SLEEP, PHYSICAL ACTIVITY, AND CARDIOMETABOLIC HEALTH IN ADOLESCENTS

SLEEP, PHYSICAL ACTIVITY, AND CARDIOMETABOLIC HEALTH IN OVERWEIGHT/OBESE ADOLESCENTS

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## ABSTRACT

Childhood obesity is currently at its highest. This is a concern due to the increased risk of developing type 2 diabetes, high blood pressure, and dyslipidemia. Previous findings suggest an association between sleep and obesity, as well as sleep and metabolic health. This study examined the relationship between objectively measured sleep and physical activity (PA), as well as sleep and cardiometabolic health in overweight/obese adolescents recruited from lifestyle clinics focused on behavioural change. Adolescents (N= 79, 10-17 yrs old) were given a Fitbit activity monitor to wear for 8 days and 7 nights to objectively track their sleep and physical activity. Exposure sleep variables included sleep duration, sleep latency, and sleep fragmentation. PA outcomes were step count and very active minutes. Body fat was measured with bioelectric impedance. Blood pressure z-score (normalized for age, gender, and height) and homeostatic model assessment-insulin resistance (HOMA-IR) were measured as well. PA (step count) was weakly associated with sleep duration (p=.087) after controlling for age, gender, adiposity, and wear time. Sleep latency predicted insulin resistance (HOMA-IR) after adjusting for age and gender (p=.047). This was a weak relationship after adjusting for age, gender, and body fat (p=.092) Sleep variables did not significantly correlate with blood pressure z-scores. It may be beneficial to target sleep in overweight/obese youth in lifestyle clinics in order to improve PA levels and cardiometabolic health.

## DECLARATION OF ACADEMIC ACHIEVEMENT

I have recruited participants and conducted visits with occasional assistance from either Scott Lynch or Selina Zheng. Scott Lynch, Selina Zheng, and Waleed Ahmed assisted with data entry. Matt Driedger assisted with wear time calculations. All statistical analysis was performed by me.

## TABLE OF CONTENTS

## LIST OF FIGURES

## LIST OF TABLES

## LIST OF ABBREVIATIONS

| | |
|---|---|
| BP | blood pressure |
| BMI | body mass index |
| GH | growth hormone |
| HOMA-IR | homeostatic model assessment-insulin resistance |
| METs | metabolic equivalent tasks |
| MVPA | moderate to vigorous physical activity |
| NREM | non-rapid eye movement |
| PA | physical activity |
| PSG | polysomnography |
| REM | rapid eye movement |
| SWS | slow wave sleep |
| TST | total sleep time |
| WHO | World Health Organization |

# CHAPTER 1: INTRODUCTION AND OBJECTIVE

Obesity has been drastically rising, affecting not only adults but children and adolescents as well (Ebbeling, Pawlak, & Ludwig, 2002). Roughly 1.6 million, or 31.5%, of Canadian children were classified as overweight or obese in the years 2009-2011 (Roberts, Shields, de Groh, Aziz, & Gilbert, 2012). Once obese, adolescents are at increased risk of developing type 2 diabetes (Steinberger & Daniels, 2003), high blood pressure (BP), and dyslipidemia (Williams, et al., 1992). High body mass index (BMI) and BP during childhood has been associated with poor cardiovascular health in adulthood (Laitinen, et al., 2013)

As obesity is a multifactorial disease (Grundy, 1998), researching factors that can improve adverse health consequences in overweight and obese adolescents will help identify potential targets for intervention. Traditional lifestyle behaviours such as physical activity and diet which are linked to obesity are targets for change in weight management programs (Whitlock, O'Connor, Williams, Beil, & Lutz, 2010). Sleep is a lifestyle behaviour that can be a target for change as well. Given the number of recent findings linking sleep duration and sleep disturbances to adverse metabolic health outcomes, understanding the relationship between sleep, physical activity (PA), and obesity related consequences among overweight and obese youth could lead to potential non-pharmacological methods of treatment. Moreover, as not all overweight/obese adolescents develop poor cardiometabolic health researching sleep may help us understand why some do. The goal of this study was to examine the relationship between sleep, PA, and adverse cardiometabolic health in overweight/obese youth using objective measurements.

# CHAPTER 2: BACKGROUND

## *2.1 Sleep Architecture*

Sleep is a natural state of altered consciousness with specific characterization that is self-controlled and is easily reversed by awakening. Sleep includes the decline in both voluntary motor activity and response to arousal activity, along with specific electroencephalographic, electro-oculographic, and electromyographic signals (Fuller, Gooley, & Saper, 2006). Sleep architecture is the structure and pattern of sleep, and is complex due to two distinct oscillating stages with a period of 90 minutes: non-rapid eye movement (NREM) and rapid eye movement sleep (REM) (Fuller, Gooley, & Saper, 2006); (Stiller & Postolache, 2005). Once sleep commences, stage 1 of NREM begins followed by stage 2, stage 3, and stage 4 of NREM sleep, after which REM sleep continues the state of sleep (Stiller & Postolache, 2004). Stages 3 and 4 are also known as slow wave sleep (SWS), characterized by slow brain wave activity (Fuller, Gooley, & Saper, 2006). SWS is a time when there are transient changes in metabolism, hormonal profiles, and neurophysiology (Tasali, Leproult, Ehrmann, & Van Cauter, 2008).

## *2.2 Sleep Disturbances*

Sleep deprivation can either be partial or total, and there are sleep disturbances, or arousals, which occur throughout the night (Bonnet & Arand, 2003). Sleep disturbances can be a cause for sleep deprivation. There are several sleep disturbance measurements. Sleep efficiency is a ratio measure of total time spent sleeping to time spent in bed. Sleep fragmentation is a ratio measure of the number of awakenings to total sleep time.

### *2.3 Sleep, Physical Activity, and Metabolism in Adults*

#### *2.3.1 Sleep and obesity*

Obesity is tagged to be a growing epidemic and it has also been suggested that sleep deprivation is an epidemic. It has been proposed that sleep loss and obesity may be "interacting epidemics" (Laposky, Bass, Kohsaka, & Turek, 2008). Studies have shown an association between short sleep duration and obesity (Beccuti & Pannain, 2011). A meta-analysis revealed that a reduction of the amount of sleep is associated with a increased BMI (Cappuccio, et al., 2008). Poor sleep quality has also been associated with obesity (Beccuti & Pannain, 2011).

#### *2.3.2 Sleep and Physical Activity (PA)*

A potential mechanism linking sleep and obesity is via changes in physical activity levels (Taheri, 2006) (Lucassen, Rother, & Cizza, 2012). This relationship could be explained by fatigue after sleep loss, therefore causing one to participate in less activity throughout the day (Taheri, 2006). Although the exact reasoning for the necessity of sleep is still unknown, it is predicted that sleep plays a role in body restoration (Fuller, Gooley, & Saper, 2006). It has been proposed that with the reduction of sleep and no proper energy restoration, PA during the day will be greatly decreased (Driver & Taylor, 2000). A sleep restriction study in 15 healthy men reported that compared to sleep duration of 8 hours, after sleep duration of 4 hours participants spent less time in more intense habitual PA (25.4% compared to 22.6%) measured by Acti-Watch (Schmid, et al., 2009). As this was a short term sleep deprivation exposure, more sleep experimental studies are necessary to determine whether sleep deprivation causes lower PA the next day.

### *2.3.3 Sleep and Blood Pressure (BP)*

In healthy individuals BP declines 10-20% during sleep and increases towards wakefulness (Smolensky, Hermida, Castriotta, & Portaluppi, 2007). A possible explanation for this trend is that sympathetic nerve activity which raises BP is lower during NREM than during wakefulness, due to increased vagal tone (Smolensky, Hermida, Castriotta, & Portaluppi, 2007). It is thought that autonomic changes which occur after a stress response are similar to those which occur after sleep disturbances (Mullington, Haack, Toth, Serrador, & Meier-Ewert, 2009). Reduced time spent in SWS could increase catecholamine release (Palagini, et al., 2013). Figure 2 summarizes the potential pathways by which sleep disturbances could result in increased BP.

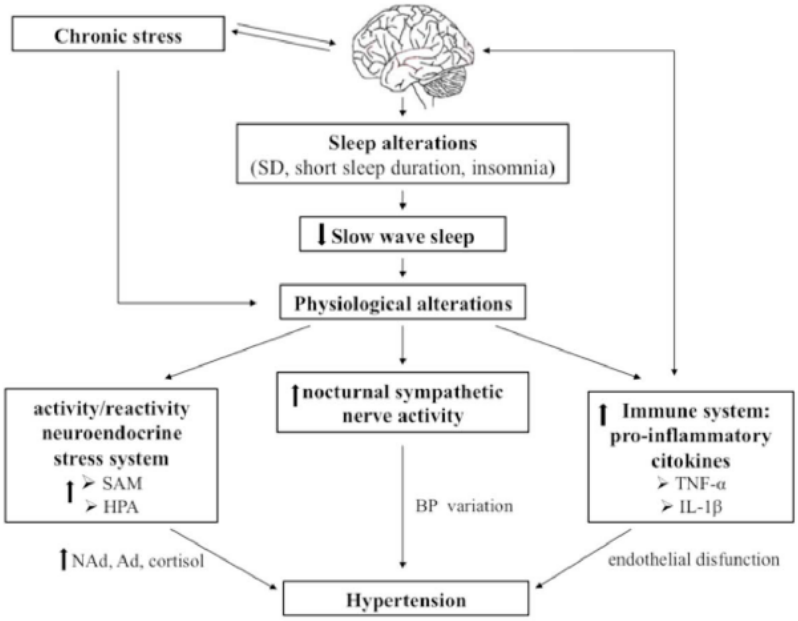



**Figure 1.** Mechanisms and pathways through which sleep deprivation and short sleep duration could cause hypertension (Palagini, et al., 2013).

Friedhelm et al. (2009) found that when there is no SWS, BP during sleep did not dip as much as it did when SWS is intact. Other sleep deprivation studies in adults show that after 24 hours of partial sleep deprivation (4 hours), BP values increased in the morning as well as

throughout the day compared to normal sleep (8 hours) in healthy participants (Lusardi, et al., 1996) and hypertensive participants (Lusardi, et al., 1999). Overall, sleep experimental studies consistently show an effect on BP levels caused by sleep deprivation.

### *2.3.4 Sleep and Glucose Regulation*

Blood sugar homeostasis largely depends on the equilibrium between liver glucose production and glucose utilization by insulin-dependent tissue and non-insulin-depended tissues (Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005). Insulin is central to glucose homeostasis inhibiting hepatic glucose production and stimulating glucose uptake predominantly by skeletal muscle and adipose tissue (Van Cauter, Polonsky, & Scheen, 1997). Glucose is regulated in a circadian manner (Rudic, et al., 2004).

Glucose levels are tightly regulated within a narrow normal range (Van Cauter, Polonsky, & Scheen, 1997). Within that range glucose levels are higher in the afternoons and evenings than in mornings (Van Cauter, Polonsky, & Scheen, 1997). The variations noticed between daytime and nighttime glucose levels are due to the reduction in glucose tolerance across the day (Van Cauter, Polonsky, & Scheen, 1997). During the state of sleep, blood glucose levels stay relatively constant in healthy people in spite of the prolonged fasting timeframe, mostly due to decreased energy requirements (Lucassen, Rother, & Cizza, 2012). This implies that there are nocturnal mechanisms which maintain the homeostatic glucose levels (Van Cauter, Polonsky, & Scheen, 1997).

Nighttime brain glucose metabolism decreases by roughly 30-40% during NREM and is responsible for two thirds of the reduction in glucose utilization at night (Van Cauter, Polonsky, & Scheen, 1997). The remaining third comes from reduced glucose utilization partially caused by reduced muscle tone (Van Cauter, Polonsky, & Scheen, 1997).When observing NREM and

REM sleep, it was found that NREM sleep was associated with increased glucose levels (Scheen, Byrne, Plat, Leproult, & Cauter, 1996). Reduced SWS worsens the body's ability to maintain glucose homeostasis and reduced SWS for 3 nights resulted in decreased insulin sensitivity, possibly due to increased sympathetic activity (Tasali, Leproult, Ehrmann, & Van Cauter, 2008).

Other hormonal changes occur with sleep deprivation and these may also influence glucose homeostasis. Hormones such as glucagon, catecholamines, and GH also influence glucose homeostasis (Van Cauter, Polonsky, & Scheen, 1997). GH undergoes variation during sleep, reaching maximum at the beginning of SWS (Lucassen, Rother, & Cizza, 2012). GH has anti-insulin effects which also plays a role in the reduction of glucose uptake (Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005). GH's possible ability to increase blood glucose concentrations is due to the mechanisms of increased hepatic glucoeneogensis and reduced insulin release from beta cells (Laposky, Bass, Kohsaka, & Turek, 2008). As adolescents undergo puberty, GH levels reach peak and decline with age (Fritz & Speroff, 2010). Cortisol undergoes circadian variation and increases glucose utilization towards awakening (Laposky, Bass, Kohsaka, & Turek, 2008). During sleep deprivation cortisol levels increase and decline at a slower rate than during normal sleep leading to greater cortisol levels are higher in the afternoon and evening (McNeil, Doucet, & Chaput, 2013). This increase in cortisol levels during the evening has been associated with decreased insulin sensitivity in the morning (McNeil, Doucet, & Chaput, 2013)

#### *2.3.4.1 Sleep Disturbances and Glucose Regulation*

Sleep disturbances can impact glucose regulation. Buxton et al. (2012) had normal weight healthy adults participants undergo sleep deprivation (5.6hrs of sleep/24hrs) for 3 weeks and observed significantly higher fasting postprandial glucose peaks and lower postprandial insulin

secretion compared to baseline (Buxton, et al., 2012). With the increase in glucose peak and the reduction in insulin secretion, glucose levels were maintained at higher levels, with 3 out of the 21 participants reaching prediabetic glucose levels (Buxton, et al., 2012). The authors suggest that a metabolic dysregulation lead to an abnormal response to food intake (Buxton, et al., 2012). Other studies have also reported greater glucose intolerance, higher glucose levels, and lower insulin sensitivity with sleep deprivation (Spiegel, Leproult, & Van Cauter, 1999) (Donga, et al., 2010). These possible hypothesized mechanisms are in Figure 2. Overall, sleep experimental studies consistently report sleep deprivation worsening metabolic response.

