many hours per night do you think is optimal?**

**What do you do to facilitate your sleep?**
(i.e., yoga, exercise, drinking tea, meditation, medication, etc.)

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td></td>
</tr>
</tbody>
</table>
Do you use recreational drugs or prescription medication to help you fall asleep? (i.e. marijuana, ativan, etc.)

Yes ☐
No ☐

If yes, please specify: ____________________________________________

Health & Wellness

Are you currently on any medications? (Please indicate all medications, including contraceptives, non-prescription [i.e. cough or allergy medicine], etc.)

1. 5.
2. 6.
3. 7.
4. 8.

Do you exercise?

If yes, please indicate:

How many times per week you exercise: _____________________________

Duration: ____________________________ (minutes)

On average, how many cigarettes do you smoke per day?
0 4) 11-20
1-5 5) 21-29
3) 6-10 6) Over 30

Please indicate your height and weight:

Height: _______
Weight: _______
Appendix B: Sleep Diary

Instructions:

Please fill out this sleep diary in the morning as soon as you awake!

Subject Number:

Time you got up:
Date: __________________________
Time:__________________________

Did this represent a “typical” day for you?
Yes □
No □

Sleep Details

Last night I got into bed at: _____:____ PM AM
I actually tried to go to sleep at: _____:____ PM AM
I think it took me about ______ Minutes to fall asleep
This morning, I finally woke at _____:____ AM PM

I actually got out of bed to start my day at _____:____ AM PM

Last night after I finally fell asleep, I woke up this many times during the night (circle one number)
0 1 2 3 4 5 or more.

The longest time I spent awake at one time was ______ minutes.
Altogether, these awakenings lasted a total of ______ minutes.

Of these awakenings (circle one number for each line):
  I woke to use the bathroom this many times 0 1 2 3 4 5 or more
  I woke due to noises, child, or bedpartner this many times 0 1 2 3 4 5 or more
  I woke due to discomfort or a physical complaint this many times 0 1 2 3 4 5 or more
  I woke due to another or no special reason this many times 0 1 2 3 4 5 or more
(Please describe the other reason if indicated)

Did you nap at any point during the previous day? *If yes, please indicate,*

Number of naps: ____________

Time of day when naps taken: *(i.e., 1pm-2pm in the afternoon)*

1. Start Time: _______ End Time: _______ AM PM
2. Start Time: _______ End Time: _______ AM PM
3. Start Time: _______ End Time: _______ AM PM

*Lifestyle Factors*

Did you consume any *caffeinated* beverages during the previous day? *(i.e., coffee, tea (non-herbal), hot-chocolate, Coca-cola, etc.)*

Yes □
No □

Please list the type of beverage(s), amount and the time consumed:

Beverage Type 1: _______________
Amount: ______________________
Time: ________________________

Beverage Type 2: _______________
Amount: ______________________
Time: ________________________

Beverage Type 3: _______________
Amount: ______________________
Time: ________________________
Did you consume any **alcoholic** beverages during the **previous day**? (i.e., wine, spirits, beer, etc.)

Yes ☐
No ☐

Please list the type of beverage(s), amount (i.e., can, glass, etc.) and the time consumed:

Beverage Type 1:________________
Amount: __________________
Time:__________________________

Beverage Type 2:______________
Amount: __________________
Time:__________________________

Beverage Type 3:________________
Amount: _______________________
Time:__________________________

Did you consume any **prescription or non-prescription** drugs the **previous evening**? (i.e., ativan, marijuana, etc.)

Yes ☐
No ☐

Please list the type of drugs and the time consumed:

Drug Type 1: ________________
Dose: _____________
Time: __________________________

Drug Type 2: ________________
Dose: _____________
Time: __________________________

Drug Type 3: ________________
Dose: _____________
Time: __________________________
Did you do any **exercise** during the previous day?

Yes □
No □

*If yes, please indicate:*

- **Type of activity:**
  (i.e., jogging, yoga, aerobics, weight lifting, etc.)

- **Duration (minutes):**

- **Intensity:**
  (i.e., walking=low, sprinting=high)

  - Low □
  - Moderate □
  - High □

- **Level of general daily physical activity:**
  (i.e., sitting all day=low, physical labor job=high)

  - Low □
  - Moderate □
  - High □

- **Time of day:**
  (i.e., 1pm-2pm in the afternoon)
Did you use any **electronic entertainment** in the previous day (e.g., videogames, TV, Computer)?

Yes ☐

No ☐

**Video games?**

Yes ☐

No ☐

Duration *(minutes):* ______________

Time of day:
*please report all the times played:*

1. Start Time: End Time: AM PM

2. Start Time: End Time: AM PM

3: Start Time: End Time: AM PM

**Watch any TV or movies?**

Yes ☐

No ☐

Duration *(minutes):* ______________

Time of day:
*please report all the times watched*

1. Start Time: End Time: AM PM

2. Start Time: End Time: AM PM

3: Start Time: End Time: AM PM

**Go on the computer?**

Yes ☐

No ☐

Duration *(minutes):* ______________

Time of day *(please report all the times used)*
1. Start Time:          End Time:             AM PM
2. Start Time:          End Time:             AM PM
3: Start Time:          End Time:             AM PM

Emotional Well-Being

Did you experience any **stressful events** during the **previous day**?

Yes ☐
No ☐

(i.e., argument with friend or family member, exam, date, etc.)

If yes, please indicate,

What was the event?
What time did this occur?

If Female:  Are you menstruating?

Yes ☐
No ☐

Please provide any comments below regarding concerns you may have regarding your sleep patterns:

____________________________________________________________________________________________________________________________

________________________________________

________________________________________

________________________________________

________________________________________