DISSERTATION

SLEEP AND INSULIN SENSITIVITY IN ADOLESCENTS AT RISK FOR TYPE 2 DIABETES

Submitted by

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ABSTRACT

SLEEP AND INSULIN SENSITIVITY IN ADOLESCENTS AT RISK FOR TYPE 2 DIABETES

Background: Type 2 diabetes (T2D) is a chronic disease that is the 7th leading cause of death in the United States, and rates of adolescent-onset (<20 years of age) T2D are rising. Adolescent-onset T2D is associated with accelerated cardiometabolic comorbidities and shorter life expectancy compared to adult-onset T2D. As traditional behavioral weight loss approaches to T2D prevention show insufficient effectiveness in adolescents, it is critical to investigate novel, potentially modifiable factors that relate to poor insulin sensitivity, a key precursor of T2D. Poor sleep health is one such potentially modifiable contributor to poor insulin sensitivity and consequently, T2D; however, most research on sleep and T2D is in adults, and the specific characteristics of sleep health that relate to poor insulin sensitivity in adolescents at risk for T2D have not been thoroughly investigated. Further, research suggests that individual characteristics related to stress vulnerability, including dispositional mindfulness (i.e., non-judgmental awareness of the present moment) and self-compassion (i.e., treating oneself with an attitude of kindness and compassion), could alter the association of sleep characteristics with insulin sensitivity. In theory, dispositional mindfulness and/or self-compassion may act as a buffer in the association of poor sleep health and metabolic consequences. Thus, the specific research aims of this dissertation project were to determine to what extent objective characteristics of weekday and weekend sleep health, (1a) wake after sleep onset, (1b) sleep onset latency, (1c) time in bed, (1d) sleep duration, and (1e) sleep efficiency, were associated with insulin sensitivity, and (2) to evaluate mindfulness and self-compassion as moderators of the associations between sleep health and insulin sensitivity.

Methods: A total of 128 adolescent girls (*M* ± *SD* age 14.40 ± 1.81 years) at risk for T2D participated in the cross-sectional, baseline phase of a parent study. Sleep disturbances were assessed with actigraphy over one week. Mindfulness was assessed with the Mindful Attention and Awareness Scale and self-compassion with the Self-Compassion Scale. The whole body insulin sensitivity index assessment of insulin sensitivity was determined from a 7-draw, 2-hour oral glucose tolerance test. Linear regressions were used to examine the links between sleep characteristics and insulin sensitivity, accounting for the potentially confounding variables of age, BMIz, race/ethnicity, and puberty. Dispositional mindfulness and self-compassion were tested as moderators of the association between sleep characteristics and insulin sensitivity.

Results: Despite bivariate associations of insulin sensitivity with weekend wake time after sleep onset and weekend time in bed, after accounting for covariates, there were only two trend-level associations. Specifically, longer weekday sleep efficiency was related to greater insulin sensitivity at trend levels, and longer weekend wake time after sleep onset tended to be related to poorer insulin sensitivity at trend levels, accounting for covariates. Mindfulness and self-compassion moderated the associations of weekend sleep efficiency and weekday sleep efficiency, respectively, with insulin sensitivity at trend levels. Higher weekend sleep efficiency was associated with greater insulin sensitivity, only for adolescents with above-average levels of mindfulness. Higher weekday sleep efficiency was associated with greater insulin sensitivity, but only for adolescents with average or above-average levels of self-compassion.

Conclusion: Sleep is an important area for future research in the prevention of T2D in at-risk adolescents. Mindfulness and self-compassion may moderate the associations between

adolescent sleep quality and insulin sensitivity; however, these processes need further investigation. A comprehensive understanding of adolescent sleep will advance knowledge of sleep health, insulin sensitivity, and mindfulness/self-compassion in the prevention of adolescent-onset T2D.

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INTRODUCTION

Sleep and Insulin Sensitivity in Adolescents at Risk for Type 2 Diabetes

Adolescent-onset (<20 years of age) type 2 diabetes (T2D) is a major public health concern, and rates of onset have increased 5% annually over the last 20 years in the United States (Divers et al., 2020; Mayer-Davis et al., 2017). Although traditional T2D prevention approaches focus on improving insulin sensitivity, a key precursor to T2D, through behavioral weight loss, unfortunately, traditional approaches show insufficient effectiveness (Al-Khudairy et al., 2017; Hannon & Arslanian, 2015). Therefore, investigation of novel, potentially modifiable factors that relate to insulin sensitivity is critical to inform alternative T2D prevention approaches in adolescents.

The natural course of insulin dynamics in adolescence makes this age span a sensitive window for understanding potentially changeable factors that influence insulin sensitivity (Alberga et al., 2012; Divers et al., 2020). Poor sleep health may play a role in worsening or decreasing of insulin sensitivity in youth (Cappuccio et al., 2010; Cedernaes et al., 2015; Depner et al., 2014), and therefore is an important component to address in lifestyle T2D preventative approaches in adolescents (Castorani et al., 2020; Chaput et al., 2016; Cree-Green et al., 2019; Dorenbos et al., 2015; Javaheri et al., 2011; Kelly et al., 2010; Leproult & Van Cauter, 2010; Matthews et al., 2012; Wheaton et al., 2016; Zeitler, 2019). However, adolescence is also a particularly vulnerable period for sleep disruptions, and these developmental changes, partially influenced by puberty, can lead to reduced sleep duration and sleep quality, despite adolescents' need for more sleep than adults (Dahl & Lewin, 2002; Keyes et al., 2015; Maslowsky & Ozer, 2014). Concerningly, insufficient and poor-quality sleep during adolescence have been associated with adverse cardiometabolic outcomes (e.g., worse or lower insulin sensitivity) that are risk factors for T2D (Chaput et al., 2016; Dorenbos et al., 2015). Yet, despite the connections of sleep and T2D in adolescents, research specifying dimensions of sleep health that relate to insulin sensitivity in adolescents, including those at risk for T2D, is lacking. Moreover, although sleep health is a multifaceted construct, comprised of sleep duration and disruption (e.g., wake after sleep onset, sleep onset latency, and sleep efficiency,) (Matthews et al., 2012; Simon et al., 2020; Wheaton et al., 2018), the majority of research in adults and adolescents focus on one aspect of sleep, namely, sleep duration, as opposed to utilizing a complex assortment of sleep-related characteristics (Chaput et al., 2023). Thus, understanding how different dimensions of sleep health (i.e., weekday and weekend wake after sleep onset, sleep onset latency, time in bed, sleep duration, and sleep efficiency), measured in the natural/home environment over the course of a whole week, relate to insulin sensitivity in adolescent girls at risk for T2D is needed (illustrated in **Figure 1**), and offers the potential to inform our understanding of how sleep health affects heightened adolescent-onset T2D risk in this population.

Although sleep may affect insulin sensitivity through a variety of pathways, the effect of poor sleep health on reduced insulin sensitivity may be modified by individual-level characteristics (Blake et al., 2016). In particular, research suggests that individual characteristics related to stress vulnerability, including dispositional mindfulness (i.e., non-judgmental awareness of the present moment) (Kabat-Zinn, 2009) and self-compassion (i.e., treating oneself with an attitude of kindness and compassion) (Friis et al., 2015), could alter the association of sleep characteristics with insulin sensitivity reducing the adverse impact of stress (e.g. insufficient or disrupted sleep) on health (e.g. insulin sensitivity) (Creswell & Lindsay, 2014). Thus, as seen in the conceptual models below, we hypothesize that the association between sleep and insulin sensitivity will be moderated by mindfulness (**Figure 2**) and/or self-compassion

(**Figure 3**). Yet, no studies to our knowledge have tested potentially modifiable individual-level characteristics that could mitigate associations of poor sleep health with lowered insulin sensitivity in this population.

In this paper, we will first examine the serious chronic disease of T2D and how biophysiological and behavioral sleep components may contribute to adolescent risk for T2D. Next, we will investigate the roles mindfulness and self-compassion may play in the associations between poor sleep and insulin sensitivity in this adolescent population. Identification of any underlying explanatory and potentially modifiable mechanisms for the association of sleep impairment with T2D among adolescents is essential to promote their future cardiometabolic health.

Serious Public Health Problem of Adolescent-Onset Type 2 Diabetes

Adolescent-onset (<20 years of age) T2D is a major public health problem (Divers et al., 2020; Mayer-Davis et al., 2017). T2D is a serious, chronic disease, affecting over 37 million Americans and costing over $327 billion annually (Centers for Disease Control and Prevention, 2022). T2D is a leading cause of severe health complications including cardiovascular and peripheral vascular disease and stroke, retinopathy and blindness, renal failure, and amputations (Cheung et al., 2010; Moss et al., 1991; Rettig & Teutsch, 1984). Individuals with T2D have a twofold higher risk of mortality than those without T2D, and mortality linked to T2D is the 7th leading cause of death in the United States (Centers for Disease Control and Prevention, 2022). Although T2D was previously limited to older adults, there has been an alarming rise in rates of adolescent-onset T2D that presents a serious healthcare challenge (Divers et al., 2020; Mayer-Davis et al., 2017; Nadeau et al., 2016). Adolescent-onset T2D appears to have an exacerbated disease course with more rapid deterioration, greater health comorbidities, and earlier mortality, as compared to adult-onset T2D, thus underlying the importance of addressing this serious disease in adolescents (Divers et al., 2020; Nadeau et al., 2016; Reynolds et al., 2018; Zeitler et al., 2012).

Adolescence as a Key Period for Targeting T2D

T2D can be potentially averted through preventing worsening insulin sensitivity, a key precursor in the path to T2D (Diabetes Prevention Program Research et al., 2009). Yet, a rise in the earlier manifestations of T2D over the past 50 years unfortunately has paralleled the dramatic increases in pediatric obesity (Ogden et al., 2015; Ogden et al., 2014). These population trends are related, given the established effect of excess adiposity on lowered insulin sensitivity (Franks et al., 2007). Low insulin sensitivity increases the demand on the β-cell (cells in the pancreas that produce insulin), causing deterioration in insulin secretory capacity, and ultimately, β-cell failure in at-risk populations (Reaven, 1988). Low insulin sensitivity is a prospective risk factor for T2D onset in young and middle adulthood, even after accounting for adiposity (Morrison et al., 2010; Nguyen et al., 2008; Nguyen et al., 2010; Reaven, 1988; Thearle et al., 2009). The natural course of insulin dynamics in adolescence makes this age span a sensitive window for the course of insulin sensitivity (Alberga et al., 2012). Puberty is characterized by a transient decline in insulin sensitivity (Kelsey & Zeitler, 2016). Yet, in adolescents at risk for T2D, insulin resistance of puberty is compounded by insulin resistance of obesity, and this high demand for compensatory insulin secretion may cause permanent β-cell failure (Kelsey & Zeitler, 2016). For example, in Hispanic/Latino adolescents with overweight and a family history of T2D, a 24% decline in insulin sensitivity was observed in 1 year (Goran et al., 2006). Those in early puberty showed compensatory increases in acute insulin secretion in response to glucose, but those in late puberty showed worsening of β-cell function (Goran et al., 2006). Thus, pubertal changes are thought to trigger a trajectory toward T2D in at-risk youth (Goran & Gower, 2001; Goran et al., 2006; Moran et al., 1999). Adolescence, therefore, is a key developmental period for understanding changeable factors that may influence insulin sensitivity and, in turn, T2D (Divers et al., 2020).

Treatment for T2D is Challenging

Effective treatment options for adolescent-onset T2D remain elusive, highlighting the critical need for innovative, effective prevention strategies (Fedewa et al., 2014; Hannon & Arslanian, 2015; Zeitler et al., 2012). In adults, the Diabetes Prevention Program, an intensive, structured program of individualized lifestyle modification, reduced the odds of developing T2D 10 years later (Diabetes Prevention Program Research et al., 2009; Knowler et al., 2002). Reduction in T2D risk was achieved through sustained weight loss that improved insulin sensitivity (Kitabchi et al., 2005). In adolescents, lifestyle approaches have demonstrated more limited long-term success (Stice et al., 2006; Whitlock et al., 2008). Despite some positive short-term effects, weight loss is difficult to achieve and sustain in the absence of extraordinary effort (Rosenbaum et al., 2007; Savoye et al., 2014; Savoye et al., 2011; Savoye et al., 2007; Shaw et al., 2009). Further, program adherence is often problematic in adults (Grey et al., 2009; Sigal et al., 2014), and weak sustainability and poor adherence are particularly apparent in the adolescent age group (Knop et al., 2015). Thus, there is a critical need to identify novel, potentially modifiable antecedents to T2D to inform targeted preventative approaches, particularly for racial and ethnic minority adolescent girls who are most affected by adolescent-onset T2D (Divers et al., 2020).