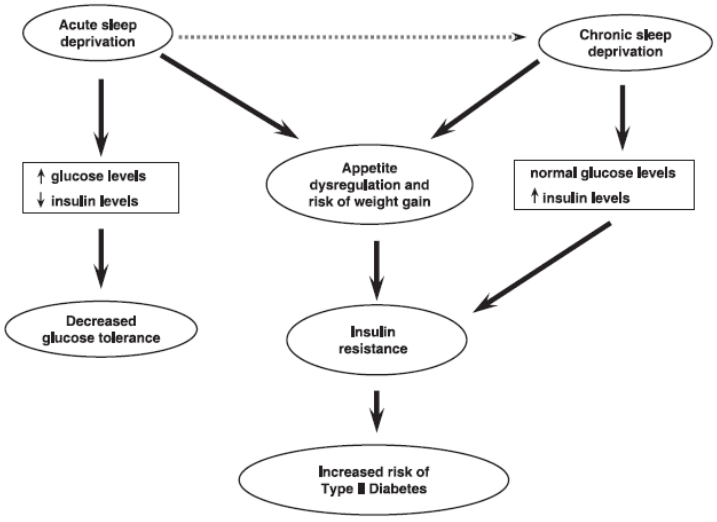



**Figure 2.** Mechanisms by which acute and chronic sleep deprivation may result in weight gain, altered glucose metabolism and type 2 diabetes (Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005).

### *2.3.4.2 Other Sleep Disturbances and Glucose Regulation*

Other sleep disturbances aside from sleep deprivation have been associated with glucose dysregulation. Longer self-reported sleep latency has been associated with developing type 2 diabetes (Kawakami, Takatsuka, & Shimizu, 2004). Lastly, sleep quality independent of sleep duration has been linked to obesity and insulin resistance in adults in epidemiological evidence

(Lucassen, Rother, & Cizza, 2012). Experimental studies are required to further understand this relationship.

## CHAPTER 3: SLEEP, PA, AND METABOLISM IN ADOLESCENTS

### *3.1 Sleep and Adolescence*

The sleep/wake cycle becomes delayed with maturity in adolescents, with weekend bedtime later than weekday and sleep extension on weekends (Crowley, Acebo, & Carskadon, 2007); (Giannotti, Cortesi, Sebastiani, & Ottaviano, 2002). Environmental influences prevalent during adolescence, such as academic requirements, caffeine consumption, and technology use, can influence sleep architecture (Carskadon, Acebo, & Jenni, 2004), (Calamaro, Mason, & Ratcliffe, 2009). Although it has been thought that environmental factors were the sole reason for the delay in bedtime, change in adolescent circadian timing is now an acceptable theory (Carskadon, Vieira, & Acebo, 1992). Adolescents experience a delay in sleep onset with age, shorter sleep duration, and prefer more evening activities than children (Colrain & Baker, 2011). Carksadon et al. (1992) noticed an inverse association between physical maturation and the tendency to have an evening preference and be later riser, concluding that puberty has an effect on circadian phase delay seen in adolescents. This evening preference occurs at around the age of 13, reaching maximum evening tendencies at around the age of 20 (Colrain & Baker, 2011). Upon reaching 20, morning tendencies increase again marking the end of adolescence (Colrain & Baker, 2011). It is not until the full development of the cortex that sleep becomes similar to adults (Brown, Basheer, McKenna, Strecker, & McCarley, 2012). Laboratory studies have also shown that circadian timing is later with increasing pubertal stage; the reasoning behind this observation though is still unclear (Carskadon, Acebo, & Jenni, 2004). Early pubertal

adolescents have smaller sleep latency than adolescents entering later stages of puberty (Crowley, Acebo, & Carskadon, 2007). There is also a correlation between puberty stage and reduced SWS (Carskadon, Acebo, & Jenni, 2004). It is evident that sleep architecture differs across one's lifetime therefore it is imperative to conduct research on adolescents' sleep separately from adults and children.

### *3.2 Sleep and Obesity in Adolescents*

Although the relationship between sleep and obesity is less studied in adolescents (Lytle, Pasch, & Farbakhsh, 2011) studies have revealed an association between short sleep duration and obesity in children (Chaput, et al., 2011); (Gupta, Mueller, Chan, & Meininger, 2002). This association seems to be stronger in children than in adults, possibly suggesting that children are more sensitive to short sleep duration (McNeil, Doucet, & Chaput, 2013). A limitation of these studies is that these studies relied on self-reported sleep and measured BMI adjusted for age (Xiaoli, Beydoun, & Wang, 2008). The limitations of self-reported sleep include recall bias and may overestimate PA, as discussed in Appendix A. BMI measurement has the limitation of not differentiating between fat mass and fat-free mass, and is a measure of excess body mass as opposed to body composition (adiposity) which could be a more important predictor of poor health outcomes (Frankenfield, Rowe, Cooney, Smith, & Becker, 2001).

In a study of 1171 adolescents (13 yrs old), self-reported sleep duration was inversely associated with BMI z-score, and positively associated with adiposity (Araujo, Severo, & Ramos, 2012). Gupta et al. (2012) (n=383, 11-16 yrs old, 26% obese) objectively measured sleep for 24 hours and reported that obese adolescents slept less than non-obese adolescents, and for every 1 hour of lost sleep the odds of obesity increased by 80%. There are limited studies looking at the relationship between sleep and the extent of obesity in an overweight/obese

population. Sung et al. (2011) (n=122, 10-16.9 years old) found no significant relationship between objectively measured sleep duration and BMI, BMI z-score, or waist circumference. More studies using objective measures are needed to prove the relationship between sleep and obesity. Moreover, more research is needed in solely overweight/obese populations.

### *3.3 Sleep and Physical Activity in Adolescents*

In a population based study, Gupta et al. (2002) (n=238, 11-16 years old) showed a reciprocal association between sleep disturbance and PA counts the following day in a sample containing 26% obese adolescents. Objective measures of PA and sleep were used but monitored for only 24 hours (Gupta et al., 2002). For every hour increase in sleep disturbance, PA decreased by 3% the next day. Pesonen et al. (2011) (n=297, 8 years old) objectively measured sleep and moderate-to-vigorous PA for 7 days and found the opposite; i.e. increased sleep duration and sleep efficiency were correlated with reduced PA the next day. For every standard deviation unit increase in sleep duration and efficiency, PA decreased by 0.09 and 0.16 standard deviations the next day. It was also reported that increased sleep latency and fragmentation were associated with increased PA the next day. Stone et al. (2013) (n=856, 10-12 years old) reported that adolescents who sleep less than 9 hours are less active than those who slept more than 10 hours. Pesonen et al. (2011) did not include obese adolescents, while Stone et al. (2012) had a more variable sample including lean, overweight, and obese adolescents. Overall, results regarding the association between sleep and PA are inconclusive, and samples of adolescents with obesity are lacking. Moreover, as these are cross-sectional studies it is difficult to conclude the direction of the relationship, ie. sleep impact PA or vice versa. Therefore, several studies attempted to delineate this relationship.

Brand et al. (2010) (n=38) reported that high exercisers had longer total sleep time, more SWS, and better overall sleep efficiency compared to low exercisers. However, sleep was measured by one night sleep-EEG, and as previously mentioned is not indicative of regular sleep patterns, while PA was assessed via a questionnaire. Furthermore, they only studied lean adolescents (Brand et al., 2010).

Nixon et al. (2009), Nixon et al. (2008), and Pesonen et al. (2011) used objective measurements for sleep and PA to evaluate their inter-relationships; Nixon et al. (2009) (n=579, 7 years old) measured sleep and PA with an actigraph for a 24 hour period. They reported an inverse association between PA and sleep latency, and time spent sedentary was associated with increased sleep latency (Nixon et al., 2009). As for sleep duration, Nixon et al. (2008) and Pesonen et al. (2011) examined the relationship between daytime PA and subsequent night sleep. Nixon et al. (2008) (n=579, 7 years old) did not find a significant association between PA and sleep duration while using an actigraph for 24 hours. Studies that looked at day to day relationships between sleep and PA are inconclusive, as is the directionality.

### *3.4 Sleep and Blood Pressure in Adolescents*

Though there appears to be an inverse relationship between BP and sleep among adults, fewer studies have been conducted involving children, with mixed results (Bayer, Hannelore, & Kris, 2009). Javaheri et al. (2008) (n=238, normal weight 13-16 years old) measured sleep via actigraphy for 5-7 days. After dividing the sample into prehypertensive and normotensive, it was found that low sleep duration (< 6.5 hours) and low sleep efficiency (<85%) are more prevalent in prehypertensive lean adolescents. It was also found that those with low sleep efficiency and those with short sleep were more likely hypertensive (Javaheri, Storfer-Isser, Rosen, & Redline,

2008). Bayer et al. (2009) suggest that the sleep duration effect on BP is only seen in adults. Furthermore, the relationship of sleep to BP in obese adolescents has not been reported.

### *3.5 Sleep and Insulin Resistance in Adolescents*

Experimental sleep lab studies are limited in the pediatric population. A study by Klingenberg et al. (2013) had 21 healthy, lean male adolescents (15-19 years old) undergo short sleep (4 h) and long sleep (9 h) while monitoring their sleep by a polysomnography (PSG). Insulin sensitivity measured with the matsuda index and IR measured with homeostatic model assessment-insulin resistance (HOMA-IR) worsened during the short sleep period (Klingenberg, et al., 2013). The authors concluded that acute sleep restriction decreases insulin sensitivity.

Several cross-sectional studies have shown an association between sleep restriction and insulin resistance in youth. Matthews et al. (2012) (n=245, 14-19 years old) used actigraphy (an objective measurement tool for sleep), and reported that the shorter the sleep during weekdays the higher the HOMA-IR, and higher glucose levels among adolescents with greater sleep fragmentation. This study included 117 obese adolescents (Matthews, Dahl, Owens, Lee, & Hall, 2012).

A study by Flint et al. (2007) included obese children (n=40, 3.5-18 years old) and found an association between sleep duration less than 6 hours and an increase in HOMA-IR. The objective measurement of PSG was only used for 1 night (Flint, et al., 2007).

Sung et al. (2011) and Koren et al. (2011) studied obese adolescents and found no association between sleep and insulin resistance, however both studies had limitations. The subjects in the study by Koren et al. (2011) (n=62, 8-17.5 years old) underwent an overnight PSG which may not be indicative of usual sleeping patterns (Koren, et al., 2011). The participants in the study by Sung et al. wore an actigraph (n=122, 10-16.9 years old) for 7 days,

however only sleep duration was reported on and not sleep fragmentation. It is apparent that sleep fragmentation may have a more pronounced effect on insulin resistance than sleep duration; therefore the lack of sleep fragmentation variables in the study could have created a false negative association (Morselli, Knutson, & Mokhlesi, 2012). Another limitation in this study, as noted by the authors, is that sleep, insulin, and anthropometric measures were done at separate times (Sung, et al., 2011). Height and weight were recorded at the initial visit, blood work was done within a month of the initial visit, and the actigraphy was distributed 14 weeks after the initial visit (Sung et al., 2011).The large time gap may have ultimately altered the results. As the evidence linking sleeping patterns to insulin resistance in obese adolescents is inconclusive, this study is essential particularly since it will use objective measures for 7 nights in overweight/obese youth.

## CHAPTER 4: HYPOTHESIS & OBJECTIVES

A hypothesized mechanism linking sleep and obesity is the reduction of PA levels due to fatigue, as lower PA levels are a widely known contributor to obesity (Sallis, Prochaska, & Taylor, 2000). As previously mentioned, obese adolescents are at greater risk of developing high BP and insulin resistance than lean adolescents. However, not all obese adolescents develop cardiometabolic problems, suggesting that other factors contribute to cardiometabolic disturbances. From literature we believe that adiposity is inversely related to PA and directly related to BP and insulin resistance, and we want to examine whether adiposity explains any identified relationship between sleep and either PA or cardiometabolic health. We will evaluate the relationship of sleep disturbance with 1) PA levels and 2) cardiometabolic health in

overweight/obese adolescents (figure 3). Furthermore, we will evaluate the extent of adiposity as a mediator of these relationships Figure 3 [3), 4)].

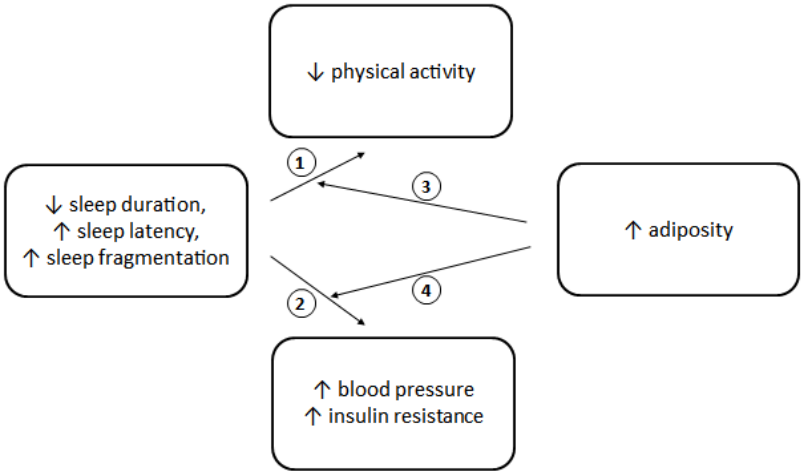



**Figure 3.** Proposed hypothesis of how sleep may play a role in cardiometabolic health and PA levels in overweight and obese adolescents.

**Primary Hypothesis:**

Sleep patterns will be associated with objectively measured PA in overweight/obese adolescents. Lower sleep duration and higher sleep latency and/or fragmentation will be associated with lower PA in overweight/obese adolescents. These relationships will be independent of adiposity.

**Secondary Hypothesis:**

Sleep patterns will be associated with cardiometabolic health, specifically insulin resistance and BP, in overweight/obese adolescents. Lower sleep duration and higher sleep latency and/or fragmenation will be associated with higher insulin resistance and BP. These relationships will be independent of adiposity.

# CHAPTER 5: METHODLOGY

## *5.1 Study Design*

The Sleep and Physical Activity in Youth (SPA) Study is a cross-sectional study of 10-17 year olds recruited from the Growing Health Weight Management Program and the Pediatric Lipid Clinic at McMaster's Children Hospital. The SPA study included both lean and overweight participants. A subset of SPA participants were included in this analysis.

