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PATHWAYS LINKING SLEEP TIMING TO OBESITY IN MIDLIFE WOMEN

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science  
at Virginia Commonwealth University.

By: DANA R. SCHREIBER  
Bachelor of Science, University of Pittsburgh, May 2011

Director: Natalie D. Dautovich, Ph.D.  
Assistant Professor of Psychology  
Department of Psychology

Virginia Commonwealth University  
Richmond, Virginia  
December, 2016

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## **Abstract**

### **PATHWAYS LINKING SLEEP TIMING TO OBESITY IN MIDLIFE WOMEN**

By: Dana R. Schreiber, B.S.

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

Virginia Commonwealth University, 2016

Director: Natalie D. Dautovich, Ph.D.,  
Assistant Professor of Psychology  
Department of Psychology

Midlife women are vulnerable to developing obesity. Both sleep timing and negative emotion are risk factors, yet limited research has explored their role on weight outcomes in this population. The current investigation explored the association of sleep timing (i.e., mean sleep time, sleep time variability) and obesity (i.e., BMI, waist circumference) in midlife women, considering emotional pathways (i.e., depressive symptoms, anger) that might mediate this association. PROCESS parallel mediation models assessed direct and indirect pathways. In follow-up analyses, sleep duration was explored as an indirect pathway linking sleep timing to obesity. Results demonstrated that sleep timing does not directly predict obesity. Emotion was also not a significant indirect pathway. Conversely, sleep duration emerged as an indirect link in the sleep timing and obesity association. Future work is needed to further disentangle the impact

of sleep on weight in midlife women using prospective, well-controlled studies, implementing daily assessments of sleep behavior.

## Pathways Linking Sleep Timing to Obesity in Midlife Women

Women in midlife are at risk for the development of obesity due to the many physiological and psychological changes that occur during this time period. Although hormonal (Shi & Clegg, 2009) and lifestyle factors such as diet and physical activity (Campbell & Samaras, 2000) have been explored as mechanisms leading to weight gain in this population, sleep, and more recently, sleep timing, has come under focus as a novel mechanism linked to obesity in midlife women (Taylor et al., 2016). Women in midlife commonly report disturbed sleep patterns, and poor sleep in this population is linked to weight gain and changes in adiposity (Choi et al., 2011). These changes subsequently increase obesity risk (Hall et al., 2012; Jennings et al., 2007; Taylor et al., 2016). In addition to affecting weight gain, poor sleep is linked with depressed mood and anger in midlife women (Meliska et al., 2011; Thomas & Donnellan, 1991). These psychological variables, in turn, are also linked to obesity in this population (Clum, Rice, Broussard, Johnson, & Webber, 2014; Crawford, Casey, Avis, & McKinley, 2011). Although the associations between sleep, emotional factors, and obesity have been separately identified, the concurrent links between these three variables remains unclear, especially in midlife women.

The current study investigated the inter-connectedness among sleep, emotion, and weight in midlife women by examining the association between sleep timing and obesity in this population and, additionally, examining the role of emotions in mediating this association. The role of sleep timing in predicting weight outcomes is crucial to explore in this population, given that midlife women have issues with sleep initiation and sleep regulation (Owens & Matthews, 1998; Polo-Kantola, 2011; Shneerson, 2005). Despite the relevance of sleep timing for weight

outcomes in midlife women, few studies using controlled conditions have examined the role of sleep timing in predicting emotional factors and weight outcomes in midlife women. The limited work in this domain has also resulted in inconsistent and discrepant findings. Therefore, the current study aimed to contribute to the understanding of pathways linking to obesity in midlife women through a comprehensive consideration of sleep timing (i.e., mean sleep time, sleep time variability), emotion (i.e., depressive symptoms, anger), and obesity (i.e., BMI and waist circumference) using a combination of well-validated, objective, and daily measures of behavior and health outcomes. The ultimate aim of this work is to add to the limited existing knowledge base to inform sleep timing recommendations and clinical judgment regarding emotional health for midlife women at risk for negative weight outcomes.

### **Prevalence and Negative Outcomes Associated with Obesity**

Obesity is a major public health issue in the United States. Thirty-six percent of adults in the United States are classified as obese, and this condition has serious implications for both physical and psychological health (Flegal, Kit, Orpana, & Graubard, 2013). Obesity, defined as having a body mass index (BMI) of 30 or above, is associated with adverse physical health outcomes including early mortality as well as serious health conditions such as metabolic syndrome (Flegal et al., 2013; Grundy et al., 2005; World Health Organization [WHO], 2000). Metabolic syndrome is a composite group of risk factors including abdominal adiposity, impaired glucose tolerance, and high blood pressure which increases risk of serious health conditions such as type 2 diabetes, stroke, cardiovascular disease, and all-cause mortality (Grundy et al., 2005; Mottillo et al., 2010; Xi, He, Zhang, Xue, & Zhou, 2014). In addition to physiological health outcomes, obesity has negative psychological implications for emotional well-being, quality of life, and mental health outcomes (Garipey, Nitka, & Schmitz, 2010;

Luppino et al., 2010; Mather, Cox, Enns, & Sareen, 2009; Vallis, 2016). Obesity is associated with anxiety and depression, and individuals who are overweight or obese have an increased likelihood of developing depressive symptoms (Garipey, et al., 2010; Luppino et al., 2010; Mather, et al., 2009), giving rise to a unique illness subtype referred to as “metabolic-mood syndrome” (Mansur, Brietzke, & McIntyre, 2015).

Although obesity is commonly linked to poor physical and psychological health problems, there are limitations of using BMI as an exclusive measurement of obesity. BMI does not differentiate between lean and fat tissue and cannot distinguish placement of adiposity (Gurruci, Hartrivanti, Hautvast, & Deurenberg, 1999; Wells, 2000). As central adiposity, also known as central obesity, has been found to influence physical and psychological health outcomes, often independent of BMI, many researchers are investigating the impact of central obesity on health outcomes in addition to BMI (Ashwell & Hsieh, 2005; McCarthy & Ashwell, 2006). Central obesity, defined as excessive abdominal fat around the stomach and abdomen area, and quantified as having a waist circumference  $> 88$  cm, is associated with several negative physical and mental health outcomes such as the development of metabolic syndrome, cardiovascular disease, type II diabetes, and depression (Grundy et al., 2006; Meisinger, Doring, Thorand, Heier, & Lowel, 2006; Walton et al., 1995; Xu, Anderson & Lurie-Beck, 2011).

**Obesity in midlife women.** Although rates of obesity and central obesity are high for both genders, women experience significantly higher rates of obesity than men. Specifically, recent data from the Centers for Disease Control and Prevention indicate that 38% of women are obese compared with 34% of men (Ogden, Carroll, Fryar, & Flegal, 2015). One segment of the population that is particularly vulnerable to obesity is women in midlife. In 2014, 42% of midlife women (between the ages of 40-60) were reported as obese as indicated by having a BMI equal

to or above 30 (Ogden et al., 2015). Women in midlife go through many physiological and psychological changes, with one significant change being the menopausal transition. The menopausal transition is associated with significant weight gain, with women gaining 2 to 2.5 kg on average, over three years' time, and experiencing significant changes in body fat distribution (Ogden et al., 2015). As such, women in the menopausal transition have increased total body fat and visceral fat (Heymsfield et al., 1994; Ho Wu, Chan, & Sham, 2010), which are risk factors for the progression to obesity and diseases such as metabolic syndrome (Su, Lin, Chu, Huang, & Tsao, 2015). In addition to menopause as a risk factor for weight gain, other factors such as financial strain, quality of social support (e.g., marital status), and stress due to multiple role management (e.g., work, social, familial obligations) are specifically related to weight outcomes in midlife women (Lachman, 2004; Umberson, Liu, & Powers, 2009).

Although many factors have been proposed to account for the increase of weight in midlife women, the etiology of this weight gain is not completely understood. Identified physiological mechanisms include changes in hormonal levels, changes in resting metabolic rate, changes in body fat distribution, and disruption of circadian rhythms which can impact food intake (Fonkin et al., 2010; Shi & Clegg, 2009; Simkin-Silverman & Wing, 2000). Lifestyle factors such as smoking and sedentary behavior are also linked to weight gain in midlife (Campbell & Samaras, 2000; Heymsfield et al., 1994; Wing, Matthews, Kuller, Meilahn, & Plantinga, 1991a) with low levels of physical activity being a consistent behavioral factor associated with weight gain in midlife women (Wing, Matthews, Kuller, Meilahn, & Plantinga, 1991b). Other contributing factors in this population include socio-demographic characteristics such as low socioeconomic status and lower levels of education, and psychological factors such as emotion regulation, coping strategies, and mood (Campbell & Samaras, 2000; Crawford, et

al., 2000; Lahman, Lissner, Gullberg, & Berglund, 2000; Pan et al. 2012; Serlachius, Hamer, & Wardle, 2007). In addition, one promising, yet understudied, link to obesity in midlife women is sleep (Appelhans et al., 2013; Hall et al., 2012).

### **Sleep as a Mechanism Leading to Obesity in Midlife Women**

Sleep is a universal behavior that is linked to obesity and central obesity, and that disproportionately affects women in midlife. Women are more likely than men to develop sleep problems throughout the lifespan and menopause is a particularly vulnerable time for experiencing sleep problems (Polo-Kantola, 2011). Overall, women are 1.4 times more times likely than men to report sleep disturbances (Krystal, 2003), with midlife and menopause having specific biological and psychological implications for sleep disruption (Kravitz et al., 2003). Common causes of sleep disturbance in this population include vasomotor symptoms, changes in hormone levels, mood, perceived health, and stress (Ameratunga, Goldin, & Hickey, 2012; Kravitz et al., 2008; Woods & Mitchell, 2010). Between 33-51% of women report sleep problems during this time, and women in the menopausal transition often rate sleep concerns as one of the most bothersome symptoms associated with menopause (Ford, Sowers, Crutchfield, Wilson, & Jannausch, 2005). The high prevalence of sleep issues in this population resulted in the recognition of sleep as a core symptom of menopause in the 2005 National Institute of Health's (NIH) State-of-the-Science Conference panel report on menopause-related symptoms (National Institute of Health, 2005). Poor sleep leads to adverse physiological and psychological outcomes in midlife including poor mood, reduced quality of life, and disability (Arigo, Kloss, Knjele, & Gilrain, 2007; Strauss, 2011). Furthermore, poor sleep has major health implications, including increased risk for obesity and abdominal adiposity (Hall et al., 2012).

Given that poor sleep can lead to poor weight outcomes including obesity and central obesity in midlife women, many researchers have investigated this association. As sleep is a multifaceted construct, it encompasses many dimensions and can be measured in a variety of ways. Although sleep dimensions such as duration have received the majority of attention in regard to weight outcomes, additional dimensions including quality, efficiency, and timing have also been linked to obesity, body mass index, and weight-related outcomes (Buysse, 2014).

**Sleep Duration.** Sleep duration is defined as the total amount of sleep obtained per 24 hours (Buysse, 2014) and has consistently been linked to higher rates of obesity in both child and adult samples (Vgontzas et al., 2014). In particular, both adults and children with short sleep duration (i.e., less than seven hours per night) have 55-89% higher odds of developing obesity than those who sleep greater than seven hours per night (Appelhans et al., 2013; Gangwisch, Malaspina, Boden-Albala, & Heymsfield, 2005). The relation between sleep duration and obesity has also been described as a U-shaped curve with lowest risk at about seven to eight hours of sleep per night with increased risk of higher weight for both shorter and longer sleepers (Youngstedt & Kripke, 2004). This nonlinear distribution highlights the role of both sleep restriction and sleep extension as a risk factors for obesity (Knutson, Spiegel, Penev, & Van Cauter, 2007; Taheri, 2007; Youngstedt & Kripke, 2004). Furthermore, the relation between sleep duration and weight has been specifically examined in midlife women. Short sleep duration has been linked to metabolic syndrome and higher weight outcomes in midlife women (Hall et al., 2012). Similarly, women who reported sleeping less than six hours per night were significantly more likely to experience metabolic disturbances and have higher weight outcomes than those who reported more sleep (Choi et al., 2011).



**Sleep Quality.** In addition to duration, sleep quality, defined as the subjective assessment of “good” or “poor” sleep, has also been linked to higher weight outcomes. In particular, in a study of Korean adults, those with poorer self-reported sleep quality had a higher prevalence of abdominal obesity (Lee et al., 2013). Additionally, in a sample of midlife women, poorer sleep quality was linked to higher weight outcomes (Jennings et al., 2007).

**Sleep Efficiency.** In addition to quality, sleep efficiency has also been tied to obesity. Sleep efficiency is defined as the ease of falling asleep and returning to sleep and is quantitatively defined as the ratio of time spent sleeping to time spent in bed (Bei, Ong, Rajaratnam, & Manber, 2015; Buysse, 2014). Sleep efficiency has been inversely associated with adiposity in young adult women (Bailey et al., 2014). Similarly, in midlife women, poor sleep efficiency was associated with an increased risk for poor weight outcomes and metabolic disturbances including increases in central adiposity (Hall et al., 2012).

