THE HEALTH IMPACT OF SEDENTARY BEHAVIOUR IN CHILDREN AND YOUTH

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Abstract

Emerging evidence suggests that sedentary behaviour is independently associated with cardiometabolic disease risk in school-aged children and youth. This thesis includes 4 related studies in the pursuit of 2 objectives: 1) To determine the cross-sectional association of sedentary time, interruptions in sedentary time, sedentary bout length, and total movement variability with markers of cardiometabolic disease risk among children and youth, and 2) To examine the impact of 1-day of prolonged sedentary behaviour, with and without interruptions or structured physical activity, on markers of cardiometabolic disease risk, hunger, food intake and spontaneous physical activity levels in children and youth. In Study 1, we found that interruptions in sedentary time and short bouts of sedentary time were beneficially associated with clustered cardiometabolic disease risk in boys and girls aged 8-11 years, independent of total sedentary time, moderate-and-vigorous physical activity (MVPA), and other confounders (all $p<0.05$), while the opposite was true for screen based sedentary behaviours. In Study 2, we found that movement variability (minute-to-minute changes in movement intensity) was negatively associated with clustered cardiometabolic disease risk and systolic blood pressure independent of MVPA, sedentary time and other covariates in a representative sample of American children and youth aged 12-17 years (all $p<0.05$). In Studies 3 and 4, we found that prolonged sitting, with or without interruptions and structured MVPA did not result in acute changes in markers of cardiometabolic disease risk, nor subsequent *ad libitum* food intake or physical activity levels in healthy children aged 10-14 years (all $p \geq 0.05$). Taken together, the studies that make up this thesis suggest that optimal levels of cardiometabolic disease risk are most likely to be seen in children who limit their time engaging in screen-based sedentary behaviours, who frequently interrupt their sedentary time, and who have high levels of variability in their movement behaviours.

Contributions

The work in this thesis is my own, and I take full responsibility for its contents. Ethics applications were required for Studies 1, 3 and 4, and details of ethics approval are provided in Appendix A. A list of co-authors from the studies that make up this thesis can be found below. Details of the contributions of individual authors (including myself) to specific manuscripts can be found at the start of Chapters 2-6. At the time of final submission, 4 manuscripts in this thesis have been accepted for publication in the *British Journal of Nutrition,* the *Canadian Journal of Diabetes, Metabolism* and *PLOS ONE*. Both *Metabolism* and *CJD* are Elsevier journals, and therefore do not require a copyright form to be included in this thesis (See Appendix B). As the author I retain the copyright for manuscripts published in *BJN,* and *PLOS ONE* uses a Creative Commons Attribution license, therefore copyright forms are not required for these manuscripts either.

Co-authors of works included in this thesis:

Mark Tremblay (Chapters 2, 3, 4, 5 & 6)

Jean-Philippe Chaput (Chapters 2, 3, 5 & 6)

Gary Goldfield (Chapters 5 & 6)

Rachel Colley (Chapters 5 & 6)

Éric Doucet (Chapters 5 & 6)

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Mélanie Henderson (Chapter 3)

Marie-Ève Mathieu (Chapter 3)

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Valerie Carson (Chapter 4)

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The HALO Research Group (and friends)

While I take full responsibility for the works that make up this thesis, none of the studies that follow would have been possible without the help of many friends and colleagues. I ask that the reader bear with me, as this section is one of the few opportunities that I will have to formally acknowledge and thank those individuals.

I would like to begin by thanking the many participants (and their families) who took part in the studies that make up this thesis. They volunteered a great deal of time, energy, and even blood, and without them none of these studies would have been possible. Not only that, but they helped make the long hours of data collection enjoyable, for which I am extremely grateful.

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However, after just a few months working in the Healthy Active Living and Obesity Research Group that he oversees, my decision to pursue a PhD was both easy and obvious. Mark has been fiercely supportive of myself and my labmates, and has consistently provided me with rapid, detailed and (when necessary) blunt feedback throughout my PhD. I don't know that many graduate students are excited to go to work in the morning, but thanks to Mark (and the fantastic group he has assembled), I've always been able to look forward to work in "The Hub". Given the experience I have had working with Mark and the rest of the HALO team, I would also like to thank Michelle Lafrance, Doug Willms, and my uncle Patrick Flanagan for directly and/or indirectly sending me in this direction.

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Although not officially part of my thesis committee, aside from Mark no one has had more impact on my PhD research than Dr Jean-Philippe Chaput. It was JP's Young Investigator Award that funded Studies 3 and 4 of this thesis, and it was his colleagues in Quebec that gave us access to the data used in Study 1. He has been an excellent colleague throughout, providing rapid and helpful feedback and support, and including me on a wide range of side projects. His

productivity and work/life balance have also served as a model for myself and the other graduate students in the HALO lab.

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In addition to the aforementioned colleagues, I would also like to thank all of the collaborators who contributed to the manuscripts included in this thesis, as well as the side projects that I have been involved with over the years. They have all been extremely giving with their time, helpful in their feedback, and patient in responding to my questions (Drs Rachel Colley and Val Carson were especially helpful in this regard).

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job than Helene Sinclair, and it's been a pleasure to work with her these last few years. Additional thanks also go to Niko Tzakis, Mike Borghese, Dr Zach Ferraro, Richard Larouche, Joel Barnes, Charles Boyer, and Allana Leblanc for their help with data collection and to Dr Kristi Adamo for always taking time from her busy schedule to help me navigate the research process. Thanks also to former HALOite Dr Meghann Lloyd for getting me involved in the CAPL study early in my time at HALO, and for Dr Pat Longmuir for keeping me involved since joining the group.

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I would be remiss if I did not also thank and acknowledge the funding agencies that have made my thesis possible. My PhD has been funded by Doctoral Research Awards from the Canadian Institutes of Health Research and the Canadian Diabetes Association, as well as an Excellence Scholarship from the University of Ottawa. Studies 3 and 4 in this thesis were also funded by a Young Investigator Award from the CHEO Research Institute to Dr JP Chaput.

Last, but certainly not least, I would like to thank my family for all the love and support that they have provided throughout graduate school, and life in general. Mom, Dad and my sister Katie have always been incredibly supportive, with everything from hockey to juggling to graduate studies, and I am very proud to be a member of their family. No one has contributed more to this

PhD than my wife Daun, who I met shortly after entering graduate school in late 2006. Thanks to her (and her alone) I've been able to maintain a sustainable level of work/life balance throughout my PhD. She has made my PhD (and more importantly, life) a wonderful experience, and I can't thank her enough for her love and support during the past 4 years (and the 2 before that). I would also like to thank Daun's parents and the rest of the Lynch/Newland/Montgomery clan for welcoming me into their family, which has added so much to our time in Ottawa. Special thanks to Daun's stepfather, Dr Hugh Montgomery, for lending the famous wheelchair that was used to transport participants to and from the washroom in Studies 3 and 4.

Prelude to Thesis

While the health importance of physical activity has been accepted for decades, recent evidence suggests that sedentary behaviour (e.g. sitting, and activities done while sitting) may be a unique and deleterious risk factor for chronic disease, independent of physical activity and other established risk factors. However, while numerous reports suggest a link between sedentary behaviour and health in the pediatric age group, several important questions remain unanswered. The following thesis attempts to advance our understanding of the relationship among sedentary behaviour, physical activity and health in the pediatric age group through the use of literature reviews, cross-sectional analyses of large datasets, and a lab-based randomized crossover study.

In Chapter 1 I offer an overview of the rationale, objectives and hypotheses of the current thesis. In Chapter 2 I provide a comprehensive overview of research into the health impact of sedentary behaviour in the pediatric age group. Building on the work presented in Chapter 2, Chapter 3 examines the association of total sedentary time, sedentary bout length, and breaks in sedentary time with markers of cardiometabolic disease risk in large cohort of children with a family history of obesity. Chapter 4 examines similar relationships for a novel characteristic of human movement; total movement variability. Chapters 5 and 6 examine the acute impact of prolonged sitting on markers of cardiometabolic disease risk, physical activity and energy intake. Finally, Chapter 7 discusses the implications of the findings laid out in Chapters 3-6, as well as opportunities for future research. Related studies that provide context for the findings presented in this thesis, as well as supporting documentation, can be found in the Appendices. It is my hope that the research included in this thesis will improve our understanding of the health impact of sedentary behaviour, and aid in the prevention of chronic disease among children and youth.

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Chapter 1 - Thesis Introduction

While the pediatric health benefits of physical activity are well-established (1–3), emerging evidence suggests that sedentary behaviour (activities that involve sitting or reclining while expending ≤ 1.5 metabolic equivalents(4)) may be independently associated with *increased* health risk among children and youth (5–12)**.**

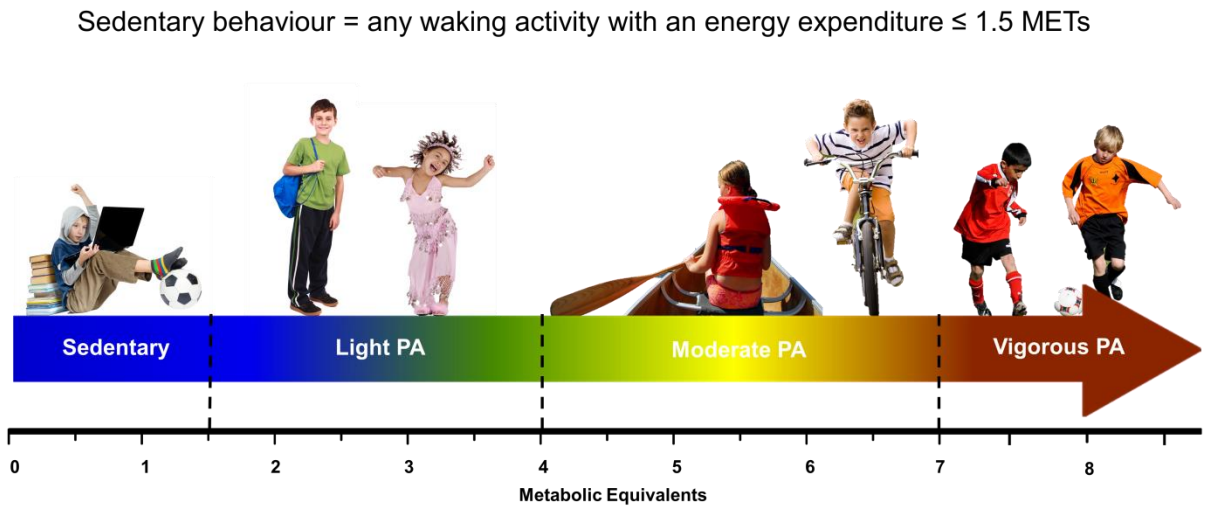

Figure 1.1 Sedentary behaviour and physical activity according to energy expenditure.

Despite a growing body of evidence suggesting that sedentary behaviour is associated with risk factors for cardiovascular disease and diabetes (hereafter referred to as cardiometabolic risk) in the pediatric population, several important questions remain. The purpose of the present thesis was to improve our understanding of the relationship between sedentary behaviour and cardiometabolic disease risk in school-aged children and youth. To do this, I have focused on 2 core areas of research:

1. The cross-sectional relationship between characteristics of sedentary behaviour/human movement and markers of cardiometabolic disease risk.
2. The metabolic and behavioural impact of prolonged sitting.

1.1 Characteristics of Sedentary Time

While accumulating evidence suggests a relationship between total sedentary time and markers of cardiometabolic disease risk in the pediatric population, recent evidence in adults suggests that the manner in which sedentary time is accumulated may also have an important health impact. Interruptions in sedentary time (e.g. standing or walking for brief periods of time) are associated with reduced body weight, abdominal fat, and cardiometabolic disease risk independent of both total sedentary time and physical activity in adults (13,14). Although a handful of studies have examined the association between breaks in sedentary time and markers of cardiometabolic disease risk in the pediatric population (15–18), none have been able to replicate the results observed in adults. Thus, at present it is unclear whether frequent interruptions in sedentary time are associated with reduced cardiometabolic disease risk among children and youth. However, it should be noted that past studies in this area have focused primarily on representative samples of children and youth (15, 16), who typically have low levels of cardiometabolic disease risk. It is possible that associations between characteristics of sedentary behaviour and cardiometabolic disease risk may be easier to detect among children with increased risk of cardiometabolic diseases, although this has not yet been investigated in any pediatric population.

Another unexamined aspect of human movement that may account for variation in cardiometabolic disease risk is total movement variability, defined as minute-to-minute changes in accelerometer counts per minute (CPM). It is also possible that variability *per se* may also be beneficial to health (19–23). For example, altering the output of mechanical ventilators to include random variations in breathing rate and volume results in improved oxygen saturation and organ

function, when compared to ventilators that provide constant output (19,23). Further, for a given level of physical activity, individuals with high amounts of movement variability are likely to have lower levels of sedentary behaviour, and more frequent breaks in their sedentary time. To date no studies have examined the relationship between movement variability and cardiometabolic disease risk in any population.

1.2 The impact of prolonged sitting on markers of cardiometabolic disease risk

As mentioned above, there is an accumulating body of research in adults and children suggesting that excessive sedentary behaviour is associated with increased health risk. Further, evidence from both adults (24–26) and animal models (27) suggests that just a few hours of prolonged sedentary behaviour can result in dramatic changes in markers of cardiometabolic disease risk. Bey and Hamilton (27) have reported that just 6 hours of sedentary behaviour resulted in dramatic reductions in lipoprotein lipase activity in rat skeletal muscle, and that just one day of sedentary behaviour resulted in a 20% reduction in plasma HDL-cholesterol levels. Similarly, it has recently been reported that in comparison to a day that includes minimal sitting, a day of constant sitting reduced insulin action by 39% in healthy, recreationally active adults (24). In support of these findings, a recent systematic review from our group concluded that acute bouts of uninterrupted sedentary behaviour result in rapid and deleterious changes in insulin sensitivity, glucose tolerance, and lipid levels among adults (26).

Taken together, the above results suggest that as little as 1 day of uninterrupted sedentary behaviour may have a measureable and deleterious impact on markers of cardiometabolic disease risk. However, no study has examined the immediate cardiometabolic impact of a laboratory-controlled bout of sedentary behaviour in children and youth. Therefore, despite evidence in both

adult (24–26) and animal models (27), the acute influence of sedentary behaviour on markers of cardiometabolic disease risk in the pediatric population is unknown.

1.3 The impact of prolonged sitting on energy intake and physical activity

In addition to questions regarding the direct relationship between sedentary time and markers of cardiometabolic disease risk in youth, the relationship between sedentary behaviour and behavioural compensation (e.g. increases or decreases in physical activity or food intake associated with acute sedentary behaviour exposure) in the pediatric population is also unclear, and is another mechanism that may link excess levels of sedentary behaviour with increased health risk. For example, it has been reported that every one-hour increase in daily TV viewing among school-children is associated with an extra consumption of 167 calories per day (28). However, it is unclear whether this is due to factors related to chronic TV viewing (for example, exposure to advertisements for nutrient-poor foods (29), by distracting children from feelings of hunger and satiety (30)), or whether simply sitting for extended periods of time results in increased hunger and food intake in this population. It is worth noting that engaging in seated video game playing (31), has been reported to increase spontaneous food intake in adolescents, and a number of studies suggest that screen-based sedentary behaviours may lead to increased caloric consumption through a variety of mechanisms (32-34). However, despite the reported relationships between specific sedentary behaviours and subsequent food intake in adults, to date no study has investigated the impact of sitting *per se*, with or without breaks or structured physical activity, on hunger or food intake in children and youth.

As with hunger and food intake, to date no studies have examined the impact of prolonged sitting on subsequent levels of spontaneous physical activity. It has been suggested that physical activity may be centrally controlled by an "activitystat", such that individuals unconsciously increase or

decrease their activity level to match energy intake or other internal cues (35). If true, this would suggest that children may increase their physical activity levels following a period of prolonged sitting. In support of this hypothesis, it has been reported that the introduction of structured physical activity may fail to increase (36), or may even reduce (37) total physical activity levels in the pediatric population, as a result of reductions in spontaneous physical activity and increases in sedentary behaviour. However, the impact of prolonged sitting on subsequent energy intake, physical activity and sedentary behaviour levels is presently unknown.

1.4 Objectives

The general objective of this thesis was to improve our understanding of the relationship between sedentary behaviour and markers of health in children and youth. In particular, it focused on 2 broad objectives that address the key gap areas described previously.

1. To determine the cross-sectional association of sedentary time, interruptions in sedentary time, sedentary bout length, and total movement variability with markers of cardiometabolic disease risk among children and youth.
2. To examine the impact of 1-day of prolonged sedentary behaviour, with and without interruptions or structured physical activity, on markers of cardiometabolic disease risk, hunger, food intake and spontaneous physical activity levels in children and youth.

1.5 Hypotheses

The general hypothesis of this thesis was that sedentary time would be positively associated with increased health risk among children and youth in both cross-sectional and intervention analyses. The specific hypotheses, and the individual studies that they accompany, are found below.

Study 1. We hypothesized that markers of cardiometabolic disease risk would be negatively associated with interruptions in sedentary time and positively associated with long bouts of uninterrupted sedentary time in cross-sectional analyses. We hypothesized that these relationships would be independent of total sedentary behaviour and physical activity levels.

Study 2. We hypothesized that markers of cardiometabolic disease risk would be negatively associated with total movement variability (defined as the minute-to-minute variation in movement intensity as assessed using accelerometer counts per minute) in children and youth. We hypothesized that this relationship would be independent of total sedentary time and physical activity levels.

Study 3. We hypothesized that a day which included short breaks in sedentary time or structured physical activity would result in significantly lower levels of cardiometabolic disease risk (defined as insulin, glucose and lipid area-under the curve), in healthy children and youth, as compared to a day of prolonged sitting. Further, we hypothesized that a day which included both interruptions in sedentary time *and* structured physical activity would result in greater reductions in markers of cardiometabolic disease risk than a day which included interruptions in sedentary time but no structured physical activity.

Study 4. We hypothesized that a day which included short breaks in sedentary time or structured physical activity would not influence subsequent levels of energy intake and hunger in children and youth, when compared to a day of prolonged sitting. We further hypothesized that participants would compensate for a day of prolonged

sitting by reducing their level of sedentary time, and increasing their level of physical activity, in the subsequent 24 hours.

1.6 Relevance

At present it is unclear whether characteristics of sedentary time or total movement variability are associated with markers of cardiometabolic disease risk in children and youth. It is also unknown whether prolonged sitting results in acute changes in markers of cardiometabolic disease risk or behavioural compensation. These critical gaps in knowledge are extremely important from a public health perspective, as recent evidence suggests that Canadian children spend roughly 8 hours per day - more than half of their waking hours – sitting (38–40). More than 70% of the average school day, including physical education, is sedentary (39), while the average Canadian child reports accumulating more than 6 hours per day of screen time during their discretionary leisure time (40). Given the ubiquity of sitting in the Canadian pediatric population, it is critical that we improve our understanding of the relationship between sedentary behaviour and health in pediatric populations.

The knowledge gaps described above limit our understanding of the health impact of sedentary behaviour in children and youth, and preclude the development of maximally efficacious interventions in this age group. Although cross-sectional evidence in adults suggests that characteristics of sedentary time may account for cardiometabolic disease risk beyond that explained by total sedentary time alone, these findings have yet to be replicated in children and youth. Similarly, while accumulating evidence suggests that sedentary behaviour is associated with increased health risk in children, it is unknown whether a single session of lab-controlled sedentary behaviour has a measurable metabolic impact in the pediatric population, or whether characteristics of sedentary time influence this impact. The studies contained within this thesis

are an attempt to address these knowledge gaps, and will assist in the development of improved clinical and public health interventions targeting chronic disease risk in this age group.

Chapter 2 - Literature Review: Sedentary behaviour as an emerging risk factor for cardiometabolic diseases in children and youth

The following article is has been accepted for publication in the Canadian Journal of Diabetes, and has been formatted according to their requirements

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Author Contributions

The article was conceived and designed by TJS, JPC and MST. TJS wrote the initial draft of the manuscript, while MST and JPC provided critical edits and additions. All authors approved the final manuscript.

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Abstract

Sedentary behaviour (e.g. TV viewing, seated video game playing, prolonged sitting) has recently emerged as a distinct risk factor for cardiometabolic diseases among children and youth. This narrative review provides an overview of recent evidence in this area, and highlights research gaps. Current evidence suggests that North American children and youth spend between 40 and 60% of their waking hours engaging in sedentary pursuits. Although data are lacking on temporal trends of objectively measured sedentary time, self-reported sedentary behaviours have increased over the last half-century, with a rapid increase since the late 1990's. Excessive sedentary behaviour has been found to have independent and deleterious associations with markers of adiposity and cardiometabolic disease risk. These associations are especially consistent for screen-based sedentary behaviours (TV viewing, computer games, etc), with more conflicting findings observed for overall sedentary time. The above associations are possibly mediated by the influence of screen-based sedentary behaviours on energy intake. Although excessive sitting has been reported to have adverse acute and chronic metabolic impacts among adults, research on children is lacking. Research is particularly needed to investigate the impact of characteristics of sedentary behaviour (i.e. type/context, sedentary bout length, breaks in sedentary time, etc), as well as interventions that examine the health and behavioural impact of sitting *per se*.

Keywords: Sedentary behaviour, pediatrics, cardiometabolic disease

Introduction

It is well established that high levels of physical activity are associated with reduced health risk in children and youth (1–3). Physical activity exhibits a dose-response relationship with health indicators in the pediatric population, and even modest amounts of physical activity can result in improved health for those at greatest risk (1). However, in addition to the consistent association between physical activity and health in the pediatric population, accumulating evidence suggests that the amount of time children and youth spend engaging in sedentary behaviours (i.e. activities that involve sitting or reclining while expending ≤ 1.5 metabolic equivalents (4)) may be associated with *increased* cardiometabolic disease risk independent of other factors such as physical activity and abdominal obesity (5–12). In response to this new research, Canada has recently created pediatric sedentary behaviour guidelines, which are separate from (but complementary to) physical activity guidelines for this age group (11). These guidelines recommend that school-aged children and youth accumulate no more than 2 hours of recreational screen time each day, and that they also limit periods of prolonged sitting and motorized transport (11). Although a number of recent narrative reviews have examined the health impact of sedentary behaviour in adults (13–17), there is a lack of such a review in the pediatric population. Thus, this article aims to provide a comprehensive overview of the available evidence concerning sedentary behaviour and markers of cardiometabolic disease risk in school-aged children and youth.

What is sedentary behaviour?

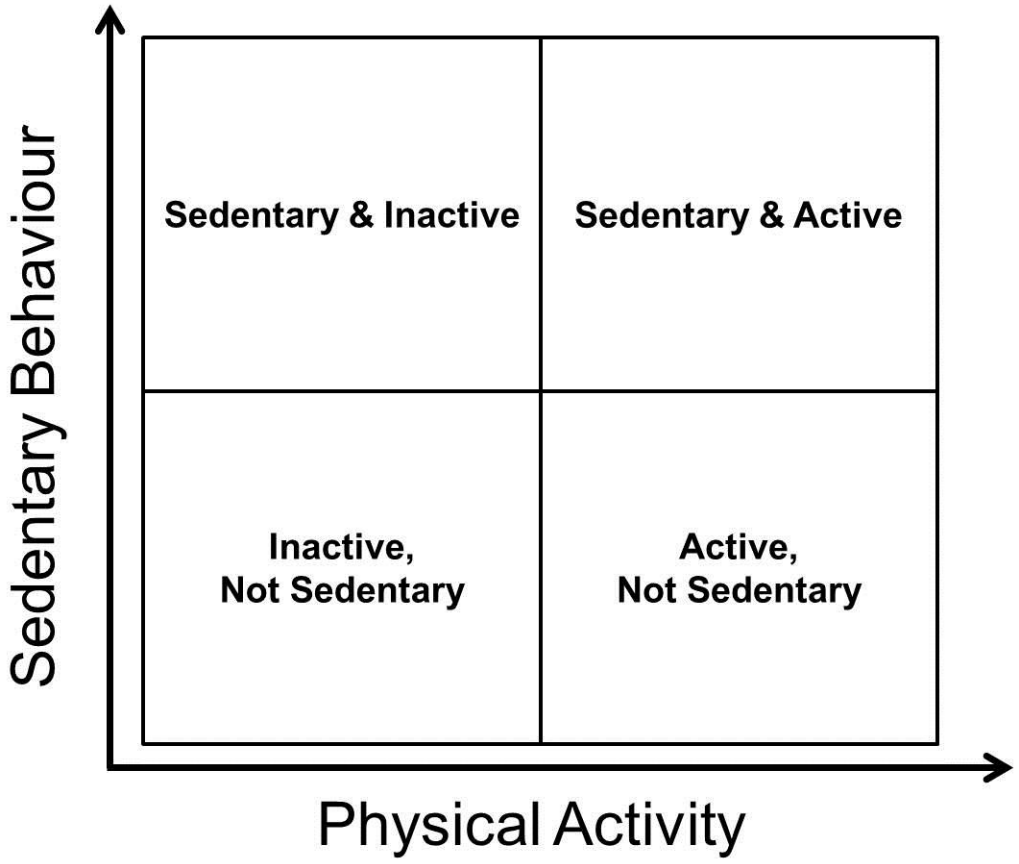

Figure 2.1 Sedentary behaviour and physical activity as distinct constructs.

What is sedentary behaviour?

The meaning of the word "sedentary" has evolved rapidly in recent years (18). Although the Latin root of the word sedentary literally means "to sit" (15), the term has historically been used by health researchers to refer to an individual who is not sufficiently physically active (4). Similarly, the phrase "sedentary lifestyle" has typically been used to refer to a lifestyle which includes little or no physical activity (19). It has therefore been relatively common for researchers to refer to individuals as "sedentary" due to their lack of physical activity, rather than the amount of time they spend sitting. However, recent evidence suggests that sitting too much and

exercising too little are separate and distinct risk factors for chronic diseases including cancer, cardiovascular disease, and diabetes (15,16,20,21). Further, an individual can easily meet physical activity guidelines while spending the vast majority of their day engaging in seated activities, or vice versa (see Figure 2.1). As a result, it has been proposed that the term "sedentary" should be used only to refer to activities which are defined by both a seated or reclining posture, and an energy expenditure at or near resting levels (4). Therefore, in this review the term sedentary will be used to specifically refer to waking behaviours characterized by energy expenditure ≤1.5 METs while in a sitting or reclining posture (4). In contrast, the term "inactive" will be used to refer to an individual who is not sufficiently physically active (e.g. not meeting physical activity guidelines).

How is pediatric sedentary behaviour measured?

As with physical activity, sedentary behaviour can be assessed using a variety of self- and proxy-report questionnaires, or by direct measurement tools (15,22,23). Self- and proxy-report tools typically take one of two approaches: 1) asking children or their parents to estimate the amount of time that they spend engaging in common sedentary behaviours (e.g. watching television, using a computer, playing passive video games, driving in a car, etc) which may be reflective of total sedentary time, or 2) asking them to estimate the amount of total time that they spend sitting on a daily basis. These tools are attractive because they are inexpensive and result in data that are relatively simple to analyze, while providing information related to specific modalities or contexts of sedentary behaviour (e.g. television viewing vs. reading). A recent systematic review suggests that self- and proxy-report tools generally display acceptable reliability and validity in assessing sedentary behaviour (22). However, these measures have a number of limitations. First and foremost, they are known to be limited by high levels of error and recall bias (23–26). Further, no single sedentary activity is representative of an individual's total sedentary behaviour

profile (23,27,28), which can pose an issue when data collection focuses on a limited number of sedentary behaviour modalities.

In contrast to self-report tools, accelerometers and inclinometers allow for the direct measurement of sedentary behaviour in childhood (15,22,23). Accelerometers assess the number of movement "counts" in a given time period, and their use has increased rapidly in recent years (29). A variety of thresholds have been proposed to distinguish between sedentary behaviour and light-intensity physical activity, with a threshold of 100 counts per minute (CPM) being shown to have high sensitivity and specificity for the measurement of sedentary behaviour in pediatric populations using both Actigraph (Actigraph, Pensacola, USA) and Actical (Philips Respironics, Andover, USA) accelerometers (22,30–36). Accelerometers can also be used to assess the frequency of breaks in sedentary time and the duration of sedentary bouts, neither of which can be determined easily via self-report tools (37–39). However, a key limitation of accelerometers is their inability to distinguish between sitting and stationary standing (40), and the lack of information regarding the modality of sedentary behaviour (e.g. TV viewing vs. reading). Inclinometers such as the activPAL (PAL Technologies, Glasgow, UK) have been reported to be more accurate than accelerometers in differentiating between sitting and standing (40,41), with Aminian and Hinckson reporting that the activPAL was able to perfectly distinguish between the two postures in healthy elementary school children (41). As with accelerometers, however, inclinometers are unable to provide information on the modality of sedentary behaviour, and have been used far less frequently. As a result of the limitations of both self-report and direct measurement tools, researchers have therefore advocated for the concurrent use of both strategies whenever possible (22,23).

Prevalence of sedentary behaviour in the pediatric population

Available evidence suggests that children and youth in developed nations currently spend 40-60% of their waking hours engaging in sedentary pursuits. Colley et al. used accelerometers to assess total sedentary time in a representative sample of 1,608 Canadians between the ages of 6 and 19 years (37,42). They estimate that girls and boys respectively accumulate 7.4 and 8.5 hours of daily sedentary time, roughly half of which is accumulated during school hours (37,42). Sedentary time also tends to increase with age; children under 11 years averaged approximately 1.3 hours less daily sedentary time than those aged 11-14 years, and roughly 2 hours less than those aged 15-19 years (42). Similar levels and trends for accelerometer-derived sedentary behaviour have been reported in cross-sectional examinations of American (43) and European (44) children and youth.

The above-mentioned findings are also supported by longitudinal studies, which suggest that both screen time and total sedentary time increase with age (45,46). For example, a longitudinal study of 759 Vietnamese students observed that boys and girls increased their daily sedentary time by more than 1 hour between the ages of 13 and 16 years (45). Similarly, Brodersen et al. found that self-reported screen time increased by more than 2.5 hours/week during a 5-year period in a study of 5,863 British adolescents (46). It is worth noting that the frequency of breaks in sedentary time also appears to decrease with age; a longitudinal study of roughly 500 children found a decrease of approximately 2 breaks/hour/year from age 5 to age 15 (47). These findings suggest that children become more sedentary with age, and also accumulate their sedentary time in increasingly prolonged bouts.

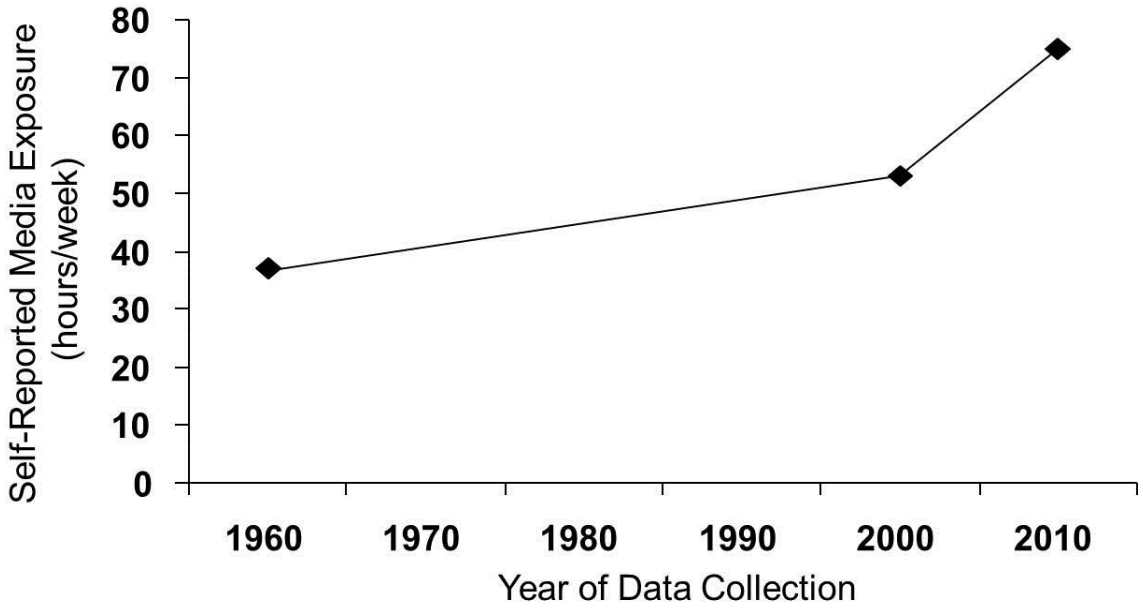

Figure 2.2 Self-reported media exposure of American youth over time.

Data from Schramm et al. (45) and Rideout et al. (46). Data have not been adjusted for multi-tasking (e.g. engaging with multiple media simultaneously).

Given the relatively recent introduction of accelerometry in population-based research, it is difficult to assess temporal trends in objectively measured sedentary time. However, self-reported media use (including TV, radio, audio, reading, etc) appears to have increased since the 1960's, with rapid increases observed in the past decade. Schramm et al. (48) reported that American children in grades 6 and 12 averaged roughly 37 hours/week of total media exposure in 1961. In contrast, recent evidence from the Kaiser Family Foundation reported that American children between the ages of 8 and 18 years averaged 53 hours/week of total media exposure in 1999, and 75 hours in 2009 (49) (Figure 2.2). After adjusting for multi-tasking (e.g. engaging with two forms of media simultaneously), the average American youth currently spends 54 hours engaging with media each week (49). The same report estimated that total media use increased by 1.5 hours/day among White American children between 1999 and 2009, and by more than 3 hours/day among Black and Hispanic children over the same period (49). A nationally

representative study of 52,000 children and youth produced a similar estimate of total daily screen time among contemporary Canadian students (50), and temporal increases in self-reported screen time have also been reported in Czech girls (but not boys) (51) and Chinese children and youth (52,53) during the late 1990's and early 2000's, although reductions in total screen time have been reported in Norwegian children (54) and Czech boys (51). Consequently, it is not surprising that the majority of children in developed nations currently exceed pediatric screen time recommendations (11,55).

Along with the reported increases in total screen time, there has also been a shift away from TV viewing, and towards increased computer and video game use in recent decades (49,51,56,57). In a study of Czech children between 1998 and 2008, the percent of sedentary time accounted for by TV viewing decreased from 17 to 12% among girls, and from 24 to 15% among boys (51). During the same period, the proportion of sedentary time accounted for by computer use more than doubled in both sexes (51). Finally, evidence suggests that sedentary modes of transportation (e.g. driving) have also increased dramatically since the 1960's throughout the Western world (58–60). Taken together, the above evidence suggests that the volume of total daily sedentary time has likely increased in the past 50 years, with computer and video game use playing a larger role in recent years.

Sedentary time and markers of adiposity in children and youth

A recent systematic review by Tremblay et al. (61) examined the relationship between sedentary behaviour (typically assessed via self- or proxy-reported screen time) and adiposity in 170 separate studies of school-aged children. Among 119 cross-sectional studies, 94 observed positive associations between sedentary behaviour and markers of adiposity. Further, the risk of being identified as obese increased in a dose-response manner with sedentary time. For example, in a sample of 461 Mexican children and youth, Hernández and colleagues observed that the odds

of being classified as obese increased by 12% for every hour of self-reported television viewing (62). These cross-sectional findings are also supported by longitudinal evidence (61,63,64). Mitchell et al. (64) showed that objectively measured sedentary time was independently associated with increased weight gain between 9 and 15 years of age among children at the 50^{th}, 75^{th} and 90^{th} body weight percentiles, independent of other covariates including physical activity, sleep, and diet.

Finally, evidence from randomized controlled trials demonstrates that reductions in sedentary time may result in reductions in adiposity (61,65,66). For example, Robinson reported that elementary school children who were randomized to receive an intervention aimed at reducing screen time experienced a 0.45 kg/m^2 reduction in body mass index (BMI) and a 2.30 cm reduction in waist circumference when compared to control students over a 6 month period (65). These findings are supported by a recent systematic review and meta-analysis, which concluded that interventions that reduce sedentary behaviour in children result in a mean decrease in BMI of 0.89 kg/m^2 (61). These results suggest that sedentary behaviour (especially screen time) has an independent and causal influence on the risk of excess weight gain and obesity in the pediatric age group (61,67).

Sedentary time and markers of cardiometabolic disease risk in children and youth

Although it has been the focus of less research than adiposity, emerging evidence suggests that sedentary behaviour is also independently associated with other markers of cardiometabolic disease risk in children and youth (5,8–10,12,61,68–71). Goldfield et al. have recently reported that television viewing and video game playing are independently associated with risk factors for diabetes and cardiovascular disease, respectively, independent of physical activity in overweight and obese adolescents (9,10). Similarly, Kriska et al. (71) observed that in comparison to obese youth without diabetes, those recently diagnosed with diabetes accumulated roughly 1 additional

hour of objectively measured sedentary time each day. These results are also supported by a recent report by Wennberg et al. (72), who found that self-reported TV viewing at age 16 is prospectively associated with the risk of metabolic syndrome at age 43 years. Participants who reported watching "several TV shows a day" at baseline had twice the odds of having metabolic syndrome at follow-up, independent of physical activity, socioeconomic status, and family history of diabetes. Associations were also seen for individual metabolic syndrome components including central obesity, lipids, and blood pressure (72). As with adiposity, these findings suggest that sedentary behaviour (typically measured as self-reported screen time) is independently associated with increased cardiometabolic disease risk in the pediatric population.

The role of sedentary behaviour modality: screen based vs. non-screen based sedentary time

In the 5 year span between 2005 and 2010, the number of investigations assessing sedentary behaviour using objective measures doubled (29). As the volume of studies using both objective measures of sedentary behaviour (which assess total sedentary time) and self-reported sedentary behaviour (which typically focus on specific sedentary behaviours like screen time) has increased, a surprising trend has become apparent in the literature. While self-reported screen time is consistently associated with increased adiposity and cardiometabolic disease risk in children and youth independent of physical activity levels (7–10,61,68,70,73–75), the relationship between objectively measured sedentary time and health indicators is far less clear. Of the numerous studies examining the relationship between objectively measured sedentary time and markers of adiposity and cardiometabolic disease risk in the pediatric population (5,12,37,64,68,69,71,74–82), only a small number (64,69,79) have detected associations which remained significant after adjustment for physical activity (Table 2.1). These findings raise questions about the health impact of sitting *per se*, in comparison to the impact of specific screen-based sedentary behaviours.

The differences between self-reported screen time and directly measured total sedentary time are most apparent when examined using a single cohort. For example, Carson and Janssen observed that self-reported TV viewing was independently associated with clustered cardiometabolic disease risk in a nationally representative sample of American children and youth (70). In contrast, the authors observed no independent associations between accelerometer-derived sedentary time and markers of cardiometabolic risk in the same sample. Similar findings have also been reported in other cohorts (68,73–75,77). Chaput and colleagues found that self-reported screen time (but not objectively measured total sedentary time) was independently associated with increased waist circumference and reduced HDL-cholesterol concentrations in a cohort of 536 children at-risk for obesity (68). Similarly, Martinez-Gomez et al. (75) reported that several biomarkers were independently and deleteriously associated with self-reported TV viewing, but not objectively measured sedentary time, in a group of Spanish adolescents.

Given the bias and error that are known to be associated with self-report measures (24,26), it is somewhat surprising that self-reported screen time appears to be more closely associated with health indicators than an objective measure of total sedentary time such as accelerometry. It is not uncommon for studies to report levels of self-reported screen time that seem highly implausible (26,83), a characteristic that has been observed in other forms of self-report data collection as well (84). For example, a recent study found that a group of highly active and highly sedentary 10-11 year old students self-reported an average of 13.9 hours *per day* of screen time, and another 5.9 hours of physical activity (26,83). If this were true, it would leave only 4.2 hours each day for eating, sleeping, and attending school, which seems unlikely. It has also been noted that the association between self-reported and directly measured sedentary behaviour can be extremely small (23,70). Carson and Janssen observed a correlation of just 0.08 between self-reported TV viewing and accelerometer-derived sedentary time in a nationally representative sample of American children and youth (70), suggesting little overlap between the two measures.

There are a number of factors that could explain the differences observed between self- or proxy-reported screen time and objectively measured sedentary time in children and youth. Self- and proxy-reports of sedentary behaviour typically only provide information on a single behaviour (a subset of total sedentary time), while objective measures of sedentary time provide a global measure of time spent sedentary. The two types of measures are therefore assessing different things (23). For this reason, self- and proxy-reported sedentary behaviour typically account for far less total time than do objective measures; Colley et al. found that parent-reported screen time was equivalent to only a third of total sedentary time assessed via accelerometry in a representative sample of Canadian children (23). Given the highly sedentary nature of contemporary life (42,43,47,85,86), it has also been suggested that the weak associations seen with objective measures of sedentary behaviour and different health indicators in children and youth may be due to a lack of inter-individual variability (23). Further, it has been noted that a variety of methods have been used to process accelerometer data in the pediatric population, and this may have a significant impact on the results of individual studies, making it difficult to directly compare results from separate investigations (29,79). For example, studies have excluded data as "non-wear" time when there are as few as 10 (5,75), or as many as 100 (79) consecutive minutes with accelerometer values of 0 CPM. Further, although an accelerometer threshold of 100 CPM is used most commonly to identify sedentary behaviour, thresholds as high as 1100 CPM have been used in studies examining the relationship between sedentary time and markers of adiposity and cardiometabolic risk in the pediatric age group (79). While the impact of such methodological issues is not certain, a recent report by Atkin et al. (79) suggests that discrepancies in sedentary thresholds (e.g. 100 CPM vs 1100 CPM) are likely to have a much larger impact than differences in non-wear time. Counter intuitively the same authors reported that *higher* thresholds, which therefore classify higher intensities of movement as sedentary time, result in stronger associations between sedentary time and markers of cardiometabolic disease

risk in the pediatric population. Further research into the impact of such methodological issues, and techniques for comparing across studies employing different methodologies, is clearly warranted.

Finally, as discussed below, it is also possible that certain forms of sedentary behaviour (e.g. TV viewing and other forms of screen time) may disproportionately promote other unhealthy behaviours such as excess food intake, which may explain why they are more consistently associated with health risk in the pediatric population (67,87). Taken together, these findings suggest that screen time (and especially TV viewing time (88)) may be more closely associated with markers of cardiometabolic disease risk than total objectively measured sedentary time in the pediatric population, and this reinforces the notion that researchers should collect data using both measures whenever possible (22,23).

Characteristics of sedentary behaviour: impact on health indicators

In addition to the health impact of total sedentary time and specific sedentary behaviours (e.g. screen time), recent evidence in adults suggests that certain patterns of sedentary behaviour may also have an important health impact (38,39,89,90). A recent systematic review (89) concluded that prolonged bouts of uninterrupted sedentary behavior have a rapid and deleterious impact on insulin sensitivity, glucose tolerance, and triglyceride levels in adults. Further, interruptions in sedentary time have been shown to be beneficially associated with body weight, abdominal fat, triglycerides and glucose metabolism in adults (38,39,90). These findings have yet to be replicated in the pediatric population.

Carson and Janssen failed to detect any associations between breaks in sedentary time, sedentary bout length, and cardiometabolic disease risk in a nationally representative sample of 2,527 American children and youth after adjustment for potential confounders (70). Kwon et al. (47)

also reported no association between breaks in sedentary time and fat mass in a sample of 544 boys and girls in the Iowa Bone Development Study, with similar findings being reported among children from the Pacific Islands (91). To our knowledge, only one study to date has reported an association between characteristics of sedentary time and health indicators in children and youth; Colley and colleagues found that prolonged bouts of sedentary behaviour (those lasting 80+ minutes) are positively associated with waist circumference in boys aged 11-14 years from the Canadian Health Measures Survey, while the opposite is true for breaks in sedentary time (37). However, these associations were not observed in older or younger boys, or in girls of any age.

The limited evidence available to date suggests that characteristics of sedentary behaviour may be less closely associated with cardiometabolic disease risk in children than has previously been reported in adults. However, it should be noted that the studies that have been published to date have focused primarily on nationally representative samples of children and youth (37,70). It is possible that associations between characteristics of sedentary behaviour and cardiometabolic disease risk may be stronger in populations with a family history of obesity, as this has previously been associated with increased childhood cardiometabolic risk (92–94).

Mechanisms by which sedentary behaviour can lead to poor health outcomes in children and youth

A number of mechanisms have been suggested that could explain the reported associations between sedentary behaviour and cardiometabolic disease risk in the pediatric population. Sedentary behaviours are defined by their low energy expenditure, and it has historically been assumed that they displace physical activity (4). This view is supported by a recent randomized crossover study which observed that exposing children to several hours of prolonged sitting did not result in any changes in physical activity levels in the subsequent 24-hour period (95). This suggests that when children engage in a bout of prolonged sedentary behaviour they do not

compensate by increasing physical activity levels later on, thereby promoting positive energy balance (95). However, other evidence suggests that the displacement of physical activity plays a relatively small role in mediating the relationship between sedentary time and cardiometabolic disease risk in children and youth (56,67,88,96,97). In a systematic review and meta-analysis examining the relationship between sedentary behaviours and physical activity, Marshall et al. (96) reported that while the two are negatively associated, the magnitude of the relationship is too small to be of clinical significance. As noted above, numerous studies have also observed significant associations between sedentary behaviour (whether self-reported or directly measured) and markers of cardiometabolic disease risk independent of physical activity levels in the pediatric population (9,10,61,70). These findings suggest that a lack of physical activity is not the primary factor linking sedentary behaviour with health indicators in this age group.

