ASSOCIATIONS BETWEEN SLEEP DURATION AND INDICATORS OF CARDIO-METABOLIC DISEASE IN CANADIAN CHILDREN AND ADOLESCENTS

by

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Abstract

Rates of obesity and type 2 diabetes in Canadian children and adolescents have increased rapidly in recent years, so research exploring modifiable risk factors is critical. Partial sleep loss has been linked with deteriorations in indicators of cardio-metabolic health. The objectives of this study were 1) to examine associations among short sleep duration and indicators of cardio-metabolic disease in Canadian children and adolescents, and 2) to identify determinants of short sleep duration. Using the 2007-2009 Canadian Health Measures Survey dataset as a data source, logistic regression models were developed to examine associations among sleep duration and indicators of cardio-metabolic disease and to identify predictors of short sleep duration. Short sleep duration was linked with greater odds of overweight/obesity in boys and adolescents only. Age was a strong predictor of inadequate sleep duration. Interventions designed to address sleep loss should focus on adolescents.

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Associations Between Sleep Duration and Indicators of Cardio-Metabolic Disease in Canadian Children and Adolescents

Chapter 1: Introduction

A large body of research has demonstrated that short sleep duration is linked to indicators of cardio-metabolic disease, including overweight/obesity (OWOB), hyperinsulinemia and dyslipidemia (Knutson, 2010; Knutson, Spiegel, Penev, & Van Cauter, 2007). However, the majority of these studies focused on adults. Only a single study examining associations between sleep duration and OWOB has been conducted with Canadian children (Chaput, Brunet, & Tremblay, 2006), and that study was not nationally representative. No studies have explored associations between sleep duration and OWOB in Canadian adolescents. Additionally, although a number of studies have examined associations between sleep duration and OWOB, fewer studies have examined links among sleep duration and other indicators of cardio-metabolic health.

To address these knowledge gaps, this study had two objectives, including 1) to develop nationally representative estimates for associations between sleep duration and a range of indicators of cardio-metabolic disease in Canadian children and adolescents aged 6-17, and 2) to identify determinants of short sleep duration in this population. This thesis begins with a review of the literature on sleep duration and cardio-metabolic health, including theories that explain the pathophysiology of linkages between sleep duration and cardio-metabolic disease, the role of covariates in these relationships, and a summary of research supporting the measures and methods selected for this research.

Indicators of Cardio-Metabolic Disease in Children and Adolescents

Obesity, dyslipidemia, and impaired carbohydrate metabolism are strongly associated with increased risks for coronary artery disease, stroke, and type 2 diabetes (International Diabetes Federation, 2011). Increased rates of OWOB have been observed in Canadian children and adolescents in recent years. For example, between 1981 and 2009, the prevalence of OWOB in Canadian youth aged 15-19 increased from 14% to 31% in boys and from 14% to 25% in girls (Statistics Canada, 2010c). Similarly, cases of type 2 diabetes in youth were almost non-existent 30 years ago but the prevalence has increased dramatically in recent years, especially among certain groups such as Aboriginal populations (Canadian Diabetes Association, 2012).

Trends reflecting the rising prevalence of indicators of cardio-metabolic disease in younger populations are concerning for a number of reasons. Firstly, the development of obesity, dyslipidemia, or impaired carbohydrate metabolism at a younger age increases the potential years of risk exposure, thereby lowering the age of potential disease onset, increasing years of morbidity over the lifespan, and reducing life expectancy. Further, children and adolescents who exhibit milder stages of cardio-metabolic risk factors tend to progress to more severe categories of risk over time. For example, overweight children are at high risk of advancing from overweight to obesity (Reilly et al., 2010), and youth with pre-hypertension tend to progress to adulthood hypertension (Tirosh et al., 2010). For these reasons, early intervention is beneficial. Despite significant research activity in this area, the prevalence of cardio-metabolic risk factors in children and youth has continued to rise. Further research is needed to develop strategies that can successfully address this health issue.

Sleep Duration in Children and Adolescents

In addition to this rising prevalence of obesity and type 2 diabetes has been a trend of decreasing total sleep time in Canadian children and adolescents (Matricciani, Olds, & Petkov, 2011). Based on an analysis of administrative data from 20 countries and 690,747 children and adolescents, Matricciani et al. (2011) determined that sleep duration declined by a median of 0.75 min/year between 1905-2008, for a total decline of over one hour during the last century. Declines in sleep duration were larger for older children (median −0.41 min per day per year for early primary school children to −0.91 min per day per year for older adolescents). Analysis by time period indicated that median sleep time increased from 1900–1939 (+2.57 min/day/year), but declined thereafter (-0.78 min/day/year from 1940–1969, -0.35 min/day/year from 1970–1989 and -0.40 min/day/year from 1990–2008). Sex-based analyses indicated that boys had larger declines in sleep duration (median −0.77 min/day/year) compared to girls (median, −0.67 min/day/year). Sleep duration decreased most on school days (−0.74 min/day/year) compared to non-school days (−0.30 min/day/year). In Canadian children and adolescents specifically, there was a median decline of 1.10 min/day/year over the study period. These findings from the Canadian data were similar to rates in the USA (-1.14 min/day/year) and Europe (-1.17 min/day/year), but contrasted with increases observed in Scandinavia (+0.65 min/day/year), Australia (+0.40 min/day/year), and the UK (+0.57 min/day/year).

Declining sleep time in children and adolescents has been attributed to various aspects of modern society (Matricciani et al., 2011). For example, the introduction of electricity and corresponding exposure to artificial light has been proposed as a plausible mechanism for extended waking hours (Gangwisch, Malaspina, Boden-Albala, & Heymsfield, 2005). A rapid expansion in use of technology has also been identified as a culprit for sleep loss among children

and adolescents, including television, internet, and mobile phone usage, all of which are increasingly available in the bedroom (Taheri, 2006).

Canadian adults also report sacrificing sleep time in order to meet the demands of modern society (C. Williams, 2001). A 2005 Canadian study found that sleep loss in adults was related to long working hours, family obligations, long commuting times, and feeling time-stressed (Hurst, 2008). Fifty percent of Canadian adults report that they restrict their sleep to gain more time during the day when daily demands become heavy (Hurst, 2008). Sleep quality has also been affected by modern life: Canadians report that shift work and lack of physical exercise are linked with decreased sleep quality (Hurst, 2008). In order to compensate for week-night sleep loss, Canadian adults report sleeping in on the weekends (Hurst, 2008).

Although the function of sleep is not entirely understood, clues have emerged through the examination of what happens when people do not get enough sleep. Medical guidelines for human sleep requirements are informed by the number of hours of sleep needed to feel refreshed (National Sleep Foundation, 2012). While to some degree, sleep requirements vary from individual to individual, most adults require between 7-9 hours of sleep (National Sleep Foundation, 2011b). In children and youth, sleep requirements vary substantially by developmental stage. Children aged 5-12 require about 10 hours of sleep per day, while adolescents need nine hours (Table 1). Researchers have examined the consequences of acute sleep loss occurring over several days, as well as chronic sleep loss occurring over longer periods such as weeks, months, or years.

Table 1

Sleep Requirements Across the Lifespan (National Sleep Foundation, 2011a)

Age group	Recommended sleep duration
0-2 months*	10.5-18.5 hours
2-12 months*	14-15 hours
18 months-3 years*	12-14 hours
3-5 years*	11-13 hours
5-12 years*	9-11 hours
13-19 years	8.5-9.5 hours
Adults	7-9 hours on average

Note. *Sleep time includes naps.

Sleep loss can also be categorized as partial or total. Partial sleep loss means getting fewer hours than needed to feel refreshed. In population-based research, people who chronically experience partial sleep loss can also be thought of as "short sleepers" or those with "short sleep duration". Partial sleep loss has been linked to declines in human functioning and health. For example, acute periods of partial sleep loss are associated with declines in cognitive functioning (Pilcher & Huffcutt, 1996) and alterations in hormones involved in glucose regulation such as insulin and cortisol (Knutson et al., 2007). Given that long-term experimental studies examining partial sleep loss would be difficult due to ethical and methodological reasons, epidemiological studies have been conducted to explore the effects of chronic partial sleep loss. Findings from these population-based studies have also linked short sleep duration to poorer physical health (e.g., cardio-metabolic diseases) (Knutson, 2010) and declines in functioning (e.g., reaction time) (Pilcher & Huffcutt, 1996).

By comparison, total sleep deprivation during a period of time refers to a complete lack of sleep over that time frame. However, studies exploring the effects of total sleep deprivation in

humans can only be conducted on a short term basis, because humans exhibit involuntary 'microsleeps' after a few days of total sleep deprivation (Durmer & Dinges, 2005). Experimentally, these short periods of total sleep deprivation are associated with declines in mental, emotional, and physical health and functioning (Alhola & Polo-Kantola, 2007; Durmer & Dinges, 2005; Knutson et al., 2007; Pilcher & Huffcutt, 1996). For example, the results of a meta-analysis indicated that acute total sleep deprivation resulted in declines in cognitive function, including attention, processing speed, memory, and reasoning (Durmer & Dinges, 2005). In a British study, a period of 17-19 hours without sleep induced declines in accuracy and response time comparable to measurements taken in people with a blood alcohol level of 0.05% (Williamson & Feyer, 2000). Short periods of total sleep deprivation have also been linked to deteriorations in indicators of cardio-metabolic health (Knutson, 2010) and immune function (Walsh et al., 2011).

Concurrent Trends of Cardio-Metabolic Disease and Decreasing Sleep Duration

Experimental and epidemiological lines of evidence suggest that the concurrent trends of increasing sleep restriction and rising rates of obesity and diabetes are not coincidental. Laboratory studies indicate that sleep restriction induces physiological and behavioural changes that negatively influence indicators of cardio-metabolic health. In the 1990's, landmark experimental research demonstrated that sleep restriction (four hours sleep/night) over a period of six days could induce obesogenic changes in appetite- and weight-related hormones in healthy adults (Spiegel, Leproult, & Van Cauter, 1999). Hormone levels returned to baseline after a seven day period of sleep recovery (12 hours sleep/night) (Spiegel et al., 1999). Since then, a number of other experimental studies have similarly demonstrated that partial sleep loss can induce adverse endocrine changes (Buxton et al., 2012; Nedeltcheva, Kessler, Imperial, & Penev,

2009; Schmid, Hallschmid, Jauch-Chara, Born, & Schultes, 2008; Spiegel, Tasali, Penev, & Van Cauter, 2004). Experimental studies also demonstrated that sleep restriction is related to obesogenic behaviours such as increased snacking (Nedeltcheva, Kilkus, et al., 2009) and decreased physical activity (Schmid et al., 2009). Reinforcing these findings are epidemiological studies indicating short sleepers are more likely to be overweight compared to longer sleepers (Cappuccio, Taggart, Kandala, & Currie, 2008; Chen, Beydoun, & Wang, 2008; Leproult & Van Cauter, 2010; Marshall, Glozier, & Grunstein, 2008) and exhibit other cardio-metabolic risk factors such as hypertension, dyslipidemia, and impaired carbohydrate metabolism (Ayas, White, Al-Delaimy, et al., 2003; Buxton et al., 2012; Buxton et al., 2010; Chaput, Despres, Bouchard, Astrup, & Tremblay, 2009; Flint et al., 2007; Gottlieb et al., 2005; Javaheri, Storfer-Isser, Rosen, & Redline, 2011).

Evidence suggests that a bi-directional relationship links short sleep duration to cardio-metabolic risk factors (Magee, Huang, Iverson, & Caputi, 2010). For example, carrying excess weight may adversely affect sleep quality; conversely, poor sleep quality could result in weight gain (Nieto, Surani, Huerta-Alardin, & Varon, 2006). Another example is sleep disordered breathing, a cause of sleep loss resulting from frequent interruptions in respiration during sleep (Nieto et al., 2006). Sleep disordered breathing is strongly associated with indicators of cardio-metabolic disease (Leinum, Dopp, & Morgan, 2009). It is thought that obesity plays a causal role in sleep disordered breathing because it frequently occurs among obese individuals, and weight loss can result in improvements in both sleep disordered breathing and cardio-metabolic health (Leinum et al., 2009). On the other hand, in studies that controlled for obesity, it was determined that sleep disordered breathing and concurrent sleep loss are independently associated with poorer cardio-metabolic health (Flint et al., 2007; Punjabi et al., 2004).

Additionally, improvements in cardio-metabolic health have been demonstrated after treatment of sleep disordered breathing with continuous positive airway pressure (Dorkova, Petrasova, Molcanyiova, Popovnakova, & Tkacova, 2008) and tonsillectomy (Gozal, Capdevila, & Kheirandish-Gozal, 2008). Continuous positive airway pressure has not been demonstrated to result in weight loss (Redenius, Murphy, O'Neill, Al-Hamwi, & Zallek, 2008), suggesting that the pathophysiological mechanisms underlying the linkage between sleep disordered breathing and cardio-metabolic disease are at least partly attributable to factors other than obesity. In summary, although any relationship between cardio-metabolic risk factors and short sleep duration is likely bidirectional, the proposed study builds on the formidable body of evidence supporting short sleep duration as an etiological factor in poor cardio-metabolic health. Given that sleep duration could potentially be modifiable in many individuals, further research into sleep loss as a risk factor for cardio-metabolic disease is worthwhile.

The relationship between sleep duration and cardio-metabolic risk factors is complex. Similarly, any mechanisms that underlie these associations are potentially numerous. As such, the discussion provided in this literature review of potential mechanisms by which short sleep could influence indicators of cardio-metabolic disease is not intended to be comprehensive. Rather, this literature review focuses on the leading theories with the strongest supporting evidence.

Short Sleep Duration and Cardio-Metabolic Disease Outcomes

Based on data from longitudinal epidemiological studies, chronic short sleep duration predicts the future development of cardiovascular disease. Specifically, in a meta-analysis involving 15 prospective epidemiological studies with a total of 474,684 participants, short sleep duration at baseline was associated with a greater risk of developing or dying of coronary heart

disease (relative risk=1.48) and stroke (relative risk=1.15) at the follow up time point (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011). In a longitudinal study of 71,617 female participants in the US-based Nurses' Health Study, short sleep duration at baseline was associated with an increased risk of cardiovascular events 10 years later (relative risk=1.82 for 5 or fewer hours/night) (Ayas, White, Manson, et al., 2003).

Short sleepers are also more likely to have type 2 diabetes and impaired carbohydrate metabolism. In a cross-sectional study of 1,486 adult participants in the American Sleep Heart Health Study, those sleeping five or fewer hours per night were 2.51 times more likely to have type 2 diabetes and 1.33 times more likely to have impaired glucose tolerance (Gottlieb et al., 2005). In a longitudinal study, females who slept less than nine hours per night at baseline had a relative risk of 1.34 for developing symptomatic diabetes 10 years later (Ayas, White, Al-Delaimy, et al., 2003).

Short Sleep Duration and Overweight/Obesity

Obesity is a well-known risk factor for cardiovascular disease and diabetes (International Diabetes Federation, 2011), and the results of both laboratory experiments and population studies support a link between short sleep duration and obesity (Chen et al., 2008; Spiegel et al., 1999). So, short sleep duration also is related to early indicators that predict the future development of cardiovascular disease and diabetes.

Measuring overweight and obesity. Body mass index (BMI) is a frequently used measure of weight status in epidemiological research, and was used in this study. BMI is calculated by dividing mass (kg) by height (in metres squared). In adults, BMI categories include underweight ($<18 kg/m^2$), normal weight ($18<25 kg/m^2$), overweight ($25-30 kg/m^2$), and obese ($>30 kg/m^2$) (World Health Organization, 1995). The development of these definitions was

informed by linking BMI scores to risks for morbidity and mortality (World Health Organization, 1995). In children and adolescents, BMI categories are developed using normative data (Cole, Bellizzi, Flegal, & Dietz, 2000). This study used the BMI categorization system developed by Cole et al. (2000) which is based on international normative data standardized for age and sex. BMI categories used in this research include obese, overweight, or neither (Cole et al., 2000).