**Sleep Timing.** Sleep timing, defined as the placement of sleep within the 24 hour day (Buysse, 2014), has also been linked to poor weight outcomes. Sleep timing is a relatively new area of research that is related to social factors and biological processes. Socially, sleep timing is impacted by factors such as occupation. Shift workers are a commonly studied population in this area with delays in sleep timing leading to poor health outcomes such as obesity (Kim et al., 2013; Suwazono et al., 2006). Biologically, sleep timing is governed by circadian rhythms. Circadian rhythms are defined as an approximate 24-hour endogenous cycle driven by an internal “master clock” that impacts physical, mental, and behavioral processes (Delezie & Challet, 2011). Disruption of this rhythm has been found to be a key component in the regulation of metabolism and energy homeostasis, which have been found to impact weight outcomes (Delezie & Challet, 2011; Gonnissen, Hulshof, & Westerterp-Plantenga, 2013).

Sleep timing can be characterized in different ways. One of which is through the measurement of mean sleep timing or the overall mean bedtime of individuals quantified across a number of days. Research has demonstrated that individuals with preference for late bedtimes have higher weight outcomes and poorer metabolic profiles. Individuals with consistently late bedtimes (initiating sleep between 1:00 am – 3:00 am) were more likely to have central obesity and other symptoms of metabolic syndrome than individuals with earlier bedtimes (initiating sleep between 9:00 pm – 11:00 pm; Lee & Shin, 2015). In a primary care sample, individuals with later bedtimes were at an increased risk for the development of obesity (Logue, Scott, Palmieri, & Dudley, 2014). The link between mean bedtime and weight has also been found in a sample of midlife women, with evidence for higher BMIs in depressed peri and post-menopausal women with later sleep timing (Meliska et al., 2011).

In addition to the measurement of mean sleep timing, daily variability in sleep timing has also been linked to obesity. Similar to the investigation of mean sleep timing and weight, variability in daily sleep timing can also disrupt circadian rhythms and lead to poor weight outcomes (Delezie & Challet, 2011; Gonnissen, et al., 2013). Daily variability in individual bed timing, known as intraindividual variability (IIV), is quantified as the daily variation around mean bedtime values. Research has demonstrated that IIV in bed timing leads to poor weight outcomes. In a study of adults in an urban primary care setting, less stable bedtimes were associated with obesity (Logue et al., 2014). In addition, in a study of young adult females, bed timing inconsistency across a seven day time period was found to predict body fat percentage (Bailey et al., 2014). The relation between bedtime variability and weight has also been observed in midlife women samples with variability in sleep timing, particularly deviation from typical bedtime, being associated with higher BMI (Taylor et al, 2016).

Although recent work investigating sleep timing and weight is promising, research in this area is limited. In particular, less is known about sleep timing and weight in midlife women samples. The study of sleep timing in midlife women is warranted, given the unique sleep difficulties for this population. For example, women in midlife have issues initiating sleep, which is particularly true during the menopausal transition (Owens & Matthews, 1998). There are multiple explanations for this, including psychosocial factors such as stress due to work, finances, and family, as well as increases or changes in parental and caregiving responsibilities (Hislop & Arber, 2003). In addition to psychosocial stressors, women in midlife also experience many hormonal changes with primary changes in estrogen and progesterone levels. These hormones have an impact on stress, mood, and body temperature and may also play a role in sleep regulation (Polo-Kantola, 2011; Shneerson, 2005). Although current research suggests a relationship between late bed timing, intraindividual variability in daily bedtimes, and weight outcomes, these investigations are limited, and more work is needed to examine these associations in non-shift work samples and, specifically, in samples of midlife women (Bei et al., 2016; Lee & Shin, 2015; Taylor et al., 2016).

### **Mechanisms Linking Sleep to Obesity**

Although there are clear connections between sleep and obesity, there is debate concerning the individual factors and specific mechanisms that connect sleep to weight problems. Not all individuals and, specifically, not all midlife women who report sleep problems are obese (Nishiura, Noguchi, & Hashimoto, 2010; Schmid et al., 2009), suggesting a role for additional factors. As such, biological, cognitive, behavioral, and emotional mechanisms have been investigated as links between poor sleep and obesity.

Biological mechanisms have been identified as one pathway linking sleep to weight outcomes. Short sleep duration and sleep variability can disrupt hormonal regulation of hunger and satiety signaling by decreasing leptin, a hormone involved in metabolism and appetite and by increasing ghrelin, a hormone involved in appetite stimulation (Spiegel, Tasali, Penev, & Van Cauter, 2004). Sleep timing in particular has been found to disrupt hormonal regulation of appetite by disrupting biological circadian rhythms. Misaligned sleep timing has been found to disrupt endogenous circadian rhythms, which leads to alterations in leptin and glucose levels (Scheer, Hilton, Mantzoros, & Shea, 2009). Short sleep duration and reductions in total sleep time have also been shown to activate brain regions associated with reward processing, increasing preferences for energy dense foods (Benedict et al., 2012; Nedeltcheva et al., 2009) and decreasing physical activity (Markwald et al., 2013; Schmid et al., 2009).

In addition to biological mechanisms, sleep can impact cognitive and behavioral mechanisms. Short sleep duration and increased variability in sleep duration influence cognitive mechanisms, including executive functioning, by impairing the prefrontal cortex. Impairment of executive functioning can impact goal-directed behavior, executive control, and impulse control, all which can influence eating behavior. (Beebe et al., 2012; Dahl, 1996). Disrupted sleep can also alter behavioral mechanisms including impulse control, which can lead to an increase in food consumption due to an inability to inhibit eating behavior (Anderson & Platten, 2011; Guerrieri et al., 2007).

Emotional regulation and processing are also impacted by sleep. Short sleep duration influences emotional reactivity, leading to heightened feelings of emotional distress (Vgontzas, Bixler, Chrousos, & Pejovic, 2008). Additionally, sleep restriction can create heightened reactions of the amygdala, leading to amplification of negative emotions (Walker & Van der

Helm, 2009; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). Higher levels of negative emotion and emotional distress have been linked to the consumption of food, as food can improve mood and decrease stress through its effect on dopamine, serotonin, and opioid systems (Gibson, 2006). However, more work is needed to understand how emotional pathways link sleep and weight.

### **Emotional Mechanisms Linking Sleep and Obesity in Midlife Women**

Although certain pathways such as biological mechanisms linking sleep and weight have been identified and studied extensively, less work has explored the role of emotional mechanisms linking sleep to weight, especially among midlife women. Emotion is important to investigate in midlife women given that emotional concerns and mood fluctuations are a primary complaint of women in this population. In addition to menopause and the menopausal transition, which has been found to lead to increases in depressive symptoms in this population (Avis, Brambilla, McKinlay, & Vass, 1994; Bromberger et al., 2001, Bromberger et al., 2007; Cohen, Soares, Vitonis, Otto, & Harlow, 2006), women in midlife can also experience poor emotional outcomes due to factors such as dual-role responsibilities (i.e., working outside the home and having primary responsibility of household responsibilities), changes in caregiving roles (i.e., need to care for both children and parents), marital dissatisfaction, lack of social support, and psychological concerns due to the aging process (i.e., negative changes in self-perception and self-worth; Earle, Smith, Harris, & Longino, 1997). Factors such as marital dissatisfaction, dual-role conflict, and aging-related changes in self-perception have been shown to influence emotional well-being and lead to emotional disturbances including depression, anger, and irritability in midlife women (Baram, 2005; Born, 2004; Bromberger et al., 2003; Earle, et al., 1997; Powell, 1996). As both depression and anger are prevalent in this population, with midlife women being at an increased risk for the development of depression and with 70% of women in

the midlife rating irritability as their primary emotional disturbance, both depression and anger are relevant to explore in this population (Born, 2004; Bromberger et al., 2007; Cohen et al., 2006).

**The Impact of Sleep on Emotions.** Poor sleep impacts an individual's ability to process emotion, and individuals with poor sleep tend to amplify negative emotions (Yoo et al., 2007; Walker & Van der Helm, 2009). Accordingly, individuals with sleep disturbances have increased self-report ratings of depression, frustration, anger, anxiety, and emotional lability, all of which might be partially explained by disturbances in emotional processing (Durmer & Dinges, 2005; Horne, 1985; Kahn-Greene, Lipizzi, Conrad, Kamimori, & Killgore, 2006; Pilcher & Huffcutt, 1996). Sleep disturbances also increase negative emotionality, attenuate emotional expressiveness, and put individuals at risk for depression (Minkel, Htaik, Banks, & Dinges, 2011; Wichniak, Wierzbicka, & Jernajczyk, 2013).

Although sleep disturbances are linked to negative emotional outcomes, sleep timing and variability in sleep timing also put individuals at risk for mood disturbances. A large body of literature demonstrates a greater risk for depressed mood and greater severity of depression for individuals with preference for later timing of sleep (Duffy, Rimmer, & Czeisler, 2001; Mongrain, Carrier, & Dumont, 2006). Variability between weekday versus weekend bed timing has also been linked to depressive symptoms above and beyond sleep duration, reflecting the unique impact of sleep variability on mood and emotion (Levandovski et al., 2011). The relation between sleep timing and depression has been observed in midlife women. Peri- and post-menopausal women with preference for later bedtimes were more likely to be depressed, indicating that menopausal women, with preference for later bedtimes, are vulnerable to mood disturbances (Meliska et al., 2011).

In addition to depressed mood, poor sleep is associated with other negative mood states including anger. In healthy German individuals, poorer sleep was associated with higher endorsement of aggressive tendencies and lower frustration tolerance (Schubert, 1977). Poor sleep quality and short sleep duration are also positively correlated with anger, hostility, and aggressive behavior in adult populations (Brisette & Cohen, 2002; Grano, Vahtera, Virtanen, Keltikangas-Jarvinen, & Kivimaki, 2008; Pilcher, Ginter, and Sadowsky, 1997; Taylor, Fireman, & Levin, 2013). The relation between poor sleep and anger has also been found in midlife women (Thomas & Donnellan, 1991). In a sample of midlife Korean men and women, trait anger was associated with both sleep disturbances and early morning awakenings (Shin, Kim, Lee, Lee, & Shin, 2004).

**The Impact of Emotions on Obesity.** There is also evidence that emotional states lead to poor weight outcomes. A large body of evidence supports depression as a risk factor for obesity (Blaine, 2008; Luppino et al., 2010). Additionally, in a smaller number of studies, positive associations between anger and weight have been identified (Lewis et al., 2009; Niaura et al., 2002).

The link between depression and obesity has been extensively studied in child and adult populations with depression associated with an increased risk for obesity (Blaine, 2008; de Wit et al., 2010; Luppino et al., 2010; Strine et al., 2008). This finding has also been demonstrated in samples of midlife women with depression and low mood associated with higher caloric intake and higher rates of obesity (Clum et al., 2014; Crawford et al., 2011; Simon et al., 2008). Although depression can lead to weight gain and obesity in midlife women, depressive symptoms also have implications for waist circumference and visceral adipose tissue in midlife, both of which are risk factors for central obesity and metabolic syndrome. Older men and women

diagnosed with a major depressive episode had a greater increase in visceral adipose tissue over time, independent of body mass index and weight change as compared to controls (Weber-Hamann et al., 2006). Depressive symptoms also predicted a longitudinal increase in waist-hip-ratio (Nelson, Palmer, Pederson, & Miles, 1999) and were associated cross-sectionally with greater waist circumference in midlife women (Raikkonen, Matthews & Kuller, 1999a).

In addition to depression, anger is an emotional state that has been linked to obesity and weight outcomes. Although there is more modest support for the association between anger and weight outcomes, positive associations between anger, hostility, and adiposity have been identified, with evidence that anger and hostility predict increases in weight over time (Bunde & Suls, 2006; Everson et al., 1997; Niaura et al., 2002; Nelson, et al., 1999). This association has also been demonstrated specifically in midlife women (Lewis et al., 2009). The Pittsburgh Healthy Women Study showed that baseline levels and changes in trait anger predicted increases in waist circumference and visceral adipose tissue over time in midlife women. The study also found overt anger expression predicted greater visceral adipose tissue at follow-up (Raikkonen et al., 1999a; Raikkonen, Matthews, Kuller, Reiber, & Bunker, 1999b). Trait anger, or a combination of trait anger and anger expression, has also been associated with waist-circumference in women (Wing et al., 1991b).