In contrast to a lack of physical activity, a number of studies suggest that screen-based sedentary behaviours may lead to increased caloric consumption through a variety of mechanisms (98,99). A recent intervention by Harris et al. (100) observed that exposing children to televised food advertisements increases subsequent *ad libitum* food intake by 45%. Similar results have also been reported by Halford and colleagues (101,102), who reported that the impact of advertisements on increased food intake is seen across all body weight categories, although it is most pronounced among children with obesity (101). They also noted that the ability to recognize food advertisements was positively associated with food intake, and that children with overweight and obesity were more likely to remember food advertisements after being exposed to them, when compared to their lean peers (101). It is possible that television viewing may also result in increased food intake by inducing "mindless eating" (103). An intervention study by Temple et al. (104) found that children spend more time eating and consume roughly twice as many calories while watching a continuous television program, in comparison to a control condition without entertainment. Passive video game playing has also been shown to increase

food intake and result in a positive energy balance in the pediatric population. A randomized crossover study by Chaput et al. (105) observed that compared to sitting quietly, 1 hour of passive video game play resulted in an 80 kcal increase in *ad libitum* food intake in adolescent boys. The collective findings suggest that sedentary screen-based sedentary behaviours (in particular television viewing) are likely to result in increased energy intake and positive energy balance in the pediatric population .

Finally, studies in adults suggest that prolonged sitting may have a rapid and direct impact on metabolic health, independent of changes in body weight or other health behaviours (15,89). For example, intervention studies report that even relatively short bouts of uninterrupted sedentary behaviour result in reduced insulin sensitivity, glucose tolerance, and increased triglyceride levels in adults (20,89,90,106,107). In comparison to a day of sitting that included periodic light-intensity walk breaks, Dunstan et al. reported that a day of uninterrupted sitting resulted in a 30% increase in insulin resistance in a group of overweight and obese adults (90). Similar results have also been reported in normal weight adults (106,107) and may be due to reductions in lipoprotein lipase and glucose transport protein activity at the level of the skeletal muscle (13,15).

To date only one intervention study has examined the acute effect of prolonged sitting in a pediatric population. Saunders et al. (108) exposed healthy 10-14 year olds to a day of uninterrupted sitting, as well as days with periodic interruptions of light and moderate intensity physical activity. In contrast to previous reports in adults (90), they reported that uninterrupted sitting did not have any impact on the insulin, glucose or lipid response to a standardized meal in this population. Although further intervention studies in children and youth are needed, the available evidence suggests that sitting *per se* may not have a direct deleterious impact on cardiometabolic health in healthy children and youth. Though it could be that the inherent

metabolic health of children is such that current analytical methods have limited sensitivity to detect subtle, but adverse, physiological changes.

Opportunities for future research

Although the relationship between certain sedentary behaviours (e.g. screen time) and cardiometabolic disease risk among children and youth are well-established, the impact of sitting *per se* is far less clear. As discussed above, independent associations between objectively measured sedentary time and cardiometabolic risk have been reported by some, but not all studies. More research is therefore needed to clarify the relationship between objectively measured sedentary behaviour and health indicators in the pediatric age group. Systematic reviews and meta-analyses focusing specifically on objectively measured sedentary behaviour (as opposed to previous reviews that have focused on *all* sedentary behaviours) may be especially useful in this regard. Standardization of accelerometry methodology would also allow for much easier comparisons across studies. Future research should also examine whether any personal factors such as sex or body weight influence the reporting of screen time, and why some screen-based sedentary behaviours are associated with health outcomes in certain populations, but not others (9,10).

More research is also needed into the role played by specific characteristics of sedentary behaviour in the pediatric population. Only a small number of studies have investigated the impact of sedentary bout length or breaks in sedentary time in the pediatric population, or the importance of sedentary behaviour during different periods of the day (37,70). A better understanding of the characteristics of sedentary behaviour that are most closely associated with cardiometabolic disease risk is needed in order to develop interventions that are maximally efficacious in reducing cardiometabolic risk among children and youth. It is also possible that

previously unexamined aspects of accelerometry data, such as total movement variability, may provide additional valuable information on movement patterns in the pediatric age group.

Finally, intervention studies are needed that examine the health and behavioural impact of prolonged sitting in the pediatric population. A recent systematic review concluded that uninterrupted sedentary behaviour results in rapid and deleterious changes in insulin sensitivity, glucose tolerance, and lipid levels in adults (89). However, as noted above, these findings have yet to be replicated in children or youth (108). By extension, it is also unclear whether substituting sedentary behaviour for standing or light-intensity physical activity can lead to improvements in cardiometabolic disease risk among the pediatric population. Further research on the relationship between sedentary behaviours and sleep quality and quantity is also required given the importance of a good night's sleep for overall health (109). Additionally, given the decline of outdoor active play observed over recent decades in children and youth (110), more research is urgently needed to better understand the implications of excessive indoor time and its associated sedentary, technology-centered activities on children's health.

Conclusions

Available evidence suggests that North American children and youth spend between 40 and 60% of their waking hours engaging in sedentary behaviours (42–44). Markers of adiposity and cardiometabolic risk are positively associated with sedentary behaviour in general, and with screen-based sedentary behaviours in particular. These relationships appear to be due to the influence of screen-based sedentary behaviours on food intake, and may also be due to a direct metabolic impact of prolonged sitting, although this has received little research attention in the pediatric population. More research is needed to investigate the impact of characteristics of sedentary behaviour (sedentary bout length, breaks in sedentary time, etc), and interventions that examine the health and behavioural impact of sitting *per se*. Despite limited evidence in children

and youth, reducing sedentary time in addition to increasing physical activity may have a significant role in the prevention of chronic diseases, including diabetes.

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Author Contributions

The article was conceived and designed by TJS, JPC and MST. TJS wrote the initial draft of the manuscript, while MST and JPC provided critical edits and additions. All authors approved the final manuscript.

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Tables

Table 2.1 Comparison of reports examining the association between objectively measured sedentary time and markers of adiposity and cardiometabolic disease risk among children and youth.

Reference	Setting (Population)	Age	N (M/F)	Accelerometer	Sedentary Cut-Point	Key Findings
No significant associations reported						
Colley et al. (37)	Canada (General Population)	6-19 years	1,608 (809/799)	Actical	<100 CPM	Sedentary time was not associated with BMI, waist circumference, HDL-cholesterol or systolic or diastolic blood pressure independent of age, wear time and MVPA.
Carson and Janssen (70)	USA (General Population)	6-19 years	2,527 (1,284/1,243)	Actigraph AM-7124	<100 CPM	Sedentary time was not associated with clustered cardiometabolic risk after adjustment for age, gender, race, SES, smoking, total fat, saturated fat, cholesterol, and sodium, or after additional adjustment for MVPA.
Martinez-Gomez et al. (75)	Spain (General Population)	13-17 years	183 (95/88)	ActiGraph GT1M	<100 CPM	Sedentary time was not associated with CRP, adiponectin or other adipokines after adjustment for sex, age, and pubertal status, or after further adjustment for BMI and MVPA.
Kwon et al. (78)	USA (General Population)	8-15 years	554 (277/277)	Actigraph 7164	<100 CPM	Sedentary time was not associated with fat mass after adjustment for height and MVPA.

Chaput et al. (74)	Canada (Children with a family history of obesity)	8-11 years	550 (299/251)	Actigraph LS 7164	<100 CPM	Sedentary time was not associated with body fat percentage or waist-to-height ratio with or without adjustment for age, sex, sleep duration, energy intake, sexual maturation, parental SES and BMI or MVPA.
Martinez-Gomez et al. (77)	USA (General Population)	3-8 years	111 (57/54)	Actigraph 7164	<100 CPM	Sedentary time was not associated with systolic or diastolic blood pressure after adjustment for age, sex, height or body fat percentage.
Significant associations reported for at least one outcome, not independent of physical activity.						
Sardinha et al. (5)	Portugal (General Population)	9-10 years	308 (161/147)	MTI Actigraph	<500 CPM	Sedentary time positively associated with insulin resistance after adjustment for sex, sexual maturity, birth weight, measurement time and both total and central adiposity.
Cliff et al. (12)	Australia (Overweight and Obese)	5-10 years	126	Actigraph 7164	<100 CPM	Sedentary time was negatively associated with HDL-cholesterol, but not triglycerides, total- or LDL-cholesterol after adjustment for age, sex, adiposity, and diet. Sedentary time was not associated with any outcome after additional adjustment for MVPA.
Kriska et al. (71)	USA (Youth with obesity and type 2 diabetes)	10-18 years	551	ActiGraph AM7164	< 1 MET	Obese youths with T2D were sedentary for 56 more minutes/day than obese youth without T2D.

Ekelund et al. (82)	UK, Switzerland, Belgium, USA, Australia, Denmark, Estonia, Norway, Brazil, Portugal (General Population)	4-18 years	20,870 (10,097/10,773)	Various types of Actigraph	<100 CPM	Sedentary time was associated fasting insulin, but not waist circumference, systolic blood pressure, triglycerides or HDL-cholesterol after adjustment for age, sex, wear time, waist circumference and height. Sedentary time was not associated with any outcome after further adjustment for MVPA.
Chaput et al. (68)	Canada (Children with a family history of obesity)	8-11 years	536 (292/244)	Actigraph LS 7164	<100 CPM	Sedentary time was positively associated with diastolic blood pressure, but not waist circumference, triglycerides, systolic blood pressure, fasting glucose, or HDL-cholesterol, after adjustment for age, sex, waist circumference, sleep duration, energy intake, sexual maturation, parental SES and BMI. Sedentary time was not associated with any outcome after further adjustment for MVPA.
Basterfield et al. (76)	UK (General Population)	7-9 years	377 (186/191)	Actigraph GT1M	<1100 CPM	Changes in sedentary time were associated with increased fat gain in the entire sample independent of SES, baseline sedentary time, and baseline fat mass index. This association was no longer significant after additional adjustment for MVPA.

Mitchell et al. (80)	UK (General Population)	12 years	5,434 (2,950/2,844)	Actigraph AM7164	≤199 CPM	Sedentary time was significantly associated with increased risk of obesity independent gender, SES, pubertal status and early life sleep and TV habits. These associations were no longer significant after adjustment for MVPA.
Steele et al. (81)	UK (General Population)	9-10 years	1,862 (820/1042)	Actigraph GT1M	<100 CPM	Sedentary time was positively associated with waist circumference and fat mass index (but not BMI) in unadjusted analyses. Sedentary time remained associated with fat mass index after adjustment for age, sex, SES, birth weight, sleep duration or maternal BMI. Sedentary time was not associated with any outcome after further adjustment for MVPA.
Hsu et al. (73)	USA	8-19 years	105 (26/79)	Actigraph GT1M	<100 CPM	Sedentary time was positively associated with waist circumference and systolic blood pressure, but not triglycerides, fasting glucose, HDL-cholesterol or diastolic blood pressure in unadjusted analyses. After adjustment for MVPA, sedentary time was not associated with any outcome.

Significant associations reported for at least one outcome, independent of physical activity.						
Henderson et al. (69)	Canada (Children with a family history of obesity)	8-11 years	424 (222/202)	Actigraph LS 7164	<100 CPM	Sedentary time was positively associated with insulin resistance after adjustment for sex, age, pubertal stage, fitness and MVPA, but not after additional adjustment for adiposity.
Mitchell et al. (64)	USA (General Population)	9-15 years	798 (391/407)	ActiGraph 7164 and GTM1	<100 CPM	Sedentary time was associated with weight gain the 50th 75th and 90th BMI percentiles independent of gender, race, maternal education, hours of sleep, healthy eating scores and MVPA. No significant associations were observed at lower BMI percentiles.
Atkin et al. (79)	Denmark, Estonia and Portugal (General Population)	9, 15 years	2,327(1,059/1,268)	MTI Actigraph	<100, <500, <800, and <1100 CPM	In meta-regression using data from all cut-points, sedentary time was associated with increased clustered cardiometabolic disease risk (but not adiposity) independent of age group, age, sex, study location, sexual maturity, day of the week, season, wear time, adiposity and total physical activity. The relationship between sedentary time and clustered risk was stronger at higher accelerometry thresholds.

CPM, counts per minute; MVPA, moderate and vigorous physical activity; SES, socioeconomic status; HDL, high density lipoprotein; BMI, body mass index; CRP, C-Reactive Protein.

Chapter 3 - Study 1: Associations of sedentary behavior, sedentary bouts and breaks in sedentary time with cardiometabolic risk in children with a family history of obesity

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Author Contributions

Data collection was conducted by members of TEAM PRODIGY, an inter-university research team including Université de Montréal, Concordia University, Université Laval, and McGill University. The analyses included in this article were conceived and designed by TJS, JPC and MST. TJS performed statistical analyses and wrote the initial draft of the manuscript. MST,

MEM, MH, JO, AT and JPC provided critical edits and additions. All authors approved the final manuscript.

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Abstract

Background: Although reports in adults suggest that breaks in sedentary time are associated with reduced cardiometabolic risk, these findings have yet to be replicated in children.

Purpose: To investigate whether objectively measured sedentary behavior, sedentary bouts or breaks in sedentary time are independently associated with cardiometabolic risk in a cohort of Canadian children aged 8-11 years with a family history of obesity.

Methods: Data from 286 boys and 236 girls living in Quebec, Canada, with at least one biological parent with obesity (QUALITY cohort) were collected from 2005-2008, and analyzed in 2013. Sedentary behavior, light and moderate-to-vigorous physical activity were measured over 7 days using accelerometry. Leisure time computer/video game use and TV viewing over the past 7 days were self-reported. Outcomes included waist circumference, body mass index Z-score, fasting insulin, fasting glucose, triglycerides, HDL-cholesterol, C-reactive protein and a continuous cardiometabolic risk score.

Results: After adjustment for confounders, breaks in sedentary time and the number of sedentary bouts lasting 1-4 minutes were associated with reduced cardiometabolic risk score and lower BMI Z-score in both sexes (all $p<0.05$). The number of sedentary bouts lasting 5-9 minutes was negatively associated with waist circumference in girls only, while the number of bouts lasting 10-14 minutes was positively associated with fasting glucose in girls, and with BMI Z-score in boys (all $p<0.05$). Leisure time computer/video game use was associated with increased cardiometabolic risk score and waist circumference in boys, while TV viewing was associated with increased cardiometabolic risk, waist circumference, and BMI Z-score in girls (all $p<0.05$).

Conclusions: These results suggest that frequent interruptions in sedentary time are associated with a favourable cardiometabolic risk profile and highlight the deleterious relationship between screen time and cardiometabolic risk among children with a family history of obesity.

Background

Sedentary behavior (e.g. sitting or reclining while expending ≤ 1.5 metabolic equivalents) [1] is independently associated with increased cardiometabolic risk in children and youth [2-10]. Recent systematic reviews have reported that sedentary behavior is associated with reduced cardiorespiratory fitness, increased adiposity and elevated risk of metabolic syndrome in the pediatric age group [3,4]. However, while a growing body of evidence suggests that sedentary behavior represents a novel risk factor for chronic disease among children and youth, it is unclear which characteristics and modalities of sedentary behavior are most closely associated with increased health risk in this population [5,7,8,11].

Self-reported screen-based sedentary behaviors (e.g. television viewing, computer use, video game playing, etc.) have been consistently associated with increased markers of cardiometabolic risk in children and youth, independent of physical activity levels [3,5,7-9]. In contrast, studies examining accelerometer-derived measures of sedentary behavior in this age group have often failed to detect a significant association with markers of cardiometabolic risk after adjustment for confounders [5,11-14]. Similarly, while interruptions in objectively measured sedentary time are beneficially associated with markers of cardiometabolic risk in adults [15,16], these findings have yet to be replicated in the pediatric age group [5,11] where activity profiles are highly intermittent [17]. A better understanding of the relationship between characteristics of sedentary behavior and markers of cardiometabolic risk is necessary to inform lifestyle interventions and public health policies aimed at reducing chronic disease risk in children and youth.

The purpose of the present study was to investigate whether objectively measured sedentary time, or characteristics related to the accumulation of sedentary behavior (e.g. breaks in sedentary time or the accumulation of sedentary time in bouts of various lengths) are independently associated with cardiometabolic risk in a cohort of Canadian children aged 8-11 years with a family history

of obesity. It was hypothesized that a continous cardiometabolic risk score would be positively associated with sedentary behavior, and negatively associated with breaks in sedentary time in this population.

Materials and Methods

Study population

The sample consisted of 630 children enrolled in the QUebec Adiposity and Lifestyle InvesTigation in Youth (QUALITY) cohort, which has been described previously [18]. Briefly, participants in the QUALITY cohort are white and aged 8-11 years at study entry, and all participants have at least one biological parent with obesity (i.e. a body mass index (BMI) ≥30 kg/m^2 or abdominal waist circumference ≥88 cm for women or ≥102 cm for men). Children were excluded from the cohort if they were consuming a very low calorie diet (≤600 kcal/day), had a serious physical or mental health condition that could compromise participation in the study, had diabetes (type 1 or type 2), or were currently taking steroids, β-blockers, thiazides or other drugs for hypertension.

Roughly 400 000 flyers were distributed between 2005 and 2008 to families with children in Grades 2–5, in 1040 primary schools within 75 km of Montreal, Quebec City and Sherbrooke in Quebec, Canada. Of 3350 families who contacted the study coordinator, 1320 met all inclusion criteria. Reasons for non-participation at baseline among eligible families were: (i) not interested, 81%; (ii) at least one parent did not agree to participate or was unavailable, 11%; (iii) child declined to participate, 4%; (iv) lived too far from a study centre, 2%; (v) insufficient time, 1%; and (vi) other, 1%.

All data included in the present analysis were collected during baseline examinations between 2005 and 2008. The present cross-sectional analysis was performed in 2013 and includes 522 participants with complete data for all variables of interest.

Ethics Statement

This project was approved by the institutional ethics review boards at Centre Hospitalier Universitaire Sainte-Justine and Laval University. Written informed parental consent and child assent were obtained for all participants, in accordance with the principles expressed in the Declaration of Helsinki.

Outcome Measures

All markers of cardiometabolic risk were assessed during a hospital visit. Height was measured to the nearest millimeter using a wall-mounted stadiometer. Weight was assessed to the nearest 0.1 kg using a spring scale that was calibrated daily. Waist circumference was assessed at the midpoint of the lowest rib and iliac crest at the end of a normal exhalation. Body mass index (BMI) was calculated by dividing body mass (kg) by height in meters squared, and converted to a BMI Z-score based on values published by the Centers for Disease Control and Prevention [19]. All anthropometric measurements were taken in duplicate with participants wearing indoor clothing without shoes or sweaters and measured according to standardized methods by trained research assistants [18].

Metabolic markers were assessed using venous blood samples collected following a 12-hour overnight fast, analyzed in batches at a single site (CHU Sainte-Justine Clinical Biochemistry laboratory) [18]. Plasma insulin was measured with the ultrasensitive Access immunoassay system (Beckman Coulter, Brea, CA, USA). Glucose (oxidase method), HDL-C and triglycerides (enzymatic method) were measured using a Synchron LX, while high sensitivity C-Reactive

Protein (hs-CRP) (immunoassay method) was measured using a Synchron CX (Beckman Coulter, Brea, CA, USA). Blood pressure was measured on the right arm, with the child in a sitting position and at rest for at least 5 min, using an oscillometric instrument (Dinamap model CR9340, GE Healthcare, Mississauga, ON). Three consecutive measures were obtained with a 1 minute break between each measure. The average value of the 3 measures was used in the present analyses.

Calculation of a Continuous Cardiometabolic Risk Score

A sex-specific continuous cardiometabolic risk score was calculated for each participant as follows:

Continuous Cardiometabolic Risk Score = -zHDL + zInsulin + zGlucose + zTriglycerides + (zBMI + zWC)/2 + (zSBP + zDBP)/2

This cardiometabolic risk score was used as a means of estimating an individual's global cardiometabolic risk. In contrast to a dichotomous metabolic syndrome diagnosis, this approach results in a continuous risk score that increases statistical power, and has been used in several recent investigations in the pediatric population [6,20,21].

Physical activity and sedentary behavior

Objectively measured sedentary behavior and physical activity were assessed using the Actigraph LS 7164 accelerometer (Actigraph, Pensacola, FL, USA) for one week. Participants were instructed to wear the accelerometer on the right hip during all waking hours, except during bathing or aquatic activities such as swimming. Acclerometry data were downloaded as 1-min epochs and were processed using SAS 9.2 (SAS Institute, Cary, NC, USA) and Microsoft Excel (Microsoft, Redmond, WA, USA) according to standardized quality control and data reduction procedures [22]. Non-wear time was defined as at least 60 consecutive minutes of zero counts, with allowance for up to 2 minutes of counts between 0 and 100 [22]. A valid day was defined as

≥ 10 hours of monitor wear time, and only participants with 4 or more valid days (including at least one weekend day) were included in the present analyses. There were no significant differences in any marker of cardiometabolic risk between participants with and without valid accelerometer data (data not shown).

Sedentary behavior was defined as all minutes with an average activity count of less than 100 counts/minute, light physical activity (LPA) as all minutes with an activity count of 100-2296 counts/minute, and moderate-to-vigorous intensity physical activity (MVPA) as any minute with an activity count greater than 2296 counts/minute [23]. A sedentary bout was defined as 1 or more consecutive minutes with less than 100 counts/minute. The number of daily bouts of sedentary time lasting 1-4 minutes, 5-9 minutes, 10-14 minutes, 15-29 minutes, and 30+ minutes were calculated for each participant. Breaks in sedentary time were calculated as any interruption in sedentary time lasting one minute or longer in which the accelerometer counts per minute rose up to or above 100 [15]. Daily television (TV) viewing, and leisure time computer/video game use (surfing the internet, playing video games on a computer or other device, etc.) were assessed using self-report questionnaires. Participants were asked how many hours they spent watching TV and using the computer for fun on weekdays and weekend days, and a mean score over the 7 days was computed. These questions are similar to those used in the Youth Risk Behavior Survey, and have been shown to be valid and reliable in the pediatric age group [24].

Covariates

Sexual maturation was assessed by a research nurse and was scored from 1 (pre-pubertal) to 5 (adult) according to Tanner stages [25,26]. Ten percent of boys and 35% of girls had a Tanner stage of 2 or higher, indicating that they had begun puberty. Baseline questionnaires ascertained highest educational level of the parents (high school, pre-university level [Collège

d'enseignement général et professionnel for Quebec], university) and total annual family income (categorized into 12 groups ranging from <$10,000 to $140,000 CAD or more).

Statistical Analyses

Sex-by-sedentary behavior interactions were investigated for all outcomes of interest. Significant sex interactions were observed for waist circumference, BMI Z-score, glucose, insulin, and hs-CRP, therefore all analyses have been performed in boys and girls separately. Fasting insulin and plasma triglycerides were non-normally distributed and were therefore transformed using a Box-Cox transformation prior to their inclusion in statistical analyses.

Independent t-tests were performed to assess differences in behavioral and cardiometabolic risk factors between boys and girls. Simple correlations were used to examine the relationship between self-reported and accelerometer-derived sedentary behavior. Regression analyses were performed to determine the associations between sedentary behavior and both the continuous cardiometabolic risk score and individual markers of cardiometabolic risk. Initial models were unadjusted, while subsequent analyses adjusted for accelerometer wear time, age, light and moderate-to-vigorous physical activity, total sedentary time, BMI Z-score (unless included in the outcome), Tanner stage, parental income and level of education. These covariates were chosen as they were associated with multiple markers of cardiometabolic risk in both sexes (all $p<0.05$). Statistical significance was set at a p value of <0.05. All statistical analyses were performed using SAS version 9.2 (SAS Institute, Cary, NC).

Results

Characteristics of study participants are presented in Table 3.1. In comparison to girls, boys were significantly more physically active and spent more time using computers/playing video games in their leisure time (all $p<0.01$). Boys also had higher concentrations of fasting glucose and HDL-

Cholesterol and lower diastolic blood pressure, triglycerides and fasting insulin (all $p < 0.01$). There were no differences between boys and girls in age, objectively measured sedentary time, LPA, self-reported television viewing, continuous cardiometabolic risk score or any anthropometric measurement (all $p > 0.05$). The number of daily sedentary bouts of each length was similar for both sexes. Boys accumulated fewer bouts of sedentary behavior lasting 1-4 minutes ($p<0.05$) while there were no differences between sexes for the number of sedentary bouts lasting 5-9 minutes, 15-29 minutes, or 30+ minutes (all $p>0.05$). Accelerometer-derived sedentary time was positively associated with leisure time computer/video game use in boys only ($r=0.20$, $p=0.008$), but was not associated with self-reported TV viewing in either sex (all $p>0.10$).

Unadjusted Associations

Associations between characteristics of sedentary behavior and markers of cardiometabolic disease risk are presented in Tables 3.2 and 3.3. In boys, the continuous cardiometabolic risk score was positively associated with total sedentary time, the number of sedentary bouts lasting 10-14 minutes, the number of bouts lasting 15-29 minutes, and both TV viewing and leisure time computer/video game use, while it was negatively associated with the number of sedentary bouts lasting 1-4 minutes (all $p < 0.05$). Among girls, the continuous cardiometabolic risk score was positively associated with total sedentary time, sedentary bouts lasting 5-9, 10-14 minutes, and 15-29 minutes, as well as both TV viewing and leisure time computer/video game use (all $p < 0.05$).

Adjusted Associations

In the fully adjusted model, breaks in sedentary time were negatively associated with the continuous cardiometabolic risk score (boys: $\beta = -0.057$, 95% CI= -0.106, -0.008; girls: $\beta = -0.084$, 95% CI= -0.143,-0.024) and BMI Z-scores (boys: $\beta = -0.026$, 95% CI=-0.040, -0.012;

girls: β = -0.032, 95% CI=-0.048, -0.016) in both sexes (all $p<0.05$). Similar associations were also observed for the number of sedentary bouts lasting 1-4 minutes. The number of sedentary bouts lasting 5-9 minutes was negatively associated with waist circumference in girls only (β = -0.355, 95% CI=-0.686, -0.025) ($p<0.05$). The number of sedentary bouts lasting 10-14 minutes was positively associated with fasting glucose in girls (β = 0.078, 95% CI=0.024, 0.133), and with BMI Z-score in boys (β = 0.169, 95% CI=0.035, 0.302). The number of sedentary bouts lasting 15-29 minutes was negatively associated with fasting triglycerides (β = -0.072, 95% CI=-0.140, -0.003) and hs-CRP (β = -0.279, 95% CI=-0.498, -0.060) in boys only (all $p<0.05$). Finally, leisure time computer/video game use was positively associated with continuous cardiometabolic risk (β = 0.485, 95% CI=0.084, 0.886) and waist circumference (β = 0.799, 95% CI=0.141, 1.457), and negatively associated with HDL-cholesterol (β = -0.041, 95% CI=-0.070, -0.012) in boys only, while TV viewing was positively associated with continuous cardiometabolic risk (β = 0.736, 95% CI=0.404, 1.068), waist circumference (β = 0.664, 95% CI=0.153, 1.174) and BMI Z-score in girls only (β = 0.197, 95% CI=0.099, 0.294) (all $p<0.05$).

Discussion

The results of the present study demonstrate that breaks in sedentary time and short bouts of sedentary behavior (e.g. those lasting 1-4 minutes) are associated with reduced cardiometabolic risk and BMI Z-scores in children aged 8-11 independent of total sedentary time and physical activity. These cross-sectional results suggest that children who frequently interrupt their sedentary time may experience lower levels of cardiometabolic risk than those who accumulate sedentary behavior with less frequent interruptions. Markers of cardiometabolic risk were also more closely associated with self-reported leisure time computer/video game use and TV viewing than with objectively measured total sedentary time in this population.

To our knowledge, this is the first study to report a beneficial association between breaks in sedentary time and global cardiometabolic risk in the pediatric population. Healy and colleagues have previously reported that breaks in sedentary time are independently and beneficially associated with adiposity, glucose metabolism, triglyceride levels and hs-CRP in adults [15,16] although recent studies have generally failed to detect similar associations in children and youth [5,11]. Carson and Janssen [5] did not observe any association between breaks in sedentary time and continuous cardiometabolic risk in a representative sample of American children and youth aged 6-19 years. Examining another representative sample Canadian youth aged 6-19 years, Colley et al [11] found that breaks in sedentary time accumulated after 3pm on weekdays were associated with lower waist circumference in boys aged 11-14 years. However, they reported that breaks in sedentary time were not significantly associated with any other outcome in older or younger boys, or in girls of any age.

The explanation for this discrepancy between the present findings and previous investigations in the pediatric age group is not immediately clear. While the present analysis focused on children with a parental history of obesity, previous investigations into the role of breaks in sedentary behavior among children and youth have focused on representative samples of the Canadian [11] and American [5] pediatric populations. Due to differences in study methodology (e.g. participant age range, accelerometer model, etc) it is not possible to directly compare levels of overweight/obesity, markers of cardiometabolic disease risk or MVPA across the three studies. However, it is possible that associations between breaks in sedentary time and cardiometabolic risk may be stronger in the present population with a family history of obesity, as parental obesity has been associated with increased childhood cardiometabolic risk by some [27-29] but not all studies [30]. This difference in study population may help to explain why the present results are more similar to those reported previously by Healy and colleagues in adults [15,16], rather than other investigations in children and youth [5,11].

Several mechanisms have been proposed which could explain the beneficial associations between breaks in sedentary time, short bouts of sedentary time, and continuous cardiometabolic risk observed in the present study. Imposed bouts of prolonged sedentary behavior have been shown to acutely reduce insulin sensitivity and increase triglyceride levels in adults [31], effects which are likely due to reductions in lipoprotein lipase and glucose transport protein activity in skeletal muscle [32, 33]. Similarly, frequent walk breaks have been shown to greatly reduce the acute metabolic impact of prolonged sitting in overweight adults [34]. If the impact of chronic breaks in sedentary time are similar to those observed acutely in adults, this could provide a plausible mechanism linking frequent interruptions in sedentary behavior with lower levels of cardiometabolic disease risk. However, a recent study by Saunders and colleagues failed to detect any acute impact of prolonged sitting, with or without interruptions, on markers of cardiometabolic risk in healthy children and youth [35]. Therefore, given that breaks and short bouts of sedentary behavior were not independently associated with any individual markers of cardiometabolic risk other than BMI Z-score in the present study, it is also possible that excess body weight may simply predispose children toward less frequent interruptions in sedentary time.

The current finding that cardiometabolic risk appears to be more closely associated with self-reported TV viewing and leisure time computer/video game use than with objectively measured sedentary time is consistent with other findings in the pediatric population [5]. As noted recently by Pereira and Power, self-reported sedentary behaviours are poorly understood at present [36]. As a result, the reason for the discrepancy between objective and subjective measures of sedentary behavior in the present study is not clear. Given that self-report measures often differ dramatically from those based on accelerometry [37,38], it is somewhat surprising that it is self-reported sedentary behaviors which are more consistently associated with health risk in the pediatric population. However, it should be noted that self-reported screen time is only able to

assess a single form of sedentary behaviour, while accelerometry provides a global measure of time spent sitting. As noted elsewhere, the two measures are therefore assessing different constructs [39,40]. This point is underscored by the recent findings of Carson and Janssen, who reported a correlation of just 0.08 between self-reported TV viewing and objectively measured sedentary time in a large sample of American children and youth [5].

The present findings suggest that it may be the behaviors children engage in while seated (e.g. increased food intake), rather than the act of sitting *per se*, that most strongly influences the development of cardiometabolic risk in the pediatric age group [39, 41-44]. For example, it has been reported that exposure to both video games [44] and television commercials [43] result in increased *ad libitum* food intake in children and youth. In contrast, sitting passively appears to have no impact on subsequent food intake or other forms of behavioural compensation (39,43-45]. The relationship between screen-based sedentary behaviours and excess food intake may therefore help to explain the associations observed between TV viewing, leisure time computer/video game use, and markers of cardiometabolic disease risk in the present study. More research into the mechanisms linking self-reported and directly measured sedentary behavior with markers of cardiometabolic risk is clearly warranted.

It is interesting to note that the associations between both self-reported and objectively measured aspects of sedentary behaviour appear to be more closely associated with measures of adiposity than with other markers of cardiometabolic disease risk in the present sample. This may be due to the fact that excess adiposity typically precludes the development of cardiometabolic dysfunction in children and youth [46]. For example, it has been reported that just 4% of obese adolescents have type 2 diabetes, whereas greater than 90% of youth with diabetes are overweight or obese [46]. Furthermore, it is known that the duration of obesity is strongly related to the risk of cardiometabolic dysfunction [47]. This may help to explain why sedentary behaviours are

consistently associated with diabetes and cardiovascular disease in adults [48], despite the relatively few significant associations observed for markers of cardiometabolic disease risk in the present study.

The present study includes several strengths and limitations that warrant mention. The present study included objectively measured sedentary time and cardiometabolic risk factors. However, it was also cross-sectional in nature, precluding the determination of causality. Screen-based sedentary behaviours were assessed via self-report, which may have introduced additional error into the current analyses, when compared with more objective measures. Self-report measures have been shown to systematically over-estimate physical activity in children and youth [38], and it is possible that screen-based sedentary behaviours may be similarly over- or underestimated in this population. However, it should be noted that any error or response bias would be likely to bias the associations between screen-based sedentary behaviours and markers of cardiometabolic disease risk towards the null, which underscores the associations observed in the present analyses. It should also be noted that the accelerometer protocol employed by the present study may have resulted in some light physical activities (e.g. standing still) being inadvertently identified as sedentary behavior. Future studies which employ inclinometers may therefore be able to more accurately distinguish between seated and standing activities [49]. These findings are also based on a sample of white youth with a family history of obesity, and therefore may not generalize to all children or to other age groups.

Conclusions

The results of the present study demonstrate that breaks in sedentary time and short bouts of sedentary behavior are independently and beneficially associated with markers of cardiometabolic risk in children with a family history of obesity. These results also suggest that cardiometabolic risk is more closely associated with measures of self-reported leisure time screen time than with

objectively measured sedentary time in this population. Future studies should investigate whether minimizing screen time or introducing frequent interruptions in sedentary time prevent the development of cardiometabolic risk among children with a family history of obesity.

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Tables

Table 3.1 Characteristics of study participants.

	Boy (n=286)	Girl (n=236)	*P value*
Age (years)	9.2 (9.1, 9.3)	9.1 (9.0, 9.2)	0.55
Height (cm)	139.3 (138.4, 140.2)	138.4 (137.3, 139.5)	0.20
Weight (kg)	38.2 (36.9, 39.5)	38.1 (36.7, 39.6)	0.94
BMI (kg/m^2)	19.4 (18.9, 19.9)	19.6 (19.0, 20.1)	0.61
Waist Circumference (cm)	67.6 (66.2, 69.0)	67.3 (65.8, 68.8)	0.82
Sedentary Time (min/day)	363.5 (354.9 372.1)	366.7 (358.1 375.4)	0.61
Number of valid days of accelerometry (days)	6.5 (6.4, 6.6)	6.5 (6.4, 6.6)	0.98
Number of hours of accelerometry data (hours/day)	13.8 (13.7, 13.9)	13.6 (13.5, 13.7)	0.02
LPA (min/day)	403.9 (397.1, 410.6)	409.5 (402.9, 416.1)	0.24
MVPA (min/day)	61.2 (57.8, 64.6)	41.2 (38.8, 43.6)	<0.01
Sedentary Bouts 1-4 Minutes (number/day)	67 (66, 68)	70 (69, 72)	<.01
Sedentary Bouts 5-9 Minutes (number/day)	13 (12, 14)	13 (13, 14)	0.58
Sedentary Bouts 10-14 Minutes (number/day)	4 (4, 5)	4 (4, 5)	0.88
Sedentary Bouts 15-29 Minutes (number/day)	3 (3, 3)	3 (3, 3)	0.92
Sedentary Bouts 30+ Minutes (number/day)	2 (2, 2)	2 (2, 2)	0.22
TV viewing (hours/day)	2.0 (1.8, 2.2)	1.8 (1.6, 2.0)	0.12
Computer/video game use (hours/day)	1.1 (0.9, 1.2)	0.6 (0.5, 0.7)	<0.01
Systolic BP (mmHg)	95 (94, 96)	94 (93, 95)	0.23
Diastolic BP (mmHg)	49 (49, 50)	50 (50, 51)	0.01
Insulin (pmol/L)	30.1 (27.9, 32.3)	38.2 (34.9, 41.5)	<0.01
Glucose (mmol/L)	5.00 (4.96, 5.04)	4.90 (4.85, 4.94)	<0.01
HDL-Cholesterol (mmol/L)	1.22 (1.19, 1.25)	1.16 (1.13, 1.19)	<0.01
Triglycerides (mmol/L)	0.76 (0.72, 0.80)	0.89 (0.84, 0.95)	<0.01
hs-CRP (mg/L)	1.09 (0.82, 1.36)	1.20 (0.92, 1.48)	0.57
Continuous Cardiometabolic Risk Score	0.03 (-0.41, 0.48)	0.03 (-0.47, 0.53)	0.98

Data presented as means (95% confidence intervals).
P values represent sex differences assessed using an independent Student's t-test.
Clustered cardiometabolic risk score was calculated by summing z-scores for insulin, glucose, triglycerides, inverted HDL-cholesterol, blood pressure, BMI, and waist circumference for each participant.
BMI, body mass index; LPA, light intensity physical activity; MVPA, moderate-to-vigorous physical activity; TV, television; BP, blood pressure; HDL, high density lipoprotein; hs-CRP, high sensitivity C-reactive protein.

Table 3.2 Associations of sedentary behavior and physical activity with markers of cardiometabolic risk in boys.

	Continuous Cardiometabolic Risk	WC (cm)	BMI (Z-Score)	Insulin (pmol/L)	Glucose (mmol/L)	HDL-C (mmol/L)	Triglycerides (mmol/L)	hs-CRP (mg/L)
Model 1								
Sedentary Time (min/day)	0.010 (0.004, 0.016)*	0.040 (0.022, 0.059)*	0.002 (0.001, 0.004)*	0.001 (0.001, 0.002)*	0.0001 (-0.0004, 0.001)	-0.0003 (-0.0007, 0.0001)	0.001 (-0.0002, 0.001)	0.004 (0.001, 0.006)*
Breaks in Sedentary Time (number/day)	-0.020 (-0.059, 0.018)	-0.093 (-0.215, 0.030)	-0.006 (-0.017, 0.004)	-0.004 (-0.010, 0.001)	-0.002 (-0.005, 0.002)	-0.001 (-0.004, 0.001)	-0.004 (-0.008, 0.001)	0.002 (-0.014, 0.019)
Sedentary Bouts 1-4 Minutes (number/day)	-0.065 (-0.100, -0.003)*	-0.271 (-0..82, -0.161)*	-0.019 (-0.028, -0.009)*	-0.009 (-0.014, -0.005)*	-0.002 (-0.005, 0.002)	0.001 (-0.002, 0.003)	-0.005 (-0.010, -0.001)*	-0.017 (-0.032, -0.002)*
Sedentary Bouts 5-9 Minutes (number/day)	0.069 (-0.076, 0.214)	0.357 (-0.103, 0.816)	0.010 (-0.028, 0.049)	0.006 (-0.015, 0.026)	-0.004 (-0.018, 0.009)	-0.008 (-0.018, 0.002)	0.003 (-0.015, 0.021)	0.077 (0.014, 0.139)*
Sedentary Bouts 10-14 Minutes (number/day)	0.594 (0.279, 0.908)*	2.526 (1.547, 3.506)*	0.158 (0.074, 0.241)*	0.078 (0.034, 0.121)*	-0.010 (-0.041, 0.020)	-0.026 (-0.048, -0.004)*	0.038 (-0.001, 0.076)	0.219 (0.084, 0.354)*
Sedentary Bouts 15-29 Minutes (number/day)	0.391 (0.059, 0.723)*	2.071 (1.034, 3.107)*	0.122 (0.034, 0.210)*	0.063 (0.017, 0.108)*	0.006 (-0.025, 0.038)	-0.004 (-0.026, 0.019)	0.006 (-0.035, 0.047)	0.126 (-0.016, 0.269)
Sedentary Bouts 30+ Minutes (number/day)	0.6620 (-0.015, 1.256)	2.973 (0.962, 4.984)*	0.217 (0.048, 0.386)*	0.109 (0.021, 0.197)*	-0.006 (-0.065, 0.053)	-0.012 (-0.056, 0.031)	0.049 (-0.029, 0.127)	0.066 (-0.209, 0.341)
TV Viewing (hours/day)	0.465 (0.204, 0.726)*	0.904 (0.068, 1.740)*	0.088 (0.017, 0.160)*	0.041 (0.005, 0.078)*	0.024 (-0.0001, 0.049)	-0.015 (-0.033, 0.003)	0.027 (-0.006, 0.060)	0.130 (0.017, 0.243)*
Computer/Video Game Use (hours/day)	0.687 (0.300, 1.073)*	1.629 (0.394, 2.863)*	0.058 (-0.049, 0.164)	0.066 (0.012, 0.119)*	0.032 (-0.004, 0.068)	-0.043 (-0.069, -0.017)*	0.028 (-0.021, 0.076)	0.173 (0.007, 0.340)*

	Continuous Cardiometabolic Risk	WC (cm)	BMI (Z-Score)	Insulin (pmol/L)	Glucose (mmol/L)	HDL-C (mmol/L)	Triglycerides (mmol/L)	hs-CRP (mg/L)
Model 2								
Sedentary Time (min/day)	0.011 (-0.019, 0.041)	0.038 (-0.012, 0.087)	-0.001 (-0.009, 0.008)	0.001 (-0.003, 0.004)	0.002 (-0.001, 0.005)	-0.001 (-0.003, 0.001)	-0.0004 (-0.004, 0.003)	0.003 (-0.009, 0.015)
Breaks in Sedentary Time (number/day)	-0.057 (-0.106, -0.008)*	-0.027 (-0.110, 0.057)	-0.026 (-0.040, -0.012)*	-0.002 (-0.008, 0.004)	0.001 (-0.005, 0.006)	-0.001 (-0.005, 0.002)	-0.005 (-0.011, 0.001)	0.005 (-0.014, 0.025)
Sedentary Bouts 1-4 Minutes (number/day)	-0.063 (-0.111, -0.015)*	-0.052 (-0.133, 0.030)	-0.028 (-0.041-0.016)*	-0.001 (-0.007, 0.005)	-0.001 (-0.006, 0.004)	-0.001 (-0.004, 0.003)	-0.002 (-0.008, 0.004)	-0.00002 (-0.019, 0.019)
Sedentary Bouts 5-9 Minutes (number/day)	-0.048 (-0.245, 0.148)	0.080 (-0.244, 0.404)	-0.039 (-0.094, 0.015)	-0.007 (-0.030, 0.017)	-0.005 (-0.025, 0.015)	-0.011 (-0.025, 0.003)	0.005 (-0.019, 0.029)	0.047 (-0.029, 0.123)
Sedentary Bouts 10-14 Minutes (number/day)	0.473 (-0.006, 0.952)	0.334 (-0.468, 1.135)	0.169 (0.035, 0.302)*	0.001 (-0.057, 0.060)	-0.041 (-0.091, 0.009)	-0.030 (-0.065, 0.004)	0.018 (-0.041, 0.077)	-0.073 (-0.262, 0.115)
Sedentary Bouts 15-29 Minutes (number/day)	-0.165 (-0.735, 0.405)	-0.721 (-1.653, 0.211)	0.128 (-0.029, 0.285)	-0.033 (-0.101, 0.035)	-0.004 (-0.063, 0.055)	0.026 (-0.015, 0.067)	-0.072 (-0.140, -0.003)*	-0.279 (-0.498, -0.060)*
Sedentary Bouts 30+ Minutes (number/day)	0.321 (-0.411, 1.054)	0.194 (-1.021, 1.409)	0.153 (-0.051, 0.357)	0.038 (-0.050, 0.126)	0.004 (-0.070, 0.077)	0.007 (-0.046, 0.060)	0.017 (-0.072, 0.106)	-0.171 (-0.447, 0.106)
TV Viewing (hours/day)	0.249 (-0.020, 0.519)	-0.176 (-0.623, 0.272)	0.050 (-0.026, 0.125)	0.003 (-0.029, 0.036)	0.020 (-0.008, 0.048)	-0.001 (-0.021, 0.018)	-0.006 (-0.039, 0.027)	0.050 (-0.055, 0.155)
Computer/Video Game Use (hours/day)	0.485 (0.084, 0.886)*	0.799 (0.141, 1.457)*	0.016 (-0.097, 0.128)	0.031 (-0.017, 0.079)	0.013 (-0.028, 0.055)	-0.041 (-0.070 -0.012)*	0.015 (-0.034, 0.064)	0.145 (-0.011, 0.300)

Model 1. Unadjusted analyses.