Although measures of fat distribution such as waist circumference and waist-hip ratio have also received significant attention, many studies suggest that these measures offer no advantage over BMI for predicting cardio-metabolic risk in children and adolescents (Garnett, Baur, Srinivasan, Lee, & Cowell, 2007; Johnson et al., 2010; Qiao & Nyamdorj, 2010; Vazquez, Duval, Jacobs, & Silventoinen, 2007; Ying, Song, Zhao, & Jiang, 2010). Additionally, because norms for body mass vary with age and gender in growing children and adolescents, standardized BMI scores must be used to account for this in epidemiological studies (Knutson, 2005). Because more research is available to support age- and sex- standardized percentiles for BMI compared to similarly standardized measures of adiposity (Cole et al., 2000), BMI was selected as the preferred measure for this study.

Energy imbalance theory of obesity. The energy imbalance theory of obesity can be used to explain the mechanisms that underlie the observed connections between short sleep duration and obesity. For weight gain to occur, an imbalance between energy intake and energy expenditure must exist (Chaput, Klingenberg, & Sjodin, 2010). Food consumption is mediated homeostatically (i.e., through perceived sensations of appetite/hunger) as well as through hedonic, or pleasure-based eating behaviour (Chaput, Klingenberg, & Sjodin, 2010). Energy expenditure can occur voluntarily through physical activity or involuntarily through basal

metabolism and thermogenesis (Chaput, Klingenberg, & Sjodin, 2010). Short sleep duration could cause weight gain through both sides of this energy balance equation (Chaput, Klinenberg, and Sjodin, 2010).

Short sleep duration dysregulates homeostatically mediated energy intake. Homeostatic control over appetite is orchestrated through complex interactions among many neuroendocrine hormones (Morselli, Leproult, Balbo, & Spiegel, 2010). Key hormones thought to be involved in the sleep-obesity relationship include leptin, ghrelin, insulin, and cortisol (Morselli et al., 2010). Leptin and ghrelin have opposite functions in regulating appetite (Pocock & Richards, 2006). Leptin is released from fat cells and is thought to suppress appetite, while ghrelin is secreted from the stomach, and stimulates hunger (Pocock & Richards, 2006). Insulin plays an important role in the regulation of blood glucose levels (Pocock & Richards, 2006). When blood glucose levels rise, insulin is secreted by the pancreas and glucose is transported out of the blood and into the cells (Pocock & Richards, 2006). Cortisol is part of the body's stress response and results in increased arousal, elevations in blood glucose, and suppression of the immune system (Pocock & Richards, 2006).

Experimental studies have demonstrated that partial sleep loss disrupts levels of leptin, ghrelin, cortisol, and insulin (Nedeltcheva, Kessler, et al., 2009; Schmid et al., 2008; Spiegel, Leproult, et al., 2004; Spiegel, Tasali, et al., 2004). For example, in healthy young men, when sleep time was restricted to four hours sleep/night in bed over two days, participants exhibited an 18% decrease in leptin and a 28% increase in ghrelin compared to when they spent 10 hours in bed over two days (Spiegel, Tasali, et al., 2004). Participants also experienced a 24% increase in hunger after the period of sleep loss, with larger increases in appetite for high carbohydrate foods (Spiegel, Tasali, et al., 2004). Similar findings were demonstrated by Schmid et al. (2008), with

a single night of partial sleep loss resulting in elevations in both ghrelin and sensations of hunger.

Increased waking time = increased eating time. Initially, the idea that sleep loss could cause obesity may seem non-intuitive because sleep is a sedentary act, and having more time awake could provide more time for energy expenditure. Interestingly, research suggests that the opposite is true. In modern societies, people tend to consume more calories than they expend (Chaput, Klingenberg, Astrup, & Sjodin, 2010). In other words, spending more time awake tends to worsen the energy imbalance that underlies obesity because people tend to overcompensate for any increased energy expenditure that occurs during the additional hours spent awake (Chaput, Klingenberg, Astrup, et al., 2010). This observed increase in energy intake resulting from sleep loss may be a consequence of increased appetite as well as non-appetite-based eating behaviour (Chaput, Klingenberg, Astrup, et al., 2010).

Several aspects of modern lifestyles have been shown to stimulate non-appetite mediated food consumption (Chaput, Klingenberg, & Sjodin, 2010). Modern lifestyles facilitate obesity through the combination of easy access to calorie-dense foods and frequent engagement in sedentary activities that are associated with eating (Chaput, Klingenberg, Astrup, et al., 2010). Thus short sleep (i.e., increased time awake) translates to increased exposure to the obesogenic modern environment. While it is intuitive that sedentary activities such as television watching and computer use are associated with reduced energy expenditure, research also suggests that these activities also result in increased food consumption independent of appetite (Chaput, Klingenberg, & Sjodin, 2010). Many sedentary activities are thought to result in increased calorie intake due to disconnection from internal satiety signals, reducing conscious control over food consumption, and exposure to food-related advertising (Chaput, Klingenberg, Astrup, et al.,

2010). For example, Blass et al. (2006) found that meals consisting of macaroni and cheese or pizza consumed over a 30 minute period of television watching resulted in an additional 288 kcal consumed compared to meals consumed while not watching television. Similar findings have been demonstrated with increased food consumption during meals consumed after video game playing (+80 kcal) (Chaput, Klingenberg, Astrup, et al., 2010), and after cognitive working such as reading and writing (+229 kcal) (Chaput, Klingenberg, Astrup, et al., 2010; McCann, Warnick, & Knopp, 1990). Listening to music while eating has also been associated with increased calorie consumption (+107 kcal) (Stroebele & de Castro, 2006).

Non-appetite mediated eating behaviour also links short sleep duration to obesity. In a crossover study of 11 healthy participants, eating behaviour was compared between a sleep restriction condition (5.5 hours of sleep/night for 14 days) and an adequate sleep condition (8.5 hours of sleep/night for 14 days) (Nedeltcheva, Kilkus, et al., 2009). While sleep restriction did not affect leptin or ghrelin levels in study participants, increased calorie consumption was observed. Additionally, energy expenditure did not increase to compensate for the increased energy intake. Interestingly, while energy consumption during meals was similar in both conditions, increased snacking behaviour was observed during the sleep restriction period, especially during times when study participants would normally be sleeping. Several other studies have confirmed this finding that partial sleep loss is followed by increased calorie consumption (Brondel, Romer, Nougues, Touyarou, & Davenne, 2010; Hicks, McTighe, & Juarez, 1986).

Short sleepers may also eat more in an attempt to offset fatigue associated with sleep debt. Short sleepers frequently report fatigue and daytime sleepiness (Durmer & Dinges, 2005)

and it has been hypothesized that sleep restricted individuals might increase calorie intake in an attempt to counteract the fatigue associated with sleep loss (Magee et al., 2010).

Short sleep duration reduces voluntary energy expenditure. Sleep restriction may also contribute to the other side of the energy balance equation through decreased energy expenditure. Energy expenditure occurs both voluntarily (e.g., through physical activity), and non-voluntarily (e.g., to power basic metabolic processes). Fatigue consequent to sleep restriction could result in reduced motivation to participate in vigorous physical activity (Magee et al., 2010). Schmid et al. (2009) found that after two nights of sleep loss (four hours of sleep/night), physical activity was reduced in healthy men during free-living conditions.

Short sleep duration reduces involuntary energy expenditure. Involuntary energy expenditure occurs to support basic metabolic processes, to maintain body temperature, and in the disposal of excess energy intake (Tan, Manchester, Fuentes-Broto, Paredes, & Reiter, 2011). Although research in this area is sparse, it has also been hypothesized that sleep duration could affect involuntary energy expenditure (Taheri, 2006). Melatonin plays an important role in the regulation of involuntary energy expenditure (Tan et al., 2011). As such, the suppression of melatonin due to late night light exposure is another proposed mechanism that could explain the linkages between sleep loss and obesity (Reiter et al., 2011).

Melatonin also could influence involuntary energy expenditure via the growth and activity of brown adipose tissue. The human body contains two types of fat: white adipose tissue and brown adipose tissue (Pocock & Richards, 2006). While the function of white adipose tissue is energy storage and padding (Martini, Timmons, & Tallitsch, 2006), brown adipose tissue is metabolically active (Pocock & Richards, 2006). Brown adipose tissue is thought to have several roles in human physiology, including heat generation to maintain body temperature (i.e.,

non-shivering thermogenesis) (Pocock & Richards, 2006) and maintenance of energy balance by disposing of excess energy intake through heat production (i.e., diet-induced thermogenesis) (Tan et al., 2011). Through these mechanisms, brown adipose tissue burns energy and reduces white adipose tissue stores (Tan et al., 2011). In animals, melatonin stimulates the activity and growth of brown adipose tissue (Tan et al., 2011). As such, it is possible that melatonin suppression linked with late night light exposure could result in decreased energy expenditure and an increase in white adipose tissue reserves (Tan et al., 2011). In humans, melatonin administration has a hypothermic effect on core body temperature, but proportionally larger increases in peripheral body temperature and peripheral heat loss occur simultaneously (Tan et al., 2011). No studies thus far have examined the effect of sleep loss on brown adipose tissue activity in humans.

Epidemiological evidence for sleep-obesity link.

Review articles. Review articles report that a sizable body of research has explored the relationship between sleep duration and overweight/obesity in children and adults (Cappuccio et al., 2008; Chen et al., 2008; Marshall et al., 2008; Patel & Hu, 2008). Meta-analyses of epidemiological studies involving children and adolescents observed a significantly increased risk of OWOB in short sleepers compared to longer sleepers (Cappuccio et al., 2008; Chen et al., 2008). For example, in a meta-analysis of 13 studies involving 30,002 children and adolescents aged 2-20 years, short sleepers had a pooled odds ratio of 1.89 for obesity (Cappuccio et al., 2008). Similarly, in a systematic review of 11 studies, youth falling into the shortest-duration category of sleepers had an odds ratio of 1.92 for being OWOB compared to the longest sleepers (Chen et al., 2008).

Cross-sectional studies – adults. The majority of cross-sectional studies involving adults have demonstrated either a 'U' shaped or negative linear association between sleep duration and OWOB (Patel & Hu, 2008), with the lowest risk of OWOB occurring in adults who achieve 7-9 hours of sleep/night (Singh, Drake, Roehrs, Hudgel, & Roth, 2005; Taheri, Lin, Austin, Young, & Mignot, 2004). While some cross-sectional studies on adults have found no association between sleep duration and obesity (e.g., Gottlieb et al., 2005), the results of age-stratified analyses demonstrate that the relationship is more consistent in younger adults (Gangwisch et al., 2005).

The only Canadian research examining the relationship between sleep duration and OWOB in adults is by Chaput and colleagues in Québec (2007). In a study of 714 adults, participants who slept 5-6 hours/night had an odds ratio of OWOB of 1.38 compared to those who slept 7-8 hours per night. Participants sleeping 5-6 hours/night also had higher adiposity and lower leptin levels. These Canadian data are supported by international studies. In a cohort study of 8,073 people, initial cross-sectional analyses determined that short sleepers had an increased risk of obesity compared to longer sleepers (Gangwisch et al., 2005).

Cross-sectional studies – children and adolescents. The results of cross-sectional research examining the association between sleep duration and OWOB in children and adolescent populations are more consistent than findings in adults (Chen et al., 2008; Patel & Hu, 2008). The only pediatric obesity-sleep research study that has been completed in Canada is the 'Québec en Forme' project which involved 422 Québec children aged 5-10 years (Chaput et al., 2006). After adjusting for age, sex, and obesity risk factors, a negative linear relationship was observed between OWOB and sleep duration (Chaput et al., 2006). Compared to children

sleeping 12-13 hours/night, odds ratios for OWOB were 1.42 for those with 10.5-11.5 hours of sleep/night and 3.45 for those with 8-10 hours of sleep/night.

Cross-sectional studies from other countries support these findings from Québec. In an Australian study, Eisenmann and colleagues (2006) studied the relationship between sleep duration, waist circumference, and BMI in 6,324 youth aged 7-15. Compared to youth who slept more than 10 hours/night, youth who slept less than eight hours/night had an odds ratio for OWOB of 3.1. In a cross-sectional study of 4,486 adolescent participants in the US National Longitudinal Study of Adolescent Health, Knutson (2005) identified a negative relationship between sleep duration and BMI in males only. While many of these large population-based studies used self-reported measures of sleep duration, smaller studies using objective measures of sleep duration such as actigraphy also support these findings, providing even stronger evidence for the obesity-sleep duration relationship (Beebe et al., 2007; Gupta, Mueller, Chan, & Meininger, 2002).

Longitudinal studies – adults. Similar to the results of cross-sectional research, longitudinal studies in adults tend to reveal a U-shaped or negative linear association between sleep duration and overweight. In a longitudinal study of 276 Canadian adults, those who slept 5-6 hours/night were 35% more likely to gain weight over a six year period compared to those who slept 7-8 hours/night (Chaput, Després, Bouchard, & Tremblay, 2008). The risk of becoming overweight over the six year period was 27% higher in short sleepers compared to those sleeping 7-8 hours/night (Chaput et al., 2008). In a retrospective analysis of 68,183 women in the US Nurses' Health Study, Patel et al. (2006) found that after adjusting for age, the shortest sleepers (<5 hours of sleep/night) weighed the most at every future time point. While all women in the study tended to gain weight over time, a dose-response relationship between sleep

duration and weight gain was demonstrated: fewer hours of sleep per night were associated with larger weight gain four and 10 years later (Patel et al., 2006). In a prospective cohort study of 496 young American adults, Hasler et al. (2004) found a negative relationship between sleep duration and weight gain between ages 27 and 40. Another US-based prospective cohort study of 8,073 participants demonstrated that each additional hour of sleep at baseline was negatively associated with weight gain over the study period (Gangwisch et al., 2005).

Longitudinal studies – children and adolescents. Compared to studies on adults, fewer longitudinal studies have been conducted on children and adolescents, and none have been conducted in Canada. In a British study, Reilly et al. (2005) studied 8,234 seven-year-old children over four years; short sleep duration predicted higher BMI at follow-up (odds ratio=1.45). Similar associations have been observed in studies undertaken in the USA with 2,281 children aged 3-12 years over a 5 year period (Snell, Adam, & Duncan, 2007) as well as 9-year olds over 4.5 years (Agras, Hammer, McNicholas, & Kraemer, 2004). A long term prospective New Zealand cohort study had similar results: short sleep in childhood predicted higher BMI at age 32 (Landhuis, Poulton, Welch, & Hancox, 2008).

Obesity is only one of several established risk factors for cardiovascular disease and diabetes. While a substantial body of literature has explored short sleep duration as a risk factor for obesity, fewer studies have examined connections among sleep loss and other early indicators of cardio-metabolic disease such as impaired carbohydrate metabolism, low HDL cholesterol, or elevated triglycerides.

Short Sleep Duration and Impaired Carbohydrate Metabolism

Dysregulation of carbohydrate metabolism is thought, at least in part, to mediate the relationship between obesity and cardio-metabolic disease (Kahn & Flier, 2000). Dysregulation

of carbohydrate metabolism may be an even more sensitive predictor of cardio-metabolic disease than is obesity (Bacha, Saad, Gungor, & Arslanian, 2006). The body maintains blood glucose levels within a narrow range to avoid the life threatening consequences of hypoglycemia and hyperglycemia (Pocock & Richards, 2006). While type 2 diabetes is diagnosed by elevated blood glucose levels, hyperglycemia is the end result of progressive deteriorations in insulin sensitivity and insulin secretion (Cali & Caprio, 2008). Deteriorations in insulin sensitivity are frequently accompanied by elevations in insulin levels, or hyperinsulinemia (Kim & Reaven, 2008). The presence of hyperinsulinemia provides early information about future risk of developing diabetes as well as cardio-metabolic disease in later life (Nguyen et al., 2010).