**Connecting Sleep, Emotions, and Obesity in Midlife Women.** Although there is evidence that: 1) sleep impacts depression and anger, and 2) depression and anger impact weight, there is limited work exploring these emotions as a mechanism linking sleep to weight outcomes. Existing research has primarily focused on the interconnectedness of sleep, mood, and weight outcomes without specifying mechanistic roles. For example, the combination of sleep disturbances and depressed mood was shown to lead to increased leptin levels in women, a



hormone which is linked to poor weight outcomes (Häfner et al., 2012). Relatedly, the combination of sleep, depression, and stress was found to influence weight loss outcomes for women (Elder et al., 2012). Lastly, poor sleep quality was strongly associated with mood disturbances in an obese population (Araghi et al., 2013). Although it is clear that there is a connection between sleep, mood, and weight in women, more research, and specifically research involving midlife women, is needed to understand these associations. Women in midlife are unique in that sleep, emotional health, and weight are in fluctuation during this time period as a result of changing developmental, biological, and psychosocial features (Earle et al., 1997; Hislop & Arber, 2003). Although sleep influences emotions (Meliska et al., 2011; Shin et al., 2004), emotions influence weight (Clum et al., 2014; Raikkonen et al., 1999a), and sleep influences weight (Bei et al., 2016; Lee & Shin, 2015; Taylor et al., 2016), it is unclear how these factors are inter-related in this population. Thus, given the sparse literature on this topic, more work is needed to further disentangle these relations. Furthermore, developing a greater understanding of emotional pathways linking sleep to weight is vital for this population as it is possible that interventions focused on sleep and/or emotional health might be relevant for improving health outcomes in this population.

### **Summary and Purpose of Current Study**

Women in midlife are at risk for the development of obesity as a result of physiological and psychological changes that occur in this time period. As of 2014, 42% of women between the ages of 40-60 were classified as obese as indicated by having a BMI equal to or above 30 (Ogden et al., 2015). In addition to BMI as an indicator of weight gain, accumulated adiposity around the midsection during midlife also increases the risk for central obesity and poor weight-related health outcomes (Heymsfield et al., 1994; Ho et al., 2010). Sleep is a factor that has

recently been implicated in obesity for midlife women. Women in midlife are vulnerable to sleep disturbances and many facets of sleep are related to weight outcomes in this population (Hall et al. 2012; Jennings et al. 2007; Taylor et al. 2016). One aspect of sleep that has received recent attention is sleep timing and sleep timing variability. Sleep timing and variability in sleep timing are important to investigate given that midlife women report a high level of sleep disturbances and specifically report trouble initiating sleep (Hislop & Arber, 2003; Owens & Matthews, 1998). Hormonal changes during midlife have also been shown to impact the circadian timing of sleep, making sleep timing a relevant area to explore in this population (Polo-Kantola, 2011; Shneerson, 2005).

Although sleep has been linked to obesity in midlife women, the mechanisms linking sleep and obesity are unclear. Biological mechanisms have been studied extensively, with the identification of specific hormones and brain areas that are associated with poor sleep and weight gain (Benedict et al., 2012; Nedeltcheva et al., 2009; Spiegel et al., 2004). However, less is known about the role of emotion as a mediating factor. Emotional fluctuations are prevalent in midlife women, with depression and anger being primary areas of concern (Bromberger et al. 2003; Bromberger et al. 2007). Poor sleep is a factor that can lead to attenuated emotional responses, increased negative emotionality, and, therefore, poor eating behaviors and weight outcomes in this population (Gibson, 2006; Walker & Van der Helm, 2009; Yoo et al., 2007). However, the mediating role of emotions in the association between sleep and weight has not yet been investigated in midlife women.

Therefore, the overall objective of the current study was to explore the association between sleep timing and obesity outcomes in a sample of midlife women. The association between sleep timing and weight outcomes is crucial to explore in this population, given that

limited research has examined this connection in midlife women despite the fact that midlife women frequently have difficulties with sleep initiation and sleep regulation (Owens & Matthews, 1998; Polo-Kantola, 2011; Shneerson, 2005). Additionally, the limited body of research in this area is inconsistent, with methodological variability in the measurement and operationalization of sleep timing producing discrepant findings. The current study addresses these methodological concerns by comprehensively investigating sleep timing (i.e., mean sleep time, sleep time variability) using well-validated, daily measures of sleep behavior (i.e., actigraphy, daily sleep diaries). Furthermore, the current study is the first to use actigraphy to investigate sleep timing and weight outcomes in midlife women.

In addition to the emphasis on a comprehensive assessment of sleep timing, the current study will also employ multiple measures of obesity (i.e., obesity, central obesity) through the assessment of both BMI and waist circumference as weight outcomes. The differentiation between obesity and abdominal obesity in midlife women is necessary, given that women in midlife experience redistribution of body fat during this time period, although many studies fail to recognize waist circumference as an independent outcome (Wells, 2000). The current study is the first to examine both BMI and waist circumference as outcomes in the sleep timing—obesity association in midlife women.

Lastly, sparse research exists using controlled analyses to examine pathways linking sleep timing and weight outcomes in midlife women. This study explores emotional factors as a potential pathway in this association. Work in this domain is warranted given that prior research had determined the negative impact of poor sleep and negative emotional factors on weight outcomes in this population (Choi et al., 2011; Clum et al., 2014; Hall et al., 2012; Meliska et al.,

2011; Raikkonen et al., 1999a), although limited research has explored the connection between sleep, emotions, and weight in controlled studies.

As such, this study had two aims: (1) to examine the association between sleep timing and obesity outcomes in midlife women, and, (2) to investigate depressive symptoms and anger as mediators of the sleep timing and obesity associations in midlife women. The current study is novel in that it is the first to: a) explore emotional factors as a potential pathway between sleep timing and weight outcomes in midlife women using a controlled model, b) investigate multiple indices of sleep timing (i.e., mean sleep time, sleep time variability) in this population using actigraphy, and, c) include both BMI and waist circumference as obesity outcomes. In addition to examining the association between sleep timing, emotion, and obesity in this population, the current study addresses discrepant findings in this domain and increases knowledge of this area to inform preventive interventions for weight gain in midlife women.

## **Method**

### **Design**

A secondary data analysis was performed using cross-sectional data from the Midlife in the United States (MIDUS-II) dataset. MIDUS-II is funded through the National Institute on Aging at the University of Wisconsin Madison (P01-AG020166).

### **Participants**

Participants completed the Midlife in the United States (MIDUS) II study, a longitudinal telephone and paper-and-pencil follow-up study of the original MIDUS I study (N = 7108). Data collection for MIDUS-II took place from 2004-2006. All eligible participants were non-institutionalized, English-speaking adults, and between 35-86 years of age. The MIDUS-II sample included approximately 4963 participants and consisted of four sub-projects.

The current study consists of a sub-sample of 139 female MIDUS-II participants between the ages of 40-64 who completed both Project 1 and Project 4 (biomarker project) of the MIDUS-II study. Demographic information and participant characteristics including, race, income, education, and self-rated health was collected during Project 1 of the MIDUS-II study. Daily physical activity, sleep behaviors, depressive symptoms, anger symptoms, BMI, and waist circumference were collected during Project 4.

### **Exclusionary Criteria**

In order to represent a normal range of sleeping behavior in midlife women, the current study controlled for possible effects of shiftwork and abnormal sleeping behavior due to irregular work schedules. Thus, we excluded participants if they indicated “yes” on the item, “Does participant show idiosyncratic sleep pattern due to work schedule, illness or travel.” This item was obtained from the MIDUS-II actigraphy dataset and was calculated to assess irregular sleeping patterns. Participants were also excluded if they scored greater than 1 on the item, “How many nights in the past 12 months did your work require you to be away from home overnight?” This question was taken from a self-administered phone questionnaire from MIDUS-II Project 1. As a result of excluding irregular sleep patterns due to irregular work and sleep schedules, 7 participants were excluded from the study. The final sample size included 132 participants.

### **Procedure**

In MIDUS-II, Project 1, participants completed a phone interview and two self-administered questionnaires (SAQs) measuring psychological constructs, demographic variables, and mental and physical health. Additionally, subsets of participants in MIDUS-II, Project 1, were selected to complete additional projects (e.g., daily diary study, cognitive functioning, biomarkers, and neuroscience projects). The current study used data from participants who were

involved in both Project 1 (completion of the phone and self-administered questionnaires) and were selected to participate in Project 4's (biomarker project) University of Wisconsin-Madison site project. Project 4 involved the completion of self-administered questionnaires (i.e., CES-D, Spielberger Trait Anger Inventory), an in-person physical examination (BMI, waist circumference measurements), and the completion of a daily sleep diary and wrist actigraphy protocol.

### **Measures**

**Sleep.** Daily sleep behaviors were assessed using self-report daily sleep diaries and wrist actigraphy (Actiwatch-64®, Philips Respironics). The sleep diary had participants answer questions regarding bedtime, amount of time to fall asleep, wake time, out of bed time, and sleep quality ratings across a continuous seven day time period. Participants also wore actigraphy watches for the same continuous seven day time period where they indicated when they tried to fall asleep (bedtime) and when they awoke in the morning (wake time) by pressing an event marker button on the Actiwatch®. At the conclusion of data collection, event marker and sleep diary information were employed to assist in the automatic detection of sleep intervals by Actiwatch® algorithms (Ancoli Israel et al., 2003). Anomalous or incomplete actigraphy intervals were reviewed further by study staff and hand scored or deleted as appropriate. Average sleep duration and sleep onset latency were determined by averaging respective values from each of the seven study nights for each participant. Both actigraphy and sleep diaries are a primary form of sleep assessment and have been found to be both reliable and valid (Carney, Lajos, Waters, 2004; Lichstein, Riedel, & Means, 1999). Additionally, sleep diaries and actigraphy provide a repeated assessment of sleep behavior, which can incorporate variability across days

(Carney et al., 2012). All participants in the current study completed all seven days of sleep diaries and there is no missing data for the sleep variables.

The primary variables of interest for the present study that were assessed from actigraphy include sleep timing (sleep onset) and sleep timing variability. In addition to sleep timing variables, additional variables from actigraphy including sleep duration and sleep onset latency were included in the current study as covariates. The additional sleep covariate of sleep quality was calculated from the daily sleep diary.

*Sleep timing.* Sleep timing was assessed by calculating mean sleep time values and sleep time variability. Mean sleep time was calculated as the mean time of sleep start (sleep onset) across seven days. Sleep start time was defined as the first 10 minute period in which no more than one epoch (i.e., 30 second time period) was scored as mobile using Actiwatch® software. Variability in sleep time was quantified by calculating intraindividual variability in sleep timing for each participant's sleep start time (sleep onset) across seven days. This resulted in intraindividual standard deviations. All intraindividual standard deviations variables were detrended for time to control for any variations due to the effects of observing behaviors over time. Specifically, detrending for time produces a variable that precisely reflects variability over time that is not due to the effects of time, per se (e.g., practice effects, participation fatigue, etc.), but rather due to inherent variations in the behavior. Detrending was conducted via linear regression analyses for all participants with time (linear, quadratic, and cubic functions) as the independent variables and the sleep timing variables as the dependent outcomes. Intraindividual standard deviations values were then calculated for the sleep timing variables using the time-independent residuals from the aforementioned linear regression analyses. This detrending

process resulted in a sleep time variability variable of within-person standard deviations that was independent of any influences of time.

***Sleep covariates.*** Sleep covariates included sleep duration, sleep onset latency and sleep quality. Sleep duration and sleep onset latency were calculated using actigraphy. Sleep quality was assessed with daily sleep diaries. Sleep duration was defined as the amount of time between sleep start and sleep end taking into account wake after sleep onset values. Sleep onset latency was determined by calculating the mean amount of time, in minutes, it took participants to fall asleep and was defined as the amount of time elapsed for the onset of sleep (sleep start) after attempting to get to sleep (as indicated in sleep diary). Lastly, sleep quality was determined from taking the mean score of how participants rated their overall sleep quality using daily sleep diaries. Sleep quality was assessed with a 1 to 5 scale with 1 indicating very good and 5 indicating very poor. All sleep variables were averaged across the seven nights of data collection to create a mean for each sleep variable.

**Depressive symptoms.** Depressive symptoms were measured with the Center for Epidemiologic Studies Depression Scale (CES-D), a 20-item self-report scale that assesses symptoms associated with depression such as poor appetite and feeling lonely (Radloff, 1977). Participants were asked to rate the frequency of experiencing 20 items during the prior week on a 4-point scale ranging from 0 (rarely or none of the time) to 3 (most or all of the time). Scores were summed to create a total score ranging from 0 – 60. One of the items, “my sleep was restless” was excluded from the total score calculation due to its similarity to the sleep constructs assessed by the sleep diary and actigraphy measures. Therefore, the total score possible from the current sample was 57. The exclusion of the sleep item from the CES-D is well-validated and commonly utilized in other studies assessing sleep (Dirksen, Epstein, & Hoyt, 2009; Levine et



al., 2003; Nadorff, Nazem, & Fiske, 2013; Wilcox et al., 2000). Scores for the CES-D ranged from 0-57. The CES-D is a well-established measure designed for use in epidemiologic studies. It has high internal consistency (Cronbach's  $\alpha = 0.89$ ) and has shown good sensitivity and specificity for identifying individuals at risk for clinical depression (Radloff, 1977). Cronbach's alpha value for the current sample was .76.