## *5.2 Sample Population Inclusion/Exclusion*

Participants were only recruited during the school year to avoid unstructured schedules during holiday breaks (winter, spring, and summer breaks). Participants were classified as overweight/obese using body mass index (BMI) z-scores based on height, weight, gender, and age (World Health Organization, 2007) (WHO). The inclusion criteria were participants with a BMI z-score>1. Participants were excluded if they had a BMI z-score<1, <4 days of sleep data including at least 1 weekend day, or reported use of melatonin. In addition, if participants had <4 days of PA measurement including at least 1 weekend day, they were excluded from the analysis for hypothesis 1 (n=3). For the BP analysis, if a valid BP measurement were unavailable or the use of BP lowering medication was reported, participants were excluded (n=6). For the IR analysis, if a valid glucose/insulin levels were unavailable or the use of insulin was reported, participants were excluded (n=34)

## *5.3 Sleep Measures (exposure variable)*

The exposure variables for both hypotheses were sleep characteristics:

1) Daily sleep duration, 2) Daily sleep latency, and 3) Daily sleep fragmentation

Sleep duration was recorded by the Fitbit (Fitbit Inc., San Francisco, USA) (as the difference in time between sleep time and wake up time, and is the minutes spent sleeping. Sleep latency was measured by the Fitbit as the time difference between bed time and sleep onset. Number of awakenings throughout sleep was determined by the Fitbit based on the amount of movement throughout the night. Sleep fragmentation was calculated as the number of awakenings per hour spent in bed post sleep onset divided by the hours of sleep. All sleep variables were averaged in order to account for weekdays and weekend distribution, see Table 2 for specific calculations. The algorithms used by Fitbit to measure sleep characteristics are not released, therefore the measurement cut-offs for these variables are unknown. However, these sleep measures were comparable to Actigraph sleep measures (Appendix A).

### *5.4 Outcome Measures*

**Physical Activity**

**Primary outcome:** daily step count

**Secondary outcome:** daily very active minutes, daily step count/wear time, daily very active minutes/wear time

There are multiple ways to measure PA. These include daily step count (primary outcome) and daily very active minutes, daily steps/wear time, and daily very active minutes/wear time as our secondary outcomes.

Fitbit PA algorithms are not released; therefore the Fitbit activity measures were compared to accelerometer PA measures (Appendix A). Step count between the accelerometer and Fitbit correlated highly for each participant (Appendix A). Very active minutes did not match with Actigraph moderate or vigorous minutes (Appendix A) and therefore cannot be considered as a measure of moderate or vigorous activity as recommended by the Canadian

Physical Activity Guidelines (Janssen, 2007). We considered very active minutes as a measure of PA intensity. The PA outcomes were averaged across the number of days the Fitbit was worn (see Table 2 for calculations), to appropriately account for the distribution of weekdays and weekends. We also adjusted the PA variables for wear time, as step count and very active minutes could be influenced by the length of time the Fitbit was worn.

**Blood Pressure**

**Primary outcome:** systolic BP

**Secondary outcome:** diastolic BP

BP was recorded from the participant's clinic chart and was considered valid if measured within 8 weeks of the study visit. BP was converted to z-scores using National Institutes of Health (2005) blood pressure tables which are age, height, and gender specific. A z-score of 1.28 and 1.64 placed categorized the participants as prehypertensive or hypertensive, respectively (National Institutes of Health, 2005).

**Insulin Resistance**

**Outcome:** HOMA-IR

Fasting glucose and insulin were recorded from the participant's medical chart and were considered valid if measured within 8 weeks of the study visit. HOMA-IR was calculated as:

Fasting glucose (mmol/L) x Fasting insulin (μIU/mol) ÷ 22.5.

HOMA-IR was chosen because it is the most accurate method to use in large populations (Kurtoglu, et al., 2010), and is highly correlated ($r$=0.88) with the gold standard euglycemic clamp (Singh & Saxena, 2010).

### *5.5 Anthropometry/Adiposity Measures*

The participants' height and weight within 4 weeks of the study visit were recorded from their clinical charts (Refer to Table 1 for height and weight measurement). A z-score of >1 or >2 placed the participant in the overweight and obese category, respectively. These growth standards are the current recommendations by the WHO and the Canadian Pediatric Society. Methodologies for adiposity, BP, fasting glucose, and fasting insulin are described in Table 1. Bioelectrical impedance is safe and non-invasive. It detects differences in electrical conductivity between fat and water components (Bolanowski & Nilsson, 2001).

### *5.6 Sleep and Physical Activity*

Adolescents were given a Fitbit Activity Monitor to wear for 8 days and 7 nights. This device objectively tracks physical activity and sleep throughout the day, and the data is uploaded online. Fitbit Ultra was given out to the participants; however with the progression of the study several participants were given the Fitbit One as the Ultra was discontinued. The different Fitbit models were used interchangeably due to high similarities between their outputs. Refer to Appendix A for comparison of the Fitbit One and the Fitbit Ultra.

Upon the return of the Fitbit from the participant, if sleep mode was initiated by the participant by pushing a button to denote going to bed, recorded sleep times were used. If sleep mode was not initiated by the participant, the log sheet was used to manually enter wake up and bed time. Sleep data was considered valid if the participant had sleep data for 4 days, with at least 1 weekend day. Weekend days for sleep measures include Friday and Saturday nights, as these are non-school nights. It is necessary to include a weekend day in our analysis as wake up time and bed time are delayed on weekends, and differ from weekdays, as previously discussed.

Thus, it is imperative for us to account this variability in our calculations of average sleep characteristics.

PA data was considered valid if the participants had PA data for 4 days (Colley, et al., 2011) with at least 1 weekend day. Weekend days for PA include Saturday and Sunday. A weekend day is necessary because adolescents exhibit variability between weekdays and weekends PA, with lower PA during weekends (Trost, Pate, Freedson, Sallis, & Taylor, 1999). Thus, it is imperative for us to account this variability in our calculations of average PA characteristics.

### *5.7 Sleep and Physical Activity Analysis*

Fitabase (Small Steps Lab LLC, California, USA) is a platform designed to collect data from internet connected devices, with the ability to export minute by minute data recorded by the Fitbit. This service was purchased in order to help with analysis of wear time to determine whether the Fitbit was worn throughout the majority of the day. With Fitabase we were able to export minute by minute step count, as well as minute by minute intensity. However, intensity is not exported as counts as is done by accelerometers such as Actigraph, but rather as digits 1-3 representing light activity, moderate activity, and very active activity, respectively.

We defined a valid day of PA with steps recorded in at least 10 hours of wear time (Colley, et al., 2011). Nonwear time in accelerometers such as Actigraph and Acticals is often defined as at least 60 consecutive minutes of 0 counts, with allowance for 1-2 minutes of counts between 0 and 100 (Colley, et al., 2011). As Fitbit does not release raw activity measures, we considered activity in steps. Thus a day with at least 10 valid hours was considered a valid PA day. To calculate non wear time, we identified periods with at least 60 consecutive minutes of 0 steps and added these minutes together. We then calculated wear time as:

Non-bed time = Bed time – wake up time

Wear time = Non-bed time – non wear time

**Table 1.** Method of measurement for height, weight, blood pressure, and HOMA-IR

| | **Method of Measurement** |
|---|---|
| **Height** | Standing height was measured with the subject in bare feet, back square against the wall and eyes looking straight ahead. A Harpenden stadiometer was used to measure height to the nearest 0.5 cm. The horizontal board was brought down to rest on top of the participant's head. Procedure was repeated and recorded. |
| **% body fat** | Participants stood in bare feet on bioelectrical impedance (InBody520). |
| **Weight** | Weight was measured in light clothing using an electronic scale, to the nearest 200 grams. The scales were standardized to 0 before each use. |
| **Blood Pressure** | Participant must have been resting for ≥ 5 minutes, and should not have smoked for at least 30 minutes before this measurement. Oscillometric model blood pressure machines were used. Adequate cuff size was ensured. Bladder encircled and covered 2/3 of length of arm with the bladder over the brachial artery. Its lower border was 1 inch above the anticubital space. Two readings on the right arm were taken, at least 5 minutes apart, and values were recorded. |
| **Fasting glucose** | Blood was drawn after a 12 hour fast. Glucose was measured by enzymatic reference method with hexokinase on the Roche INTEGRA analyzer. |
| **Fasting Insulin** | Blood was drawn after a 12 hour fast. Insulin was measured by immunometric Assay on the IMMULITE analyzer. |

**Table 2.** Daily sleep characteristics and physical activity calculations.

| | |
|---|---|
| **Daily sleep variables calculations** | Weekday average*:* sleep characteristic averaged across the number of days with Fitbit data from Sunday to Thursday night.<br><br>Weekend average*:* sleep characteristic averaged across the number of days with Fitbit data from Friday to Saturday night.<br><br>Average sleep variable:<br>([Weekday average of sleep variable × 5] + [Weekend average of sleep variable × 2]) ÷ 7 |
| **Daily physical activity variables calculations** | Weekday average*:* PA characteristic averaged across the number of days with Fitbit data from Monday to Friday.<br><br>Weekend average*:* PA characteristic averaged across the number of days with Fitbit data from Saturday to Sunday.<br><br>Average PA variable:<br>([Weekday average of PA variable × 5] + [Weekend average of PA variable × 2]) ÷ 7 |

### *5.8 Statistical Analysis*

Comparison of sample characteristics by gender was completed using an independent t-test for normally distributed data. For all variables not normally distributed in both groups (daily sleep duration, daily sleep latency, daily step count, daily very active minutes, and HOMA-IR) a non-parametric Mann-Whitney U test was used. Similarly, when comparing characteristics between prehypertensive/hypertensive and normotensive participants an independent t-test for normally distributed data was performed. For non-normally distributed variables in both groups (daily sleep latency, daily sleep fragmentation, and gender distribution) a non-parametric Mann-Whitney U test was used. In the univariate analysis, non-normally distributed data was transformed in order to use Pearson correlations. The square root of daily sleep latency, log of daily sleep fragmentation, log of daily very active minute, and the inverse of daily step count were computed to normalize the variables.

Multivariate regression models using the hierarchical method were used. Refer to Table 3 for regression models summary. Age and gender were controlled for. Age was included as sleep architecture changes with age (previously discussed), and physical activity decreases with age (Colley, et al., 2011). Physical activity also varies with gender (Colley, et al., 2011), therefore gender was controlled for. Adiposity was a variable of interest as there is no conclusive evidence that adiposity has an impact on sleep or physical activity in overweight/obese children. Age and gender were put in the model first (model A), followed by sleep characteristic (model B), followed by %BF (model C) for hypotheses 1 and 2. This allowed us to understand whether sleep characteristics are associated with each PA and cardiometabolic health outcome, independently of adiposity. In total there were 3 (A-C) models (Table 3).

Following the rule of thumb of 10 subjects/variable for multivariate regression analysis, as there are 6 variables, a sample size of 60 was needed for appropriate statistical power (Field & Miles, 2010). This was achieved for all of our outcomes, except for the insulin resistance analysis for which our sample size was 45 participants. All analysis was completed in SPSS version 21 (IBM, Armonk, New York), with statistical significance defined as p value<0.05.

**Table 3.** Regression models per outcome. Sleep characteristics for each model: 1) daily sleep duration, 2) daily sleep latency, and 3) daily sleep fragmentation. Each outcome was an outcome for 3 separate regression models/sleep characteristic.

| **Hypothesis Outcomes:** Daily step counts/, Daily very active minutes, systolic BP z-score, diastolic BP z-score, HOMA-IR | | |
|---|---|---|
| **Model A** | **Model B** | **Model C** |
| Age | Age | Age |
| Gender | Gender | Gender |
| | Sleep characteristic | Sleep characteristic |
| | | %BF |

# CHAPTER 6: RESULTS

## *6.1 Sample Characteristics*

Of the 155 participants recruited for the SPA study, 79 were included in this analysis (refer to Figure 4). The most common reason for exclusion was lack of sleep data. Participants excluded did not differ by age or extent of obesity but males were more likely excluded than females (Table 5). There was however no uneven gender distribution in our final sample (47% males). Sample characteristics of included participants are summarized in Table 6. By default of our inclusion, our participants were overweight/obese adolescents with an average age of 13.8 ±2.4, an average BMI z-score of 2.7 ±0.7 and an average %BF of 39.6 ±6.6. Females had greater body fat ($p=0.017$) yet BMI z-scores were similar to the males.

The average daily sleep duration was 489 min ±70 min. Only 9% of the participants met the recommended guidelines by the National Sleep Foundation of at least 510 min of sleep every night (National Sleep Foundation, 2013) they wore the Fitbit. On average, the recommended sleep duration guidelines were met on 43% of the nights with no differences between genders ($p=0.192$). The average daily sleep latency was 21 min ±13 min, and the average daily sleep fragmentation was 2.22 awakenings/hour ±1.08 awakenings/hour.

The average daily step count was 9038 ±2962. As for the recommended 12,000 steps/day guidelines (Colley, Janseen, & Tremblay, 2012), no participant met this recommendation every day when wearing the Fitbit. Moreover, 42% never met this recommendation on any day. The recommended steps/day guidelines were met on 25% of the days with no differences between genders ($p=0.193$). Correlations between sample characteristics sleep characteristics, and PA characteristics are shown in Table 6.