Sleep loss could influence dysregulation of carbohydrate metabolism through increases in obesity, sympathetic nervous system activity, evening cortisol and growth hormone, and decreased glucose uptake (Knutson, 2010). While the increased adiposity associated with obesity induces impairments in carbohydrate metabolism (Kahn & Flier, 2000), sleep loss also impairs carbohydrate metabolism independent of obesity (Flint et al., 2007; Spiegel et al., 1999). Consequences of partial sleep loss include reduced brain glucose utilization as well as increases in growth hormone and evening cortisol, both of which result in impaired carbohydrate metabolism (Knutson, 2010). In addition, sleep restriction induces insulin resistance (Buxton et al., 2012; Nedeltcheva, Kessler, et al., 2009) and may also result in increased sympathetic nervous activity which results in reduced insulin secretion (Knutson, 2010).

Experimental studies have demonstrated that acute, partial sleep loss can have a negative influence on carbohydrate metabolism (Spiegel et al., 1999), and population-based studies indicate that short sleep duration is associated with future risk of diabetes (Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005; Spiegel, Tasali, Leproult, & Van Cauter, 2009). There are

a number of potential explanations for these observed linkages between sleep loss and impaired carbohydrate metabolism. Firstly, a consequence of sleep loss is increased secretion of growth hormone and cortisol as well as circulating pro-inflammatory cytokines, all of which reduce insulin sensitivity (Flint et al., 2007). Further, sleep restriction results in increased sympathetic nervous system activity, a state shown to inhibit pancreatic beta cell function and decrease insulin secretion (Flint et al., 2007). Finally, short sleepers are likely to be exposed to light at night and could subsequently exhibit suppression of melatonin secretion. In animal studies, administration of melatonin results in improvements in carbohydrate metabolism (Wolden-Hanson et al., 2000). Another explanation for the beneficial effect of melatonin is that this hormone leads to improved sleep quality, which then leads to the observed improvements in metabolic parameters (Van Cauter, 1998). The deteriorations in carbohydrate metabolism resulting from sleep loss are also likely to have negative consequences for other cardio-metabolic risk factors such as dyslipidemia and hypertension (Ferrannini, 2006).

Experimental studies involving humans have demonstrated that impairments in carbohydrate metabolism could be induced by acute partial sleep deprivation. Spiegel, Leproult, and Van Cauter (1999) demonstrated that healthy young men restricted to four hours in bed per night over six nights had significant impairment in glucose metabolism, exhibiting measurements characteristic of older adults with impaired glucose tolerance. After a seven-day period of sleep recovery (12 hours in bed per night), glucose tolerance values returned to healthy ranges. A similar study that utilized a randomized crossover design to test the effect of sleep restriction (four hours in bed/night for two nights) vs. adequate sleep (10 hours in bed/night for two nights) had similar findings (Spiegel et al., 2005). More recent studies involving both men and women confirm the findings from these early studies. For example, Buxton et al. (2010) determined that

a week-long period of sleep restriction (five hours of sleep/night) resulted in decreased glucose tolerance. In a study of 11 healthy men and women, Nedeltcheva et al. (2009) observed reduced glucose tolerance and reduced insulin sensitivity after a 14 day period of sleep restriction (5.5 hours of sleep/night). In a study of nine healthy men and women, a single night of partial sleep loss (four hours of sleep/night) resulted in decreased insulin sensitivity (Donga et al., 2010). Likewise, in a study involving shift workers in the Antarctic, workers demonstrated decreased carbohydrate tolerance after meals consumed during night shifts (Lund, Arendt, Hampton, English, & Morgan, 2001). Carbohydrate tolerance returned to normal after two days of working day shifts (Lund et al., 2001).

Epidemiological research also supports a link between sleep loss and deteriorations in carbohydrate metabolism (Knutson, 2010). In a longitudinal study of 276 Canadian adults, Chaput, Despres, Bouchard, Astrup, and Tremblay (2009) determined that over the six year study period short sleepers had a relative risk of 2.78 for developing impaired glucose tolerance or diabetes compared to those who slept 7-8 hours/night. In an African-American population with type 2 diabetes, short sleepers had an increased severity of impaired glucose regulation (Knutson, Ryden, Mander, & Van Cauter, 2006).

Apparently, only a single study has examined links between carbohydrate metabolism and sleep duration in children or adolescents. In a laboratory study of 40 obese children and adolescents ages 3-18, participants who slept fewer than six hours per night had increased insulin resistance (Flint et al., 2007). No difference in obesity was observed between the shorter and longer sleepers, indicating that the relationship between short sleep and impaired carbohydrate metabolism was independent of obesity.

Measuring Carbohydrate Metabolism. While the gold standard for assessing carbohydrate metabolism is the euglycemic clamp method, this procedure is not feasible for large population surveys because it is expensive, invasive, and time consuming (Chiarelli & Marcovecchio, 2008). Lower-cost and less invasive surrogate measures that have been validated in children and adolescents are based on simpler measures of fasting insulin and/or glucose (Chiarelli & Marcovecchio, 2008). High fasting insulin in childhood and adolescence is strongly associated with cardio-metabolic risk factors such as obesity, hypertension and dyslipidemia (Bao, Srinivasan, & Berenson, 1996; Lambert et al., 2004; Nguyen et al., 2010). Because fasting insulin is convenient to measure and has strong associations with future cardio-metabolic disease, it was chosen to represent carbohydrate metabolism for this study.

Short Sleep Duration and High Triglycerides

Although elevated triglycerides are considered a marker of increased risk for cardiovascular disease (International Diabetes Federation, 2011), relatively few studies have examined associations between sleep duration and triglyceride levels. In a US-based cross-sectional study of 1,214 adult participants, Hall et al. (2008) found that short sleep duration was associated with elevated triglycerides. In a cross-sectional study of 8,860 Norwegian participants aged 40-45, short sleep duration, compared to longer sleep duration, was related to higher triglycerides (Bjorvatn et al., 2007). Given that shift workers frequently experience sleep loss, epidemiological examination of cardio-metabolic risk factors in shift workers provides additional insight. Shift workers exhibit higher triglycerides even after adjusting for socioeconomic factors (Karlsson, Knutsson, & Lindahl, 2001). During night shifts, workers have increased post-meal triglyceride levels compared to the same meal consumed during the day (Lund et al., 2001).

Short Sleep Duration and Low HDL Cholesterol

Low HDL cholesterol is also linked to the future development of cardio-metabolic disease (International Diabetes Federation, 2011). Only a few epidemiological studies have examined relationships between sleep duration and lower HDL cholesterol. In a cross-sectional study of 8,860 Norwegian participants aged 40-45, short sleepers had lower HDL cholesterol compared to longer sleepers (Bjorvatn et al., 2007). In a prospective study involving 935 women from the Nurses' Health Study, among women who had type 2 diabetes at baseline, short sleep duration was associated with decreased HDL cholesterol four years later (C. J. Williams, Hu, Patel, & Mantzoros, 2007).

Covariates Involved in the Sleep Duration/Cardio-Metabolic Disease Relationship

Covariates could influence the relationship between sleep duration and indicators of cardio-metabolic disease through confounding and/or interaction (Szklo & Nieto, 2007). In order to better estimate the potential causal influence of sleep duration on indicators of cardio-metabolic disease in this observational study, the influence of additional variables must be accounted for. In this study this was done by selecting covariates from available Canadian Health Measures Survey (CHMS) variables that were likely to have confounding and/or interaction effects, based on evidence from the literature.

Confounding occurs when an observed association between the predictor and outcome variables is the result of the influence of a third variable, or confounder (Szklo & Nieto, 2007). Confounding variables are related to both the predictor variable and the outcome variable (Szklo & Nieto, 2007). A confounding effect can be illustrated by using a fictitious example of an observed association between inadequate sleep duration and OWOB in a population. In this example, further exploration of the data reveals that increasing age is associated both with an

increased risk of obesity and with inadequate sleep. As such, analyses must account for the confounding effect of age on the relationship between sleep duration and obesity. Inclusion of confounding variables in multivariate models is an effective method of adjusting for the effect of confounders on the relationship between a predictor variable and the outcome variable (Hosmer & Lemeshow, 2000; Szklo & Nieto, 2007).

Several techniques can be used to identify appropriate confounders to include in multivariate models. First, a clinical or scientific rationale should exist that supports the potential impact of confounders on both predictor and outcome variables (Szklo & Nieto, 2007). While significance testing has also been used as a method of searching for confounders, this method has been criticized because exhaustive searching is likely to uncover statistically significant but spurious associations (Pace, 2008). Additionally, sometimes a combination of variables, taken together, can have a confounding effect, while the individual variables do not (Hosmer & Lemeshow, 2000). A disadvantage of relying on clinical relevance alone to select variables is that the inclusion of too many variables can result in unstable estimates, or 'overfitting.' This problem can be recognized through the appearance of large standard errors and/or unrealistic coefficients (Hosmer & Lemeshow, 2000). A final strategy to identify confounders is to add the potential confounding variable(s) to the bivariate model. If addition of the variable(s) results in a change in the direction and/or magnitude of the crude association, this is indicative of a confounding effect (Szklo & Nieto, 2007).

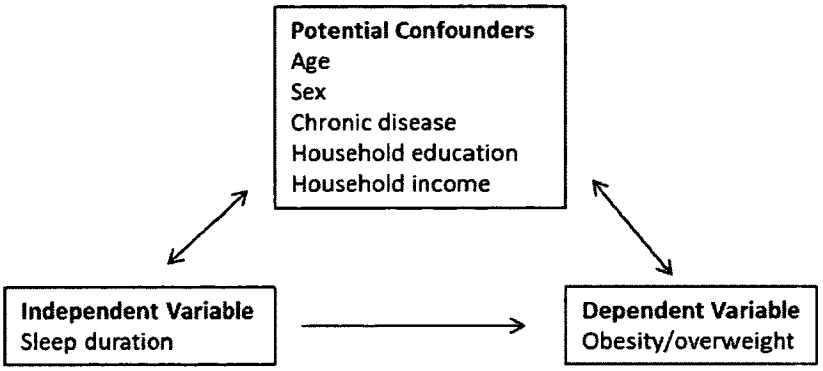
When the effect of a predictor variable on an outcome variable varies at different levels of a third variable, this is called 'interaction', 'effect modification', or 'heterogeneity of effect' (Hosmer & Lemeshow, 2000; Szklo & Nieto, 2007). For example, if short sleep duration predicts obesity in boys but not girls, then it could be said that sex modifies the relationship between

sleep duration and obesity. Interaction can be additive or multiplicative (Szklo & Nieto, 2007). In additive interaction, the difference observed for a main effect at different levels of a third variable is equal to the sum of the effects of the individual variables (Szklo & Nieto, 2007). In multiplicative interaction, a heterogeneous main effect is larger (synergistic) or smaller (antagonistic) than the individual effects of the predictor variable and the third variable together (Szklo & Nieto, 2007).

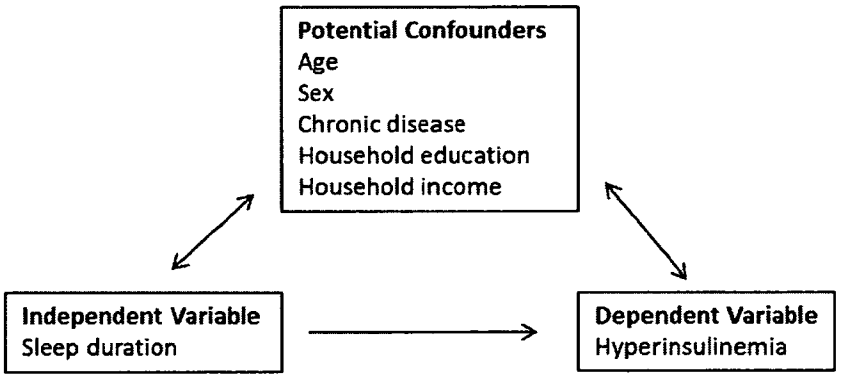
Several strategies exist to identify and address interactions in logistic regression models. To identify interactions, a list of scientifically plausible interactions is created (Hosmer & Lemeshow, 2000). Next, interaction terms are added to the main effects model one at a time and tested for significance (Hosmer & Lemeshow, 2000). Hosmer and Lemeshow (2000) suggest that interaction terms that are significant at $p<0.10$ and add to the overall model fit (i.e., likelihood ratio test) should be included in the final model to adjust for their effect. Another strategy to address interactions in logistic regression models is to stratify analyses (Szklo & Nieto, 2007). Using interaction terms confers the advantage of a larger sample size when data are sparse (Szklo & Nieto, 2007).

Main Relationships of Interest. The following diagrams illustrate hypothesized relationships among the variables of interest in this study (Figure 1). As described in further detail in chapter 2, this study utilized logistic regression models to examine the hypothesized associations among the predictor variables and the outcome variables. Note that in each of the diagrams below, the relationship shown among the predictor variables and the dependent variables is illustrated with a uni-directional arrow. However, this observational, cross-sectional study's limitations do not permit determination of the directionality of these associations, so the arrows are intended to describe only the hypothesized relationships of interest.

A. Association between sleep duration and overweight/obesity.



B. Association between sleep duration and hyperinsulinemia.



C. Association between sleep duration and low HDL cholesterol.

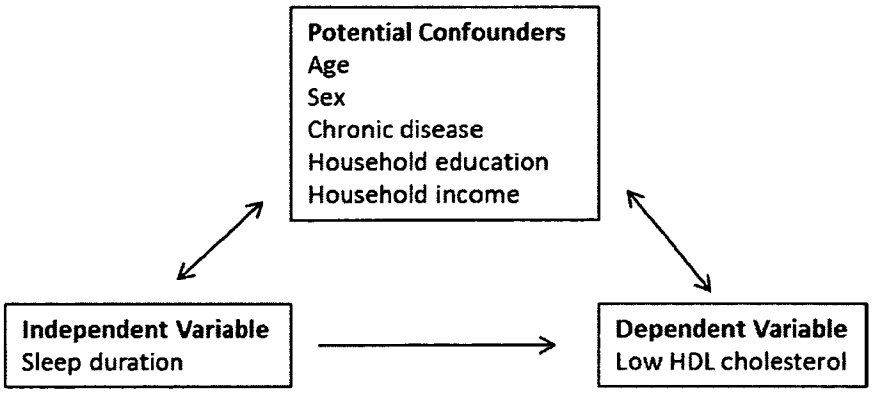
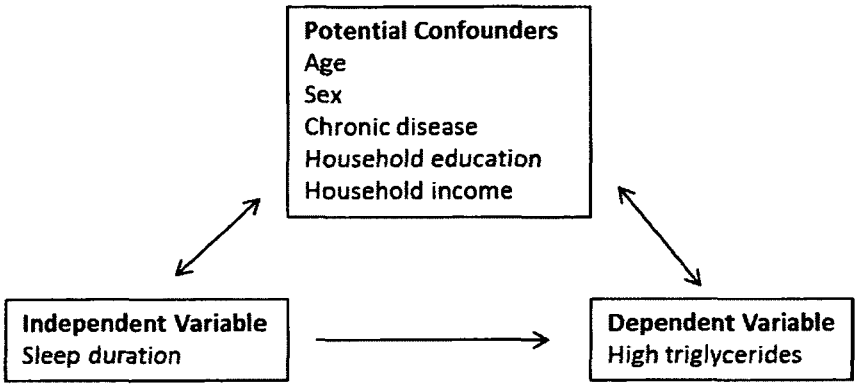


Figure 1. Main relationships of interest explored using logistic regression models.

D. Association between sleep duration and high triglycerides.



E. Predictors of sleep duration.

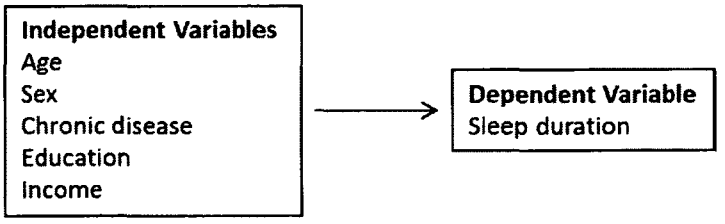


Figure 1 (continued). Main relationships of interest explored using logistic regression models.