**Anger.** Anger in the current study was measured using the trait anger scale of Spielberger's State-Trait Anger Expression Inventory (STAXI; Spielberger, 1996), a measure of the experience and expression of anger. Respondents indicated how they "generally felt" to 15 given statements (e.g., "I have a fiery temper," "I feel irritated"). For each item, participants rated themselves on a 4-point scale from 1 (almost never) to 4 (almost always). Total scores were obtained by summing the items of the scale. Scores range from 15 – 60 with higher scores indicating a greater tendency to experience and express anger. Cronbach's alpha value for the current sample was .85.

**Obesity and central obesity.** Obesity and central obesity were measured during a physical exam using a standardized procedure. Obesity, defined as having a body mass index (BMI) greater than or equal to 30 (WHO, 2000), was calculated by dividing body weight (in kilograms) by height (in meters squared). Central obesity, defined as having a waist circumference > 88 cm (Grundy et al., 2004), was measured at the narrowest point between the ribs and iliac crest. Both obesity and central obesity variables were analyzed as continuous variables.

**Additional covariates.** In addition to sleep covariates (i.e., sleep duration, sleep onset latency, sleep quality), other covariates examined in the current study included age, race, educational level, annual income, self-rated health, and physical activity. Age, race, educational

level, annual income and self-rated health variables were obtained from self-administered phone questionnaires from Project 1 of the MIDUS-II study. Self-rated health was assessed by a self-report measure from Project 1, where participants were asked to rate their general physical health. The questionnaire consisted of a scale ranging from 1 to 5. Items were reversed scored such that a score of “1” indicates “excellent” and a score of “5” indicates “poor”. Physical activity was assessed by daily sleep diaries with the question, “How many minutes of moderate to vigorous exercise did you engage in today?” A score of weekly physical activity was obtained by summing the score of physical activity, in minutes, across the seven days of data collection.

### **Statistical Analyses**

**Descriptive statistics and covariate evaluation.** Several covariates were entered into analyses given their known associations with sleep, weight, and emotional factors in this population. Specifically, age, weekly physical activity, self-rated health, annual income, race, educational level, and specific sleep variables including sleep duration, sleep onset latency, and sleep quality were considered as covariates (Campbell & Samaras, 2000; Choi et al., 2011; Crawford, et al., 2000; Jennings et al., 2007; Lahman et al., 2000; Owens & Matthews, 1998; Wing et al., 1991a).

First, Pearson correlation analyses were calculated to identify significant bivariate associations between covariates and main variables of interest (i.e., mean sleep time, sleep time variability, depressive symptoms, anger, BMI, waist circumference). To increase power and degrees of freedom for analyses, only variables with significant bivariate associations were included in the final statistical models ( $p < .05$ ). In final analyses, covariates for BMI analyses included sleep duration and educational level. For waist circumference analyses, sleep duration was included as a covariate. Other covariates including race, annual income, weekly physical

activity, age, self-rated health, sleep onset latency, and sleep quality were not included in statistical models given that they were not statistically related to outcomes variables in the sample (Table 3).

**Aim 1.** The first aim of the study was to investigate whether sleep timing and sleep timing variability predicted obesity and/or waist circumference. Informed by existing research, I hypothesized that:

1. Mean sleep timing (i.e., later sleep timing) would be associated with higher levels of obesity (i.e., greater BMI) in the current sample after adjusting for select covariates.
2. Greater variability in sleep timing would be associated with higher levels of obesity (i.e., greater BMI) in the current sample after adjusting for select covariates.
3. Mean sleep timing (i.e., later sleep timing) would be associated with larger waist circumference in the current sample after adjusting for select covariates.
4. Greater variability in sleep timing would be associated with larger waist circumference in the current sample after adjusting for select covariates.

**Aim 2.** The second aim of the study was to investigate whether depressive symptoms and/or anger mediated the association between sleep timing variables (mean sleep timing and sleep timing variability) and obesity and/or waist circumference. Based on existing literature, I hypothesized that:

1. Later sleep timing and greater sleep timing variability would be associated with higher levels of depressive symptoms and higher levels of anger.
2. Higher levels of depressive symptoms and anger would be associated with higher rates of obesity (greater BMI) and larger waist circumference.

3. Depressive symptoms and anger would mediate the sleep timing (both mean and variability) and obesity and waist circumference associations.

**Parallel mediation analyses.** To assess aims 1 and 2, Hayes' SPSS PROCESS macro (Hayes, 2013) was used to run four parallel mediation models to test the direct association of sleep timing (mean sleep time, sleep time variability) and weight outcomes (BMI, waist circumference), and the mediating role of depressive symptoms and/or anger in the sleep timing—obesity association. The BMI outcome models controlled for possible confounders including sleep duration and educational level. The waist circumference models controlled for the possible confounder of sleep duration. Using the PROCESS macro, the indirect effect of depressive symptoms and/or anger was tested using a non-parametric, bias-corrected bootstrapping procedure that provided an empirical approximation of the sampling distribution of the product of the estimated coefficients in the indirect paths using 5,000 resamples from the data set.

## **Results**

### **Data preparation and data cleaning**

SPSS 23.0 was used for all data analyses. Data were cleaned and descriptive statistics (means, standard deviations, and frequencies) calculated to verify that data met the assumptions of the planned analyses. A review was conducted to assess skewness, kurtosis, and outliers for all main variables and covariates of interest. Skewness and kurtosis values for mean sleep time, anger, BMI, and waist circumference were close to or below an absolute value of 1, indicating that they were approximately normally distributed. Sleep time variability and sleep duration were positively skewed and had positive kurtosis (i.e., values greater than one). Outliers were removed from the dataset to reduce skewness. Depressive symptoms also had skewness and kurtosis

values greater than one, indicating positive skewness and kurtosis. Outliers were removed from the dataset and a square root transformation was performed to create a normal distribution.

Transformed values of depressive symptoms, sleep time variability, and sleep duration were used for all correlational analyses, parallel mediation analyses, and follow-up analyses. Demographic information does not contain transformed values. In addition to a review of skewness, kurtosis, and outliers, assumptions of independence, normality, multicollinearity, and homoscedasticity were assessed. All values were sufficiently met. Power calculations using G\*Power (Faul, Erdfelder, Buchner, & Albert-Georg, 2009) suggested that for a mediation analysis with five predictors, a sample size of at least 92 participants was needed to predict an  $R^2$  of at least .15 at an alpha level of .05, with a power of .80. In the current study, assuming a medium effect size, 132 participants was sufficient to detect an effect.

### **Descriptive and correlational results**

First, sociodemographic and health characteristics were examined (Table 1). On average, participants were 52.9 years old ( $SD = 6.94$ ), primarily White, with over 50% of participants having a two-year associate's degree or greater. The average annual income of participants was \$66,449 ( $SD = \$53,563$ ). Women in the sample were generally overweight and obese, with high levels of abdominal obesity ( $BMI = 31.04 \pm 6.96$ , waist circumference =  $99.61 \pm 17.64$ ; Table 2). Levels of obesity are slightly higher in the current sample (i.e., 48.6%) compared with the national average of midlife women in 2014 (i.e., 42.1%; Ogden et al., 2015). Waist circumference values in the current sample were also slightly higher (i.e., 99.45) than the national average for women 18 years and older in the United States as of 2012 (i.e., 96.0; Ford, Maynard, & Li, 2014). Pearson correlations were conducted to examine bivariate associations

between all main variables of interest (i.e., mean sleep time, sleep time variability, depressive symptoms, anger, BMI and waist circumference) and all covariates (Table 3).

Table 1

<i>Participant Sociodemographic and Health Characteristics (M, SD)</i>		
Variable	N	Percentage (%)
Age (52.90, 6.94)		
40-49 years	51	34.23
50-59 years	61	40.94
60-64 years	37	24.83
Race		
White/Caucasian	131	87.9
Black/African American	7	4.7
Asian American	2	1.3
Native American/Alaska Native	1	0.7
Other/Don't Know	8	5.4
Income (\$66,449, 53,563)		
0 – \$20,000	24	19.4
\$20,000 – \$39,999	20	16.1
\$40,000 – \$59,999	18	14.5
\$60,000 – \$79,999	19	15.3
\$80,000 - \$100,000	9	7.3
Greater than \$100,000	34	27.4
Education		
Professional Degree	4	2.7
Master's Degree	4	2.7
Some Graduate School	4	2.7
Bachelors	30	20.1
Two-Year	15	10.1
3+ college	9	6.0
1-2 college	29	19.5
High school	35	23.5
GED	7	0.7
Did not graduate high school	7	4.7
Physical Activity (244.79, 414.52)		
Less than 150 minutes	70	53.4
150 – 299 minutes	34	26.0
300 minutes or greater	27	20.6
Self-Rated Health (2.37, .97)		
Excellent (1)	25	16.8
Very Good (2)	67	45.0
Good (3)	38	25.5
Fair (4)	15	10.1
Poor (5)	4	2.7

*Note.* Self-rated health consists of scale ranging from 1 to 5 with items reversed scored such that 1 indicates “excellent” and 5 indicates “poor”. Physical Activity variable indicates sum of daily physical activity in minutes per week.

Table 2

*Participant Weight, Emotion Characteristics, and Sleep Time (M, SD, range)*

Variable	N	Percentage (%)
BMI (31.05, 6.89, 17.13 – 52.00)		
Less than 18.5	1	0.7
18.6 - 25	24	16.4
25.0 - 29.9	50	34.2
Greater than or equal to 30	71	48.6
Waist Circumference (99.45, 17.52, 66.00 – 158.00)		
Less than 80.00 cm	16	10.7
80.0 – 87.99	24	16.1
88 cm and above	109	73.2
Depressive Symptoms (14.68, 5.19, 4.00 – 31.20)		
Anger (24.00, 4.99, 15 – 41)		
Mean Sleep Time (11:36 pm, 84 minutes, 7:48 pm – 3:54 am)		
Sleep Time Variability (43 minutes, 51 minutes, 0 – 412 minutes )		

*Note.* A BMI level less than 18.5 indicates underweight status, a level 25 or greater indicates overweight status, and a level greater than or equal to 30 indicates obese status. A waist circumference greater than or equal to 88 cm indicates central obesity.

Table 3

*Pearson Correlation Coefficients among Weight, Sleep Timing, Emotion, and Covariate Variables*

	BMI	Waist Circumference	Mean Sleep Time	Sleep Time Variability	Depressive Symptoms	Anger
BMI	-					
Waist Circumference	.847**	-				
Mean Sleep Time	-.004	.017	-			
Sleep Time Variability	.133	.164*	.187*	-		
Depressive Symptoms	.087	.083	.227**	.204*	-	
Anger	.048	.037	.187*	.065	.444**	-
$C_1$ Sleep Duration	-.200*	-.283**	-.264**	-.529**	-.190*	-.006
$C_2$ Education	-.101*	-.082	-.023	.097	.007	.072
$C_3$ Sleep Onset Latency	-.057	.007	.337**	.406**	.315**	.052
$C_4$ Sleep Quality	.051	.088	.148	.144	.322**	.290**
$C_5$ Physical Activity	-.063	-.107	-.034	-.027	-.261**	-.145
$C_6$ Self-Rated Health	.139	.078	-.045	-.131	-.083	-.061
$C_7$ Annual Income	-.104	-.139	-.102	.056	-.021	-.082
$C_8$ Age	.033	.076	-.135	-.120	-.150	-.119
$C_9$ Race	-.145	-.110	.130	.022	.060	-.055

*Note.*  $C_1$  –  $C_9$  indicate covariates.

\* $p < .05$ . \*\*  $p < .001$ .

### The direct association between sleep timing and BMI

Two parallel mediation models were run with BMI as the outcome to examine depressive symptoms and anger as indirect pathways between sleep timing (mean sleep time and sleep time variability) and obesity (BMI; Figure 1).

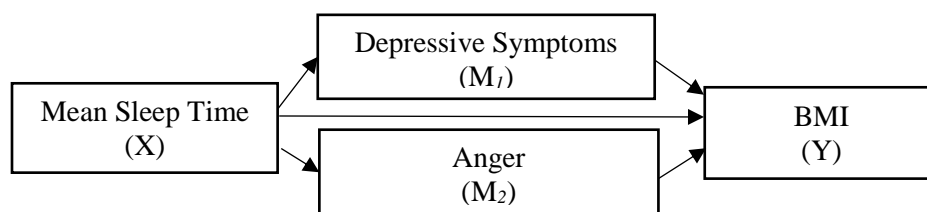


Figure 1. Example of parallel mediation model.