Model 2. Adjusted for accelerometer wear time, age, light and moderate-to-vigorous physical activity, total sedentary time (except when exposure), BMI Z-score (except when included in outcome), Tanner stage, parental income and level of education. Data are presented as beta coefficients (95% confidence intervals). n=286.

Associations assessed using linear regression analysis. Data are presented as beta coefficients (95% confidence intervals). n=286.

*=$p<0.05$

Fasting insulin and plasma triglycerides have been transformed using a Box-Cox transformation.

Continous cardiometabolic risk score was calculated by summing z-scores for insulin, glucose, triglycerides, negative HDL-cholesterol, blood pressure, BMI, and waist circumference for each participant.

BMI, body mass index; HDL-C, high density lipoprotein cholesterol; hs-CRP, high sensitivity c-reactive protein; MVPA, moderate-to-vigorous physical activity; TV, television; WC, waist circumference.

Table 3.3 Associations of sedentary behavior and physical activity with markers of cardiometabolic risk in girls.

	Continuous Cardiometabolic Risk	WC (cm)	BMI (Z-Score)	Insulin (pmol/L)	Glucose (mmol/L)	HDL-C (mmol/L)	Triglycerides (mmol/L)	hs-CRP (mg/L)
Model 1								
Sedentary Time (min/day)	0.010 (0.002, 0.017)*	0.034 (0.013, 0.056)*	0.001 (-0.001, 0.003)	0.020 (0.001, 0.003)*	0.001 (-0.00004, 0.0010)	-0.0002 (-0.001, 0.0002)	0.0001 (-0.001, 0.001)	-0.001 (-0.004, 0.002)
Breaks in Sedentary Time (number/day)	0.013 (-0.039, 0.065)	-0.020 (-0.177, 0.137)	-0.009 (-0.023, 0.005)	0.004 (-0.004, 0.012)	0.004 (-0.001, 0.009)	0.001 (-0.002, 0.004)	-0.00004 (-0.006, 0.006)	-0.009 (-0.030, 0.011)
Sedentary Bouts 1-4 Minutes (number/day)	-0.016 (-0.064, 0.032)	-0.124 (-0.266, 0.017)	-0.009 (-0.022, 0.004)	-0.004 (-0.011, 0.004)	-0.00001 (-0.004, 0.004)	0.001 (-0.002, 0.004)	0.0004 (-0.005, 0.006)	-0.005 (-0.024, 0.014)
Sedentary Bouts 5-9 Minutes (number/day)	0.211 (0.031, 0.391)*	0.527 (-0.009, 1.063)	0.024 (-0.026, 0.074)	0.043 (0.016, 0.071)*	0.012 (-0.004, 0.029)	-0.004 (-0.015, 0.007)	0.001 (-0.021, 0.022)	0.013 (-0.058, 0.085)
Sedentary Bouts 10-14 Minutes (number/day)	0.510 (0.120, 0.899)*	1.025 (-0.141, 2.191)	-0.016 (-0.124, 0.092)	0.087 (0.027, 0.146)*	0.052 (0.017, 0.087)*	-0.016 (-0.039, 0.008)	0.012 (-0.034, 0.058)	-0.057 (-0.211, 0.097)
Sedentary Bouts 15-29 Minutes (number/day)	0.413 (0.012, 0.814)*	1.512 (0.338, 2.686)*	0.017 (-0.093, 0.126)	0.086 (0.025, 0.146)*	0.026 (-0.010, 0.062)	-0.012 (-0.036, 0.012)	-0.006 (-0.053, 0.040)	-0.048 (-0.206, 0.110)
Sedentary Bouts 30+ Minutes (number/day)	0.442 (-0.314, 1.198)	1.346 (-0.906, 3.597)	0.033 (-0.174, 0.241)	0.130 (0.015, 0.246)*	0.012 (-0.056, 0.080)	0.014 (-0.032, 0.059)	0.044 (-0.044, 0.133)	-0.248 (-0.544, 0.047)
TV Viewing (hours/day)	0.774 (0.475, 1.072)*	2.458 (1.557, 3.359)*	0.176 (0.090, 0.261)*	0.099 (0.053, 0.145)*	0.023 (-0.005, 0.051)	-0.030 (-0.048, -0.011)*	0.050 (0.014, 0.087)*	0.196 (0.074, 0.319)*
Computer/Video Game Use (hours/day)	0.902 (0.314, 1.490)*	3.215 (1.456, 4.974)*	0.191 (0.026, 0.356)*	0.133 (0.044, 0.222)*	0.010 (-0.043, 0.064)	-0.037 (-0.073, -0.001)*	0.058 (-0.011, 0.127)	0.203 (-0.033, 0.440)

	Continuous Cardiometabolic Risk	WC (cm)	BMI (Z-Score)	Insulin (pmol/L)	Glucose (mmol/L)	HDL-C (mmol/L)	Triglycerides (mmol/L)	hs-CRP (mg/L)
Model 2								
Sedentary Time (min/day)	0.014 (-0.353, 0.957)	-0.015 (-0.056, 0.027)	0.005 (-0.003, 0.014)	-0.0001 (-0.003, 0.003)	-0.0001 (-0.003, 0.003)	-0.0001 (-0.002, 0.002)	0.001 (-0.003, 0.004)	-0.006 (-0.016, 0.005)
Breaks in Sedentary Time (number/day)	-0.084 (-0.143, -0.024)*	-0.012 (-0.102, 0.079)	-0.032 (-0.049, -0.015)*	-0.001 (-0.008, 0.007)	0.002 (-0.004, 0.008)	0.0001 (-0.003, 0.004)	-0.002 (-0.010, 0.005)	0.009 (-0.014, 0.031)
Sedentary Bouts 1-4 Minutes (number/day)	-0.097 (-0.153, -0.041)*	-0.066 (-0.152, 0.019)	-0.032 (-0.048, -0.016)*	-0.004 (-0.010, 0.003)	-0.001 (-0.006, 0.005)	-0.0003 (-0.004, 0.003)	-0.003 (-0.010, 0.004)	0.008 (-0.013, 0.029)
Sedentary Bouts 5-9 Minutes (number/day)	0.041 (-0.192, 0.274)	-0.355 (-0.686, -0.025)*	0.020 (-0.047, 0.087)	0.004 (-0.023, 0.031)	0.002 (-0.020, 0.024)	0.004 (-0.010, 0.018)	-0.009 (-0.036, 0.018)	0.012 (-0.072, 0.096)
Sedentary Bouts 10-14 Minutes (number/day)	0.484 (-0.106, 1.073)	-0.603 (-1.451, 0.245)	-0.010 (-0.181, 0.162)	0.017 (-0.053, 0.086)	0.078 (0.024, 0.133)*	-0.026 (-0.062, 0.009)	0.049 (-0.020, 0.118)	-0.017 (-0.230, 0.196)
Sedentary Bouts 15-29 Minutes (number/day)	0.512 (-0.209, 1.233)	-0.073 (-1.107, 0.962)	0.157 (-0.049, 0.363)	-0.028 (-0.112, 0.056)	-0.001 (-0.069, 0.067)	-0.020 (-0.064, 0.023)	0.012 (-0.071, 0.096)	-0.067 (-0.328, 0.194)
Sedentary Bouts 30+ Minutes (number/day)	-0.260 (-1.112, 0.592)	0.241 (-0.980, 1.463)	-0.061 (-0.308, 0.185)	0.043 (-0.057, 0.144)	-0.032 (-0.111, 0.046)	0.043 (-0.008, 0.093)	0.062 (-0.038, 0.162)	-0.240 (-0.542, 0.062)
TV Viewing (hours/day)	0.736 (0.404, 1.068)*	0.664 (0.153, 1.174)*	0.197 (0.099, 0.294)*	0.016 (-0.026, 0.058)	0.005 (-0.028, 0.039)	-0.009 (-0.030 0.013)	0.030 (-0.012, 0.072)	0.108 (-0.020, 0.235)
Computer/Video Game Use (hours/day)	0.560 (-0.076, 1.197)	0.548 (-0.373, 1.468)	0.141 (-0.043, 0.325)	0.021 (-0.054, 0.096)	0.006 (-0.055, 0.066)	-0.025 (-0.064, 0.013)	0.020 (-0.055, 0.095)	0.105 (-0.126, 0.335)

Model 1. Unadjusted analyses.

Model 2. Adjusted for accelerometer wear time, age, light and moderate-to-vigorous physical activity, total sedentary time (except when exposure), BMI Z-score (except when included in outcome), Tanner stage, parental income and level of education. Data are presented as beta coefficients (95% confidence intervals). n=286.

Associations assessed using linear regression analysis.

Associations assessed using linear regression analysis. Data are presented as beta coefficients (95% confidence intervals). n=236.

*=p<0.05

Fasting insulin and plasma triglycerides have been transformed using a Box-Cox transformation.

Continuous cardiometabolic risk score was calculated by summing z-scores for insulin, glucose, triglycerides, negative HDL-cholesterol, blood pressure, BMI, and waist circumference for each participant.

BMI, body mass index; HDL-C, high density lipoprotein cholesterol; hs-CRP, high sensitivity c-reactive protein; MVPA, moderate-to-vigorous physical activity; TV, television; WC, waist circumference.

Chapter 4 - Study 2: Movement variability is associated with clustered cardiometabolic disease risk in American youth.

The following article was originally submitted to the journal Diabetes Care, and has been formatted according to their requirements. Data from this article has been submitted for presentation at the Canadian Society for Exercise Physiology Annual Scientific Meeting in October, 2013.

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TJS, VC and MST designed the current analysis. TJS conducted the statistical analyses and wrote the manuscript. All authors helped revise the manuscript. TJS had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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Abstract

Objective: Both the intensity and volume of physical activity are associated with reduced health risk, while the opposite may be true for prolonged bouts of sedentary behavior. This suggests that movement variability, defined as minute-to-minute changes in accelerometer counts per minute, may also be associated with cardiometabolic disease risk. The purpose of this study was to determine whether movement variability was independently associated with markers of cardiometabolic disease risk in a representative sample of American youth aged 12-17 years.

Research Design and Methods: This study included 1460 adolescents from the 2003/04 and 2005/06 National Health and Nutrition Examination Surveys. Physical activity, sedentary behaviour, and movement variability (defined as minute-to-minute changes in accelerometer counts) were measured over 7 days using accelerometry. Outcomes included waist circumference, body mass index Z-score, fasting insulin, fasting glucose, triglycerides, HDL- and LDL-cholesterol, systolic and diastolic blood pressure, and a clustered cardiometabolic disease risk score.

Results: Participants in the highest tertile of movement variability were characterized by relatively low levels of sedentary time and high levels of moderate and vigorous physical activity and breaks in sedentary time (all $p<0.05$). Movement variability was negatively associated with clustered cardiometabolic disease risk and systolic blood pressure in both sexes independent of physical activity, sedentary time and other covariates (all $p<0.05$).

Conclusions: These findings provide evidence that movement variability is independently associated with clustered cardiometabolic disease risk in American youth. Therefore, in addition to targeting increases in physical activity and decreases in sedentary time, frequent changes in movement intensity may be important for optimal health among youth.

Introduction

Whether accumulated sporadically or in bouts lasting several minutes, moderate and vigorous physical activity (MVPA) is consistently associated with reduced cardiometabolic disease risk in youth (1–3). Further, the benefits of a given volume of physical activity appear to increase with exercise intensity (2,4–8). Recent findings suggest that even short bouts of vigorous intensity physical activity are likely to have a positive impact on adiposity, cardiorespiratory fitness, blood pressure, vascular function, and insulin sensitivity (5–8). These findings suggest that intense bouts of activity, even when brief in duration, have an important impact on fitness and health.

In contrast to MVPA, emerging research suggests that sedentary behavior (activities that involve sitting or reclining while expending ≤ 1.5 metabolic equivalents(9)) may be independently associated with *increased* health risk in youth (10–13). Furthermore, research among adults suggests the manner in which sedentary behavior is accumulated may also influence health risk. In particular, prolonged bouts of uninterrupted sedentary behavior appear to have a rapid and deleterious impact on health (14,15), while interruptions in sedentary time are beneficially associated with body weight, abdominal fat, triglycerides and glucose metabolism in adults (15–17) and with waist circumference in boys (18).

The above findings suggest that movement variability, defined as minute-to-minute changes in accelerometer counts per minute (CPM), may account for variation in cardiometabolic disease risk beyond that accounted for by MVPA and sedentary time. In comparison to individuals with low levels of movement variability, individuals with high movement variability are likely to accumulate higher amounts of MVPA, and lower amounts of sedentary behavior. For a given volume of sedentary behavior and MVPA, an individual with a high level of movement variability is also likely to have a greater number of breaks in sedentary time, and higher average physical activity intensity (See Figure 4.1). Furthermore, it has recently been proposed that with

respect to various biological processes, variability *per se* may also be beneficial to health (19–23). For example, mechanical ventilators that include random variations in breathing rate and volume result in improved oxygen saturation and organ function, when compared to ventilators that provide constant output (19,23). To date no studies have examined the relationship between movement variability and cardiometabolic disease risk in any population.

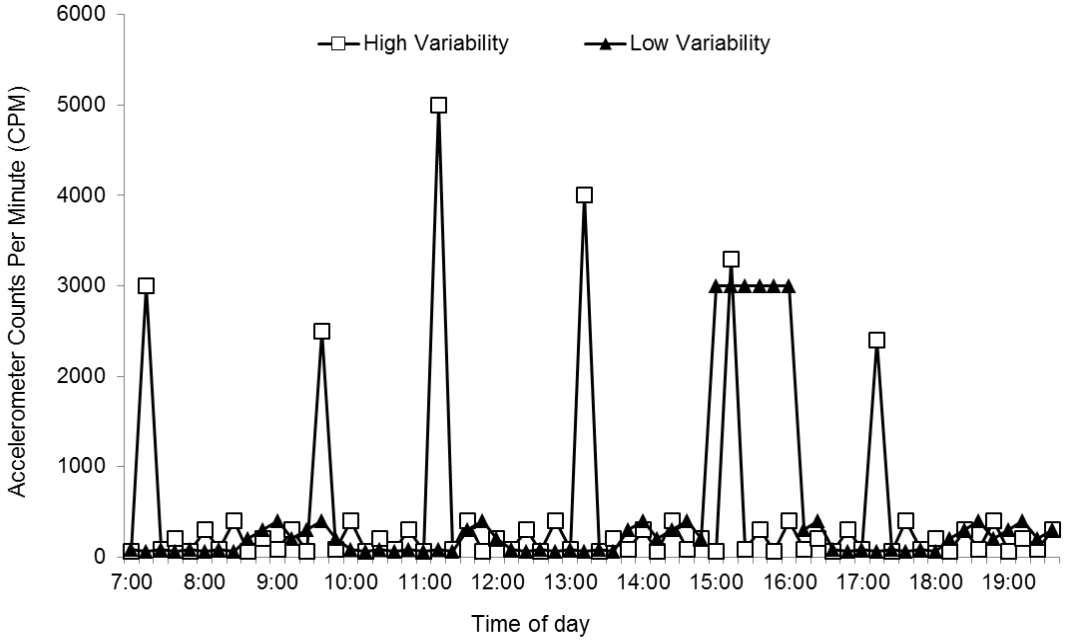

Figure 4.1 Examples of high and low movement variability.

Both individuals have the same amount of total MVPA and sedentary time, although the high variability individual has a cumulative variability of 51080 CPM, while the low variability individual has a cumulative variability 10180 CPM.

The purpose of the present study was to investigate the relationship between minute-to-minute movement variability and markers of cardiometabolic disease risk in a representative sample of American youth aged 12-17 years. We hypothesized that movement variability would be associated with improved cardiometabolic disease risk independent of MVPA and sedentary behavior in this population.

Research Design and Methods

Participants

The present study is based on the 2003-2004 and 2005-2006 cycles of the Nutrition Health and Nutrition Examination Survey (NHANES), a nationally representative cross-sectional survey of Americans, which includes both a detailed health interview and a series of direct physical measures. The study was approved by the National Center for Health Statistics, and consent was obtained from all participants and their parents/guardians. A total of 3428 participants aged 12-17 took part in the 2003-2004 and 2005-2006 iterations of the NHANES.

Measurement of Physical Activity, Sedentary Behavior, and Movement Variability

Physical activity, sedentary behavior, and movement variability were collected using the Actigraph AM-7124 accelerometer (Actigraph, Ft. Walton Beach, FL), which participants were asked to wear on their right hip for 7 consecutive days except when sleeping or engaging in aquatic activities such as swimming or bathing. Acclerometry data were downloaded as 1-min epochs and were processed using SAS 9.2 (SAS Institute, Cary, NC, USA) according to standardized quality control and data reduction procedures (24). Non-wear time was defined as at least 60 consecutive minutes of zero counts, with allowance for 1 to 2 minutes of counts between 0 and 100 (24). A valid day was defined as ≥10 hours of monitor wear time, and only participants with 4 or more valid days, including at least 1 weekend day, were included in the present analyses. Time spent in moderate (≥ 4 metabolic equivalents) and vigorous intensity physical activity (≥ 7 metabolic equivalents) was determined using age-specific accelerometer cut-points (25,26). Sedentary behavior was defined as < 100 counts per minute (CPM). A break in sedentary time was defined as any episode lasting 1 minute or longer where the accelerometer count rose to 100 CPM or higher (16). MVPA, VPA, sedentary time, breaks in sedentary time,

and movement variability were adjusted by wear time by standardizing the variables using the residuals obtained when regressing the variables on wear time (27).

Movement variability was defined as the absolute minute-to-minute variability in accelerometer CPM, for all wear-time on valid days. For example, consecutive minutes with accelerometer counts of 85, 2000 and 150 CPM, have a cumulative variability score of 3765 CPM ((|2000-85|) + (|150-2000|)). Variability scores were summed for each valid day, and an average daily variability score was calculated for each participant.

Outcome Measures

All markers of cardiometabolic disease risk, including anthropometric measures, fasting insulin and glucose, triglycerides, HDL- and LDL-cholesterol, and blood pressure were taken by trained personnel at the mobile examination center visit (28). Weight was assessed using a Toledo digital scale (Mettler-Toledo, LLC, Columbus, OH) while participants wore a paper gown. Height was assessed using a fixed stadiometer. BMI was calculated by dividing body mass (kg) by height in meters squared, and converted to a BMI Z-score based on values published by the CDC (29). Waist circumference was made at the level of the iliac crest following a normal expiration.

HDL-cholesterol was measured using the direct HDL immunoassay method, while total cholesterol and triglycerides were measured enzymatically. LDL-cholesterol was calculated from measured values of total cholesterol, triglycerides, and HDL-cholesterol according to the Friedewald equation (30). Glucose and insulin measurements differed slightly between 2003-2004 and 2005-2006; glucose was assessed via the hexokinase method using the Roche Cobas Mira (F. Hoffmann-La Roche Ltd, Basel, Switzerland) in 2003-2004, and with the Roche/Hitachi 911 in 2005-2006. Insulin was analyzed via immunoenzymometric assay in 2003-2004 and via ELISAin 2005-2006 (31). A validated regression equation was used to convert values from

2005-2006 to those which were directly comparable to values from 2003-2004 (31). Blood pressure was measured manually four consecutive times on the right arm while seated, and the average blood pressure was calculated after excluding the first reading, except when only one reading was taken (n=86 for systolic blood pressure and n=113 for diastolic blood pressure).

Calculation of Clustered Cardiometabolic Disease Risk Score

An age and sex-specific clustered cardiometabolic disease risk score was calculated for each participant as follows:

Clustered Cardiometabolic Disease Risk Score = -zHDL + zInsulin + zGlucose + zTriglycerides + (zBMI + zWC)/2 + (zSBP + zDBP)/2 (32)

This clustered risk score was used as a means of estimating an individual's global cardiometabolic disease risk, and is based broadly on the metabolic syndrome (32).

Covariates

Age, sex, self-ascribed ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, other), poverty-to-income ratio (a measure of socioeconomic status (SES), calculated as the ratio between family income and poverty threshold), smoking status, and energy intake were included as covariates. Ethnicity was self-reported by participants, and was included because previous research has observed significant interactions between sedentary behavior and ethnicity in the past (17). Smoking was assessed by asking participants "Have you ever tried cigarette smoking, even 1 or 2 puffs?", and participants were dichotomized to categories of "yes" or "no". Energy intake was assessed by 24-hour recall.

Statistical Analyses

Insulin, glucose, triglycerides, waist circumference, MVPA, vigorous physical activity (VPA), and breaks in sedentary time were non-normally distributed, and therefore log transformed prior

to inclusion as the outcome in any statistical analysis. Sex-by-variability and ethnicity-by-variability interactions were investigated for all outcomes of interest. We observed significant sex interactions but not ethnicity interactions for clustered risk, HDL-Cholesterol and triglycerides; therefore, all analyses have been performed in boys and girls separately. T-tests were used to assess differences in movement variability between boys and girls. Linear regression analyses were used to examine the association between movement variability and markers of cardiometabolic disease risk after adjustment for age, ethnicity, SES, energy intake, smoking, and waist circumference (when not an outcome) (Model 1), and after additional adjustment for sedentary behavior and MVPA (Model 2). All regression results are presented as the change in the outcome per 10 000 CPM increase in movement variability.

Participants were also divided into sex-specific tertiles of movement variability. Initial ANCOVAs were used to examine whether MVPA, VPA, sedentary behavior or breaks in sedentary time differed by tertile of movement variability, with adjustment for age, ethnicity, SES, energy intake and smoking status. Subsequent ANCOVAs were performed to assess whether the clustered cardiometabolic disease risk score varied according to tertiles of movement variability, after adjustment for age, ethnicity, SES, energy intake, smoking, sedentary behavior and MVPA. A Bonferroni correction was used to adjust for multiple comparisons in post hoc tests following ANCOVAs to assess differences between individual tertiles.

Data are presented as means and 95% confidence intervals (CI) unless otherwise noted. Statistical significance was set at a p value of <0.05. All statistical analyses were performed using SAS version 9.2 (SAS Institute, Cary, NC) and accounted for the complex design and sample weights of NHANES. Due to missing data and significant differences in age and ethnicity between included and excluded participants, sample weights were re-weighted for non-response to achieve a representative sample.

Results

Subject characteristics are presented in Table 4.1. Full accelerometry and non-fasting blood sample data were available for 1460 participants (707 girls, 753 boys), however only 656 participants (314 girls, 342 boys) had full data for fasting blood samples (insulin, glucose, triglycerides, and LDL-Cholesterol). Thus analyses involving these outcomes and the clustered risk score have a sample of 656 participants, while analyses involving non-fasting outcomes (body mass index (BMI), waist circumference, HDL-Cholesterol, and systolic and diastolic blood pressure) include the full sample of 1460.

Approximately 52% of the sample was male, with an average age of 14.9 years. Participants spent 57% of their time engaging in sedentary behavior, and 3% engaging in MVPA. On average, participants accumulated 276976 CPM of movement variability during each valid day, with boys accumulating significantly more than girls (Girls: 245747 (238025, 253469); Boys: 306911 (296630, 317193), $p< 0.0001$).

Physical activity and sedentary behavior across tertiles of movement variability are presented in Table 4.2. In both sexes MVPA, VPA and breaks in sedentary time significantly increased across tertiles of movement variability, while sedentary time significantly decreased (all $p<0.0001$).

The associations between movement variability and markers of cardiometabolic disease risk are presented in Table 4.3. In both sexes, movement variability was associated with reduced clustered risk and systolic blood pressure independent of MVPA, sedentary time, and other covariates (all $p<0.05$). Movement variability was also independently associated with reduced diastolic blood pressure in girls, reduced fasting glucose and insulin in boys, and increased BMI in boys (all $p<0.05$). After back-transforming glucose and insulin from the log scale in boys, an

additional 10 000 CPM of movement variability was associated with a 0.1 (0.0, 0.2)% lower fasting glucose level, and with a 1.9 (0.8, 3.0)% lower fasting insulin.

The association between tertiles of movement variability and clustered risk is shown in Figure 4.2. There was a significant trend for reduced clustered risk with increasing tertiles of movement variability independent of MVPA, sedentary time, and other covariates (all $p<0.05$) in both sexes. Clustered risk was significantly reduced in the highest tertile of movement variability compared to the lowest tertile in both boys and girls, while risk was also reduced in the middle tertile for boys only (all $p<0.05$).

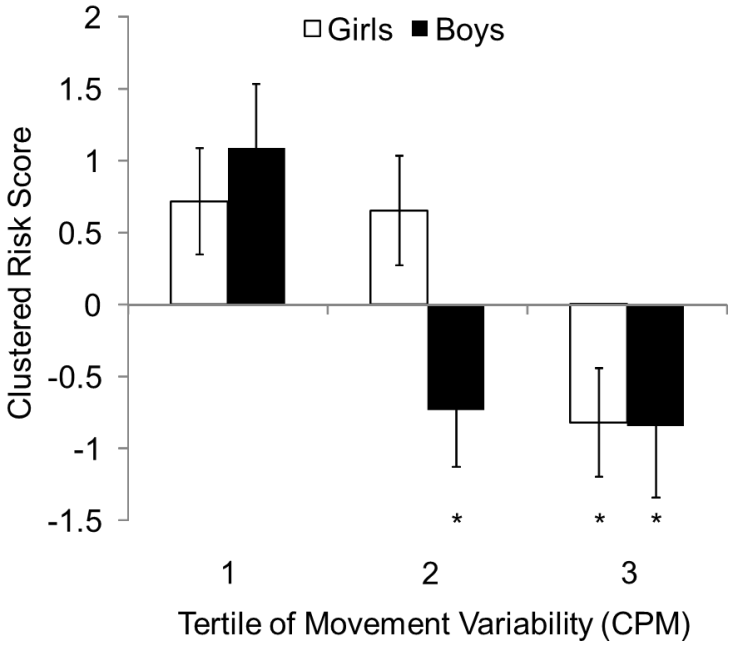

Figure 4.2 Clustered cardiometabolic disease risk across tertiles of movement variability.

Associations assessed using ANCOVAs with adjustment for age, ethnicity, SES, energy intake and smoking status. Data are presented as mean ± standard error.
***=significantly different from Tertile 1 of same sex, $p<0.05$.**

Conclusions

The results of the present study support our hypothesis, and demonstrate that total movement variability is beneficially associated with clustered cardiometabolic disease risk in youth independent of covariates including energy intake, MVPA, and sedentary behavior. These findings also illustrate that individuals with high levels of movement variability are characterized by relatively low levels of sedentary behavior, and high amounts of MVPA, VPA, and breaks in sedentary time. These findings suggest that youth with high levels of variability in their movement patterns are likely to experience lower clustered risk than participants with lower levels of movement variability.

This is the first study to investigate the relationship between movement variability and cardiometabolic disease risk in any population, and therefore makes a novel contribution to the literature. However, the clinical and public health significance of these findings is not yet clear. These results suggest that a 100 000 CPM increase in daily movement variability could reduce clustered metabolic risk by approximately 1.5 units in boys, and by 1 unit in girls. To put this in perspective, this could be achieved by just 50 transitions from sedentary behavior to light physical activity, or an equivalent number of transitions from light to moderate activity. This increase in variability might be most easily achieved by activities which include alternations of high and low intensity movement, such as soccer or basketball (33,34). Guagliano and colleagues (34) report that during basketball, soccer and netball games, adolescent girls spend roughly 40% of their time being sedentary, 30% engaging in light activity, and 15% in both MPA and VPA. A similar range of movement behaviors have been reported during soccer games involving adolescent boys (33). It is conceivable that household chores such as gardening or sweeping could also contribute to increased variability (35).

The present findings suggest that for a given energy expenditure, interventions that produce greater increases in total movement variability may bring about greater health benefits in youth. Future research should investigate the health impact of variability *per se*, and examine whether different methods of increasing movement variability (e.g. frequent breaks in sedentary time vs increased time spent in variable MVPA) bring about comparable improvements in cardiometabolic health. If interventions targeting movement variability are shown to be efficacious in improving cardiometabolic health in at-risk youth, this could lead to interventions which are more feasible and attractive to non-exercisers than traditional programs focusing on structured bouts of MVPA.

There are a number of mechanisms that could explain the results observed in the present study. Variability is a key component of all forms of interval training – by repeated exposure to brief but intense stressors, an individual is exposed to a greater total load than would be possible if the intensity were consistent over time (5). Similar effects are likely to contribute to the beneficial associations observed in the present study. Movement variability was associated with reduced sedentary time, increased breaks in sedentary time, and increased volume of both MVPA and VPA – all factors that have been individually linked with improved health. This suggests that individuals with high levels of movement variability may benefit from the same mechanisms associated with these other movement behaviors, including increased lipoprotein lipase and glucose transport protein activity (36,37), increased mitochondrial capacity (5) and vascular function (5,37). Future research is needed to investigate the mechanisms underlying the relationships observed in the present study, and to determine whether there are benefits related to movement variability *per se*, or whether it is simply a proxy measure for other important movement behaviors.

It has recently been argued that with respect to various forms of biological stimuli, variability *per se* may be beneficial to health (19–23). For example, it has been shown that randomly varying the rate and volume of breaths provided by a mechanical ventilator results in enhanced gas exchange and reduced stress in the lung, heart and brain when compared to ventilators that provide breaths at a constant rate and volume (19,23). Further, it has been noted that disease states such as Parkinson's disease (gait patterns) and congestive heart failure (heart rate variability) are characterized by dramatic reductions in variability (21). Finally, some have suggested that variations in energy intake via intermittent fasting may lead to improvements in markers of cardiometabolic disease risk in adults (20,22). Interestingly, children living in traditional lifestyles, such as Old Order Amish and Mennonite communities, appear to have greater variability in their movement profiles than children living a contemporary lifestyle (38). Given the current findings and those cited above, it is plausible that total movement variability may have a direct influence on markers of cardiometabolic disease risk in youth.

The present study has several important strengths and limitations that warrant mention. Strengths include the objective measurement of key movement variables and markers of cardiometabolic disease risk. Our study also included a large and representative sample of American youth aged 12-17. Limitations include the cross-sectional design, which precludes determinations of causality. Further, several important confounders included in the present study (SES, smoking status, and energy intake) were self-reported, which may have resulted in residual confounding (39,40).

In conclusion, the present study provides evidence that movement variability is associated with improved clustered cardiometabolic disease risk as well as physical activity and sedentary behavior profiles in American youth. These results suggest that independent of MVPA and total sedentary time, increases in movement variability may result in improved health in this age

group. Further research is needed to investigate the mechanisms underlying these relationships, and to determine whether increased movement variability *per se* is sufficient to improve health in this population.

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Tables

Table 4.1 Subject characteristics.

Variables	Total (N=1460)
Age (years)	14.9 (14.8, 15.0)
Sex (%)	
Male	51.6 (753/1460)
Female	48.4 (707/1460)
Race (%)	
Non-Hispanic white	23.1 (337/1460)
Non-Hispanic black	35.1 (512/1460)
Hispanic	38.4 (561/1460)
Other	3.4 (50/1460)
Accelerometer Derived Variables*	
Total wear time (minutes/day)	860 (849, 871)
Sedentary behavior (minutes/day)	488 (479, 498)
Moderate and vigorous physical activity (minutes/day)	29 (26, 32)
Vigorous physical activity (minutes/day)	5 (3, 6)
Breaks in sedentary time (number/day)	98 (97, 100)
Movement variability (counts/minute)	276976 (269637, 284315)
Markers of cardiometabolic disease risk	
BMI (kg/m^2)	22.6 (22.2, 23.0)
Waist Circumference (cm)	78.9 (77.8, 79.9)
Insulin (pmol/L)	66 (60, 72)
Glucose (mmol/L)	5.05 (5.00, 5.11)
Triglycerides (mmol/L)	0.93 (0.86, 1.01)
HDL-Cholesterol (mmol/L)	1.36 (1.34, 1.39)
LDL-Cholesterol (mmol/L)	2.29 (2.20, 2.38)
Systolic Blood Pressure (mmHg)	108 (107, 109)
Diastolic Blood Pressure (mmHg)	59 (58, 60)

Continuous data are presented as mean (95% confidence interval). Percentages are presented as percent (numerator/denominator).
*Corrected for wear time using the residuals method

Table 4.2 Physical activity and sedentary behavior across tertiles of movement variability.

	Tertile 1	Tertile 2	Tertile 3	*p* for trend
Females				
Movement variability (counts/min)	181354 (177470, 185239)	242329 (238467, 246191)	326946 (315218, 338675)	
MVPA (mins/day)	10 (8, 11)[*]	19 (16, 21)[†]	34 (31, 37)[‡]	<0.001
VPA (mins/day)	2 (0, 4)[*]	2 (1, 3)[†]	5 (4, 6)[‡]	<0.001
Sedentary time (mins/day)	550 (535, 565)[*]	518 (500, 536)[†]	459 (439, 479)[‡]	<0.001
Breaks in sedentary time (number/day)	96 (94, 97)[*]	96 (95, 97)[*]	99 (98, 100)[‡]	<0.001
Males				
Movement variability (counts/min)	212089 (202663, 221515)	305792 (301729, 309855)	414946 (401964, 427928)	
MVPA (mins/day)	20 (13, 28)[*]	32 (29, 36)[†]	64 (56, 72)[‡]	<0.001
VPA (mins/day)	4 (0, 8)[*]	4 (3, 4)[*]	11 (7, 16)[‡]	<0.001
Sedentary time (mins/day)	530 (509, 552)[*]	461 (444, 478)[†]	399 (380, 419)[‡]	<0.001
Breaks in sedentary time (number/day)	97 (93, 102)[*]	97 (97, 99)[*]	105 (101, 110)[‡]	0.0002

Associations assessed using ANCOVAs with adjustment for age, ethnicity, SES, energy intake and smoking status. Data are presented as mean (95% confidence interval). n=1460.
Columns with different superscript symbols are significantly different, $p<0.05$.
Although raw values are displayed in the above table, log transformed MVPA, VPA, and breaks in sedentary time were included in statistical analyses.
MVPA: moderate and vigorous physical activity; VPA: vigorous physical activity

Table 4.3 Associations between movement variability and markers of cardiometabolic disease risk in participants aged 12-17 years.

Outcomes	Model 1	Model 2
Females		
Clustered Risk	-0.063 (-0.114, -0.012)*	-0.072 (-0.124, -0.020)*
BMI z-Score	-0.000 (-0.020, 0.010)	-0.000 (-0.020, 0.010)
Log transformed waist circumference	-0.001 (-0.005, 0.002)	-0.003 (-0.008, 0.001)
Log transformed insulin	-0.009 (-0.022, 0.004)	-0.009 (-0.023, 0.004)
Log transformed glucose	-0.000 (-0.002, 0.002)	0.000 (-0.002, 0.002)
Log transformed triglycerides	-0.002 (-0.016, 0.011)	-0.003 (-0.017, 0.011)
HDL-Cholesterol (mmol/L)	0.002(-0.004, 0.007)	0.001(-0.005, 0.008)
LDL-Cholesterol (mmol/L)	0.007 (-0.010, 0.023)	0.005(-0.010, 0.021)
Systolic Blood Pressure (mmHg)	-0.261 (-0.411, -0.110)*	-0.262 (-0.442, -0.080)*
Diastolic Blood Pressure (mmHg)	-0.184 (-0.348, -0.021)*	-0.102 (-0.289, 0.086)
Males		
Clustered Risk	-0.091 (-0.171, -0.011)*	-0.119 (-0.193, -0.046)*
BMI z-Score	0.010 (0.000, 0.010)*	0.010 (0.000, 0.010)*
Log transformed waist circumference	-0.001 (-0.004, 0.001)	-0.001 (-0.004, 0.001)
Log transformed insulin	-0.010 (-0.020, 0.000)	-0.019 (-0.030, -0.008)*
Log transformed glucose	-0.000 (-0.001, 0.001)	-0.001 (-0.002, -0.000)*
Log transformed triglycerides	-0.007 (-0.016, 0.001)	-0.004 (-0.014, 0.006)
HDL-Cholesterol (mmol/L)	0.003(-0.001, 0.007)	0.004(-0.001, 0.010)
LDL-Cholesterol (mmol/L)	-0.002(-0.014, 0.010)	0.002(-0.016, 0.019)
Systolic Blood Pressure (mmHg)	-0.108 (-0.239, 0.023)	-0.171 (-0.308, -0.033)*
Diastolic Blood Pressure (mmHg)	-0.015 (-0.150, 0.116)	0.065 (-0.140, 0.268)

Associations assessed using linear regression.
Data are presented as the change in outcome associated with a 10 000 CPM increase in movement variability (95% confidence interval).
Model 1: Adjusted for age, ethnicity, SES, kcal, smoking and WC (when not in outcome).
Model 2: Adjusted as above, as well as sedentary behavior and moderate and vigorous physical activity.
Insulin, glucose, triglycerides, and waist circumference were log transformed prior to analyses.
Analyses involving BMI, WC and HDL-cholesterol and blood pressure included 707 females and 753 males participants, while all others included 314 females and 342 males.
*=statistically significant, $p<0.05$.
BMI: body mass index; WC: waist circumference; HDL-C: HDL-cholesterol; LDL-C: LDL-cholesterol.

Chapter 5 - Study 3: Prolonged sitting and markers of cardiometabolic disease risk in children and youth: a randomized crossover study

The following article is in press at the journal Metabolism: Clinical and Experimental, and has been formatted according to their requirements. Data from this article were presented at the International Congress on Physical Activity and Public Health in October, 2012, and at the American College of Sports Medicine AGM in May, 2013.

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Study design: TJS, JPC, RCC, GSG, GPK, ED, MST; data collection: TJS, JPC, ED; Data analysis and interpretation: TJS, JPC, MST; manuscript writing and editing: TJS, JPC, RCC, GSG, GPK, ED, MST.

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Structured Abstract

Objective: Recent evidence suggests that short bouts of uninterrupted sedentary behavior reduce insulin sensitivity and glucose tolerance while increasing triglyceride levels in both healthy and overweight/obese adults. To date no study has examined the acute impact of uninterrupted sitting in children and youth. The objective of the present study was to determine whether 8 hours of uninterrupted sitting increase markers of cardiometabolic disease risk in healthy children and youth, in comparison to 8 hours of sitting interrupted by light intensity walk breaks or structured physical activity.

Materials/Methods: 11 healthy males and 8 healthy females between the ages of 10 and 14 years experienced 3 conditions in random order: (1) 8 hours of uninterrupted sitting (Sedentary); (2) 8 hours of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes (Breaks); and (3) 8 hours of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes as well as 2 x 20 minutes of moderate-intensity physical activity (Breaks+Physical Activity). Insulin, glucose, triglyceride, HDL and LDL cholesterol area under the curve were calculated for each condition.

Results: We observed no significant differences in the area under the curve for any marker of cardiometabolic disease risk across the 3 study conditions (all $p > 0.09$).

Conclusions: These results suggest that in comparison to interrupted sitting or structured physical activity, a single bout of 8 hours of uninterrupted sitting does not result in measurable changes in circulating levels of insulin, glucose, or lipids in healthy children and youth.

Key Terms: Sedentary behavior, insulin sensitivity, glucose tolerance, pediatric population

Abbreviations

BMI: Body mass index

REE: Resting Energy Expenditure

iAUC: Incremental area under the curve

HDL: High density lipoprotein

LDL: Low density lipoprotein

Introduction

Prolonged bouts of uninterrupted sedentary behavior (sitting or reclining while expending ≤ 1.5 metabolic equivalents [1]) result in deleterious changes in insulin sensitivity, glucose tolerance, and plasma triglyceride levels in both healthy and overweight/obese adults [2–8]. Although initial studies in this area focused primarily on long-term bed rest and other restrictive forms of sedentary behavior [8], more recent studies have found that prolonged sitting may also result in significant reductions in insulin sensitivity and glucose tolerance in adult participants [3–5]. Dunstan and colleagues have recently reported that insulin and glucose responses to a standardized meal were elevated by nearly 25% following 7 hours of uninterrupted sitting in overweight and obese adults, in comparison to sitting with periodic light-intensity walk breaks [5]. Moreover, Stephens et al [4] reported that a single day of sitting reduced insulin action by 39% among a group of recreationally active young adults.

Despite the recent findings in adults, to date the effects of uninterrupted sitting in the pediatric population remain unexamined. Epidemiological studies have reported consistent associations between sedentary behavior and metabolic dysfunction in children and youth [9–11], suggesting that prolonged sitting may have a measurable health impact in this population. Given that the average child in North America spends more than half their waking hours sitting down [12–14], any cardiometabolic disease risk resulting from uninterrupted sedentary behavior in this age group would be of great public health importance. The objective of the present randomized crossover study was to determine whether 8 hours of uninterrupted sitting would result in increased concentrations of common markers of cardiometabolic disease risk in healthy children and youth, in comparison to a day of sitting interrupted by light intensity walk breaks, with and without structured physical activity.

Methods

Participants

Nineteen healthy children and youth (11 male, 8 female) aged 10-14 years were recruited for this study. There were no limits placed on body weight or physical activity levels prior to study entry. Written consent was obtained from the parents of all participants prior to participation. Oral assent was obtained from participants aged 10-13 years, while participants aged 14 years provided written consent. This study conformed to the ethical standards outlined in the Declaration of Helsinki and was approved by the Research Ethics Boards at the Children's Hospital of Eastern Ontario Research Institute and the University of Ottawa.

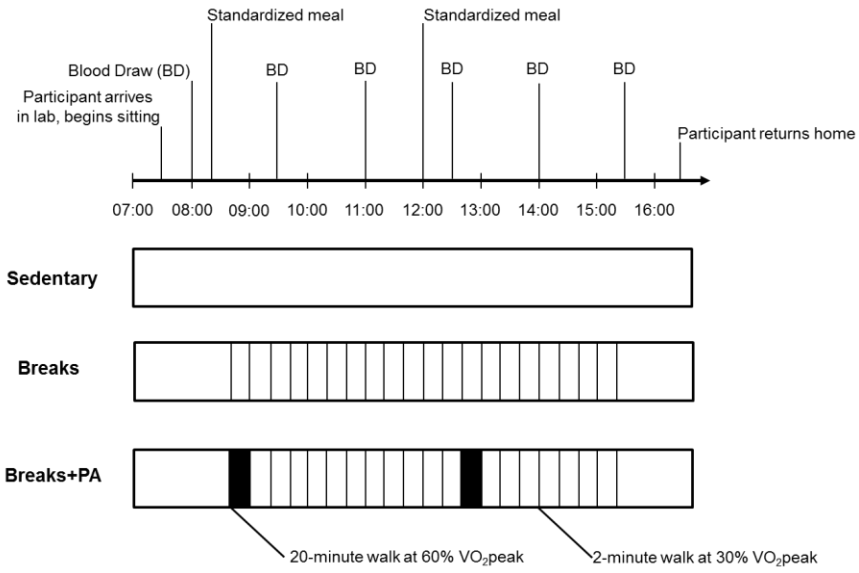

Figure 5.1 Overview of the study protocol.

Baseline Testing Session

Participants visited the Behavioral and Metabolic Research Unit at the University of Ottawa on 4 occasions – 1 baseline session and 3 experimental sessions – each separated by at least one week. Participants arrived for all sessions at 07:30, and were instructed to fast and abstain from structured exercise for 12 hours prior to each visit. The baseline session included measurements

related to anthropometry, physical activity, sedentary behavior, cardiorespiratory fitness (VO_2peak), and resting energy expenditure (REE). Weight was measured to the nearest 0.1 kg using a calibrated electronic scale. Standing height was measured to the nearest 0.5 cm using a wall-mounted stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Waist circumference was measured at the midpoint between the lower border of the last rib and the upper border of the iliac crest after a gentle expiration. Pubertal development was assessed using self-reported Tanner stages as previously validated by Taylor et al. [15].

REE and VO_2peak were assessed using an Ultima PF/PFX (MedGraphics, St Paul, USA) metabolic cart. VO_2peak was measured using the Dubowy graded treadmill protocol [16]. Participants wore an Actical accelerometer (Philips Respironics, Andover, USA) on their right hip for a total of five week days and two weekend days following baseline testing. Accelerometer data were processed using standardized reduction procedures [12] in SAS version 9.2 (SAS Institute, Cary, USA) and were used to assess baseline levels of physical activity and sedentary behavior. Pediatric accelerometer cut-points of 100, 1,500 and 6,500 counts per minute were used to identify light-, moderate- and vigorous-intensity physical activity, respectively [17]. Daily energy requirements were estimated as the sum of REE and average daily physical activity-related energy expenditure as calculated using the Actical 2.12 software (Philips Respironics, Andover, USA).