Age. Age is likely to be a confounder in the relationship between sleep duration and indicators of cardio-metabolic disease in children and adolescents. Progression through puberty is associated with deteriorations in indicators of cardio-metabolic health (Lambert et al., 2004). Hormonal changes that transpire during puberty arise concurrently with increases in body mass and blood pressure as well as deteriorations in blood lipid levels and carbohydrate metabolism (Lambert et al., 2004).

At the same time, epidemiological data suggest that sleep duration tends to decrease through adolescence, outpacing decreases in developmentally appropriate sleep requirements (Iglowstein, Jenni, Molinari, & Largo, 2003). For example, in a population of German youth, age was the strongest predictor of sleep duration, explaining 57.0% and 41.2%, respectively, of the variance in sleep length for girls and boys (Hitze et al., 2009). No Canadian study has

examined age as a determinant of sleep duration in adolescents or children. Although it seems plausible that sleep requirements and sleep duration should be related not only to biological age but also to developmental stage because circadian rhythms are thought to vary with developmental stage (Wolfson, 2010), evidence does not support an association between pubertal stage and sleep duration (Flint et al., 2007). Thus, social factors seem to be important determinants of sleep duration. Age-related factors that influence sleep duration include altered physiological sleep requirements, social and cultural factors specific to adolescence, and advances in circadian rhythm phase that favour alertness later at night (Knutson, 2005).

It is also plausible that age could be an effect modifier in the relationship between sleep duration and cardio-metabolic disease (i.e., the associations among short sleep duration and indicators of cardio-metabolic disease might be stronger or weaker at different ages). One possible reason why this might occur is that older children with inadequate sleep may have been short sleepers since childhood. If this is the case, then adolescents with short sleep should have had a greater exposure time to the damaging effects of inadequate sleep, and then a relationship between short sleep and indicators of cardio-metabolic disease would only surface in older children, or be stronger in older children. Analyses were stratified by age to test for this effect.

Sex/gender. Sex and gender may also confound the relationship between sleep duration and cardio-metabolic disease because biological and social factors linked to sex and gender may influence both sleep duration and indicators of cardio-metabolic disease. Data from a recent Canadian health survey found that adolescent boys were more likely to be OWOB compared to girls (31% vs. 25%) (Statistics Canada, 2010c). The sex-specific hormone changes that occur during adolescence could have different effects on weight gain (Knutson, 2005). For example,

while adolescent males exhibit increases in testosterone and muscle mass, adolescent females demonstrate increases in estrogen and fat tissue (Martini et al., 2006).

For adults, social factors mediate observed sex differences in sleep duration (Hurst, 2008), but for children the relationship between sex and sleep duration or quality is less well studied. There is no difference in total sleep time for adolescent girls versus adolescent boys, but females and males have different sleep patterns, with girls awaking earlier on weekdays and later on weekends compared to boys (Lee, McEnany, & Weekes, 1999). Sleep architecture also differs by sex, with females spending proportionally more time in the slow wave sleep stage compared to males (Briere, Forest, Chouinard, & Godbout, 2003).

Interestingly, the obesity-sleep duration relationship seems to differ by sex (Patel & Hu, 2008). While many studies have found positive associations between sleep duration and obesity for both sexes (Patel & Hu, 2008), the risk for obesity with short sleep duration seems to be higher for boys (Chaput et al., 2006; Eisenmann et al., 2006; Gibson, Lambert, & Neate, 2004; Knutson, 2005; Sekine et al., 2002). In the only existing study conducted in Canada, Chaput et al. (2006) found that while short sleep duration was positively associated with OWOB in 5-10 year-olds for both sexes combined, stratified analyses showed that the relationship was only statistically significant in boys. Indeed, in a meta-analysis of 11 studies, the pooled odds ratio for OWOB in children and adolescents with short sleep duration was 2.50 for boys, compared to 1.24 for girls (Chen et al., 2008). Because the effect of sleep loss on cardio-metabolic risk appears to be sex-specific, analyses were stratified by gender.

There are several possible explanations for why the sleep duration-obesity relationship seems to be modified by sex (Knutson, 2005). Compared to boys, girls may demonstrate unfavourable cardio-metabolic changes only with larger magnitudes of sleep loss. Females may

have better sleep quality as they spend more time in slow wave sleep (Briere et al., 2003). Further, females are more resistant to environmental stress, and have lower mortality rates in response to malnutrition, injury, and infectious disease (Eisenmann et al., 2006; Wells, 2000).

Socioeconomic status. Literature suggests that socioeconomic status is also a confounder in the relationship between sleep duration and cardio-metabolic disease. Internationally, research has demonstrated that lower socioeconomic status is related to poorer health outcomes, including indicators of cardio-metabolic risk (Marmot, 2003). Low socioeconomic status populations also tend to get less sleep than more affluent groups (Jarrin, Silverstein, & McGrath, 2009; Moore, Adler, Williams, & Jackson, 2002). Van Cauter and Spiegel (1999) postulate that socioeconomic status is linked to short sleep duration through poor housing conditions (e.g., crowded and/or unsafe housing, noise, and cold or hot temperatures) and increased stress and anxiety, which are also related to poor sleep. Another potential mechanism is related to the observation that patterns of cortisol secretion tend to be altered in low-socioeconomic populations (Dowd, Simanek, & Aiello, 2009; Lupie, King, Meaney, & McEwen, 2001). Given that high cortisol levels at night are known to cause sleep difficulties (Rodenbeck, Huether, Rüther, & Hajak, 2002), these alterations in cortisol secretion could affect sleep quality and duration.

Epidemiological research in both Canadian and international populations has examined socioeconomic status as a determinant of sleep duration in children and adolescents. The nature of the relationship between socioeconomic status and sleep duration differs from country to country. Internationally, the extent to which social and health policies address socioeconomic inequities differs significantly, and thus it is logical that the influence of socioeconomic status on sleep duration would likewise vary between countries.

In a study of 183 Canadian youths aged 8-18 years, linear relationships were observed between socioeconomic status and each of sleep duration (r = .20), sleep quality (r = .25), and daytime sleepiness (r = -.25) (Jarrin et al., 2009). In an American study of children aged 2-7 years old, McLaughlin Crabtree et al. (2005) observed that lower socioeconomic status was associated with impaired "sleep behaviour scores", a composite measure of healthy sleep behaviours and characteristics (e.g., bedtime hour, having nightmares, daytime sleepiness). In contrast, other studies have not found associations between sleep duration and socioeconomic status. In a German study of 414 boys and girls aged 6-19 years, no significant association was observed between socioeconomic status and sleep duration (Hitze et al., 2009). In an Australian study of 591 seven-year old children, Nixon et al. (2008) also did not find a relationship between sleep duration and socioeconomic status.

The possibility that the association between sleep duration and cardio-metabolic indicators could differ at various levels of socioeconomic status is also conceivable. No research could be located that examined whether the relationship between sleep duration and cardio-metabolic risk varies at different levels of socioeconomic status. The possibility of an interaction effect was explored through testing of interaction terms.

Chronic conditions. Chronic health conditions could be a confounder in the sleep/cardio-metabolic disease relationship through a number of complex pathways. Firstly, symptoms associated with chronic health conditions cause sleep difficulties. For example, people suffering from chronic pain are more likely to report sleep disturbance (Smith & Haythornthwaite, 2004). Similarly, while deterioration in cardio-metabolic health may be a consequence of chronic sleep loss, type 2 diabetes and obesity may also play an etiologic role in sleep disturbance (Nieto et al., 2006). For example, type 2 diabetes induces changes in nervous

system function which are thought to cause sleep apnea (Nieto et al., 2006). Finally, some medications can affect both sleep and cardio-metabolic health. For example, inflammatory diseases are often treated with glucocorticoid medications that, similar to the effects of endogenous cortisol described above, cause side-effects that include insomnia, hypertension, weight gain, and alterations in carbohydrate metabolism (Mayo Clinic, 2010; U. S. National Library of Medicine, 2011).

A complex relationship connects psychiatric disorders to sleep disturbances and cardio-metabolic risk factors. Patients with psychiatric disorders frequently report sleep disturbances (Nieto et al., 2006; Smith & Haythornthwaite, 2004; Wulff, Gatti, Wettstein, & Foster, 2010) including both insomnia and hypersomnia (Benca, 2008). While behavioural factors related to psychiatric disorders can cause sleeping problems, physiological abnormalities can also be a common pathogenic factor in the etiology of both psychiatric conditions and sleep disturbance (Benca, 2008). Some psychiatric disorders are also linked to factors leading to poor cardio-metabolic health. For example, night-eating syndrome and seasonal affective disorder are both linked to overeating and weight gain (Benca, 2008). Further, symptoms associated with psychiatric disorders could lead to reduced motivation and/or ability to exercise. Medications used to treat psychiatric disorders (e.g., selective serotonin reuptake inhibitors, antipsychotics) can have deleterious effects on body mass and carbohydrate metabolism (Allison et al., 1999; Kivimäki et al., 2010). Finally, stimulant medications used to treat attention deficit/hyperactivity disorder can cause sleep disturbances (Corkum, Panton, Ironside, MacPherson, & Williams, 2008; Schwartz et al., 2004).

Behaviour covariates. Knowledge of behaviours that influence sleep duration and cardio-metabolic risk factors is important because behaviours are potentially modifiable.

Moreover, behaviours that independently influence cardio-metabolic risk are potential confounders in this study. Relationships among behaviours, sleep duration and cardio-metabolic risk are complicated. Behaviours may be causes or consequences of short sleep duration and/or cardio-metabolic risk factors. Several behaviours have been explored as covariates in the sleep-obesity relationship, including physical activity, TV watching, video game playing, and computer time.

Physical activity. Physical activity is a likely confounder in the sleep duration/cardio-metabolic risk relationship. While it is fairly well established that engagement in vigorous physical activity is related to favourable cardio-metabolic health (Yassine et al., 2009), evidence also suggests that exercise has an independent, favourable impact on sleep duration and quality. In a laboratory study of 11 children, a period of high-intensity aerobic exercise during the day was followed by increased time spent in deeper sleep stages (slow wave sleep) and improved ease of falling asleep compared to sleep after less intense exercise (Dworak et al., 2008). Epidemiological studies also show that physically active youth sleep better. In a study involving 434 Swiss adolescents, teen athletes had better sleep quality, fell asleep faster, awakened less often during the night, and had less daytime tiredness than the comparison group (Brand et al., 2010). In an American study, physical activity was related to decreased sleep disruption (Gupta et al., 2002). Similarly, cardiorespiratory fitness and physical activity are related to better sleep quality and longer sleep duration, respectively, in Portuguese adolescent females and German girls (Hitze et al., 2009; Mota & Vale, 2010). Long sleepers are also more likely to be involved in organized sports (von Kries, Toschke, Wurmser, Sauerwald, & Koletzko, 2002). Additionally, vigorous physical activity improves mood and reduces stress and anxiety (Strong et

al., 2005) which should improve sleep quality and duration (Uhde, Cortese, & Vedeniapin, 2009).

Television watching. Television watching is an independent risk factor for cardio-metabolic risk factors (Chaput, Klingenberg, Astrup, et al., 2010). Not only is television watching a sedentary activity, but television time may also increase food consumption independently of appetite (Blass et al., 2006; Chaput, Klingenberg, Astrup, et al., 2010). While largely unstudied, potential relationships between television watching and sleep duration are likely complex. Firstly, television watching could be a cause of sleep loss. Specifically, people may give up sleep in order to watch more television, or television viewing could decrease ease of falling asleep by having a stimulating effect. Watching television could also be a consequence of sleep difficulty (people watch television due to being unable to fall asleep). Finally, television watching could also be a reflection of an unhealthy lifestyle. Other screen-related activities such as video game playing and computer usage are similarly linked with sleep difficulties (Garrison, Liekweg, & Christakis, 2011).

Food intake. As with other covariates examined here, the connections among food intake, sleep duration, and cardio-metabolic risk factors are complex. Eating behaviour plays a role in the development of obesity, cardiovascular disease, and diabetes, and eating habits can also be a reflection of overall lifestyle. Further, sleep duration may also independently influence eating habits. As described earlier, experimental research on healthy adults demonstrates that chronic partial sleep deprivation results in changes in appetite-related hormones (Spiegel et al., 1999; Spiegel, Tasali, et al., 2004) as well as in increases in subjective appetite (Brondel et al., 2010) and more calories consumed from snacks (Nedeltcheva, Kilkus, et al., 2009).

No experimental studies have explored the effect of sleep loss on eating behaviour in children or adolescents, but epidemiological studies have explored this linkage. For example, in a Finnish study of 1,265 10-11 year old children, short sleep duration was related to increased consumption of energy-rich foods and lower consumption of nutrient-dense foods (Westerlund, Ray, & Roos, 2009). Similarly, Hitze et al. (2009) found that German children who were short sleepers were more likely to consume soft drinks and fast food. In a population of American adolescents aged 16-19, short sleep duration was linked to increased snacking behaviour and higher fat and carbohydrate intake (Weiss et al., 2010).

Other variables. This study does not account for all variables potentially related to cardio-metabolic disease or sleep duration. For example, other covariates in the sleep-obesity relationship include ethnicity, parental obesity, breastfed status, dietary habits, and substance use (Chaput et al., 2006; Hasler et al., 2004; Knutson, 2005). These variables are not examined in this study because the Canadian Health Measures Survey did not collect data on these items, or the variables could not be used for methodological reasons such as sample size, age range coverage, or missing data.

Gaps in the Literature

Based on the literature reviewed here, several knowledge gaps were illuminated, providing a rationale for this research. Firstly, while some international research exists on the relationship between sleep duration and obesity in children and adolescents, Canadian research is sparse. Only one study has been conducted with Canadian children, and this was a relatively small study restricted to Québecois children aged 5-10 years (Chaput et al., 2006). No study has examined sleep duration and obesity among Canadian adolescents. Additionally, while experimental and epidemiological research on adults supports linkages among sleep loss,

hypertension, dyslipidemia, and impaired carbohydrate metabolism, few studies have examined these relationships in children or adolescents. Only a single study with a relatively small sample size could be located that examined relationships between insulin resistance and sleep duration in a population of obese children and adolescents (Flint et al., 2007). No such research has been undertaken in order to explore similar relationships to other cardio-metabolic risk factors. Finally, no study could be located that identified the determinants, or predictors, of short sleep duration in Canadian children and adolescents.

Study Objectives and Hypotheses

There were two main objectives of this study. Firstly, this study investigated associations between short sleep duration and indicators of cardio-metabolic disease in a nationally representative sample of Canadian children and adolescents. Secondly, this study explored determinants of short sleep duration in this population. To achieve these research objectives, this study tested two hypotheses. First, it was hypothesized that Canadian youth with short sleep duration exhibit greater odds of having indicators of cardio-metabolic disease, including obesity, dyslipidemia, and dysregulation of carbohydrate metabolism compared to longer sleepers. Secondly, it was hypothesized that increasing age, low socioeconomic status, low household education, and having a chronic condition are all associated with increased odds of short sleep duration.

Chapter 2: Methods

Participants

Data for this research were obtained from the 2007-2009 Canadian Health Measures Survey (CHMS) (Statistics Canada, 2010b), a nationally representative Canadian population health survey. Further details regarding the CHMS are included in Appendix A. Inclusion criteria were participants aged 6-17 years old; participants with diabetes were excluded. Two data files were used for analyses, including the full sample (n=1690), and the fasting subsample that only included participants who were fasting (n=735).

Materials and Procedures

Measures.

Sleep duration. Sleep duration was gathered by self-report through the survey question "How many hours do you usually spend sleeping in a 24 hour period, excluding time spent resting?" Parents/guardians answered the question for children aged 6-11 years. Sleep duration was recoded from a continuous variable to a dichotomous variable ('short sleeper' vs. 'not a short sleeper') based on age-specific sleep requirements (Chen et al., 2008). According to Chen et al. (2008), children aged 6-10 years need at least 10 hours of sleep per night, and those aged 11-17 years need at least nine hours of sleep per night. Participants who did not meet these sleep requirements were assigned to the 'short sleeper' category, while those who met sleep requirements were categorized as 'not a short sleeper'.