First, the direct association between mean sleep time and BMI was investigated. Although the overall model, after controlling for sleep duration and education was significant,  $F(3, 126) = 5.55, p = .001, R^2 = .117$ , mean sleep time was not significantly associated with BMI (95% CI =  $-.0146, .0075$ ; Table 4). In the investigation of sleep time variability and BMI, the overall model was significant after controlling for sleep duration and education,  $F(3, 126) = 5.74, p = .001, R^2 = .120$ , however sleep time variability was not significantly associated with BMI (95% CI =  $-.0148, .0417$ ; Table 5).

### The direct association between sleep timing and waist circumference

Next, two mediation models were run with waist circumference as the outcomes to examine depressive symptoms and anger as indirect pathways between sleep timing (i.e., mean sleep time and sleep time variability) and obesity (i.e., waist circumference). First, the direct association between mean sleep time and waist circumference was investigated. Although the overall model was significant after controlling for sleep duration,  $F(5, 124) = 3.73, p = .003, R^2 = .131$ , mean sleep time was not significantly associated with waist circumference (95% CI = -



.0367, .0207; Table 6). Similarly, although the overall model of the association between sleep time variability and waist circumference was significant after controlling for sleep duration,  $F(2, 127) = 6.43, p = .002, R^2 = .092$ , sleep time variability was not significantly associated with waist circumference (95% CI = -.0589, .0890; Table 7).

### **The relation between sleep timing and sleep duration**

After running initial analyses examining the direct association of sleep timing and weight outcomes, it appeared that sleep duration might be a factor affecting the association between sleep timing variables and weight. This was apparent when all PROCESS models were significant despite the non-significant direct association between sleep timing and weight outcomes. Upon closer inspection, sleep duration was significantly related to weight outcomes in the mean sleep time—BMI (95% CI = -.0429, -.0041), mean sleep time—waist circumference (95% CI = -.1345, -.0338), and sleep time variability—waist circumference (95% CI = -.1334, -.0198) models when controlling for the corresponding sleep time variable. Sleep duration was not found to be significant in the mean sleep time—BMI association when controlling for mean sleep time (95% CI = -.0387, .0044; Tables 4-7). Given that prior work has demonstrated a similar relation between sleep timing and sleep duration (Baron, Reid, Kern, & Phyllis, 2011), it was of interest to further explore the relation between sleep timing and sleep duration.

First, we ran the original four parallel mediation models without controlling for sleep duration to examine the direct association between sleep timing and weight. Prior work has conducted similar analyses to disentangle the relation between variables (Baron et al., 2011). When not controlling for sleep duration, sleep time variability was significantly associated with waist circumference (95% CI = .0011, .1257). However, sleep time variability was not associated with BMI (95% CI = -.0039, .1242). Mean sleep time was also not associated with BMI (95% CI

= -.0108, .0102), or waist circumference (95% CI = -.0024, .0472) when sleep duration was removed as a covariate.

Table 4

*Coefficients for Mean Sleep Time Parallel Mediation Model with BMI as Outcome*

Predictor	<i>M</i> <sub>1</sub> (Depressive Symptoms)			<i>M</i> <sub>2</sub> (Anger)			Y (BMI)		
	Coeff.	SE	<i>p</i>	Coeff.	SE	<i>p</i>	Coeff.	SE	<i>p</i>
Constant	3.32	.99	.001	11.44	10.19	.125	50.20	10.19	.000
X (Mean Sleep Time)	.00	.00	.241	.01	.00	.115	-.00	.01	.579
<i>M</i> <sub>1</sub> (Depressive Symptoms)	--	--	--	--	--	--	.72	1.06	.500
<i>M</i> <sub>2</sub> (Anger)	--	--	--	--	--	--	-.12	.14	.369
<i>C</i> <sub>1</sub> Sleep Duration	-.00	.00	<b>.040</b>	.00	.01	.733	-.02	.01	<b>.018</b>
<i>C</i> <sub>2</sub> Education	.03	.02	.229	.23	.18	.133	-.73	.25	<b>.004</b>
	<i>R</i> <sup>2</sup> = .07			<i>R</i> <sup>2</sup> = .04			<i>R</i> <sup>2</sup> = .12		
	<i>F</i> (3,126) = 3.03,			<i>F</i> (3,126) = 1.62,			<i>F</i> (5,124) = 3.47,		
	<b><i>p</i> = .032</b>			<i>p</i> = .189			<b><i>P</i> = .006</b>		

Note. *M*<sub>1</sub> and *M*<sub>2</sub> indicate mediators. *C*<sub>1</sub> and *C*<sub>2</sub> specify covariates. Bolded *p* values designate values < .05.

Table 5

*Coefficients for Sleep Time Variability (IIV) Parallel Mediation Model with BMI as Outcome*

Predictor	<i>M</i> <sub>1</sub> (Depressive Symptoms)			<i>M</i> <sub>2</sub> (Anger)			Y (BMI)		
	Coeff.	SE	<i>p</i>	Coeff.	SE	<i>p</i>	Coeff.	SE	<i>p</i>
Constant	3.96	.47	.000	21.08	3.56	.000	43.56	5.84	.000
X (IIV)	.00	.00	.104	.01	.01	.565	-.74	.25	.373
<i>M</i> <sub>1</sub> (Depressive Symptoms)	--	--	--	--	--	--	.57	1.07	.600
<i>M</i> <sub>2</sub> (Anger)	--	--	--	--	--	--	-.13	.14	.355
<i>C</i> <sub>1</sub> Sleep Duration	-.00	.00	.210	.00	.01	.829	-.02	.01	.119
<i>C</i> <sub>2</sub> Education	.03	.02	.290	.27	.19	.150	-.74	.25	<b>.003</b>
	<i>R</i> <sup>2</sup> = .28			<i>R</i> <sup>2</sup> = .08			<i>R</i> <sup>2</sup> = .13		
	<i>F</i> (3, 126) = 3.48,			<i>F</i> (3, 126) = .88,			<i>F</i> (5, 124) = 3.58,		
	<b><i>p</i> = .018</b>			<i>p</i> = .456			<b><i>p</i> = .005</b>		

Note. *M*<sub>1</sub> and *M*<sub>2</sub> indicate mediators. *C*<sub>1</sub> and *C*<sub>2</sub> specify covariates. Bolded *p* values designate values < .05.

Table 6

*Coefficients for Mean Sleep Time Parallel Mediation Model with Waist Circumference as Outcome*

Predictor	$M_1$ (Depressive Symptoms)			$M_2$ (Anger)			Y (Waist Circumference)		
	Coeff.	SE	<i>p</i>	Coeff.	SE	<i>p</i>	Coeff.	SE	<i>p</i>
Constant	3.32	.99	.000	11.44	7.40	.125	151.61	26.43	.000
X (Mean Sleep Time)	.00	.00	.241	.01	.00	.115	-.01	.01	.620
$M_1$ (Depressive Symptoms)	--	--	--	--	--	--	1.74	2.76	.530
$M_2$ (Anger)	--	--	--	--	--	--	-.26	.37	.474
$C_1$ Sleep Duration	-.00	.00	<b>.040</b>	.00	.01	.733	-.08	.03	<b>.001</b>
	$R^2 = .07$			$R^2 = .04$			$R^2 = .13$		
	$F(3, 126) = 3.03,$			$F(3, 126) = 1.62,$			$F(5, 124) = 3.73,$		
	<b><math>p = .032</math></b>			$p = .189$			<b><math>p = .004</math></b>		

Note.  $M_1$  and  $M_2$  indicate mediators.  $C_1$  specifies covariate. Bolded *p* values designate values < .05.

Table 7

*Coefficients for Sleep Time Variability (IIV) Parallel Mediation Model with Waist Circumference as Outcome*

Predictor	$M_1$ (Depressive Symptoms)			$M_2$ (Anger)			Y (Waist Circumference)		
	Coeff.	SE	<i>p</i>	Coeff.	SE	<i>p</i>	Coeff.	SE	<i>p</i>
Constant	4.12	.44	.000	22.72	3.39	.000	131.33	15.12	.000
X (IIV)	.00	.00	.084	.01	.01	.476	.02	.04	.704
$M_1$ (Depressive Symptoms)	--	--	--	--	--	--	1.32	2.82	.641
$M_2$ (Anger)	--	--	--	--	--	--	-.35	.37	.351
$C_1$ Sleep Duration	-.00	.00	.234	.00	.01	.759	-.08	.03	.009
	$R^2 = .26$			$R^2 = .00$			$R^2 = .10$		
	$F(2, 127) = 4.66,$			$F(2, 127) = .26,$			$F(4, 125) = 3.41,$		
	<b><math>p = .011</math></b>			$p = .772$			<b><math>p = .011</math></b>		

Note.  $M_1$  and  $M_2$  indicate mediators.  $C_1$  specifies covariate. Bolded *p* values designate values < .05.

### **Sleep duration as a mediator in the sleep timing-obesity association**

Given that the preceding analyses suggested that sleep duration might influence the association between particular sleep timing and weight outcomes, additional follow-up analyses were conducted to explore the relation between sleep timing (i.e., mean sleep time and sleep time variability), sleep duration, and obesity. To disentangle the association between sleep timing and sleep duration further, four mediation models were run with BMI and waist circumference as outcomes to examine sleep duration as an indirect pathway between sleep timing and obesity. The aim of this analysis was to determine if sleep timing was related to weight outcomes indirectly through sleep duration given previous work which has identified the impact of late sleep timing on shorter sleep duration (Knutson, 2012). Next, four analyses were run to examine the potential moderating influence of sleep duration on the association between sleep timing and obesity. The aim of the moderation analyses was to determine whether there is an interaction between duration and timing given that previous work has proposed that they operate by similar biological mechanisms. Specifically, short sleep duration and late and inconsistent sleep timing have both been proposed to influence hormonal factors and metabolism to influence weight outcomes (Skeldon et al., 2016). Thus, it was of interest to explore interacting vs. individual effects between sleep timing and sleep duration on obesity.

Bootstrapping analyses were conducted to assess the indirect effect of sleep duration as a mediator of the sleep timing—obesity association. Sleep duration was found to be a significant mediator in the mean sleep time—BMI (95% CI = .0001, .0123; Figure 2, Table 8), mean sleep time—waist circumference (95% CI = .0007, .0378; Figure 4, Table 9), and sleep time variability—waist circumference associations (95% CI = .0079, .1006; Figure 5, Table 9), meaning that later sleep timing led to shorter duration in sleep, which in turn, led to higher BMI

and waist circumference. In addition, more variability in sleep time led to shorter sleep duration and thus, higher waist circumference. Sleep duration was not found to be a significant mediator in the sleep time variability—BMI association (95% CI =  $-.0124, .0438$ ; Figure 3, Table 8).

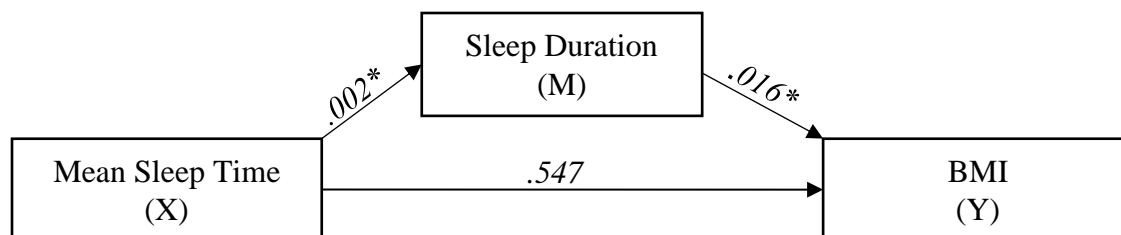


Figure 2. Simple mediation model for the association between mean sleep time and BMI as mediated by sleep duration.  
\* $p < .05$ .

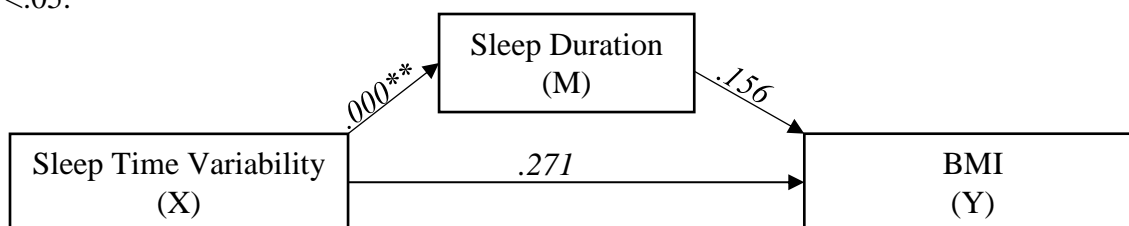


Figure 3. Simple mediation model for the association between sleep time variability and BMI as mediated by sleep duration.  
\* $p < .05$ , \*\* $p < .001$ .