Poor Sleep Health and Type 2 Diabetes Risk

Poor sleep health is gaining attention as a novel, possible risk factor for poor insulin sensitivity and T2D, independent of adiposity, and is of particular concern in adolescent girls at risk for T2D (Cappuccio et al., 2010; Cedernaes et al., 2015; Depner et al., 2014; Koren et al., 2015). Poor sleep health refers to both insufficient sleep, meaning shortened total sleep, and sleep disturbance, referring to shortened, disrupted, and/or mistimed sleep (Koren et al., 2015). Adolescence is a particularly vulnerable period for sleep disruptions (Dahl & Lewin, 2002; Keyes et al., 2015; Maslowsky & Ozer, 2014), and the physical changes of puberty further increase an adolescent's risk for T2D development as puberty is also characterized by a transient decline in insulin sensitivity (Kelsey & Zeitler, 2016). Although research has shown that poor sleep health is related T2D (Cappuccio et al., 2010; Cedernaes et al., 2015; Depner et al., 2014), the majority of research on the connection between sleep health and insulin sensitivity or T2D has been carried out in adults as opposed to adolescents.

Sleep during Adolescence

Sufficient sleep during adolescence is an essential component for healthy development and wellbeing (Short et al., 2013); however, adolescents typically experience shorter sleep duration compared to childhood, and developmental changes in sleep have been attributed to the pubertal transition (Maslowsky & Ozer, 2014). Over two-thirds of U.S. high school students report insufficient sleep (<8 hours) on school nights, and adolescent girls are more likely to report insufficient sleep than boys (Paruthi et al., 2016; Wheaton et al., 2018). In addition to insufficient sleep time, adolescence is also a hallmark period for heightened sleep disturbances, such as increased rates of insomnia, referring to difficulty falling or staying asleep, despite the opportunity to sleep, with daytime impairment (Hysing et al., 2013; Roth, 2007). As with insufficient sleep, sleep disturbances like insomnia appear to be more common in girls than boys. For instance, a study of clinical, diagnostic insomnia, according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria, found that out of the adolescents who met criteria for insomnia at some point during their adolescence, a striking 24% of girls and 13% of boys had a full-syndrome insomnia disorder (Hysing et al., 2013).

Adolescence as a "Perfect Storm"

According to the "Perfect Storm" model, adolescent insufficient sleep may result from evolving psychosocial factors in conjunction with the maturation of sleep bio-regulation systems (Carskadon, 2011). Specifically, the onset of puberty initiates a normative shift in adolescent circadian rhythm that may result in later bedtimes; however, early school start times, academic pressures, and social commitments compound this shift resulting in further insufficient sleep for this already at risk population (Crowley et al., 2018). As adolescence is a key developmental period where youth gain independence to explore their autonomy which involves decision-making surrounding their bedtimes, this increased autonomy at bedtime also often results in later bedtimes and shorter sleep durations (Tashjian et al., 2019). This risk may be further compounded by their high cellphone use and other screen-based electronic devices in the evenings prior to bedtime, which can further offset circadian rhythms resulting in even later bedtimes and less sleep (Mireku et al., 2019; Tashjian et al., 2019).

Concurrently, adolescents experience changes in the sleep/wake homeostasis process that enable them to stay awake longer which is accompanied by a physiological shift in circadian rhythm that delays sleep and wake onset (Crowley et al., 2018). Cortisol, a naturally-occurring steroid hormone that helps regulate multiple body processes including the stress response, naturally secretes in a circadian pattern in which highest concentrations are in the early morning and at midnight (Nader et al., 2010). However, this circadian rhythm, regulated by the central and peripheral nervous systems, is highly sensitive, such that even slightly elevated chronic stress with elevated evening cortisol levels, in conjunction with an increased sensitivity to glucocorticoids, which are a type of steroid hormone that includes cortisol, in the evening, has the potential to influence the development of metabolic challenges (Mousikou et al., 2021; Nader et al., 2010). Poor sleep health also has been proposed to amplify sympathetic nervous symptom activity, which may become heightened in response to the stressor of insufficient or disturbed sleep, and exerts downstream effects on inhibiting appetitive hormones such as leptin, disturbing appetite and satiety regulation that can directly and indirectly, via increased energy intake, lower insulin sensitivity (Dutil & Chaput, 2017). Thus, poor sleep health as a stressor may alter the functioning of the hypothalamic-pituitary-adrenocortical (HPA) axis (i.e., the neuroendocrine system that, with the autonomic nervous system, governs the body's peripheral physiologic stress response) (Chrousos, 2009), consequently dysregulating cortisol output, and thus potentially lowering insulin sensitivity (Adam et al., 2010; Huybrechts et al., 2014; Misra et al., 2008; Prodam et al., 2013; Reinehr & Andler, 2004). Taken together, when these typical biophysiological changes during adolescence are combined with psychosocial and environmental pressures (e.g., social media use, social expectations, academic pressures, socioeconomic status, health disparities, and early school start times), it increases burden on adolescents, may affect their ability to obtain quality, sufficient sleep, and may increase their risk for poor insulin sensitivity (Crowley et al., 2018).

Poor Sleep and Insulin Sensitivity

The prevalence of poor sleep health in adolescent girls is particularly worrisome for adolescents at risk for T2D, as the extant literature supports a connection between objectively and subjectively measured insufficient sleep and low insulin sensitivity in generally healthy adolescents (Chaput et al., 2016; Dorenbos et al., 2015). Findings from an observational, cross-sectional study of high school students indicated that objectively measured shortened sleep, particularly during the weekdays, was associated with lower insulin sensitivity (Matthews et al., 2012). Likewise, in an experimental study of healthy adolescent boys using polysomnography, shortening sleep acutely decreased insulin sensitivity (Klingenberg et al., 2013). Longer actigraphy measured sleep duration, even after adjusting for adiposity, has also been related to higher insulin sensitivity in a community cohort of healthy adolescents (Javaheri et al., 2011) and in a small sample of treatment-seeking adolescents with overweight and obesity (Simon et al., 2019). Further, prior literature, including several meta-analyses, shows that in healthy adults, short sleep and mistimed sleep relate to acute and long-term declines in insulin sensitivity, β-cell function, and a greater risk for T2D onset (Cappuccio et al., 2010; Cedernaes et al., 2015; Depner et al., 2014; Eckel et al., 2015; Jung et al., 2011; Markwald et al., 2013). As research has shown that insufficient and poor quality sleep during adolescence have been associated with adverse cardiometabolic outcomes (e.g., T2D) and key precursors to T2D (i.e., insulin sensitivity and insulin resistance) (Castorani et al., 2020; Chaput et al., 2016; Cree-Green et al., 2019; Dorenbos et al., 2015; Javaheri et al., 2011; Kelly et al., 2010; Leproult & Van Cauter, 2010; Matthews et al., 2012; Wheaton et al., 2016; Zeitler, 2019), thus, these age-related changes in sleep during the high-risk adolescence period highlight the importance of this developmental period for understanding possible effects of sleep health on insulin sensitivity adolescence (Divers et al., 2020; Maslowsky & Ozer, 2014).

More Sleep Measurements are Needed

However, many prior adolescent studies are limited by either reliance on self-reported sleep, which may measure time in bed rather than time spent asleep, or utilization of laboratory paradigms that, although objective, do not capture typical sleep in the natural environment (Lauderdale et al., 2008). For instance, while laboratory paradigms are ideal for objective measurements of sleep, they may not capture real life sleep onset latency, or the amount of time spent in bed prior to falling sleep, and other naturalistic sleep quality characteristics (Sadeh, 2011; Short et al., 2012). Additionally, past research also primarily focuses on the links between a singular aspect of sleep, namely sleep duration, with insulin sensitivity as opposed to utilizing the complex assortment of sleep-related characteristics (i.e., wake after sleep onset, sleep onset latency, time in bed, and sleep efficiency) (Chaput et al., 2023). Although the construct of sleep duration measured with actigraphy may be most in agreement with polysomnography (i.e., the gold standard of sleep measurement), it is still important to investigate these other sleep variables to gain a more comprehensive view of sleep in an at-home setting (Ancoli-Israel et al., 2015; Meltzer et al., 2012). Building upon the existing literature, understanding how different dimensions of sleep health (e.g., weekday and weekend: wake after sleep onset, sleep onset latency, time in bed, sleep duration, and sleep efficiency), measured in a natural, home environment over 7 consecutive days relate to insulin sensitivity in at-risk adolescent girls is needed and offers the potential to inform our understanding of how sleep health may influence health outcomes like T2D risk.

Individual Moderating Influences: Mindfulness and Self-Compassion

Considering the adverse health consequences of poor sleep in adolescents, it is essential to also identify potential protective factors that may buffer the negative effects of poor sleep.

From a biopsychosocial framework in line with psychological stress and coping theory (Lazarus & Folkman, 1984), although sleep may affect insulin sensitivity through a variety of stress-mediated pathways, individual-level differences in stress vulnerability may be protective or aversive to altering the association of sleep health characteristics with insulin sensitivity (Chi et al., 2018; Dunning et al., 2019). Thus, the effect of poor sleep health on reduced insulin sensitivity can potentially be modified by individual-level characteristics, such as mindfulness and self-compassion (Blake et al., 2016). Dispositional mindfulness is the propensity to purposefully pay attention to the present moment with an attitude of non-judgment and equanimity (Kabat-Zinn, 2009). An individual attitude of self-compassion, the propensity to treat oneself with kindness when facing personal difficulties (Neff, 2003), is a related, but distinct construct from mindfulness that also may offer stress-buffering effects with potential to the potential to reduce self-criticism, improve mood, and increase intrinsic self-care motivation (Friis et al., 2016). Enhanced monitoring and self-acceptance of thoughts, emotions, and body sensations as they unfold moment-to-moment is theorized to buffer individuals against adverse effects of stress exposure through a number of mechanisms.

In particular, the mindfulness stress-buffering account posits that the individual trait of higher dispositional mindfulness is protective by reducing the adverse impact of stress (e.g. poor sleep) on physical health (e.g. insulin sensitivity) (Creswell & Lindsay, 2014). In this context, higher dispositional mindfulness and self-compassion have the potential to decrease insulin resistance through their effect on lowering adverse stress-related symptoms; however, mindfulness and self-compassion may improve one's awareness of and changes to health-related behaviors, like sleep, as well as its underlying stress-related physiology (Turner & Hingle, 2017). Another possibility is that mindfulness and self-compassion act directly on reducing stress and mitigating adverse effects of stress physiology (e.g., decreasing stress reactivity and cortisol dysregulation) (Epel et al., 2009). These constructs also have been postulated to indirectly protect against the adverse effects of stress on health outcomes through altering stress-related behaviors (e.g. decreasing stress-eating or eating in the absence of hunger, increasing physical activity) (Schneider et al., 2019).

Mindfulness and self-compassion may not only work at a physiological level but also through supporting cognitive and emotional coping and self-regulatory strategies. Although, the directionality of the association between sleep and mindfulness is currently unknown, according to the meta-cognitive model of sleep, greater mindfulness can potentially change cognitions and emotions around poor sleep (Garland et al., 2015; Ong et al., 2012). Specifically, greater mindfulness may improve sleep by improving cognitions and emotions typically related to sleep difficulties and negative sleep-related thoughts (Garland et al., 2015; Ong et al., 2012). Alternatively, the developmental model of regulation and sleep suggests that insufficient sleep produces emotional, neurobiological, behavioral, and cognitive regulatory deficits, particularly for adolescents, and mindfulness/self-compassion, as meta-cognitive and regulatory processes, may thus be adversely affected by poor sleep (Dahl, 1996; Palmer & Alfano, 2017). Overall, in line with stress and coping theory (Lazarus & Folkman, 1984) and the mindfulness stress-buffering hypothesis (Creswell & Lindsay, 2014), it could be anticipated that adolescents at risk for T2D who have higher mindfulness and higher self-compassion will show dampened associations between poor sleep health characteristics and low insulin sensitivity, whereas teenagers with relatively lower mindfulness and lower self-compassion will show the strongest associations of poor sleep health and low insulin sensitivity.