Overweight/obesity. In the clinical exam, height and weight measurements were performed by trained professionals. Weight was measured with a Mettler Toledo digital scale, and standing height was measured with a stadiometer. From these measurements, body mass index (BMI) was calculated by dividing body mass (kg) by height squared (m^2). The original

CHMS variable classified participants as obese, overweight, or neither using cut-points based on z-scores standardized for age and sex from pooled international data (Cole et al., 2000). To create a dichotomous variable, the original three categories were recoded to two categories, with participants in either the overweight or obese categories assigned to the 'overweight or obese' category, and no change made to those originally designated as 'neither'.

Insulin, HDL cholesterol, and triglycerides were measured using fasting blood tests. Cut-points for the associated dichotomous variables were established using criteria used by Lambert et al. (2004). The continuous insulin data were recoded into a dichtomous variable, with the 75th percentile designated as the cut-point for hyperinsulinemia versus no hyperinsulinemia (Lambert et al., 2004). For low HDL cholesterol, the 25th percentile was designated as the cut-point, with those falling below the 25th percentile assigned to the 'low HDL cholesterol' category (Lambert et al., 2004). The cut point for 'high triglycerides' was set at the 75th percentile.

Age. Age was self-reported, and was defined as the age, in years, given at the time of the clinical examination. In logistic regression, predictor variables must have a linear relationship with the outcome variable (Hosmer & Lemeshow, 2000). To meet this assumption, the age variable was recoded from a continuous variable to a dichotomous variable with categories for ages 6-11 and ages 12-17.

Sex. Sex was self-reported at the time of the clinical examination and consisted of two categories (male/female).

Chronic conditions. Chronic health conditions were self-reported and based on survey questions that probed for clinically diagnosed conditions (Table 19, Appendix A). Respondents with at least one chronic condition were coded as having a chronic condition. Chronic health

conditions with the highest frequencies included 'other' (13.1%), asthma (11.2%), learning disability (8.1%), back problems (4.2%), and mood disorders (1.6%).

Education. Household education was self-reported, and was defined as the highest level of education achieved by any member of the household. To preserve stability of the estimates, the education variable was collapsed from four strata to two strata as per recommendations from Statistics Canada. For education, the upper-most stratum (has post-secondary degree/diploma) was recoded as 'higher education' and the bottom three strata (less than secondary, secondary, or some post-secondary) were assigned to the 'lower education' category.

Income. Income was self-reported, was originally a dichotomous variable ('low income' vs. 'middle/high income') and was not altered.

Data Analyses

Data were analyzed using the statistical software packages SPSS 20.0 and WesVar 5.1. Descriptive statistics were calculated to provide an overall view of the study population characteristics (Table 2). Initial checks were conducted to ensure that the data met the assumptions for the statistical tests. Statistical significance was assessed at $p < .05$. In logistic regression models, statistical significance was assessed for the overall model (likelihood ratio test) and for the predictive ability of the individual variables (odds ratio with confidence interval). Population weights were utilized to develop nationally representative estimates. To account for the complex CHMS sampling design, bootstrapping was used to calculate the coefficient of variation and confidence intervals.

Testing for interaction terms was a two-step process recommended by Hosmer and Lemeshow (2000). The first step involved identifying scientifically plausible interaction terms. Each interaction term was then added to a bivariate logistic regression model. Interaction terms

associated with the outcome variable with a significance level of at least $p < .10$ were then added to multivariate models with all covariates. If the interaction term added to the predictive ability of the model, then it was addressed in the analyses. Models were also stratified by age and sex to further test for heterogeneity of effect. Although the full set of coefficients for each of the models is presented in tabular format throughout the results section, the interpretation of coefficients was restricted to key predictor variables relevant to the original research questions.

Objective 1. To examine associations between sleep duration and the four indicators of cardio-metabolic disease, four logistic regression models were developed. These models are represented in Figures 1A-1D (Chapter 1). The first regression model included sleep duration as the predictor variable and OWOB as the outcome variable, with having a chronic condition, household education, and household income included as covariates (Figure 1A). This model was tested using both the full sample and the fasting subsample. Using the fasting subsample, subsequent parallel regression models were tested with each of hyperinsulinemia (Figure 1B), high triglycerides (Figure 1C), and low HDL cholesterol (Figure 1D) as the outcome variable respectively.

Although both the full and fasting samples could have been utilized for the models predicting OWOB, the full sample (n=1690) was used for OWOB initially because the larger sample size in the full sample provided more statistical power. However, in order to compare the hypothesized relationships between short sleep duration and each of the different cardio-metabolic indicators that require the fasting sample (hyperinsulinemia, low HDL, and high triglycerides), the logistic regression analyses for OWOB were repeated using the fasting sample (n=735).

Objective 2. To identify determinants of sleep duration, logistic regression models were developed, with age, sex, chronic conditions, education and income entered as predictor variables, and sleep duration included as the outcome variable. This model is represented in Figure 1E (Chapter 1). Models were also stratified by age and sex to explore whether the predictors of short sleep duration are different for girls compared to boys, and for children compared to adolescents. The full sample was utilized for these regression models because the sample size was larger and it contained all variables necessary for the analyses.

Ethics

Several measures were taken to ensure this research project was conducted ethically, including following Statistics Canada policies and procedures, and ensuring the data were collected ethically (Statistics Canada, 2010d). To get permission to access the CHMS data, a proposal was submitted to the Social Sciences and Humanities Research Council of Canada (SSHRC) for review, which included an evaluation of threats to confidentiality, as well as the feasibility and utility of the research. All researchers involved in the project underwent a security screening check, and agreed to abide by the Statistics Act (Statistics Canada, 2010a, 2010e). Data were accessed from within the secure Research Data Centres (RDCs) at the University of Victoria and the University of British Columbia. No raw data were removed from the RDC: only the results of analyses were removed after first being vetted by Statistics Canada analysts (Statistics Canada, 2010d). Numerous measures were in place to ensure that the CHMS data collection and storage was conducted ethically, including working closely with partners such as the Health Canada Research Ethics Board and the Office of the Privacy Commissioner of Canada (Day, Langlois, Tremblay, & Knoppers, 2007). Day et al. (2007) provide extensive detail regarding the ethical measures in place for the CHMS.

Chapter 3: Results

Data Screening and Cleaning

The full CHMS sample included 1690 participants aged 6-17 years with complete data, and the fasting subsample had 735 participants with complete data. Characteristics of the study participants are documented in Table 2, including proportions and standard errors (SE).

Table 2

Characteristics of the Study Population (Bootstrapped Data)

	Full sample (n=1690)	Fasting sample (n=735)
	% (SE)	% (SE)
Age category		
6-11 years	49.8 (0.8)	49.4 (1.3)
12-17 years	50.2 (0.8)	50.6 (1.3)
Sex		
Male	53.0 (0.9)	53.2 (1.2)
Female	47.0 (0.9)	46.8 (1.2)
Sleep adequacy		
Short sleeper	44.0 (1.4)	44.7 (2.5)
Not a short sleeper	56.0 (1.4)	55.3 (2.5)
OWOB status		
OWOB	25.5 (2.2)	28.1 (2.7)
No OWOB	74.5 (2.2)	71.9 (2.7)
Insulin levels		
Hyperinsulinemia	-	23.0 (1.8)
No hyperinsulinemia	-	77.0 (1.8)
Triglyceride levels		
High triglycerides	-	23.8 (2.0)
No high triglycerides	-	76.2 (2.0)
HDL levels		
Low HDL	-	24.7 (2.0)
No low HDL	-	75.3 (2.0)
Household education		
Below post-secondary	20.2 (2.7)	20.3 (3.5)
Post-secondary	79.8 (2.7)	79.7 (3.5)
Household income		
Low	23.9 (2.9)	24.8 (2.8)
Middle/high	76.1 (2.9)	75.2 (2.8)
Chronic condition		
Yes	30.5 (2.9)	33.0 (3.4)
No	69.5 (2.9)	67.0 (3.4)

Note. OWOB=Overweight or obese, HDL=High density lipoprotein.

Missing Data. Participants with missing data for any of the study variables were excluded from analyses. In the full sample, of the 1876 participants who met inclusion criteria, 186 had missing data and were excluded, leaving a final sample size of 1690. In the fasting

subsample, 828 participants met inclusion criteria, and 93 with missing data were excluded. Based on recommendations from Statistics Canada, for participants with insulin levels coded as below the lowest detectable limit of 14.43 pmol/L, data were imputed using the $L/\sqrt{2}$ method (Hornung & Reed, 1990). Using the $L/\sqrt{2}$ method, the geometric standard deviation (GSD) was first obtained by log-transforming the insulin data then calculating the exponential value of the standard deviation. Because the calculated GSD was below 3.0, the lowest detectable limit for insulin was divided by $\sqrt{2}$ to get 10.20 pmol/L. This figure was subsequently imputed for all participants with insulin data coded as below the detectable limit.

Chi-square tests were performed to examine differences between participants with and without missing data for the full sample and fasting subsample (Table 3). In the full sample, analyses indicated that participants with missing data were older, more likely to be short sleepers, more likely to have a chronic condition, and more likely to have low household education levels. The same pattern was observed in the fasting subsample, with the exception that education level and sleep duration did not differ according to missing data status. In the fasting subsample, participants with missing data were more likely to have hyperinsulinemia.

Model Building Process. During the initial data exploration process, several variables were eliminated from the models. Although it had been hoped that findings from the activity monitor data could provide information on physical activity and sleep duration, these data were excluded because of a large amount of missing data. Self-reported measures of physical activity were different for children and adolescents, so these variables were also eliminated from the models.

Table 3

Characteristics of Participants with Complete vs. Missing Data (Bootstrapped Data)

Characteristic	Full sample (n=1876)			Fasting subsample (n=828)		
	Complete (n=1690)	Missing (n=186)		Complete (n=735)	Missing (n=93)	
	% (SE)	% (SE)	p^a	% (SE)	% (SE)	p^a
Age			**<.001**			**.001**
6-11 years	**49.8 (0.8)**	**21.0 (3.2)**		**49.4 (1.3)**	**31.9 (4.3)**	
12-17 years	**50.2 (0.8)**	**79.0 (3.2)**		**50.6 (1.3)**	**68.1 (4.3)**	
Sex			.305			.401
Boys	53.0 (0.9)	48.9 (3.8)		53.2 (1.2)	48.8 (5.5)	
Girls	47.0 (0.9)	51.1 (3.8)		46.8 (1.2)	51.2 (5.5)	
OWOB status			.862			.257
OWOB	25.5 (2.2)	26.1 (4.9)		28.1 (2.7)	33.5 (7.6)	
Not OWOB	74.5 (2.2)	73.9 (4.9)		71.9 (2.7)	66.5 (7.6)	
Sleep duration			**<.001**			.536
Inadequate	**44.0 (1.4)**	**56.2 (5.9)**		44.7 (2.5)	48.0 (8.9)	
Adequate	**56.0 (1.4)**	**43.8 (5.9)**		55.3 (2.5)	52.0 (8.9)	
Insulin			-			**.001**
Hyperinsulinemia	-	-		**23.0 (1.8)**	**39.4 (7.8)**	
No hyperinsulinemia	-	-		**77.0 (1.8)**	**60.6 (7.8)**	
Triglycerides			-			.111
High triglycerides	-	-		23.8 (2.0)	31.1 (6.9)	
No high triglycerides	-	-		76.2 (2.0)	68.9 (6.9)	
HDL			-			.053
Low HDL	-	-		24.7 (2.0)	33.7 (6.9)	
No low HDL	-	-		75.3 (2.0)	66.3 (6.9)	
Household education			**.004**			.974
Below post secondary	**20.2 (2.7)**	**29.7 (5.3)**		20.3 (3.5)	19.7 (5.1)	
Post secondary	**79.8 (2.7)**	**70.3 (5.3)**		79.7 (3.5)	80.3 (5.1)	
Household income			.109			.614
Low	23.9 (2.9)	31.7 (12.4)		24.8 (2.8)	21.8 (9.4)	
Middle/high	76.1 (2.9)	68.3 (12.4)		75.2 (2.8)	78.2 (9.4)	

Table 3 (continued)

Characteristics of Participants with Complete vs. Missing Data (Bootstrapped Data)

Characteristic	Full sample (n=1876)			Fasting subsample (n=828)		
	Complete (n=1690)	Missing (n=186)		Complete (n=735)	Missing (n=93)	
	% (SE)	% (SE)	p^a	% (SE)	% (SE)	p^a
Chronic condition			**.008**			.628
Yes	**30.5 (2.9)**	**39.6 (6.9)**		33.0 (3.4)	35.6 (7.8)	
No	**69.5 (2.9)**	**60.4 (6.9)**		67.0 (3.4)	64.4 (7.8)	

Note. **Bold=$p < .05$.** OWOB=Overweight or obese, HDL=High density lipoprotein.
[a]X^2 test for differences between participants with full and missing data for at least one variable.

Assumptions. The data were checked to ensure that the assumptions for the analyses were met. Using the 90-10 split method (Tabachnick & Fidell, 2007) no univariate outliers were detected. The unstratified multivariate models had adequate fit, indicating no need to search for multivariate outliers (Tabachnick & Fidell, 2007). No multicollinearity was identified after examining the standard errors and correlations among the independent variables from the logistic regression output. All models satisfied the minimum ratio of at least 20 cases per independent variable (Hosmer & Lemeshow, 2000). Although all interaction terms tested were not significant and thus were not included in the final models, models were stratified by age and sex to further examine for heterogeneity of effect and to provide estimates comparable to other studies.

Objective 1: To examine associations between short sleep duration and indicators of cardio-metabolic disease.

Associations Between Short Sleep Duration and Overweight/Obesity. Based on CHMS data, the national prevalence of OWOB in the Canadian population aged 6-17 was 25.5%. Characteristics of participants classified as either OWOB or Not OWOB are outlined in

Table 4. Chi square analyses indicated that participants with short sleep duration, adolescent age, and lower household education were more likely to be OWOB.

Table 4

Characteristics of Participants With and Without OWOB (Bootstrapped, Full Sample)

Characteristic	OWOB (n=393) % (SE)	Not OWOB (n=1297) % (SE)	p^a
Sleep duration			**<.001**
Inadequate	**52.5 (2.8)**	**41.1 (1.7)**	
Adequate	**47.5 (2.8)**	**58.9 (1.7)**	
Age			**.047**
6-11 years	**45.7 (2.1)**	**51.2 (1.5)**	
12-17 years	**54.3 (2.1)**	**48.8 (1.5)**	
Sex			.068
Male	56.8 (2.1)	51.7 (1.1)	
Female	43.2 (2.1)	48.3 (1.1)	
Household education			**.004**
Below post secondary	**25.0 (4.0)**	**18.5 (2.7)**	
Post secondary	**75.0 (4.0)**	**81.5 (2.7)**	
Household income			.427
Low	25.3 (4.0)	23.5 (3.2)	
Middle/high	74.7 (4.0)	76.5 (3.2)	
Chronic condition			.088
Yes	33.7 (3.8)	29.3 (3.0)	
No	66.3 (3.8)	70.7 (3.0)	

Note. **Bold=$p < .05$**. OWOB=Overweight or obese. [a]X^2 test for differences in characteristics among those with and without OWOB.

The results of logistic regression analyses with sleep duration as the predictor variable and OWOB as the outcome variable are presented in Table 5. In a bivariate logistic regression model with sleep duration as the predictor variable and OWOB as the outcome variable, short sleepers had an odds ratio of 1.59 (95% CI 1.19-2.88) for OWOB compared to children and

adolescents who reported meeting recommended sleep duration guidelines. In a multivariate model adjusting for the effect of age, sex, chronic conditions, education, and income, the odds ratio for OWOB in short sleepers were slightly attenuated to 1.56 (95% CI=1.15-2.19). In sex-stratified models, boys with short sleep had increased odds of OWOB (OR=1.85, 95% CI=1.19-2.88), but girls did not (OR=1.27, 95% CI=0.63-2.57). In age-stratified analyses, short sleepers aged 12-17 had increased odds of OWOB (OR=1.77, 95% CI=1.05-2.99) but no significant effect was observed in children aged 6-11 years (OR=1.37, 95% CI=0.84-2.22). Table 6 presents the coefficients for all variables included in the models.