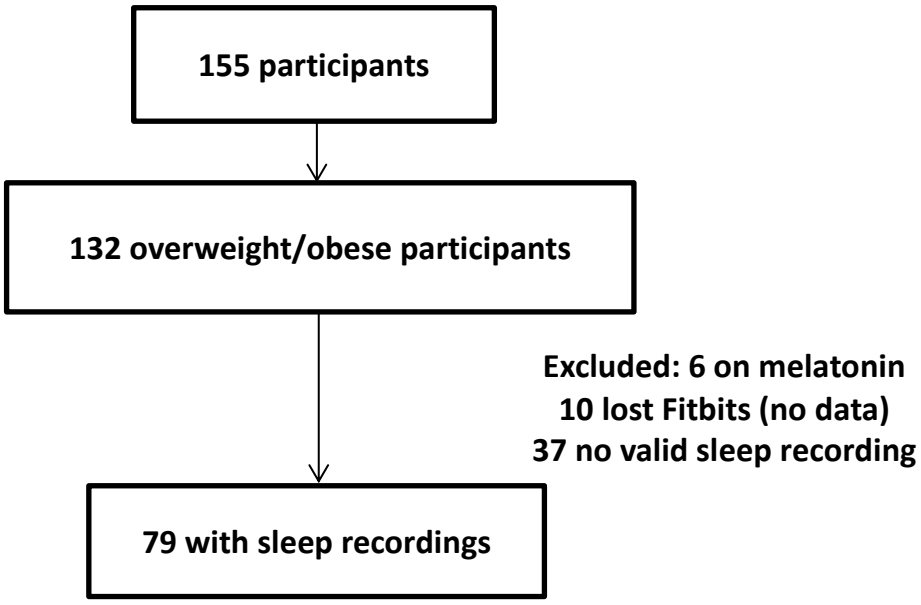



**Figure 4.** Breakdown of the number of participants included/excluded from sample.

**Table 4.** Comparison of age, gender, BMI z-score, and %BF between participants who were excluded and included in the sample.

| | **Included participants (n=79)** | **Excluded participants (n=53)** | **P value** |
|---|---|---|---|
| **Age mean (SD)** | 13.80 (2.4) | 13.89 (2.3) | .864 |
| **Gender #M, F** | 37 (47%), 42 | 39 (74%), 14 | **.002*** |
| **BMI Z-score mean (SD)** | 2.69 (.7) | 2.99 (1.0) | .116 |
| **%body fat mean (SD)** | 39.6 (6.6) | 39.11 (10.6) | .597 |

**Table 5.** Unadjusted sample characteristics of participants. BP=blood pressure.

| | ALL | | | Males | | Females | | |
|---|---|---|---|---|---|---|---|---|
| **Sample characteristics** | **Valid N** | **Mean (SD)** | **Range** | **Valid N** | **Mean (SD)** | **Valid N** | **Mean (SD)** | **P value** |
| Age | 79 | 13.8 (2.4) | 10-17 | 37 | 13.6 (2.4) | 42 | 13.9 (2.4) | .656 |
| Height (cm) | 79 | 162.5 (10.6) | 140-186 | 37 | 164.7 (10.8) | 42 | 160.5 (10.2) | .082 |
| Weight (kg) | 79 | 82.4 (22.9) | 47-138 | 37 | 84.1 (23) | 42 | 81 (23) | .539 |
| Height Z-score | 79 | .77 (1.23) | -2.1 – 3.9 | 37 | .89 (1.23) | 42 | .66 (1.25) | .404 |
| BMI Z-score | 79 | 2.7 (0.7) | 1.03-4.65 | 37 | 2.8 (0.6) | 42 | 2.6 (0.8) | .180 |
| % body fat | 79 | 39.6 (6.6) | 23.3-53.3 | 37 | 37.9 (6.2) | 42 | 41.2 (6.2) | **.017*** |
| Days of valid sleep/participant | 79 | 6.2 (0.9) | 4-7 | 37 | 6.1 (0.9) | 42 | 6.3 (0.9) | .400 |
| Daily sleep duration (min) | 79 | 489 (70) | 330-737 | 37 | 480.9 (74) | 42 | 495.4 (66) | .280 |
| Daily sleep latency (min) | 79 | 21 (13) | 0.71-74.2 | 37 | 20.6 (13.9) | 42 | 20.7 (12.2) | .768 |
| Daily sleep fragmentation (awakenings/hour/day) | 79 | 2.22 (1.08) | 0.6-6 | 37 | 2.28 (1.02) | 42 | 2.15 (1.14) | .259 |
| Days of valid PA/participants | 76 | 6.3 (0.87) | 4-7 | 35 | 6.3 (0.83) | 41 | 6.3 (0.91) | .725 |
| Daily step count (steps/day) | 76 | 9038 (2962) | 3010-15287 | 35 | 9581 (3215) | 41 | 8575 (2681) | .149 |
| Daily very active minutes (min/day) | 76 | 22 (17) | 2.24-89 | 35 | 26.5 (21) | 41 | 18 (11) | .122 |
| Daily wear time (min/day) | 76 | 728 (108) | 458-956 | 35 | 737.9 (102.8) | 41 | 719.6 (114.3) | .379 |
| Daily step count/min wear time (steps/min/day) | 76 | 13.6 (6.1) | 5.67-47.01 | 35 | 15.08 (8.2) | 41 | 12.26 (2.9) | .117 |
| Daily very active (min/ min wear time) | 76 | 0.030 (.03) | 0-0.11 | 35 | 0.038 (0.03) | 41 | 0.026 (0.015) | .221 |
| Systolic BP (mm Hg) | 73 | 116 (9.5) | 92-137 | 34 | 116.6 (10) | 39 | 114.8 (9.1) | .440 |
| Diastolic BP (mm Hg) | 73 | 69 (6.9) | 53-88 | 34 | 68 (6) | 39 | 70.2 (7.5) | .171 |
| Systolic BP z-score | 73 | 0.47 (0.76) | -1.77-2.12 | 34 | 0.42 (0.8) | 39 | 0.51 (0.7) | .589 |
| Diastolic BP z-score | 73 | 0.38 (0.57) | -0.81-1.87 | 34 | 0.27 (0.5) | 39 | 0.49 (0.6) | .097 |
| Fasting glucose (mmol/L) | 45 | 4.8 (0.56) | 3.4-6.8 | 22 | 4.9 (0.6) | 23 | 4.8 (.56) | .370 |
| Fasting insulin (pmol/L) | 45 | 118.7 (132) | 14-654 | 22 | 113 (128) | 23 | 124 (137) | .946 |
| HOMA-IR | 45 | 2.2 (2.2) | 0.42-11.11 | 22 | 2.1 (2.1) | 23 | 2.2 (2.4) | .820 |

**Table 6.** Univariate analysis of relationship between age, %BF, sleep characteristics, PA characteristics, and cardiometabolic health outcomes. Sqrt=square root, SBP= systolic blood pressure, DBP=diastolic blood pressure. * p<0.05 level.** p< 0.01 level.

| | **Age** | **%BF** | **Average sleep duration (min)** | **Sqrt average sleep latency (min)** | **Log average sleep fragmentation** |
|---|---|---|---|---|---|
| **%body fat** | **.24*** | | | | |
| **Daily sleep duration (min)** | **-.52**** | -.03 | | | |
| **Square root daily sleep latency (min)** | -.04 | .07 | -.04 | | |
| **Log daily sleep fragmentation** | -.04 | -.07 | .003 | .12 | |
| **Daily step count** | -.05 | -.18 | -.11 | -.18 | -.02 |
| **Log daily very active minutes** | -.005 | **-0.26*** | -.18 | -.07 | -.07 |
| **Inverse daily step count/wear time** | -.02 | .21 | .20 | .19 | -.11 |
| **Log daily very active minutes/wear time** | .03 | **-.24*** | -.19 | -.10 | -.04 |
| **SBP z-score** | .06 | -.17 | .04 | -.10 | .04 |
| **DBP z-score** | .14 | .23 | .08 | -.14 | -.16 |
| **Log HOMA-IR** | **.29*** | **.30*** | -.11 | .26 | -.04 |

### *6.2 Sleep and Physical Activity*

To determine the relationship between sleep and PA for hypothesis 1, both univariate and multivariate analyses were done. There was no significant relationship between sleep characteristics (sleep duration, sleep latency, and sleep fragmentation) and PA (average step count or very active minutes) in our univariate analysis (Table 7). Sleep characteristics remained insignificant correlates of PA even when corrected for age and gender in our multivariate regression models (Table 8 and 9). Daily very active minutes was significantly correlated with %BF (p=.022) in our univariate analysis. Body fat continued to be related to daily very active minutes when corrected for age, gender, and sleep, but inclusion of body fat did not change the relationship of sleep to PA (Table 9). Interestingly when corrected for wear time, daily steps was weakly influenced by both sleep duration and sleep latency (Table 10 and 11). These were very small effects however and predicted 4.3% and 3.9% of the variance in daily steps, respectively.

### *6.3 Sleep and Blood Pressure*

To determine the relationship between sleep and cardiometabolic health for hypothesis 2, both univariate and multivariate analyses were done. Sleep characteristics were not related to systolic BP z-score or diastolic BP z-score in either univariate analysis (Table 7) or multivariate analyses (Table 12 and Table 13). Roughly 15% of our sample was classified as prehypertensive/hypertensive. When comparing sleep characteristics of prehypertensive and hypertensive participants to normotensive participants, there were no significant sleep characteristic differences (Table 14).

### *6.4 Sleep and Insulin Resistance*

Sleep latency was directly related to HOMA-IR independently of age and gender (Table 14). For every 10 minute increase in sleep latency, HOMA-IR increased by 0.55. Gender did not

influence HOMA-IR but there was a weak effect of age with an increase in age predicting increased insulin resistance. This may be expected given the known influence of puberty on insulin resistance (Bloch, Clemons, & Sperling, 1987), (Moran, et al., 1999). Age, gender, and sleep latency explained 17% of the variance in HOMA-IR. After adjusting for %BF, the relationship between sleep latency and HOMA-IR was weak and no longer significant. The relationship between sleep latency and HOMA-IR may be therefore partially explained by %BF.

**Table 7.** Multivariate regression analysis examining daily step count. N=76.

| | **Model 1A** | | | | **Model 1B** | | | | **Model 1C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Daily step count** | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | 10223.5 | 1995.4 | | <0.001 | 15104.0 | 4883.2 | | .003 | 16506.3 | 5052.6 | | .002 |
| **Age** | -47.2 | 142.2 | -.04 | .741 | -152.9 | 171.7 | -.12 | .376 | -109.0 | 176.4 | -.09 | .539 |
| **Gender** | -990.5 | 682.2 | -.17 | .151 | -832.8 | 696.4 | -.14 | .236 | -651.0 | 716.3 | -.11 | .366 |
| **Daily sleep duration** | | | | | -7.2 | 6.6 | -.16 | .277 | -6.7 | 6.6 | -.15 | .304 |
| **% body fat** | | | | | | | | | -58.0 | 54.4 | -.13 | .290 |
| $R^2$ | | .031 | | | | .046 | | | | .061 | | |
| Adju $R^2$ | | .004 | | | | .007 | | | | .009 | | |
| P value | | .322 | | | | .328 | | | | .335 | | |

| | **Model 2A** | | | | **Model 2B** | | | | **Model 2C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | 10223.5 | 1995.4 | | <.001 | 12077.1 | 2284.1 | | <.001 | 13558.9 | 2711.2 | | <.001 |
| **Age** | -47.2 | 142.2 | -.04 | .741 | -54.5 | 140.7 | -.04 | .700 | -18.1 | 145.2 | -.02 | .901 |
| **Gender** | -990.5 | 682.2 | -.17 | .151 | -997.5 | 674.9 | -.17 | .144 | -817.6 | 697.7 | -.14 | .245 |
| **Daily sleep latency** | | | | | -401.5 | 248.9 | -.18 | .111 | -381.9 | 249.6 | -.18 | .130 |
| **% body fat** | | | | | | | | | -54.7 | 54.9 | -.12 | .314 |
| $R^2$ | | .031 | | | | .064 | | | | .078 | | |
| Adju $R^2$ | | .004 | | | | .025 | | | | .026 | | |
| P value | | .322 | | | | .185 | | | | .213 | | |

**Table 7.**Continued.

| Daily step count | Model 3A | | | | Model 3B | | | | Model 3C | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | 10223.5 | 1995.4 | | <.001 | 10052.2 | 3092.7 | | .002 | 11703.1 | 3420.1 | | .001 |
| **Age** | -47.2 | 142.2 | -.04 | .741 | -47.1 | 142,1 | -.04 | .743 | -6.6 | 147.4 | -.01 | .964 |
| **Gender** | -990.5 | 682.2 | -.17 | .151 | -998.1 | 694.8 | -.17 | .155 | -801.7 | 715.3 | -.14 | .266 |
| **Daily sleep fragmentation** | | | | | -118.3 | 1623.7 | -.01 | .942 | -196.0 | 1622.3 | -.01 | .904 |
| **% body fat** | | | | | | | | | -61.4 | 54.7 | -.14 | .266 |
| $R^2$ | | .031 | | | | .031 | | | | .047 | | |
| Adju $R^2$ | | .004 | | | | -.010 | | | | -.006 | | |
| P value | | .322 | | | | .521 | | | | .478 | | |

**Table 8.** Multivariate regression analysis examining daily very active minutes. N=76. Sqrt=square root.

| Daily very active minutes | Model 1A | | | | Model 1B | | | | Model 1C | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | 1.27 | .22 | | <.001 | 2.01 | .54 | | <.001 | 2.4 | .55 | | <.001 |
| **Age** | .001 | .02 | .01 | .962 | -.02 | .02 | -.12 | .378 | -.01 | .02 | -.06 | .691 |
| **Gender** | -.09 | .08 | -.14 | .234 | -.07 | .08 | -.09 | .400 | -.03 | .08 | -.04 | .728 |
| **Daily sleep duration** | | | | | -.001 | .001 | -.23 | .104 | -.001 | .001 | -.22 | .121 |
| **% body fat** | | | | | | | | | -.01 | .01 | -.25 | **.042*** |
| $R^2$ | | .019 | | | | .055 | | | | .109 | | |
| Adju $R^2$ | | -.007 | | | | .016 | | | | .059 | | |
| P value | | .489 | | | | .250 | | | | .081 | | |

| | Model 2A | | | | Model 2B | | | | Model 2C | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | 1.27 | .22 | | <.001 | .001 | .26 | | <.001 | 1.7 | .30 | | <.001 |
| **Age** | .001 | .02 | .01 | .962 | -.09 | .02 | .003 | .979 | .01 | .02 | .06 | .590 |
| **Gender** | -.09 | .08 | -.14 | .234 | -.02 | .08 | -.14 | .234 | -.05 | .08 | -.08 | .513 |
| **Sqrt daily sleep latency** | | | | | | .03 | -.08 | .524 | -.01 | .03 | -.06 | .624 |
| **% body fat** | | | | | | | | | -.01 | .01 | -.25 | **.041*** |
| $R^2$ | | .019 | | | | .025 | | | | .081 | | |
| Adju $R^2$ | | -.007 | | | | -,016 | | | | .029 | | |
| P value | | .489 | | | | .608 | | | | .193 | | |