Experimental Sessions

The 3 experimental conditions were performed in random order, as determined by TJS using a random number generator in Microsoft Excel (Microsoft Corporation, Redmond, USA) (Figure 5.1). Participants were blinded to the order of conditions, and only told which condition they

would experience upon arrival in the lab each morning. Upon arrival to the lab at 07:30 a catheter was inserted into an antecubital vein for blood sampling. During the Sedentary condition, participants remained seated at all times from 07:30 until 15:30 (when necessary, participants were transported to the washroom via wheelchair). The Sedentary With Breaks (Breaks) condition was similar to the Sedentary condition, with the exception that participants walked for 2 minutes on a treadmill at an intensity equivalent to 30% of VO_2peak every 20 minutes beginning at 8:40 (i.e. 08:40, 09:00, 9:20, etc). Finally, the Sedentary With Breaks and Physical Activity (Breaks+PA) condition was similar to the Breaks condition, but in addition to walking at a light-intensity every 20 minutes, participants also performed two 20-minute bouts of moderate-intensity physical activity by walking or jogging on a treadmill at 60% of VO_2peak from 08:40-09:00 and from 12:40-13:00. During all 3 conditions, participants engaged in a standardized set of common sedentary behaviors in identical order – 4 hours of watching movies and television programs, 2 hours of puzzles and other forms of mental work, and 1.5 hours of video games.

Standardized Meals

Standardized meals were provided at breakfast (08:15) and lunch (12:00), using a menu developed for the pediatric population. Breakfast consisted of white bread, butter, peanut butter, cheddar cheese, and orange juice, while lunch included chicken strips, tortilla chips, grapes, baby carrots, 2% milk, lemonade, ketchup, and Oreo cookies [18]. Both meals were standardized relative to estimated daily energy requirements (rather than macronutrient intake) with breakfast and lunch respectively providing 25% and 40% of estimated daily needs. The mean±SD intake at breakfast and lunch were 2322±410 and 3669±799 kJ, respectively. The proportion of calories from carbohydrate, fat, and protein respectively at breakfast were 52±5%, 36±5% and 12±1% while at lunch they were 57±2%, 31±3% and 12±3%. Participants with allergies or food

intolerances (n=3) had individual food items replaced. However, each participant received identical meals at each of their 3 visits, and was asked to consume all food that was provided.

Markers of Cardiometabolic Disease Risk

Six blood draws were performed during each experimental day, with each sample requiring approximately 6 ml of blood. The first blood draw occurred at 08:00, and further draws were performed every 90 minutes until 15:30. All markers of cardiometabolic disease risk were assessed in duplicate using heparinized plasma, which was stored at -80 °C prior to analysis. There were no missing samples for any participant or variable of interest. Insulin was assayed by enzyme-linked immunosorbent assay (ALPCO Diagnostics, Salem, USA). Glucose, triglycerides, HDL and LDL cholesterol were assessed on the Ortho Vitros 5.1FS (Ortho-Clinical Diagnostics, Rochester, NY). The inter-assay precision for each test was as follows: insulin 11%; glucose 2%; triglycerides 2%; HDL-Cholesterol 3%, LDL-Cholesterol 3%. Net incremental area under the curve (iAUC) was calculated for all cardiometabolic disease risk factors using the trapezoid rule [19]. This approach was used rather than the positive iAUC since HDL- and LDL-Cholesterol curves were expected to have negative values [19].

Statistical Analyses

Sample size calculations were based on a recent study using a similar crossover design in overweight and obese adults [5]. Although this differs from the current study population, it is the only human study with a design similar in nature to the present study [5]. We estimated that 13 paired observations would provide 90% power to detect an absolute difference as small as 3,000 pmol/L·min in our primary outcome of insulin incremental area under the curve (iAUC) across conditions with a standard deviation of 3,000 pmol/L·min at an alpha level of p=0.05 and a two-tailed distribution.

Insulin, triglyceride, HDL and LDL cholesterol iAUC were non-normally distributed, and therefore transformed using a Box-Cox transformation. To determine if males and females could be combined into one analysis, sex-by-condition interactions were assessed for all dependent variables. No significant interactions were detected, therefore males and females were combined for all analyses to maximize statistical power. A linear mixed-model was fitted for the iAUC of each risk factor, with effects for condition, age, sex, BMI, waist circumference, Tanner stage, and baseline physical activity and sedentary behavior. Statistical significance was defined as a p value of 0.05 or less, and a Bonferroni correction was used to adjust for multiple comparisons in *post hoc* tests following the mixed-effect model. A similar linear mixed-model for raw levels of each risk factor over time was also fitted to assess temporal differences between conditions. This model included effects for condition, time, time-by-condition interaction, age, sex, BMI, waist circumference, Tanner stage, and baseline physical activity and sedentary behavior. Data are presented as mean ± standard deviation. All statistical tests were performed in SAS 9.2.

Results

Subject characteristics are presented in Table 5.1. In comparison to female participants, males were significantly older and more sedentary (all $p< 0.01$). However, there were no differences between males and females in terms of BMI, self-reported Tanner stage, moderate-to-vigorous physical activity, or any marker of cardiometabolic disease risk at baseline (all $p\geq 0.15$) There were no significant differences in baseline markers of cardiometabolic disease risk across the 3 experimental conditions (all $p>0.25$).

iAUC values for the 3 experimental conditions are presented in Table 5.2. We did not observe significant differences for any marker of cardiometabolic disease risk (all $p>0.09$). This finding

remained consistent with or without adjustment for age, sex, Tanner stage, BMI, waist circumference, and baseline physical activity and sedentary behavior. Separating analyses by sex did not materially change these results (data not shown).

When examining temporal changes in markers of cardiometabolic disease risk across conditions, we observed a significant time-by-condition interaction for plasma glucose concentrations only ($p = 0.001$). Post-hoc tests determined that the glucose concentrations were significantly greater at Time 2 during the Breaks+PA condition than during the Breaks condition ($p=0.004$) but not the Sedentary condition ($p=0.051$) (Figure 5.2). Glucose levels were not significantly different across conditions at any other time point (all $p>0.40$).

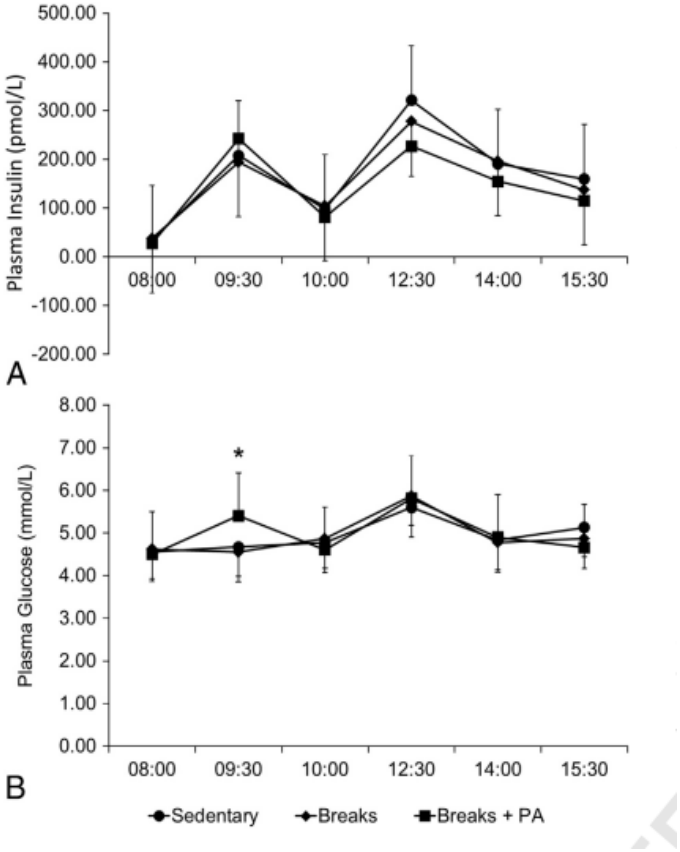

Figure 5.2 Insulin (A) and glucose (B) concentrations during 8 hours of prolonged sitting, with or without interruptions or physical activity (n=19).

Sedentary: 8 hours of uninterrupted sitting.Breaks: 8 hours of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes. Breaks + Physical Activity: 8 hours of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes as well as 40 minutes of moderate-intensity physical activity.
Data are presented as mean and standard deviation. Significance was assessed by a linear mixed-model with effects for condition, time, time-by-condition interaction, age, sex, BMI, waist circumference, Tanner stage, and baseline physical activity and sedentary behavior. A Bonferroni correction was used to adjust for multiple comparisons in post hoc tests following the mixed-effect model. Only plasma glucose showed a significant time-by-condition interaction (p = 0.001). * = significant difference between Breaks and Breaks + PA condition, p<0.01.

Discussion

The results of the present randomized crossover study suggest that an acute prolonged bout of uninterrupted sitting does not result in deleterious changes in traditional markers of cardiometabolic disease risk in healthy children and youth. Although we observed a small increase in glucose levels following breakfast in the Breaks+PA condition, we observed no other differences across the three study conditions for any outcome of interest. These results are in contrast to those reported in both healthy and overweight/obese adults, where uninterrupted sitting has been reported to result in acute and deleterious changes in insulin sensitivity and glucose tolerance [5].

There are several factors which could explain the discrepancy between the current findings and those observed in adults [2-5]. The current study focused on healthy boys and girls who were more physically active than the average Canadian youth [12]. Girls, but not boys, were also less sedentary than the national average [12]. The participants were also aerobically fit and metabolically healthy at baseline, which is underscored by the relatively small insulin and glucose responses following both breakfast and lunch. It is probable that a similar investigation in physically inactive youth, a highly sedentary population, or those with obesity or other elevated cardiometabolic disease risk factors, may produce results more similar to those observed in adults. Employing a more sensitive measure of metabolic disease risk (i.e. continuous glucose measurements), or a larger food challenge (i.e. liquid meal high in fat and/or sugar) may also have more clearly differentiated between the study conditions. However, these techniques are substantially more burdensome than those used in the current study, which may impede their use in studies of the pediatric population. Examining the expression of genes related to carbohydrate and lipid metabolism may also have revealed differences between conditions, and is worth exploring in future studies in this population. However, given the present results it seems likely

that the acute impact of a single bout of uninterrupted sitting on cardiometabolic disease biomarkers is simply smaller (or absent) in the pediatric population, as compared to adults.

It is not immediately clear why a transient increase in plasma glucose levels was observed at 9:30 during the Breaks+PA condition, but not during the Sedentary or Breaks conditions in the present study. It is worth noting that this increase occurred at the blood draw following the first 20-minute bout of exercise at 60% of VO_2peak. Aerobic exercise has been reported to increase hepatic glucose production and plasma glucose levels in adults [20], and it is possible that this was the cause of the increase observed in the present study. However, there is unlikely to be any clinical significance to this brief and relatively small increase in glucose levels.

The current study has several strengths and limitations that warrant mention. This is the first investigation into the acute impact of uninterrupted sedentary behavior in the pediatric population, and employed a rigorous randomized crossover design. Further, in contrast to the liquid meals that are sometimes used in adult studies of this nature [5], the standardized meals employed in the current study were similar to the food eaten by children on a normal basis, increasing the ecological validity of our study. However, it is also possible that a liquid meal that is high in sugar and fat may also have provided a greater metabolic stimulus, which may have more effectively differentiated the impact of our three study conditions. A longer exposure to uninterrupted sitting may also have resulted in different results, although the ecological validity of such an approach would be questionable. All participants in the current study were healthy at baseline, and the majority were both lean and physically active. It is therefore unclear whether similar results would be observed among a population of overweight or obese youth, or those showing signs of metabolic dysfunction. The present findings are also limited by our small sample size, and by the relatively small number of outcomes which were examined. Finally, the

present study did not examine whether fluctuations in energy balance across the three conditions may have influenced metabolic risk [4].

It is possible that the present findings may have differed had our study included a large number of participants, although our sample size calculation suggests that we had sufficient power for our primary outcome of insulin iAUC [5]. It is also worth noting that there were no consistent trends across the various outcomes measured, regardless of statistical significance. Although the insulin iAUC during the Sedentary condition was the highest of the 3 conditions, this was not the case for any other risk marker. This lends support to our conclusion that prolonged sitting does not result in significant increases in markers of cardiometabolic disease risk in this age group, and suggests that our results would not have been appreciably different with a larger sample size.

In conclusion, our findings suggest that in comparison to light walk breaks with or without structured physical activity, 8 hours of uninterrupted sitting do not result in measurable changes in circulating levels of insulin, glucose, or lipids in healthy children and youth. This suggests that the relationship between sedentary behavior and increased health risk observed in epidemiological studies may be due to the behaviors children engage in while seated, rather than any direct metabolic impact of sitting *per se* [21-23]. Future research should involve children at risk of cardiometabolic abnormalities to determine whether the results are comparable.

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Tables

Table 5.1 Participant characteristics.

	Male (n=11)	Female (n=8)	*p* value
Age (years)	12.9 (0/8)	11.3 (0.7)	<0.01
BMI (kg/m^2)	18.7 (4.5)	17.4 (2.9)	0.49
Waist Circumference (cm)	66.6 (15.8)	59.8 (5.7)	0.26
Tanner Stage	1.9 (1.0)	1.5 (0.8)	0.41
Sedentary behavior (min/day)	539.4 (48.3)	461.1 (66.0)	<0.01
MVPA (min/day)	66.8 (28.5)	59.5 (23.8)	0.56
Insulin (pmol/L)	42.6 (27.7)	33.1 (18.8)	0.42
Glucose (mmol/L)	4.7 (0.4)	4.6 (0.3)	0.71
LDL-Cholesterol (mmol/L)	1.9 (0.6)	2.0 (0.7)	0.91
HDL-Cholesterol (mmol/L)	1.3 (0.3)	1.3 (0.4)	0.15
Triglycerides (mmol/L)	0.7 (0.2)	0.7 (0.2)	0.81

BMI: body mass index; MVPA: moderate-and-vigorous physical activity; LDL: low density lipoprotein; HDL: high density lipoprotein. Date are presented as mean (SD). Significance was assessed using an independent measures t test.

Table 5.2 Net incremental area under the curve (iAUC) values for biomarkers of cardiometabolic disease risk during 8 hours of prolonged sitting, with or without interruptions or physical activity (n=19).

	Sedentary	Breaks	Breaks + Physical Activity	*p* for trend
Insulin (pmol/L·min)	80,559.3 (66380.3)	78,707.3 (83074.5)	64,270.8 (42272.4)	0.552
Glucose (mmol/L·min)	185.3 (171.7)	163.2 (148.8)	248.0 (220.4)	0.091
Triglycerides (mmol/L·min)	99.8 (114.4)	101.5 (69.0)	65.2 (66.4)	0.106
HDL-Cholesterol (mmol/L·min)	-38.7 (39.9)	-32.2 (40.2)	-52.3 (41.4)	0.431
LDL-Cholesterol (mmol/L·min)	-62.9 (67.2)	-50.0 (28.9)	-76.5 (51.4)	0.400

Sedentary: 8 hours of uninterrupted sitting.
Breaks: 8 hours of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes.
Breaks + Physical Activity: 8 hours of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes as well as 40 minutes of moderate-intensity physical activity.
Data are presented as mean (standard deviation). Significance was assessed by a linear mixed-model with effects for condition, age, sex, BMI, waist circumference, Tanner stage, and baseline physical activity and sedentary behavior. Although raw values are presented above, all statistical analyses have been performed using normalized data.

Chapter 6 - Study 4: Children and youth do not compensate for an imposed bout of prolonged sitting by reducing subsequent food intake or increasing physical activity: a randomized crossover study

The following article has been accepted for publication in the British Journal of Nutrition (Cambridge University Press), and has been formatted according to their requirements. **Data from this article were presented at the American College of Sports Medicine AGM in May, 2013.**

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Author Contributions

All authors contributed to the design of the research project; research was conducted by TJS and JPC; lab space and equipment were contributed by ED; TJS performed statistical analyses, and wrote the first draft of the manuscript with JPC and MST; all authors provided critical feedback to the manuscript and approved the final version; TJS takes primary responsibility for manuscript content. The authors have no potential conflicts of interest to report.

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Abstract

The behavioural impact of an imposed bout of prolonged sitting has yet to be investigated in the pediatric population. Our objective was to determine the acute effect of prolonged sitting on *ad libitum* food intake and spontaneous physical activity in healthy children and youth. A total of 20 healthy youth (12 males, 8 females) aged 10-14 years, with a mean±SD BMI of 18.6±4.3 kg/m^2, experienced 3 conditions in random order: (1) a day of uninterrupted sitting (*Sedentary*); (2) a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes (*Breaks*); and (3) a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes as well as 2 x 20 minutes of moderate-intensity physical activity (*Breaks+PA*). Food intake (*ad libitum* buffet meal) and physical activity (accelerometry for 24 hours) were assessed following each condition. Despite significant differences in sedentary behaviour and activity levels during the 3 in-lab sessions (all $p<0.01$), we observed no differences in *ad libitum* food intake immediately following each condition, nor any changes in the level of sedentary behaviour or physical activity in the 24-hours following each condition (all $p>0.25$). These findings suggest that children and youth may not compensate for an imposed bout of sedentary behaviour by reducing subsequent food intake or increasing physical activity.

Introduction

Both acute and chronic exposure to some sedentary behaviours (activities that involve sitting or reclining while expending ≤ 1.5 metabolic equivalents[1]) have been associated with excess food intake and weight gain in children and youth[2–5]. Chaput *et al.* [2] reported that in comparison to seated rest, 45 minutes of seated video game play resulted in significant increases in acute food intake and positive energy balance in adolescent males. Similarly, a recent systematic review by Tremblay and colleagues[3] concluded that sedentary behaviour (generally measured as time spent watching TV) was consistently associated with increased body weight and other markers of adiposity among school-aged children. This evidence has led some to suggest that sedentary behaviour may be a key contributor to increasing pediatric obesity rates[6–8]. However, while there is evidence that some common modalities of sedentary behaviour are likely to increase energy intake in children and youth, the impact of sitting *per se* has yet to be investigated[6].

The influence of an imposed bout of prolonged sitting on subsequent physical activity in children and youth is also unclear. It has previously been suggested that physical activity levels among this population are regulated by an "activitystat"[9–11]. In support of this view, several reports suggest that in response to an imposed bout of physical activity, youth may consciously or unconsciously compensate by reducing their physical activity levels throughout the rest of the day[9–12]. However, no study has yet examined whether an imposed bout of sedentary behaviour (i.e. sitting) results in a similar behavioural compensation in free-living conditions. If activity levels are regulated by a central mechanism similar to the "activitystat", it is plausible that youth may compensate for a prolonged period of sitting or inactivity by reducing their level of sedentary behaviour and increasing their level of physical activity later in the day. Given that North American children spend most of their waking time engaging in sedentary behaviours[13–15], it is pertinent to investigate the impact of prolonged sitting on subsequent food intake and physical activity, both of which are important health-related behaviours.

The objective of this randomized crossover study was to determine whether one day of uninterrupted sitting would result in different compensatory changes in *ad libitum* food intake and/or spontaneous physical activity in healthy children and youth, in comparison to a day of sitting interrupted by light intensity walk breaks, with and without structured physical activity. Based on the available evidence, we hypothesized that prolonged sitting would result in a compensatory increase in subsequent spontaneous physical activity, a reduction in sedentary behaviour, and no change in *ad libitum* food intake.

Experimental Methods

Subjects

Twenty healthy children and youth (12 males, 8 females) aged 10-14 years participated in this intervention study. There were no limits placed on participant weight or activity levels. This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects/patients were approved by the institutional Research Ethics Boards at the Children's Hospital of Eastern Ontario and the University of Ottawa. Written informed consent was obtained from the parents of all participants. Oral assent was obtained from participants aged 10-13 years (assent was witnessed and formally recorded), while participants aged 14 years provided written consent prior to participation.

Baseline Testing Session

The current analysis is part of a larger study examining the metabolic impact of prolonged sitting in children and youth, which has been described previously[16]. Participants attended 1 baseline session and 3 experimental sessions, each separated by at least one week. All sessions began at 07:30, and participants were instructed to fast and abstain from structured exercise for 12 hours prior to each visit. The baseline session included measurements related to anthropometry, physical activity, sedentary behaviour, cardiorespiratory fitness (VO_2peak), and resting energy expenditure (REE). At this initial visit participants were asked to identify any food allergies or intolerances that might impact the standardized breakfast and buffet meals during the experimental sessions. Weight was measured to the nearest 0.1 kg using a BWB-800AS calibrated electronic scale (Tanita Corporation of America Inc., Arlington Heights, IL). Standing height was measured to the nearest 0.5 cm using a Tanita HR-100 wall-mounted stadiometer (Tanita Corporation of America Inc., Arlington Heights, IL). Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). Children were categorized as overweight/obese using the International Obesity Taskforce (IOTF) cut-points.[17] Waist circumference was measured at the midpoint between the lower border of the last rib and the upper border of the iliac crest after a gentle expiration. Pubertal development was assessed using self-reported Tanner stages as previously validated by Taylor *et al* [18].

REE and VO_2peak were measured using an Ultima PF/PFX (MedGraphics, St Paul, USA) metabolic cart. VO_2peak was assessed using the Dubowy graded treadmill protocol[19]. Participants wore an Actical accelerometer (Philips Respironics, Andover, USA) on their right hip

for seven consecutive days following baseline testing. Accelerometer data were processed using standardized reduction procedures[13] in SAS version 9.2 (SAS Institute, Cary, USA) and used to assess baseline levels of physical activity and sedentary behaviour. Accelerometer cut-points of 100, 1500 and 6500 counts per minute were used to identify light-, moderate-, and vigorous-intensity physical activity, respectively[20]. Total energy expenditure during each of the experimental conditions was estimated using the following formula, where the thermic effect of food is fixed at 10%: (REE + physical activity energy expenditure during the session) x 1.11[21].

Experimental Sessions

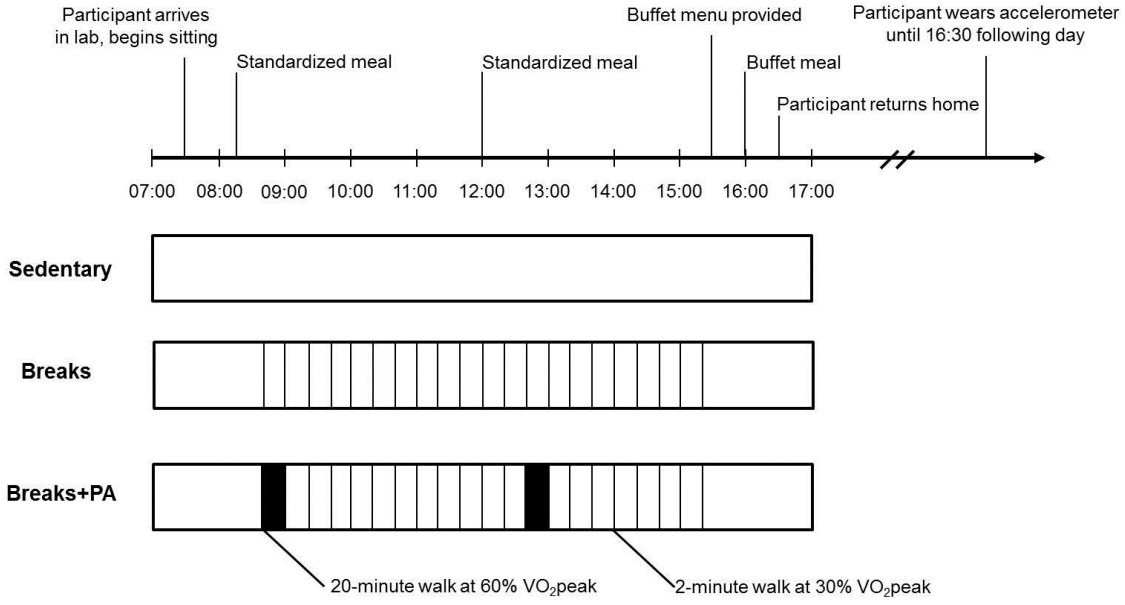

Figure 6.1 Overview of the study protocol (modified from Saunders et al.[16]).

Sedentary: a day of uninterrupted sitting.
Breaks: a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes.
Breaks + Physical Activity: a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes as well as 40 minutes of moderate-intensity physical activity.

The 3 experimental conditions were performed in random order, as determined using a random number generator in Microsoft Excel (Microsoft Corporation, Redmond, USA). Participants arrived at the lab at 07:30 for all experimental conditions and began sitting. During the *Sedentary condition*, participants remained seated without interruption until 16:30 (when necessary, participants were transported to the washroom via wheelchair) (**Figure 6.1**). The Sedentary With

Breaks (*Breaks*) condition was similar to the *Sedentary condition*, with the exception that participants walked for 2 minutes on a treadmill at an intensity equivalent to 30% of VO_2peak every 20 minutes beginning at 08:40 (i.e. 08:40, 09:00, 09:20, etc). Finally, the Sedentary With Breaks and Physical Activity (*Breaks+PA*) condition was similar to the *Breaks condition*, but in addition to walking at a light-intensity every 20 minutes, participants also performed two 20-minute bouts of moderate-intensity physical activity by walking or jogging on a treadmill at 60% of VO_2peak from 08:40-09:00 and from 12:40-13:00.

During all 3 conditions, participants engaged in a standardized set of common sedentary behaviours in identical order – 4 hours of watching movies and television programs, 2 hours of puzzles and other forms of mental work, and 2 hours of video games. Each experimental condition concluded with a buffet meal which lasted from 16:00-16:30. Participants wore accelerometers for the duration of each experimental condition and the 24-hours following each condition to assess levels of physical activity and sedentary behaviour.

Standardized Meals

Standardized meals were provided at breakfast (08:15) and lunch (12:00), using a menu developed for the pediatric population[22]. Breakfast consisted of white bread, butter, peanut butter, cheddar cheese, and orange juice, while lunch included chicken strips, tortilla chips, grapes, baby carrots, 2% milk, lemonade, ketchup, and Oreo cookies. Both meals were standardized relative to estimated daily energy requirements (rather than macronutrient intake) with breakfast and lunch respectively providing 25% and 40% of estimated daily needs. Daily energy requirements were estimated as the sum of REE and average daily physical activity-related energy expenditure recorded at baseline. The mean±SD intake at breakfast and lunch were 2322±410 and 3669±799 kJ, respectively. The proportion of kilojoules from carbohydrate, fat, and protein respectively at breakfast were 52±5%, 36±5% and 12±1% while at lunch they were 57±2%, 31±3% and 12±3%. Participants with allergies or food intolerances (n=3) had individual food items replaced. However, each participant received identical meals at each of their 3 visits, and was asked to consume all food that was provided.

Visual Analog Scales (VAS)

Hunger and prospective food consumption were assessed immediately before participants were provided with the buffet food menu at 15:30 and again immediately following the buffet meal which occurred from 16:00-16:30. This was done using 100 mm Visual Analog Scales (VAS)

adapted from those described by Hill and Blundell[23], which are reliable both before and after a meal[24] and have been employed previously in pediatric populations[2,25]. Subjects were asked to place a mark at the position which approximated their level of hunger and the amount of food they thought they could eat at that time.

Buffet Meals

Spontaneous food intake was assessed using an *ad libitum* buffet meal at 16:00 during each experimental condition. The buffet has been validated previously[26], and allowed for assessment of total energy intake as well as macronutrient composition. The meal consisted of a variety of foods differing in macronutrient composition. Participants selected items from a written menu, were instructed to eat *ad libitum*, and were provided with additional servings on request. Participants were given 30 minutes for this meal, and all foods were weighed to the nearest 0.1 g before and after ingestion. Energy and macronutrient intake were calculated using The Food Processor (ESHA Research, Salem, Oregon).

Statistical Analyses

As described above, the current analysis is part of a larger investigation of the metabolic impact of prolonged sitting in the pediatric population[16]. The primary outcome of the study was insulin sensitivity, which was used to estimate the necessary sample size to assess significance. The sample size for the present analyses was therefore predetermined. However, given the levels of variability observed in the present study, a post hoc sample size calculation revealed that we had greater than 80% power to detect a difference of 12 minutes/day in moderate physical activity, 5 minutes/day in vigorous physical activity, or 600 kJ in energy intake across study conditions.

Buffet food intake (both in kilojoules and grams), absolute protein intake, percent fat intake, and both VAS following the buffet meal were non-normally distributed, and were transformed using Box-Cox transformations to improve normality. Baseline differences between male and female participants were assessed by independent samples t test for continuous variables and by chi square test for proportions.

To determine if males and females could be combined in subsequent analyses, sex-by-condition interactions were assessed for all dependent variables. No significant interactions were detected, therefore males and females were combined for all analyses to maximize statistical power and improve clarity. A linear mixed-model was fitted for each food intake-related outcome, with

effects for condition, age, sex, Tanner stage, BMI and baseline physical activity and sedentary behaviour. Similar models were used for physical activity and sedentary behaviour-related outcomes, with additional adjustment for accelerometer wear-time. This study was not sufficiently powered to investigate the impact of BMI on these results, and therefore BMI-by-condition interactions were not examined. Statistical significance was defined as a two-sided alpha level of 0.05, and a Bonferroni correction was used to adjust for multiple comparisons in *post hoc* tests following the mixed-effect model. Data are presented as mean (SD). All statistical tests were performed in SAS 9.2.

Results

Participant characteristics are presented in **Table 6.1**. In comparison to their female counterparts, male participants were significantly older, spent more time engaging in sedentary behaviour, and less time engaging in light-intensity physical activity at baseline (all $p<0.03$). In contrast, at baseline there were no differences between males and females with respect to BMI, waist circumference, self-reported Tanner stage, or daily moderate-and-vigorous intensity physical activity (MVPA) (all $p > 0.15$).

The amounts of sedentary behaviour, light-, and moderate-intensity physical activity accumulated during each experimental condition are presented in **Table 6.2**. As imposed, the 3 conditions varied significantly with respect to sedentary time, light- and moderate-intensity physical activity and total steps during the in-lab portion of the study (all $p< 0.01$). According to accelerometer data, during the *Sedentary condition* participants spent 97.1% of lab time engaging in sedentary behaviour, compared to 86.5% and 81.0% in the *Breaks* and *Breaks+PA* conditions, respectively. As expected, there were no differences in vigorous physical activity across the three study conditions ($p=0.18$), nor did we observe differences for any measure related to hunger, food intake, or satiety across the three study conditions during the in-lab portion of the study (all $p>0.06$) (Table 6.2 and **Figure 6.2**). These results were similar with and without adjusting for age, sex, Tanner stage, BMI and baseline physical activity and sedentary behaviour. The estimated energy expenditure during the in-lab portion of the study differed significantly across the 3 conditions (all $p<0.01$), and is presented with energy intake in Figure 2.

The volume of sedentary behaviour and physical activity accumulated during the 24-hour period immediately following each experimental condition is presented in **Table 6.3**. We observed no significant differences for any activity-related variable (all $p>0.25$). These results were consistent

whether examining absolute levels of activity, as a percent of total wear-time, as a change score relative to baseline levels, or restricting analyses to only those participants who had 10 or more hours of wear time (data not shown). These results were not impacted by adjustment for age, sex, Tanner stage, BMI, baseline physical activity and sedentary behaviour or accelerometer wear-time.

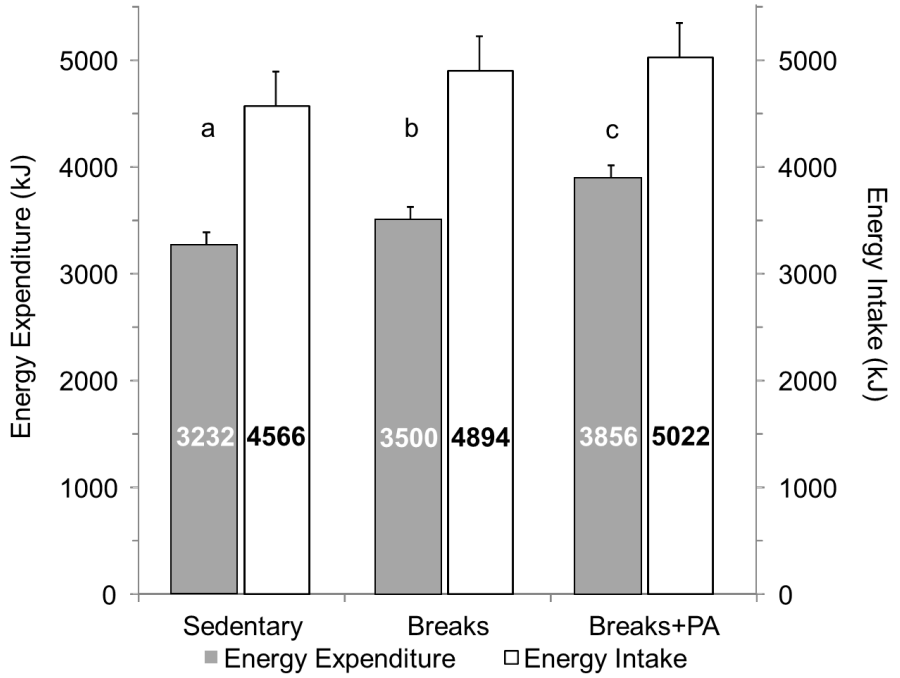

Figure 6.2 Energy intake and estimated energy expenditure while in lab during a day of sitting with or without interruptions and structured physical activity.

Sedentary: a day of uninterrupted sitting.
Breaks: a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes.
Breaks + Physical Activity: a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes as well as 40 minutes of moderate-intensity physical activity.
Energy intake was assessed using an *ad libitum* buffet meal, while energy expenditure was estimated as (REE + Physical activity energy expenditure) x 1.11.
Data are presented as mean±SEM. Significance was assessed by a linear mixed-model with effects for condition, accelerometer wear time, age, sex, Tanner stage, BMI and baseline physical activity and sedentary behaviour. Bars with different superscript letters are significantly different at $p<0.05$ level with Bonferroni correction.

Discussion

The findings of the present study, although exploratory and hypothesis generating, suggest that children may not compensate for an acute bout of prolonged sitting by reducing subsequent food intake or increasing physical activity levels. Although there were differences in the level of sedentary behaviour, physical activity, and estimated energy expenditure during the 3 study conditions, we observed no differences in *ad libitum* food intake immediately following each session, nor were there any differences in physical activity or sedentary behaviour levels in the subsequent 24-hour period. Future studies are needed to examine whether prolonged sitting results in sustained positive energy balance, or whether subsequent adaptations in energy intake or expenditure are able to maintain energy homeostasis.

These results suggest that it is the behaviours that youth commonly engage in while seated (e.g. watching television[4], playing video games[2], or doing mental work[27]), rather than sitting *per se*, that result in the increased food intake associated with sedentary behaviour. This is supported by the work of Epstein and colleagues [28-30], who have reported that reductions in screen-based sedentary behaviours have an important influence on both energy intake and body weight among children and youth. For example, Epstein et al. reported that reducing daily screen time by 25-50% resulted in a spontaneous reduction in energy intake of 1938 kJ/day in a group of non-overweight teens over a 3 week period.[28] Although physical activity-related energy expenditure also increased following the reduction in screen time, it was of a much smaller magnitude than the reduction in energy intake (474 kJ/day).[28] Collectively, these findings suggest that focusing on a reduction in screen-based sedentary behaviours may have a greater impact on energy balance than a similar focus on total sedentary time.

The present findings also support the assertion that energy intake is not acutely coupled with energy expenditure in the pediatric population[6, 31]. Instead, the available evidence suggests that any acute influence of physical activity on food intake in children and youth is likely to be related to the intensity of the activity, rather than the associated energy expenditure. For example, Thivel and colleagues[12] recently compared the impact of high- (75% VO_2max) and low-intensity (40% VO_2max) exercise on *ad libitum* food intake in obese adolescents. They reported that despite both activity bouts expending roughly 1400 kJ of energy, only the high-intensity bout reduced subsequent food intake at lunch and dinner, in comparison to a day without structured exercise. While the *Breaks+PA* condition of the present study did include a total of 40 minutes of structured exercise at 60% of VO_2peak, it may be that this intensity was insufficient to influence

subsequent food intake. It is also possible that the acute influence of exercise on caloric intake in this age group may be different among healthy weight compared to overweight/obese populations,[32] although the present study was not sufficiently powered to examine such body weight interactions. Future studies should also investigate variations in the magnitude and direction of behavioural compensation (or lack thereof) following prolonged sitting, as exercise-induced variations in energy expenditure and body weight have been shown to vary considerably among adults.[33]

The current findings also suggest that physical activity levels are not acutely regulated by an internal "activitystat"[9, 10], as we observed no difference in physical activity or sedentary behaviour levels in the 24-hour period following each experimental condition. Instead, these results support the recent findings of Goodman and colleagues[34] who found no evidence that a bout of physical activity during one portion of the day was compensated for with reduced physical activity later in the day among a cohort of British children. These results are in contrast to those of Thivel and colleagues[12], who reported that an imposed bout of high- and low-intensity physical activity did not significantly increase 24-hour energy expenditure above that observed during an inactive day among obese teenagers. However, it should be noted that Thivel and colleagues assessed energy expenditure by placing participants in calorimetric chambers, which is likely to have substantially reduced their opportunities for spontaneous physical activity outside of their bouts of structured exercise. In contrast, following the in-lab portion of each condition, the present study examined physical activity levels in free-living conditions, which may help to explain these discrepant findings.

Taken together, the above findings suggest that acute sedentary behaviour may contribute to a positive energy balance due to its low level of energy expenditure, and by failing to produce a compensatory reduction in energy intake or increase in energy expenditure subsequent to the behaviour. This effect is likely to be exacerbated through the increased caloric intake that is associated with many common sedentary behaviours such as TV viewing and video game playing[2,4,27]. However, the current findings also suggest that the introduction of periodic bouts of light- and moderate-intensity physical activity throughout the day may increase energy expenditure without resulting in compensatory changes in energy intake or spontaneous physical activity. It is worth noting that physical activity intensity has been negatively associated with adiposity in the pediatric age group, and therefore the impact of breaks of vigorous intensity on energy balance are worthy of future study[35]. The current results suggest that activity breaks of at

least light- or moderate intensity spread throughout the day may be a simple way to promote or maintain energy balance in the current sedentary and obesogenic environment[36].

The present study has several strengths and limitations which warrant mention. The study employed a rigorous randomized crossover design, which strictly controlled participants' energy intake, sedentary behaviour and physical activity across the 3 study conditions. However, energy intake was measured only once at the end of each in-lab session, and physical activity and sedentary behaviour were only assessed in the 24-hour period immediately following each lab session. It is therefore unclear whether similar results would be seen in response to chronic exposure to prolonged sedentary behaviour. The present findings are also limited by the small sample size, and therefore the possibility of a Type 2 error cannot be ruled out. It is also worth noting that participants were required to eat standardized meals at both breakfast and lunch, which may have been different from the amount or type of food they would consume on a normal day (habitual diet was not assessed in the current study). Similarly, the buffet meal in the present study took place at 16:00, which is earlier than the typical evening meal in North America. Further, participants in the present study were healthy, and more physically active at baseline than the general Canadian population[13]. Thus these results may not generalize to physically inactive, obese or diseased participants, or to other age groups. Physical activity and sedentary behaviour levels in the present study were assessed via accelerometers, which is not able to accurately measure all forms of activity (e.g. swimming, cycling). However, the use of accelerometry allowed for the assessment of sedentary behaviour and physical activity in free-living conditions, increasing the ecological validity of these findings. Finally, the buffet in the present study included palatable items such as pizza and potato chips, which may have itself influenced *ad libitum* intake or reduced differences across conditions[6].

In conclusion, we found no evidence that children and youth compensate for an imposed bout of prolonged sitting, with or without breaks and structured physical activity, by decreasing their subsequent energy intake and/or increasing their physical activity levels. These findings suggest that a sedentary day may lead to a positive energy balance through reduced energy expenditure without compensatory reductions in energy intake or subsequent increases in physical activity energy expenditure. They also suggest that the introduction of light- or moderate-intensity activity breaks throughout an otherwise sedentary day may help to increase energy expenditure with no compensatory increase in food intake, thus promoting energy balance in the pediatric age

group. Future studies with larger sample sizes are needed to further investigate the impact of prolonged sitting on energy balance in the pediatric population.

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Tables

Table 6.1 Characteristics of study participants at baseline.

	Male (n=12)		Female (n=8)		
	Mean	SD	Mean	SD	*p* value
Age (years)	12.8	(1.0)	11.3	(0.7)	<0.01
BMI (kg/m^2)	19.4	(5.0)	17.4	(2.9)	0.31
Proportion Overweight/Obese	2/12		1/8		0.80
Waist Circumference (cm)	68.7	(16.5)	59.8	(5.7)	0.16
Tanner Stage	2.0	(1.0)	1.5	(0.8)	0.27
Sedentary Behaviour (min/day)	536.4	(47.2)	461.6	(66.0)	<0.01
LPA (min/day)	209.6	(45.6)	256.8	(33.8)	0.02
MVPA (min/day)	64.0	(28.8)	59.5	(23.8)	0.72

Date are presented as mean (SD).
BMI: Body mass index; LPA: light physical activity; MVPA: moderate-and-vigorous physical activity.
Baseline differences between male and female participants were assessed by independent samples t test (continuous variables) and chi square test (proportions).

Table 6.2 Measures of sedentary behaviour, physical activity, hunger and caloric intake during time spent in lab engaging in prolonged sitting, with and without breaks and structured physical activity (n=20).

	Sedentary		Breaks		Breaks+PA		
	Mean	SD	Mean	SD	Mean	SD	*p* for trend
Sedentary Behaviour (min)	498.9	(19.2)[a]	444.3	(19.2)[b]	416.0	(19.6)[c]	<0.01
Light Physical Activity (min)	12.3	(19.8)[a]	58.6	(19.8)[b]	55.4	(20.3)[b]	<0.01
Moderate Physical Activity (min)	2.3	(14.5)[a]	10.5	(14.5)[a]	40.9	(14.9)[b]	<0.01
Vigorous Physical Activity (min)	0.1	(2.3)	<0.1	(2.3)	1.3	(2.4)	0.18
Steps (steps)	687	(1979)[a]	4482	(1979)[b]	8658	(2042)[c]	<0.01
Pre-Buffet Prospective Food Consumption (mm)	56	(20)	66	(20)	61	(19)	0.07
Pre-Buffet Hunger (mm)	55	(19)	63	(19)	56	(18)	0.16
Post-Buffet Prospective Food Consumption (mm)	13	(15)	14	(15)	7	(14)	0.18
Post-Buffet Hunger (mm)	9	(11)	8	(11)	6	(10)	0.25
Food intake in the buffet (g)	782	(254)	839	(254)	767	(254)	0.37
Calories from carbohydrates (%)	56	(9)	57	(9)	55	(9)	0.62
Calories from fat (%)	33	(9)	32	(9)	36	(9)	0.32
Calories from protein (%)	10	(4)	11	(4)	10	(4)	0.44

Sedentary: a day of uninterrupted sitting.
Breaks: a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes.
Breaks + Physical Activity: a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes as well as 40 minutes of moderate-intensity physical activity.
Significance was assessed by a linear mixed-model with effects for condition, age, sex, Tanner stage, BMI and baseline physical activity and sedentary behaviour. Columns with different superscript letters are significantly different at $p<0.05$ level with Bonferroni correction.

Table 6.3 Sedentary behaviour and physical activity levels in the 24-hours immediately following prolonged sitting with or without breaks and structured physical activity (n=20).

	Sedentary		Breaks		Breaks+PA		
	Mean	SD	Mean	SD	Mean	SD	*p* for trend
Wear time (min)	700.0	(235.0)	728.7	(240.9)	701.1	(247.0)	0.90
Sedentary Behaviour (min)	518.5	(73.0)	514.4	(73.3)	501.5	(74.8)	0.67
Sedentary Behaviour (% wear time)	73.6	(10.1)	71.1	(10.2)	70.9	(10.4)	0.60
Light Physical Activity (min)	139.5	(46.2)	140.8	(46.4)	152.5	(47.3)	0.53
Light Physical Activity (% wear time)	19.6	(6.1)	20.2	(6.1)	21.3	(6.2)	0.60
Moderate Physical Activity (min)	48.1	(30.8)	48.0	(30.9)	52.7	(31.7)	0.85
Moderate Physical Activity (% wear time)	6.4	(4.8)	7.5	(4.8)	7.4	(5.0)	0.73
Vigorous Physical Activity (min)	4.2	(10.1)	6.9	(10.2)	3.7	(10.4)	0.54
Vigorous Physical Activity (% wear time)	0.5	(1.5)	1.2	(1.5)	0.5	(1.6)	0.26
Steps (steps)	10596	(5414)	11172	(5439)	10485	(5561)	0.89

Sedentary: a day of uninterrupted sitting.
Breaks: a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes.
Breaks + Physical Activity: a day of sitting interrupted with a 2-minute light-intensity walk break every 20 minutes as well as 40 minutes of moderate-intensity physical activity.
Significance was assessed by a linear mixed-model with effects for condition, accelerometer wear time, age, sex, Tanner stage, BMI and baseline physical activity and sedentary behaviour. There were no significant differences between experimental conditions.

Chapter 7 - General Discussion

Sedentary behaviour has been linked with increased cardiometabolic disease risk among children and youth in multiple cross-sectional and longitudinal studies(5–12,41–45). The purpose of the present thesis was to clarify the relationship between sedentary behaviour and health in the pediatric age group by addressing two key objectives:

1. To determine the cross-sectional association of sedentary time, interruptions in sedentary time, sedentary bout length, and total movement variability with markers of cardiometabolic disease risk among children and youth.
2. To examine the impact of 1-day of prolonged sedentary behaviour, with and without interruptions or structured physical activity, on markers of cardiometabolic disease risk, hunger, food intake and spontaneous physical activity levels in children and youth.