Mindfulness

Research has suggested that higher dispositional mindfulness in adult samples was associated with lower perceived stress (Brown & Ryan, 2003). Similarly, in an adolescent sample at-risk for adult obesity, a mindfulness-induction was associated with a reduced anxiety response to stress compared to adolescents who received a neutral-induction prior to stress (Miller et al., 2021). Recent research investigating dispositional mindfulness and stress buffering for adolescents has further suggested that high levels of dispositional mindfulness may have a stress buffering or protective “effect,” at trend levels, for the connection between adolescent general perceived stress and internalizing symptoms for specific stressors (Lucas-Thompson et al., 2021).

Other prior mindfulness interventions have shown that increasing mindfulness, compared to active control groups, is related to decreasing depression symptoms in both adolescents and adults (Dunning et al., 2019; Khoury et al., 2013). Further interventions investigating increasing mindfulness, compared to active comparisons, have also found that increasing mindfulness helped to decrease depression symptoms in adolescents in the short-term (Dunning et al., 2019). Furthermore, pilot studies suggest that mindfulness-based interventions have potential to decrease depression symptoms and reduce insulin resistance in adolescents at risk for type 2 diabetes (Shomaker et al., 2019; Shomaker et al., 2017). Similarly, a cognitive-behavioral and mindfulness-based group intervention for adolescents with depression or anxiety, resulted in increases perceived sleep quality (Blake et al., 2017).

Past observational research in adults consistently indicates that those who report being more mindful also report better and/or longer sleep (Bogusch et al., 2016; Brisbon & Lachman, 2017; Howell et al., 2010; Howell et al., 2008). Cross-sectional studies on adolescents also have found that greater mindfulness is associated with higher self-reported sleep quality (Howell et al., 2008; Liu et al., 2018; Murphy et al., 2012). Interestingly, mindfulness-based interventions in young adults have suggested that mindfulness may also improve sleep quality and reduce daytime sleepiness (Bogusch et al., 2016; Caldwell et al., 2010; Howell et al., 2010). Other research in adult samples found similar results in which mindfulness-based interventions improved sleep (Ong et al., 2012) and that individuals randomly assigned to a sleep deprivation condition had lower levels of mindfulness than those in a control condition (Campbell et al., 2018).

Furthermore, self-reported insufficient sleep duration in adolescents with overweight/obesity was related to poorer insulin sensitivity; however these effects were greater among adolescents with lower dispositional mindfulness, compared to average or higher levels of mindfulness, suggesting that an individual's dispositional mindfulness may be potentially modified to mitigate the adverse effects of poor sleep health on lowered insulin sensitivity (Clark et al., 2021). As mindfulness may be trained and has been proposed to improve physical health among populations with high stress, such as adolescents, it would be valuable to increase investigation of mindfulness and sleep in adolescents (Creswell et al., 2019).

Self-Compassion

Self-compassion may influence an individual's psychological health and wellbeing by alleviating the adverse effects of stress (Homan & Sirois, 2017). A meta-analysis investigating the association between self-compassion and psychopathology found that across 14 studies, higher self-compassion was associated with lower depression, anxiety, and stress (MacBeth & Gumley, 2012). Similarly, in a study of adults with chronic illnesses, self-compassion was associated with engaging in more adaptive coping, and less maladaptive coping, which were linked to less stress (Sirois et al., 2019). Higher levels of self-compassion were also associated with lower levels of perceived stress in a sample of college students (Hu et al., 2018). This is in line with another study on adults found that those with higher levels of self-compassion was associated with lower levels of negative affect, higher levels of positive affect, and less perceived stress in daily life (Krieger et al., 2015). Moreover, results from this study also suggested that self-compassion buffered the adverse effect of stress on daily negative affect (Krieger et al., 2015). Similarly, higher self-compassion was significantly associated with lower distress and higher wellbeing in an adolescent sample (Prentice et al., 2021).The extant literature further suggests that self-compassion may buffer the effects of perceived stress in adolescents, such that high self-compassion, as opposed to low self-compassion, was associated with lower levels of perceived stress, depressive symptoms, and anxiety symptoms (Bluth et al., 2016; Lathren et al., 2019).

Self-compassion has been consistently associated with enhanced sleep and physical wellbeing in adults; however, research is lacking on these associations in adolescents. For example, higher levels of self-compassion have also been associated with lower subjective sleep disturbances in adult samples (Kemper et al., 2015). This is consistent with a study of university students which suggested that higher levels of trait self-compassion were positively associated with better subjective sleep quality (Butz & Stahlberg, 2018). Additionally, Butz and Stahlberg (2018) further reported that students who were randomly assigned to practice self-compassion prior to bedtime, compared to a control condition, reported better sleep quality the next morning. Other researchers explained that higher levels of self-compassion buffered the adverse effects of daily stress on subjective sleep latency in a sample of college students (Hu et al., 2018), and a meta-analysis found that adults with higher self-compassion levels reported better self-reported sleep quality (Brown et al., 2021). Moreover, consistent associations of self-compassion with physical health and health promoting behaviors (e.g., exercising and eating nutritiously) exist in adults (Phillips & Hine, 2021), and even data from adults with type 1 diabetes and T2D suggest that self-compassion may be protective against the negative effects of distress in these groups (Friis et al., 2015; Friis et al., 2016). However, further research is needed investigating the associations of self-compassion with sleep and insulin sensitivity in at-risk adolescents.

Current Study

In summary, adolescent girls with high weight and obesity are at high risk for poor sleep health, low insulin sensitivity, and consequently, adolescent-onset T2D. Data from adults and a small body of data from healthy adolescents support a connection between poor sleep health and low insulin sensitivity and T2D risk. Yet, there is a dearth of research on sleep health in adolescents at risk for T2D, and no studies testing potentially modifiable individual-level characteristics that could mitigate adverse effects of poor sleep health on lowered insulin sensitivity in this population.

Therefore, the aims of this dissertation were to determine to what extent characteristics of weekday and weekend sleep health, including (1a) wake after sleep onset, (1b)sleep onset latency, (1c) time in bed, (1d) sleep duration, and (1e) sleep efficiency), are associated with insulin sensitivity in adolescent girls at risk for T2D and 2) to evaluate mindfulness and self-compassion as moderators of the associations of sleep health dimensions with insulin sensitivity in teenage girls at risk for T2D. Findings will deepen our understanding of the role of sleep in T2D risk among a group at risk for adolescent-onset T2D and potentially inform future, preventative interventions targeting sleep for adolescent T2D prevention.

METHODS

Overview of the Study

Data for this project are derived from a larger National Institute of Diabetes, Digestive and Kidney Disease (NIDDK) parent study (Shomaker et al., 2018) with distinct aims from the current dissertation project. The parent study (R01DK111604) is an ongoing, active randomized controlled trial with adolescent girls at risk for T2D. Although there is a longitudinal component to the larger study, I utilize only the baseline dataset in order to test my specific aims. I determine the characteristics of sleep health associated with insulin sensitivity in N=128 adolescent girls at risk for T2D (Aim #1) and evaluate the individual attributes of mindfulness and self-compassion as possible moderators of the associations between sleep health dimensions and insulin sensitivity (Aim #2).

Participants and Recruitment

Adolescents are girls, ages 12-17 years, who are predisposed to low insulin sensitivity and heightened T2D risk based upon having overweight/obesity (BMI $\geq 85^{th}$ percentile) (Ogden et al., 2002) and a family history of T2D (Shomaker et al., 2018). Also, all participants have some degree of elevated depressive symptoms, determined by a Center for Epidemiologic Studies-Depression Scale (CES-D) total score >20 (Stice et al., 2009), a frequent cut-point used to identify “at-risk” adolescents (Garber et al., 2009; Stice et al., 2008). This sample characteristic maximizes the likelihood of observing a range of sleep disturbances, given that depression and sleep health are inter-connected (Lovato & Gradisar, 2014). We exclude, and provide referrals, to any participant with active suicidal ideation or with a current DSM-5 psychiatric disorder that would impede compliance and necessitate more intensive treatment, including major depressive disorder, substance abuse, psychosis, bipolar disorder, conduct disorder, panic disorder, and obsessive-compulsive disorder. Likewise, adolescents with T2D or another major medical illness are excluded and referred to their primary care physician. Further, youth taking medications potentially affecting mood, weight, insulin sensitivity, or cortisol secretion (e.g., stimulants, anti-depressants, anti-psychotics, insulin sensitizers, or chronic steroids) are excluded. Finally, adolescents with an active pregnancy or who are breastfeeding are excluded.

Key recruitment methods are: i) outreach to patients receiving care at Children's Hospital Colorado identified through the electronic medical record as potentially eligible, ii) advertisements on local school parent Listservs, iii) notices to community research networks in surrounding areas; and iv) direct mailings to area families with adolescent daughters. The total sample to be recruited for the parent study is N=150. As of March 2023, we have enrolled N=128 eligible adolescents.

Procedure

Study procedures reported in this dissertation were carried out at the outpatient Pediatric Clinical and Translational Research Center (CTRC) at Children's Hospital Colorado or remotely, at participants' homes. Prior to the first screening visit, a phone screen was used to evaluate initial eligibility, and adolescents were directed to complete a depression symptoms screener. Families meeting initial eligibility criteria were invited to complete an in-person screening and baseline measurement visit, collected during two outpatient research visits to the CTRC.

At the first screening visit, all participants and parents/guardians had the study described to them in detail by a trained study staff member and signed IRB-approved assent/consent forms, respectively, prior to participation. Enrolled participants were then instructed to fast starting at 10:00 pm the night prior to a second, in-person visit. Between the two laboratory screening visit appointments, participants completed ambulatory measures of sleep. At the second laboratory screening, they completed an oral glucose tolerance test (OGTT), surveys/questionnaires, interviews, and dual-energy x-ray absorptiometry (DXA).

Due to the COVID-19 pandemic, for a period of time adolescents and their parent/guardian were sent remote REDCap consents and had the study described to them in detail in a remote study visit via Vidyo to provide informed consent/assent, determine eligibility, complete surveys, and participate in a mental health interview. Participants were then still instructed to return for a second study visit at the hospital to complete an OGTT during COVID-19. Adolescents were financially compensated for their time.

Measures

Primary Outcome Variables

Sleep Disturbance.

Actiwatch (Spectrum Plus, Phillips), a small device worn on the nondominant wrist continuously for 7 days, was used to objectively quantify key dimensions of sleep health including weekday and weekend: wakefulness after sleep onset (the amount of time a person spends awake between fully asleep to fully awake], sleep onset latency [the amount of time to go from fully awake to fully asleep], time in bed [total time spent in bed], sleep duration [total amount of sleep], sleep efficiency [the percentage of time spent in bed fully asleep], and social jetlag [the difference in sleep amount between weekdays and weekends]) (Walia & Mehra, 2019). Following standard guidelines to increase compliance, participants maintained a concurrent daily sleep log reporting their bedtimes and wake times. Sleep start and wake times were manually selected to facilitate accurate scoring of sleep/wake episodes (Ancoli-Israel et al., 2015). Parent reports and adolescent reports of sleep duration show weak associations with objective sleep measures (Arora et al., 2013; Dayyat et al., 2011). In contrast, Actiwatch has good concordance with polysomnography and has less subject burden than laboratory sleep studies (Cellini et al., 2013). Objective sleep measures have been related to insulin sensitivity in healthy adolescents (Matthews et al., 2012). Using standard protocols, Actiwatch data, in conjunction with daily diary logs, were cleaned and prepared for analysis (Ancoli-Israel et al., 2015).

Insulin Sensitivity.