**Table 8.** Continued

| **Daily very active minutes** | **Model 3A** | | | | **Model 3B** | | | | **Model 3C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | 1.3 | .22 | | <.001 | 1.1 | .34 | | <.001 | 1.4 | .37 | | <.001 |
| **Age** | .001 | .02 | .01 | .962 | .001 | .02 | .01 | .957 | .01 | .02 | .07 | .560 |
| **Gender** | -.09 | .08 | -.14 | .234 | -.10 | .08 | -.15 | .198 | -.06 | .08 | -.09 | .450 |
| **Log daily sleep fragmentation** | | | | | -.14 | .18 | -.09 | .448 | -.15 | .18 | -.1 | .385 |
| **%body fat** | | | | | | | | | -.01 | .01 | -.26 | **.033*** |
| $R^2$ | | .019 | | | | .027 | | | | .088 | | |
| Adju $R^2$ | | -.007 | | | | -.013 | | | | .036 | | |
| P value | | .489 | | | | .572 | | | | .158 | | |

**Table 9.** Multivariate regression analysis examining average step count/wear time and sleep characteristics. N=76. Sqrt=square root.

| | **Model 1A** | | | | **Model 1B** | | | | **Model 1C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Daily step count/wear time** | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | .08 | .018 | | <.001 | .011 | .04 | . | .800 | -.01 | .05 | | .853 |
| **Age** | .001 | .001 | -.03 | .797 | .001 | .002 | .11 | .427 | .001 | .002 | .06 | .692 |
| **Gender** | .01 | .01 | .13 | .253 | .005 | .01 | .09 | .443 | .002 | .01 | .04 | .719 |
| **Daily Sleep duration** | | | | | .001 | .001 | .25 | .075 | .001 | .001 | .24 | .087 |
| **% body fat** | | | | | | | | | .001 | .001 | .20 | .099 |
| $R^2$ | | .018 | | | | .061 | | | | .096 | | |
| Adju $R^2$ | | -.009 | | | | .022 | | | | .045 | | |
| P value | | .510 | | | | .208 | | | | .121 | | |

| | **Model 2A** | | | | **Model 2B** | | | | **Model 2C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | .08 | .02 | | <.001 | .07 | .021 | | .002 | .04 | .03 | | .074 |
| **Age** | .01 | .001 | -.03 | .797 | .001 | .001 | -.02 | .837 | -.001 | .001 | -.07 | .546 |
| **Gender** | .01 | .01 | .13 | .253 | .01 | .01 | .14 | .242 | .005 | .01 | .09 | .463 |
| **Sqrt daily sleep latency** | | | | | .004 | .002 | .19 | .091 | .004 | .002 | .18 | .114 |
| **% body fat** | | | | | | | | | .001 | .001 | .19 | .107 |
| $R^2$ | | .018 | | | | .057 | | | | .091 | | |
| Adju $R^2$ | | -.009 | | | | .017 | | | | .040 | | |
| P value | | .510 | | | | .238 | | | | .144 | | |

**Table 9.**Continued.

| | Model 3A | | | | Model 3B | | | | Model 3C | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Daily step count/wear time** | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | .08 | .02 | | <.001 | .07 | .03 | | .021 | .04 | .03 | | .159 |
| **Age** | .001 | .001 | -.03 | .797 | .001 | .001 | -.03 | .803 | -.001 | .001 | -.08 | .509 |
| **Gender** | .01 | .01 | .13 | .253 | .01 | .01 | .12 | .313 | .004 | .01 | .07 | .564 |
| **Log daily sleep fragmentation** | | | | | -.01 | .02 | -.09 | .419 | -.01 | .02 | -.08 | .455 |
| **% body fat** | | | | | | | | | .001 | .001 | .21 | .092 |
| $R^2$ | .018 | | | | .027 | | | | .066 | | | |
| Adju $R^2$ | -.009 | | | | -.013 | | | | .013 | | | |
| P value | .510 | | | | .572 | | | | .300 | | | |

**Table 10.** Multivariate regression analysis examining daily very active minutes/wear time. N=76. Sqrt=square root.

| | **Model 1A** | | | | **Model 1B** | | | | **Model 1C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Daily very active minutes/wear time** | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -1.6 | .21 | | <.001 | -.84 | .51 | | .107 | -.57 | .52 | | .271 |
| **Age** | .01 | .02 | .04 | .751 | -.01 | .02 | -.09 | .492 | -.004 | .02 | -.03 | .813 |
| **Gender** | -.09 | .07 | -.15 | .214 | -.07 | .07 | -.10 | .376 | -.03 | .07 | -.05 | .671 |
| **Daily sleep duration** | | | | | -.001 | .001 | -.24 | .093 | -.001 | .001 | -.22 | .109 |
| **% body fat** | | | | | | | | | -.01 | .01 | -.23 | .060 |
| $R^2$ | | .022 | | | | .060 | | | | .106 | | |
| Adju $R^2$ | | -.005 | | | | .020 | | | | .055 | | |
| P value | | .447 | | | | .216 | | | | .090 | | |

| | **Model 2A** | | | | **Model 2B** | | | | **Model 2C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -1.6 | .21 | | <.001 | -1.5 | .25 | | <.001 | -1.2 | .29 | | <.001 |
| **Age** | .01 | .02 | .04 | .751 | .004 | .02 | .03 | .774 | .01 | .02 | .09 | .451 |
| **Gender** | -.09 | .07 | -.15 | .214 | -.09 | .07 | -.15 | .212 | -.06 | .07 | -.09 | .454 |
| **Sqrt daily sleep latency** | | | | | -.02 | .03 | -.10 | .374 | -.02 | .03 | -.09 | .450 |
| **% body fat** | | | | | | | | | -.01 | .01 | -.23 | .060 |
| $R^2$ | | .022 | | | | .033 | | | | .080 | | |
| Adju $R^2$ | | -.005 | | | | -.008 | | | | .028 | | |
| P value | | .447 | | | | .494 | | | | .199 | | |

**Table 10.**Continued.

| Daily very active minutes/wear time | Model 3A | | | | Model 3B | | | | Model 3C | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -1.6 | .21 | | <.001 | -1.8 | .33 | | <.001 | -1.5 | .36 | | <.001 |
| **Age** | .01 | .02 | .04 | .751 | .01 | .02 | .04 | .749 | .01 | .02 | .09 | .422 |
| **Gender** | -.09 | .07 | -.15 | .214 | -.09 | .07 | -.16 | .193 | -.06 | .07 | -.09 | .420 |
| **Log daily sleep fragmentation** | | | | | -.09 | .17 | -.06 | .594 | -.11 | .17 | -.07 | .530 |
| **% body fat** | | | | | | | | . | -.01 | .01 | -.24 | **.049*** |
| $R^2$ | | .022 | | | | .026 | | | | .078 | | |
| Adju $R^2$ | | -.005 | | | | -.015 | | | | .026 | | |
| P value | | .447 | | | | .597 | | | | .213 | | |

**Table 11.** Multivariate regression analysis examining systolic blood pressure (SBP) z-score.N=73. Sqrt=square root.

| | **Model 1A** | | | | **Model 1B** | | | | **Model 1C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Systolic BP z-score** | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | .15 | .54 | | .783 | -.72 | 1.3 | | .568 | .01 | 1.3 | | .992 |
| **Age** | .02 | .04 | .06 | .607 | .04 | .05 | .13 | .388 | .06 | .05 | .18 | .228 |
| **Gender** | .09 | .18 | .06 | .605 | .08 | .18 | .05 | .682 | .17 | .19 | .11 | .364 |
| **Daily Sleep duration** | | | | | .001 | .002 | .11 | .445 | .001 | .002 | .13 | 381 |
| **% body fat** | | | | | | | | | -.03 | .02 | -.23 | .068 |
| $R^2$ | | .008 | | | | .016 | | | | .064 | | |
| Adju $R^2$ | | -.020 | | | | -.026 | | | | .008 | | |
| P value | | .757 | | | | .766 | | | | .339 | | |

| | **Model 2A** | | | | **Model 2B** | | | | **Model 2C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | .15 | .54 | | .783 | .39 | .61 | | .519 | 1.1 | .74 | | .128 |
| **Age** | .02 | .04 | .06 | .607 | .02 | .04 | .06 | .601 | .03 | .04 | .10 | .394 |
| **Gender** | .09 | .18 | .06 | .605 | .09 | .18 | .06 | .607 | .19 | .19 | .12 | .322 |
| **Sqrt avg. SL** | | | | | -.06 | .07 | -.1 | .389 | -.05 | .07 | -.08 | .478 |
| **%BF** | | | | | | | | | -.03 | .02 | -.22 | .088 |
| $R^2$ | | .008 | | | | .019 | | | | .060 | | |
| Adju $R^2$ | | -.020 | | | | -.024 | | | | .005 | | |
| P value | | .757 | | | | .728 | | | | .372 | | |

**Table 11.** Continued.

| **Systolic BP z-score** | **Model 3A** | | | | **Model 3B** | | | | **Model 3C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | .15 | .54 | | .783 | .41 | .78 | | .606 | 1.2 | .88 | | .190 |
| **Age** | .02 | .04 | .06 | .607 | .02 | .04 | .07 | .585 | .04 | .04 | .11 | .380 |
| **Gender** | .09 | .18 | .06 | .605 | .11 | .18 | .07 | .567 | .19 | .19 | .13 | .296 |
| **Log daily sleep fragmentation** | | | | | .19 | .42 | .06 | .651 | .15 | .41 | .04 | .716 |
| **% body fat** | | | | | | | | | -.03 | .02 | -.22 | .080 |
| $R^2$ | | .008 | | | | .011 | | | | .055 | | |
| Adju $R^2$ | | -.020 | | | | -.032 | | | | -.001 | | |
| P value | | .757 | | | | .859 | | | | .422 | | |

**Table 12.** Multivariate regression analysis examining diastolic blood pressure. N=73. Sqrt=square root.

| | **Model 1A** | | | | **Model 1B** | | | | **Model 1C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Diastolic blood pressure z-score** | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -.16 | .39 | | .682 | -1.4 | .90 | | .126 | -1.8 | .95 | | .066 |
| **Age** | .03 | .03 | .13 | .268 | .06 | .04 | .26 | .076 | .05 | .04 | .22 | .129 |
| **Gender** | .22 | .13 | .19 | .105 | .19 | .13 | .17 | .154 | .14 | .14 | .13 | .304 |
| **Daily sleep duration** | | | | | .002 | .001 | .22 | .133 | .002 | .001 | .21 | .150 |
| **% body fat** | | | | | | | | | .01 | .01 | .16 | .206 |
| $R^2$ | | .055 | | | | .086 | | | | .107 | | |
| Adju $R^2$ | | .028 | | | | .046 | | | | .055 | | |
| P value | | .137 | | | | .101 | | | | .098 | | |

| | **Model 2A** | | | | **Model 2B** | | | | **Model 2C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -.16 | .39 | | .682 | .09 | .45 | | .839 | -.38 | .54 | | .491 |
| **Age** | .03 | .03 | .13 | .268 | .03 | .03 | .13 | .261 | .02 | .03 | .09 | .407 |
| **Gender** | .22 | .13 | .19 | .105 | .22 | .13 | .19 | .104 | .16 | .14 | .14 | .247 |
| **Sqrt daily sleep latency** | | | | | -.06 | .05 | -.14 | .227 | -.07 | .05 | -.16 | .177 |
| **% body fat** | | | | | | | | | .02 | .01 | .18 | .144 |
| $R^2$ | | .055 | | | | .075 | | | | .104 | | |
| Adju $R^2$ | | .028 | | | | .035 | | | | .051 | | |
| P value | | .137 | | | | .143 | | | | .109 | | |

**Table 12.** Continued.

| **Diastolic blood pressure z-score** | **Model 3A** | | | | **Model 3B** | | | | **Model 3C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -.16 | .39 | | .682 | -.61 | .57 | | .286 | -1.0 | .65 | | .121 |
| **Age** | .03 | .03 | .13 | .268 | .03 | .03 | .12 | .308 | .02 | .03 | .09 | .445 |
| **Gender** | .22 | .13 | .19 | .105 | .19 | .13 | .17 | .145 | .15 | .14 | .13 | .290 |
| **Log daily sleep fragmentation** | | | | | -.33 | .30 | -.13 | .275 | -.31 | .30 | -.12 | .305 |
| **% body fat** | | | | | | | | | .01 | .01 | .16 | .201 |
| $R^2$ | | .055 | | | | .071 | | | | .094 | | |
| Adju $R^2$ | | .028 | | | | .031 | | | | .040 | | |
| P value | | .137 | | | | .161 | | | | .148 | | |

**Table 13.** Characteristics comparison between prehypertensive+hypertensive participants and normotensive participants. N=73

| | **Prehypertensive + hypertensive (n=11)** | **Normotensive (n=62)** | **P value** |
|---|---|---|---|
| **Age mean (SD)** | 14.4 (2.5) | 13.4 (2.3) | .180 |
| **Gender #M, F** | 4, 7 | 30, 32 | .464 |
| **BMI z-score mean (SD)** | 2.7 (0.7) | 2.7 (0.7) | .950 |
| **% body fat mean (SD)** | 38.9 (7.1) | 39.2 (6.2) | .887 |
| **Daily sleep duration (min) mean (SD)** | 493 (123) | 487 (61) | .881 |
| **Daily sleep latency (min) mean (SD)** | 21 (14) | 21 (13) | .638 |
| **Daily sleep fragmentation mean (SD)** | 2.14 (1.0) | 2.26 (1.1) | .805 |