To achieve these objectives, we have performed 4 studies, using 3 datasets, and employed both cross-sectional and interventional research methodologies. Our findings, which are summarized in Table 7.1, make an important contribution to our understanding of the health impact of sedentary behaviour among children and youth. In the discussion that follows, I will examine our results in the context of my two key objectives, and in relation to the published literature.

Table 7.1 Summary of thesis key findings and contributions to the literature.

Study 1	Short bouts of sedentary behaviour, breaks in sedentary time, and screen-based sedentary behaviours are independently associated with cardiometabolic disease risk among children with a family history of obesity.
Study 2	Total movement variability is independently associated with reduced cardiometabolic disease risk among a representative sample of American children and youth.
Study 3	Uninterrupted sitting does not result in acute changes in markers of cardiometabolic disease risk among healthy children and youth aged 10-14 years.
Study 4	Uninterrupted sitting does not result in compensatory changes in subsequent energy intake or physical activity levels in health children and youth aged 10-14 years.

7.1 Characteristics of sedentary behaviour: associations with health indicators

The studies presented in Chapters 3 and 4 of this thesis demonstrate that breaks in sedentary time, short bouts of sedentary behaviour (e.g. those lasting 1-4 minutes), and total movement variability are associated with reduced cardiometabolic disease risk in children and youth, independent of total sedentary time and physical activity. These cross-sectional findings suggest that, all else being equal, children who have high amounts of movement variability and frequent interruptions in their sedentary time may experience reduced cardiometabolic disease risk when compared to children with less frequent interruptions in sedentary time or reduced movement variability. The findings presented in Chapters 3 and 4 also highlight the contrasting relationships of self-reported screen time and directly measured sedentary time with markers of cardiometabolic disease risk in the pediatric population. While TV viewing and computer time were independently associated with increased cardiometabolic disease risk in girls and boys respectively, daily sedentary time assessed via accelerometer was not associated with any marker of cardiometabolic disease risk in the fully adjusted model in either sex.

To our knowledge, the studies contained in Chapters 3 and 4 are the first to report a favourable association between breaks in sedentary time, total movement variability and global cardiometabolic disease risk in the pediatric population. These studies add to a growing body of research in adults which suggests that prolonged bouts of uninterrupted sedentary time are associated with increased health risk, while the opposite is seen for breaks in sedentary time (13,14,25,26,46–48). For example, breaks in sedentary time have been reported to be independently and beneficially associated with multiple markers of cardiometabolic disease risk in adults (13, 14). However, recent studies in pediatric populations have failed to detect any association between breaks in sedentary time and markers of cardiometabolic disease risk (15,16), with the exception of waist circumference (in boys only) in one study (15). As noted in Chapter 3, the association between breaks in sedentary time and cardiometabolic disease risk in this thesis was examined in children with a family history of obesity, while previous studies on this topic have focused primarily on nationally representative samples of Canadian and American youth (15,16). It is possible that this difference in sample populations may help to explain why the findings reported in Chapter 3 diverge from previous investigations in the pediatric population.

While the associations between characteristics of sedentary time and cardiometabolic disease risk presented in Chapter 3 were examined using a cohort of children with a family history of obesity, the role of total movement variability was investigated using a representative sample of American children and youth via the National Health and Nutrition Examination Survey. The independent associations observed between movement variability and clustered cardiometabolic disease risk in Chapter 4 are

especially noteworthy, given that previous research using this same dataset has failed to detect any association between characteristics of sedentary time and markers of cardiometabolic disease risk (16). When combined with the findings in Chapter 3 and elsewhere in the published literature (1,9,13,14,25,26), a picture begins to emerge which suggests that an "ideal" movement profile may be more complicated than simply meeting national physical activity guidelines.

Table 7.2 Healthy and deleterious movement profiles for children and youth.

Ideal Movement Profile	Deleterious Movement Profile
↑ Movement variability	↓ Movement variability
↑ Moderate and vigorous physical activity	↓ Moderate and vigorous physical activity
↑ Breaks in sedentary time	↓ Breaks in sedentary time
↓ Total sedentary time	↑ Total sedentary time
↓ Prolonged bouts of sedentary time	↑ Prolonged bouts of sedentary time
↓ Screen time	↑ Screen time

As outlined in Table 7.2 and Figure 7.1, the ideal movement profile for children and youth appears to be one which includes large amounts of physical activity, relatively little uninterrupted sitting or screen time, and frequent changes in movement intensity. However, at present the clinical relevance of breaks in sedentary time and increased movement intensity remain largely unclear in the pediatric age group. If future studies support the notion that increasing movement variability or breaks in sedentary time are associated with reduced health risk in the pediatric population, it could lead to the development of novel interventions with increased efficacy and/or effectiveness for

treating or preventing chronic disease risk among children and youth. For example, data from the studies that make up this thesis suggest that for a given level of activity and sedentary time, interventions that produce greater increases in total movement variability and/or more frequent breaks in sedentary time could result in greater health benefits among children and youth. Both epidemiological and lab-based studies should examine the health impact of such changes in movement patterns,in order to determine whether they result in clinically significant improvements in cardiometabolic health. If intervention studies demonstrate that such changes are efficacious in improving cardiometabolic health in at-risk youth, this could lead to the development of interventions that are more practical for non-exercisers than programs which focus exclusively on structured exercise.

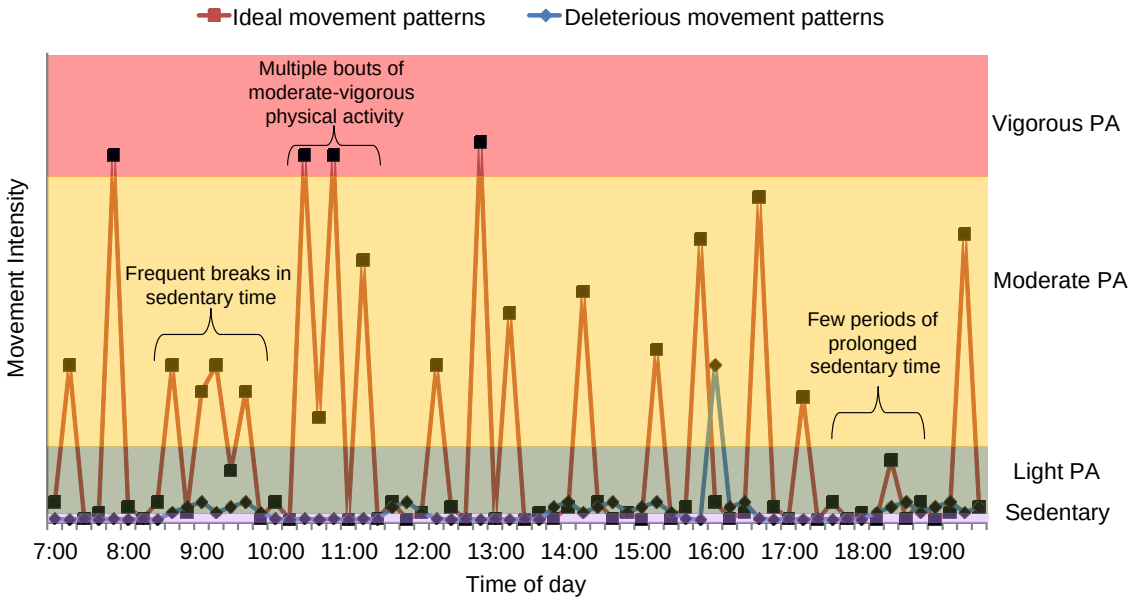

Figure 7.1 Examples of healthy and deleterious movement patterns.

In addition to any potential direct health impact of movement variability, the findings of Chapter 4 also suggest that this new metric may serve as a simple means of assessing the

quality of individual's overall movement patterns. For example, Chapter 4 found that total movement variability is associated with a constellation of desirable movement behaviours, including high levels of physical activity, low levels of sedentary time, and frequent interruptions in sedentary time. When compared to individuals with low levels of movement variability, individuals with high movement variability are likely to accumulate higher amounts of MVPA, and lower amounts of sedentary behavior. Further, at any combination of total sedentary time and MVPA, individuals with high levels of movement variability are also likely to have more frequent breaks in sedentary time and/or higher average physical activity intensity. Rather than assessing each of these movement behaviours in isolation, movement variability could therefore be used as a global measure of a "healthy" movement profile. While further research is clearly necessary before such applications of movement variability can be made in a clinical setting, these examples nonetheless highlight the potential value of this novel movement characteristic.

7.2 The impact of prolonged sedentary behaviour on markers of cardiometabolic disease risk, energy intake, and physical activity in children and youth

Chapters 5 and 6 examined the metabolic and behavioural impact of prolonged sitting among a group of healthy children aged 10-14 years. These studies failed to detect any significant impact of prolonged sitting on markers of cardiometabolic disease risk in the pediatric age group. Further, they also demonstrated that children did not compensate for an acute bout of prolonged sitting by modifying their subsequent levels of physical activity, sedentary behaviour, or food intake. As with the cross-sectional association between breaks in sedentary time and markers of cardiometabolic disease risk (which has been observed more consistently in adult populations than in children and youth), the

results from Chapters 5 and 6 suggest that the acute impact of prolonged sitting may also differ according to sample population. For example, interventions which impose prolonged sitting have resulted in acute reductions in insulin sensitivity and glucose tolerance in multiple studies among adults (25,46,49). We did not observe any such change among healthy children and youth in the present thesis, despite sitting for a comparable amount of time. It is possible that changes in markers of cardiometabolic disease risk may have been observed had the present intervention focused on a different participant population, such as children and youth with elevated risk of chronic disease, or those with a family history of obesity (similar to the participants in Chapter 3). As discussed below, this is an important area for future research in the pediatric age group.

The data presented in Chapters 5 and 6 also help to explain the disparate associations of objectively measured sedentary time and screen time observed in Chapter 3 and in the field more generally. Numerous studies (9,11,12,16,50) have found that screen-based sedentary behaviours are associated with markers of cardiometabolic disease risk in children and youth. However, although some studies have reported a significant association between objectively measured sedentary time and markers of cardiometabolic disease risk in this population (5,10,44,51–53), many others (including Chapter 3 of this thesis) have failed to detect such a relationship (15,16,50,54–59). In Chapter 6 of this thesis we report that prolonged sitting did not result in significant changes in markers of cardiometabolic disease risk or subsequent food intake or physical activity in children and youth. In contrast, both observational and experimental evidence suggests that screen-based sedentary behaviours result in increased *ad libitum* food intake in this population (29,34,60–62) Thus, while prolonged sitting and screen-based sedentary behaviours may result in less movement and promote incidental snacking, the resulting

positive energy balance appears to be more related to screen-based sedentary behaviours. This is likely one reason why cardiometabolic disease risk is more consistently associated with screen-based sedentary behaviours than with total sedentary time in the pediatric population, as observed in Chapter 3.

7.2.1 Does sitting *per se* have a negative impact on pediatric health?

Given that neither objectively measured total sedentary time nor prolonged sitting were associated with increased cardiometabolic disease risk in the present thesis, some may ask whether it is worth promoting reductions in sedentary time in the pediatric age group. However, the balance of evidence continues to suggest that high levels of total sedentary time should be avoided by all age groups, including children and youth. Though not equivocal, several cross-sectional and longitudinal studies have found significant associations between sedentary behaviour and cardiometabolic disease risk among children and youth (5,10,44,51–53). As discussed previously, results from Chapter 6 suggest that even in the absence of acute changes in markers of cardiometabolic disease risk, prolonged sitting is still likely to lead to positive energy balance, and potential weight gain. Further, as reported in Chapter 3, certain characteristics of sedentary behaviour (e.g. breaks and/or bout length) may be associated with risk factors for chronic disease in this age group, even if total sedentary time is not. It is also possible that longer (e.g. >8 hours) exposure to uninterrupted sedentary behaviours, or repeated exposures over a period of several days, may result in detectable adverse changes in cardiometabolic disease risk biomarkers.

Even if the potential immediate health impact of total sedentary time in the pediatric age group is disregarded, excessive sitting in childhood may still set children on a path for

poor health in later life. Both total sedentary time and the number of prolonged bouts of sedentary behaviour increase with age (56,63,64); therefore participants with high levels of sedentary behaviour in childhood are likely to experience even greater levels in adulthood. This is troubling, since total (self-reported) sedentary time in adulthood has been linked with increased risk of chronic disease morbidity and mortality (65). Similarly, while we failed to detect an acute deleterious effect of prolonged sitting in the present thesis, it has been reported to reduce insulin sensitivity and glucose tolerance in several studies among adults (25,46,49,66). Further, sedentary time appears to track moderately well through childhood, such that the most sedentary individuals among a population at one time point are likely to remain so later on (67,68). Therefore, while the immediate health impact of prolonged sitting in childhood may be small, it may nevertheless result in increased cardiometabolic disease risk in adulthood.

The findings of the present thesis also lend support to the wording used in Canada's Sedentary Behaviour Guidelines, which were initially released in 2011 (41). The current guidelines focus on screen-based sedentary behaviours, stating that school-aged children and youth should "limit recreational screen time to no more than 2 hours per day; lower levels are associated with additional health benefits". However, they also state that children and youth should "limit sedentary (motorized) transport, extended sitting and time spent indoors throughout the day." Given the findings presented in this thesis and elsewhere in the published literature, the current approach which focuses primarily on screen-based sedentary behaviours, but which also suggests limiting prolonged sitting more generally, seems appropriate (41).

7.3 Opportunities for future research

The results of the present thesis suggest a number of potential areas for future research, several of which have been briefly alluded to previously. These studies are discussed below and grouped according to study methodology.

7.3.1 Observational studies

The results of the present thesis raise several questions that can be explored further using existing cross-sectional databases. Chapter 4 of this thesis is the first study to examine the impact of movement variability in any population. Therefore, replication studies are needed to examine the association between movement variability and health in other populations of children, as well as examining similar relationships in adults, and in populations that are already at elevated risk for chronic disease. Longitudinal studies that examine the relationship between movement variability and health over time could also yield interesting results, and are now feasible given a growing number of longitudinal databases that include objective measures of sedentary time at multiple time points (13,17,44). These studies will help to determine whether movement variability represents a novel and distinct risk factor for chronic disease, and establish whether interventions targeting movement variability are worthy of investigation.

In addition to total movement variability, further studies are also needed to clarify the association of breaks in sedentary time and sedentary bout length with health in the pediatric age-group. Although the study presented in Chapter 3 found significant associations between breaks in sedentary time and clustered cardiometabolic disease risk in our sample of children with a family history of obesity, other studies have failed to detect such an association in samples of the general population (15,16). This suggests

patterns of sedentary behaviour may be particularly important in specific groups of children, and future studies should examine whether this is the case. As with total movement variability, longitudinal studies are likely to be useful in deciding whether interventions targeting breaks in sedentary time are warranted in pediatric or adult populations.

7.3.2 Intervention studies

The results presented in Chapters 5 and 6 suggest that prolonged sitting has a minimal acute impact on metabolic health, energy intake and physical activity in healthy children and youth between the ages of 10 and 14 years. However, it is unclear whether such findings would be observed in older youth, or in those with increased markers of cardiometabolic disease risk. Studies have suggested that prolonged sitting and other forms of sedentary behaviour result in acute increases in markers of cardiometabolic disease risk in healthy young adults (46,49,69–72). It is therefore possible that similar findings may be observed in older teens as well.

It is also possible that the majority of participants in Studies 5 and 6 were in such robust health that 1 day of prolonged sitting was insufficient to result in changes in markers of cardiometabolic disease risk. Acute changes in markers of cardiometabolic disease risk following prolonged sitting may be more consistent, and more clinically relevant, in children and youth with elevated cardiometabolic disease risk at study entry. It is worth noting that individuals living with chronic diseases such as the metabolic syndrome, cardiovascular disease and type 2 diabetes have been largely overlooked by intervention studies targeting sedentary behaviour in both adults and children (26). This omission is

especially glaring given that these populations may be most likely to gain from lifestyle interventions that promote reductions in sedentary behaviour.

When compared to traditional lifestyle interventions that focus on structured physical activity, reductions in sedentary time (or increasing the frequency of breaks in sedentary time) are likely to be easier to implement and may promote better adherence, given that they require few skills, resources or physical effort. Although several studies have examined the impact of reduced screen-based sedentary behaviours on body weight in children and youth (9,73,74), to date no studies have examined the metabolic impact of reducing total sedentary time in any pediatric population. Similarly, while a small number of studies have examined the impact of reducing total sedentary time in adult populations, most interventions to date have focused on feasibility rather than health outcomes (75–82). As a result, the efficacy and effectiveness of reducing total sedentary time remains unclear in both pediatric and adult populations. Studies examining the cardiometabolic impact of reducing total sedentary time, or specific sedentary behaviours, are therefore greatly needed moving forward.

It is also necessary to further investigate whether the health impact of sedentary behaviour can be modified through the adoption of other health behaviours. For example, Stephens et al. reported that the impact of prolonged sitting on insulin sensitivity was cut in half by simply reducing energy intake to match expenditure (46). Similarly, a recent intervention by Duvivier and colleagues (49) reported that an hour of daily vigorous exercise does not eliminate the cardiometabolic impact of prolonged sitting. Aside from the studies presented within the present thesis, to date no studies have examined such issues in the pediatric age group. A better understanding of the

interaction between sedentary behaviour and other health behaviours is needed in order to tailor both clinical and public health messages targeting the prevention of chronic diseases.

Finally, interventions are needed that directly compare the health and behavioural impact of common sedentary behaviours. For example, previous studies have suggested that both video games and TV viewing increase *ad libitum* food intake in children and youth (29,34,60,61). However, to our knowledge these behaviours have yet to be compared to each other, or with other common forms of sedentary behaviour such as reading or texting. As with projects discussed previously, these comparative studies may help target clinical and public health interventions towards those forms of sedentary behaviour which are most closely associated with deleterious health or health behaviours.

7.4 Thesis strengths and limitations

The current thesis has a number of strengths and weaknesses that warrant mention.The studies in this thesis focus on North American children and youth between the ages of 8 and 17 years. Thus, the findings generated from these studies may not generalize to other populations or age-groups. The first two investigations in this thesis (Chapters 3 and 4) were cross-sectional in nature, and therefore cannot make conclusions related to causality. Further, these studies examined only a brief snapshot of each individual's exposure to sedentary behaviour, and thus may not represent the impact of chronic sedentary behaviour.

While the design of the third and fourth studies in this thesis (tightly controlled randomized crossover studies) allow for conclusions related to causality, the results may not generalize to more ecologically valid situations, or to cumulative bouts of prolonged sitting performed over a period of days or weeks. It should also be noted that participants in these studies were healthy and more active than the typical Canadian child, which may have influenced our findings towards the null. As discussed previously, future research should examine whether similar findings are observed in children and youth who are physically inactive, highly sedentary, or who have elevated markers of cardiometabolic disease risk.

Despite the abovementioned limitations, the studies that form this thesis have a number of key strengths which allow them to make an important contribution to our understanding of the health impact of sedentary behaviour in the pediatric age-group. The primary strength of this thesis is that it employed a variety of approaches ranging from large-scale nationally representative surveys to rigorously controlled lab-based intervention studies. This allowed for the examination of associations at the population level, as well as examining causal relationships and possible mechanisms in a more controlled setting. This approach provided insight into the mechanisms underlying key issues (for example, the frequent discrepancies in the strength of health-related associations for self-reported and objectively measured sedentary time in Chapter 3 of this thesis and in the literature more generally) which would not have been possible had only one methodology been applied to all studies. Finally, each study within this thesis also included the direct measurement of important markers of cardiometabolic disease risk, as well as objective measurement of physical activity and sedentary behaviour.

7.5 Conclusions

The present thesis demonstrates that breaks in sedentary time, short bouts of sedentary behaviour and total movement variability are cross-sectionally associated with reduced cardiometabolic disease risk independent of physical activity and sedentary behaviour in certain populations of children and youth. Self-reported TV viewing and computer use were also strongly and independently associated with increased cardiometabolic disease risk in girls and boys, respectively. Despite these cross-sectional findings, in our intervention studies we found no acute impact of prolonged sitting, with or without breaks and structured physical activity, on markers of cardiometabolic disease risk, subsequent energy intake, or physical activity levels in healthy youth aged 10-14 years. The findings of these intervention studies show that prolonged sitting does not have an immediate impact on markers of cardiometabolic disease risk in children and youth, although it may predispose to positive energy balance. Collectively, these findings suggest that optimal levels of cardiometabolic disease risk are most likely to be seen in children who limit their time engaging in screen-based sedentary behaviours, who frequently interrupt their sedentary time, and who have high levels of variability in their movement behaviours.

Chapter 8 – Bibliography for Chapters 1 and 7

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Chapter 9 - Appendices

Appendix A: Ethics approval notices for thesis research projects

9.1 Study 1

File Number: H06-12-20 Date (mm/dd/yyyy): 07/05/2012

Université d'Ottawa University of Ottawa
Bureau d'éthique et d'intégrité de la recherche Office of Research Ethics and Integrity

Ethics Approval Notice

Health Sciences and Science REB

Principal Investigator / Supervisor / Co-investigator(s) / Student(s)

First Name	Last Name	Affiliation	Role
Mark	Tremblay	Health Sciences / Human Kinetics	Supervisor
Travis	Saunders	Health Sciences / Others	Student Researcher
Angelo	Tremblay	Others / Others	Other Collaborator

File Number: H06-12-20

Type of Project: Secondary use of data

Title: The Health Impact of Sedentary Behaviour in Children and Youth

Approval Date (mm/dd/yyyy)	Expiry Date (mm/dd/yyyy)	Approval Type
07/05/2012	07/04/2013	Ia

(Ia: Approval, Ib: Approval for initial stage only)

Special Conditions / Comments:
N/A

[redacted]

File Number: H06-12-20 Date (mm/dd/yyyy): 07/05/2012

Université d'Ottawa University of Ottawa
Bureau d'éthique et d'intégrité de la recherche Office of Research Ethics and Integrity

This is to confirm that the University of Ottawa Research Ethics Board identified above, which operates in accordance with the Tri-Council Policy Statement and other applicable laws and regulations in Ontario, has examined and approved the application for ethical approval for the above named research project as of the Ethics Approval Date indicated for the period above and subject to the conditions listed the section above entitled "Special Conditions / Comments".

During the course of the study the protocol may not be modified without prior written approval from the REB except when necessary to remove subjects from immediate endangerment or when the modification(s) pertain to only administrative or logistical components of the study (e.g. change of telephone number). Investigators must also promptly alert the REB of any changes which increase the risk to participant(s), any changes which considerably affect the conduct of the project, all unanticipated and harmful events that occur, and new information that may negatively affect the conduct of the project and safety of the participant(s). Modifications to the project, information/consent documentation, and/or recruitment documentation, should be submitted to this office for approval using the "Modification to research project" form available at:
http://www.rges.uottawa.ca/ethics/application_dwn.asp

Please submit an annual status report to the Protocol Officer 4 weeks before the above-referenced expiry date to either close the file or request a renewal of ethics approval. This document can be found at:
http://www.rges.uottawa.ca/ethics/application_dwn.asp

[redacted]

Signature:

[redacted]

Riana Marcotte
Protocol Officer for Ethics in Research
For Daniel Lagarec, Chair of the Sciences and Health Sciences REB

[redacted]

9.2 Study 2 (Data publically available, therefore no approval required)

From: Ethics [mailto:ethics@uottawa.ca]
Sent: September 27, 2011 3:16 PM
To: Saunders, Travis
Subject: RE: Secondary data sets

Hello Mr. Saunders,

Thank you for your email.

You will in fact need to submit for Secondary use of data for the studies from Quebec and from CHEO. Because the data from United States is publicly available, it will not be necessary to get a certificate for that data.

Secondary use of data refers to the use in research of information originally collected for a purpose other than the current research purpose (p. 62 of the Tri-Council Policy Statement on Ethics in Research).

Along with the submission form, you will need to submit a 5-page research proposal, as well as a permission letter from the original owner of the data indicating that they grant you permission to use the data for your research purposes.

If you have any questions or comments, please do not hesitate to contact me via email or at extension 5387,

Best regards,

[redacted]

9.3 Studies 3 & 4

CHEO Children's Hospital of Eastern Ontario
Centre hospitalier pour enfants de l'est de l'Ontario

2011 FINAL APPROVAL LETTER

CHEO Site Investigator: Dr. Mark Tremblay	**Department/PSU:** HALO
Date of Final Approval Letter: February 24, 2011	
Protocol Title: The Metabolic Effects of Breaks in Sedentary Time	**REB #:** 11/01E **Other Study #:**
Original Approval Date and REB Meeting Date: January 05, 2011	**Valid until:** January 04, 2012
c.c.: Travis Saunders, CHEO RI Administration	
Protocol Date: February 10, 2011	**Protocol Version:** Version #2
Consent Form Version and Date: Version #2, February 11, 2011	
Date of Health Canada Non-Objection Letter, and Control #: Not Applicable	**Assent Form Version and Date:** Version #2, February 11, 2011
Investigators Brochure/Drug Monograph Version and Date: Not Applicable	**Recruitment Poster Version and Date:**
SITE Specific Restrictions:	**Recruitment Pamphlet Version and Date:**
	Other Study Documentation Received by REB:

This protocol was approved at a meeting of the CHEO Research Ethics Board in which the quorum rules were met and only those REB members who were independent of the investigator(s) conducting the study voted on the final decision.

In fulfilling its mandate, the CHEO REB is guided by: Tri-Council Policy Statement; ICH Good Clinical Practice Practices: Consolidated Guideline; Applicable laws and regulations of Ontario and Canada (e.g., Health Canada Division 5 of the Food and Drug Regulations & the Food and Drugs Act - Medical Devices Regulations).

Final approval is granted with the understanding that the investigator agrees to comply with the following requirements:

- The investigator must conduct the study in compliance with the protocol and any additional conditions set out by the Board.
- The investigator must not implement any deviation from, or changes to, the protocol without the approval of the REB except where necessary to eliminate an immediate hazard to the research subject, or when the change involves only logistical or administrative aspects of the study (e. g., change of telephone number or research staff). As soon as possible, however, the protocol deviation form and, if appropriate, the proposed protocol amendment(s) should be submitted to the Board for review.
- The investigator must, prior to use, submit to the Board changes to the study documentation, e.g., changes to the informed consent letters, recruitment materials. Should major revisions to the consent form be made, the investigator agrees to re-consent those subjects who have originally consented to the study and who wish to continue on the study.
- For clinical drug or device trials, investigators must promptly report to the REB all adverse events that are both serious and unexpected (SAEs). For SAE reports on CHEO patients, the investigator must also comply with the hospital-wide Policy regarding, Procedures For Considering Medical Error In The Differential Diagnosis of Severe Adverse Events (SAE) Associated with the Drugs Administered in a Clinical Trial (see http://cheonet/data/1/rec_docs/3792_Medical%20Error%20Policy%20revised%20january%2020061.doc).
- For all other research studies, investigators must promptly report to the REB all unexpected and untoward occurrences (including the loss or theft of study data and other such privacy breaches).
- Investigators must promptly report to the REB any new information regarding the safety of research subjects (e.g., changes to the product monograph or investigator's brochure for drug trials). Where available, any reports produced by Data Safety Monitoring Board should be submitted to the REB.
- Investigators must notify the REB of any study closures (temporary, premature or permanent), in writing along with an explanation of the rationale for such action.
- Investigators must submit an annual renewal report to the REB 30 days prior to the expiration date stated above.
- Investigators must submit a final report at the conclusion of the study.
- Investigators must provide the Board with French versions of the consent form, unless a waiver has been granted.

[redacted]

Dr. Carole Gentile, C.Psych.
Chair, Research Ethics Board
CG/smeh

File Number: H05-11-11

Date (mm/dd/yyyy): 07/13/2011

Université d'Ottawa
Bureau d'éthique et d'intégrité de la recherche

University of Ottawa
Office of Research Ethics and Integrity

Ethics Approval Notice

Health Sciences and Science REB

Principal Investigator / Supervisor / Co-investigator(s) / Student(s)

First Name	Last Name	Affiliation	Role
Mark	Tremblay	Health Sciences / Others	Principal Investigator
Jean-Philippe	Chaput	Medicine / Medicine	Other Collaborator
Rachel	Colley	Others / Others	Other Collaborator
Éric	Doucet	Health Sciences / Human Kinetics	Other Collaborator
Gary	Goldfield	Medicine / Medicine	Other Collaborator
Glen	Kenny	Health Sciences / Human Kinetics	Other Collaborator
Travis	Saunders	Health Sciences / Others	Project Coordinator

File Number: H 05-11-11

Type of Project: Professor

Title: The Metabolic Impact of Breaks in Sedentary Time

Approval Date (mm/dd/yyyy)	Expiry Date (mm/dd/yyyy)	Approval Type
06/13/2011	06/12/2012	Ia

(Ia: Approval, Ib: Approval for initial stage only)

Special Conditions / Comments:
N/A

[redacted]

File Number: H05-11-11

Date (mm/dd/yyyy): 07/13/2011

Université d'Ottawa University of Ottawa
Bureau d'éthique et d'intégrité de la recherche Office of Research Ethics and Integrity

This is to confirm that the University of Ottawa Research Ethics Board identified above, which operates in accordance with the Tri-Council Policy Statement and other applicable laws and regulations in Ontario, has examined and approved the application for ethical approval for the above named research project as of the Ethics Approval Date indicated for the period above and subject to the conditions listed the section above entitled "Special Conditions / Comments".

During the course of the study the protocol may not be modified without prior written approval from the REB except when necessary to remove subjects from immediate endangerment or when the modification(s) pertain to only administrative or logistical components of the study (e.g. change of telephone number). Investigators must also promptly alert the REB of any changes which increase the risk to participant(s), any changes which considerably affect the conduct of the project, all unanticipated and harmful events that occur, and new information that may negatively affect the conduct of the project and safety of the participant(s). Modifications to the project, information/consent documentation, and/or recruitment documentation, should be submitted to this office for approval using the "Modification to research project" form available at:
http://www.rges.uottawa.ca/ethics/application_dwn.asp

Please submit an annual status report to the Protocol Officer 4 weeks before the above-referenced expiry date to either close the file or request a renewal of ethics approval. This document can be found at:
http://www.rges.uottawa.ca/ethics/application_dwn.asp

[redacted]

[redacted]

Germain Zongo
Protocol Officer for Ethics in Research
For Dr. Daniel Lagarec, Chair of the Health Sciences and Sciences REB

[redacted]

Appendix B: Permission for republication

Chapters 2 and 5 have been accepted for publication in Elsevier journals, which do not require permission in order to include a manuscript in a thesis document (see below). Chapters 3 and 6 are published in journals which allow the author to maintain copyright, therefore permission is not required for these studies either.

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Use for internal training by author's company	Yes	Yes with full acknowledgement of final article	Yes with full acknowledgement of final article
Distribution to colleagues for their research use	Yes	Yes	Yes
Use in a subsequent compilation of the author's works	Yes	Yes with full acknowledgement of final article	Yes with full acknowledgement of final article
Inclusion in a thesis or dissertation	Yes	Yes with full acknowledgement of final article	Yes with full acknowledgement of final article
Reuse of portions or extracts from the article in other works	Yes	Yes with full acknowledgement of final article	Yes with full acknowledgement of final article
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Appendix C: Related publications

All of the publications in this appendix have been authored or co-authored by Travis Saunders during his PhD studies. Although they are not a part of the thesis itself, they are related to the health impact of sedentary behaviour, and are therefore included to provide additional context to the thesis. All of these papers have been published in journals with Creative Commons Attribution licenses, and therefore do not require permission for republication.

Hindawi Publishing Corporation
Journal of Nutrition and Metabolism
Volume 2012, Article ID 712435, 12 pages
doi:10.1155/2012/712435

Review Article

Acute Sedentary Behaviour and Markers of Cardiometabolic Risk: A Systematic Review of Intervention Studies

Travis J. Saunders,[1,2] Richard Larouche,[1,2] Rachel C. Colley,[1] and Mark S. Tremblay[1,2,3]

[1] *Healthy Active Living and Obesity Research Group, Children's Hospital of Eastern Ontario Research Institute, Ottawa, ON, Canada K1H 8L1*
[2] *School of Human Kinetics, University of Ottawa, Ottawa, ON, Canada K1N 6N5*
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Correspondence should be addressed to Travis J. Saunders, [redacted]

Received 24 January 2012; Accepted 28 March 2012

Academic Editor: Maria Luz Fernandez

North Americans spend half their waking hours engaging in sedentary behaviour. Although several recent interventions suggest that short bouts of uninterrupted sedentary behaviour may result in acute increases in cardiometabolic risk, this literature has not been reviewed systematically. This study performed a systematic review of the impact of uninterrupted sedentary behaviour lasting ≤7 days on markers of cardiometabolic risk (insulin sensitivity, glucose tolerance, and fasting insulin, glucose, and lipid levels) in humans. Interventions were identified through systematic searches of Medline and Embase and screened by 2 independent reviewers. A total of 25 interventions were identified that examined the impact of imposed sedentary behaviour on biomarkers of interest. The majority of these studies focused on healthy young men, with very little identified research on females or other age groups. We found consistent, moderate quality evidence that uninterrupted sedentary behaviour ≤7 days results in moderate and deleterious changes in insulin sensitivity, glucose tolerance, and plasma triglyceride levels. In contrast, there is inconsistent, very low-quality evidence linking uninterrupted sedentary behaviour with changes in insulin, glucose, and HDL- and LDL-cholesterol levels. These findings suggest that uninterrupted bouts of sedentary behaviour should be avoided in order to prevent or attenuate transient increases in metabolic risk.

1. Introduction

High levels of chronic sedentary behaviour are associated with increased risk of obesity [1, 2], diabetes [1, 3], cardiovascular disease [4–6], some cancers [7], and even mortality [3–5]. For example, it has been reported that sedentary behaviour is prospectively associated with increased risk of all-cause and cardiovascular disease mortality, and that these associations remain significant after control for physical activity, diet, and smoking [4]. These findings suggest that sedentary behaviour should not be viewed as simply the lack of physical activity but may instead represent an independent and distinct risk factor for chronic disease.

In addition to the health impact of chronic sedentary behaviour, recent evidence suggests that increases in metabolic risk may be apparent following bouts of uninterrupted sedentary behaviour lasting just a few days in length [8–11]. Stephens et al. reported that a single day of uninterrupted sitting resulted in a 39% reduction in whole body insulin action in healthy adults [11]. Similarly, Hamburg et al. observed that 5 days of continuous bed rest produced deleterious changes in cholesterol, triglyceride, glucose, and insulin levels and reduced insulin sensitivity [10]. These findings are supported by work in animal models, which suggest that just 6–24 hours of sedentary behaviour results in significant reductions in lipoprotein lipase activity [12] and insulin sensitivity [13, 14] in skeletal muscle.

Although several narrative reviews have discussed the acute changes in metabolic risk following short-term exposure to uninterrupted sedentary behaviour [8, 9, 15, 16],

the published literature in this area has yet to be examined systematically. Therefore, we conducted a systematic review examining the impact of uninterrupted sedentary behaviour lasting ≤7 days (operationally defined as an "acute" bout) on insulin sensitivity, glucose tolerance, and lipid, glucose, and insulin levels in humans.

2. Methods

2.1. Study Criteria. To be included in this paper, a study had to examine at least one of the following risk markers in humans: insulin sensitivity, glucose tolerance, or fasting insulin, glucose, or lipid levels. Uninterrupted sedentary behaviour had to be imposed by the researchers for a period lasting 7 days or less. Studies examining longer (e.g., chronic) bouts of sedentary behaviour were excluded as it was felt that it would be inappropriate to consider the impacts of both acute and chronic sedentary behaviour in a single systematic review, given the large amount of heterogeneity that this would introduce into the methodologies of included studies. Only intervention studies (both randomized and nonrandomized) that imposed on participants a controlled bout of sedentary behaviour were included in this paper.

For the purposes of this paper, sedentary behaviour was defined as a distinct class of waking behaviours characterized by little physical movement and low-energy expenditure (≤1.5 METs), as well as a sitting or reclining posture [9]. Eligible forms of sedentary behaviour included sitting, bed rest (head-up, horizontal, and head-down), and casting (e.g., having one or more legs immobilized in a cast). There were no restrictions placed on the age or sex of participants. Only articles published in English or French were included in the present review, and no limits were placed on the date of publication. The review methodology was prospectively registered in PROSPERO (Registration number: CRD42011001431).

2.2. Search Strategy. Literature searches were performed using Ovid Medline and Ovid Embase in March of 2012. The search strategy was created with the help of a research librarian and run by TJS (see **Algorithm 1**). Potentially relevant articles were also identified by 6 key informants and through the authors' personal reference libraries. Articles were extracted as text files from the Ovid interface and imported into Reference Manager (Thompson Reuters, San Fransisco, CA, USA). Duplicate articles were first removed using the Ovid interface, and any remaining duplicates were removed manually. Once imported into Reference Manager, conference abstracts were also removed from the database.

Titles and abstracts of articles identified through the search were reviewed by two authors (T. J. Saunders and R. Larouche) using Reference Manager. Any articles identified as being potentially relevant by either reviewer were obtained for further screening. The full text of these articles was then reviewed independently by TJS and RL to determine whether the article met the *a priori* review inclusion criteria. All decisions at this stage were made by consensus and any discrepancies between the two reviewers were resolved through discussion. In this paper consensus was reached for all included articles.

2.3. Data Extraction and Analysis. Data was extracted by T. J. Saunders and verified by R. Larouche. Information was extracted regarding study design (year, methodology, country, number of participants, duration of sedentary behaviour, age), modality of sedentary behaviour, risk factors examined, and main findings. Reviewers were not blinded to the authors or journals when extracting data. The primary summary measure was the mean difference in each outcome measure (or mean change in nonrandomized interventions) following exposure to acute sedentary behaviour. Where possible, effect sizes were calculated using Cohen's *d*. We defined an effect size of ≤0.20 as small, an effect of 0.21–0.80 as moderate, and effects ≥0.81 as large. For the purposes of this paper, positive effect sizes represent increased cardiometabolic risk (e.g., increased fasting triglyceride levels), while negative effect sizes represent reduced risk.

Following data extraction it became clear that the interventions included in the present paper were very heterogeneous in terms of the length of exposure, the type of sedentary behaviour that was examined, and even the measurement of individual risk factors (e.g., insulin sensitivity was assessed using HOMA, QUICKI, whole body insulin action, oral glucose tolerance tests, and hyperinsulinemic clamps). Thus, we believe that meta-analyses or pooling of data across studies would be inappropriate and have therefore performed a qualitative synthesis of the evidence instead.

Forest plots were created using Review Manager 5.1 (The Nordic Cochrane Centre, The Cochrane Collaboration) to display the relationship between sedentary behaviour and each outcome of interest. Studies assessing glucose tolerance and insulin sensitivity employed a wide range of methodologies and units of measurement, and plots for these outcomes are therefore presented as percent mean difference, while all other outcomes are presented as mean difference with 95% confidence interval. Studies which did not provide raw data were not included in forest plots.

2.4. Quality of Evidence. The risk of bias and strength of evidence from individual studies was assessed using the Downs and Black Checklist [17]. This 27 point checklist assesses the strength of reporting, external validity, internal validity, and power. As some questions are worth more than one point, the maximum score that a study can receive is 32.

The quality of evidence for each outcome was assessed as high, moderate, low, or very low using the GRADE approach [18]. In this approach, randomized trials begin as high-quality evidence and observational studies begin as low-quality evidence. For the purposes of this paper, nonrandomized interventions were considered as observational studies. Following the initial rating based on study design, the quality of evidence was then rated up or down for apparent risk of bias, imprecision, inconsistency, indirectness, or suspicion of publication bias. Risk of bias was assessed using Review Manager Version 5.1 (The Nordic Cochrane Centre, The

(1) sedentar$.tw.
(2) ((chair or sitting or car or automobile or auto or indoor or in-door or screen or computer) adj time).tw.
(3) bed rest/
(4) weightlessness simulation/
(5) physical inactivit* .tw.
(6) sedentary lifestyle/
(7) weightlessness/
(8) sitting/
(9) Suspension/
(10) Weight bearing/
(11) Head down tilt/
(12) posture/
(13) immobilization/
(14) or/(1)–(13)
(15) cardiovascular fitness.tw.
(16) metabolic syndrome x/
(17) Insulin Resistance/ or insulin/
(18) (metabolic cardiovascular syndrome or metabolic syndrome or syndrome x).tw.
(19) exp cholesterol, hdl/ or exp lipoproteins, ldl/ or exp lipoproteins, vldl/
(20) Triglycerides/
(21) Glucose Intolerance/ or Glucose Clamp Technique/ or Glucose Tolerance Test/ or Blood Glucose/ or glucose homeostasis/
(22) lipid metabolism/
(23) or/(15)–(22)
(24) (14) and (23)
(25) (24) not (animal/ not human/)
(26) remove duplicates from (25)

Algorithm 1: Medline search strategy.

Cochrane Collaboration), and GRADE was assessed using GRADEpro Version 3.6 (GRADE Working Group).

3. Results

3.1. Description of Studies. After deduplication and the removal of conference abstracts the search strategy retrieved 5,670 articles for initial screening (**Figure 1**). To this, 16 additional articles that were identified through key informants were added, bringing the total number of potential articles to 5,686. Initial screening of titles and abstracts identified 85 articles that received a detailed assessment of the full text article. Reasons for excluding studies included an ineligible exposure (e.g., the bout of sedentary behaviour exceeded 7 days, or simply investigated the impact of reducing structured physical activity in active individuals, without actually imposing sedentary behaviour) ($n = 26$), the article being written in a language other than English or French ($n = 12$), ineligible outcome ($n = 7$), the article being a review or commentary ($n = 10$), and "other" ($n = 2$). Some articles were excluded for multiple reasons.

A total of 29 articles reporting data from 25 independent interventions met all inclusion criteria and are presented in the current review. Nineteen of the identified interventions were nonrandomized trials, 4 were randomized crossover studies (e.g., participants served as their own controls), and 2 were randomized controlled trials. The studies included a total of 368 participants (309 males and 59 females), who were recruited from 12 countries across North America (USA), Europe (Denmark, France, Bulgaria, Russia, Greece, Sweden, Poland, and Slovakia, Norway), Asia (Japan), and Oceania (Australia). Participants ranged from 18 to 72 years of age, although the average age of participants was under 35 years for all but 3 studies, and under 30 for all but 7 studies. Sixteen studies employed head-up or horizontal bed rest, 5 employed head-down bed rest, 4 employed sitting, and one employed casting (one employed both sitting and head-down bed rest). The smallest studies had 5 participants [**19**, **20**] and the largest had 38 [**21**]. The mean number of participants per study was 15.1 ± 10.1, and the median was 10.

Three studies examined the impact of 2, 4, and 5 hours of uninterrupted sedentary behaviour on biomarkers of interest, respectively; all other studies examined the impact of 1 day or more. Six studies examined the impact of 1 day of sedentary behaviour, 4 examined 2 days, 7 examined 3 days, 2 examined 4 days, 6 examined 5 days, 2 examined 6 days, and 8 examined the impact of 7 days of sedentary behaviour (6 studies collected data at multiple time-points). Characteristics of individual studies are presented in **Table 1**.

3.2. Fasting Insulin. Two randomized crossover studies ($n = 22$) [**11**, **22**] and 12 nonrandomized intervention studies ($n = 185$) [**10**, **21**, **23–32**] examined the impact of sedentary behaviour on fasting insulin levels (**Figure 2**). Neither

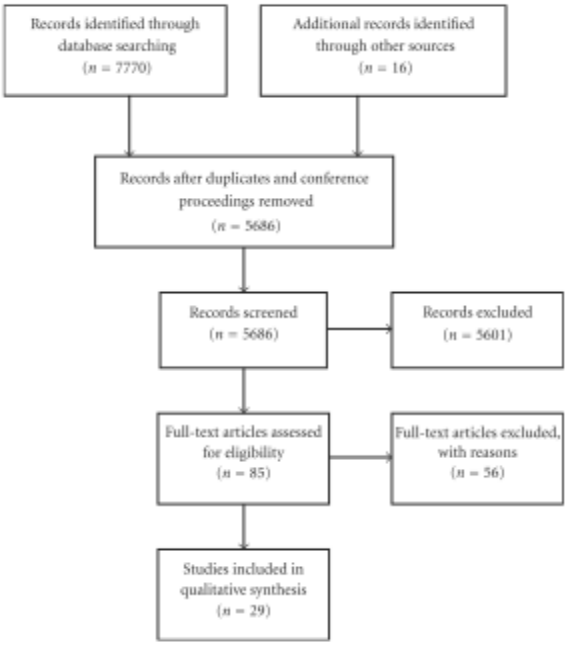

FIGURE 1: Flow of articles through the search process.

randomized crossover study observed significant changes in insulin levels as a result of uninterrupted sedentary behaviour, despite having effect sizes in the moderate range. Stephens et al. [11] reported that fasting insulin levels were 47.6 ± 11.6 pmol/L following one full day (17 hours) of uninterrupted sitting, compared to 39.3 ± 16.3 pmol/L following a day that included as little sitting as possible (~ 6 hours of sitting spread throughout the day) in a group of 12 healthy young adults. Duran-Valdez et al. [22] also observed nonsignificant increases following 2 days of strict bed rest in both healthy participants (71.4 ± 42.6 versus 84.1 ± 36.4 pmol/L) and those with type 2 diabetes (79.2 ± 50.3 versus 106.3 ± 46.6 pmol/L).