An oral glucose tolerance test (OGTT) is a well validated, yet less invasive, costly, and labor-intensive approach than clamps or IVGTT. OGTT yields more valid estimates of insulin sensitivity than fasting values only (Eldredge & Agras, 1997). Following a 10-hour overnight fast, participants receive 1.75 g/kg of glucola (max=75g). Using an intravenous line, blood was sampled for insulin, glucose, and C-peptide at fasting, 10, 20, 30, 60, 90, and 120 minutes after glucola. Insulin sensitivity was estimated as whole body insulin sensitivity index (WBISI) (Matsuda & DeFronzo, 1999), which has excellent convergent validity with clamps in youth who have obesity but who do not have diabetes (Yeckel et al., 2004).

Dispositional Mindfulness.

Participants complete the Mindful Attention and Awareness Scale (MAAS)-Adolescent Version – a 15-item questionnaire of general, dispositional mindfulness, in which one assesses their present-moment experiences in everyday activities (Brown & Ryan, 2003). Items are rated on a Likert-scale from “1” (almost always) to “6” (almost never) with questions such as “I find it difficult to stay focused on what’s happening in the present.” The total score is the sum of all items, with higher scores indicating relatively higher, or more positive, dispositional mindfulness (Brown & Ryan, 2003). In a systematic review, the MAAS demonstrated reliability and validity in adolescent samples (Brown et al., 2011).

Self-Compassion.

Participants completed the Self-Compassion Scale (SCS) to assess the individual attribute of self-compassion (Neff, 2003). The 26-item SCS generates 6 subscales: self-kindness, self-judgement, common humanity, isolation, mindfulness, and over-identification. In response to the probe, "how I typically act towards myself in difficult times," participants are asked to indicate how often they behave in a certain manner (e.g., "I'm disapproving and judgmental about my own flaws and inadequacies") using a Likert-scale response rating from "1" (almost never) to "5" (almost always). Scores are computed by calculating the mean of subscale item responses, and a total score also is calculated as the sum of all items, with relevant items reverse-scored (Neff, 2003). Higher total scores indicate greater self-compassion. The SCS is reliable and has been validated in adolescent samples (Cunha et al., 2016).

Control Variables

BMI Indices.

BMI (kg/m^2) was derived from height in triplicate by stadiometer and fasting weight by calibrated digital scale. BMI z-score and BMI %ile (Ogden et al., 2002) were computed to determine eligibility (BMI ≥85th percentile).

Body Composition.

Dual-energy x-ray absorptiometry (DXA) was conducted to assess total fat/lean mass using Hologic QDR Discovery A (S/N81337; Bedford, MA). DXA offers a gold-standard for fat distribution assessment with strong predictive validity for youths' obesity-related co-morbidities (Bauer et al., 2012).

Health History/Puberty.

A brief psychiatric and medical health history, including T2D history, of the participant and family was conducted with a parent/guardian to determine eligibility. Physical examination, including blood pressure in triplicate and waist circumference, was conducted to evaluate adolescent health status. Tanner staging for breast and pubic hair was performed by a pediatric endocrinologist.

Perceived Stress.

Participants completed the Perceived Stress Scale (PSS) (Cohen, 1988; Kupst et al., 2015). This scale is a 10-item questionnaire that measures perceptions of stress. Items are rated on a 5-point Likert-scale from "0" (Never) to "4" (Very Often) with questions such as, "In the last month, how often have you been upset because of something that happened unexpectedly?" The total score is calculated as the sum of all items, with higher scores indicating greater stress. This measure has demonstrated good internal reliability in adolescent samples (Liu et al., 2020; Quach et al., 2016).

Depressive Symptoms.

Participants completed the Center for Epidemiologic Studies-Depression Scale (CES-D) to determine eligibility (total score >20) and to assess depressive symptoms. The 20-item CES-D is a widely-used continuous measure of depression symptoms experienced over the past week (Radloff, 1977). Each item is scored on a Likert scale from "0" (rarely or none of the time; less than 1 day) to "3" (all of the time; 5-7 days) with items such as, "I was bothered by things that usually don't bother me." The total score is calculated as the sum of all items, with higher values indicative of greater depression symptomatology (Radloff, 1977). The CES-D total score is reliable and validated in adolescent samples (Phillips et al., 2006).

Data Analysis

Data were analyzed using SPSS for Windows, version 28 (IBM Corporation, Armonk, NY, USA). Preliminary analyses involved data cleaning (e.g., checking for outliers and normality) followed by understanding simple bivariate associations in the data.

Aim and Hypothesis #1

Aim 1 was to determine characteristics of sleep health associated with insulin sensitivity in adolescent girls at risk for T2D (**Figure 1**). For both weekday and weekend sleep indices, greater wakefulness after sleep onset (1a), longer sleep onset latency (1b) greater alterations in time in bed (1c), greater alterations in sleep duration (1d), and reduced sleep efficiency (1e), were expected to be associated with lower insulin sensitivity, even after accounting for age and adiposity, as well as race/ethnicity and pubertal stage. To examine this first aim, the associations between different characteristics of sleep disturbance with insulin sensitivity, linear regressions adjusted for age, BMIz, race/ethnicity, and puberty were performed. Age, BMIz score, race/ethnicity, and puberty were included a priori as covariates, due to their known relationships with sleep and insulin sensitivity and to ensure that results were independent of these variables.

The independent variables (IVs) were weekday and weekend, wakefulness after sleep onset (1a), sleep onset latency (1b), time in bed (1c), sleep duration (1d), and sleep efficiency (1e). The dependent variable (DV) was insulin sensitivity. Covariates were entered in the first level of the regression. Next, the IVs were examined first in isolation (separate regression models), and then, significant IVs were included in the same regression model to determine the unique associations of each sleep characteristic with insulin sensitivity. Finally, we also adjusted for perceived stress and depression as a sensitivity analysis, as negative mood (e.g., stress depressive symptoms) could be confounders of the association between sleep and insulin sensitivity.

Aim and Hypothesis #2

To address the second study objective, moderation models (PROCESS Model 1; **Figure 2 and 3**) were used to test if the associations of poor sleep health characteristics and insulin sensitivity were moderated by mindfulness (**Figure 2**) and self-compassion (**Figure 3**), adjusted for age, BMIz, race/ethnicity, and puberty, using the PROCESS SPSS macro by Hayes (v3.0; 2018) which uses a product-of-coefficients approach with 5,000 bias-corrected bootstrapped estimates to assess the significance of the indirect association or intervening variable (Preacher & Hayes, 2004). Age, BMIz, race/ethnicity, and puberty were included a priori as covariates, due to their known relationships with sleep and insulin sensitivity and to ensure that results were independent of these variables. Analyses proceeded from simple to more complex. Each moderator was tested singularly. Likewise, all sleep health IVs were tested separately. Following standard procedures, in order to interpret the meaning of any significant moderation findings, conditional associations were examined at low (-1 SD below the mean), average (mean), and high (+1 SD above the mean) values of dispositional mindfulness and self-compassion (Preacher et al., 2007).

Power Analyses

For this dissertation, power analyses were conducted based upon the primary aim #1. To calculate power, PASS software (NCSS, LLC, Kaysville, Utah) was used. Given the sample size of 128 participants, the detectable effect size (R^2) in multiple regression corresponding to 80% power was calculated. Assuming that the R^2 between the sleep outcome IV and the covariates of age, adiposity, puberty, and depression is equal to 0.273, a sample size of 120 results in a detectable increase in the R^2 equal to 0.043 using a test with a significance level of 0.05 at 80% power. Throughout the results, standardized coefficients are included as an estimate of effect size effects (small effects = 0.2; medium effects = 0.5; large effects = 0.8) (Cohen, 1988).

RESULTS

Descriptive Information

A total of N = 128 adolescent females ($M \pm SD$ age 14.40 ± 1.81 years) participated in the study. Study sample descriptive information on socio-demographic characteristics and key variables is provided in **Table 1**. Eighty-two percent of the sample reported insufficient weekday sleep (<8 hours) and 64.2% reported insufficient weekend sleep (<8 hours).

Bivariate Correlations between Key Variables

Table 2 summarizes the results from the Pearson bivariate correlations that describe cross-sectional associations among key variables. There was one trend level association and two significant associations between the adolescent sleep variables and insulin sensitivity. Specifically, there was a trend level, small negative association between adolescents' weekday time in bed and insulin sensitivity (r = -.18, p = .05), meaning that longer weekday time in bed tended to be related to lower insulin sensitivity. Weekend wake time after sleep onset and insulin sensitivity were inversely correlated with each other (r = -.23, p = .01), such that longer weekend wake time after sleep onset was related to lower insulin sensitivity (small effect). Finally, weekend time in bed and insulin sensitivity were negatively correlated with each other (r = -.19, p < .05), such that longer weekend time in bed was related to lower insulin sensitivity (small effect).

Mindfulness was significantly, inversely associated with perceived stress (r = -.43, p < .001; moderate effect) and self-compassion (r = .38, p < .001; small-to-moderate effect). Self-compassion was significantly, positively correlated with weekday wake time after sleep onset (r = .20, p = .03; small effect), weekday time in bed (r = .24, p = .01; small effect), weekday sleep duration (r = .24, p = .01; small effect), weekend time in bed (r = .20, p = .03; small effect), weekend sleep duration at trend levels ($r = .18$, $p = .05$; small effect), and inversely correlated with perceived stress ($r = -.52$, $p < .001$; moderate effect) and depression ($r = -.27$, $p < .01$; small effect).

Regression Analysis with Insulin Sensitivity

A series of linear regression analyses adjusting for age, BMIz, race/ethnicity, and puberty was performed to examine the associations of sleep indices with adolescent insulin sensitivity, after adjusting for these covariates. A summary of the findings is shown in **Table 3** (see below). The entry order into the regression equations was as follows. In the first step, the covariates for insulin sensitivity (i.e., age, BMIz, race/ethnicity, and puberty) were entered. The model yielded a significant finding for the overall association of the covariates, $F(4, 118) = 10.48$, $R^2 = .26$, $p < .001$, with both age ($B = .19$, $p = .03$; small effect) and BMIz ($B = -.36$, $p < .001$; moderate effect) contributing significantly to variability in adolescent insulin sensitivity. Conversely, race/ethnicity ($B = .09$, $p = .26$; small effect) and puberty ($B = .12$, $p = .16$; small effect) were not significantly associated with adolescent insulin sensitivity, accounting for age and BMIz in the model. The total amount of variance accounted for by the covariates combined was 26.2%, adjusted $R^2 = .24$.

In the second step, adolescent sleep variables (i.e., weekday and weekend: wake after sleep onset, sleep onset latency, time in bed, sleep duration, and sleep efficiency) on adolescents' insulin sensitivity were examined one at a time. After controlling for the covariates, no associations of sleep indices with insulin sensitivity were significant.

There were two trend-level associations. After controlling for the covariates, weekday sleep efficiency tended to be related to variability in insulin sensitivity, $F(1, 114) = 2.87$, $B = .14$, $\Delta R^2 = .02$, $p = .09$, such that greater weekday sleep efficiency tended to be related to greater (better) insulin sensitivity (small effect). Similarly, adolescent weekend wake time after sleep onset, after controlling for the effect of the covariates, tended to be associated with insulin sensitivity, $F(1, 109) = 2.82$, $B = -.15$, $\Delta R^2 = .02$, $p = .09$, such that longer weekend wake time after sleep onset tended to be related to poorer insulin sensitivity (small effect). Overall, however, these non-significant results indicated that the relation of the covariates (i.e., age, BMIz, race/ethnicity, and puberty) may be more related than sleep indices on adolescent insulin sensitivity.

Sensitivity Analyses with Depression and Perceived Stress

As a sensitivity analysis, we evaluated the similarities/differences if depression and perceived stress were entered separately in Step 3 with the sleep and adolescent insulin sensitivity relationship (**Table 3**). No sleep index became significant after controlling for perceived stress or depression in the model. Additionally, depression ($B = -.02$, $\Delta R^2 = .00$; $p = .82$) and perceived stress ($B = .11$, $\Delta R^2 = .01$; $p = .20$) did not relate to insulin sensitivity. Thus, as depression and perceived stress were not significant contributing variables to insulin sensitivity, we chose to not include these as control variables.