**Table 14.** Multivariate regression analysis examining HOMA-IR.N=45. Sqrt=square root.

| | **Model 1A** | | | | **Model 1B** | | | | **Model 1C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **HOMA-IR** | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -.298 | .26 | | .265 | -.65 | .61 | | .292 | -.99 | .62 | | .117 |
| **Age** | .047 | .02 | .29 | .050 | .05 | .02 | .37 | .057 | .04 | .02 | .31 | .105 |
| **Gender** | -.02 | .09 | -.03 | .819 | -.02 | .09 | -.03 | .854 | -.06 | .09 | -.11 | .487 |
| **Daily sleep duration** | | | | | .001 | .001 | .12 | .526 | .001 | .001 | .11 | .555 |
| **%BF** | | | | | | | | | .01 | .01 | .28 | .083 |
| $R^2$ | | .089 | | | | .098 | | | | .164 | | |
| Adju $R^2$ | | .045 | | | | .032 | | | | .080 | | |
| P value | | .142 | | | | .234 | | | | .119 | | |

| | **Model 2A** | | | | **Model 2B** | | | | **Model 2C** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -.298 | .26 | | .265 | -.61 | .296 | | .046 | ..88 | .34 | | .015 |
| **Age** | .047 | .02 | .29 | .050 | .04 | .02 | .33 | **.026*** | .04 | .02 | .28 | .058 |
| **Gender** | -.02 | .09 | -.03 | .819 | -.001 | .08 | -.002 | .989 | -.04 | .09 | -.07 | .640 |
| **Sqrt daily sleep latency** | | | | | .055 | .03 | .295 | **.047*** | .05 | .03 | .25 | .092 |
| **% body fat** | | | | | | | | | .01 | .01 | .22 | .152 |
| $R^2$ | | .089 | | | | .173 | | | | .215 | | |
| Adju $R^2$ | | .045 | | | | .113 | | | | .137 | | |
| P value | | .142 | | | | **.048*** | | | | **.042*** | | |

**Table 14.** Continued.

| | Model 3A | | | | Model 3B | | | | Model 3C | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **HOMA-IR** | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value | Unstd β | Std Error | Std β | P value |
| **Constant** | -.298 | .26 | | .265 | -.23 | .47 | | .538 | -.56 | .39 | | .157 |
| **Age** | .047 | .02 | .29 | .050 | .04 | .02 | .31 | .053 | .03 | .02 | .27 | .089 |
| **Gender** | -.02 | .09 | -.03 | .819 | -.01 | .09 | -.02 | .896 | -.05 | .09 | -.09 | .573 |
| **Log daily sleep fragmentation** | | | | | .07 | .24 | .05 | .786 | .14 | .24 | .09 | .558 |
| **% body fat** | | | | | | | | | .01 | .01 | .29 | .068 |
| $R^2$ | | .089 | | | | .090 | | | | .164 | | |
| Adju $R^2$ | | .045 | | | | .024 | | | | .08 | | |
| P value | | .142 | | | | .269 | | | | .120 | | |

# CHAPTER 7: DISCUSSION

## *7.1 Sleep Characteristics*

The average daily sleep duration of our sample was 489 min (8.15 hours). The National Sleep Foundation recommends 8.5-9.25 hours for this age group (National Sleep Foundation, 2013), and on average our population was slightly below these recommendations. A very small proportion of our population, 9%, met these guidelines every night. Although our sample's sleep duration is below the recommended guidelines, it is slightly above the average American adolescent self-reported sleep of roughly 8 hours. This was a population poll performed in 1,602 adolescents ages 11-17 (National Sleep Foundation, 2006). Although it is self-reported, it is one of the few studies looking at sleep in a large population. Large population studies of accelerometer sleep measurement in adolescents are lacking.

Though not conducted in such large populations, studies have used objective sleep tools in adolescents and have reported lower sleep duration than what we found. Gupta et al. (2014) (N=383, 11-16 yrs, Motionlogger accelerometer) reported an average of 7.68 hours of sleep and Javaheri et al. (2008) (N=238, 13-15 yrs, Actiwatch accelerometer) reported an average of 7.71 hours of sleep. Both IglayReger et al. (2014) (N=37, 11-17 yrs, Sensewear accelerometer) and Sung et al. (2011) (N=133, 10-16.9, Mini Motionlogger) measured sleep duration using accelerometers in overweight/obese adolescents recruited from weight management programs. These populations were very similar to ours in terms of age, overweight status, and type of clinic recruited from. The average sleep duration reported by IglayReger et al. was 7.1 hours and by Sung et al. was 7.8 hours, both shorter than the sleep duration in our sample. Furthermore, the ranges reported by IglayReger et al. (216-558) and Sung et al. (294-570) were considerably lower than the range in our sample. In comparison, our population appears to have greater sleep

duration than both adolescents who self-reported sleep, and adolescents who had sleep objectively measured (both overweight/obese and lean). The variation in objective sleep measurements between studies may have played a role in these differences.

### *7.2 Physical Activity Characteristics*

The average daily step count in our sample (9038) was lower than the average step count in a population representative sample of 11-19 year old Canadian adolescents (10,674) (Colley, et al., 2011). The average daily step count of males in our sample (9581) was lower than the average step count of males in the general population of overweight and obese males: 11, 188 and 10, 256, respectively. Similarly, the average daily step count of the females in our sample (8575) was also lower than the average step count of the general population of females and of overweight and obese females: 10, 560 and 11, 159, respectively (Colley, et al., 2011). Our population had a lower average step count than an average Canadian adolescent, an average Canadian overweight/obese adolescent, and lower than the recommended guidelines of 12, 000 steps/day. The inclusion criteria of at least 4 days with 1 weekend day was similar in this population based study, however average wear time was 13.9 hours, slightly above the 12.1 hours of wear time in our sample. Therefore, our sample may have had a lower average daily step count due to less activity, or less wear time.

An additional reason for the observed step count difference could be the different monitors used. While we used the Fitbit Ultra and the Fitbit One, Colley et al. (2011) used an Actical accelerometer (Phillips Respironics, Oregon, USA). Noah et al. (2013) compared both the Fitbit Ultra to an Actical accelerometer (Phillips Respironices, Inc., Andover, MA). As discussed in Appendix A, a significant difference was found between the Fitbit Ultra and Actical while jogging or walking on an incline as the Actical generated a higher step count.

### *7.3 Sleep and Physical Activity*

Although there were no significant relationships between sleep characteristics and PA measured either as daily step count or as daily very active minutes, when daily step count was corrected for wear time we did find a weak relationship between sleep duration and step count, as well as sleep latency and step count. It appears that sleep may influence total steps, and not more intense activity. However, as this is a cross-sectional study the direction of the relationship of sleep and PA cannot be determined.

As mentioned in section 1, the relationship between sleep and PA has been inconsistent in the literature. Moreover, few studies examined the relationship between sleep and PA using objective tools for >24hrs for both measures. Gupta et al. (2012) measured both sleep and PA for 24 hrs using accelerometers and reported a significant inverse relationship between sleep disturbance and total activity counts. The length of measurement and PA variable differ from our study, and may explain the differences in findings. Furthermore, their sample population was a mixture of lean/overweight/obese adolescents with a wider %BF range (1.6-43.9) than ours. Stone et al. (2013) used an accelerometer for PA (7 day wear) and questionnaire for sleep measurement in 10-12 yr olds, and reported similar findings to Gupta et al. (2012). They found that children who slept <9 hrs spent less time in more intense PA than those who slept ≥10 hrs. Pesonen et al. (2011) objectively measured sleep and PA for 24 hrs in 8 year olds. Contrasting Gupta et al., they reported that sleep duration and efficiency was associated with decreased PA levels the next day.

Our findings may differ from these studies for several reasons. We used objective measurement for both sleep and PA, and we used these for a longer time period which may be able to better represent the participants' general trends of sleep and PA. Furthermore, while we

observed a weak relationship between step count and sleep variables, the aforementioned studies did not measure step count. Lastly, while we adjusted for wear time these studies have not.

We also examined an overweight/obese population. It is possible that we were unable to detect a significant relationship between sleep and PA because it is not applicable to overweight/obese children. A polysomnographic study comparing overweight and lean 10-16.9 yr olds reported that the overweight adolescents spent more time in stage 3 sleep even though sleep duration was not significantly different (Beebe, et al., 2007). As overweight adolescents may spend more time in SWS, it may be that overweight/obese adolescents do not feel as tired as lean adolescents. Thus, the hypothesis that poor sleep does not restore body energy causing lower PA levels may not be applicable in overweight/obese adolescents. However, more polysomnographic studies need to be conducted in comparing lean and overweight/obese adolescents for a further understanding of differences in sleep stages.

### *7.4 Sleep and Blood Pressure*

We found no significant relationship between sleep characteristics and BP in either our univariate or multivariate. Javaheri et al. (2012) objectively measured sleep for 7 days and reported that adolescents with low sleep efficiency were at 3.5-fold and adolescents with short sleep duration (<6.5 hrs) were at 2.5-fold increased odds of being prehypertensive/hypertensive. We did not see a significant difference in systolic BP z-score ($p=0.059$) and diastolic BP z-score ($p=0.517$) between participants who slept <6.5 hrs ($n=8$) and those who slept >6.5 hrs ($n=65$). As the difference in systolic BP z-score is approaching significance, perhaps with a larger sample of sleepers with <6.5 hrs, a significant difference would have been found.

Referring back to Figure 3, it is the SWS that could have an impact on BP, and as mentioned earlier overweight adolescents may have greater SWS, possibly explaining why we

did not see a relationship. Furthermore, perhaps cortisol was not heightened in our sample, similarly seen in healthy adolescent boys by Klingenberg et al. (2013). The results found by Sung et al. (2011) supported our findings that sleep duration measured by accelerometers was not associated with BP in obese adolescents recruited from a tertiary care weight-management clinic. When we compared normotensive and prehypretensive+hypertensive, we did not find significant differences in sleep characteristics. The number of participants in the 2 groups was uneven (62 and 11); therefore it would be beneficial to reproduce this analysis in a larger group of prehypertensive/hypertensive participants.

Interestingly %BF was not associated with BP z-scores, which could be partially explained by the fact that our population‘s %BF is on the higher end on the spectrum and are in pubertal age range. The alternative is that once an adolescent is overweight/obese the %BF variation is not a predictor of hypertension, and there are other factors playing a role

### *7.5 Sleep and Insulin Resistance*

Sleep latency was a predictor of increased insulin resistance. Longer sleep latency has been previously reported in overweight adolescents (Beebe, et al., 2007). When the model was adjusted for %BF, this relationship was no longer significant suggesting that body fat may in some way be mediating this relationship. We saw no relationship of sleep fragmentation or sleep duration on HOMA-IR.

Studies looking at the relationship between sleep latency and insulin resistance in the pediatric population are lacking. Moreover, the majority of adult studies look at sleep duration and not sleep quality (McNeil, Doucet, & Chaput, 2013). An 8 year prospective study followed 2,265 Japanese males and reported that participants who reported greater difficulty falling asleep had a higher hazard ratio for type 2 diabetes (Kawakami, Takatsuka, & Shimizu, 2004).

Sung et al. (2011) did not find a significant relationship between HOMA-IR and sleep duration either in obese adolescents recruited from a tertiary care weight-management clinic. Koren et al. (2011) performed PSG tests on obese adolescents and reported a U-shaped relationship between sleep and glucose homeostasis (2 hr glucose and HbA1c). They also reported that stage 3 was significantly inversely correlated with insulin secretory measures. When analyzing HOMA-IR measures, they found a marginal inverse relationship between duration of stage 2 and it was the BMI z-score that was the strongest predictor. Flint et al. (2007) also studied a population of obese children and reported that participants who slept <6 hrs had higher HOMA-IR than those which slept >6 hrs. They reported that sleep duration is a predictor of insulin resistance even after controlling for BMI z-score. On average, our population did not have such severe sleep deprivation. When we compared HOMA-IR between participants who slept >6.5 hrs (n=39) and those who slept <6.5 hrs (n=6), we found no significant difference (p=0.422). However, due to the small sample sizes in both groups this is worth repeating in a larger sample.

The relationship between sleep latency and HOMA-IR may be due to obstructive sleep apnea being more prevalent in overweight/obese children compared to lean (Narang & Mathew, 2012). Insomnia, defined as difficulty to fall and stay asleep, often co-occurs with obstructive sleep apnea. (Luyster, Buysse, & Strollo, 2010). Caffeine consumption has also been shown to increase sleep latency (McHill, Smith, & Wright, 2014). Caffeine has also been shown to decrease insulin sensitivity (Keijzers, Tack, De Galan, & Smits, 2002).

Technology use has also been linked to longer sleep latency. Playing computer games has been shown to increase sleep latency due to increased activity of the autonomic nervous system, evident by increased heart rate (Higuchi, Motohashi, Liu, & Maeda, 2005). Moreover, the uses of

computers and cell phones in bedrooms have been reported to influence the sleep-wake cycle (Brunborg, et al., 2010). Compared to non-light-emitting diode computer screens, exposure to a white light-emitting diode backlit computer screen has been shown to suppress melatonin production (Cajochen, et al., 1985). Self-luminous tablets with blue light-emitting diodes have also shown to suppress melatonin production (Wood, Rea, Plitnick, & Figuerio, 2012). Melatonin is a hormone signalling the onset of sleep by reducing heat production and increasing heat loss (Cajochen, Krauchi, & Wirz-Justice, 2003). Therefore, delaying melatonin production can ultimately delay sleep latency. Technology use is very prevalent in Canadian youth, as less than 20% of met the total screen time guidelines of 2 hours or less/day (Mark, Boyce, & Janssen, 2006). The most common devices used by Canadian youth include television, followed by computers and video games (Active Healthy Kids Canada, 2012). Tablets, specifically iPads, are the top choice for future purchase. (Active Healthy Kids Canada, 2012). Limiting screen time, especially before bed, and caffeine intake may help reduce sleep latency.

Age was a significant predictor of HOMA-IR, but this relationship was weakened after adjusting for %BF. This relationship between age and HOMA-IR could be partially explained by puberty and partially by the fact that older adolescents have greater range of %BF. Interestingly, we did not see that %BF was associated with HOMA-IR as is commonly seen in the general population. This could have occurred due to our narrow range of %BF as this is an overweight/obese population. Another possibility, as discussed with BP, once an adolescent is overweight/obese %BF variation is not a predictor of HOMA-IR and other factors play a role.

### *7.6 Strengths/Limitations/Future Direction*

The biggest strength of our study is the use of objective measures for both sleep and PA. The use of objective sleep measurement allowed us to analyze the relationship between sleep

characteristics such as sleep fragmentation that cannot be measured by subjective measures and allowed for more accurate measures of sleep duration and sleep latency. We also contributed to literature by looking at an adolescent population, and a solely overweight/obese population. Moreover, we contributed to literature by validating a commercially available product that is user friendly and discrete. Data collection using monitoring devices may not be as feasible in overweight/obese children compared to lean children due to insecurities of being bullied due to the stigma associated with PA and obesity, leading them to not wear the device (Ellery, Weiler, & Hazell, 2013). It has been suggested that discrete devices would be more helpful in this population, and the Fitbit allows for that.