Table 5

Odds Ratios For OWOB in Short Sleepers (Bootstrapped Data)

Model	Model[d]	OR (95% CI)
Bivariate	**<.001**	**1.59 (1.19-2.88)**
Multivariate[a]	**<.001**	**1.56 (1.15-2.19)**
Boys multivariate[b]	**<.001**	**1.85 (1.19-2.88)**
Girls multivariate[b]	**.007**	1.27 (0.63-2.57)
Age 6-11 multivariate[c]	**.040**	1.37 (0.84-2.22)
Age 12-17 multivariate[c]	**.001**	**1.77 (1.05-2.99)**

Note. **Bold=$p < .05$.** OWOB=Overweight or obese
[a]Odds ratio adjusted for age, sex, chronic conditions, education and income.
[b]Odds ratio adjusted for age, chronic conditions, education and income.
[c]Odds ratio adjusted for sex, chronic conditions, education and income.
[d]Likelihood ratio test for significance of full models vs. constant-only models.

Table 6

Odds Ratios (95% CI) for All Variables Included in Multivariate Models Predicting OWOB (Bootstrapped Data, Full Sample)

Predictor variable	Unstratified Model[a]	Sex-stratified Models[b]		Age-stratified Models[c]	
		Boys	Girls	6-11 years	12-17 years
Short sleeper	**1.56 (1.15-2.19)**	**1.85 (1.19-2.88)**	1.27 (0.63-2.57)	1.37 (0.84-2.22)	**1.77 (1.05-2.99)**
Adolescent age	1.09 (0.78-1.52)	1.11 (0.75-1.65)	1.07 (0.57-1.99)		
Male sex	0.80 (0.64-1.00)	-	-	0.87 (0.63-1.19)	0.75 (0.46-1.21)
Chronic condition	0.88 (0.62-1.25)	0.92 (0.58-1.45)	0.86 (0.54-1.38)	1.21 (0.73-2.01)	0.70 (0.38-1.29)
Low education	0.68 (0.44-1.07)	0.68 (0.30-1.54)	**0.67 (0.47-0.94)**	0.74 (0.31-1.82)	0.64 (0.34-1.22)
Low income	0.97 (0.59-1.62)	1.41 (0.62-3.22)	0.66 (0.44-1.01)	0.78 (0.34-1.76)	1.20 (0.74-1.95)
Model[d]	**<.001**	**<.001**	**.007**	**.040**	**.001**

Note. **Bold=$p < .05$**

[a]Odds ratio adjusted for age, sex, chronic conditions, education and income.

[b]Odds ratio adjusted for age, chronic conditions, education and income.

[c]Odds ratio adjusted for sex, chronic conditions, education and income.

[d]Likelihood ratio test for significance of full models vs. constant-only models.

Associations Between Short Sleep Duration and Hyperinsulinemia. The fasting subsample (n=735) was used to conduct all analyses involving hyperinsulinemia, low HDL cholesterol, and high triglycerides. Based on the CHMS dataset, the national prevalence of hyperinsulinemia in the Canadian population aged 6-17 was 23.0%. Characteristics of participants with and without hyperinsulinemia are outlined in Table 7. Chi square analyses indicated that participants with short sleep duration, aged 12-17 years, with lower household education, or with a chronic condition were more likely to have hyperinsulinemia (Table 7).

Table 7

Characteristics of Participants With and Without Hyperinsulinemia (Bootstrapped Data, Fasting Subsample)

Characteristic (n=735)	Hyperinsulinemia (n=158) % (SE)	No Hyperinsulinemia (n=577) % (SE)	p^a
Sleep duration			**.001**
Inadequate	**55.6 (4.2)**	**41.5 (3.2)**	
Adequate	**44.4 (4.2)**	**58.5 (3.2)**	
Age			**<.001**
6-11 years	**23.5 (3.9)**	**57.1 (2.4)**	
12-17 years	**76.5 (3.9)**	**42.9 (2.4)**	
Sex			.588
Male	51.4 (3.1)	53.8 (1.3)	
Female	48.6 (3.1)	46.2 (1.3)	
Household education			**.013**
Below post secondary	**27.0 (6.4)**	**18.3 (3.2)**	
Post secondary	**73.0 (6.4)**	**81.7 (3.2)**	
Household income			.278
Low	21.6 (4.4)	25.7 (2.8)	
Middle/high	78.4 (4.4)	74.3 (2.8)	
Chronic condition			**.002**
Yes	**42.7 (7.7)**	**30.1 (3.5)**	
No	**57.3 (7.7)**	**69.9 (3.5)**	

Note. **Bold=$p < .05$** [a]X^2 test for differences in characteristics among participants with and without hyperinsulinemia.

In a bivariate logistic regression model with sleep duration as the predictor variable and hyperinsulinemia as the outcome variable, short sleepers had significantly greater odds of having hyperinsulinemia (OR=1.76, 95% CI=1.07-2.92) (Table 8). However, after adjusting for age, sex, chronic condition, education and income, short sleep duration was not significantly linked to increased odds of hyperinsulinemia (OR=1.27, 95% CI=0.73-2.19). Similarly, short sleep

duration was not a significant predictor of hyperinsulinemia either in age- or in sex-stratified models. Table 9 presents the coefficients for all variables included in the models.

Table 8

Odds Ratios For Hyperinsulinemia in Short Sleepers (Bootstrapped Data, Fasting Subsample)

Regression Model	Model[d]	OR (95% CI)
Bivariate	**.001**	**1.76 (1.07-2.92)**
Multivariate[a]	**<.001**	1.27 (0.73-2.19)
Boys multivariate[b]	**<.001**	1.36 (0.73-2.54)
Girls multivariate[b]	**<.001**	1.21 (0.58-2.50)
Age 6-11 multivariate[c]	**.030**	1.63 (0.87-3.06)
Age 12-17 multivariate[c]	**<.001**	1.19 (0.62-2.30)

Note. **Bold=$p < .05$**

[a]Odds ratio adjusted for age, sex, chronic conditions, education and income.
[b]Odds ratio adjusted for age, chronic conditions, education and income.
[c]Odds ratio adjusted for sex, chronic conditions, education and income.
[d]Likelihood ratio test for significance of full models vs. constant-only models.

Table 9

Odds Ratios (95% CI) for All Variables Included in Multivariate Models Predicting Hyperinsulinemia (Bootstrapped Data, Fasting Subsample)

Predictor variable	Unstratified Model[a]	Sex-stratified Models[b]		Age-stratified Models[c]	
		Boys	Girls	6-11 years	12-17 years
Short sleeper	1.27 (0.73-2.19)	1.36 (0.73-2.54)	1.21 (0.58-2.50)	1.63 (0.87-3.06)	1.19 (0.62-2.30)
Adolescent age	**4.03 (2.02-8.04)**	**6.39 (2.16-18.89)**	**2.73 (1.14-6.57)**	-	-
Male sex	1.27 (0.89-1.82)	-	-	2.41 (0.93-6.28)	1.03 (0.58-1.83)
Chronic condition	0.59 (0.26-1.30)	0.71 (0.31-1.59)	0.54 (0.19-1.54)	0.71 (0.30-1.67)	0.55 (0.18-1.75)
Low education	0.70 (0.31-1.58)	0.47 (0.21-1.05)	0.83 (0.33-2.10)	1.18 (0.39-3.58)	0.59 (0.20-1.75)
Low income	1.45 (0.85-2.48)	**3.78 (1.14-12.47)**	0.84 (0.38-1.82)	0.65 (0.24-1.74)	2.09 (0.91-4.82)
Model[d]	**<.001**	**<.001**	**<.001**	**0.030**	**.013**

Note. **Bold=$p < .05$**

[a]Odds ratio adjusted for age, sex, chronic conditions, education and income.

[b]Odds ratio adjusted for age, chronic conditions, education and income.

[c]Odds ratio adjusted for sex, chronic conditions, education and income.

[d]Likelihood ratio test for significance of full models vs. constant-only models.

Associations Between Short Sleep Duration and Low HDL Cholesterol. Based on the CHMS dataset, the national prevalence of low HDL cholesterol in the Canadian population aged 6-17 was 24.7%. Comparisons of characteristics of participants with and without low HDL cholesterol are outlined in Table 10. Chi square analyses indicated that participants with low HDL were more likely to be aged 12-17 years, have lower household education and income levels, and were more likely to have a chronic condition compared to those without low HDL.

Table 10

Characteristics of Participants With and Without Low HDL (Bootstrapped Data, Fasting Subsample)

Characteristic (n=735)	Low HDL (n=160) % (SE)	No Low HDL (n=575) % (SE)	p^a
Sleep duration			.069
Inadequate	50.6 (6.9)	42.8 (1.9)	
Adequate	49.4 (6.9)	57.2 (1.9)	
Age			**<.001**
6-11 years	**32.2 (4.2)**	**55.0 (2.3)**	
12-17 years	**67.8 (4.2)**	**45.0 (2.3)**	
Sex			.936
Male	53.0 (4.3)	53.3 (2.5)	
Female	47.0 (4.3)	46.7 (2.5)	
Household education			**<.001**
Below post secondary	**30.5 (8.3)**	**16.9 (2.4)**	
Post secondary	**69.5 (8.3)**	**83.1 (2.4)**	
Household income			**.004**
Low	**32.7 (5.1)**	**22.1 (3.1)**	
Middle/high	**67.3 (5.1)**	**77.9 (3.1)**	
Chronic condition			**<.001**
Yes	**45.1 (8.0)**	**29.0 (3.3)**	
No	**54.9 (8.0)**	**71.0 (3.3)**	

Note. **Bold=$p < .05$.** $^aX^2$ test for differences in characteristics among those with and without low HDL. HDL=High density lipoprotein.

Short sleep duration was not a statistically significant predictor of low HDL in multivariate or stratified logistic regression models (Table 11). Table 12 presents the coefficients for all variables included in the models.

Table 11

Odds Ratios For Low HDL in Short Sleepers (Bootstrapped, Fasting Subsample)

Regression Model	Model[d]	OR (95% CI)
Bivariate	.069	n/a[e]
Multivariate[a]	**<.001**	1.06 (0.58-1.92)
Boys multivariate[b]	**<.001**	1.60 (0.74-3.47)
Girls multivariate[b]	**.048**	0.73 (0.37-1.42)
Age 6-11 multivariate[c]	**.014**	0.89 (0.43-1.85)
Age 12-17 multivariate[c]	**<.001**	1.22 (0.55-2.72)

Note. **Bold=$p < .05$.** HDL=High density lipoprotein.
[a]Odds ratio adjusted for age, sex, chronic conditions, education and income.
[b]Odds ratio adjusted for age, chronic conditions, education and income.
[c]Odds ratio adjusted for sex, chronic conditions, education and income.
[d]Likelihood ratio test for significance of full models vs. constant-only models.
[e]Odds ratio not displayed because overall model was not significant.

Table 12

Odds Ratios (95% CI) for All Variables Included in Multivariate Models Predicting Low HDL (Bootstrapped, Fasting Subsample)

Predictor variable	Unstratified Model[a]	Sex-stratified Models[b]		Age-stratified Models[c]	
		Boys	Girls	6-11 years	12-17 years
Short sleeper	1.06 (0.58-1.92)	1.60 (0.74-3.47)	0.73 (0.37-1.42)	0.89 (0.43-1.85)	1.22 (0.55-2.72)
Adolescent age	**2.52 (1.41-4.52)**	**4.37 (1.52-12.61)**	1.66 (0.70-3.91)	-	-
Male sex	1.05 (0.54-2.02)	-	-	2.21 (0.88-5.56)	0.62 (0.22-1.73)
Chronic condition	0.57 (0.30-1.09)	**0.47 (0.23-0.99)**	0.75 (0.30-1.86)	0.55 (0.22-1.37)	0.61 (0.33-1.13)
Low education	0.58 (0.30-1.12)	0.51 (0.13-1.97)	0.70 (0.24-2.05)	0.78 (0.23-2.59)	0.50 (0.20-1.27)
Low income	0.65 (0.39-1.06)	0.59 (0.22-1.54)	0.61 (0.20-1.84)	0.91 (0.40-2.06)	0.46 (0.20-1.05)
Model[d]	**<.001**	**<.001**	**.048**	**.014**	**<.001**

Note. **Bold=$p < .05$.** HDL=High density lipoprotein.

[a]Odds ratio adjusted for age, sex, chronic conditions, education and income.

[b]Odds ratio adjusted for age, chronic conditions, education and income.

[c]Odds ratio adjusted for sex, chronic conditions, education and income.

[d]Likelihood ratio test for significance of full models vs. constant-only models.

Associations Between Short Sleep and High Triglycerides. Based on the CHMS dataset, the national prevalence of high triglycerides in the Canadian population aged 6-17 was 23.8%. Comparisons of characteristics of participants with and without high triglycerides are outlined in Table 13. Chi square analyses indicated that participants with high triglycerides were more likely to fall into the 12-17 year old age category.

Table 13

Characteristics of Participants With and Without High Triglycerides (Bootstrapped Data, Fasting Subsample)

Characteristic (n=735)	High Triglycerides (n=171) % (SE)	Not High Triglycerides (n=564) % (SE)	p[a]
Sleep duration			.397
Inadequate	47.5 (4.1)	43.8 (2.9)	
Adequate	52.5 (4.1)	56.2 (2.9)	
Age			**.001**
6-11 years	**38.3 (3.5)**	**52.9 (1.6)**	
12-17 years	**61.7 (3.5)**	**47.1 (1.6)**	
Sex			.393
Male	50.4 (3.9)	54.1 (1.5)	
Female	49.6 (3.9)	45.9 (1.5)	
Household education			.263
Below post secondary	23.3 (6.9)	19.4 (3.3)	
Post secondary	76.7 (6.9)	80.6 (3.3)	
Household income			.058
Low	19.4 (3.9)	26.4 (3.4)	
Middle/high	80.6 (3.9)	73.6 (3.4)	
Chronic condition			.070
Yes	38.6 (5.4)	31.3 (3.8)	
No	61.4 (5.4)	68.7 (3.8)	

Note. **Bold=$p < .05$** [a]X^2 test for differences in characteristics among those with and without high triglycerides.

Short sleepers did not have increased odds of high triglycerides in multivariate models accounting for age, sex, chronic conditions, education and income (OR=0.99, 95% CI=0.64-1.53). Similarly, sex- and age-stratified models did not demonstrate any significant associations between sleep duration and high triglycerides (Table 14). Table 15 presents the coefficients for all variables included in the models.

Table 14

Odds Ratios For High Triglycerides in Short Sleepers (Bootstrapped, Fasting Subsample)

Regression Model	Model[d]	OR (95% CI)
Bivariate	.398	n/a[e]
Multivariate[a]	**.002**	0.99 (0.64-1.53)
Boys multivariate[b]	**<.001**	1.45 (0.85-2.47)
Girls multivariate[b]	.088	n/a[e]
Age 6-11 multivariate[c]	**.012**	1.16 (0.56-2.43)
Age 12-17 multivariate[c]	.112	n/a[e]

Note. **Bold=$p < .05$.**
[a]Odds ratio adjusted for age, sex, chronic conditions, education and income.
[b]Odds ratio adjusted for age, chronic conditions, education and income.
[c]Odds ratio adjusted for sex, chronic conditions, education and income.
[d]Likelihood ratio test for significance of full model vs. constant-only model.
[e]Odds ratio not displayed because overall model was not significant.