Moderation Analyses

A set of analyses was performed using Hayes' (2021) PROCESS macro to investigate whether mindfulness and self-compassion moderated the associations of sleep indices with insulin sensitivity in adolescents, covarying for age, BMIz, race/ethnicity, and puberty. Moderating effects were indicated when the interaction term, the product of sleep and mindfulness or the product of sleep and self-compassion, was significant. Only the models including the weekend sleep efficiency x mindfulness interaction term and the weekday sleep efficiency x self-compassion interaction term were at trending levels of significance in the regression predicting insulin sensitivity. All other model's moderation interaction terms were not significant.

Mindfulness as a Moderator

A summary of the moderation results with dispositional mindfulness as a moderator is provided in **Table 4a**. Dispositional mindfulness moderated the association between weekend sleep efficiency and insulin sensitivity at trend level ($\Delta R^2 = .02$, $b = .004$, $SE = .002$, $p = .09$; small effect). Further, as in the linear regression model, weekend sleep efficiency and insulin sensitivity were not significantly related ($p = .15$).

To probe the meaning of this trending interaction, we further examined the conditional relationships between weekend sleep efficiency and insulin sensitivity at different levels of dispositional mindfulness (**Table 4b; Figure 4**). For participants with above-average levels of dispositional mindfulness (total score = 70, with possible range of 15 [least mindful] to 90 [most mindful]), the slope of weekend sleep efficiency and insulin sensitivity was significantly different from zero ($b = 0.09$, $SE = 0.04$), $p < .05$. For participants with average levels of mindfulness (total score = 57), the slope was not significantly different from zero ($b = 0.04$, $SE = 0.03$), $p = .18$. Participants with lower-than-average levels of mindfulness (total score = 45) also did not have a slope of weekend sleep efficiency and insulin sensitivity significantly different from zero ($b = -0.01$, $SE = 0.04$), $p = .76$. These results suggest that a higher level of weekend sleep efficiency was associated with greater insulin sensitivity, only for adolescents with above-average levels of mindfulness (see **Figure 4**).

Self-Compassion as a Moderator

A summary of the moderation results with self-compassion as a moderator is provided in **Table 5a**. Self-compassion only moderated the association between adolescent weekday sleep efficiency and insulin sensitivity at trending levels of significance (ΔR^2 = .02, *b* = 0.14, *SE* = 0.07, $p > .05$; small effect). Further, as in the first regression model, weekday sleep efficiency and insulin sensitivity were not significantly related ($p = .12$).

To further investigate the meaning of this trending interaction effect, we examined the conditional relationships between weekday sleep efficiency and insulin sensitivity at different levels of self-compassion (**Table 5b; Figure 5**). For participants with average levels of self-compassion (total score = 2.92, with a possible range of 1 [lowest self-compassion] to 5 [highest self-compassion]), the slope of weekday sleep efficiency and insulin sensitivity was significantly different from zero (*b* = 0.10, *SE* = 0.04), $p = .02$. Participants with above average levels of self-compassion (3.35) also demonstrated a slope significantly different form zero (*b* = 0.16, *SE* = 0.06), $p = .01$. However, for participants with below-average levels of self-compassion (2.23), the slope of weekday sleep efficiency and insulin sensitivity was not significantly different from zero (*b* = 0.01, *SE* = 0.05), $p = .90$. These results indicate that a higher level of weekday sleep efficiency was associated with greater insulin sensitivity for adolescents with average or above-average levels of self-compassion (see **Figure 5**).

DISCUSSION

Many adolescents do not obtain sufficient sleep, a factor that is associated with an increased risk for adverse health outcomes such as T2D. Indeed, results showed that 82% of adolescent participants reported insufficient sleep (<8 hours) on school nights, which is slightly above the U.S. average (~70%) of insufficient sleep in adolescents (Paruthi et al., 2016; Wheaton et al., 2018). In this project, we evaluated the associations of a comprehensive battery of objectively assessed sleep health indices (i.e., weekday and weekend: wake after sleep onset, sleep onset latency, time in bed, sleep duration, and sleep efficiency) with insulin sensitivity in teenagers at-risk for T2D. Further, we investigated if mindfulness and self-compassion moderated these relationships. These interpretations must be undertaken with caution as they are for the purposes of hypothesis generation.

Hypothesis 1: Sleep and Insulin Sensitivity

We predicted that greater alterations in weekday and weekend (1a) greater wakefulness after sleep onset, (1b) longer sleep onset latency, (1c) reduced time in bed, (1d) shorter sleep duration, and (1e) reduced sleep efficiency would be associated with lower insulin sensitivity, even after accounting for age, adiposity, race/ethnicity, and pubertal stage. There were significant bivariate inverse associations between sleep variables of weekend wake time after sleep onset and weekend time in bed and insulin sensitivity. However, after adjusting for covariates that are highly related to insulin sensitivity (i.e., age, BMIz, race/ethnicity, puberty), no sleep variable was significantly related to insulin sensitivity. Instead, in models accounting for these covariates, weekday sleep efficiency and weekend wake time after sleep onset were related to insulin sensitivity at trending levels of significance. Thus, in this sample of adolescents at-risk for T2D with mood concerns, the associations of measured sleep behavior (e.g., weekday time in bed) with insulin sensitivity primarily were accounted for by BMIz. This finding is in agreement with a study on adult populations that found an association between total sleep time and insulin and HbA1c levels only in models not adjusted for BMI (Ford et al., 2014), suggesting that BMI may be a intervening factor. A study on preadolescent children found similar results indicating that longer sleep duration and better insulin sensitivity were partially mediated through adiposity (Alves et al., 2022). This lack of an association between sleep and insulin sensitivity, independent of BMIz, could also be due to the specificity of our sample that included youth with a heighted T2D risk and mood concerns, many of whom had heightened insulin resistance/poor insulin sensitivity, which perhaps limited the utility of variations in sleep to explain variability in insulin sensitivity. Additionally, some data suggest a U-shaped relationship of sleep duration and insulin sensitivity in adolescents as well as adults, with both too short and too long sleep being problematic for metabolic health (Javaheri et al., 2011; Koren et al., 2011), indicating the need for further exploration into this relation. It is also possible that there could be more complicated pathways at work that our analyses were not able to detect, such as through stress-related physiology (e.g., excess cortisol), and stress-related behavioral factors (e.g., emotional eating) (Bjorntorp, 2001; Pervanidou & Chrousos, 2012; Rosmond, 2003; Stetler & Miller, 2011; Stuart & Baune, 2012), or appetite-regulating hormones (Dutil & Chaput, 2017). We also predicted that depression and stress would be important covariates in the sleep and insulin sensitivity association; however, these variables were not significantly associated, suggesting that depression and stress may work through more complicated mechanisms in this relationship. Thus, it may be beneficial for future work to investigate further pathways and mechanisms of the sleep and insulin sensitivity association in adolescence using more varied measures of mood and stress.

Despite overall small effect sizes and the lack of significant findings, it is worth noting that directions of the trending level relationships were in alignment with our initial hypothesis, which suggest that higher weekday sleep efficiency (better sleep quality) is related to better insulin sensitivity whereas higher weekend wake time after sleep onset (worse sleep quality) is associated with worsened insulin sensitivity. These findings add to our understanding of objectively measured naturalistic sleep with multiple additional dimensions of sleep behavior (i.e., weekday and weekend: wake after sleep onset, sleep onset latency, time in bed, sleep duration, and sleep efficiency) and how they may relate to insulin sensitivity. Moreover, the sleep variables at trending levels in this study, namely weekday sleep efficiency and weekend wake after sleep onset, are similar constructs to those used in the extant literature; however, much of this research includes sleep duration as the primary outcome. These trending results are similar to prior literature, including several meta-analyses, that suggest in healthy adults, short sleep and mistimed sleep relate to acute and long-term declines in insulin sensitivity, β-cell function, and a greater risk for T2D onset (Cappuccio et al., 2010; Cedernaes et al., 2015; Depner et al., 2014; Eckel et al., 2015; Jung et al., 2011; Markwald et al., 2013). These results are also in alignment with research in adolescents suggesting that insufficient and poor quality sleep have been associated with adverse cardiometabolic outcomes (e.g., T2D) (Castorani et al., 2020; Chaput et al., 2016; Cree-Green et al., 2019; Dorenbos et al., 2015; Javaheri et al., 2011; Kelly et al., 2010; Leproult & Van Cauter, 2010; Matthews et al., 2012; Wheaton et al., 2016; Zeitler, 2019). Interestingly, relatively longer sleep duration, after adjusting for adiposity, has been related to better insulin sensitivity in healthy adolescents (Javaheri et al., 2011) and in treatment-seeking adolescents with overweight and obesity (Simon et al., 2019). Taken together, these findings suggest that future research may benefit from further investigation into specific sleep characteristics as potential modifiable factors to prevent cardiometabolic disease prevention during adolescence.

As these results were only significant at trending levels and with relatively small effect sizes, past research has also noted that actigraphy may not be sufficiently sensitive to reliably measure individuals with symptoms of insomnia (Rösler et al., 2023); therefore it would be beneficial to utilize multiple alternative ways to best capture sleep constructs, such as self-reported sleep, perceived sleep, and polysomnography. Research in adolescents has also found that actigraphy measured wake after sleep onset was substantially greater than sleep diary estimates, whereas actigraphy measured total sleep time was substantially less than sleep diary estimates and parent report (Short et al., 2012). This indicates that there may be salient differences in adolescent sleep that can result in more or less actigraphy scored sleep than perceived self-reported sleep necessitating further examination (Short et al., 2012). Thus, future research investigating links between sleep and insulin sensitivity should aim to include multiple, complementary measures of sleep such as actigraphy, self-report, and polysomnography to develop a more comprehensive understanding of both adolescent objective and subjective sleep patterns in this context.

Hypothesis 2: Mindfulness and Self-Compassion as Moderators

We further predicted dispositional mindfulness and self-compassion would moderate the association of sleep and insulin sensitivity. There were no significant moderators. Instead, models including the interaction of weekend sleep efficiency by mindfulness and the interaction of weekday sleep efficiency by self-compassion on insulin sensitivity were at trending levels of significance with small effect sizes.

Mindfulness as a Moderator

The association between weekend sleep efficiency and insulin sensitivity was moderated by dispositional mindfulness at trending levels of significance. Specifically, when teens had better sleep and had higher dispositional mindfulness, compared to average or lower levels of mindfulness, they were more likely to be more insulin sensitive. These results suggest that high levels of mindfulness may only be able to moderate the associations of some types of poor sleep (i.e., weekend sleep efficiency) on insulin sensitivity. However, in contrast to our predictions based on the mindfulness stress buffering hypothesis (Creswell & Lindsay, 2014), dispositional mindfulness did not buffer the negative influences of poor sleep on insulin sensitivity. Rather than finding that higher mindfulness acted as a buffer, or dampened the associations between sleep and insulin sensitivity, our results showed the opposite in which the associations of sleep quality and insulin sensitivity were significant only for adolescents with higher relative to low or average levels of dispositional mindfulness, at trending levels of significance.

One possible explanation of this finding is that, as adolescents in our sample had elevated depression symptomology, they may have engaged in greater rumination around sleep compared to adolescents in the general population with lower levels of depression symptoms. Higher uncontrolled rumination in adolescent samples has been associated with greater depression and anxiety symptoms (Wilkinson et al., 2013). Additionally, rumination has been shown to be uniquely associated with the judgment of inner experiences in adolescent samples such that adolescents who engage in non-judgement, or who have a less negative view of their experiences, may be less likely to engage in rumination as a response to stress or sadness (Swords & Hilt, 2021). However, as greater depression symptoms may be associated with higher self-criticism, as opposed to engaging in non-judgmental acceptance associated with mindfulness

(Ehret et al., 2015), adolescents in this study could have engaged in these less adaptive responses to the stress of poor sleep, despite high levels of overall dispositional mindfulness.