Three nonrandomized interventions reported significant increases in insulin levels ranging from 26 to 47% following uninterrupted sedentary behaviour [10, 23, 28] while 8 reported no change [21, 24–27, 29, 30]. The interventions that observed a change in insulin levels tended to impose uninterrupted sedentary behaviour for a longer period of time than those that found no change (6.0 ± 1.0 days versus 3.9 ± 2.6 days), although the mean number of participants (14.0 ± 7.0 versus 16.4 ± 13.3) and the quality of the studies did not appear to differ across the interventions. Effect sizes for these nonrandomized interventions ranged from −0.16 to 0.95, with all but one study reporting effect sizes in the small and moderate ranges.

Given the aforementioned evidence from both randomized and nonrandomized interventions, we conclude that an acute bout of uninterrupted sedentary behaviour may result in a small-to-moderate increase in fasting insulin levels. However, the inconsistency of this effect and the lack of a statistical significance in randomized interventions leads us to conclude that the quality of this evidence is very low (Table 2).

3.3. Fasting Glucose. Two randomized crossover studies (n = 22) [11, 22], one randomized controlled trial (n = 20) [33], and 14 nonrandomized intervention studies (n = 149) [10, 20, 23–30, 34–36] examined the impact of uninterrupted sedentary behaviour on fasting glucose levels. The one randomized controlled trial reported that, in comparison to ambulatory controls, plasma glucose levels were elevated by 34% following 7 days of uninterrupted bed rest [33]. The effect size in this randomized controlled trial was greater than 1, signifying a large effect. In contrast, neither randomized crossover study reported any change in fasting glucose levels following uninterrupted sedentary behaviour [11, 22]. One of these randomized crossover studies reported a moderate effect size of 0.43, [11], while the other reported small effect sizes of 0.07 and 0.14 in healthy participants and those with type 2 diabetes, respectively [22].

Table 1: Characteristics of included studies.

Reference	Design	First author	Year	Country	n (M/F)	Age range	Mean age	Modality	Duration	Outcomes	Downs and Black
[37]	RCT	Yaroshenko	1998	Greece	30 (30/0)	22–26	24.8	BR	7 days	TG	24
[33]	RCT	Zorbas	1999	Bulgaria	30 (30/0)	22–26	24.3	BR	7 days	FG	24
[22]	RCO	Duran-Valdez	2008	USA	10 (2/8)	24–72	46.2	BR	2 days	FI, FG, IS	21
[11]	RCO	Stephens	2010	USA	12 (6/6)	19–32	26.1	SIT	1 day	FI, FG, IS	24
[39]	RCO	Dunstan	2012	Australia	19 (11/8)	45–65	53.8	SIT	5 hours	IS, GT	27
[42]	RCO	Nygaard	2009	Norway	13 (0/13)	>50		SIT	2 hours	GT	23
[21]	NT	Alibegovic	2010	Denmark	38 (38/0)		25.0	BR	7 days	FI	23
[31]	NT	Alibegovic	2009	Denmark	33 (33/0)		25.6	BR	7 days	FI	23
[23, 47]	NT	Blanc	2000	France	16 (8/8)		30.2	HDBR	6 days	FI, FG, IS, GT	23
[35]	NT	Dolkas	1977	USA	7 (7/0)	19–22	20.0	BR	4 days	FG	21
[25]	NT	Barbe	1999	France	8 (8/0)	23–31	27.1	HDBR	5 days	FI, FG	21
[10]	NT	Hamburg	2007	USA	20 (14/6)		30.7	BR	5 days	FI, FG, IS, TG	23
[20]	NT	Katkov	1979	Russia	5 (5/0)		34.0	BR	5 days	FG	17
[41]	NT	Kiilerich	2011	Denmark	6 (6/0)	22–36	28.7	BR	7 days	IS, GT	
[36]	NT	Ksinantova	2002	Slovakia	15 (15/0)		34.0	HDBR	4 days	FG	20
[29]	NT	Lipman	1972	USA	7 (7/0)	18–20		BR	3 days	FI, IS, GT	18
[32]	NT	Kanikowska	2010	Japan	8 (8/0)		27.0	HDBR	5 days	FI, FG, IS	
[27, 40]	NT	Mikines	1989	Denmark	6 (6/0)		25.0	BR	7 days	FI, FG, IS, GT	20
[30]	NT	Moro	2007	France	8 (8/0)	22–27	23.0	HDBR, SIT	4 hours	FI, FG	20
[34]	NT	Navasiolava	2010	Russia	8 (8/0)		23.0	BR	7 days	FG, TG, HDL, LDL	22
[26]	NT	Nygren	1997	Sweden	6 (6/0)		24.1	BR	1 day	FI, FG, IS	22
[19]	NT	Richter	1989	Denmark	5 (5/0)	22–24		CAST	7 days	IS	20
[24, 48, 49]	NT	Smorawinski	1996	Poland	29 (29/0)		20.10	BR	3 days	IS, GT	19
[28]	NT	Stuart	1988	USA	6 (6/0)	21–28	23.0	BR	7 days	FI, FG, IS, GT	22
[38]	NT	Yanagibori	1997	Japan	23 (13/10)	19–25		BR	3 days	IS, GT, TG, HDL, LDL	22

RCT: randomized controlled trial; RCO: randomized crossover; NT: nonrandomized trial; M: male; F: female; HDBR: head-down bed rest; 22BR: horizontal or head-up bed rest; SIT: sitting; CAST: casting; FG: fasting glucose; FI: fasting insulin; TG: triglycerides; HDL: HDL-cholesterol; LDL: LDL-cholesterol; IS: insulin sensitivity; GT: glucose tolerance.
When an intervention was described in more than one paper, the author name and year are taken from the earliest publication.

One nonrandomized intervention observed a significant increase in glucose levels of moderate size [10], one reported a significant reduction of moderate size [36], and one intervention observed moderate and large reductions in males and females, respectively, although this change was only significant in females [23]. The 11 other intervention studies did not observe any significant change in fasting glucose levels following uninterrupted sedentary behaviour [20, 24–30, 32, 34, 35]. The effect sizes among these 11 studies ranged from −1.21 to 0.47.

Given the evidence provided by 17 separate intervention studies, we conclude that an acute bout of uninterrupted sedentary behaviour may result in a small-to-moderate increase in fasting glucose levels. However, the high level of inconsistency from both randomized and nonrandomized interventions leads us to conclude that this evidence is of very low quality.

3.4. Fasting Triglycerides. One randomized controlled trial ($n = 30$) [37] and 3 nonrandomized interventions ($n = 51$) [10, 34, 38] assessed the impact of uninterrupted sedentary behaviour on fasting triglyceride levels (Figure 3). The randomized controlled trial [37] exposed 20 men to one week

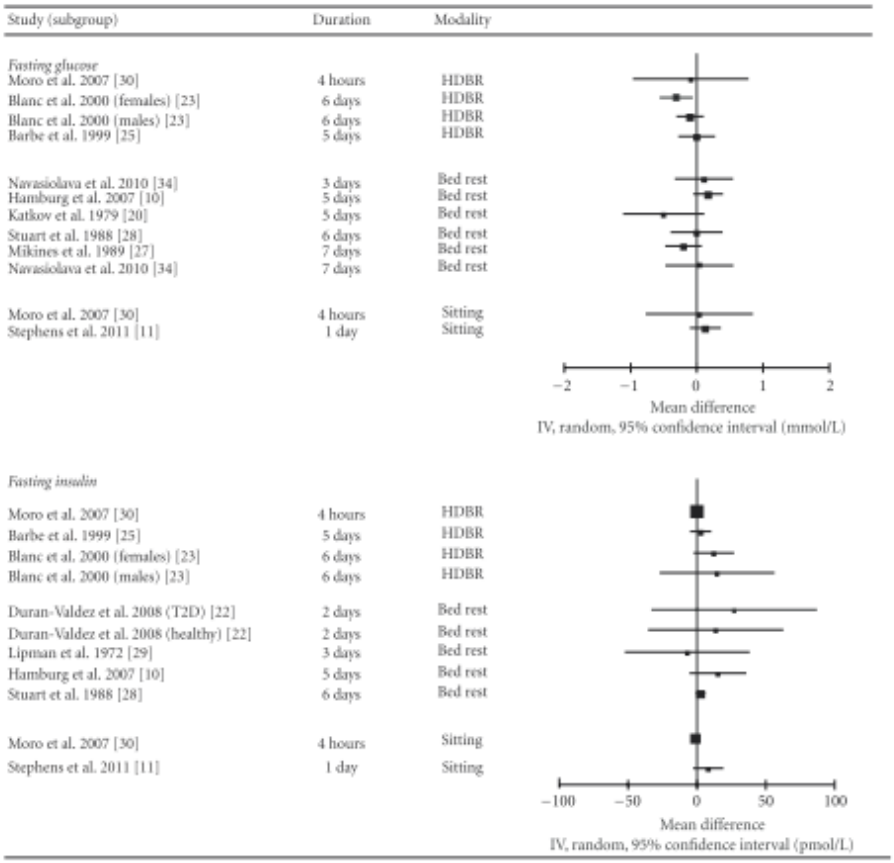

Figure 2: Forest plot of mean differences of fasting glucose and insulin values between sedentary behaviour and control conditions (sedentary behaviour-control).

of bed rest and assessed triglyceride levels on days 1, 3, and 7. The 20 men in the experimental group were further split into two groups of 10—those who knew when their bed rest would begin (*acute* bed rest), and those who were not told when it would begin (*rigorous* bed rest). In comparison to the control group, triglyceride levels were significantly elevated by 30.2% in the acute group after one day, although no change was observed in the rigorous group. Following 3 days of bed rest, triglyceride levels were elevated by 15.2% and 23.6% in the acute and rigorous bed rest groups, respectively. At the completion of 1 week of bed rest, triglyceride levels remained elevated by 36.8% and 31.9% in the acute and rigorous bed rest groups in comparison to the control group. The effect size for sedentary behaviour in this intervention was above 1 for both intervention groups on days 1, 3, and 7, indicating a large effect.

The three nonrandomized interventions also found that acute sedentary behaviour resulted in significant increases in triglyceride levels [10, 34, 38]. Hamburg et al. [10] reported that triglyceride levels were elevated by 34.8% following 5 days of bed rest in 20 healthy men and women. Navasiolava et al. [34] observed that although no change in triglyceride levels was observed following 3 days of acute sedentary behaviour in a group of 8 male participants, triglyceride levels were 58.9% higher than baseline on day 7. Finally, Yanagibori et al. [38] found that triglyceride levels were elevated by 38.1% following 3 days of bed rest in men, but not women. With the exception of male participants in one study [38], the effect sizes reported in these nonrandomized interventions were all moderate to large.

Given the large and relatively consistent changes in triglyceride levels reported by both a randomized controlled

Table 2: Summary of key evidence.

Risk factor	Number of studies	Number of participants (M/F)	Size of effect	Quality of evidence
Insulin sensitivity	11	161 (118/43)	Moderate-to-Large	Moderate quality
Triglycerides	4	81 (65/16)	Moderate-to-Large	Moderate quality
Glucose tolerance	6	119 (83/36)	Moderate-to-Large	Moderate quality
HDL-cholesterol	3	51 (35/16)	Moderate	Very low quality
Fasting insulin	14	207 (187/20)	Small-to-Moderate	Very low quality
Fasting glucose	17	191 (163/28)	Small-to-Moderate	Very low quality
LDL-cholesterol	2	28 (22/6)	Moderate	Very low quality

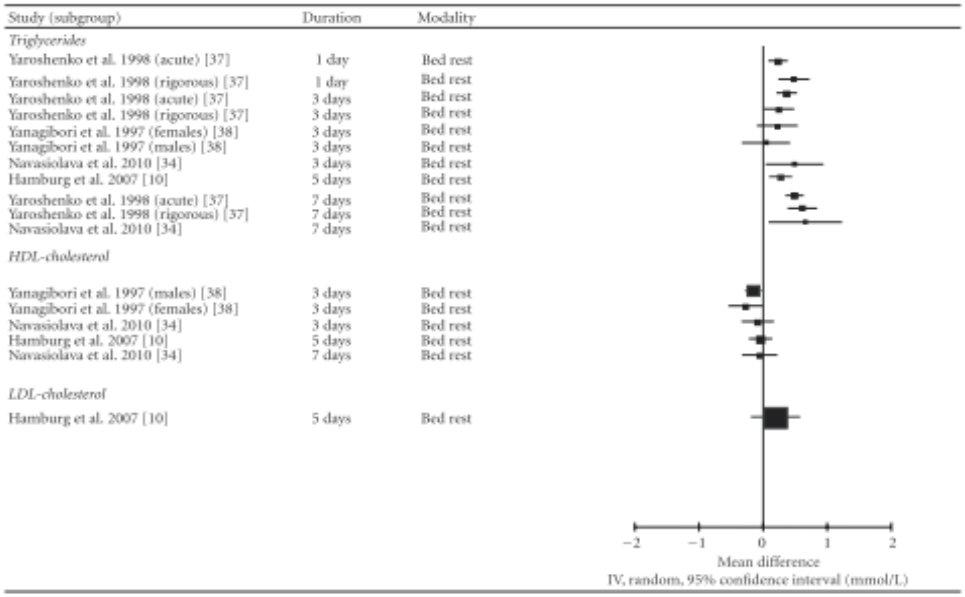

Figure 3: Forest plot of mean differences of fasting lipid levels between sedentary behaviour and control conditions (sedentary behaviour-control).

trial and nonrandomized interventions, we conclude that acute bouts of uninterrupted sedentary behaviour result in a moderate-to-large increase in circulating triglyceride levels and that the available evidence is of moderate quality.

3.5. Fasting HDL-Cholesterol. Three nonrandomized interventions ($n = 51$) [10, 34, 38] reported on the effect of uninterrupted sedentary behaviour ranging from 3 to 7 days on HDL-cholesterol levels. Two interventions reported non-significant reductions in HDL-cholesterol levels following sedentary behaviour [10, 34] while one study [38] reported significant reductions of 11.5% and 19.3% in men and women, respectively, following 3 days of bed rest. The effect sizes in these studies ranged from 0.09 to 0.84, suggesting that acute bouts of uninterrupted sedentary behaviour may result in moderate reductions in HDL-cholesterol levels. However, given the inconsistency of these findings and the lack of data from randomized interventions, we conclude that the available evidence is of very low quality.

3.6. LDL-Cholesterol. Two nonrandomized interventions ($n = 28$) [10, 34] examined the relationship between uninterrupted sedentary behaviour and changes in LDL-cholesterol levels following 3 [34], 5 [10], and 7 [34] days of sedentary behaviour. Although the studies reported moderate-sized increases in LDL-cholesterol levels at all time points,

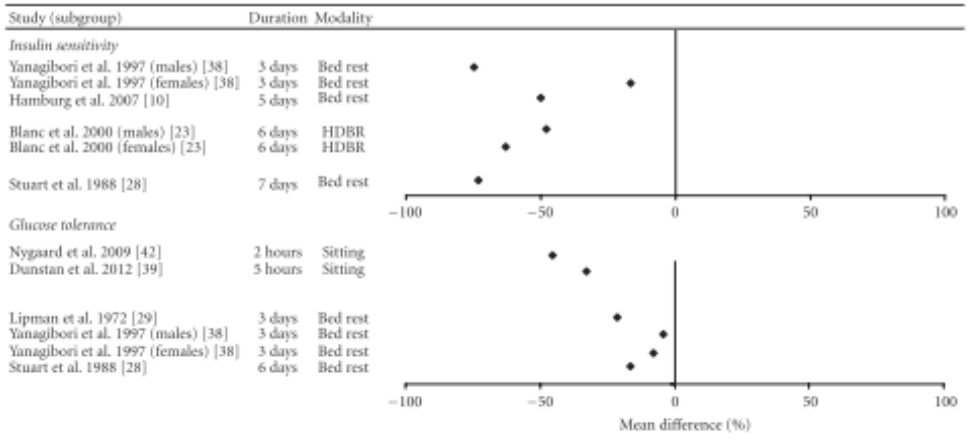

Figure 4: Forest plot of percent mean differences of insulin sensitivity and glucose tolerance between sedentary behaviour and control conditions (sedentary behaviour-control).

none of these increases were statistically significant. Thus, while the available evidence suggests that an acute bout of uninterrupted sedentary behaviour may result in a moderate increase in LDL-cholesterol levels, the quality of this evidence is very low.

3.7. Insulin Sensitivity. Three randomized crossover studies ($n = 41$) [11, 22, 39] and 10 nonrandomized interventions ($n = 120$) [10, 19, 23, 24, 28, 29, 32, 38, 40, 41] examined the relationship between acute bouts of uninterrupted sedentary behaviour and measures of insulin sensitivity in healthy adults (Figure 4). The measures employed included HOMA [10, 32, 41], QUICKI [22], insulin-stimulated glucose uptake [11], insulin sensitivity index [10], insulin area under-the-curve (AUC) during oral glucose tolerance tests or standardized meals [23, 24, 28, 38, 39, 41], and hyperinsulinemic euglycemic clamps [19, 28, 40]. The crossover studies measured insulin sensitivity during 5 hours of sedentary behaviour [39], as well as before and after 1 [11, 22] and 2 [22] days of sedentary behaviour. The nonrandomized interventions assessed insulin sensitivity before and after 3 [24, 29, 38], 5 [10], 6 [23], and 7 [19, 28, 40, 41] days of sedentary behaviour.

Two of the three randomized crossover studies [11, 39] reported that uninterrupted sedentary behaviour had a deleterious effect on insulin sensitivity. Stephens et al. reported that insulin-stimulated glucose uptake was 39% lower following a day of acute sitting in a group of 12 healthy adults, in comparison to a day that minimized sitting [11]. Similarly, Dunstan et al. reported that the insulin AUC following a standardized meal was increased by 30% following 5 hours of uninterrupted sitting in a group of 19 overweight adults, in comparison to 5 hours of sitting which was broken up with periodic light-intensity walk breaks [39]. The third crossover study [22] reported that 1 day of sedentary behaviour resulted in a nonsignificant reduction in QUICKI scores in 5 healthy adults and a nonsignificant increase in 5 adults with type 2 diabetes. A significant reduction in insulin sensitivity was observed following two days of bed rest in participants with type 2 diabetes, but not in healthy adults. The significant reduction in participants with type 2 diabetes following 2 days of bed rest was of moderate size, while the nonsignificant reduction in healthy participants was small.

Eight of the 10 nonrandomized trials reported significant reductions in insulin sensitivity ranging from 12.5% to 100% following uninterrupted sedentary behaviour. For example, Hamburg et al. [10] reported that HOMA insulin sensitivity was reduced by 50% following 5 days of bed rest in 20 healthy adults, while the insulin sensitivity index was reduced by 12.5% in the same group of subjects. Similarly, Yanagibori et al. [38] report that insulin AUC during an oral glucose tolerance test was increased by 16.6% in 10 men and 74.9% in 7 women following 3 days of bed rest. The effect sizes from these nonrandomized interventions ranged from 0.34 to 3.3.

Although the majority of studies (9/12) examining insulin sensitivity had no control group, the effect sizes of the sedentary behaviour interventions were consistently moderate to large. The results were also consistent, with 10 of 12 published studies reporting a reduction in insulin sensitivity in at least one subgroup of participants. Thus, we conclude that acute bouts of uninterrupted sedentary behaviour are likely to result in a moderate-to-large reduction in insulin sensitivity and that the available evidence is of moderate quality.

3.8. Glucose Tolerance Tests. Two randomized crossover studies ($n = 32$) [39, 42] examined the impact of 2 [42]

and 5 hours [39] of prolonged sitting on glucose AUC in response to a standard meal, while seven nonrandomized interventions ($n = 87$) [23, 24, 27–29, 38, 41] examined the impact of uninterrupted sedentary behaviour lasting 3 [24, 38], 6 [23], and 7 [27, 28, 41] days on measures of glucose tolerance.

Both of the randomized crossover studies reported that uninterrupted sitting resulted in significant increases in glucose AUC in response to a standardized meal. Nygaard et al. reported that 2 hours of sitting resulted in a 45% increase in the glucose response to a standard meal in a group of 13 elderly women, in comparison to a combination of sitting and walking at a self-selected "very light" intensity [42]. Similarly, Dunstan et al. reported that the glucose AUC following a test meal was 33% higher following 5 hours of prolonged sitting, in comparison to 5 hours of sitting which was broken up with periodic light-intensity walk breaks [39].

Five of seven nonrandomized studies reported significant reductions in glucose tolerance in at least some participants, ranging from 7.8 to 30%. For example, Smorawiński et al. [24] reported that glucose AUC during oral glucose tolerance tests was 30% higher following three days of bed rest in inactive young men, although there were no change in endurance- or strength-trained athletes. Yanagibori et al. [38] observed significant 7.8% reductions in oral glucose tolerance in women, but not men, following 3 days of bed rest. The effect sizes in these studies ranged from −0.03 to 1.4 and were in the moderate or high range for all but one study.

The available evidence suggests that acute bouts of uninterrupted sedentary behaviour may result in moderate-to-large reductions in oral glucose tolerance. Given the relatively consistent findings and the strong evidence from randomized crossover studies, we conclude that the evidence linking acute sedentary behaviour with reductions in glucose tolerance is of moderate quality.

3.9. Quality Assessment. Downs and Black scores assessing the risk of bias for individual studies are presented in Table 1. The average score was 21.4 ± 2.3, out of a maximum of 32. The three randomized crossover studies had the highest quality (25.0 ± 1.7), followed by the randomized controlled trials (22.5 ± 2.1) and the nonrandomized interventions (20.8 ± 1.9). The overall quality of evidence related to each outcome is presented in Table 2.

4. Discussion

Based on our systematic review of data from 25 independent interventions, we found moderate quality evidence suggesting that acute bouts of uninterrupted sedentary behaviour lasting 2 hours to 7 days result in rapid and deleterious changes in triglyceride levels, insulin sensitivity, and glucose tolerance. We also found very low-quality evidence that it results in changes in fasting glucose, fasting insulin, and HDL- or LDL-cholesterol.

The findings of the current paper have important public health implications. Recent estimates suggest that on average North American adults and children spend 7–10 hours per day—more than half their waking hours—engaging in sedentary behaviour [43–46]. This suggests that many individuals likely spend several consecutive hours sitting down on a regular basis, which is not dissimilar to the protocol employed by 3 randomized crossover studies in this paper that resulted in significant reductions in insulin sensitivity and glucose tolerance [11, 39, 42]. Individuals who perform long bouts of uninterrupted sedentary behaviour on a regular basis may therefore be exposing themselves to higher levels of circulating triglycerides, as well as reduced insulin sensitivity and glucose tolerance, which may help to explain the prospective associations between sedentary behaviour and chronic disease morbidity and mortality [3–5].

Research in animal models suggests mechanisms that may explain our observation of consistent changes in both insulin sensitivity and plasma triglyceride levels in response to uninterrupted sedentary behaviour. Bey and Hamilton reported that just 18 hours of hindlimb unloading results in near total cessation of lipoprotein lipase activity and roughly 75% reduction in triglyceride uptake in rat skeletal muscle [12]. Similarly, it is also well established that skeletal muscle denervation results in rapid changes in glucose transport protein expression and reductions in insulin sensitivity [13, 14]. These findings suggest that rapid and deleterious changes in skeletal muscle metabolic function may underlie the relationship between sedentary behaviour, triglyceride levels, and insulin sensitivity observed in the present review.

4.1. Strengths and Limitations. The major strength of this paper is its rigorous systematic methodology. The search strategy was developed in consultation with a research librarian with expertise in search creation, and the screening process included two independent reviewers who came to consensus on all included studies. Strength of evidence was assessed using GRADE in order to increase the transparency of the grading process. Finally, the paper was prospectively registered with PROSPERO.

The limitations of this paper relate primarily to the quality of evidence that is presently available. Of 25 independent interventions identified by this paper, only 6 employed a randomized design. Further, although fasting glucose, glucose tolerance, insulin, and insulin sensitivity have each been examined by 9 or more investigations, lipid levels have received little attention by comparison. Given the small number of studies and the low quality of evidence currently available for these outcomes, it is difficult to determine their relationship with sedentary behaviour with any certainty.

There has also been a large amount of heterogeneity in the modality of sedentary behaviour (e.g., sitting versus bed rest) and in the way that outcome measures are calculated, which precluded the use of meta-analyses in the present paper. Only 5 studies identified by the current paper examined a modality of sedentary behaviour other than bed rest. The modality of sedentary behaviour which is most common in daily life is undoubtedly sitting, yet the acute impact of sitting has only been examined in four interventions. In contrast the metabolic impact of bed rest has received far more attention in the published literature [16], despite the

fact that prolonged periods of bed rest are uncommon in day-to-day life. Given that it is unclear whether sitting and bed rest have a similar impact on markers of cardiometabolic risk, it is important that future studies focus on the impact of sitting to determine whether it has an impact which is similar to that of bed rest.

The sample size of most interventions identified by this paper was quite small, and the vast majority of studies were performed in physically fit, healthy young adult males between the ages of 20 and 30. We only identified two interventions focused on individuals above the age of 50 [39, 42], or those with elevated body weight [22, 39] and we were not able to identify any interventions focusing on pediatric populations. Further, females made up just 16% of the participants in the identified interventions, which makes it unclear whether the relationships observed in the current paper will generalize to females of any age.

It is also worth noting that, at present, it is difficult to differentiate the impact of sedentary behaviour *per se* from that of a positive energy balance. If energy intake is maintained at an individual's habitual level, it can be assumed that an imposed bout of sedentary behaviour is likely to result in positive energy balance. However, to our knowledge only one intervention [11] has attempted to separate the impact of an acute bout of sedentary behaviour from that of acute positive energy balance. Interestingly, Stephens et al. report that reducing energy intake to match energy expenditure during a bout of prolonged sedentary behaviour reduced the deleterious impact on insulin sensitivity by roughly 50% [11]. Further, no studies identified in the current paper reported adjusting results for baseline physical activity, fitness, or diet. Future work should investigate these issues further, in order to determine the relative contributions of sedentary behaviour and positive energy balance to changes in cardiometabolic risk factors.

To date only three studies have examined the impact of uninterrupted sedentary behaviour lasting less than 1 day on markers of metabolic risk. Given that healthy individuals rarely spend 24 hours engaging in uninterrupted sedentary behaviour, it is important that future studies investigate whether shorter bouts of sedentary behaviour also have a measurable impact on metabolic health. Future work should also investigate the acute impact of sedentary behaviour on nontraditional markers of cardiometabolic risk including adipokines and markers of inflammation. Finally, none of the studies identified in the current paper examined whether these deleterious changes in risk markers persisted once participants returned to free living conditions. Thus, it is unclear whether the changes observed in the reviewed studies endure for several days following the cessation of sedentary behaviour, or whether they are rapidly resolved. Assessing the clinical significance of these changes will be difficult until their time-course has been more carefully examined.

5. Conclusions

This study demonstrates that, at present, there is moderate quality evidence that acute bouts of uninterrupted sedentary behaviour result in significant and deleterious changes in insulin sensitivity, glucose tolerance, and plasma triglyceride levels. There is currently very low-quality evidence linking uninterrupted sedentary behaviour with changes in circulating insulin, glucose, and HDL- and LDL-cholesterol levels. There is no evidence that acute bouts of uninterrupted sedentary behaviour provide any positive changes in markers of cardiometabolic risk. However, the majority of studies identified by this paper focused on healthy young men, and it is therefore unclear whether these results will generalize to females or to other age groups. These findings suggest that uninterrupted bouts of sedentary behaviour should be avoided in order to prevent transient increases in metabolic risk.

Abbreviations

HDL-cholesterol:	High-density lipoprotein cholesterol
LDL-cholesterol:	Low-density lipoprotein cholesterol
MET:	Metabolic equivalent
HOMA:	Homeostasis model of assessment
QUICKI:	Quantitative insulin sensitivity check index
AUC:	Area-under-the-curve.

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Conflict of Interests

The authors report no conflict of interests.

Authors' Contribution

T. J. Saunders, R. Larouche, R. C. Colley, and M. S. Tremblay developed the study rationale and criteria for inclusion and exclusion. T. J. Saunders developed and executed the literature search. T. J. Saunders and R. Larouche screened all potentially relevant articles. T. J. Saunders extracted data from included studies and wrote the first draft of the paper. R. Larouche checked data extraction, then reviewed, and edited the paper with R. C. Colley and M. S. Tremblay.

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Sedentary Behaviour, Visceral Fat Accumulation and Cardiometabolic Risk in Adults: A 6-Year Longitudinal Study from the Quebec Family Study

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Abstract

Background: Sedentary behaviour has recently emerged as a unique risk factor for chronic disease morbidity and mortality. One factor that may explain this relationship is visceral adiposity, which is prospectively associated with increased cardiometabolic risk and mortality. The objective of the present study was to determine whether sedentary behaviour was associated with increased accumulation of visceral fat or other deleterious changes in cardiometabolic risk over a 6-year follow-up period among adult participants in the Quebec Family Study.

Methods: The current study included 123 men and 153 women between the ages of 18 and 65. Total sedentary time and physical activity were assessed by self-report questionnaire. Cross-sectional areas of visceral and subcutaneous abdominal adipose tissue were assessed using computed tomography. Cardiometabolic biomarkers including fasting insulin, glucose, blood lipids, HOMA-Insulin Resistance, and oral glucose tolerance were also measured. All variables of interest were collected at both baseline and follow-up.

Results: After adjustment for age, sex, baseline BMI, physical activity, energy intake, smoking, education, income and menopausal status, baseline sedentary behaviour was not associated with changes in visceral adiposity or any other marker of cardiometabolic risk. In the longitudinal model which adjusted for all studied covariates, every 15-minute increase in sedentary behaviour from baseline to follow-up was associated with a 0.13 cm increase in waist circumference (95% CI = 0.02, 0.25). However, there was no association between changes in sedentary behaviour and changes in visceral adiposity or other markers of cardiometabolic risk.

Conclusion: These results suggest that neither baseline sedentary behaviour nor changes in sedentary behaviour are associated with longitudinal changes in visceral adiposity in adult men and women. With the exception of waist circumference, the present study did not find evidence of a relationship between sedentary behaviour and any marker of cardiometabolic risk in this population.

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Introduction

Sedentary behaviour (e.g. sitting, reclining) has recently emerged as a unique risk factor for chronic disease [1,2] and is consistently associated with increased risk of both obesity and mortality [3–6]. Excess sedentary time has also been associated with increased accumulation of central adiposity and other markers of cardiometabolic risk [7,8]. For example, Wijndaele and colleagues reported that increases in television (TV) viewing during a 5-year follow-up period were associated with significant increases in waist circumference in both men and women, and increases in blood pressure and clustered cardiometabolic risk among women [8].

One factor that may link sedentary behaviour with increased morbidity and mortality is the accumulation of visceral adipose tissue, which is prospectively associated with mortality and increased cardiometabolic risk [9–11]. Despite the hypothesized link between high levels of sedentary behaviour and both obesity and central adiposity, the association between sedentary behaviour and the accumulation of visceral adipose tissue remains largely unexamined. To our knowledge, only one cross-sectional study has examined this question, and reported no association between sedentary behaviour and visceral fat levels in physically inactive adults [12].

A longitudinal study of the association between sedentary behaviour and the accumulation of visceral fat could therefore make an important contribution to our understanding of the relationship between sedentary behaviour and chronic disease morbidity and mortality. The objective of the present study was to determine whether sedentary behaviour was associated with increased accumulation of visceral fat or other deleterious changes in cardiometabolic risk among adult participants in the Quebec Family Study.

Materials and Methods

Ethics Statement

All participants provided written informed consent to participate in the study. The project followed guidelines of the Medical Research Council of Canada, and was approved by the Medical Ethics Committee of Laval University.

Subjects

The Quebec Family Study was initiated at Laval University in 1978. The primary goal of this project was to investigate the role of genetics in the development of obesity and related cardiovascular risk factors. A total of 1650 individuals from 375 families were recruited and assessed in Phase 1 of the study (1978 to 1981). In this initial phase recruitment was conducted irrespective of body weight, resulting in a cohort with body mass index (BMI), ranging

Table 1. Baseline subject characteristics across tertiles of baseline sedentary behaviour in males.

	n (L/M/H)	Low	Medium	High
Age (years)	39/33/51	39 (15)	39 (13)	39 (16)
Baseline sedentary time (min/day)	39/33/51	305 (62)	472 (50)	667 (89)
MVPA (min/day)	39/32/51	50 (60)	41 (47)	25 (36)
BMI (kg/m^2)	39/33/51	25.8 (4.4)	26.5 (4.6)	26.4 (5.5)
Waist circumference (cm)	39/33/51	89.4 (12.6)	91.3 (12.1)	90.9 (15.6)
Body fat (%)	37/31/46	21.3 (7.5)	22.5 (7.4)	21.5 (8.6)
Visceral AT (cm^2)	29/29/36	112.5 (61.1)	124.9 (67.0)	114.7 (85.5)
Subcutaneous AT (cm^2)	29/29/36	208.2 (130.0)	235.5 (132.1)	191.6 (129.6)
Total abdominal AT (cm^2)	29/29/36	320.7 (176.9)	360.4 (189.5)	306.3 (197.6)
Fasting glucose (mmol/L)	39/33/51	5.00 (0.59)	4.93 (0.57)	4.97 (0.52)
HOMA-IR Index	28/30/39	2.32 (1.72)	2.96 (2.77)	2.87 (2.46)
Glucose AUC (mmol/L)	30/28/37	1224 (244)	1126 (197)	1143 (253)
Insulin AUC (pmol/L)	28/28/36	69249 (67670)	65615 (43890)	73260 (50413)
Total cholesterol (mmol/L)	39/22/50	4.95 (0.86)	5.15 (0.90)	4.89 (1.02)
HDL-cholesterol (mmol/L)	39/33/50	1.16 (0.30)	1.13 (0.29)	1.08 (0.25)
LDL-cholesterol (mmol/L)	38/32/50	3.17 (0.72)	3.25 (0.73)	3.13 (0.88)
Triglycerides (mmol/L)	39/33/50	1.49 (0.90)	1.77 (1.18)	1.56 (0.76)
Energy intake (kcal/day)	39/32/51	2910 (598)	2714 (715)	2672 (803)
Total family income in Canadian dollars (n (%))				
<10,000$		0 (0)	0 (0)	0 (0)
10,000–29,000$		1 (2.6)	0 (0)	0 (0)
30,000–49,000$		13 (34.2)	13 (37.1)	8 (17.0)
50,000–69,000$		13 (34.2)	9 (25.7)	13 (27.7)
70,000$+		11 (28.95)	13 (37.1)	26 (55.3)
Education level				
High School		20 (48.8)	9 (25.7)	10 (21.7)
College*		14 (34.2)	12 (34.3)	21 (45.7)
University		7 (17.1)	14 (40.0)	15 (32.6)

Data are expressed as mean (SD) unless otherwise specified.
L = low sedentary behaviour at baseline; M = medium sedentary behaviour at baseline; H = high sedentary behaviour at baseline; MVPA, moderate-to-vigorous physical activity; BMI, body mass index; AT, adipose tissue; HOMA-IR, homeostasis model assessment of insulin resistance; AUC, area under-the-curve.
*In Québec, there is a level of education generally lasting 2 to 3 years between high school and university termed CEGEP (*Collège d'Enseignement Général et Professionnel*), an acronym that does not have any translation in English.
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Table 2. Baseline subject characteristics across tertiles of baseline sedentary behaviour in females.

	n (L/M/H)	Low	Medium	High
Age (years)	54/58/41	41 (12)	42 (15)	32 (13)
Baseline sedentary time (min/day)	54/58/41	305 (70)	463 (41)	620 (69)
MVPA (min/day)	53/57/30	21 (26)	10 (15)	14 (16)
BMI (kg/m^2)	54/58/41	25.2 (6.0)	25.7 (6.3)	24.5 (4.7)
Waist circumference (cm)	54/58/41	77.9 (14.5)	79.0 (13.8)	76.8 (11.9)
Body fat (%)	45/50/40	30.8 (10.1)	31.2 (9.0)	28.4 (8.3)
Visceral AT (cm^2)	41/42/29	87.7 (61.8)	101.2 (55.9)	64.4 (44.0)
Subcutaneous AT (cm^2)	41/42/29	282.8 (154.8)	312.5 (162.6)	268.1 (147.4)
Total abdominal AT (cm^2)	41/42/29	370.5 (202.2)	413.7 (200.9)	332.5 (180.6)
Fasting glucose (mmol/L)	54/56/40	4.66 (0.51)	4.81 (0.52)	4.69 (0.40)
HOMA-IR Index	44/45/32	2.29 (1.96)	2.27 (2.00)	1.94 (1.27)
Glucose AUC (mmol/L)	40/41/29	1075 (209)	1162 (257)	1101 (186)
Insulin AUC (pmol/L)	39/41/29	66290 (46483)	79134 (74763)	61007 (42899)
Total cholesterol (mmol/L)	5457/39	5.10 (1.01)	5.16 (1.03)	5.01 (2.00)
HDL-cholesterol (mmol/L)	54/5739	1.40 (0.36)	1.37 (0.32)	1.32 (0.37)
LDL-cholesterol (mmol/L)	54/5738	3.13 (0.86)	3.18 (0.86)	2.84 (1.00)
Triglycerides (mmol/L)	54/57/39	1.28 (0.59)	1.39 (0.62)	2.08 (5.11)
Energy intake (kcal/day)	54/56/41	1877 (381)	1869 (398)	2096 (434)
Total family income in Canadian dollars (n (%))				
<10,000$		1 (1.8)	2 (3.6)	1 (2.8)
10,000–29,000$		1 (1.8)	0 (0)	0 (0)
30,000–49,000$		25 (45.5)	16 (29.1)	6 (16.7)
50,000–69,000$		13 (23.6)	14 (25.5)	10 (27.8)
70,000$+		15 (27.3)	23 (41.8)	19 (52.8)
Education level				
High School		29 (50.9)	30 (50.9)	10 (27.0)
College*		15 (26.3)	21 (35.6)	16 (43.2)
University		13 (22.8)	8 (13.6)	11 (29.7)
Menopausal status				
In menopause		13 (36.1)	15 (46.9)	3 (30)
Not in menopause		23 (63.9)	17 (53.1)	7 (70)

Data are expressed as mean (SD) unless otherwise specified.
L = low sedentary behaviour at baseline; M = medium sedentary behaviour at baseline; H = high sedentary behaviour at baseline; MVPA, moderate-to-vigorous physical activity; BMI, body mass index; AT, adipose tissue; HOMA-IR, homeostasis model assessment of insulin resistance; AUC, area under-the-curve.
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from 13.8 to 64.9 kg/m^2. An additional 123 families with at least 1 parent and 1 offspring with a BMI of 32 or higher were added to the study in Phase 2 (1989–1994) and 3 (1995–2001) of the study, while also retesting 100 families from Phase 1. Families were recruited through the media and were all French Canadians from the greater Québec City area. From the sample of 223 white nuclear families (totaling 951 subjects involved in Phases 1, 2, and 3), 147 men and 169 women were eligible for longitudinal analyses between Phase 2 and 3. Longitudinal analyses were not possible with Phase 1 as assessments differed at this time point from those employed in Phases 2 and 3. Additional details about the Quebec Family Study have been previously published [13].

Baseline in the current study corresponded to Phase 2, and the mean duration of follow-up between Phase 2 and 3 was 6.0 (SD 1.0) years. The following exclusion criteria were applied: (i) aged less than 18 years or greater than 64 years (13 men and 9 women excluded); (ii) diabetic, defined as use of insulin or a hypoglycemic agent, a fasting plasma glucose level of ≥7.0 mmol/L, or a 2-hour postload plasma glucose level of ≥11.1 mmol/L (7 men and 3 women excluded) and (iii) missing data for sedentary behaviour (4 men and 4 women excluded). The final number of eligible participants within the longitudinal sample was 286 individuals (123 men and 153 women) (see Tables 1 and 2).

Sedentary Behaviour and Physical Activity

Sedentary behaviour and physical activity were estimated using a physical activity record [14]. Subjects had to complete a physical activity diary for 3 days, including 2 weekdays and 1 weekend day, with each day being divided into 96 periods of 15 minutes each. Subjects were asked to code the main activity performed during each 15-minute period using a scale from 1 to 9, ranging from sleeping (category 1) to intense manual work (category 9). Time

Table 3. Associations (95% confidence interval) of sedentary behaviour and markers of adiposity at baseline.

Model	BMI (M)	BMI (F)	WC	Fat%	TAAT	VAT	ASAT
1	−0.01 (−0.08, 0.07)	−0.02 (−0.12, 0.09)	0.07 (−0.10, 0.24)	−0.10 (−0.22, 0.01)	−1.51 (−4.09, 1.07)	−0.07 (−0.95, 0.81)	−1.45 (−3.43, 0.53)
2	−0.01 (−0.08, 0.07)	0.01 (−0.10, 0.11)	0.02 (−0.14, 0.17)	−0.01 (−0.10, 0.08)	−0.36 (−2.92, 2.21)	0.18 (−0.59, 0.94)	−0.54 (−2.51, 1.44)
3	−0.01 (−0.02, 0.01)	−0.03 (−0.05, −0.01)*	0.03 (−0.10, 0.16)	0.08 (−0.02, 0.17)	0.59 (−1.39, 2.57)	0.63 (−0.82, 2.08)	−0.04 (−1.55, 1.47)

Model 1: unadjusted.
Model 2: adjusted for age and sex.
Model 3: adjusted for age, sex, BMI, energy intake, moderate-to-vigorous physical activity, educational level, income, smoking and menopausal status.
*$p<0.05$.
M, Male; F, Female; BMI, Body Mass Index; WC, Waist Circumference; Fat%, body fat percentage; TAAT, total abdominal adipose tissue; VAT, visceral adipose tissue; ASAT, abdominal subcutaneous adipose tissue.
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spent in categories 6 to 9 was used to calculate moderate-to-vigorous physical activity (MVPA) [14]. Sedentary behaviour was calculated as the sum of time identified as being in category 2 ("Sitting: eating, listening, writing, etc"). Time spent in category 1 ("sleeping, resting in bed") was not included in the sedentary behaviour category as sedentary behaviour refers only to waking behaviours [2]. The reliability and validity of the record have been previously reported [14]. These measurements were performed both at baseline and after 6 years.

Assessment of Abdominal Fat by Computed Tomography (CT)

Cross-sectional abdominal adipose tissue areas were assessed by CT using a Siemens Somatom DRH scanner (Erlanger, Germany) as described in detail elsewhere [15]. Briefly, an abdominal scan was taken between the fourth and fifth lumbar vertebrae (L4–L5) with subjects lying in a supine position with arms stretched above the head. The position of the scan was determined using a scout radiograph of the abdomen. Total and visceral adipose tissue areas were delineated with a graph pen and then computed using an attenuation range of −190 to −30 Hounsfield units [16]. Visceral fat area was determined by drawing a line within the muscle wall surrounding the abdominal cavity. Abdominal subcutaneous fat area was obtained by computing the difference between total and visceral adipose tissue areas.

Anthropometric and Body Composition Measurements

Height was measured to the nearest 0.1 cm using a standard stadiometer, and body weight was measured to the nearest 0.1 kg using a digital panel indicator scale (Beckman Industrial Ltd, Model 610/612, Scotland, UK). BMI was calculated as body weight divided by height squared (kg/m^2). Waist circumference was measured at the line between the lower border of the last rib and the upper border of the iliac crest. All anthropometric measurements were performed according to standardized procedures recommended at The Airlie Conference [17]. Body density was obtained from the mean of 6 valid measurements derived from underwater weighing [18]. The helium dilution method of Meneely and Kaltreider [19] was used to determine the pulmonary residual volume before immersion in the hydrostatic tank,. Total body fat percentage was determined from body density with the equation of Siri [20]. Body fat mass was estimated from body weight and the percentage of body fat. These measurements were performed in the same way at both baseline and after 6 years.

Cardiometabolic Risk Factors

Total cholesterol and triglyceride concentrations were determined by use of commercial enzymatic-based methods, as described elsewhere [21]. HDL-cholesterol concentrations were analyzed after precipitation of apolipoprotein B-containing lipoproteins with heparin and manganese chloride [22]. Glucose concentrations were measured enzymatically and serum insulin

Table 4. Associations (95% confidence interval) of sedentary behaviour and markers of cardiometabolic risk at baseline.