Table 15

Odds Ratios (95% CI) for All Variables Included in Multivariate Models Predicting High Triglycerides (Bootstrapped, Fasting Subsample)

Predictor variable	Unstratified Model[a]	Sex-stratified Models[b]		Age-stratified Models[c]	
		Boys	Girls	6-11 years	12-17 years
Short sleeper	0.99 (0.64-1.53)	1.45 (0.85-2.47)	n/a[e]	1.17 (0.56-2.43)	n/a[e]
Adolescent age	**1.78 (1.27-2.51)**	**2.15 (1.18-3.92)**	n/a[e]	-	-
Male sex	1.28 (0.83-1.98)	-	-	1.73 (0.90-3.31)	n/a[e]
Chronic condition	0.71 (0.43-1.17)	1.00 (0.46-2.19)	n/a[e]	**0.53 (0.33-0.85)**	n/a[e]
Low education	0.83 (0.35-1.98)	0.60 (0.16-2.28)	n/a[e]	0.56 (0.19-1.66)	n/a[e]
Low income	1.65 (0.83-3.29)	**2.67 (1.02-7.02)**	n/a[e]	0.99 (0.38-2.57)	n/a[e]
Model[d]	**.002**	**<.001**	.088	**.012**	.112

Note. **Bold=$p < .05$**

[a]Odds ratio adjusted for age, sex, chronic conditions, education and income.

[b]Odds ratio adjusted for age, chronic conditions, education and income.

[c]Odds ratio adjusted for sex, chronic conditions, education and income.

[d]Likelihood ratio test for significance of full models vs. constant-only models.

[e]Odds ratio not displayed because overall model was not significant.

In summary, short sleepers had higher odds of OWOB (analyses conducted using the full sample, n=1690) but not hyperinsulinemia, low HDL or high triglycerides (analyses conducted using the smaller fasting subsample, n=735). To investigate whether these results may have been influenced by the lower statistical power that arose from the smaller fasting sample size, logistic regression models using OWOB as the outcome variable were repeated using the fasting sample (Table 16). The odds of OWOB in short sleepers were similar when conducted with the fasting subsample, but as expected, confident intervals were wider (e.g., unstratified multivariate model, OR 1.58, 95% CI=1.00-2.48) vs. the full sample (OR 1.56, 95% CI=1.15-2.19).

Table 16

Comparison of Odds Ratios For Indicators of Cardio-Metabolic Disease in Short Sleepers (Bootstrapped, Fasting Subsample)

	OWOB		Hyperinsulinemia		Low HDL		High Triglycerides	
Model	OR (95% CI)	Model[d]	OR (95% CI)	Model[d]	OR (95% CI)	Model[d]	OR (95% CI)	Model[d]
Bivariate	**1.73 (1.15-2.60)**	**.001**	**1.76 (1.07-2.92)**	**.001**	n/a[e]	.069	n/a[e]	.398
Multivariate[a]	**1.58 (1.00-2.48)**	**<.001**	1.27 (0.73-2.19)	**<.001**	1.06 (0.58-1.92)	**<.001**	0.99 (0.64-1.53)	**.002**
Boys[b]	**1.93 (1.04-3.56)**	**<.001**	1.36 (0.73-2.54)	**<.001**	1.60 (0.74-3.47)	**<.001**	1.45 (0.85-2.47)	**<.001**
Girls[b]	n/a[e]	.110	1.21 (0.58-2.50)	**<.001**	0.73 (0.37-1.42)	**.048**	n/a[e]	.088
Ages 6-11[c]	n/a[e]	.269	1.63 (0.87-3.06)	**.030**	0.89 (0.43-1.85)	**.014**	1.17 (0.56-2.43)	**.012**
Ages 12-17[c]	1.90 (0.91-3.97)	**<.001**	1.19 (0.62-2.30)	**.013**	1.22 (0.55-2.73)	**<.001**	n/a[e]	.112

Note. **Bold=p < .05**. OWOB=Overweight or obese, HDL=High density lipoprotein.

[a]Odds ratios adjusted for age, sex, chronic conditions, education and income.

[b]Odds ratios adjusted for age, chronic conditions, education and income.

[c]Odds ratios adjusted for sex, chronic conditions, education and income.

[d]Likelihood ratio test for significance of full model vs. constant-only model.

[e]Odds ratio not displayed because overall model was not significant.

Objective 2: To identify determinants of short sleep duration.

Based on the full sample of the CHMS, the national prevalence of short sleep duration in the Canadian population aged 6-17 was 44.0%. Comparisons of characteristics of participants with and without short sleep duration are outlined in Table 17. Chi square analyses indicated characteristics linked with short sleep duration included being aged 12-17 years, female sex, and having a chronic condition.

Table 17

Characteristics of Participants With and Without Short Sleep Duration (Bootstrapped Data, Full Sample)

Characteristic (n=1690)	Short Sleep Duration (n=743) % (SE)	Not a Short Sleeper (n=947) % (SE)	p^a
Age			**<.001**
6-11 years	**37.0 (2.0)**	**59.9 (1.6)**	
12-17 years	**63.0 (2.0)**	**40.1 (1.6)**	
Sex			**.033**
Male	**50.1 (2.8)**	**55.3 (1.7)**	
Female	**49.9 (2.8)**	**44.7 (1.7)**	
Household education			.251
Below post secondary	18.9 (3.4)	21.2 (2.6)	
Post secondary	81.1 (3.4)	78.8 (2.6)	
Household income			.346
Low/lower middle	25.0 (2.9)	23.1 (3.2)	
Upper middle/high	75.0 (2.9)	76.9 (3.2)	
Chronic condition			**.028**
Yes	**33.2 (3.3)**	**28.3 (3.4)**	
No	**66.8 (3.3)**	**71.7 (3.4)**	

Note. **Bold=$p < .05$**. [a]X^2 test for differences in characteristics between sleep duration categories.

To further explore risk and protective factors associated with being a short sleeper, logistic regression analyses were performed using the full sample (n=1690). Using short sleep

duration as the outcome variable and age, sex, chronic conditions, education, and income as predictor variables, five logistic regression models were developed (Table 18).

In the unstratified multivariate model, age was the strongest predictor of short sleep duration. Compared to children aged 6-11, teens aged 12-17 had greater odds of not meeting sleep guidelines (OR 2.62, 95% CI=1.88-3.66). Sex, chronic conditions, education and income were not significant predictors of short sleep duration. In sex-stratified analyses, age was a predictor of short sleep duration both in girls (OR=3.25, 95% CI=2.16-4.90) and in boys (OR=2.15, 95% CI=1.30-3.55). The only other significant predictor of short sleep duration was low income, which, in children aged 6-11 only, was associated with lower odds of short sleep duration (OR=0.60, 95% CI=0.40-0.91).

Table 18

Predictors of Short Sleep Duration (Full Sample, Bootstrapped Data)

Predictor variable	Unstratified model[a]	Sex-stratified models[b]		Age-stratified models[c]	
		Boys	Girls	6-11 years	12-17 years
Adolescent age	**2.62 (1.88-3.66)**	**2.15 (1.30-3.55)**	**3.25 (2.16-4.90)**	-	-
Male sex	1.30 (0.90-1.88)	-	-	1.05 (0.72-1.53)	1.64 (0.97-2.77)
Chronic condition	0.83 (0.58-1.19)	0.78 (0.49-1.23)	0.88 (0.48-1.61)	0.94 (0.56-1.56)	0.74 (0.41-1.32)
Low education	1.34 (0.92-1.94)	1.70 (1.04-2.76)	1.03 (0.61-1.74)	1.23 (0.79-1.91)	1.40 (0.74-2.66)
Low income	0.81 (0.63-1.05)	0.99 (0.70-1.41)	0.67 (0.37-1.25)	**0.60 (0.40-0.91)**	1.15 (0.71-1.87)
Model[d]	**<.001**	**<.001**	**<.001**	**.018**	**.004**

Note. **Bold=$p < .05$**.

[a]Odds ratios adjusted for age, sex, chronic conditions, education and income.

[b]Odds ratios adjusted for age, chronic conditions, education and income.

[c]Odds ratios adjusted for sex, chronic conditions, education and income.

[d]Likelihood ratio test for significance of full model vs. constant-only model.

Chapter 4: Discussion

Hypothesis 1: Compared to longer sleepers, short sleepers exhibit increased odds of having indicators of cardio-metabolic disease.

Odds of Overweight/Obesity in Short Sleepers. The first objective of this study was to explore associations among short sleep duration and indicators of cardio-metabolic disease, including a) overweight/obesity (OWOB), b) hyperinsulinemia, c) low HDL cholesterol, and d) high triglycerides in a nationally representative population Canadian children and adolescents. Age- and sex-stratified analyses indicated that Hypothesis 1a was supported for boys and adolescents only. After adjusting for the effects of age, chronic conditions, education and income on OWOB, boys with short sleep duration had 1.85 times the odds of OWOB compared to boys who met sleep guidelines; the relationship was not significant in girls (OR=1.27, 95% CI=0.63-2.57). Similarly, after adjusting for sex, chronic conditions, education and income, short sleepers aged 12-17 had increased odds of OWOB (OR=1.77, 95% CI=1.05-2.99); the relationship was not significant in children aged 6-11 years (OR=1.37, 95% CI=0.84-2.22).

As described in Chapter 1, short sleep duration may contribute to OWOB by affecting both energy intake and energy expenditure. Increased energy intake consequent to short sleep duration may occur via increased appetite as well as through increases in non-appetite regulated eating behaviour (Chaput, Klingenberg, & Sjodin, 2010). Short sleep duration is also associated with decreases in voluntary physical activity (Schmid et al., 2009), and may also be linked with reductions in non-voluntary energy expenditure via basal metabolic rate, non-exercise activity thermogenesis and the thermic effect of food (Taheri, 2006).

The finding that boys were more vulnerable to the obesogenic effect of sleep loss is consistent with data from other published literature (Chaput et al., 2006; Eisenmann et al., 2006;

Gibson et al., 2004; Knutson, 2005; Sekine et al., 2002). Several theories could explain why boys may be more vulnerable to the obesogenic effects of short sleep duration. As explained in Chapter 1, from an evolutionary perspective, girls may be more physiologically resilient to environmental stressors such as sleep loss (Eisenmann et al., 2006; Wells, 2000). Another possibility is that females reach their biological maturity earlier than males and thus their circadian rhythms may be attuned to sleep patterns that more closely match their social environment. For example, the progression from childhood to adolescence is characterized by an inclination towards 'eveningness', or a preference to be awake later at night and to sleep in later in the morning (Crowley, Acebo, & Carskadon, 2007). As adolescents proceed into adulthood, this eveningness preference tends to shift back to an increased tolerance for wakefulness earlier in the morning and a preference for earlier bedtimes (Crowley et al., 2007). In an Italian study, Tonetti, Fabbri and Natale (2008) found that adolescent females reached their peak in eveningness circadian rhythms at an earlier age compared to males. Sex differences in sleep architecture may also account for this effect. Females spend more time in slow wave sleep compared to males (Briere et al., 2003). Because slow wave sleep plays an important role in human metabolism (Spiegel et al., 2009), females may require a higher threshold of sleep loss in order to experience a negative health impact (Eisenmann et al., 2006).

Age-stratified analyses demonstrated that the risk of OWOB in short sleepers was only significant in adolescents (OR=1.77, 95% CI=1.05-2.99) but not in children (OR=1.37, 95% CI=0.84-2.22). No previous studies that calculated similar estimates could be located. A potential explanation for this finding is that adolescents who report not getting enough sleep had also been short sleepers since childhood, and subsequently had been exposed to the obesogenic effects of insufficient sleep for a longer time period. This theory is supported by longitudinal

studies on children and adolescents that demonstrated that short sleepers are more likely to gain weight over time (Reilly et al., 2005; Snell, Adam, & Duncan, 2007; Agras, Hammer, McNicholas, & Kraemer, 2004).

Stratification of analyses also resulted in smaller sample sizes and, as expected, corresponding wider confidence intervals. While short sleepers in all age and sex strata had greater odds of OWOB compared to longer sleepers, the confidence intervals overlapped the null (1.0) for girls and children. Although some schools of thought suggest that confidence intervals that overlap the null should not automatically be interpreted to indicate that no association exists (Szklo & Nieto, 2007), a conservative approach was taken in this study and these results were interpreted as sleep loss being linked to OWOB only in boys and adolescents. However, because of the limitations associated with smaller stratified sample sizes (Szklo & Nieto, 2007), this inference should be considered with caution.

Odds of Hyperinsulinemia, Low HDL, and High Triglycerides in Short Sleepers. The results of logistic regression analyses conducted on the fasting subsample did not support the hypotheses that short sleepers had greater odds of hyperinsulinemia, low HDL, or high triglycerides compared to longer sleepers. Although other studies that examined associations among short sleep duration and cardio-metabolic indicators other than OWOB in children and adolescents are sparse, a few studies have demonstrated that short sleep is linked with adverse cardio-metabolic health. In a population of obese children, Flint et al. (2007) found that short sleepers had higher fasting insulin levels. Martinez-Gomez et al. (2011) determined that adolescents with short sleep had higher C-reactive protein even after adjusting for obesity. Hitze et al. (2009) found that short sleep duration was linked to lower adiponectin in boys, and higher leptin, insulin, and degree of insulin resistance in girls.

Several explanations could account for this finding of short sleepers having increased odds of OWOB but not of other indicators of cardio-metabolic disease. First, children and adolescents may need a longer exposure time to short sleep duration to develop cardio-metabolic risk factors other than OWOB. Longitudinal studies examining the long-term effect of short sleep duration on indicators of cardio-metabolic health other than OWOB are needed to test this hypothesis.

Another potential reason why short sleepers had greater odds of OWOB only is that, in children and adolescents, cardio-metabolic health might deteriorate only as a future consequence of OWOB and not directly because of sleep loss. For example, the increased adiposity associated with OWOB is thought to result in deteriorations in other indicators of cardio-metabolic health (Kahn & Flier, 2000). Conversely, evidence also suggests that sleep loss, independently of OWOB, plays a causal role in the deterioration of cardio-metabolic health (Flint et al., 2007; Spiegel et al., 1999). The findings from this study did not support the hypothesis that short sleep duration independently increases the risk of these indicators of cardio-metabolic disease in children and adolescents.

Another potential reason that short sleepers did not have greater odds of hyperinsulinemia is that alternative measures could have provided a superior assessment of the deterioration in carbohydrate metabolism that occur secondary to sleep loss. Sleep loss affects both insulin secretion (e.g., beta cell function) and insulin resistance in numerous peripheral tissues (Spiegel et al., 2009). This study measured serum hyperinsulinemia, which is an appropriate measure to evaluate insulin resistance (Kim & Reaven, 2008). However, it is possible that a measure that evaluates both insulin resistance and beta cell function (e.g., the homeostatic model assessment

of insulin resistance) would have been more appropriate given that sleep loss affects both of these aspects of carbohydrate metabolism (Spiegel et al., 2009).

Type II error is an additional possible explanation for why this study found that short sleepers had greater odds of OWOB but not other indicators of cardio-metabolic disease. Because the fasting subsample was smaller than the full sample, it was expected that the odds ratios for these analyses would have wider confidence intervals (Szklo & Nieto, 2007). In several cases, short sleepers had greater odds of having hyperinsulinemia, low HDL, and high triglycerides, but the confidence intervals overlapped 1.0. Even though a conservative approach was taken in this study, there are differing opinions on the interpretation of confidence intervals that overlap 1.0 (Szklo & Nieto, 2007). Because of the concerns with sample size, the inference that hyperinsulinemia does not arise in children and adolescents as a consequence of short sleep duration should be considered with caution.

To further examine the effect of sample size on statistical significance, logistic regression models predicting OWOB in short sleepers were conducted on both the full and fasting samples. In a comparison of estimates obtained from the full and fasting datasets, the odds ratios were similar, but as expected, the confidence intervals were consistently wider for the fasting dataset. For example, in the unstratified analyses the OR and 95% CI for OWOB in short sleepers was 1.56 (95% CI=1.15-2.19) in the full sample versus 1.58 (95% CI=1.01-2.48) in the fasting sample. In a few cases, confidence intervals became wide enough to result in a statistically non-significant estimate. In full sample analyses, 12-17 year olds with short sleep had an odds ratio of 1.77 (95% CI 1.05-2.99) for OWOB. When this same analysis was conducted on the fasting sample, the OR for OWOB was 1.90, but the 95% CI overlapped 1.0 (0.91-3.97) and thus was not statistically significant. In summary, the limitations of sample size should be considered

when interpreting the statistical significance of odds ratios from corresponding confidence intervals.