Moreover, other research has suggested that different facets of mindfulness may have differential relationships with various psychological variables such as anxiety and depression (Desrosiers et al., 2013) and may be associated with an increased risk of experiencing distressing thoughts and feelings as well as exacerbating depression and anxiety symptoms (Lomas et al., 2015). In particular, although most aspects of mindfulness are associated with reduced psychological distress, the tendency to observe one's experience is often not associated (Desrosiers et al., 2013), or even positively associated, with stress and anxiety especially in individuals who do not meditate (Brown et al., 2015; Curtiss & Klemanski, 2014). Interestingly, in a sample of adolescents, the mindfulness aspects of awareness and non-reactivity predicted lower depression over time, yet, the aspect of observing predicted an increase in depression which was mediated by increased rumination (Royuela-Colomer & Calvete, 2016). Taken together, these results highlight the utility of utilizing multiple constructs related to mindfulness, in order to develop a more comprehensive understanding of potential adaptive and/or maladaptive effects in adolescents, especially among those with depression. Future research should consider

Another possible, alternative explanation is that our results may be in line with the differential susceptibility theory, which suggests that some individuals may have specific genetic predispositions that may influence their susceptibility to both positive and negative environments compared to other individuals (Belsky, 2013). Past research has suggested that environmental stressors may influence the risk for developing insomnia (Drake et al., 2014). The differential susceptibility theory further suggests that individuals who are more “vulnerable” may demonstrate heightened adverse effects of negative environmental experiences and demonstrate heightened beneficial effects from positive environmental experiences (Belsky et al., 2007). Therefore, the theory of differential susceptibility would suggest that mindfulness, as an adaptive internal environment, would increase the strength of the existing associations between sleep quality and insulin sensitivity, versus altering the existing associations. In this context, poor sleep quality would be associated with worsened insulin sensitivity for all individuals, but these associations would be heightened in adolescents with higher levels of dispositional mindfulness, compared to those with lower dispositional mindfulness.

The association of mindfulness and stress reactivity can also be seen neurobiologically in adults in which higher dispositional mindfulness was associated with decreased grey matter in the right amygdala, an area of the brain that contributes to the processing of emotions like fear, further providing potential explanations for why individuals with greater dispositional mindfulness may also have reduced stress reactivity (Taren et al., 2013). Further, sleep and stress are thought to interact bidirectionally influencing the central nervous system and metabolism. From a physiological perspective, poor sleep health alters the functioning of the HPA axis (Chrousos, 2009), resulting in elevated daily cortisol output, which, in turn, has been associated with lower insulin sensitivity (Huybrechts et al., 2014; Misra et al., 2008; Prodam et al., 2013; Reinehr & Andler, 2004) and predictive of worsening of insulin sensitivity over time (Adam et al., 2010), even after adjusting for adiposity. Therefore, the individual trait of higher dispositional mindfulness could help better regulate the physiological response to stress, like poor sleep, which may offer protective benefits on insulin sensitivity (Turner & Hingle, 2017). In line with this explanation, teenagers with greater mindfulness have been shown to more frequently use effective coping skills (Lucas-Thompson et al., 2019; Metz et al., 2013) and less frequently use maladaptive coping strategies such as emotional eating or overeating foods high in carbohydrates and fats, which can promote insulin resistance (Pivarunas et al., 2015).

Greater mindfulness may also improve sleep by increasing one's awareness of the mental and physical states related to sleep difficulties and thus adapting and improving cognitions and emotions typically related to sleep difficulties and negative sleep-related thoughts (Garland et al., 2015; Ong et al., 2012). For example, individuals with higher mindfulness may be more aware of their psychological and physical states at bedtime with acceptance which may promote relaxation and reduce psychological stress and arousal to improve sleep (Lau et al., 2018). However**,** the developmental model of regulation and sleep suggests that as insufficient sleep produces emotional, neurobiological, behavioral, and cognitive regulatory deficits, mindfulness, in turn, may also be reduced by poor sleep (Dahl, 1996; Palmer & Alfano, 2017) as seen in a study in which individuals randomly assigned to sleep deprivation had lower levels of mindfulness than those in a control condition (Campbell et al., 2018). Therefore, promoting quality sleep may be an essential aspect of emotional and physical health.

It is important to further note that the interpretations of these results must be undertaken with caution and for the purposes of hypothesis generation only. As our findings were only significant at trending levels, it is also possible that we are not fully capturing the construct of dispositional mindfulness in our current measure. For example, we could instead be measuring a small component of mindfulness. It has also been argued that self-reported mindfulness instruments may potentially assess attentional lapses rather than dispositional mindfulness (Grossman, 2008; Isbel et al., 2020). Other recent research has suggested that different facets of mindfulness may have differential relationships with various psychological variables such as anxiety and depression (Desrosiers et al., 2013). Interestingly, some research has found that mindfulness may be related to executive functioning abilities, such as working memory, suggesting that executive function and additional measures of mindfulness may be important additions to future research studies (Quaglia et al., 2015). As mindfulness and self-compassion may be trained and has been proposed to improve physical health among populations with high stress, such as adolescents, it is essential to increase investigation of mindfulness and stress in adolescents (Lindsay & Creswell, 2019).

These results contrast with a study I conducted with adolescents who had high weight, without elevated depression. Self-reported insufficient sleep duration was related to poorer insulin sensitivity with associations greater among adolescents with lower dispositional mindfulness, compared to average or higher levels of mindfulness, further suggesting that an individual’s dispositional mindfulness may be potentially modified to mitigate the adverse effects of poor sleep health on lowered insulin sensitivity (Clark et al., 2021). The finding of the moderating influence of mindfulness is also consistent with past research investigating links between sleep and mindfulness. Mindfulness-based interventions in young adults have suggested that mindfulness may also improve sleep quality and reduce daytime sleepiness (Bogusch et al., 2016; Caldwell et al., 2010; Howell et al., 2010), which is consistent with other research in adult samples (Ong et al., 2012). Cross-sectional studies on adolescents have also found that greater mindfulness is associated with higher self-reported sleep quality (Howell et al., 2008; Liu et al., 2018; Murphy et al., 2012). Similarly, a cognitive-behavioral and mindfulness-based group intervention for adolescents with depression or anxiety, resulted in increases perceived sleep quality (Blake et al., 2017). Past observational research consistently indicates that those who report being more mindful also report better and/or longer sleep (Bogusch et al., 2016; Brisbon & Lachman, 2017; Howell et al., 2010; Howell et al., 2008).

Self-Compassion as a Moderator

Similarly, when we assessed self-compassion as a moderator, self-compassion moderated the relationship between weekday sleep efficiency and insulin sensitivity at trending levels of significance. Specifically, if the participants were sleeping well, and had average or higher levels of self-compassion, they were more likely to be more insulin sensitive. Results suggest that average or high levels of self-compassion may only be able to moderate the associations of poor sleep (i.e., weekday sleep efficiency) on insulin sensitivity in adolescents. In contrast to our predictions informed by the mindfulness stress buffering hypothesis (Creswell & Lindsay, 2014), sleep quality was not related to insulin sensitivity when self-compassion was lower. Here, as also seen with dispositional mindfulness above, greater self-compassion seemed to amplify the association of sleep quality and insulin sensitivity, which is also consistent with the differential susceptibility theory (Belsky et al., 2007). These results contribute an additional attitudinal dimension of mindfulness that provides more important nuance of the construct. Past intervention work has emphasized the importance of experiential acceptance and self-compassion in improving emotion regulation in mindfulness interventions (Lindsay & Creswell, 2019) and these results further add to this work.

Another possible explanation of this finding is that a self-compassionate attitude may be able to influence key variables associated with poor sleep, such as stress and anxiety, and can help regulate physiological arousal associated with stress (Lindsay & Creswell, 2019). Self-compassion also has been postulated to improve an individual's ability to emotionally regulate stressful situations through adaptive cognitive reappraisal of the stressor, like sleep disturbance (Finlay-jones, 2017). For instance, individuals with higher self-compassion may be better able to relax at bedtime by reducing stressful, self-critical thoughts that could perpetuate a poor night's sleep (Butz & Stahlberg, 2018; Hu et al., 2018).

This finding is important as it suggests that self-compassion may be a modifiable factor in which improved self-compassion may facilitate better subjective sleep quality, and thus, in turn, better insulin sensitivity in adolescents. Other recent studies have found a significant association between self-compassion and sleep quality and suggest that self-compassion may play a protective role in mitigating the adverse effect of stressors on sleep quality (Butz & Stahlberg, 2018; Hu et al., 2018). Higher self-compassion has been shown to be linked to less bedtime procrastination, an important sleep-related behavior, partially due to the use of cognitive reappraisal, an adaptive emotion regulation strategy that helps downregulate negative mood (Sirois et al., 2019). A meta-analysis also found that self-compassion is associated with self-reported sleep quality in adults (Brown et al., 2021), which is consistent with a study on college students that found that a brief self-compassion induction contributed to improved sleep quality that night (Butz & Stahlberg, 2018). Another meta-analysis supported both the correlational and causal relationship between self-compassion and increased subjective sleep quality (Butz & Stahlberg, 2020). However, future research is needed to investigate possible mechanisms for relation between self-compassion and sleep to consider if self-compassion-based interventions might be effective in improving sleep quality and in turn improve insulin sensitivity.

Limitations

Although this study makes important contributions to our knowledge of a relatively specific, high-risk group of adolescent girls at risk for T2D with high weight and elevated depression, there are limitations to note. First, this study was a cross-sectional analysis that utilized only baseline data; therefore, we are currently unable to make conclusions regarding causality and see more robust buffering effects of mindfulness that may have been observed if these measures were collected longitudinally. Second, findings from this study may have limited generalizability. This specific sample of adolescents was comprised of individuals of high weight, elevated depression, and at risk for T2D; therefore, these results may not generalize to all adolescents. Additionally, although accelerometers are considered useful instruments for measuring sleep with high sensitivity and accuracy, their specificity is low compared with polysomnography, which is considered the gold standard for evaluating sleep (Ancoli-Israel et al., 2015; Marino et al., 2013). Finally, further longitudinal, experimental studies are needed to investigate how mindfulness and/or self-compassion training improves sleep health and in turn help prevent T2D in at-risk adolescents.

CONCLUSION

In conclusion, this dissertation contributes to our knowledge about the interconnections between sleep indices, dispositional mindfulness, self-compassion, and insulin sensitivity in adolescents at risk for T2D. Due to the high prevalence of insufficient sleep duration in adolescents and adolescent onset T2D combined with the continued research support on the connection between insufficient sleep and insulin sensitivity in adolescents, it is important to investigate insufficient sleep as a potentially modifiable factor in the prevention of adolescent T2D. Our results indicate that objectively measured sleep and insulin sensitivity are associated at trending levels. Moreover, the current study supports past literature documenting that mindfulness and self-compassion may moderate the associations of sleep quality on insulin sensitivity; however, these findings do not support the stress buffering hypothesis. Rather, results suggest that mindfulness and self-compassion may work through more complex mechanisms in adolescents potentially through rumination or through a differential susceptibility lens, overall necessitating further investigation. Future research should continue to explore multiple, complementary measures of sleep, as well as how mindfulness and self-compassion may operate over time. Furthermore, if researchers can identify salient mechanisms that may explain the connection between sleep insufficiency and T2D in adolescents, prevention/intervention programs can be improved to better target these mechanisms, improve sleep health, and potentially reduce adolescent T2D risk.