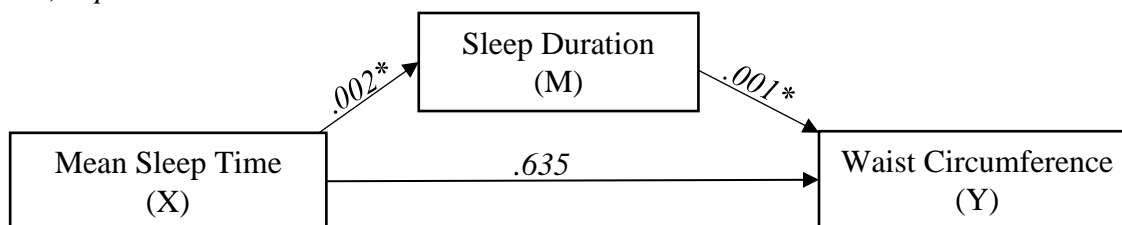


Figure 4. Simple mediation model for the association between mean sleep time and waist circumference as mediated by sleep duration.  
\* $p < .05$ .

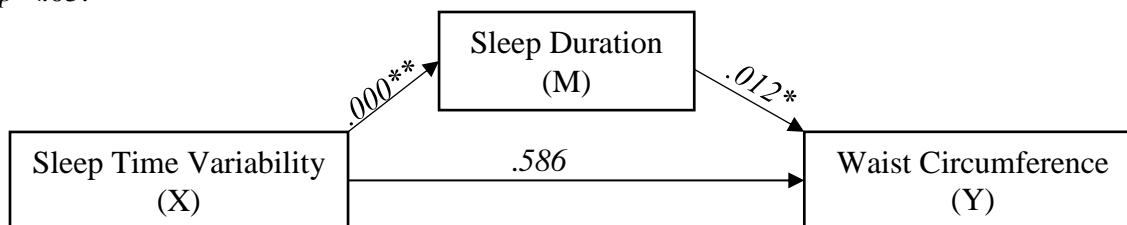


Figure 5. Simple mediation model for the association between sleep time variability and waist circumference as mediated by sleep duration.  
\* $p < .05$ , \*\* $p < .001$ .

Table 8

*Coefficients for Mean Bedtime and Bedtime Variability (IIV) Mediation Models with BMI as Outcomes*

Predictor	<i>M</i> (Sleep Duration) Mean Bedtime-Sleep Duration pathway			<i>M</i> (Sleep Duration) IIV-Sleep Duration pathway			<i>Y</i> (BMI) Mean Bedtime-BMI pathway			<i>Y</i> (BMI) IIV-BMI pathway		
	Coeff.	<i>SE</i>	<i>p</i>	Coeff.	<i>SE</i>	<i>p</i>	Coeff.	<i>SE</i>	<i>p</i>	Coeff.	<i>SE</i>	<i>p</i>
	Constant	594.29	74.72	.000	391.52	15.90	.000	49.66	9.85	.000	41.56	4.52
<i>X</i> (Mean Sleep Time)	-.16	.05	<b>.002</b>	--	--	--	-.00	.01	.547	--	--	--
<i>X</i> (IIV)	--	--	--	-.72	.10	<b>.000</b>	--	--	--	.02	.01	.271
<i>M</i> (Sleep Duration)	--	--	--	--	--	--	-.02	.01	<b>.016</b>	-.02	.01	.156
<i>C<sub>I</sub></i> Education	.14	2.30	.952	1.35	2.03	.507	-.72	.24	<b>.003</b>	-.75	.24	<b>.002</b>
	$R^2 = .07$			$R^2 = .28$			$R^2 = .11$			$R^2 = .11$		
	$F(2, 129) = 4.85,$			$F(2, 129) = 25.31,$			$F(3, 128) = 5.00,$			$F(3, 128) = 5.32,$		
	<b><math>p = .009</math></b>			<b><math>p &lt; .001</math></b>			<b><math>p = .003</math></b>			<b><math>p = .002</math></b>		

*Note.* Two mediation models are represented in table (i.e., mean sleep time—sleep duration—BMI model, sleep time variability—sleep duration—BMI model). Bolded values specify a *p* value < .05 or < .001. *C<sub>I</sub>* indicates the covariate entered into each model.

Table 9

*Coefficients for Mean Bedtime and Bedtime Variability (IIV) Mediation Models with Waist Circumference (WC) as Outcomes*

Predictor	<i>M</i> (Sleep Duration) Mean Bedtime-Sleep Duration pathway			<i>M</i> (Sleep Duration) IIV-Sleep Duration pathway			<i>Y</i> (WC) Mean Bedtime-WC pathway			<i>Y</i> (WC) IIV-WC pathway		
	Coeff.	<i>SE</i>	<i>p</i>	Coeff.	<i>SE</i>	<i>p</i>	Coeff.	<i>SE</i>	<i>p</i>	Coeff.	<i>SE</i>	<i>p</i>
	Constant	595.33	72.38	.000	401.18	6.48	.000	140.02	24.68	.000	125.21	11.23
<i>X</i> (Mean Sleep Time)	-.16	.05	<b>.002</b>	--	--	--	-.01	.01	.635	--	--	--
<i>X</i> (IIV)	--	--	--	-.71	.10	<b>.000</b>	--	--	--	.02	.04	.586
<i>M</i> (Sleep Duration)	--	--	--	--	--	--	-.08	.024	<b>.001</b>	-.07	.03	<b>.012</b>
	$R^2 = .07$			$R^2 = .23$			$R^2 = .08$			$R^2 = .08$		
	$F(1, 130) = 9.76,$			$F(1, 130) = 50.40,$			$F(2, 129) = 5.74,$			$F(2, 129) = 5.78,$		
	<b><math>p = .002</math></b>			<b><math>p &lt; .001</math></b>			<b><math>p = .004</math></b>			<b><math>p = .004</math></b>		

*Note.* Two mediation models are represented in table (i.e., mean sleep time—sleep duration—waist circumference model, sleep time variability—sleep duration—waist circumference model). Bolded values specify a *p* value < .05. No covariates were entered into waist circumference models.

### Sleep duration as a moderator in the sleep timing-obesity association

Next, moderation analyses were conducted to assess whether sleep duration strengthens or weakens the association between sleep timing and obesity (Figure 6). No significant moderating effects of sleep duration were found for the mean sleep time—BMI [-.0002, .0001], mean sleep time—waist circumference [-.0006, .0002], sleep time variability—BMI [-.0002, .0004], and sleep time variability—waist circumference associations ([-.0005, .0010])

### Depressive symptoms and anger as mediators in the sleep timing-obesity association

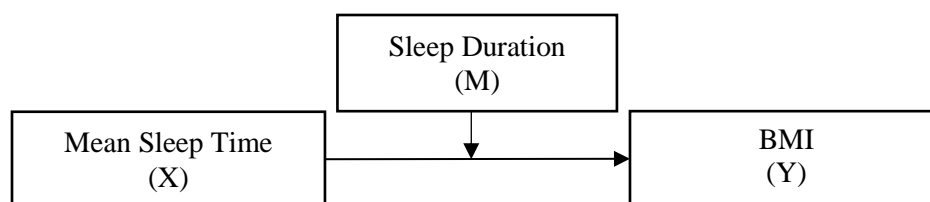


Figure 6. Example of moderation model.

Lastly, bootstrapping analyses were conducted to assess the indirect effect of depressive symptoms and anger as parallel mediators of the sleep timing—obesity association. First, the direct association between sleep timing and emotion was investigated. After controlling for selected covariates, mean sleep time was not associated with depressive symptoms (95% CI = -.0005, .0018) or anger (95% CI = -.0017, .0152; Tables 4 and 6). Similarly, sleep time variability was not associated with depressive symptoms (95% CI = -.0005, .0052) or anger (95% CI = -.0154, .0281; Tables 5 and 7) when controlling for covariates. However, mean sleep time was significantly associated with depressive symptoms (95% CI = .0004, .0024) and anger (95% CI = .0012, .0156), when sleep duration was not included in statistical models. Similarly, sleep time variability was significantly associated with depressive symptoms (95% CI = .0008, .0056) when sleep duration was absent from statistical models.



Next, the direct association between emotional factors and weight outcomes were investigated. When exploring the association between depressive symptoms and weight outcomes, depressive symptoms were not associated with weight outcomes in mean sleep time—BMI (95% CI = -1.3864, 2.8238), mean sleep time—waist circumference (95% CI = -3.7180, 7.1976), sleep time variability—BMI (95% CI = -1.5541, 2.6875) and sleep time variability—waist circumference models (95% CI = -4.2661, 6.9068; Tables 4-7). Similarly, anger was also not associated with weight outcomes in the mean sleep time—BMI (95% CI = -.4085, .1529), mean sleep time—waist circumference (95% CI = -.9915, .4638), sleep time variability—BMI (95% CI = -.4099, .1480) and sleep time variability—waist circumference models (95% CI = -1.0783, .3857; Tables 4-7).

Depressive symptoms and anger were then investigated as indirect pathways linking sleep timing to weight outcomes. Both depressive symptoms and anger were not found to be significant mediators in the mean sleep time—BMI (95% CI = -.0037, .0022), sleep time variability—BMI (95% CI = -.0065, .0115), mean sleep time—waist circumference (95% CI = -.0073, .0061), and sleep time variability—waist circumference associations (95% CI = -.0168, .0291).

## Discussion

The aims of the current study were to 1) investigate whether sleep timing predicts obesity in a sample of midlife women and, 2) investigate whether depressive symptoms and/or anger mediates this relation. The current study did not find evidence that sleep timing is directly associated with poor weight outcomes in midlife women, or that emotional factors mediate the association. Rather, sleep timing indirectly predicted obesity through sleep duration. As there is compelling theoretical rationale for examining sleep timing in relation to weight outcomes in

midlife women, findings from the current study highlight the need to examine potential pathways that link sleep timing to weight outcomes in this population. Although the primary aims of the current study resulted in insignificant associations, the current study utilized empirically strong measures and sound methodological techniques, strengthening previous work in this subject area. As such, the current study highlights methodological considerations for future research. Additionally, as the current results suggest that sleep timing may predict obesity in midlife women through indirect pathways, it is of interest to investigate pathways that may connect sleep timing to weight outcomes in this population.

Regarding the first aim to investigate the association between sleep timing and weight outcomes, the main pattern of findings from the current study indicated that sleep timing does not directly contribute to weight outcomes in this sample of midlife women. Rather, sleep timing emerged as an indirect predictor of weight outcomes through sleep duration. Sleep duration and sleep timing are often correlated, and many studies investigating the association between sleep timing and weight have found that including sleep duration as a covariate blunts the association between sleep timing and weight (Baron et al., 2011; Taylor et al., 2016). Therefore, in follow-up analyses, it was of interest to disentangle this connection. The finding that sleep timing was indirectly related to weight outcomes through sleep duration, indicates that later sleep timing and variability in sleep timing are associated with shorter sleep duration, which, in turn, predicts weight outcomes.

This finding suggests that sleep duration, rather than sleep timing, directly predicts weight outcomes in this population. When examining the pathway between sleep behavior (i.e., sleep duration, sleep timing) and weight outcomes, it is clear that the link between sleep duration and weight has been studied to a greater extent. There is evidence that decreases in sleep

duration are related to increases in appetite, and preference for energy dense food, thus influencing eating behavior and weight gain (Benedict et al., 2012; Nedeltcheva et al., 2009; Spiegel et al., 2004). Alternatively, the link between sleep timing and weight is less clear. First, there is evidence that misalignment in sleep timing influences similar processes as sleep duration (i.e., appetite, hormones, metabolism, eating behavior) which serves to influence weight outcomes (Roenneberg et al., 2012; Scheer et al., 2009; Waterhouse, Buckley, Edwards, & Reilly, 2003). However, it is unclear whether sleep duration was adequately controlled for in many instances, making it challenging to determine if sleep timing alone is associated with poor weight outcomes (Scheer et al., 2009; Waterhouse, et al., 2003). Thus, while sleep timing and sleep duration might influence similar processes, it is possible that the impact on weight is driven primarily through sleep duration, not sleep timing.

In addition to influencing similar processes, it is possible that delayed or more variable sleep timing creates less opportunity for sleep (i.e., contributes to shorter sleep duration), which then influences dietary behavior and weight outcomes (Roepke & Duffy, 2010; Wittman, Dinisch, Mellow, & Roenneberg, 2006). As such, it is not the independent contribution of sleep timing, but its influence on sleep duration that influences appetite, preference for energy dense food, and subsequent weight gain. It is also possible that in addition to creating less opportunity for sleep, late sleep timing leads to more opportunity to engage in poor health behaviors due to increased wake time (e.g., eating late at night and eating less healthful foods) which leads to poor weight outcomes (Stewart & Wahlqvist, 1985; Waterhouse et al., 2003). Accordingly, it is possible that sleep timing alone does not contribute to weight outcomes in midlife women, but instead it is a combination of sleep timing, sleep duration, and alternative biological, social and psychological factors that lead to weight gain and poor weight outcomes.