Model	HDL-C (M)	HDL-C (F)	LDL-C	TG	FG	FI	HOMA-IR	Glucose AUC	Insulin AUC
1	−0.01 (−0.01, 0.01)	−0.01 (−0.01, 0.01)	−0.01 (−0.02, 0.01)	0.01 (−0.02, 0.03)	0.01 (−0.01, 0.01)	0.02 (−0.67, 0.71)	0.01 (−0.02, 0.03)	−0.21 (−3.27, 2.85)	88.64 (−660.03, 837.32)
2	−0.01 (−0.01, 0.01)	−0.01 (−0.01, 0.01)	−0.01 (−0.01, 0.01)	0.01 (−0.02, 0.03)	0.01 (−0.01, 0.01)	−0.11 (−0.83, 0.60)	−0.01 (−0.03, 0.03)	0.47 (−2.50, 3.44)	197.84 (−571.27, 966.94)
3	−0.01 (−0.01, 0.01)	0.01 (−0.01, 0.02)	0.01 (−0.02, 0.03)	−0.01 (−0.03, 0.01)	−0.01 (−0.02, 0.01)	−0.03 (−1.17, 1.11)	−0.01 (−0.06, 0.04)	−6.37 (−13.37, 0.62)	823.09 (−453.45, 2099.64)

Model 1: unadjusted.
Model 2: adjusted for age and sex.
Model 3: adjusted for age, sex, BMI, energy intake, moderate-to-vigorous physical activity, educational level, income, smoking and menopausal status.
*$p<0.05$.
M, Male; F, Female; HDL-C, HDL-Cholesterol; LDL-C, LDL-Cholesterol; TG, triglycerides; FG, fasting glucose; FI, fasting insulin; AUC, area under-the-curve.
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Table 5. Associations (95% confidence interval) of baseline sedentary behaviour with 6-year change in markers of adiposity.

Model	BMI (M)	BMI (F)	WC	Fat%	TAAT	VAT	ASAT
1	0.01 (−0.02, 0.05)	−0.04 (−0.08, −0.01)*	−0.02 (−0.09, 0.05)	−0.03 (−0.08, 0.02)	−0.47 (−1.57, 0.63)	−0.23 (−0.73, 0.26)	−0.24 (−1.05, 0.58)
2	0.01 (−0.02, 0.05)	−0.04 (−0.08, −0.01)*	−0.02 (−0.10, 0.05)	−0.03 (−0.08, 0.02)	−0.33 (−1.47, 0.81)	−0.19 (−0.70, 0.33)	−0.14 (−0.98, 0.70)
3	−0.02 (−0.08, 0.05)	0.03 (−0.05, 0.11)	−0.07 (−0.19, 0.05)	−0.07 (−0.15, 0.02)	−0.69 (−2.92, 1.53)	−0.16 (−1.38, 1.05)	−0.53 (−1.91, 0.86)

Model 1: unadjusted.
Model 2: adjusted for age and sex.
Model 3: adjusted for age, sex, baseline BMI, energy intake, moderate-to-vigorous physical activity, educational level, income, smoking and menopausal status.
*p<0.05.
M, Male; F, Female; BMI, Body Mass Index; WC, Waist Circumference; Fat%, body fat percentage; TAAT, total abdominal adipose tissue; VAT, visceral adipose tissue; ASAT, abdominal subcutaneous adipose tissue.
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concentrations were measured by radioimmunoassay [23]. A 75 g oral glucose tolerance test (OGTT) was performed in the morning after a 12 h fast. The total areas under the curve during the OGTT for insulin and glucose were computed from the plasma levels determined at 15 min intervals during the first hour following the glucose ingestion and every 30 min for the subsequent 3 h, using the trapezoidal method as previously described [24]. Insulin sensitivity was estimated in the fasting state using the homeostasis model assessment for insulin resistance (HOMA-IR) [25].

Energy Intake

Diet was evaluated with a 3-day food record, including 2 weekdays and 1 weekend day, at baseline and year 6. Participants were shown how to complete this record by a dietician who provided instruction about measuring the quantities of ingested foods [26]. Mean daily energy intake was estimated by a dietician using a computerized version of the Canadian Nutrient File [27].

Measurement of Covariates

Several covariates were measured via self-reported questionnaires. These include age, sex, smoking habits (nonsmoker or ex-smoker, light smoker [≤10 cigarettes per day], heavy smoker [>10 cigarettes per day]), highest educational level (high school, college [CEGEP for Quebec], university), total annual family income (categorized into 5 groups ranging from < $10,000 to $70,000 or more) and menopausal status.

Statistical Analysis

Sample size calculations were performed to assess whether the present dataset was likely to provide sufficient power to detect a significant relationship between sedentary behaviour and longitudinal changes in our primary outcome of visceral adiposity, should one exist. Assuming that the fully adjusted model would account for roughly 25% of the variance in changes in visceral adiposity during the 6-year follow-up, and that sedentary behaviour would account for at least 3% of this variance, the current dataset of 206 participants with full data for our primary outcome provides more than 85% power to detect a significant association should one exist at an alpha level of 0.05.

To determine if men and women could be combined into one analysis, sex-by-sedentary behaviour interactions were assessed for all dependent variables. Significant interactions were detected for BMI and HDL-cholesterol, and thus analyses involving these variables are presented in men and women separately. There were no significant sex interactions for any other variables of interest, therefore all other analyses present men and women combined in order to maximize statistical power. Normality of distribution was assessed using the Shapiro-Wilk test and visual inspection. Moderate- and vigorous-intensity physical activity and BMI were both transformed using a log function. Regression analyses were

Table 6. Associations (95% confidence interval) of baseline sedentary behaviour with 6-year change in markers of cardiometabolic risk.

Model	HDL-C (M)	HDL-C (F)	LDL-C	TG	FG	FI	HOMA-IR	Glucose AUC	Insulin AUC
1	−0.01 (−0.01, 0.01)	0.01 (0.01, 0.01)*	−0.01 (−0.01, 0.01)	−0.01 (−0.03, 0.02)	0.01 (−0.01, 0.01)	0.51 (−0.05, 1.08)	0.02 (−0.01, 0.05)	−0.58 (−4.07, 2.92)	−8.53 (−579.88, 562.82)
2	−0.01 (−0.01, 0.01)	0.01 (−0.01, 0.01)	−0.01 (−0.01, −0.01)*	−0.01 (−0.03, 0.02)	0.01 (−0.01, 0.01)	0.67 (0.10, 1.25)*	0.03 (0.01, 0.05)*	0.06 (−3.45, 3.57)	65.56 (−524.90, 656.02)
3	−0.01 (−0.01, 0.01)	0.01 (−0.01, 0.02)	−0.01 (−0.03, 0.01)	−0.01 (−0.02, 0.02)	−0.01 (−0.03, 0.01)	0.18 (−0.99, 1.35)	0.01 (−0.07, 0.07)	−2.63 (−12.51, 7.25)	−1252.36 (−2527.59, 22.86)

Model 1: unadjusted.
Model 2: adjusted for age and sex.
Model 3: adjusted for age, sex, baseline BMI, energy intake, moderate-to-vigorous physical activity, educational level, income, smoking and menopausal status.
*p<0.05.
M, Male; F, Female; HDL-C, HDL-Cholesterol; LDL-C, LDL-Cholesterol; TG, triglycerides; FG, fasting glucose; FI, fasting insulin; AUC, area under-the-curve.
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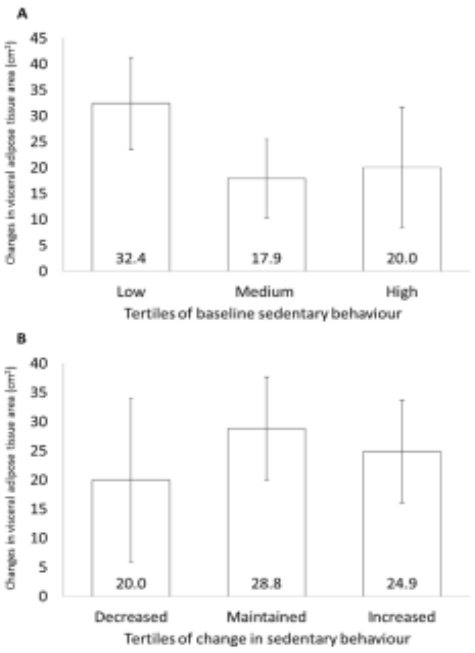

Figure 1. Changes in visceral adipose tissue across tertiles of sedentary behaviour. Changes in visceral adipose tissue cross-sectional area across tertiles of baseline sedentary behaviour (Figure 1A) or change in sedentary behaviour (Figure 1B) were compared by analysis of covariance with adjustment for age, sex, baseline BMI, energy intake, moderate-to-vigorous physical activity, educational level, income, smoking and menopausal status. Data are presented as mean ± standard error. There were no significant differences across tertiles of sedentary behaviour in either analysis.
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performed to determine the univariate and multivariate associations between sedentary behaviour and 6-year changes in markers of cardiometabolic risk. Multivariate models were adjusted for age, sex, baseline BMI, energy intake, moderate- and vigorous-intensity physical activity, education level, income, smoking and menopausal status. Participants were also divided into sex-specific tertiles of baseline sedentary behaviour and change in sedentary behaviour from baseline to follow-up, and an ANCOVA was then used to compare the change in markers of adiposity across these tertiles, adjusting for the same covariates as in the above regression analyses. It should be noted that participants with identical values were grouped into the same tertile, which resulted in unequal numbers of participants in each tertile. A Bonferroni correction was used to adjust for multiple comparisons in post hoc tests following the ANCOVA.

Data are given as mean and standard deviation unless otherwise noted. Statistical significance was set at a p value of ≤ 0.05. All statistical analyses were performed using SAS version 9.2 (SAS Institute, Cary, NC).

Results

Baseline Sedentary Behaviour and Markers of Cardiometabolic Risk

Baseline characteristics of male and female participants are presented in Tables 1 and 2. At baseline, men and women averaged 8.3 and 7.5 hours of daily sedentary behaviour, respectively. Sedentary behaviour was not associated with any marker of adiposity or cardiometabolic risk in unadjusted cross-sectional analyses at baseline (Tables 3 and 4). These results were not changed following adjustment for age and sex. Following additional adjustment for energy intake, moderate-to-vigorous physical activity, educational level, income, smoking and menopausal status, each additional 15-minutes of baseline sedentary behaviour was cross-sectionally associated with 0.03 kg/m^2 lower BMI (95% CI = −0.05, −0.01) in women, but not men. However, there were no other significant associations between sedentary behaviour and any other marker of adiposity or cardiometabolic risk in the fully adjusted model at baseline.

Tables 5 and 6 present associations of baseline sedentary behaviour with changes in measures of adiposity and markers of cardiometabolic risk, respectively. In unadjusted analyses each 15-minute increase in baseline sedentary behaviour was associated with a 0.01 mmol/L increase in HDL-cholesterol (95% CI = 0.01, 0.01) and a −0.04 kg/m^2 reduction in BMI (95% CI = −0.08, −0.01) in women, but not men. Following adjustment for age and sex, the association with BMI in women remained unchained, while each 15-minute increase in baseline sedentary behaviour was also associated with a 0.01 mmol/L reduction in LDL-cholesterol (95% CI = −0.01, −0.01), a 0.67 pmol/L increase in fasting insulin (95% CI = 0.10, 1.25), and a 0.03 unit increase in HOMA-IR (95% CI = 0.01, 0.05) in men and women combined. However, after further adjustment for baseline BMI, energy intake, moderate-to-vigorous physical activity, educational level, income, smoking and menopausal status, baseline sedentary behaviour was not associated with changes in any marker of adiposity or cardiometabolic risk.

Figure 1A presents the average accumulation of visceral adipose tissue across the three tertiles of baseline sedentary behaviour. The mean (standard deviation) reported sedentary time in the three tertiles were 305 (66), 471 (53) and 642 (89) minutes in the low, medium, and high tertiles, respectively. There were no differences in the accumulation of any abdominal fat compartment across the three tertiles of sedentary behaviour. Adjusting for covariates did not materially change the results.

Longitudinal Changes in Sedentary Behaviour and Markers of Cardiometabolic Risk

In the fully adjusted model, each 15-minute increase in sedentary behaviour was positively associated with a 0.13 cm increase in waist circumference (95% CI = 0.02, 0.25). However, there were no significant associations between the change in sedentary behaviour and the change in visceral adiposity or any other marker of cardiometabolic risk (data not shown).

Participants were also classified into tertiles based on their longitudinal changes in sedentary behaviour from baseline to follow up. One third of participants reduced their sedentary time by a mean (standard deviation) of 195 (108) minutes over the 6-year follow-up. Another third maintained roughly the same amount of sedentary behaviour throughout the study, reducing their sedentary time by an average of just 13 (39) minutes. The final third of participants increased their sedentary time by an average of 165 (97) minutes. However, there were no differences in

the accumulation of any abdominal fat compartment among these three tertiles (Figure 1B).

Discussion

Our results suggest that sedentary behaviour is not associated with 6-year changes in visceral adiposity in adult men and women. To our knowledge, this is the first longitudinal study to examine the relationship between sedentary behaviour and the accumulation of visceral adipose tissue measured by CT. These findings are consistent with a recent study that found no cross-sectional association between objectively measured sedentary behaviour and visceral adiposity in a group of 126 abdominally obese men and women [12].

It is worth noting that both our study, and the previous study by McGuire and Ross [12], examined the association of visceral fat with a measure of total sedentary time. It is unclear whether similar results would have been observed for specific modalities of sedentary behaviour (e.g. screen-based vs. non-screen sedentary behaviours). For example, prospective studies in both the US and Australia have found associations between TV viewing and increased waist circumference [7,8]. Given that TV viewing has been linked with increased energy intake [3,28,29] this modality of sedentary behaviour may be more closely associated with changes in adiposity and metabolic risk than global measures of total sedentary time [30–32]. For example, a study of 9,000 American adults found that those who watched more than 2 hours per day of television also consumed higher amounts of energy-dense snack foods and soft drinks, as well as consuming more calories during snacks and the evening meal [32]. Further, a recent intervention study by Harris et al. [33] reports that exposure to food advertisements resulted in roughly a 30% increase in food intake among adult participants. Other specific forms of sedentary behaviour such as seated mental work have also been shown to result in increased food intake, as compared to simply resting in the seated position [34]. Taken together, these findings suggest that specific modalities of sedentary behaviour are likely to impact food intake (and therefore adiposity) in different ways, and highlight the importance of assessing the impact of both global sedentary behaviour and of these specific modalities [35].

With the exception of waist circumference, the present study also failed to detect a prospective association between sedentary behaviour and several important markers of cardiometabolic risk including plasma lipids, HOMA-IR, and glucose tolerance. These findings are consistent with some, but not all, previous prospective studies in this area. For example, Ekelund and colleagues found no association between baseline sedentary behaviour and HOMA-IR at 1-year follow-up in a group of 192 men and women [36]. In contrast, Helmerhorst and colleagues reported a significant association between baseline sedentary behaviour and fasting insulin at 5-year follow-up in a cohort of 376 adults, independent of physical activity levels [37]. Of note, both of these studies used objective measures of total sedentary time at baseline. Wijndaele and colleagues have also reported prospective associations between changes in television viewing and clustered cardiometabolic risk in women, but not men [8].

As with adiposity, it is likely that the relationship between sedentary behaviour and markers of cardiometabolic risk may also vary depending on the modality of sedentary behaviour. For example, we have previously reported that seated video-game use, but not other forms of sedentary behaviour, are associated with increased metabolic risk in overweight and obese adolescents [38]. Given these and other findings, it is difficult to come to global conclusions regarding sedentary behaviour and the development of subsequent cardiometabolic risk. However, given the consistent associations between sedentary behaviour and the risk of mortality reported in other studies, public health messages promoting reductions in sedentary behaviour remain important [3].

The present study contains strengths and weaknesses that warrant mention. Limitations include the measurement of sedentary behaviour by self-report, and a lack of information related to specific modalities of sedentary behaviour. The observed results may have differed if an objective measure of sedentary behaviour had been employed, or if sedentary behaviour had been broken into specific modalities such as screen-time and non-screen sedentary behaviours. The relatively small sample size and homogeneous sample of the current study also limits our statistical power, and the generalizability of these findings. It should also be noted that this was a retrospective analysis, as the Quebec Family Study was originally designed to assess the genetic contributions to obesity. Strengths of this study include its longitudinal design and the use of computed tomography to assess visceral and subcutaneous abdominal adiposity [39]. This study also included objective measures of several important markers of cardiometabolic risk, including lipids, insulin resistance, and glucose tolerance in both men and women studied in their natural environment.

In summary, our results suggest that neither baseline sedentary behaviour nor changes in sedentary behaviour are associated with longitudinal changes in visceral adiposity in adult men and women. With the exception of waist circumference, sedentary behaviour does not appear to be associated with longitudinal changes in any marker of cardiometabolic risk in this population. These findings suggest that the development of cardiometabolic risk may be due primarily to factors other than self-reported sedentary behaviour.

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Author Contributions

Designed the statistical analysis: TJS JPC MST. Revised manuscript: JPC MST CB AT JPD TJS. Conceived and designed the experiments: CB JPD AT. Performed the experiments: CB JPD AT. Analyzed the data: TJS. Wrote the paper: TJS.

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INTERNATIONAL JOURNAL OF BEHAVIORAL NUTRITION AND PHYSICAL ACTIVITY

LETTER TO THE EDITOR Open Access

Clustering of children's activity behaviour: the use of self-report versus direct measures

Travis J Saunders*, Stephanie A Prince and Mark S Tremblay

Abstract

While we concur with the objectives of the recent *International Journal of Behavioural Nutrition and Physical Activity* paper published by Jago and colleagues titled "Physical activity and sedentary behaviour typologies of 10-11 year olds", we feel that the results as currently presented do not support their conclusions. Though the authors created groups of children with dramatically different patterns of self-reported physical activity and sedentary behaviour, an inspection of the objectively measured accelerometry data shows little difference between the groups. Further, in at least one instance the difference between groups was of the opposite direction when using objective measures, as opposed to the self-report measures used in the published analysis. Thus, we caution the authors from making conclusions based on their self-report data, and propose that they re-analyze their data using their objectively measured data instead.

To the Editor,

We read with great interest your recently published study by Jago and colleagues [1] titled "Physical activity and sedentary behaviour typologies of 10-11 year olds". The authors argue convincingly that interventions which aim to promote increased physical activity and/or reduced sedentary behaviour should focus on the specific needs and characteristics of their target populations. As such, we concur that their objective to identify clusters of children with similar patterns of physical activity and sedentary behaviour would provide key information for the design of targeted interventions. Unfortunately, we believe that the data presented in the paper suggests that the clusters created by the authors do not represent groups of children with distinct activity patterns, and that the conclusions of the paper are therefore unsupported.

In their paper, Jago et al. [1] assessed physical activity and sedentary behaviour using both self-report questionnaires and accelerometry. However, when creating clusters of children with similar behaviour, the authors relied on only the self-reported data. While this resulted in clusters of children with very distinct quantities of self-reported physical activity and sedentariness, the groups appear almost identical when compared using the objectively measured data. For example, according to the self-report data, the "High Activity/Low Sedentary" group performed an average of 3.6 hours more weekday physical activity than children in the "Low Activity/Medium Sedentary" group. However, when the accelerometer-derived values of weekday moderate- to vigorous-intensity activity are compared instead, the difference between the two groups is reduced to roughly two minutes. Thus, in this situation, the difference between the two groups using self-report measures was roughly 100 times greater than the measured difference assessed using accelerometry.

A similar problem is observed when comparing the groups for sedentary time. For example, the self-report data suggests a dramatic difference in screen time (excluding school-work) between the "High Activity/High Sedentary" group which accumulated 13.86 hours per day and the "High Activity/Low Sedentary" group which reported just 5.77 hours per day. In contrast, the objectively measured data suggests that the "High Activity/High Sedentary" group accumulated 4.7 hours of weekday sedentary time outside of class time (roughly 9 hours less than suggested by their self-reported screen-time), and only differed from the "High Activity/Low Sedentary" group by 5 minutes. Similarly, the "High Activity/High Sedentary" group actually accumulated *less* objectively-measured sedentary time than the "Low

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Activity/Medium Sedentary" group on both weekdays and on weekends. Further, it is questionable whether it would even be possible for children to accumulate the daily volume of screen time (13.86 hours) and physical activity (5.89 hours) reported by children in the "High Activity/High Sedentary" cluster. If true, this would leave the children less than 5 hours per day for both school-work and sleep, suggesting that these values are not just unlikely but impossible.

The large discrepancies between objective and self-report activity patterns observed in the present study have also been reported by others. For example, a recent systematic review by Adamo and colleagues [2] reports that, in comparison to accelerometry, self-report measures overestimate physical activity by an average of 114% in boys and 584% in girls. Recent findings also suggest that self-reported screen time is only weakly correlated with objectively measured sedentary behaviour in adults [3]. It has also been noted that few of the studies which purport to assess sedentary behaviour have actually measured it [4]. Given the discrepancies between self-report and direct measures of activity in the literature, and the availability of directly measured data in the present situation, we caution the authors from making conclusions based on their self-report classifications. Further, we would be interested to know how the behaviour clusters created in the present study might differ if they were based on the accelerometry data, and whether this might also result in more pronounced differences between the clusters in terms of body mass index or the Index of Multiple Deprivation score.

We welcome comments from the authors of the current study in order to provide further clarification of the methods employed and conclusions made.

Respectfully,

Travis J. Saunders, Stephanie A. Prince and Mark S. Tremblay.

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Authors' contributions

TJS, SAP and MST conceived of the letter, and participated in its design. TJS and SAP participated in the drafting of the manuscript, and MST critically revised it. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

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Review Article

Potential Contributors to the Canadian Pediatric Obesity Epidemic

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As a group, Canadian children and youth are heavier than at any time in the recent past. However, to date there has been no critical examination of the factors which are likely to have contributed to these deleterious trends. A review of the evidence suggests that there is robust evidence supporting the role of reduced sleep, increased sedentary time, increased consumption of sugar-sweetened beverages, and secular increases in adult obesity as contributing factors to the current epidemic of childhood obesity. There is moderate evidence that these trends are related to changes in either total energy intake or physical activity, while there is very little evidence supporting the role of maternal age, breastfeeding, exposure to endocrine disrupters, or inadequate calcium intake. These findings suggest that targeting sleep, sedentary time, and sugar-sweetened beverage intake in Canadian children and youth may help to prevent future weight gain at the population level.

1. Introduction

Available evidence suggests that Canada is in the midst of an epidemic of childhood obesity [1–4]. Between 1981 and 2007–2009, the average body mass index (BMI) of 12-year-old Canadians increased from 18.1 to 19.2 kg/m^2 in boys, and from 18.4 to 19.5 kg/m^2 in girls [4]. During this same time period the prevalence of overweight/obesity among Canadians aged 15–19 increased dramatically from 14% to 31% in boys, and from 14% to 25% in girls [4]. In fact, among Canadians aged 15–19, fully 14% of boys and 10% of girls are now considered obese [4]. Equally worrying, as BMI has increased during the past 30 years, so too has the prevalence of abdominal obesity [1, 4]. Since 1981, the average waist circumference (WC) among Canadian youths aged 12–19 has increased by more than 5 centimetres, such that approximately one-fifth of Canadians in this age group now have a WC that places them at some form of increased health risk [1].

These recent increases in both the BMI and WC of Canadian youth are a tremendous public health concern, as pediatric obesity is associated with both metabolic dysfunction in childhood [5–8], as well as an increased risk of mortality well into adulthood [9, 10]. Thus, the objective of the present paper is to give a comprehensive examination of the possible causes of our current epidemic of childhood obesity. This will be done through a discussion of the strength of the evidence base and biological plausibility for each putative factor, before finally comparing their relative contributions to these deleterious trends. This review will focus on Canadian data whenever possible. Before examining the putative causes of the epidemic, however, it is important to briefly review the factors affecting energy balance and their role in body weight.

2. Energy Balance

As noted by Jéquier and Tappy, the first law of thermodynamics—which states that energy can neither be created nor destroyed—applies to humans [11]. With respect to body weight, this means that changes in stored energy (e.g.,

adiposity) are equal to energy intake (EI) minus energy expenditure (EE) [11]. Energy expenditure can be broken down into three separate components [11]:

(i) basal metabolic rate (BMR),

(ii) diet-induced thermogenesis, and

(iii) energy used for exercise and physical activity (PA).

Energy intake, on the other hand, is simply the sum of the energy consumed by an individual, minus approximately 5–10% that is excreted in urine and feces [11]. When EI exceeds EE, the result is an increase in energy stores, and therefore weight gain. Thus, any putative cause of the childhood obesity epidemic must influence either EI, EE, or both. With that in mind, let us now evaluate the role of both conventional and unconventional factors in the etiology of childhood obesity.

3. Reduced Physical Activity

Given its role in the energy balance equation it is quite obvious that, all else being equal, a reduction in the number of calories burned through PA will directly lower EE and result in positive energy balance. Regular bouts of PA are also known to result in substantial elevations in BMR in both lean and obese individuals [12, 13], suggesting that reductions in PA may further reduce EE by deleterious changes to BMR. Similarly, it has also been suggested that regular PA results in more accurate coupling of EI and EE [13–17]. Taken together, these findings suggest that reductions in PA may negatively impact both sides of the energy balance equation by directly reducing EE and by inhibiting the proper regulation of EI. Not surprisingly, available observational evidence also suggests that PA plays a role in the prevention of excess weight gain in children and youth.

Numerous cross-sectional studies report that overweight and obese children are less active than their lean peers, while the majority of longitudinal studies report small, inverse associations between high levels of PA and the accumulation of excess body weight [18–20]. For example, Berkey and colleagues report that every hour of self-reported daily PA in girls aged 9–14 is associated with a $-0.0284\,\mathrm{kg/m^2}$ smaller increase in BMI over a one-year period (the relationship was of borderline significance in boys) [21]. Similarly, a recent systematic review by Connelly and colleagues reports that compulsory PA is the single most defining factor of controlled trials that successfully prevent the development of childhood overweight or obesity [22]. It should be noted that current findings are based mainly on self-reported levels of PA, which are known to be substantially less accurate than objective measures such as pedometry and accelerometry [23, 24]. However, despite these methodological limitations, the balance of evidence suggests that low levels of PA are likely to predispose to future weight gain.

While the above evidence suggests that low levels of PA are likely to result in increased risk of future weight gain, at present it is unclear whether current levels of PA in Canadian youth are lower than those of previous generations, which would be necessary in order for PA to play a causal role in the current obesity epidemic [25]. Self-reported leisure-time PA among Canadian adolescents actually increased during the 1980's and remained stable throughout the 1990's [26], suggesting that current PA levels may be higher than they were before the obesity epidemic. However, this data conflicts with other lines of evidence, which suggest that total PA levels among Canadian children may be lower than they were in the past. For example, it has been reported that the proportion of trips to school that involves active transportation decreased by roughly 20% between 1986 and 2006 among Canadian children in the Toronto region [27]. Similarly, children who live in Canadian Old Order Amish and Mennonite communities, where lifestyles are similar to those in contemporary Canadian society 60–100 years ago [23], accumulate roughly 50% more steps per day than their contemporary Canadian peers [28], as well as 30–50% more moderate-to-vigorous PA [29]. In the absence of more complete and objective data on the PA of past generations of Canadian youth and given that less than 10% of Canadian youth are currently meeting PA guidelines [30–32], it appears relatively safe to conclude that total PA-related EE of Canadian youth is at or near historic lows.

Thus, given the multiple biological mechanisms linking reduced PA with increased adiposity, consistent but relatively small longitudinal associations between PA and weight gain, and evidence suggesting that Canadian children are likely less active than in previous generations, there is currently moderate evidence that insufficient PA plays a causal role in the current epidemic of childhood obesity.

4. Increased Sedentary Behaviour

Sedentary behaviour is defined as "a distinct class of behaviours (e.g., sitting, watching TV, driving) characterized by little physical movement and low energy expenditure ($\leq$1.5 METs)" [33]. At present, it is unclear whether the prevalence of sedentary behaviours in Canada and other industrialized nations has increased in recent decades. For example, a review by Marshall and colleagues reports that the screen time (time spent watching television, playing videogames, or using computers) of children in modern nations has not increased since the 1950's [34]. In contrast, Nelson and colleagues report that weekly computer usage increased by roughly 4 hours/week between 1999 and 2004 in American youth [35]. Similarly, a recent report suggests that Canadian children average more than 6 hours of screen time on weekdays, and that even preschoolers watch an average of almost 2 hours of television per day [36]—amounts that seem highly unlikely 40 years ago. Similar trends are seen in sedentary modes of transportation such as driving, which have also increased dramatically in recent decades [27]. Finally, accelerometry data from the nationally representative Canadian Health Measures Survey suggests that Canadian youth spend an average of 8.6 hours per day (more than 60% of their waking hours) engaging in sedentary behaviour [30]. Taken together, these reports suggest that Canadian children are likely more sedentary than previous generations.

In addition to reports of increasing levels of sedentary behaviour in Canadian youth, there is also an accumulating body of evidence which suggests that high levels of sedentary behaviour may predispose to weight gain, especially in

young children, while reductions in sedentary behaviour may promote weight loss or weight maintenance [18, 37]. For example, Burke et al. [38] report that every hour of television watching at age 6 was associated with a 50% increased risk of overweight or obesity at age 8 in a sample of Australian children, independent of other risk factors for weight gain. Similarly, a randomized controlled trial which reduced screen time resulted in significant reductions in both weight gain and the accumulation of abdominal fat in elementary school children [39]. However, few studies have found relationships between sedentary behaviour and weight gain in older children, suggesting that sedentary behaviour may only be a risk factor for obesity in young children [18, 37].

The potential mechanisms which are thought to link sedentary behaviour and adiposity involve deleterious changes to both EE and EI. Most obviously, sedentary activities are defined by having low EE [33] and may also displace PA, although there is currently little evidence that such displacement takes place [18, 40–42]. With respect to EI, excess sedentary behaviour may also result in "uncoupling" between EE and EI [14]. Further, it has been suggested that television viewing may exert a particularly negative impact on pediatric EI. For example, Wiecha and colleagues report that every one-hour increase in daily television viewing among school children is associated with an extra consumption of 167 calories [43]. In addition, it has also been suggested that exposure to television food advertisements increases children's food intake at subsequent meals [44, 45]. Thus, through its impact on both EE and EI, it is very plausible that sedentary behaviour has a deleterious impact on energy balance, and therefore body weight.

Given the strong and consistent relationship between sedentary behaviours and weight gain in early childhood, temporal trends which suggest that sedentary behaviours have increased in recent decades, and numerous plausible biological mechanisms, there is currently strong evidence that increases in sedentary behaviour play an important role in the epidemic of childhood obesity.

5. Increased Total Energy Intake

It is well established that intentional overfeeding results in significant weight gain [46–48]. For example, Levine and colleagues report that overfeeding volunteers by 1000 kcal/day results in an average weight gain of 5 kg in just 8 weeks [47]. However, counterintuitively, recent reviews have noted that total EI has not been a consistent predictor of weight gain in prospective studies of children [19, 49]. It is worth noting however that this may be due to the limitations of self- or parent-reported caloric intake, as both adults and children are known to have great difficulty in accurately reporting EI [50–52]. For example, Huang and colleagues report that fully 55% of children aged 3–19 who participated in the Continuing Surveys of Food Intakes by Individuals study reported physiologically implausible values for energy intake, and that excluding these individuals resulted in dramatic improvements in both the strength and the consistency of the relationship between EI and body weight [52].

As with PA, there is currently little information regarding historical trends in the EI of Canadian children, and data from the United States are equivocal as some [53–56], but not all [57, 58], studies report increased EI during the past half century. For example, self-reported energy intake from the 1977-1978 Nationwide Food Consumption Survey and the 1999–2004 National Health and Nutrition Examination Survey suggest that the average daily EI of American children aged 1–10 in 1999–2004 was 15% higher than in 1977-1978, with similar increases observed in adolescents [54, 55]. In contrast to these findings, however, Troiano and colleagues report that between 1970 and 1994, EI in American youth was relatively stable [57]. Thus, given the absence of Canadian data and the ambiguity of available data from our closest neighbour, there is currently insufficient evidence to conclude that EI has increased in Canadian youth during recent decades.

While prospective studies and historical trends may lend only weak support to the putative role played by EI in the Canadian childhood obesity epidemic, it remains extremely plausible biologically. As mentioned earlier, several trials have shown that intentional overfeeding results in dramatic weight gain in adults [46–48], and there is little reason to expect this relationship to be different in children. While it should be noted that there is evidence that overconsumption results in compensatory increases in EE in some individuals, [47], this would likely be insufficient to prevent an increase in obesity rates at the population level. Further, other consistent predictors of weight gain which lend themselves to more accurate self-reporting than total EI (e.g., television watching and sugar-sweetened beverages, which will be discussed below) are thought to exert their influence through their impact on EI. Thus, despite the weak evidence presented from observational studies, the strong biological plausibility and impressive results from studies of chronic overfeeding suggest that there is currently moderate evidence that increased EI has contributed to the childhood obesity epidemic.

6. Increased Sugar-Sweetened Beverage Intake

While trends in total EI over the past 40 years are unclear, there is little ambiguity for trends in sugar-sweetened beverage (SSB) intake, which has increased dramatically in recent decades [58–60]. For example, the average self-reported soft-drink intake in American youth increased from roughly 150 mL/day in 1977 to more than 350 mL/day in 1998 [59], and recent studies suggest that total SSB intake has continued to increase into the 21st century [60]. Interestingly, while this may be partially due to increased fast food consumption, available evidence suggests that SSB intake has also increased in the home environment in recent decades [56].

Several recent systematic reviews have also concluded that there is consistent evidence that excess consumption of SSBs is associated with an elevated risk of weight gain [19, 61, 62]. For example, among longitudinal studies, Vartanian and colleagues report significant effect sizes of 0.24 and 0.09 for the relationship of SSB consumption with total EI and body

weight, respectively [62]. Similarly, a 19-month prospective study of 548 school children reports that every serving of sugar-sweetened beverages at baseline was associated with a 0.18 kg/m^2 increase in BMI at followup [63]. Finally, it has recently been estimated that removing sugar-sweetened beverages from the diet of American children and youth would reduce caloric intake by an average of 235 calories per day [64], which has the potential to dramatically reduce the risk of positive energy balance in this age group. Thus, available evidence suggests that excessive consumption of SSBs plays a strong role in the etiology of the childhood obesity epidemic.

This relationship between SSB and prospective weight gain can be explained by multiple biological mechanisms. First and foremost, SSB intake is associated with increased EI, as described earlier [62]. This is likely due to the fact that SSBs are both energy dense and have little impact on satiety, both of which could lead to increases in EI [61]. Further, many SSBs are sweetened with high fructose corn syrup (HFCS) and therefore contain a fructose fraction. This fructose fraction may also contribute to weight gain through increased lipogenesis, inhibition of satiety signals, and reductions in EE, although it should be noted that the relative importance of HFCS in the etiology of obesity is still a matter of dispute [61, 65].

7. Increased Dietary Fat Intake

Not surprisingly, as the consumption of carbohydrates has increased during the past 30 years, the relative contribution of fat to total EI has decreased, although intake remains above recommendations [57, 58]. For example, Cavadini and colleagues report that between 1965 and 1996 fat intake decreased from 39% to 32% of EI for Americans aged 11–18 [58]. However, it should be noted that this same study found that *absolute* fat intake actually increased by 4% during the 1990's [58], suggesting that the relative changes in fat intake may have more to do with increased consumption of carbohydrates than with reductions in fat consumption.

The evidence linking fat intake and obesity in prospective studies is surprisingly equivocal and provides little support for the role of fat intake in the development of obesity [19]. For example, Davis and colleagues report that of 15 longitudinal studies of childhood weight gain reviewed by the American Dietetic Association, just 4 supported the role of dietary fat intake, while 4 others showed mixed results, and 7 found no association [19]. However, as with total EI, fat intake remains an extremely plausible mechanism biologically. Similar to SSBs, dietary fat is both energy dense and relatively nonsatiating per calorie ingested, lending itself to passive overconsumption [66]. Further, increased fat consumption appears to have little impact on fat oxidation or overall EE, suggesting that excess EI related to increased fat intake is very likely to result in positive energy balance and weight gain [67]. However, despite these plausible mechanistic links, available evidence provides only weak support of the role of dietary fat intake in the current childhood obesity epidemic.

8. Reduced Calcium Intake

Along with the changes in fat and SSB intake in recent decades, there has also been a well-documented reduction in the intake of dietary calcium [58, 68]. For example, between 1965 and 1996, Cavadini et al. report that the milk consumption of American youth decreased by 36%, while total calcium intake dropped by roughly 13% [58]. Further, it has been suggested that high calcium intake may influence body weight through increases in fecal fat excretion, fat oxidation, and thermogenesis [69, 70]. However, a recent meta-analysis of childhood calcium supplementation studies reports no significant association between supplementation and any measure of weight or body composition [71], which is supported by the findings of a similar review in adults [72]. Thus, while there is some evidence for plausible mechanisms linking reduced calcium with increased adiposity, the lack of evidence linking calcium intake with changes in actual measures of body composition suggests that reductions in calcium intake do not represent an important cause of the Canadian childhood obesity epidemic.

9. Reduced Sleep

Available evidence suggests that short-sleep duration may be another important risk factor for childhood overweight and obesity. A recent systematic review and meta-analysis by Cappuccio and colleagues reports that children who sleep less than 10 hours per night are at 89% greater risk than their peers who sleep more than 10 hours/night [73]. Using this same data, it has been estimated that 5 to 13% of all childhood obesity could be due to short-sleep duration [74]. Although the vast majority of the research to date has been cross-sectional [73], there is evidence of sleep as a predictor of weight gain in prospective studies as well. For example, Reilly and colleagues report that toddlers who slept less than 11 hours per night at age 2.5 years were 35–45% more likely to be obese at age 7 than toddlers who averaged more than 12 hours of sleep [75], with similar findings reported by Bell and Zimmerman [76].

Secular trends in sleep duration also support the putative role of sleep duration in the childhood obesity epidemic. Since the 1970's, the average sleep duration of children has decreased significantly among industrialized nations. Between 1974 and 1986, the average sleep time of 2-year olds in the Zurich Longitudinal Studies decreased by 45 minutes [77], while Dollman and colleagues report a 30-minute decrease from 1985 to 2004 among South Australian teenagers [78]. Similarly, the prevalence of sleep-onset difficulties has also increased dramatically in recent years [79].

Finally, a putative role for shortened sleep in the etiology of the obesity epidemic is also supported by plausible mechanisms which are thought to influence both EE and EI [80, 81]. For example, it has been reported that sleep restriction in adults results in significant increases in hormones which promote EI including cortisol and ghrelin, along with decreases in anorectic hormones such as leptin and PYY [80, 82–84]. Not surprisingly, short-sleep duration has also

been shown to result in increased hunger and appetite, both of which were strongly associated with the changes in ghrelin and leptin mentioned earlier [84]. Given that leptin and ghrelin are thought to, respectively, promote and inhibit physical activity, it has been suggested that sleep debt could potentially result in reductions in EE as well [81, 85]. However, recent experimental evidence in young men suggests that acute sleep restriction results in relatively little change in EE [86]. Thus, at present it appears very likely that sleep deprivation results in increased EI, while there is little direct evidence that it results in reduced EE. When these biological mechanisms are considered alongside the consistent relationship between shortened sleep and obesity in prospective studies, and secular trends in sleep duration, there is currently strong evidence that shortened sleep plays a role in the childhood obesity epidemic.

10. Prenatal Exposure to Endocrine-Disrupting Chemicals

Endocrine-disrupting chemicals (EDCs) are any "compound, either natural or synthetic, which alters the hormonal and homeostatic systems that enable the organism to communicate with and respond to its environment" [87], several of which (known as obesogens) may influence body weight [88]. Limited evidence suggests that EDCs may exert a negative influence on aspects of EE. For example, it has been reported that mothers who have high levels of polychlorinated biphenyls (PCBs) in their breast milk also have low levels of plasma triiodothyronine, a thyroid hormone which is known to stimulate basal metabolism [89]. Similarly, interventions in adults which increase plasma organochlorine concentrations result in significant decreases in both triiodothyronine and resting metabolic rate [90] and may also reduce skeletal muscle oxidative capacity [91]. However, despite this limited biological evidence linking EDCs and EE, at present it is unclear whether prenatal exposure to EDCs predisposes to future weight gain [92]. For example, while some reports suggest that the concentration of PCBs in cord blood is positively associated with BMI in early childhood [92], other reports suggest no relationship [93], or even a negative relationship [94] between prenatal PCB exposure and prospective weight gain. Similar inconsistencies have also been observed for other EDCs such as DDE [92]. Thus, while being an interesting area for future research, at present there is very little evidence that EDCs play a causal role in the childhood obesity epidemic.

11. Increased Maternal Age

The average age of first pregnancy has increased dramatically in recent decades in both Canada [95–97] and around the world [98–100], and several plausible mechanisms have been suggested, which could link maternal age with increased risk of childhood obesity. For example, older mothers are known to give birth to smaller infants, which is itself a risk factor for the development of obesity [96, 101]. Similarly, older women are also likely to have both higher plasma concentrations of EDCs and higher BMIs, both of which may also predispose their children to future weight gain, as discussed elsewhere in this review [102–104]. Finally, research in sheep suggests that older maternal age may result in increased fat deposition [105], which may be related to accelerated reductions of proteins responsible for thermogenesis-related energy expenditure [25], although it is not immediately clear how or if this relates to humans.

Although the mechanisms described above are all at least somewhat plausible, the relationship between maternal age and childhood obesity in observational studies is inconsistent. For example, while Patterson and colleagues report that the odds of obesity in a cohort of American girls increased by 14% for every 5-year increase in maternal age [106], a more recent study of 8234 British children found no relationship between maternal age and risk of obesity at age 7 [75]. Given this conflicting evidence, there is currently only weak evidence that maternal age plays a role in the childhood obesity epidemic, and future prospective studies are needed to clarify this relationship.

12. Reduced Breastfeeding

Duration of breastfeeding has been strongly and consistently linked with reduced risk of childhood overweight and obesity [107]. For example, Harder and colleagues performed a meta-analysis which examined the association between duration of breastfeeding and the risk of childhood overweight in 17 independent observational studies [107]. In comparison to children who were breastfed for less than 1 month, they report that children who were breastfed for 1–3 months had 19% reduced risk of overweight. The risk of being overweight continued to decrease as the duration of breastfeeding increased—risk was reduced by 24% among those breastfed for 4–6 months, 33% among those breastfed for 7–9 months, and by 50% for those breastfed for more than 9 months. On average, each additional month of breastfeeding reduced the risk of being overweight by 4%.

Despite consistent reports of the relationship between breast feeding and reduced risk of overweight and obesity, the mechanisms underpinning this relationship remain unclear. It has been suggested that it may be due to alterations in the neuroendocrine control of appetite, although this has yet to be verified in human participants [107]. Thus, it is not possible at present to determine the precise mechanisms linking the duration of breastfeeding to body weight in childhood.

While breastfeeding appears to have a strong relationship with the risk of excess weight gain in childhood, trends in the prevalence of breastfeeding suggest that it is not a major contributor to secular increases in childhood obesity rates during the 20th century. Since the 1970's, the prevalence of breastfeeding has remained constant or increased among most western nations for which data is available [108, 109]. For example, in the early 1970's roughly 20% of American women exclusively breastfed while in the hospital, but this increased to 45% by the year 2000 [109]. Given that obesity rates continued to increase steadily throughout this period

despite increases in the prevalence of breastfeeding, there is currently weak evidence that breastfeeding plays a primary role in the childhood obesity epidemic.

13. Increased Adult Obesity Rates

Available evidence suggests that both parental obesity and gestational weight gain are risk factors for childhood obesity [75, 110, 111]. For example, Reilly and colleagues examined the relationship between parental and childhood obesity in a prospective study of nearly 9,000 British children [75]. In comparison to children born to two nonobese parents, they report that children were 2.5 times more likely to be obese when they had an obese father, and 4.3 more likely to be obese if they had an obese mother. Further, children born to two obese parents were more than 10 times more likely to develop obesity by age 7 than those born to two non-obese parents [75]. It has been reported that gestational weight gain is also a predictor of childhood obesity, and that this impact is stronger in women who were obese prior to becoming pregnant [111]. Finally, recent reports suggest that surgical weight loss prior to pregnancy dramatically reduces the risk of childhood obesity in babies born to obese women [112]. These relationships suggest that any putative cause of the increasing prevalence of adult obesity [113] including those that are unlikely to play a direct role in the epidemic of childhood obesity (e.g., iatrogenic weight gain [25]) may nonetheless play important indirect roles.

The relationship between parental and childhood obesity is likely to be linked via numerous mechanisms. For example, genetic factors are reported to account for roughly 25% of the variance in fat mass [114], which is likely to mediate some of the relationship in body composition between parent and child. Further, learned behavioural characteristics such as food choices, PA, and sedentary behaviours are also likely to mediate the transmission of intergenerational obesity [75, 115]. Finally, studies of animal models suggest that obesity or excessive weight gain during pregnancy is likely to predispose childhood obesity through deleterious changes in the central regulation of energy balance [116]. For example, lambs born to overfed ewes are less sensitive to signals of excess nutrient supply or fat mass than lambs born to control animals [116]. Taken together, the strong association between parental and childhood obesity and the numerous plausible mechanisms underlying these associations suggest that one of the most important drivers of the childhood obesity epidemic may in fact be adult obesity.