TABLES AND FIGURES

Table 1. *Study sample descriptive information*

Variable	*Mean*	*SD*	%
Age, years	14.40	1.81	--
Race/ethnicity			
Non-Hispanic White	--	--	28.1%
Hispanic	--	--	30.5%
Black	--	--	10.9%
Asian	--	--	2.3%
American Indian or Alaskan Native	--	--	3.1%
Other	--	--	25.0%
Weekday sleep duration (hours)	6.99	0.99	--
<8 hours	--	--	82.0%
8-10 hours	--	--	15.6%
>10 hours	--	--	0.0%
Weekend sleep duration (hours)	7.47	1.36	--
<8 hours	--	--	64.2%
8-10 hours	--	--	33.3%
>10 hours	--	--	2.5%
Watch Days Worn	7.05	1.69	--
<4 days	--	--	5.5%
≥4 days	--	--	94.5%
Perceived stress (PSS)	28.82	6.68	--
Insulin sensitivity (WBISI)	4.01	2.71	--
Dispositional mindfulness (MAAS)	56.94	12.53	--
Self-compassion (SCS)	2.81	0.57	--
BMI, kg/m^2	32.04	6.19	--
BMIz, standard score for age/sex	1.93	0.46	--
BMI, percentile for age/sex	96.00	3.56	--
Weight status			
Overweight (85-94th percentile)	--	--	32.8%
Obesity (≥95th percentile)	--	--	67.2%

Body composition for age/sex			
Total lean mass (kg)	43.88	8.80	--
Percent fat mass (%)	43.80	4.98	--
Tanner breast pubertal stage	4.84	0.56	--
Depression symptoms (CES-D total)	30.09	6.65	--

Note: N = 120-128; Weekday sleep duration is objectively measured by actiwatch worn by adolescents and calculated from bedtime/waketime. PSS is the 10-item Perceived Stress Scale with higher scores indicating greater stress. WBISI is the whole body insulin sensitivity index, with higher values representing better insulin sensitivity. MAAS is the Mindful Attention Awareness Scale with possible scores of 1 to 6, with higher scores indicating more mindfulness (more positive valence). SCS is the 26-item Self-Compassion Scale; higher scores indicate greater self-compassion. BMI is body mass index. BMI z-score refers to body mass index for age and sex. CES-D is the 20-item Center for Epidemiologic Studies-Depression Scale; the total sum score has a possible range of 0 to 60, with higher values representing greater depression symptomatology (more negative valence) and elevated depressive symptoms referring to a total score ≥21.

Table 2. *Bivariate correlations among key variables*

Variable	1. Age, years	2. BMIz	3. Race/ ethnicity	4. Puberty	5. Weekday wake after sleep onset	6. Weekday sleep onset latency	7. Weekday time in bed	8. Weekday sleep duration	9. Weekday sleep efficiency	10. Weekend wake after sleep onset	11. Weekend sleep onset latency	12. Weekend time in bed	13. Weekend sleep duration	14. Weekend sleep efficiency	15. Social jetlag	16. PSS	17. CESD	18. MAAS	19. SCS	20. WBISI
1.	--																			
2.	-.21*	--																		
3.	.04	-.22*	--																	
4.	.36***	-.10	.07	--																
5.	-.13	-.06	.09	.02	--															
6.	-.06	-.05	.05	-.17+	-.29**	--														
7.	-.33***	-.02	.04	-.19*	.48***	.21*	--													
8.	-.31***	.04	.01	-.15	.36***	-.15+	.85***	--												
9.	-.01	.10	-.09	.06	-.27 **	-.69***	-.23 **	.28**	--											
10.	-.30**	.16	.12	-.06	.57	-.09	.21	.09	-.26	--										
11.	-.04	-.00	.08	-.05	-.11	.48***	.15	-.06	-.43***	-.10	--									
12.	-.19*	.119	-. 07	-.12	.18*	.19*	.28**	.17+	-.21*	.46***	.29**	--								
13.	-.12	.09	-.12	-.08	.04	-.04	.15	.21*	.15	.20*	-.17+	.80***	--							
14.	.06	-.01	-.09	.08	-.19*	-.38***	-.16+	.12	.59***	-.33***	-.70***	-.17+	.44***	--						
15.	-.03	.06	-. 07	.16+	-.06	-.07	-.31***	-.32***	-.05	.16+	.12	.27**	.20*	-.05	--					
16.	.14	-.01	.06	.09	-.07	-.09	-.23**	-.21*	.10	.02	-.10	-.10	-.06	.06	.02	--				
17.	.03	.18*	-.01	.14	-.07	.03	-.06	-.07	-.04	.17+	.00	.08	.06	.02	.06	.29***	--			
18.	-.10	.10	-.05	-.14	-.00	-.14	.05	.12	.10	-.00	.06	.05	.03	-.01	.08	-.43***	-.01	--		
19.	-.13	-.08	-.18*	-.09	.20*	-.04	.24**	.24**	-.00	.06	.02	.20*	.18+	-.00	-.02	-.52***	-.27**	.38***	--	
20.	.31***	-.44***	.19*	.23*	-.08	-.13	-.18+	-.11	.10	-.23*	-.02	-.19*	-.11	.09	-.10	-.04	-.08	-.12	-.01	--
Mean (SD)	14.40 (1.81)	1.92 (0.46)	0.54 (0.55)	0.89 (0.31)	61.64 (27.59)	33.80 (32.94)	8.84 (1.28)	6.99 (0.99)	79.55 (5.62)	63.30 (35.04)	35.71 (39.94)	9.40 (1.61)	7.47 (1.36)	79.76 (8.57)	0.90 (1.10)	28.82 (6.68)	30.09 (6.65)	56.94 (12.53)	2.81 (0.57)	4.07 (2.71)

$+p \leq .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Note. N = 120-128; BMI z-score refers to body mass index for age and sex. Race/ethnicity was coded 0 = non-Hispanic White, 1 = other race/ethnicity. Puberty was coded 0 = late puberty/Tanner 5, 1 = early/mid puberty/Tanner 1-4. Weekday sleep duration is objectively measured by actiwatch worn by adolescents and calculated from bedtime/waketime. PSS is the 10-item Perceived Stress Scale with higher scores indicating greater stress. WBISI is the whole body insulin sensitivity index, with higher values representing better insulin sensitivity. MAAS is the Mindful Attention Awareness Scale with possible scores of 1 to 6, with higher scores indicating more mindfulness (more positive valence). SCS is the 26-item Self-Compassion Scale; higher scores indicate greater self-compassion. BMI is body mass index. CES-D is the 20-item Center for Epidemiologic Studies-Depression Scale; the total sum score has a possible range of 0 to 60, with higher values representing greater depression symptomatology (more negative valence) and elevated depressive symptoms referring to a total score ≥21.

Table 3. *Series of regression analyses for variables predicting adolescent insulin sensitivity*

	Insulin Sensitivity					
Predictor Variable	*b*	*SE*	*B*	*t*	*p*	ΔR^2
Step 1					.001***	.26
Age	.28	.13	.19	2.14	.03	--
BMIz	-2.09	.48	-.36	-4.38	.001***	--
Race/Ethnicity	.45	.40	.09	1.13	.26	--
Puberty	1.02	.72	.12	1.42	.16	--
Step 2						
Weekday wake after sleep onset	-.01	.01	-.09	-1.12	.27	.01
Weekday sleep onset latency	-.01	.01	-.12	-1.48	.14	.01
Weekday time in bed	-.24	.19	-.11	-.11	.19	.01
Weekday sleep duration	-.06	.23	-.02	-.02	.80	.00
Weekday sleep efficiency	.07	.04	.14	.14	.09+	.02
Weekend wake after sleep onset	-.01	.01	-.15	-1.68	.09+	.02
Weekend sleep onset latency	-.00	.01	-.02	-.23	.82	.00
Weekend time in bed	-.16	.14	-.09	-1.11	.27	.01
Weekend sleep duration	-.08	.17	-.04	-.48	.63	.00
Weekend sleep efficiency	.03	.03	.08	.94	.35	.01
Mindfulness	-.01	.02	-.04	-.48	.64	.00
Self-compassion	.07	.39	.02	.18	.86	.00
Step 3						
Depression	-.01	.03	-.02	-.23	.82	.00
Perceived stress	-.04	.03	-.11	-1.30	.20	.01

Note: *b* = unstandardized regression coefficient at each step; *SE* = standard error; *B* = standardized regression coefficient at each step; *t* = test statistic; ΔR^2 = change in variance explained by variables at each respective step +$p \leq .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

N = 128; BMI z-score refers to body mass index for age and sex. Race/ethnicity was coded 0 = non-Hispanic White, 1 = other race/ethnicity. Puberty was coded 0 = late puberty/Tanner 5, 1 = early/mid puberty/Tanner 1-4. Weekday sleep duration is objectively measured by actiwatch worn by adolescents and calculated from bedtime/waketime. PSS is the 10-item Perceived Stress Scale with higher scores indicating greater stress. WBISI is the whole body insulin sensitivity index, with higher values representing better insulin sensitivity. MAAS is the Mindful Attention Awareness Scale with possible scores of 1 to 6, with higher scores indicating more mindfulness (more positive valence). SCS is the 26-item Self-Compassion Scale; higher scores indicate greater self-compassion. BMI is body mass index. CES-D is the 20-item Center for Epidemiologic Studies-Depression Scale; the total sum score has a possible range of 0 to 60, with higher values representing greater depression symptomatology (more negative valence) and elevated depressive symptoms referring to a total score ≥21.

Table 4a. *Moderation effect of mindfulness on the association between weekend sleep efficiency and insulin sensitivity*

		b	*SE*	*t*	*p*
Constant	i_Y	10.05	10.05	1.75	.08
Weekend Sleep Efficiency (*X*)	b_1	-.18	.13	-1.46	.15
Mindfulness (*W*)	b_2	-.32	.18	-1.80	.08
Efficiency x Mindfulness (*XW*)	b_3	.00	.00	1.74	.09

$R^2 = 0.28$, $MSE = 5.80$
$F(7, 104) = 5.87$, $p < .001$

Note. N = 117 participants. *b* = unstandardized regression coefficient; *SE* = standard error; *t* = test statistic. Age, and BMIz (body mass index [BMI; k/gm^2] standardized for age/sex), race/ethnicity, and puberty were controlled for in the analyses.

Table 4b. *Conditional effects of mindfulness as a moderator in the association of weekend sleep efficiency and insulin sensitivity*

Predictor	Outcome	*b*	*SE*	*p*	LL CI	UL CI
Weekend Sleep Efficiency	Insulin Sensitivity					
-1 SD		-.01	.04	.76	-.08	.06
M		.04	.03	.18	-.02	.09
+1 SD		.09	.04	.05	.00	.17

Note. N = 117 participants. *b* = unstandardized regression coefficient; *SE* = standard error; LL CI=lower limit 95% confidence interval; UL CI=upper limit 95% confidence interval. Age, and BMIz (body mass index [BMI; k/gm^2] standardized for age/sex), race/ethnicity, and puberty status were controlled for in the analyses.

Table 5a. *Moderation effect of self-compassion on the association of weekday sleep efficiency and insulin sensitivity*

		b	*SE*	*t*	*p*
Constant	i_Y	26.31	15.49	1.70	.09
Weekday Sleep Efficiency (*X*)	b_1	-.30	.19	-1.56	.12
Self-Compassion (*W*)	b_2	-10.79	5.58	-1.94	.06
Efficiency x Self-Compassion (*XW*)	b_3	.14	.07	1.96	.05

$R^2 = 0.30$, $MSE = 5.57$
$F(7, 109) = 6.76$, $p < .001$

Note. $N = 117$ participants. *b* = unstandardized regression coefficient; *SE* = standard error; *t* = test statistic. Age, and BMIz (body mass index [BMI; k/gm^2] standardized for age/sex), race/ethnicity, and puberty were controlled for in the analyses.

Table 5b. *Conditional effects of self-compassion as a moderator in the association of weekday sleep efficiency with insulin sensitivity*

Predictor	Outcome	*b*	*SE*	*p*	LL CI	UL CI
Weekday Sleep Efficiency	Insulin Sensitivity					
-1 SD		0.01	0.05	.90	-.09	.11
M		0.10	0.04	.02	.02	.19
+1 SD		0.16	0.06	.01	.04	.28

Note. $N = 117$ participants. *b* = unstandardized regression coefficient; *SE* = standard error; LL CI=lower limit 95% confidence interval; UL CI=upper limit 95% confidence interval. Age, and BMIz (body mass index [BMI; k/gm^2] standardized for age/sex), race/ethnicity, and puberty status were controlled for in the analyses.