Hypothesis 2: Factors associated with greater odds of short sleep duration include adolescent age, chronic conditions, low household income, and low household education.

The second objective of this research was to identify risk and protective factors associated with short sleep duration. Age was the strongest predictor of short sleep duration, with teens aged 12-17 having greater odds of not getting enough sleep compared to children aged 6-11. These findings are consistent with other published research that also found that age explained most of the variance in sleep duration, with older children more likely to experience sleep loss (Hitze et al., 2009; Iglowstein et al., 2003). Adolescents are less likely to meet sleep requirements because they restrict their sleep to meet social demands, and this reduction in sleeping hours surpasses the slight decline in sleep requirement that occurs in adolescence (Iglowstein et al., 2003).

The only other significant predictor variable was low income, which was linked with lower odds of short sleep duration in 6-11 year olds only. This finding was unexpected because previous research indicated that sleep duration is inversely related to socioeconomic status in Canadian children and adolescents (Jarrin et al., 2009). It is uncertain why the results of these studies contradict each other. A possible explanation for this finding is that children of higher income families are more likely to have opportunities to participate in organized activities that encroach on sleep time. Another possibility is that children of higher income families could have more technology available to them. Use of electronic devices such as cell phones, televisions and computers may be linked to sleep debt (Taheri, 2006). Low income was not a significant predictor of sleep duration in adolescents. Possibly, this finding could be explained by the

increased independence associated with adolescence. Alternatively, it is possible that low-income teens might 'catch up' to the sleep restriction of their higher income peers by becoming involved in other activities that lead to sleep loss such as part-time employment.

Limitations

Validity. The potential for misclassification exists because data for several variables were self-reported. While it had been hoped that the CHMS activity monitor data could be used as an objective measure of sleep duration, an excess of missing data did not allow for this. Although the body of epidemiological evidence on sleep and obesity has been criticized for dependency on self-reported data (Bliwise & Young, 2007; Taheri & Thomas, 2008), comparisons of sleep duration as assessed by questionnaire and actigraphy were not significantly different in adolescents (Wolfson et al., 2003). However, parents may overestimate sleep length in younger children (Werner, Molinari, Guyer, & Jenni, 2008). Although it is unknown whether the magnitude of overestimation differs among parents of OWOB and non-OWOB children, no difference was observed among sex, age, or socioeconomic strata (Werner et al., 2008). Misclassification also may have occurred because it is uncertain how closely the respondents' perception of an average night's sleep length corresponds to actual sleep length. Other self-reported variables that are likely to have been affected by misclassification include the socioeconomic status variables education and income.

Selection biases also may have influenced the results. Respondents with missing data on any of the study variables were excluded from the analyses. Participants with missing data were more likely to be short sleepers and to have hyperinsulinemia. The exclusion of these participants from the sample may have resulted in an underestimation of the association between sleep duration and indicators of cardio-metabolic disease. Biases may have also resulted from

complete non-response. Although the survey sample was weighted to adjust for complete non-response, weighting only ensured the sample was representative for age, sex, and geographic location (Giroux, 2007). Other factors that influence participation in research would not have been adjusted for, such as income, education level, and health status. For example, because the CHMS required a thorough clinical examination including fitness testing, people with chronic conditions that limit physical activity may have opted out of the survey. Consequently, because the CHMS sample was likely healthier than the actual Canadian population, the analyses conducted in this study may have underestimated the association between sleep duration and indicators of cardio-metabolic disease.

Determination of Causation. An important limitation of this study is that determining causation is not possible when using a cross-sectional, observational study design. One of the criteria for causation is that exposure to the causal factor must precede the outcome (Szklo & Nieto, 2007). In this cross-sectional study, it is not known whether short sleep duration preceded the onset of OWOB. As such, the directionality of the observed association between short sleep duration and OWOB is uncertain. Indeed, evidence suggests that the relationship between cardio-metabolic disease and sleep loss is bi-directional (Magee et al., 2010). However, controlled experimental studies demonstrating that sleep restriction can induce deteriorations in cardio-metabolic health in adults (Knutson et al., 2007) support the possibility that short sleep duration is an etiological factor in the development of OWOB in Canadian children and adolescents.

Another limitation associated with observational study designs is that confounding variables, and not short sleep duration, could have been causal factors in the sleep duration/OWOB relationship observed in this study. For example, it is possible that short

sleepers are also more likely to engage in unhealthy behaviours linked to OWOB. Although multivariate models accounted for as many confounders as possible, it is likely that some residual confounding occurred. Similarly, models that aimed to identify predictors of short sleep duration could have been affected by residual confounding.

Despite the limitations associated with cross-sectional, population-based studies, there are several advantages to this design. First, given the existing scientific evidence linking harm to sleep loss, it would be unethical to intentionally subject a pediatric population to sleep restriction. The observational study design provides a way of measuring these associations ethically. Secondly, a goal of this study was to provide nationally representative estimates. Although controlled, experimental trials would provide strong evidence, the cost would be high because the required sample size to produce national estimates is large and dispersed geographically. The significantly lower expense of an observational study is much more feasible. Finally, although it is not possible to demonstrate that sleep loss is an etiological factor in the development of cardio-metabolic disease with this study, the results of double-blinded experimental studies on adults provide strong evidence that sleep loss can cause deteriorations in indicators of cardio-metabolic health. Moreover, the findings in this observational study support the findings from these controlled experimental studies. Taken together, this study and the corresponding body of research provide evidence that short sleep duration is a causal factor in the epidemic of OWOB in Canadian children and adolescents.

Disadvantages of Using the Odds Ratio. Another limitation of this study is the use of the odds ratio. Although odds ratios can be considered a measure of effect size, they are often incorrectly interpreted to be equivalent to the relative risk (Davies, Crombie, & Tavakoli, 1998). When the outcome of interest is rare, this is not problematic because the relative risk and odds

ratio are similar in value (Davies et al., 1998). However, when the outcome of interest is common (e.g., prevalence of more than 10%), then interpreting an odds ratio as a relative risk inflates the effect size of an association (Davies et al., 1998). This presents a problem for the present study because the prevalence of OWOB in the population was 25.5%. To address this issue, the relative risk for OWOB in short sleepers versus adequate sleepers was calculated and compared with the bivariate odds ratio. While the unadjusted odds ratio for OWOB was 1.59, the relative risk was calculated at 1.41. If the odds ratio had been interpreted as being equivalent to the relative risk (e.g., by stating that short sleepers are 59% more likely to be overweight or obese compared to longer sleepers), this would have inflated the actual risk of OWOB in short sleepers by 18%.

Despite these disadvantages, there are several advantages with using the odds ratio. First, a main goal of this research was to provide comparisons with other Canadian and international sleep duration/cardio-metabolic disease studies. Because the majority of other published studies used the odds ratio as a measure of association, it was advantageous to utilize this method to produce easily comparable results. Secondly, logistic regression models and corresponding odds ratios allow for the use of a dichotomous outcome variable. Dichotomous outcome variables were preferred because the negative health impact of indicators of cardio-metabolic disease may only occur at a certain threshold. For example, a high body mass index (BMI) is linked with health problems in the OWOB range, and not uniformly as BMI increases (Cole et al., 2000).

Alternative Statistical Approaches. Logistic regression was chosen as a measure of association for this study because of its ability to handle binary outcome variables, as well as to adjust for linear and/or categorical confounders, and also to allow for comparisons with previously published research. However, other methods could have been utilized, including chi-

square analyses, linear regression, and ordinal logistic regression. Although chi-square analyses were used to provide a crude assessment of association, multivariate analysis was essential in order to account for confounding variables. Linear regression and analysis of variance would have allowed for the use of continuous variables, which in turn could have provided a finer level of detail regarding the relationship between sleep duration and indicators of cardio-metabolic disease. However, because the relationship between sleep duration and body mass index is not linear, this would have violated one of the assumptions of linear regression. Data transformations may have been able to satisfy this assumption, but these transformations also increase the complexity of interpreting the resulting coefficients (Grissom, 2000). Logistic regression provides the advantage of not requiring this assumption.

The ability to use dichotomous outcome variables was also advantageous because indicators of cardio-metabolic health are only linked with negative health implications outside of 'normal' ranges (Cole et al., 2000). For example, because HDL cholesterol is linked with an increased risk of heart disease in the 'low' range (American Heart Association, 2012), it is advantageous to examine short sleep duration's association with low HDL, rather than with the HDL level expressed as a continuous variable. Although ordinal logistic regression would have allowed the use of additional categories linked to health risk (e.g., underweight, normal, overweight, obese), a larger sample size would have been required to obtain stable estimates.

Generalizability. Although the CHMS sampling strategy was designed to gain a nationally representative sample of the Canadian population, several populations were not included in the sample, including people living in institutions or on reserves, and full time members of the military. Institutions excluded from the CHMS include collective dwellings that provide custody or medical care such as boarding schools, hospitals, and group homes for youth.

The CHMS represents approximately 96% of the Canadian population (Tremblay, Wolfson, & Gorber, 2007), indicating high generalizability outside of the aforementioned excluded groups.

Future Research

The findings from this study illuminate several areas for further investigation. To build on the findings from this cross-sectional study, a longitudinal study could evaluate whether over time, short sleep duration predicts the development of OWOB and other indicators of cardio-metabolic disease. As well, increased understanding of the mechanisms that lead to OWOB in short sleepers is needed. For example, further study of the behavioural and physiological consequences of sleep loss and their corresponding connection to indicators of cardio-metabolic disease is needed. Further study into the mechanisms underlying sex differences in the sleep duration-OWOB relationship is also needed. Finally, the development, implementation and evaluation of health promotion strategies that facilitate healthy sleep habits in children and adolescents should be undertaken.

Conclusion

In summary, inadequate sleep duration was linked with greater odds of OWOB in boys and adolescents. Short sleepers did not have increased odds of other cardio-metabolic disease indicators, including hyperinsulinemia, low HDL, and high triglycerides. Age predicted inadequate sleep duration in both sexes and all age groups. Public health strategies that emphasize healthy sleeping habits in children and adolescents should be trialed and assessed for their affect on OWOB, especially in boys. Finally, because the progression from childhood to adolescence is characterized by a greater risk of inadequate sleep, health promotion strategies that aim to address this issue should focus on addressing the factors that affect the sleep duration of adolescents specifically.

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Appendix A: Methodological Details - Canadian Health Measures Survey

Unless otherwise specified, all methodological detail for the CHMS presented here was drawn from Statistics Canada documentation (Statistics Canada, 2010b).

Sampling Strategy

All Canadians between the ages of 6-79 were potentially part of the CHMS sample except for full-time members of the military, residents of institutions, and people living on Indian reserves or crown lands. The CHMS is representative of 96% of the Canadian population (Tremblay et al., 2007). To achieve a representative sample, 10 age-gender groups with a minimum of 500 participants per group were developed, making a total of 5,000 participants. For the clinical exam, 257 potential clinic sites across Canada were established. Of these locations, 15 sites were selected using a systematic sampling method, with probability proportional to the population of the region. Initial contact was made through a letter mailed to households requesting their participation in the study.

Response Rate

From 8,772 households contacted, 69.6% provided consent to participate. Out of the households that agreed to participate, 88.3% responded to the household survey, and 84.9% of survey respondents proceeded to complete the clinical exam. The overall response rate was 51.7% (Giroux, 2007). Weighting was used to account for non-response.

Weighting

The CHMS dataset included a weighting variable in order to calculate nationally representative estimates. Each person in the sample was assigned a weight, or number of other Canadians that he or she represented. Use of these weights in SPSS presents a problem because SPSS calculates weighted estimates based on the population size, and not the sample size.

Because estimates are erroneously based on the much larger population size, SPSS will incorrectly indicate that calculated estimates are significant (Hahs-Vaughn, 2005). To avoid this problem, 'normalized weights' were created by dividing the weighting variable by the mean of the weights (Hahs-Vaughn, 2005).

Bootstrap Method

Because the CHMS sampling design involved clustering and stratification, the calculated sample variance is likely to underestimate the actual population variance. For example, clinical exams were conducted at only 15 sites across Canada, and respondents within each site are likely to be more similar to each other than to respondents from other locations. In order to account for this design effect, the survey bootstrap method can be used to better estimate the variance in the population (Phillips, 2004). The bootstrap method is a resampling procedure in which many subsamples are created from the original sample with replacement. Bootstrap weights are created to represent the probability of selection into the subsample (Phillips, 2004). Statistics Canada provides 500 bootstrap weight variables for the CHMS which were used in the program WesVar 5.1. The desired estimates are then recalculated using each new subsample, and the variance between the estimates provides a more accurate assessment of variance.

Covariates

Age. The CHMS sample population included participants aged 6-79 years. As the goal of this research is to examine the impact of sleep duration on cardio-metabolic risk in children and adolescents, the population selected for this purpose was 6-17. Adolescents aged 18 and 19 were excluded because many variables of interest were only available for ages 6-17 (i.e., Cole's BMI classification, HWMDCOL).

Age was collected twice in the CHMS: once during the household questionnaire (DHH_AGE) and again at the time of the clinical exam (CLC_AGE). Because of a time delay between the two components, age could change between the questionnaire and clinical exam. Thus, for accuracy, it is necessary to use the clinical exam age when examining clinical exam variables (i.e. blood tests), and questionnaire age when utilizing questionnaire variables (i.e. sleep duration). In the situation where both the household questionnaire and clinical exam data are used, Statistics Canada suggests using the age variable for the module containing the most important variable of interest. Because the outcome variables for this research are all based on the clinical exam portion, the age variable selected was age at the time of the clinical exam (CLC_AGE).

Chronic conditions. As described previously, chronic illnesses are related to sleep disturbances (Nieto et al., 2006; Smith & Haythornthwaite, 2004; Wulff et al., 2010). During the household interview, participants were asked if they had ever been diagnosed with various chronic conditions. From the responses to these questions, the variable CCCF1 was derived from the answers to these questions; respondents were coded as "has at least one chronic condition" or "has no chronic conditions". Constituent questions and frequencies are listed in Table 19.

Table 19

Chronic Conditions Frequencies

Survey Question		Age range	Full Sample	Fasting Subsample
CCC_11	Do you have asthma?	all	11.2%	12.5%
CCC_21	Do you have fibromyalgia?	12+	suppressed	suppressed
CCC_22	Do you have arthritis or rheumatism, excluding fibromyalgia?	12+	suppressed	suppressed
CCC_24	Do you have back problems, excluding fibromyalgia and arthritis?	12+	4.2%	3.1%
CCC_31	Do you have high blood pressure?	12+	suppressed	suppressed
CCC_34	Have you ever been told by a health professional that your blood cholesterol was too high?	12+	suppressed	suppressed
CCC_41	Do you have chronic bronchitis?	all	suppressed	suppressed
CCC_51	Do you have diabetes?	all	suppressed	suppressed
CCC_61	Do you have heart disease?	all	suppressed	suppressed
CCC_71	Do you have cancer?	all	suppressed	suppressed
CCC_81	Do you suffer from the effects of a stroke?	12+	suppressed	suppressed
CCC_82	Do you have a thyroid condition?	12+	suppressed	suppressed
CCC_83	Do you have a mood disorder such as depression, bipolar disorder, mania or dysthymia?	all	1.6%	2.5%
CCC_84	Do you have a learning disability?	all	8.1%	8.3%
CCC_91	Do you have an eating disorder such as anorexia or bulimia?	all	suppressed	suppressed
CCC_92	Do you suffer from kidney dysfunction or disease?	12+	suppressed	suppressed
CCC_93	Do you have liver disease or gallbladder problems?	12+	suppressed	suppressed
CCC_95	Do you have hepatitis?	12+	suppressed	suppressed
CCC_101	Do you have any other long-term physical or mental health condition?	all	13.1%	14.6%

Note. Suppressed data are not displayed due to Statistics Canada minimum cell size requirements.