Although it appears that sleep timing does not directly lead to poor weight outcomes, emotional factors (i.e., depressive symptoms, anger) were not an indirect pathway between the sleep timing and weight association. Despite theoretical reasoning for this association (Yoo et al., 2007; Walker & Van der Helm, 2009), multiple explanations exist for the current findings. First, while previous work has identified the impact of sleep on emotion and the influence of emotions on weight outcomes (Meliska et al., 2011; Shin et al., 2004; Thomas & Donnellan, 1991; Wing et al., 1991b; Weber-Hamann et al., 2006), only a few investigations have identified the connection between these variables in controlled research. This study is one of the first to explore the mediating role of depressive symptoms and/or anger in the sleep timing—weight association, while controlling for potential confounders such as sleep duration and educational level. Although correlations exist between sleep, emotion, and weight in midlife women in the current study, replicating previous work in this area (Araghi et al., 2013; Elder et al., 2012; Häfner et al., 2012; Table 3), it appears that links among sleep timing, emotions, and weight are more complicated when other factors are also considered.

Furthermore, as with previous results, it is possible that sleep duration rather than sleep timing is a more relevant behavior for predicting emotions and weight outcomes in this population. Although the current study did not find that emotion mediated the association between sleep timing and weight outcomes, we did find that sleep timing was associated with emotion in the absence of sleep duration. This pattern of results is similar to those in the sleep timing—weight associations. The role of sleep timing in relation to emotion in midlife women is sparse, with most work exploring correlational associations between variables (Meliska et al., 2011). Although sleep timing may be associated with emotional factors, more work is needed to further disentangle the independent contribution of sleep timing to emotional outcomes, while

controlling for sleep duration. Furthermore, in addition to sleep duration, it is also possible that other factors (i.e., menopausal stage, lifestyle factors, caregiving roles, parental roles) may be associated with variability in sleep timing, emotion, and weight, and may be relevant to include when exploring the connections among these variables in controlled studies of midlife women (Hislop & Arber, 2003). As the current study was not able to control for factors such as menopausal stage or potentially relevant social variables, it would be of interest to include these variables in subsequent studies.

Lastly, although the current study did not uncover a mediating role for emotion in the sleep timing—obesity association, the women in the current study had very low levels of depressive symptoms and low levels of anger, which may have influenced the results by limiting the amount of variability in the sample. Previous work demonstrating a connection between sleep and emotion, as well as emotion and weight, has utilized midlife women with clinical levels of depressive symptoms and higher levels of anger (Clum et al., 2014; Crawford, et al., 2011; Lewis et al., 2009; Simon et al., 2008). Although the current study did find that sleep timing was related to emotion in specific circumstances, emotion was not related to weight outcomes. It is possible that negative weight outcomes are only linked with higher levels of depressive symptoms and anger. Depression and/or anger have been shown to influence caloric intake, eating behavior, and stress levels, all of which influence weight outcomes (Clum et al., 2014; Crawford, et al., 2011). In addition, the use of cross-sectional analyses may have reduced our power to detect a significant association. Previous work has found a connection between emotion and weight in midlife women, but used a longitudinal design (Nelson, et al., 1999; Raikkonen et al., 1999a; Raikkonen, et al., 1999b). It is possible that consistent levels of depressive symptoms and/or anger may be more relevant to explore when investigating the indirect influence of emotion in

the sleep time and weight association. As the current study used retrospective assessment of emotion combined with a seven day sleep protocol, future work examining this association in prospective analyses across time would be of interest to enhance understanding of how sleep, emotions, and weight are related in this population.

In summary, results of the current study suggest that sleep timing may indirectly relate to obesity through alternative pathways. Specifically, sleep duration was identified as a potential mediating factor. This is a novel approach compared to previous work in this area, which has solely investigated the direct link between sleep timing and obesity. However, it is important to note that only a small body of literature has explored even this direct association and, furthermore, this existing small body of literature has produced inconsistent results. Specifically, existing research has shown support both for and against the direct role of sleep timing in relation to weight outcomes. Lee & Shin (2015), for example, found support for an association between consistent late sleep timing and BMI in a sample of middle age and older men and women when controlling for relevant covariates including sleep duration. Baron et al. (2011), conversely, did not find such an association between average sleep time and BMI in a general adult population (age range = 18 - 71). Rather, sleep timing was associated with BMI only when sleep duration was not included as a covariate, indicating that sleep timing alone may not directly contribute to weight outcomes.

In research specifically focused on midlife women, a similar pattern emerges. For example, Taylor et al. (2016) found in a sample of midlife women that sleep timing was not directly associated with BMI outcomes in both cross-sectional and prospective analyses after controlling for covariates including sleep duration. Yu et al. (2015), alternatively, found that midlife women with preference for later sleep timing had higher weight outcomes even when

controlling for sleep duration. Again, although some research highlights the direct and independent role of sleep timing in predicting weight outcomes, other research suggests that timing may not directly predict weight outcomes when accounting for sleep duration.

The current study extends previous work and has considerable strengths in comparison to previous work in this field in that it utilized strong, empirically sound measures including actigraphy, daily measurement of sleep, and objective measures of obesity. The current study also comprehensively investigated multiple measurements of sleep timing (i.e., mean sleep time, sleep time variability) and obesity (i.e., BMI, waist circumference), while examining alternative pathways which may link sleep timing and obesity outcomes. Results of the current study suggest that when accurately and objectively measuring weight and sleep behavior, sleep timing may not be a direct predictor of weight. Rather than investigating the direct link between sleep timing and weight, results of the current study suggest that the investigation of indirect pathways should take precedent, using empirically sound methodology.

The current results highlight the potential role of indirect pathways linking sleep timing and poor weight outcomes. Therefore, it is of use to explore the methodological incongruities in existing research in order to provide an organizing framework to suggest a path forward for future work in this growing area of research. First, as previously noted, only a limited number of studies have explored the connection between sleep timing and weight outcomes. Although a few studies have specifically investigated timing and weight outcomes in midlife women (Taylor et al., 2016; Yu et al., 2015), generalizability is limited and more work is needed to further explore this relation. In addition to the sparse literature on this topic, there are also limits to interpretation due to methodological differences in study design. These differences include variation in the measurement of sleep timing, differences in the operationalization of sleep timing,

inconsistencies in the consideration of weekday and/or weekend sleep timing, and lastly, variations in the measurement of weight outcomes. Examination of these methodological differences is important given that differences in measurement and operationalization of both sleep timing and obesity result in varying outcomes. Thus, having a deeper understanding of discrepancies provides a framework in which to move forward in this area of work.

First, sleep timing can be measured in a variety of ways, including retrospective self-report questionnaires (i.e., recall of typical sleep times), daily sleep diaries, actigraphy, (i.e., ambulatory physiological measure of sleep activity), and chronotype questionnaires (i.e., measure of circadian preferences including sleep time; Lockley, Skene, & Arendt, 1999; Zavada, Gordijn, Beersma, Daan, & Roenneberg, 2005). After an evaluation of the present findings in the context of existing literature, certain trends emerged regarding methodological differences in the measurement of sleep timing. First, research that has found an independent and direct association between sleep timing and weight outcomes when controlling for factors such as sleep duration has utilized self-report measures of average bedtime or general bedtime preference (Lee & Shin, 2015; Yu et al., 2015). Conversely, research that has not found an independent association has utilized daily measures of sleep time in the form of daily sleep diaries or actigraphy (Baron et al., 2011; Taylor et al., 2016). These are important methodological differences and point to the importance of accurately identifying sleep patterns when considering their relation to weight outcomes. Sleep diaries and actigraphy are the gold standard of ambulatory and self-report sleep assessment given their ability to decrease recall bias, provide a more precise estimate of sleep behaviors, and capture fluctuation that can exist across daily sleep behaviors (Martin & Hakim, 2011). The current study is the first to explore the association between sleep timing and weight outcomes in midlife women using actigraphy. Current results are consistent with other findings



investigating sleep timing using daily sleep methodology. It is possible that daily sleep measurement captures sleep more accurately than self-report methods that rely on recall. Moving forward, research investigating the indirect pathways linking sleep timing to obesity should incorporate daily sleep recording methodology such as sleep diaries and actigraphy.

In addition to differences in the measurement of sleep, there are also differences in the operationalization of sleep timing. Sleep timing can be measured via mean sleep time (i.e., an individual's average bedtime across a certain time period) or variability in sleep time (i.e., daily fluctuation in sleep time across days). The majority of work in this area has investigated mean sleep time in samples including midlife women (Baron et al., 2011; Lee & Shin, 2015), and only a very small body of literature has investigated sleep time variability in relation to weight outcomes in this population (Taylor et al., 2016). Variability in sleep timing is important to investigate given that variability encompasses the day-to-day factors that can influence sleep and wake cycles, which are obscured when only the average sleep time is measured (Bei, Wiley, Trinder, & Manber, 2016). Despite research suggesting that irregularity in sleep time increases risk of health problems such as obesity (Patel et al., 2014), variability in sleep time is under-investigated in midlife women. The current study replicated Taylor et al.'s (2016) results by demonstrating that sleep time variability is not directly associated with BMI when controlling for relevant factors such as sleep duration. The current study also adds to the literature, as it is the first to study sleep time variability using actigraphy (compared to daily sleep diaries). This addition is important as actigraphy can capture sleep timing more accurately compared to diary measures (Carney, Lajos, & Waters, 2004). More work is needed to further explore the connection between variability in sleep time and weight outcomes in this population. As women in midlife may be particularly vulnerable to fluctuation in night-to-night sleep behavior due to

due to work, social demands, parental obligations and biological and psychological factors such as having issues initiating sleep (Hislop & Arber, 2003; Owens & Matthews, 1998; Shaver, Johnston, Lentz, & Landis, 2002), exploring this connection is warranted using controlled conditions and prospective analyses.

Besides differences in measurement and operationalization of sleep timing, there is also considerable variability in results depending on whether weekday or weekend sleep timing is examined. In studies that have found an independent and direct association between sleep timing and weight, only weekday sleep was measured (Lee & Shin, 2015; Taylor et al., 2016). Taylor et al. (2016) for example, found that sleep timing variability was independently associated with BMI in a sample of midlife women when only weekday sleep was considered. Lee & Shin (2015) found a similar association between consistent mean weekday sleep time and BMI in a sample of midlife and older men and women. Social jetlag, a condition where weekend sleep is misaligned from weekday sleep, has been shown to influence weight outcomes (Roenneberg et al., 2012), but the independent contribution of weekday sleep to weight outcomes is unclear. This is a potential avenue to explore in midlife women, as it is possible that weekday sleep fluctuation is more crucial than weekend sleep and/or the combination of weekday and weekend sleep in this population. Previous work demonstrates that when sleep timing is at odds with social and work demands, it can lead to circadian misalignment, which can independently influence factors such as metabolism, dietary intake, and hormones such as leptin and glucose (Ruger & Scheer, 2009; Scheer, Hilton, Mantzoros, & Shea, 2009). In midlife women, it is possible that misalignment of sleep timing occurs most often during the week, when work and social schedules are less flexible. Late sleep timing during the week may lead to increases in circadian disruption, leading to independent effects on weight outcomes. As the current study measured sleep timing across

seven days, which included both weekday and weekend sleep, the independent contributions of weekday versus weekend sleep is unknown in the current sample. More work is needed to further explore the independent and differential associations of weekday and weekend sleep timing to weight outcomes in midlife women. This is of particular importance when considering the direct pathway between sleep timing and weight outcomes.

In addition to differences regarding the measurement, operationalization, and consideration of weekday versus weekend sleep, an additional methodological difference is the measurement of obesity. The current study extended existing research by measuring waist circumference, in addition to BMI, as a measure of obesity. Measuring waist circumference when exploring sleep timing in relation to weight is novel, given that most work investigating the association between sleep timing and obesity utilizes BMI as the primary weight outcome measure (Baron et al., 2011; Taylor et al., 2016). Although BMI captures general distribution of body fat, it does not take into account location of body fat. Waist circumference, alternatively, focuses on central obesity, and it is a risk factor for poor health outcomes such as metabolic syndrome (Grundy et al., 2006; Meisinger et al., 2006; Walton et al., 1995; Xu et al., 2011). Waist circumference is of particular importance to investigate in midlife women given that they can experience a redistribution of fat in the midsection during this time (Gurrici et al., 1999; Wells, 2000). Although sleep timing has been linked to metabolic health outcomes in midlife men and women (Lee & Shin, 2015; Taylor et al., 2016), the current study is the first to investigate waist circumference as a weight outcome in the study of sleep timing and obesity in a specific midlife woman sample. Although this study did not find a direct association between sleep timing and waist circumference, it would be of interest to include waist circumference as an outcome in future studies given that circadian misalignment is an important factor

contributing to poor metabolic health outcomes including waist circumference (Delezie & Challet, 2011; Gonnissen et al., 2013).