This study is of course not without limitations. There is no consensus on how to classify non-wear time using accelerometers in overweight/obese children. A consensus regarding how many days of monitoring PA and sleep is needed for this population is also lacking (Toftager, et al., 2013). As there is no consensus it is difficult to compare studies as the methodologies used slightly vary. We attained a fairly small sample of participants with valid sleep, PA, and blood work in our insulin resistance analysis. Therefore it would be beneficial to reproduce the relationship between objectively measured sleep and insulin resistance in a bigger sample. Furthermore, perhaps doing a 2 hours oral glucose tolerance test can shed light on glucose homeostasis, specifically as Buxton et al. (2012) found that fasting postprandial glucose was impacted after sleep deprivation in adults.

It would be advantageous for futures studies to determine the differences in sleep stages between overweight/obese adolescents. Moving forward, analyze the relationship between sleep architecture and adiposity, PA, and cardiometabolic health. It would also be beneficial to determine whether participants have sleep apnea, which overweight/obese adolescents are at a

greater risk of developing (Narang & Mathew, 2012). This can help understand how sleep, sleep apnea, and our outcomes are interrelated. Adiposity distribution may be a helpful factor to analyze as well in overweight/obese adolescents, as McNeil et al. (2013) suggest that short sleep duration may be a predictor of abdominal adiposity in adults, which on its own is correlated with type 2 diabetes. This is worth exploring in children and adolescents. In the distant future the development of specific sleep recommendations for cardiometabolic health improvement would be necessary as the current guidelines do not take these into account. Koren et al. (2011) suggest 7.5-8.5 hours of sleep for optimal glucose homeostasis in obese adolescents, which on average our population has met. This finding would have to be confirmed.

### *7.7 Conclusions and Implications*

In overweight/obese adolescents recruited from a lifestyle intervention program we found a weak relationship between sleep duration and step count, and sleep latency and step count after adjusting for wear time. There was also a relationship of sleep latency and insulin resistance after adjusting for age and gender. Limiting screen time and caffeine may help with reducing sleep latency. This relationship was weak and did not remain significant after adiposity adjustment. This of course does not mean that poor sleep is not a risk factor in lean adolescents. Perhaps once an adolescent is overweight/obese sleep duration and quality does not influence these factors and other factors play a greater role. It is also important to remember that these participants were recruited from a lifestyle management program, and therefore may not be a good representation of overweight/obese adolescents.

The lack of participants meeting daily recommended steps is a great concern, and an understanding of the root cause is necessary. It appears that sleep may have a slight influence on

activity levels and cardiometabolic health; therefore sleep should not be overlooked in this population especially as there are many other benefits from proper sleep.

## Appendix A: Fitbit Validation

### *A.1.1 Introduction:*

### A. 1. 1. 2 Objective Measurements of Sleep and Physical Activity: Activity Monitors

A polysomnography (PSG) is considered as the gold standard for sleep studies, however due to its high cost and inability to measure sleep over multiple days, activity monitors are a great alternative (de Souza, et al., 2003). As such, the benefits of using accelerometers include the ability to continuously measure for 24 hours for several days, it is more cost effective, and it has the ability to record during free style living (Ancoli-Israel, et al., 2003). When wearing sleep activity monitors such as sleep ActiGraphs on the wrist, one can differentiate between sleep and wakefulness by the use of algorithms and scoring (de Souza, et al., 2003). The stored data is uploaded to a computer after which one can analyze wake/sleep (Ancoli-Israel, et al., 2003).

The most common sleep algorithms used include ones that were developed by Cole et al. (1992) and Sadeh et al. (1994); these algorithms were developed by using both an accelerometer and a PSG. Both studies provided beneficial insight with Cole et al. (1992) using participants with sleep disorders in the sample size, and Sadeh et al. (1994) using children and adolescents in the sample size. Cole et al. (1992) developed algorithms with 88% accuracy, and Sadeh et al. (1994) have shown accuracy rates of over 90%, yet both papers provide slightly different algorithms. De Souza et al. (2003) compared the two algorithms with a PSG, and looked at the algorithms' sensitivity (rate of the ability to detect epochs classified as sleep by a PSG) and specificity (rate of the ability to detect epochs classified as awake). Sensitivity values were 99% and 97% and specific values were 34% and 44%, for Cole's and Sadeh's algorithms respectively (de Souza, et al., 2003). The primary limitation is the inability to detect awakenings as well as a

PSG, however it is a very useful method for sleep measurement (de Souza, et al., 2003) as it small, light, and non-intrusive (Ellery, Weiler, & Hazell, 2013), which allows for use in large populations.

Actigraph accelerometers can also objectively measure physical activity. The GT3X actigraph accelerometer holds a triaxial capacitive accelerometer (John & Freedson, 2012). It senses variation in the electric charge storage potential, thus sensing change in acceleration (John & Freedson, 2012). The acceleration sensor consists of 3 plates: 2 fixed plates that act as electrodes with a parallel movable plate in between (John & Freedson, 2012). Movement of the central plate causes a capacitance change in the 2 fixed plates which leads to a voltage change to the current electric flow (John & Freedson, 2012). Overall, actigraph accelerometers are able to provide data on the duration, intensity, and frequency (Ellery, Weiler, & Hazell, 2013). The GT3X is also able to generate out inclinometer information by using the 3 axes and distinguishing whether the person is not wearing the device, standing, lying, or sitting (John & Freedson, 2012). Accelerometers are currently the most popular device for measurement of physical activity in a research setting (Ellery, Weiler, & Hazell, 2013). The raw data that is stored in the accelerometer is summed over a chosen epoch; after which depending on the sample population appropriate metabolic equivalent tasks (METs) are picked to translate the counts to activity intensity (Ellery, Weiler, & Hazell, 2013).

Sleep activity monitors are superior to logs; especially parental sleep logs (John & Freedson, 2012). It has been found that parental filled logs have more disagreements relative to an activity monitor in adolescents (John & Freedson, 2012). Recording physical activity with activity monitors is also superior to subjective measurements such as activity diaries, questionnaires, interviews, and surveys which could have errors due to recall bias (Ellery,

Weiler, & Hazell, 2013). Particularly in youth, subjectively measured PA was overestimated relative to objective data (Ellery, Weiler, & Hazell, 2013). Specifically, overweight/obese youth are more likely to overestimate physical activity during subjective questionnaires due to the stigma and misconception regarding greater weight and poor physical activity (Ellery, Weiler, & Hazell, 2013). Youth with low self-esteem on the other hand might underestimate physical activity levels (Ellery, Weiler, & Hazell, 2013).

#### A.1.1.3 Fitbit

The Fitbit manufacture have reported that the Fitbit is an accelerometer (Montgomery-Downs, Insana, & Bond, 2011), and a Fitbit representative has stated that the Fitbit has been tested to ensure accurate sleep and PA recordings, and that METs are used for activity intensity measurement. Fitbit's METs cut-offs and sleep algorithms are not released, nor how they were tested or determined. Few studies have compared the Fitbit to other devices.

#### A.1.1.4 Activity Monitors and Fitbit Comparison in Literature

Several authors have compared various aspects of the Fitbit to other measures, including the actigraph accelerometers. Montgomery-Downs et al. (2011) (N=24) evaluated the validity of the Fitbit Ultra comparing it to a PSG and an actigraph Actiwatch-64 (Mini Mitter, Inc., Bend, OR, USA) across 1 night. Three of the participants had also worn 2 Fitbit Ultras to measure reliability (Montgomery-Downs, Insana, & Bond, 2011). Epoch length for the Actiwatch was set to 30 seconds (Montgomery-Downs, Insana, & Bond, 2011). The algorithm chosen were not based on previous research, nor was the reasoning for this choice was explained. This is a very significant limitation, as the purpose of the study was to compare different activity monitors. Without proper and accurate sleep algorithms this will hinder the ability to state any similarities/differences observed with confidence. The authors also assumed that the Fitbit epoch

setting was 1 min (Montgomery-Downs, Insana, & Bond, 2011). This is not appropriate to assume as this might not be the case as it was not tested. Although Fitbit presents data per minute on the Fitbit website, it is not to assume that is the epoch length. The PSG epoch length was 30 seconds (Montgomery-Downs, Insana, & Bond, 2011). To overcome the problem of the use of different epoch lengths, the Fitbit epoch values were doubled to match the 30 s PSG an actigraph epochs (Montgomery-Downs, Insana, & Bond, 2011). When comparing a device to a PSG, a common problem that arises are the different epoch lengths where a standard epoch length for a PSG is 30 seconds, and devices such as actigraphy usually have a standard epoch of 1 min (Ancoli-Israel, et al., 2003). A way to overcome this problem is to have an algorithm that detects low threshold of awakening, thus even small amount of awakenings will consider the epoch as an awakening (Ancoli-Israel, et al., 2003). With the Fitbit, unfortunately, it is not possible to find out the number of awakenings/epoch. This factor supports the reason to use properly tested algorithm.

Limitations aside, inter-fitbit reliability agreement rates were quite high: 96.5%, 99.1%, and 97.6% (Montgomery-Downs, Insana, & Bond, 2011). Fitbit overestimated total sleep time by an average of 67.1 min, while the actigraphy overestimated total sleep time by 43 min (Montgomery-Downs, Insana, & Bond, 2011). Comparing the Fitbit to the Actiwatch, the Fitbit overestimate total sleep time by >30 min among 29.2% (Montgomery-Downs, Insana, & Bond, 2011). The authors conclude that the Fitbit, like the Actiwatch, has very low specificity (Montgomery-Downs, Insana, & Bond, 2011).

As for physical activity comparisons, a significant difference was found between the steps recorded by the Fitbit Ultra and by the Actical (Philips Respironics, Inc., Andover, MA) while jogging or walking on an incline (N=23) (Noah, Spierer, & Bronner, 2013). Intra device

step count was not significantly different; therefore Fitbit reliability is strong (Noah, Spierer, & Bronner, 2013). The participants only wore the devices for one day for 6 minute bouts; therefore longer wear time is necessary. Also, due to such short bouts the step counts were all under 156 steps causing even small step differences to be statistically significant differences. The maximum step differences were 3 steps, therefore although statistically significant this difference may not be of clinical significance. Due to several limitations in current literature and to ensure accurate correlation we had volunteers test the Fitbit against several devices.

### *A.1.2 Objectives:*

#### A.1.2.1 Primary Objective

To determine whether Fitbit Ultra measurements of PA and sleep correlate with devices currently used in large population samples, ActiSleep and Actigraph accelerometer

#### A.1.2.2 Secondary Objectives

To determine whether the Fitbit Ultra and the Fitbit One are in agreement prior to using the Fitbit One in the study

### *A.1.3 Methods:*

#### A.1.3.1 Fitbit Actigraph Comparison

To ensure that the Fitbit PA and sleep measures correlate with activity monitor measures, volunteers wore both for 8 days and 7 nights. For sleep, the ActiSleep monitor (ActiGraph LLC, Pensacola, FL) was used and worn on the non-dominant hand at nighttime, while the Fitbit was attached onto the wristband of the ActiSleep to ensure close proximity. Upon bed time sleep mode was initiated by holding down the button on the Fitbit. Sleep mode was turned off upon waking up. As for PA, the GT3X (ActiGraph LLC, Pensacola, FL) was worn around the waist

during the day and the Fitbit was attached onto the GT3X waistband directly near the accelerometer, to ensure close proximity. The following are the measures that were recorded and compared between the Fitbit and accelerometer:

| Sleep | Physical activity |
|---|---|
| Latency (min) | Light (min) |
| Total Sleep Time (TST) (min) | Moderate (min) |
| Awakenings | Vigorous (min) / Very Active (Fitbit) (min) |
| | Step Count |

After the timespan of 8 days, the Fitbit data was uploaded online to an account provided for the volunteer. The GT3X and ActiSleep data was downloaded using the ActiLife 6 Software. Sleep analysis for ActiSleep was completed by inputting wake up and bed times recorded by the Fitbit and stored on the Fitbit website. This ensured that the time period of recording was the same for the Fitbit and Actisleep. Sadeh and Cole sleep algorithms were then applied and the output data was exported. Sleep epoch can only be set to 60 second on the ActiSleep. PA data was downloaded and analyzed at epoch lengths of 15 and 60 seconds. The algorithm and MET cutoffs for PA used were those developed by Freedson et al. (1998).

**A.1.3.2 Statistical Analysis**:

Correlation coefficients were used to determine whether the devices correlate. Pearson correlation (r) was used on normally distributed data while Spearman correlation ($r_s$) was used for non-normally distributed data. For data with low correlation coefficients, Bland-Altman plots were used. Average of the measures versus difference of the measures was plotted. The limits of agreement chosen were ±2 SD, or 95% limit of agreements (Bland & Altman, 1999). For sleep latency, since there is a narrow range of variation and the differences were quite small, a moderate correlation could be seen due to the narrow range of variation (de Souza, et al., 2003). The same reasoning was used to create Bland-Altman plots for awakenings.

### *A.1.4 Results:*

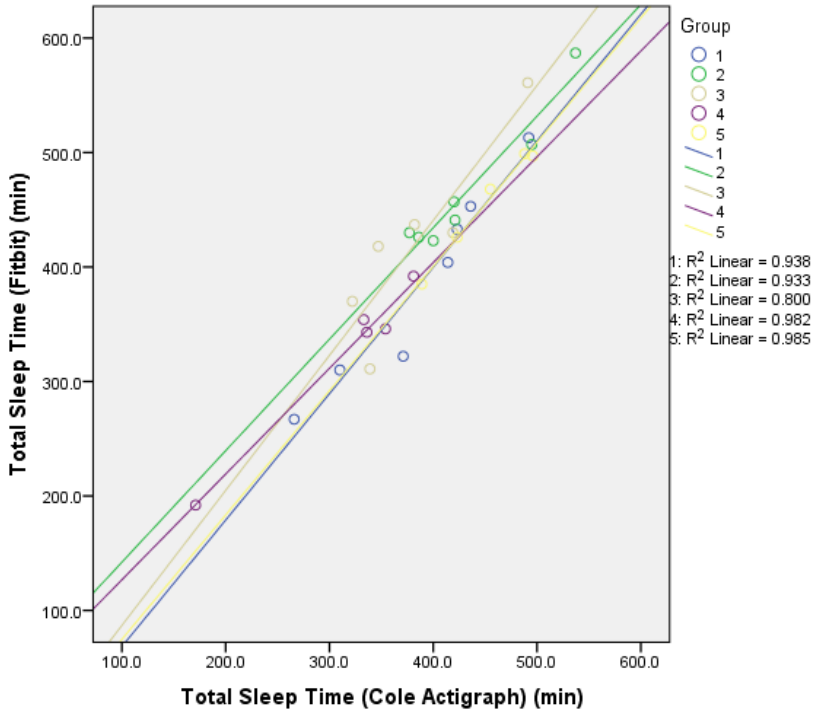
#### A.1.4.1 Sleep Variables

TST was highly correlated for both Cole and Sadeh algorithms, but stronger correlations were observed for Cole (Figure 1A). TST of the Fitbit and Cole algorithm were very similar and highly correlated for every volunteer (Fitbit-Cole $r_s$: 0.96**, 0.79*, 0.94**, 1**, 0.9*, *p<0.05 **p<0.01), with an overall correlation for all participants combined of $r_{s=}$ .926 (p< .001).