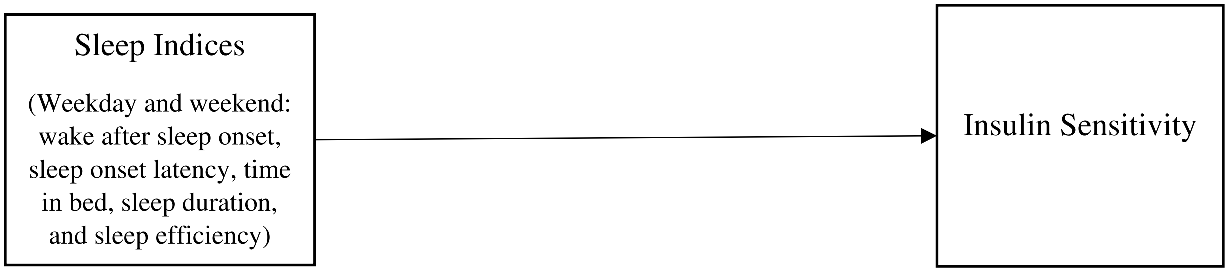

Figure 1. *Path model of the associations of sleep indices and insulin sensitivity*

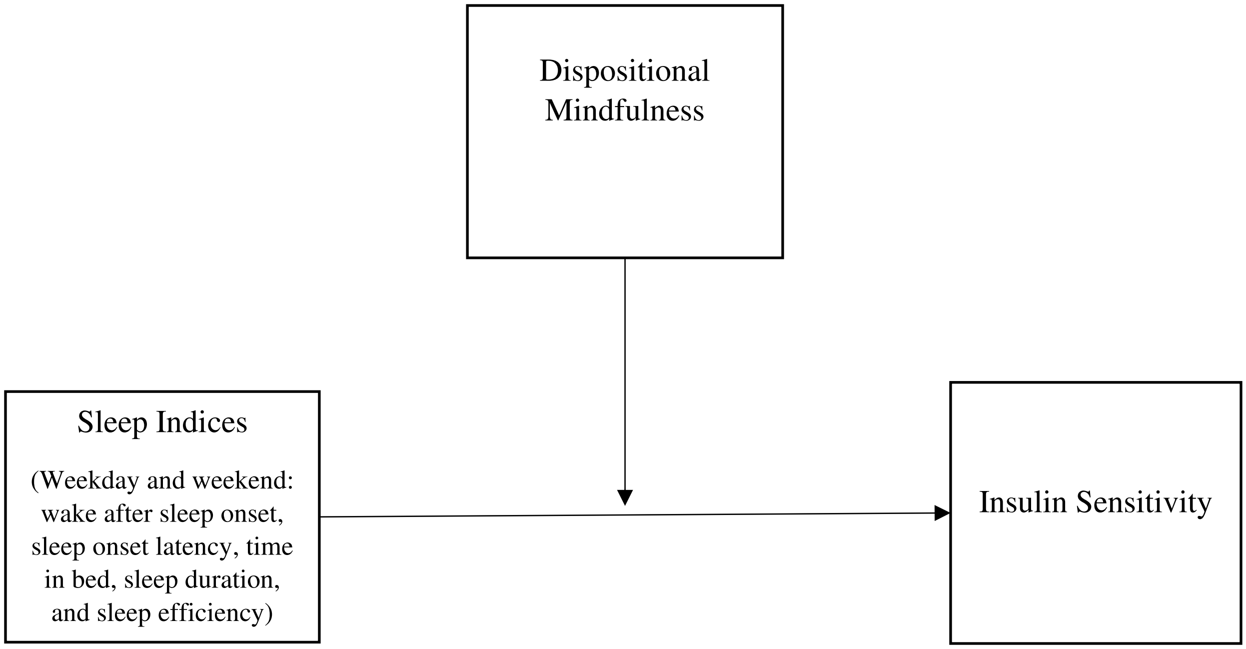

Figure 2. *Path model of the moderating role of dispositional mindfulness on sleep indices-insulin sensitivity*

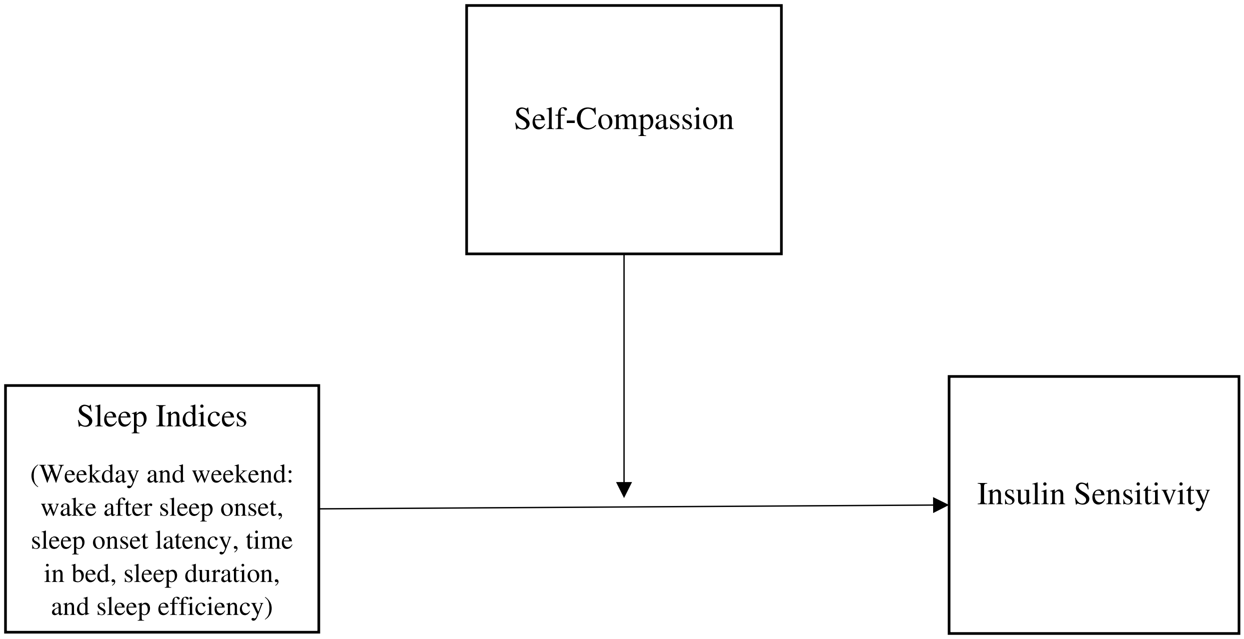

Figure 3. *Path model of the moderating role of self-compassion on sleep indices-insulin sensitivity*

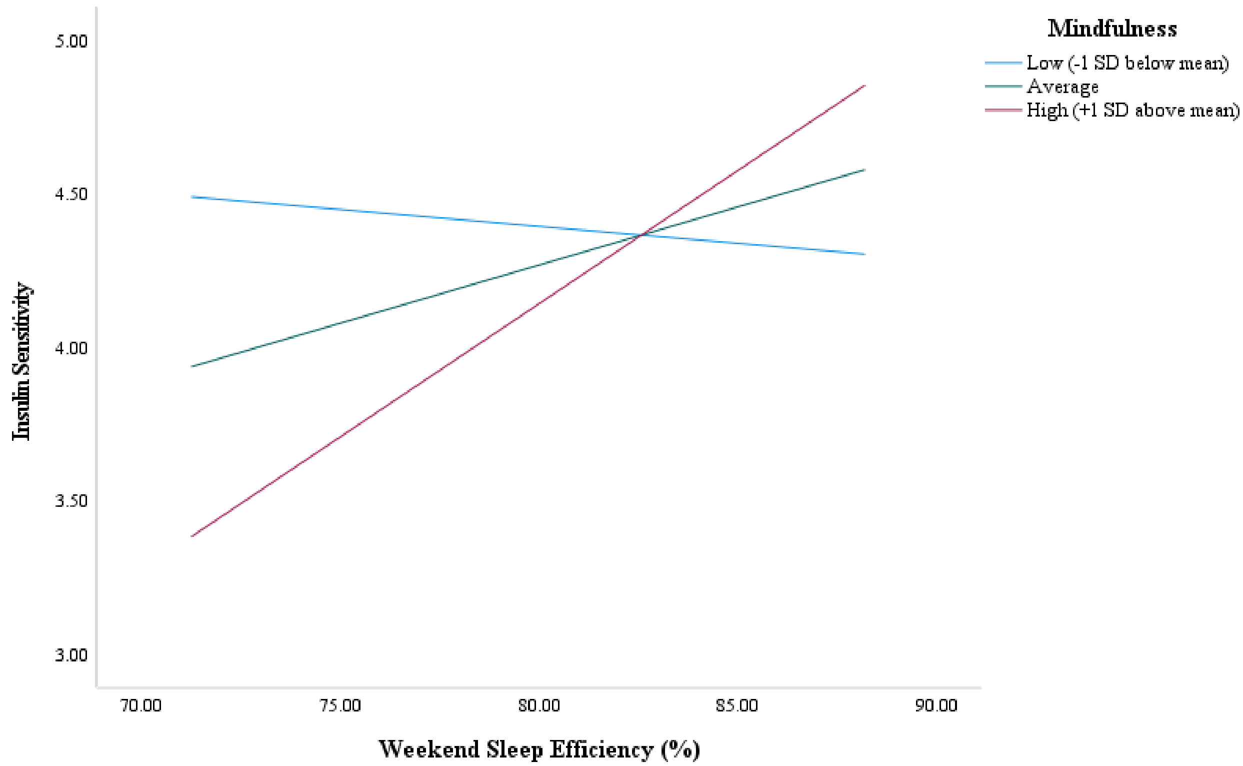

Figure 4. *The moderating role of dispositional mindfulness in the association between adolescent weekend sleep efficiency and insulin sensitivity*

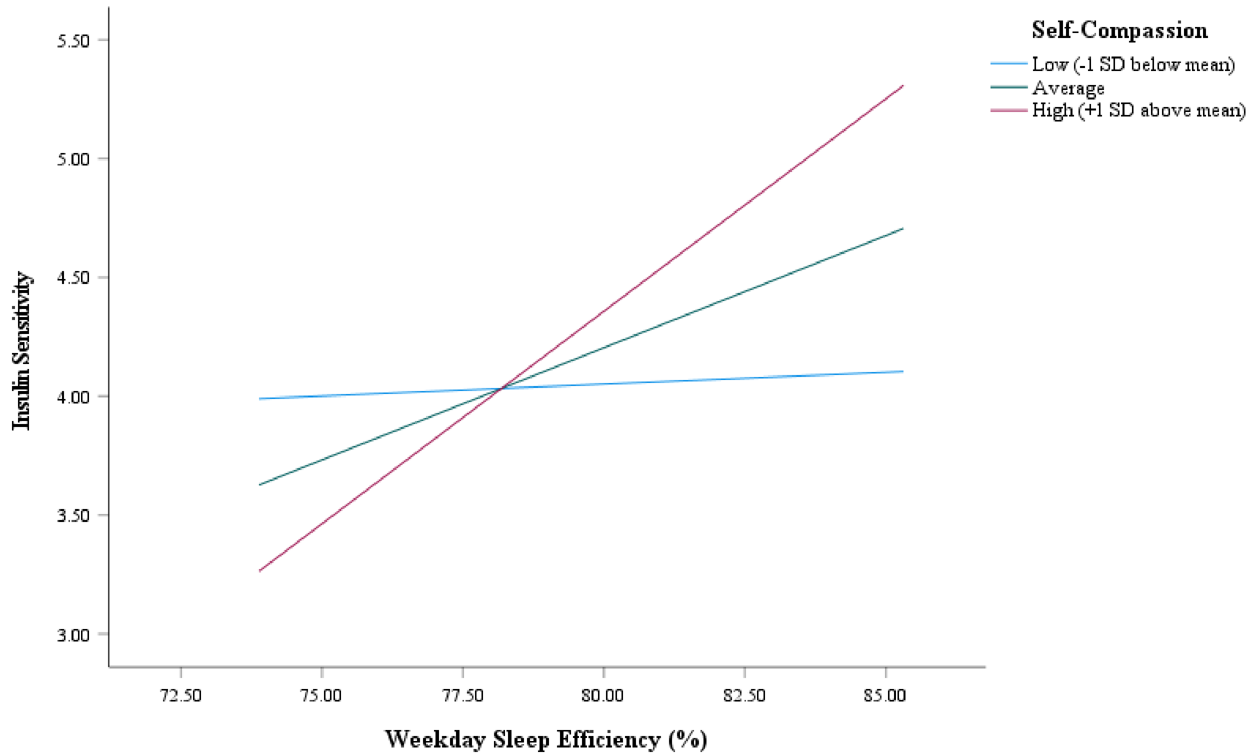

Figure 5. *The moderating role of self-compassion in the association between adolescent weekday sleep efficiency and insulin sensitivity*

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