In summary, an examination of methodological differences in this area of research demonstrates that when studying sleep timing and weight outcomes, the measurement and operationalization of sleep timing and obesity, and the consideration of weekday versus weekend sleep influences results in the association between sleep timing and obesity. As considerable variability currently exists in this area of research, with very few studies specifically examining this association in midlife women, the current study increases knowledge in this area of research using a strong methodological approach. Despite these strengths, a direct association between sleep timing and obesity was not observed. As such, future work is needed to extend current findings in this population using controlled studies and prospective analyses focusing on daily sleep behavior and weight outcomes. Additionally, research investigating indirect pathways linking sleep timing to weight outcomes is warranted given the results of the current study. Specifically, there is a need to use well-validated and accurate measure of daily sleep behavior in a prospective research design when investigating direct and indirect pathways. Additional work should also consider differences between weekday versus weekend sleep.

### **Summary and Implications**

In the investigation of sleep timing as a risk factor for obesity in midlife women, the current study did not find support for sleep timing as a direct predictor of weight outcomes in this population. Rather, sleep timing indirectly contributed to poor weight outcomes through alternative pathways. One pathway identified in the current study is sleep duration. It appears that late sleep timing and sleep time variability contribute to decreases in sleep duration, which then serve to predict weight outcomes. Although the current study investigated emotion as an

alternative pathway by which sleep timing may predict weight, emotion did not link sleep timing and weight. Future research is needed to explore pathways that link sleep timing to weight in this population. As both sleep and weight are of clinical concern for midlife women and link to poor health outcomes, it is imperative to understand pathways of this association in order to determine what modifiable behaviors are potential targets for reducing obesity and weight outcomes in this population. The current results have implications for understanding how the sleep behaviors of midlife women may contribute to weight outcomes. In addition to the known importance of sufficient sleep duration for promoting healthy weight, the current results suggest that delayed or variable bedtimes may also have important implications for weight in this population via sleep duration. As such, research that builds on the current findings is needed that can inform clinical recommendations for sleep behaviors in midlife women as a preventative or intervention approach for maintaining a healthy weight.

### **Strengths and Limitations**

Although the current study has many strengths, several limitations must be addressed. First is the overall lack of racial diversity in the current sample. As a majority of the women are White, the homogeneity of the sample prevents broad generalizability of the results to midlife women of different races and ethnicities. Additionally, due to the use of secondary data, we could not control for potentially relevant covariates. These include both biological factors (i.e., menopausal status, hormonal fluctuation, genetic factors), and psychosocial factors (i.e., parental obligations, eating behavior), which have been shown to influence sleep, emotion, and weight outcomes in midlife women. Thus, the current study cannot address biological mechanisms, menopausal factors, and social factors that may link sleep timing to emotions and weight outcomes in this population. The current study is also cross-sectional, and models used in the

current study are operating in a temporal conceptual framework. As such, causality cannot be determined by the current analyses. Future research using prospective designs would be beneficial for investigating the direct association between sleep timing and weight outcomes as well as the potential influence of emotion and sleep duration as pathways that influence this association. Specifically, repeated concurrent assessment of daily sleep behavior, emotion, and objectively measured weight across time would be particularly useful. In addition, incorporating more clinical and variable levels of emotional disturbances would be important to investigate in future work. The current study assessed emotion (i.e., depressive symptoms, anger) retrospectively and was not able to examine daily fluctuation of emotion. The current sample also had limited variability in terms of depressive symptom and anger scores. This prevents generalization of results to midlife women with more severe levels of mood symptoms and may also influence findings as it is possible that daily emotion is more relevant to explore in this population compared to a composite mean score of emotion. More research is needed to further investigate these factors.

Although the study does contain limitations, there are several strengths. First, the MIDUS II dataset represents a national sample of women. Though limited in racial diversity, the use of this dataset allowed us to examine a broad range of women living in the United States. Second, the use of actigraphy data and daily sleep diaries enabled the examination of sleep timing across multiple days (including weekdays and weekends). As such, we assessed both mean sleep timing and variability in sleep, with reduced recall bias. This is also the first study to assess sleep timing in midlife women using two measurements (i.e., mean sleep time, sleep time variability) with actigraphy which provides a more comprehensive picture of sleep. Third, we used objective measures of BMI and waist circumference. This is the first study to explore the association

between sleep timing and waist circumference in a specific sample of midlife women, which is important given that women in midlife experience a redistribution of fat in the midsection during this time, which is associated with poor health outcomes such as obesity and metabolic syndrome (Gurrichi, et al., 1999; Wells, 2000). Although sleep timing is associated with other indices of metabolic syndrome in this population (i.e., insulin resistance; Taylor et al., 2016), previous work has not exclusively measured waist circumference as a health outcome in this population. The current study was able to extend knowledge provide information regarding the association between sleep timing and waist circumference in this population. Lastly, we used well-validated measures of depressive symptoms and anger in the exploration of emotion as a mediator in the sleep timing—obesity association.

In addition to the sample and measurement strengths, this project extends the current literature in a number of novel ways. This is the first study to investigate emotion as a mediator between sleep and obesity outcomes in midlife women. This is important as all three factors are prevalent symptoms in this population and the emotional mechanisms linking sleep to weight are not fully understood (Nishiura et al., 2010; Schmid et al., 2009). Although the current study did not find a significant mediating effect of emotion in the sleep timing—weight relation, this was potentially due to the limited variability of depressive symptoms and anger in our sample, as well as limitations due to the cross-sectional study design. Despite these limitations, the current study highlights future areas of work, which could help to extend understanding of how emotion is associated with sleep timing and weight in this population. In particular, it would be useful to examine clinical levels of depressive symptoms and higher levels of anger in future investigations. Furthermore, this study investigated multiple aspects of sleep timing in relation to weight outcomes. As sleep timing is a relatively new sleep construct with sparse research

examining its impact on weight outcomes in midlife women, the current study serves to replicate findings from previous work (Taylor et al., 2016) and provide recommendations for future work on this topic.

### **Future Directions**

The current study explored the direct association between sleep timing and obesity in a sample of midlife women and also explored two potential indirect pathways linking sleep timing to weight outcomes (i.e., sleep duration and emotion). In addition to sleep duration and emotional factors, it is possible that other indirect pathways may be relevant to explore in this association. First, prior work demonstrates that sleep timing impacts weight outcomes indirectly through both physiological mechanisms (i.e., metabolism, cravings, hunger; Roenneberg et al., 2012; Scheer et al., 2009) and behavioral mechanisms (i.e., eating behavior, types of food consumed; Stewart & Wahlqvist, 1985; Waterhouse et al., 2003). Prior work demonstrates that later sleep timing and eating late at night impacts weight outcomes, however late timing in the absence of nighttime eating does not independently impact weight (Baron et al., 2011). The current study did not assess eating behavior, so it is unknown how later sleep timing and greater variability in sleep timing impact eating behavior and timing of eating in this population. It is also possible the sleep timing is indirectly related to genetic factors which make one more susceptible to later bedtimes and a higher risk for developing obesity (Turek et al., 2005; Kudo et al., 2007). The current study was not able to control for genetic and/or physiological factors, which would serve to further explore the impact of sleep time on weight. Therefore, future work should examine additional indirect links between sleep timing and weight outcomes. Exploration of the associations among: a) sleep timing, eating behavior, and weight outcomes; b) sleep timing, hormonal and/or physiological factors, and weight outcomes; and c) sleep timing, timing



of eating, and weight outcomes would be of interest to explore in midlife women in controlled studies. It would also be of importance to continue examining the possible role or indirect influence of sleep duration when examining additional factors, given that sleep duration appears to play a role in the association between sleep timing and obesity in midlife women.

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## Appendix A

### MIDUS II Biomarker Project Daily Sleep Diary (Sample of One Diary Entry)

**DATE:** \_\_\_\_\_

**BEFORE BED:** (Please complete the following before going to sleep).

1. How alert were you today? (circle a number): **most alert 1 2 3 4 5 not alert at all**
2. How many minutes of moderate or vigorous exercise did you get today? \_\_\_\_\_
3. Did you nap today? **Yes No** If yes, specify length of time: \_\_\_\_\_ (minutes)
4. How many caffeinated drinks did you have today? \_\_\_\_\_ #of drinks  
(Note: 1 mug of coffee or cup of tea, 1 can pop = 1 drink)
5. How many alcoholic drinks did you have today? \_\_\_\_\_ #of drinks  
(Note: 1 bottle beer or wine cooler, 1 glass of wine, 1 shot of liquor = 1 drink)
6. Did you take any medications today that you don't regularly take every day? **Yes No**  
(e.g. allergy or cold medicine, pain relievers, etc.)  
If yes, record the medication name(s) and dose(s): \_\_\_\_\_

**WEDNESDAY MORNING UPON AWAKENING:** (Please complete soon after waking up, waiting no more than 10 minutes).

7. Did you take any medication or supplement specifically to help you sleep? **Yes No**  
If yes, please record the medication name and dose: \_\_\_\_\_
8. What time did you go to bed and begin trying to go to sleep? \_\_\_[B4AD18]\_\_ a.m./p.m.
9. How long did it take you to get to sleep last night? \_\_\_ (minutes)
10. How difficult was it to get to sleep last night? (circle a number) **very easy 1 2 3 4 5 very difficult**
11. How many times did you wake last night (after falling asleep but before your final awakening)? \_\_\_
12. Did you wake up because of noises, lights, or some other activity? **Yes No**
13. If you woke up during the night, did you have difficulty getting back to sleep? **Yes No**
14. If you woke up during the night, during how many of these awakenings did you get out of bed? \_\_\_
15. What time did you wake up for the day and not return to sleep? \_\_\_\_\_:\_\_\_\_\_ a.m./p.m.
16. What time did you get out of bed for the day? \_\_\_\_\_:\_\_\_\_\_ a.m./p.m.

**Please rate the following:** (circle a number)

17. How deeply you slept last night: **very deeply 1 2 3 4 5 very lightly**
18. How well-rested you feel this morning: **well rested 1 2 3 4 5 poorly rested**
19. How alert you feel this morning: **very alert 1 2 3 4 5 not alert at all**
20. Overall quality of your sleep last night: **very good 1 2 3 4 5 very poor**

## Appendix B

### Center for Epidemiological Studies Depression Inventory (CES-D)

Note: throughout the following “R” indicates item is reverse coded before constructing the scale score.

Items: 20 items – Question 3 (a - t)

“During the past week...?”

- a. “I was bothered by things that usually don’t bother me.”
- b. “I did not feel like eating; my appetite was poor.”
- c. “I felt that I could not shake off the blues even with the help of my family and friends.”
- d. “I felt that I was just as good as other people.”(R)
- e. “I had trouble keeping my mind on what I was doing.”
- f. “I felt depressed.”
- g. “I felt that everything I did was an effort.”
- h. “I felt hopeful about the future.”(R)
- i. “I thought my life had been a failure.”
- j. “I felt fearful.”
- k. “My sleep was restless.”
- l. “I was happy.”(R)
- m. “I talked less than usual.”
- n. “I felt lonely.”
- o. “People were unfriendly.”
- p. “I enjoyed life.”(R)
- q. “I had crying spells.”
- r. “I felt sad.”
- s. “I felt that people dislike me.”
- t. “I could not get “going”.

Coding: 1 = Rarely or none of the time; 2 = Some or a little of the time; 3 = Occasionally or moderate amount of the time; 4 = Most or all of the time.

Scaling: To maintain consistency with the literature, all items were recoded to a 0-3 scale (1=0, 2=1, 3=2, 4=3). Items marked with (R) were then reverse-coded so that high scores reflect higher understanding in the scale. Unless otherwise indicated above, scale scores were computed by summing across all items for which there was no missing data. Mean substitution was used in cases with only one missing value.

## Appendix C

### Spielberger Trait Anger Inventory

Items: 15 items - Question 6 (a - o)

(Circle the number that best describes how you generally feel.)

- a. "I have a fiery temper."
- b. "I am quick tempered."
- c. "I am a hotheaded person."
- d. "I get annoyed when I am singled out for correction."
- e. "It makes me furious when I am criticized in front of others."
- f. "I get angry when I'm slowed down by others mistakes."
- g. "I feel infuriated when I do a good job and get a poor evaluation."
- h. "I fly off the handle."
- i. "I feel annoyed when I am not given recognition for doing good work."
- j. "People who think they are always right irritate me."
- k. "When I get mad, I say nasty things."
- l. "I feel irritated."
- m. "I feel angry."
- n. "When I get frustrated, I feel like hitting someone."
- o. "It makes my blood boil when I am under pressure."

## Vita

Dana Rae Schreiber was born on October 9, 1988 in Philadelphia, Pennsylvania and is an American citizen. She graduated from Upper Dublin High School, Fort Washington, Pennsylvania in 2007. She obtained her Bachelor of Science degree in Psychology from the University of Pittsburgh in Pittsburgh, Pennsylvania in 2011, and subsequently worked on multiple NIH-funded research grants through the University of Pittsburgh and the University of Pittsburgh Medical Center. She began her graduate work in the Counseling Psychology doctoral program at Virginia Commonwealth University in August 2015 under the mentorship of Natalie D. Dautovich, Ph.D.