Spearman's correlation coefficients were not found to be significant for between sleep latencies for either Fitbit-Cole or Fitbit-Sadeh measures. However, since the average sleep latency difference between the two devices for all the participants was only 3.9 min for Fitbit-Sadeh algorithm and 4.2 min for Fitbit-Cole algorithm, Bland-Altman plots were used (Figure A1B). Pearson's correlations were not significant for the number of awakenings; therefore Bland-Altman plots were used (Figure A1B).

**Figure A1.** Sleep comparison between Fitbit and Actisleep. A) Correlation between TST Fitbit and Actisleep at 60 seconds epoch. B) Bland-Altman plot of sleep latency measured by Fitbit and ActiSleep (Sadeh). Mean difference= 3.9, 2SD= 14.7 C) Bland-Altman plot of awakenings measured by Fitbit and ActiSleep (Sadeh). Mean difference= -9.9, 2SD= 15.2. N=5

A)




B)

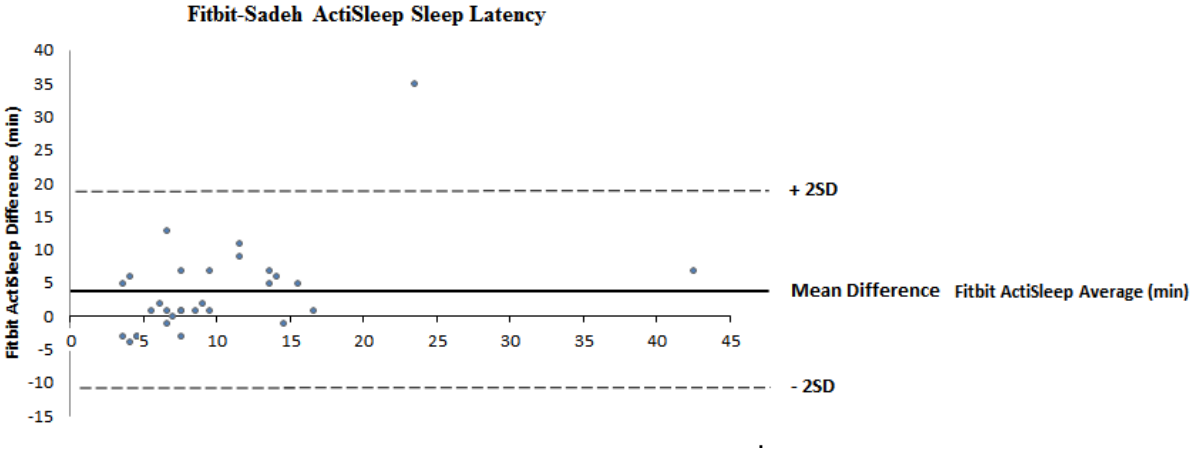
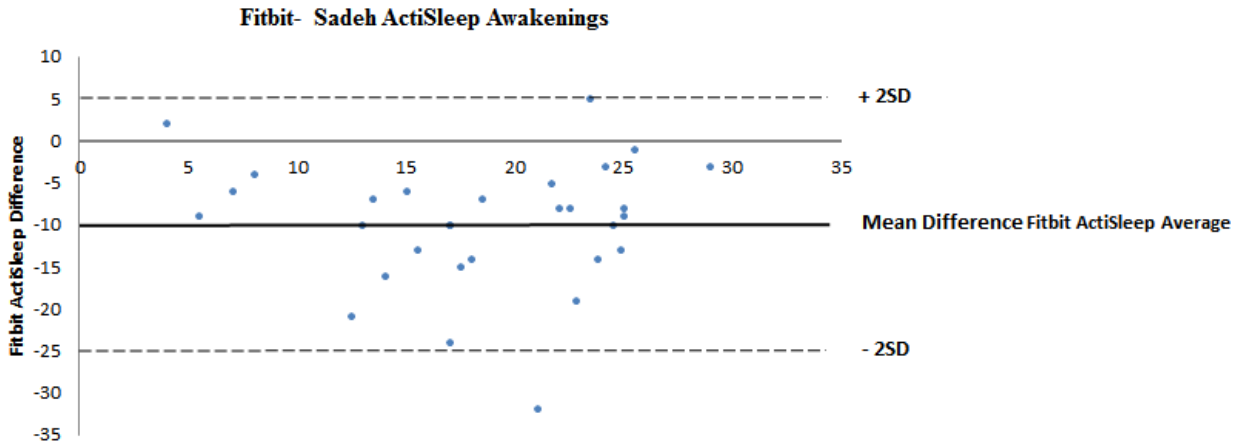


**Figure A1.** Continued.

C)



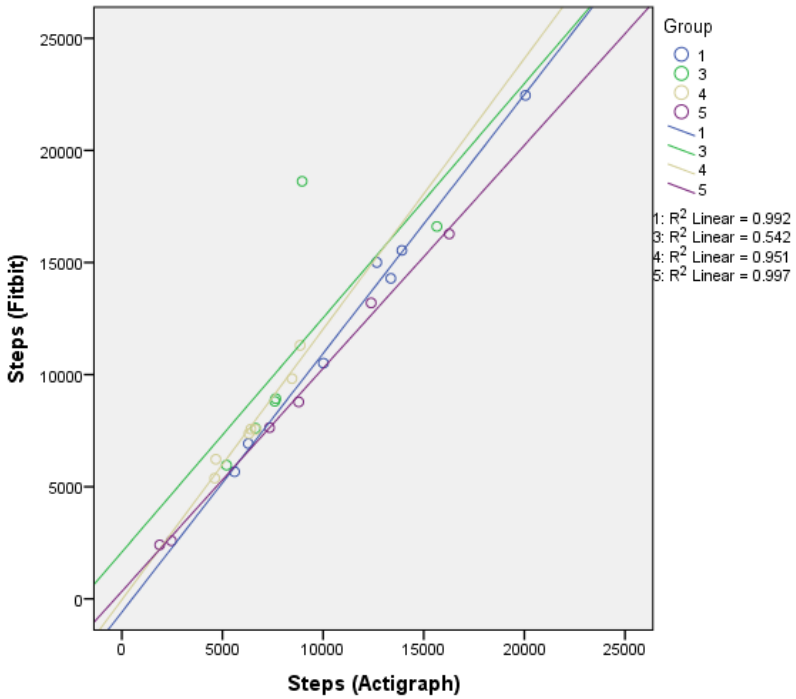
**A.1.4.2 Physical Activity Variables**:

Steps between the accelerometer and Fitbit were highly correlated for every participant (r= .996**, .736, .975**, .998**, **p<0.01 ) (Figure A2A). The outlier seen for volunteer 3 is due to the volunteer biking for several hours, possibly suggesting that the two devices pick up cycling differently. With the outlier removed, the correlation between the Fitbit and acclereomter step count was r= .999 (p< .001). When combining all the participants together the correlation between the 2 step count measurements was r= .932 (p< .001) and r=.991 (p< .001) with the outlier of volunteer 3 removed.

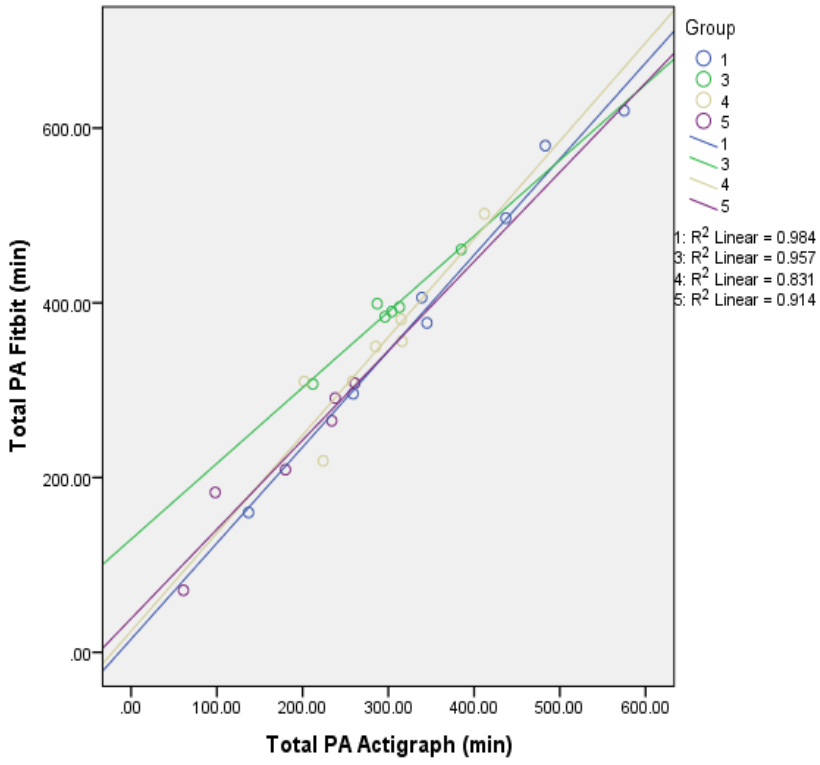
Total physical activity (ie. all minutes added for all intensities) was also highly correlated for each participant at epoch of 60 seconds; however the measurements were not identical between the two devices (Figure A2B). Moderate to vigorous physical activity (MVPA) are common guidelines set for PA (World Health Organization, 2010). Therefore, we compared MVPA as measured by the actigraph accelerometer to moderate-very intense activity as measured by the Fitbit. At 60 seconds epoch, high correlations were observed for each participants, however the measurements were not identical between the two devices (Figure A2C)

**Figure A2. Physical Activity Comparisons between Fitbit and Actigraph** A) Correlation between steps measured by the Fitbit and Actigraph accelerometer. B) Correlation between total PA measured by the Fitbit and Actigraph (60 sec epoch). C) Correlation between moderate-very intense PA as measured by the Fitbit, and MVPA, a common used guideline for PA, as measured by the Actigraph (60 sec epoch). N=4

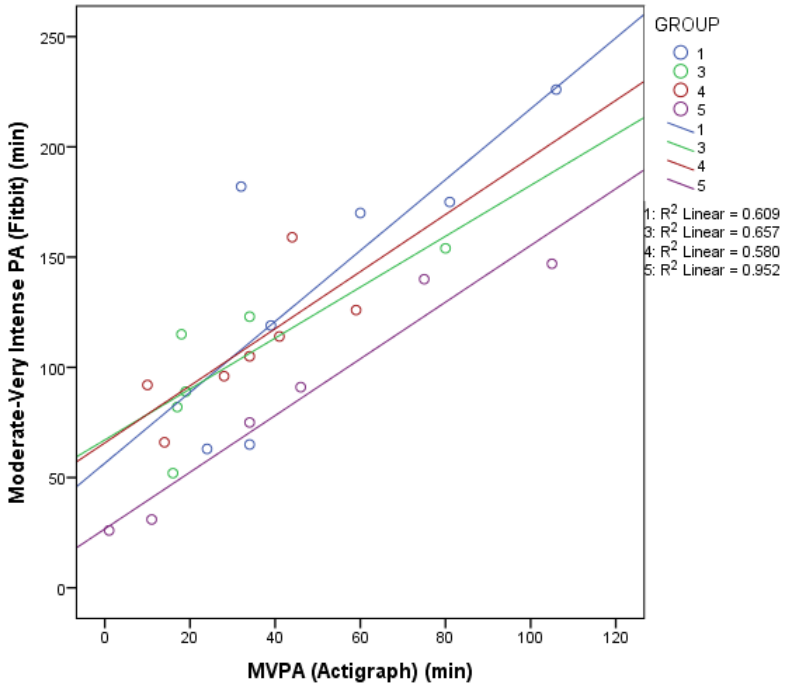
A)




B)



C)


Moderate-Very Intense PA (Fitbit) (min)
MVPA (Actigraph) (min)
0
50
100
150
200
250
0
20
40
60
80
100
120
GROUP
1
3
4
5
1: R2 Linear = 0.609
3: R2 Linear = 0.657
4: R2 Linear = 0.580
5: R2 Linear = 0.952

### A.1.4.2 Fitbit One and Fitbit Ultra Comparison

Both devices had extremely similar outputs. Total sleep time was always within 3 minutes of each other, latency was the same, and the number of awakenings either did not differ or differed by 1. Step counts were always within fewer than 50 steps of each other out of >10, 000 steps. Physical activity times at different intensities were identical.

### *A.1.5 Conclusion*

As TST highly correlated and was very similar between the 2 devices, we conclude that it is a valid variable to measure using the Fitbit. Bland Altman plots for sleep latency (Figure A1B) show that the majority of the points lie very closely to the mean difference line. As such, this allows us to conclude that there is an agreement (Bland & Altman, 1999). The Bland-Altman plot for awakenings (Figure A1C) is quite scattered, however if looking at TST, the correlations are really high. We conclude that this must mean that the Fitbit does not detect small awakenings, as the ActiSleep reports more awakenings. As these are short awakenings that the Fitbit does not detect, we conclude that the number of awakenings is a viable variable to measure with the Fitbit.

As the step count of the Fitbit and accelerometer were highly similar and highly correlated, we conclude that the Fitbit is an appropriate measure of step count. We also conclude that all moderate and very active minutes as measured by the Fitbit are not equivalent to moderate and vigorous activity as measured by the accelerometer. Therefore, we conclude that active minutes should be used as a measure of intensity. Lastly, like other studies we have also found high repeatability with the Fitbit Ultra (data not shown). We have also found that the Fitbit One and Fitbit Ultra can be used interchangeably in our study.

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