14. Relative Contributions to Childhood Obesity

As has been noted by others, there is currently insufficient information to make a truly objective ranking of the putative causes of an issue as complex as the current obesity epidemic [25]. However, the evidence presented above does allow some general conclusions to be made. This review has identified 4 factors—reduced sleep, increased sedentary time, increased consumption of sugar-sweetened beverages, and secular increases in adult obesity—which are likely to have made an important contribution to Canada's childhood obesity epidemic. Each of these factors has shown strong and consistent associations with childhood weight gain, has increased in prevalence during the obesity epidemic, and results in either biological or behavioural changes that are likely to promote positive energy balance. Of these, adult obesity appears to have the most powerful impact on childhood obesity levels, while reducing the consumption of sugar-sweetened beverages may be among the simplest ways to prevent future weight gain in individuals of all ages.

Available evidence provides only moderate support for the role of either total EI or PA in the etiology of childhood obesity. This is likely due to methodological limitations of self-reported intake and expenditure, as both of these factors are biologically plausible and have been shown to have impressive effects on adiposity in experimental studies. It is possible that methodological limitations may also explain the inconsistent relationships seen between obesity and dietary fat intake. Future studies employing more objective methods of measurement are important to determine the true role of these factors in the etiology of the childhood obesity epidemic.

Finally, although each has been linked in some way with childhood obesity, there is currently weak evidence supporting the role of maternal age, breastfeeding, exposure to endocrine disrupters, or calcium insufficiency in the etiology of the childhood obesity epidemic. Of these, maternal age, breastfeeding, and endocrine disruptors appear worthy of future study, while there is sufficient evidence to conclude that calcium intake plays little role in pediatric obesity rates at the population level.

15. Summary

Although influenced by numerous factors, available evidence suggests that the Canadian childhood obesity epidemic is most closely related to deleterious changes in sugar-sweetened beverage intake, sedentary behaviour, reduced sleep, and adult obesity. Interventions aimed at modifying these factors may help to prevent further increases in obesity rates among the Canadian pediatric population.

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REVIEW Open Access

Systematic review of sedentary behaviour and health indicators in school-aged children and youth

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Abstract

Accumulating evidence suggests that, independent of physical activity levels, sedentary behaviours are associated with increased risk of cardio-metabolic disease, all-cause mortality, and a variety of physiological and psychological problems. Therefore, the purpose of this systematic review is to determine the relationship between sedentary behaviour and health indicators in school-aged children and youth aged 5-17 years. Online databases (MEDLINE, EMBASE and PsycINFO), personal libraries and government documents were searched for relevant studies examining time spent engaging in sedentary behaviours and six specific health indicators (body composition, fitness, metabolic syndrome and cardiovascular disease, self-esteem, pro-social behaviour and academic achievement). 232 studies including 983,840 participants met inclusion criteria and were included in the review. Television (TV) watching was the most common measure of sedentary behaviour and body composition was the most common outcome measure. Qualitative analysis of all studies revealed a dose-response relation between increased sedentary behaviour and unfavourable health outcomes. Watching TV for more than 2 hours per day was associated with unfavourable body composition, decreased fitness, lowered scores for self-esteem and pro-social behaviour and decreased academic achievement. Meta-analysis was completed for randomized controlled studies that aimed to reduce sedentary time and reported change in body mass index (BMI) as their primary outcome. In this regard, a meta-analysis revealed an overall significant effect of -0.81 (95% CI of -1.44 to -0.17, p = 0.01) indicating an overall decrease in mean BMI associated with the interventions. There is a large body of evidence from all study designs which suggests that decreasing any type of sedentary time is associated with lower health risk in youth aged 5-17 years. In particular, the evidence suggests that daily TV viewing in excess of 2 hours is associated with reduced physical and psychosocial health, and that lowering sedentary time leads to reductions in BMI.

Keywords: Inactivity, sitting, TV, body composition, fitness, metabolic syndrome, cardiovascular disease, self-esteem, pro-social behaviour, academic achievement

Introduction

Engaging in regular physical activity is widely accepted as an effective preventative measure for a variety of health risk factors across all age, gender, ethnic and socioeconomic subgroups [1-6]. However, across all age groups, levels of physical activity remain low [7-12] and obesity rates continue to rise [10,11,13,14]; collectively threatening the persistent increase in life expectancy enjoyed over the past century and efforts to counteract the inactivity and obesity crisis [15].

This inactivity crisis is especially important in the pediatric population as recent data from the Canadian Health Measures Survey [8] suggest that only 7% of children and youth aged 6-19 years participate in at least 60 minutes of moderate- to vigorous-intensity physical activity per day, thus meeting the current physical activity guidelines from Canada [16], the U.S. [6], the U.K [17], Australia [18] and the World Health Organization (WHO) [5]. However, even for those children and youth who meet current guidelines, there remains 23 hours per day for school, sleep, work, and discretionary time. Several sources report that children and youth spend the majority of their

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discretionary time engaging in sedentary pursuits (e.g. watching television (TV) or playing video games) [8,19-28]. Canadian children and youth are spending an average of 8.6 hours per day, or 62% of their waking hours being sedentary [8]. Similar trends are being reported in the U.S. where children and youth spend an average of 6-8 hours per day being sedentary [22-28]. Accumulating evidence shows that, independent of physical activity levels, sedentary behaviours are associated with increased risk of cardio-metabolic disease, all-cause mortality, and a variety of physiological and psychological problems [29-31]. Therefore, to maximize health benefits, approaches to resolve the inactivity crisis should attempt to both increase deliberate physical activity *and* decrease sedentary behaviours, especially in the pediatric population. However, to date, public health efforts have focused primarily on physical activity and have paid little attention to the mounting evidence to support sedentary behaviour as a distinct behaviour related to poor health.

A recent scoping review identified review articles, meta-analyses, and grey literature that examined the relationship between sedentary behaviour and health [32]. The large majority of this information reported on the relationship between screen time and body composition and did not include other indicators of health [23-25]. Furthermore, none of these reviews followed the rigorous process of a systematic review and are therefore not able to be used to inform the development of clinical practice guidelines. As a result, to our knowledge, there are no systematic, evidence-based sedentary behaviour guidelines for any age group, anywhere in the world. Guidelines that do exist are largely based on expert opinion or narrative literature reviews [33,34].

Therefore, the purpose of this systematic review was to gather, catalog, assess and evaluate the available evidence examining sedentary behaviours in relation to selected health outcomes in children and youth 5-17 years of age and present a summary of the best available evidence. Specifically, the review presents available evidence for minimal and optimal thresholds for daily sedentary time in children and youth, and when possible, how thresholds differ across health outcome or demographic status (i.e. age, gender). The information gathered in this review can serve to guide future research and inform the development of evidence-based clinical practice guideline recommendations for safe and healthy amounts of daily sedentary behaviour in the pediatric population.

Methods

Study Inclusion Criteria

The review sought to identify all studies that examined the relationship between sedentary behaviour and a specific health outcome in children and youth (aged 5-17 years). All study designs were eligible (e.g. cross sectional, retrospective, prospective, case control, randomized controlled trial (RCT), longitudinal). Longitudinal studies were included if the data presented in the article was consistent with the age limits that were set (i.e. if the study looked at participants at age 10 and then again at age 30, only baseline measurements from age 10 were used).

Studies were included only if there was a specific measure of sedentary behaviour. Eligible exposures of sedentary behaviours included those obtained via direct (e.g., measurements of sitting, or low activity measured by accelerometer) and self-reported (e.g., questionnaires asking about TV watching, video gaming, non-school computer use, and screen time - composite measures of TV, video games, computers) methods. Sedentary behaviour was often measured as a composite measure of all time engaging in sedentary behaviours including screen time outside of school hours. Six health indicators were chosen based on the literature, expert input, and a desire to have relevant measures from a range of holistic health indicators (i.e. not only physical health, but also emotional, mental and intellectual health). The six eligible indicators in this review were:

1. Body composition (overweight/obesity measured by body mass index (BMI), waist circumference, skin folds, bio-impedance analysis (BIA), dual-energy x-ray absorptiometry (DXA or DEXA));
2. Fitness (physical fitness, physical conditioning, musculoskeletal fitness, cardiovascular fitness);
3. Metabolic syndrome (MS) and cardiovascular disease (CVD) risk factors (unfavourable lipid levels, blood pressure, markers for insulin resistance or type 2 diabetes);
4. Self-esteem (self-concept, self-esteem, self efficacy);
5. Behavioural conduct/pro-social behaviour (child behaviour disorders, child development disorder, pro-social behaviour, behavioural conduct, aggression);
6. Academic achievement (school performance, grade-point average).

No Language or date limits were imposed in the search. The following definitions were used to help guide the systematic review [31]:

- *Sedentary*: A distinct class of behaviours (e.g. sitting, watching TV, playing video games) characterized by little physical movement and low energy expenditure (≤ 1.5 METs).
- *Sedentarism*: Engagement in sedentary behaviours characterized by minimal movement, low energy expenditure, and rest.

- *Physically active:* Meeting established physical activity guidelines (e.g. see Tremblay et al. 2011 for Canadian Physical Activity Guidelines [16]).
- *Physical inactivity:* The absence of physical activity, usually reflected as the proportion of time not engaged in physical activity of a pre-determined intensity and therefore not meeting established physical activity guidelines.

Study Exclusion Criteria

As the volume of literature on sedentary behaviour was anticipated to be very high, to control the feasibility of this project, the following sample size limits were set *a priori*: population based studies (observational, cross sectional, cohort, and retrospective studies) were required to have a minimum sample size of 300 participants; RCTs, and intervention studies were required to have at least 30 participants. Studies of 'active gaming' (e.g., Nintendo Wii™, Microsoft Kinect™, Sony's Playstation Move™, video arcades, etc.) were excluded. Finally, studies that defined sedentary behaviour as 'failing to meet physical activity guidelines' were excluded from the review.

Search strategy

The following electronic bibliographic databases were searched using a comprehensive search strategy to identify relevant studies: Ovid MEDLINE(R) (1950 to February Week 2 2010), Ovid EMBASE (1980 to 2010 Week 07), and Ovid psycINFO (1806 to February Week 3 2010). The search strategy was created by a single researcher (JM) and run by a second researcher (AL). The search strategies can be found in Additional file 1. The search was limited to studies looking at 'school-aged' children and youth (mean age of 5-17 years). Articles were extracted as text files from the OVID interface and imported in to Reference Manager Software (Thompson Reuters, San Francisco, CA). Duplicate articles were first removed using Reference Manager Software, and any remaining duplicates were removed manually. All articles were given a unique reference identification number in the database.

Titles and abstracts of potentially relevant articles were screened by two reviewers (AL and one of GG, MT, RC, RL or TS) and full text copies were obtained for all articles meeting initial screening by at least one reviewer. Two independent reviewers examined all full text articles (AL and one of GG, MT, RC, RL or TS) and any discrepancies were resolved by discussion and consensus between the two reviewers. If the reviewers were unable to reach consensus, a third reviewer was asked to look at the article in question. Consensus was obtained for all included articles.

Twelve key content experts were contacted and asked to identify the most influential papers from their personal libraries examining sedentary behaviour and health in the pediatric age group. Government documents from the U.S [6], the U.K. [17], and Australia [18] were used for reference and to help guide the review process.

Data extraction

Standardized data extraction tables were created; data extraction was completed by one reviewer (AL) and checked by another (one of GG, RC, RL, or TS) for accuracy. Information was extracted regarding study characteristics (i.e. year, study design, country, number of participants, age), type of sedentary behaviour, measure of sedentary behaviour (i.e. direct, or indirect), and health outcome. Reviewers were not blinded to the authors or journals when extracting data.

Risk of bias assessment

The Downs and Black checklist was used to asses study quality [35]. This 27 point checklist assesses the *quality of reporting* (e.g. "Are the main findings of the study clearly described"); *external validity* (e.g. "Were the subjects asked to participate representative of the entire population from which they were recruited"); *internal validity* (e.g. "Were subjects randomized to intervention groups"); and *power* (e.g. "Was there sufficient power such that the difference being due to chance is less than 5%"). The maximum score a study can receive is 32, with higher scores indicating better quality. Inter-rater reliability was calculated using Cohen's kappa.

Quality of evidence was determined by the study design and by Downs and Black score. Level of evidence was used to explain the quality of available studies and the confidence of the findings [36]. RCTs were considered to have the highest level of evidence while anecdotal reports were considered to have the lowest evidence. See Table 1 for more details. When possible, studies were examined for differences among age and gender subgroups.

Analysis

A meta-analysis was performed with the data that were sufficiently homogeneous in terms of statistical, clinical, and methodological characteristics using Review Manager Software 5.0 (The Cochrane Collaboration, Copenhagen Denmark). Pooled estimates for the meta-analysis and their 95% confidence intervals were obtained using the random effects estimator of DerSimonian-Laird [37]. Studies were weighted by the inverse of their variance. Cochrane's Q was used to test for heterogeneity among studies and the I^2 (squared) index [10] was used to determine the degree of heterogeneity [38]. Funnel plots were

Table 1 Criteria for assigning level of evidence to a recommendation

Level of evidence	Criteria
Level 1	- Randomized control trials without important limitations
Level 2	- Randomized control trials with important limitations - Observational studies (non-randomized clinical trials or cohort studies) with overwhelming evidence
Level 3	- Other observational studies (prospective cohort studies, case-control studies, case series)
Level 4	- Inadequate or no data in population of interest - Anecdotal evidence or clinical experience

Adapted from: Lau DC et al. 2007 [36]

used to assess publication bias (data not shown). Qualitative syntheses were conducted for remaining studies.

Results

Description of studies

After de-duplication, the preliminary search of electronic databases, reference lists, and grey literature identified 5,291 potentially relevant articles (Figure 1). Of these, 3,299 were identified in MEDLINE, 1,016 in EMBASE, 912 in psycINFO, and 64 through key informants, government documents, and bibliographies. After a preliminary review of titles and abstracts, 828 articles were included for detailed assessment of the full text article. Of these, 232 met the criteria for study inclusion (8 RCTs, 10 intervention studies, 37 longitudinal studies and 177 cross sectional studies). Individual study characteristics can be seen in Table 2. Reasons for excluding studies included: ineligible population (e.g. ineligible age or sample size) (n = 161), ineligible exposure (e.g. diet, physical activity) (n = 145), ineligible measure of sedentary behaviour (i.e. not meeting physical activity guidelines) (n = 19), ineligible outcome (n = 60), ineligible analysis (e.g. analysis focused on content of screen time versus duration of screen time, analysis focused on active video gaming) (n = 60), and 'other' (n = 216) (e.g. commentary article or methodological paper). Some studies were excluded for multiple reasons. Some articles (n = 9) could not be retrieved due to missing or incorrect reference information.

Table 2 provides a summary of all studies included in the review. The majority of the studies included in this systematic review were cross sectional (n = 177). In total, data from 983,840 participants were included in this review. Studies ranged from 30 participants in intervention studies and RCTs, to 62,876 participants in cross sectional observational investigations. Articles were published over a 51 year period from 1958 to 2009, and included participants ranging from 2-19 years of age. Although the scope of the review focused on those 5-17 years of age, studies that had a range below 5 years or over 17 years were not excluded as long as the mean age was between 5-17 years. Included studies involved participants from 39 countries; there were a greater number of articles reporting on female-only data than those reporting on male-only data. Translators were contracted to read non-English articles and complete any necessary data extraction for studies that met inclusion criteria (n = 8).

Of the 232 studies, 170 studies reported data on body composition, 15 on fitness, 11 on MS and CVD, 14 on self-esteem, 18 on pro-social behaviour, and 35 on academic achievement. The majority of studies (n = 223) used indirect measures to assess sedentary behaviour (i.e. parent-, teacher-, or self-report questionnaires). There were 14 studies [24,27,28,39-49] that directly measured sedentary behaviour with accelerometers and one that directly measured television viewing through a monitoring device [50]. The direction of the association between increased sedentary behaviour and health outcomes were similar between direct and indirect measures. Meta-analysis was conducted for RCTs examining change in body mass index.

Risk of bias assessment

Risk of bias assessment was completed for all included studies (Additional file 2). The mean Downs and Black score was 20.7 (range = 16-26). The studies were then split into groups and labeled as 'high quality' (score 23-26, n = 36), 'moderate quality' (score 19-22, n = 169), and 'lower quality' (score 16-18, n = 27). Quality of study did not affect the outcome of the study; in other words, both lower quality and high quality studies showed a positive relationship between increased time spent sedentary and health risk. Inter-reviewer assessment using the Downs and Black tool was very high (kappa = 0.98).

Data Synthesis

Body composition

Of the 232 studies included in this review, 170 examined body composition, with the majority of these focusing on the relationship between overweight and obesity and time spent watching TV (Table 3). Body composition was measured in a variety of ways including body mass index (BMI), sum of skin folds, percent body fat and various composite measures (e.g. BMI + sum of skin folds). Of the 8 RCTs, 7 showed that decreases in sedentary time lead to reductions in body weight (see meta-analysis below for details). Intervention studies reported desirable

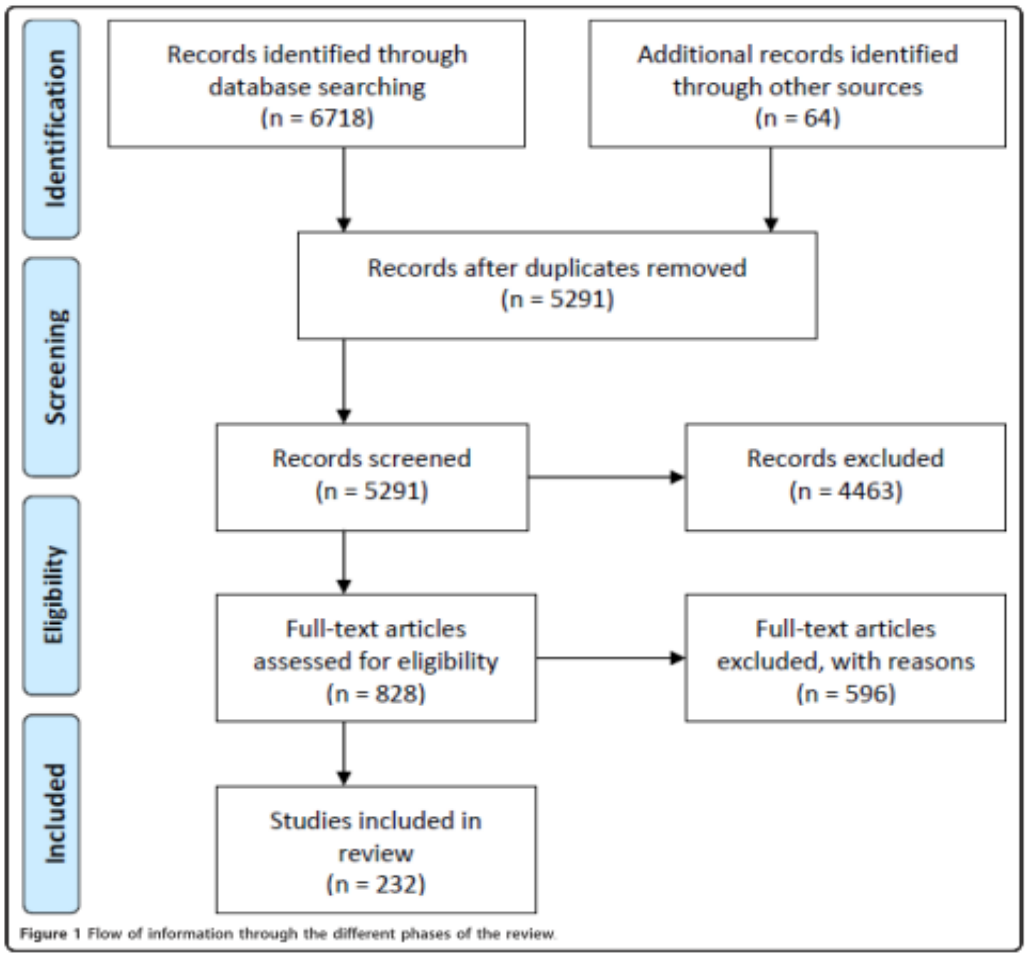

Figure 1 Flow of information through the different phases of the review.

changes in body weight, BMI, and weight status among children and youth who successfully decreased their sedentary time [51-60]. Three intervention studies [61-63] reported that although sedentary behaviour decreased, there was no change in weight status (measured through BMI and skinfold thickness); however, these studies had relatively short follow-up periods (~1 year) and no control group leading the authors hypothesized that a longer follow up period was needed to detect a significant change in body composition. While nine-teen longitudinal studies reported that children who watched greater amounts of TV at baseline saw steeper increases in BMI, body weight and fat mass over time [64-82], nine longitudinal studies reported no significant relationship between time spent sedentary and weight status or fat mass [61-63,83-89]. Of the 119 cross sectional studies, 94 reported that increased sedentary time was associated with one or more of increased fat mass, increased BMI, increased weight status and increased risk for being overweight [28,90-182]. Risk for obesity increased in a dose response manner with increased time spent engaging in sedentary behaviours [92,106,110,128, 156,178]. Twenty-five cross sectional studies reported no significant relationship between

Table 2 Summary of characteristics of included studies

First Author	Year	Country	Grade	Age Range	Mean age	*n* analyzed			Units of sedentary behaviour		Exposure	Outcome
						Total	Boys	Girls				
RANDOMIZED CONTROLLED TRIALS												
Epstein LH [265]	1995	US		8-12	10.1	61			hour	week	TV	BC
Epstein LH [50]	2008	US		4-7	6	70	37	33	hour	day	TV	BC
Goldfield GS [264]	2006	Canada		8-12	10.4	30	13	17	min	day	TV	BC
Gortmaker SL [57]	1995	US			11.7	1295	668	627	hour	day	TV	BC
Hughes AR [262]	1991	Scotland		5-11	8.8	134	59	74	hour	day	SB	BC
Robinson TN [58]	1999	US				192			hour	week	TV, GAMES	BC
Robinson TN [221]	2003	US		8-10	9.5	61	0	61	hour	week	TV	BC, SE
Shelton D [263]	2007	Australia		3-10	7.5	43	20	23	hour	day	TV	BC
INTERVENTION STUDIES												
Epstein LH [56]	2000	US		8-12	10.5	76	24	52	hour	month	SB, ST	BC, FIT
Epstein LH [59]	2004	US		8-12	9.8	60	23	39	times	week	TV	BC
Epstein LH [60]	2005	US		8-16		58	28	30	hour	day	SB, TV	BC
Gentile DA [61]	2009	US			9.6	1323	685	674	hour	day	ST	BC
Goldfield GS [52]	2007	Canada		8-12	10.4	30	13	17	hour	day	SB	BC, SE
Harrison M [62]	2003	Ireland			10.2	312	177	135	min	day	TV, ST	BC
Ochoa MC [53]	2007	Spain		6-18	11.6	370	196	174	hour	week	TV	BC
Salmon J [51]	2008	Australia		1011	10.8	311	152	159	hour	day	TV	BC
Simon C [54]	2002	France			11.7	954	468	486	hour	day	TV, COMP	BC, SE
Tanasescu M [55]	2000	Puerto Rico		7-10	9.2	53	22	31	hour	day	TV	BC
LONGITUDINAL STUDIES										hour		
Aires L [83]	2010	Portugal		11-19		345	147	198	hour	day	SCREEN	BC, FIT
Berkey CS [76]	2003	US		10-15		11887	5120	6767	hour	day	TV, GAMES	BC
Bhargava A [77]	2008	US				7635			min	day	TV	BC
Blair NJ [68]	2007	England			5.5	591	287	304	hour	day	SB, TV	BC
Borradaile KE [86]	2008	US			11.2	1092	501	591	hour	week	TV	BC
Burke V [71]	2006	Australia			7.6/10.8	1569	630	648	hour	week	SCREEN	BC
Chen JL [78]	2007	Chinese		7-8	7.52	307	147	160	hour	day	TV, GAMES	BC
Danner FW [66]	2008	US				7334	3674	3660	hour	day	TV	BC
Dasgupta K [215]	2006	Canada			12.7/15.1/17.0	662	319	343	hour	week	SB, TV	MS
Day RS [85]	2009	US		8-14		556	277	279	min	day	TV	BC
Dietz WH [181]	1985	US		12-17		2153			hour	day	TV	BC
Elgar FJ [79]	2005	Wales			11.7	654	293	361	hour	week	TV	BC
Elgar FJ [79]	2005	Wales			15.3	392	181	211	hour	week	TV	BC
Ennemoser M [237]	2007	German		6-8		332			min	day	TV	SE, AA
Fulton JE [84]	2009	US		10-18		472	245	227	min	day	TV	BC
Gable S [70]	2007	US				8000			hour	day	TV	BC
Hancox RJ [88]	2004	New Zealand		5-15		1013			hour	day	TV	BC, MS
Hancox RJ [72]	2006	New Zealand		5-15		603	372	339	hour	day	SCREEN	BC
Henderson VR [67]	2007	US		11-19		2379	0	2379	hour	day	TV, SCREEN	BC
Hesketh K [80]	1997	Australia		5-10	7.6	1278	630	648	hour	day	SCREEN	BC
Hesketh K [80]	1997	Australia		8-13	10.7	1278	630	648	hour	day	SCREEN	BC
Hesketh K [64]	2009	Australia		5-10	7.7	1943	972	971	hour	day	TV, GAMES	BC
Hesketh K [64]	2009	Australia		8-13		1569	816	753	hour	day	TV, GAMES	BC
Jackson LA [223]	2009	US			12	500	235	265	hour	day	COMP, SCREEP	SE
Jago R [82]	2005	US		5-6	6.5	138	65	73	min	hr	SB, TV	BC
Janz KF [73]	2005	US			5.6/8.6	378	176	202	hour	day	SCREEN	BC
Johnson JG [41]	2007	US							hour	day	TV	AA

Table 2 Summary of characteristics of included studies *(Continued)*

Kaur H [75]	2003	US		12-17		2223	1149	1074	hour	day	TV	BC
Lajunen HR [128]	2007	Finland		15-19		5184			hour		SB	BC
Lonner W [238]	1985	US		9-19	14.2	367			hour	day	TV	AA
Maffeis C [89]	1998	Italy			8.7	298	148	150	min	day	SCREEN	BC
Mistry K [229]	2007	US							hour	day	TV	PRO
Mitchell JA [49]	2009	UK		11-12	11.8	5434	2590	2844	hour	day	SB	BC, FIT
Must A [87]	2007	US		10-17		156	0	156	hour	day	SB, SCREEN	BC
O'Brien M [69]	2007	US		2-12		653			hour	week	TV	BC
Parsons TJ [74]	2005	England/Scotland/Wales			11/16	17733			hour	day	TV	BC
Purslow LR [63]	2008	England		8-9		345	176	169	min	day	SB	BC
Timperio A [65]	2008	Australia		10-12		344	152	192	times	week	SB, SCREEN	BC
Treuth MS [29]	2007	US			11.9	984	0	984	min	day	SB	BC
Treuth MS [27]	2009	US			13.9	984	0	984	min	day	SB	BC
WosjeKS [205]	2009	US		6.75-7.25		214			hour	day	SCREEN	FIT
CROSS SECTIONAL STUDIES												
Al SH [192]	2009	International		12-18		17715	8503	9212	hour	day	TV	BC
Albarwani S [207]	2009	Oman		15-16		529	245	284	hour	week	TV, COMP	FIT
Alves JG [191]	2009	Brazil		7-10		733	407	326	hour	day	TV	BC
Aman J [218]	2009	Sweden		11-18	14.5	2093	1016	991	hour	week	TV, COMP	MS
Andersen LF [155]	2005	Norway		8-14		1432	702	730	hour	day	TV	BC
Andersen RE [142]	1998	US		8-16		4063	1985	2071	hour	day	TV	BC
Anderson SE [103]	2008	US		4-12	8	2964	1509	1455	hour	day	TV	BC
Armstrong CA [213]	1998	US			9.28	588	304	284	hour	day	TV	FIT
Asante PA [183]	2009	US		3-13	8.5	324	182	142	hour	day	SCREEN	BC
Aucote HM [163]	2009	Australia	5-6		11.09	393	198	195	hour	week	TV, GAMES	BC
Barlow SE [151]	2007	US		6-17	12.1	52845			hour	day	TV	BC
Basaldua N [109]	2008	Mexico		6-12	8.9	551	278	273	hour	day	TV	BC
Bellisle F [123]	2007	France		9-11		1000	500	500	hour	day	TV	BC
Berkey CS [90]	2000	US			Sep-14	10769	4620	6149	hour	day	TV	BC
Beyerlein A [105]	2008	Germany		4.5-7.3		4967	2585	2382	hour	day	TV	BC
Boone JE [164]	2007	US			15.9	9155	4879	4276	hour	week	SCREEN	BC
Boone-Heinonen J [104]	2008	US		11-21		9251			hour		SB	BC
Boutelle KN [130]	2007	US		16-18		1726	890	836	hour	day	TV	BC
Brodersen NH [235]	2005	England			11.8	4320	2578	1742	hour	week	SB	SE, PRO
Bukara-Radujkovic G [96]	2009	Bosnia		11-12	11.5	1204	578	626	hour	day	TV, COMP	BC
Butte NF [119]	2007	US		6-17	10.8	897	441	456	hour	day	SCREEN	BC
Caldas S [245]	1999	US		4-19		34542			hour	day	TV	AA
Carvalhal MM [131]	2007	Portugal	10-11			3365	1755	1610	hour	day	TV, COMP	BC
Chaput J [154]	2006	Canada		5-10	6.6	422	211	211	hour	day	SCREEN	BC
Chen MY [78]	2007	Taiwan		13-18	15.03	660	351	309	hour	day	TV, COMP	BC, SE, PRO
Chowhan J [232]	2007	Canada		12-15		2666			hour	day	TV	PRO
Christoforidis A [95]	2009	Greece		4-18	11.41	1549	735	814	hour	day	SCREEN	BC, FIT
Collins AE [149]	2008	Indonesia		12-15		1758	815	916	hour	day	TV, COMP	BC
Colwell J [200]	2003	Japan		12-13		305	159	146	hour	day	SCREEN	BC, PRO
Cooper H [247]	1999	US	7-11			424	225	199	hour	day	TV	AA
Crespo CJ [177]	2001	US		8-16		4069	1994	2075	hour	day	TV	BC
Da CR [157]	2003	Brazil		7-10		446	107	107	hour	day	TV	BC
Dasgupta K [215]	2007	Canada		13-17		1267			hour	week	SCREEN	MS
Delva J [125]	2007	US				11265	5274	5991	hour	week	TV	BC
Dietz WH [181]	1985	US		12-17		6671			hour	day	TV	AA
Dietz WH [181]	1985	US		6-11		6965			hour	day	TV	BC, AA
Dollman J [211]	2006	Australia	6	10-11		843	439	404	min	Day	TV	FIT

Table 2 Summary of characteristics of included studies *(Continued)*

Dumais SA [255]	2009	US		10-12		15850			hour		TV	AA
Dominick JR [225]	1984	US	10, 11	14-18		250	110	140	hour	Day	TV, GAME	SE, PRO
Eisenmann JC [175]	2002	US		14-18		15143			hour	day	TV	BC
Eisenmann JC [113]	2008	US'			16.2	12464	6080	6384	hour	day	TV	BC
Ekelund U [134]	2006	Europe		9-16		1921	911	1010	hour	day	TV	BC, MS
Fetler M [249]	1984	US	6			10603			hour	day	SCREEN	AA
Forshee RA [201]	2004	US		12-16	14	2216	1075	1141	hour	day	TV	BC
Forshee RA [188]	2009	US		5-18		1459	734	725	hour	week	SCREEN	BC
Gaddy GD [257]	1986	US				5074			hour	day	TV	AA
Giammattei J [140]	2003	US		11-14	12.6	385	186	199	hour	day	TV	BC
Gibson S [156]	2004	England		7-18		1294	655	639	min	day	TV	BC
Gomez LF [150]	2007	Colombia		5-12		11137	5539	5598	hour	day	TV, GAMES	BC
Gordon-Larsen P [176]	2002	US		11-19	15.9	12759	6290	6496	hour	week	TV, GAMES	BC
Gortmaker SL [143]	1996	US		10-15	11.5	746	388	358	hour	day	TV	BC
Gortmaker SL [57]	1999	US		6-11		1745			min	week	TV	SE, AA
Gortmaker SL [57]	1999	US		12-17		1745			min	week	TV	SE, AA
Graf C [167]	2004	Germany			6.8	344	177	167	hour	day	TV, COMP	BC
Grusser SM [40]	2005	Germany	6		11.83	323	175	148	hour	day	TV	AA
Hardy LL [133]	2006	Australia		11-15		2750	1446	1304	hour	day	SCREEN	FIT
Hernandez B [178]	1999	Mexico		9-16		461	244	217	hour	day	TV	BC
Hirschler V [144]	2009	Argentina		7-11	8.9	330	168	162	hour	day	TV	BC
Holder MD [222]	2009	Canada		8-12		375	252	262	hour	day	SCREEN	SE
Hume C [190]	2009	Netherlands			13	580	277	303	hour	day	SCREEN	BC
Islam-Zwart K [195]	2008	US				480	198	282	hour	day	TV	BC
Jackson LA [223]	2009	US			12.18	515	259	256	hour	day	GAMES, COMP	AA
Janssen I [166]	2004	Canada		11-16		5890	2812	3078	hour	day	TV, COMP	BC
Janz K [174]	2002	US		4-6	5.3	462	216	246	hour	day	TV	BC
Jaruratanasirikul S [241]	2009	Thailand	7-12		15.9	1492	562	929	hour		GAMES	AA
Johnson CC [41]	2007	US			12	1397	0	1397	hour	day	SB	SE
Katzmarzyk PT [197]	1998	Canada		9-18		784	423	361	min	day	TV	BC, FIT
Katzmarzyk PT [184]	1998	Canada				640	356	284	hour	day	TV	BC, FIT
Kautiainen S [135]	2005	Finland		14-18		6515	2916	3599	hour	day	SCREEN	BC
Keith TZ [256]	1986	US	high school seniors			28051			hour	day	TV	AA
Klein-Platat C [165]	2005	France			12	2714	1357	1357	hour	week	SB	BC
Kosti RI [196]	2007	Greece		12-17		2008	1021	987	hour	day	TV	BC
Kristjansson AL [243]	2009	Iceland		14-15		5810	2807	3004	hour	day	TV	AA
Kuntsche E [230]	2006	International		11-15		31177			hour	day	TV	PRO
Kuriyan R [117]	2007	India		6-16		598	324	274	hour	day	TV	BC
Lagiou A [160]	2008	Greece		10-12		633	316	317	hour	day	TV, GAMES	BC
Lajous M [92]	2009	Mexico		11-18	13.9	9132	3519	5613	hour	day	TV	BC
Lajunen HR [128]	2007	Finland			17.6	4098	1981	2117	hour	week	COMP	BC
Lasserre AM [116]	2007	Switzerland		10.1-14.9	12.3	5207	2621	2586	hour	day	TV	BC
Laurson KR [107]	2008	US		7-12		709	318	391	hour	week	SCREEN	BC
Lazarou C [217]	2009	Cyprus			11.7	622	306	316	hour	day	TV	MS
Leatherdale ST [11]	2008	Canada		14-19		25416	12806	12610	hour	day	TV	BC, PRO
Lioret S [127]	2007	France		3-14		1016	528	488	hour	day	SB, TV, COMP	BC
Lobelo F [208]	2009	US		14-18		5210	0	5210	hour	day	SCREEN	FIT
Lowry R [173]	2002	US				15349	7445	7828	hour	day	TV	BC
Lutfiyya MN [118]	2007	US		5-17		7972			hour	day	TV	BC
Maffeis C [114]	2008	Italy		8-10	9.3	1837	924	913	hour	day	TV	BC
Mark AE [220]	2008	US		12-19	15.9	1803	1005	798	hour	day	TV	BC, MS
McMurray RG [187]	2000	US		10-16	12.7	2389	1149	1240	hour	day	TV	BC
Mihas C [193]	2009	Greece		12-17	14.4	2008	1021	987	hour	day	SCREEN	BC

Table 2 Summary of characteristics of included studies *(Continued)*

Mikolajczyk RT [194]	2008	Germany		11-17	13.5	4878	2433	2445	hour	low/ high	SB	BC
Moraes SA [135]	2006	Mexico		6-14	8.0/11.3	662	343	339	hour	week		
Morgenstern M [94]	2009	Germany/US		10-17	12.8	4810	2294	2516	hour	day	SCREEN	BC
Morgenstern M [94]	2009	Germany/US		12-16	14	4473	2239	2234	hour	day	SCREEN	BC
Mota J [199]	2006	Portugal			14.6	450	220	230	hour	day	TV, COMP	BC
Muller MJ [179]	1999	Germany		5-7		1468	739	729	hour	day	TV	BC
Nagel G [193]	2009	Germany		6-9	7.57	1079		498	hour	day	TV, GAMES	BC
nastassea-Vlachou K [240]	1996	Greece		6-13		4690	2279	2411	hour	day	TV	AA
Nawal LM [148]	1998	US		5-18		62976			hour	day	TV, COMP	BC
Nelson MC [233]	2006	US		7-12		11957	5979	5978	hour	day	SCREEN	PRO
Neumark-Sztainer D [224]	2004	US		11-18	14.9	4746	2382	2364	hour	week	TV	SE, PRO
Nogueira JA [45]	2009	Brazil		8.3-16.8	13	326	204	122	hour	day	SB	BC
Obarzanek E [180]	1994	US		9-10	10.1	2379	0	2379	hour	week	TV	BC
Ohannessian CM [226]	2009	US		14-16	14.99	328	138	190	hour	day	SCREEN	SE, PRO, AA
Ortega FB [122]	2007	Spain		13-18.5	15.4	2859	1357	1502	hour	day	SB	BC
Overby NC [219]	2009	Norway		6-19		723	375	348	min	day	TV	
Ozmert E [42]	2002	Turkey				689	343	346	hour	day	TV	PRO, AA
Padez C [99]	2009	Portugal		7-9		3390	1696	1694	hour	day	TV	BC
Page RM [234]	2001	Philippine			15.1	3307	1267	1819	hour	week	TV	PRO
Pate RR [210]	2006	US		12-19	15.4	3287	1686	1601	hour	day	TV	FIT
Patrick K [169]	2004	US		11-15	12.7	878	407	471	min	day	TV	BC
Pratt C [101]	2008	US			12	1458	223	1235	hour	day	SB	BC
Purath J [185]	1995	US	3-5			365	189	176	hour	day	TV	BC, MS
Ramos E [126]	2007	Portugal		13		2161	1045	1116	min	week	SB, TV, COMP	BC
Rapp K [138]	2005	Germany			6.2	2140	1015	1125	hour	day	TV	BC
Ridley-Johnson R [252]	1983	US	5-8			290			hour	day	TV	AA
Roberts DF [250]	1984	US				539			hour	week	TV	AA
Robinson TN [58]	1999	US			12.4	971	0	971	hour	day	TV	BC
Ruangdaraganon N [141]	2002	Thailand		6-12	9.4	4197	2126	2035	hour	day	TV	BC
Russ SA [147]	2009	US		6-17		54863	28153	26710	hour	day	SCREEN	BC, SE
Sakamoto A [236]	1994	Japan	4-6			307	165	142	times	week	GAMES	PRO
Sakamoto A [236]	1994	Japan	4-6			537	287	250	hour	week	COMP, GAMES	PRO
Sakamoto A [236]	1994	Japan	4-5			118	118	0	hour	week	COMP, GAMES	PRO
Salmon J [136]	2006	Australia		5-12		1560	743	817	hour	day	TV	BC
Sardinha LB [48]	2008	Portugal		9-10	9.8	308	161	147	hour	day	SB	MS
Scott LF [254]	1958	US	6-7			407			hour		TV	AA
Sharif I [244]	2006	US		10-14		6522	3169	3353	hour	day	TV, GAMES	PRO, AA
Sharif I [260]	2010	US		9-15	12	4508	2209	2299	hour	day	TV, GAMES	AA
Shejwal B [246]	2006	India			16.05	654	368	286	hour	day	TV	AA
Shields M [162]	2006	US/Can		2-17		8661			hour	day	SB, TV	BC
Shin N [239]	2004	US		6-13	9	1203	605	598	min	day	TV	AA
Singh GK [106]	2003	US		10-17		46707	24072	22635	hour	day	TV	BC
Singh GK [106]	2003	US		10-17		46707	24072	22635	hour	day	TV	BC
Skoric MM [258]	2009	Singapore		8-12	10	333	180	153	hour		TV, GAMES	AA
Smith BJ [161]	2007	Fiji		11-16		443	200	245	hour	day	TV	BC
Spinks AB [124]	2007	Australia		5-12		518	282	236	min	week	SB, SCREEN	BC
Steffen LM [98]	2009	US		8-11		526	256	270	hour	day	TV	BC
Stettler N [168]	2004	Switzerland			8	872	410	462	hour	day	TV, GAMES	BC

Table 2 Summary of characteristics of included studies *(Continued)*

Sugiyama T [47]	2007	US		12-19	15.9	4508	2295	2213	hour	day	SB	MS
Sun Y [91]	2009	Japan		12-13	.	5753	2842	2911	hour	day	TV	BC
Taylor WC [158]	2002	US		6-15	11.1	509	231	278	kcal	day	SB	BC
te Velde SJ [129]	2007	International		9-14	11.4	12538	6256	6282	hour	day	TV, COMP	BC
Thompson AM [189]	2009	Canada	3, 7, 11			1777	795	982	min	day	TV	BC
Toschke AM [112]	2008	Germany		5-6		4884			hour	day	TV	BC
Toschke AM [121]	2007	Germany		5-6		5472			hour	day	TV	BC
Trang NHHD [146]	2009	Australia		11-16		2660	1332	1328	hour	day	SCREEN	BC
Tremblay MS [172]	2003	Canada		7-11		7261			hour	day	TV	BC
Treuth MS [27]	2009	US		11-12	11.9	1579	0	1579	hour	day	SB	BC
Tsai H [153]	2007	Taiwan		11-12		2218	1146	1072	hour	day	TV	BC
Tsai H [145]	2009	Taiwan		11-12		1329	615	672	hour	day	SB, TV	BC
Tucker LA [212]	1987	US			15.7	406	406	0	hour	day	TV	FIT, SE, PRO
Tucker LA [206]	1986	US			15.7	379	379	0	hour	day	TV	FIT
Tucker LA [214]	1996	US		9-10	9.8	262	162	100	hour	day	TV	FIT
Ussher MH [231]	1007	England		13-16		2623			hour	day	TV	PRO, AA
Utter J [171]	2003	US			14.9	4480	2240	2240	hour	day	SCREEN	BC
Utter J [152]	2007	New Zealand		5-14		1743	959	784	hour	day	TV, COMP	BC
Vader AM [97]	2009	US			11, 7	11594	6162	5432	hour	day	TV	BC
van Schie EG [261]	1997	Netherlands		10-14	11.5	346	171	175	hour	day	SCREEN	PRO, AA
van Zutphen M [159]	2007	Australia		4-12	8	1926	939	987	min	day	TV	BC
Vandewater EA [170]	2004	US		1-12	6	2831	1444	1387	hour	day	SB, SCREEN	BC
Vaughan C [198]	2007	Australia		11-18	14	443	189	254	hour	day	SCREEN	BC
Vicente-Rodriguez G [110]	2008	Spain		13-18.5		1960	1012	948	hour	day	TV, GAMES	BC
Violante R [137]	2005	Mexico		6-14		8624	258	4366	hour	day	TV	BC
Wake M [186]	2003	Australia		5-13	9.1	2862	1445	1417	hour	week	SCREEN	BC
Walberg HJ [251]	1984	US	2-6		13	2890	1445	1445	hour	day	TV	AA
Walberg HJ [253]	1982	US			17	2001	1031	970	hour	day	TV	AA
Waller CE [202]	2003	China		6-11	9	880			hour	week	TV	BC
Wang Y [120]	2007	US			11.9	498	218	280	hour	day	SCREEN	BC
Welch WW [248]	1986	Australia	3-4	9	9	1960					TV	AA
Wells JC [108]	2008	Brazil		10-12		4452	2193	2258	hour	day	TV	BC, MS
Whitt-Glover MC [24]	2009	US		6-19		749	351	398	min	day	SB	BC
Wiggins J [227]	1987	US	4-12			483	252	231	min	day	TV	SE, AA
Wolf AM [203]	1998	US		11-14		552	0	552	hour	day	TV	BC
Wong SL [100]	2009	Canada			15.5	25060	12806	12254	hour	day	SB, SCREEN	BC
Zabinski MF [132]	2007	US		11-15		878	425	453	hour	day	SB	BC

SB, sedentary behaviour; TV, television viewing; COMP, computer time; GAME, video game playing; SCREEN, composite measure of 2 or more screen activities (i.e. television viewing, computer time, or video game playing); BC, body composition; MS, measures of metabolic syndrome and/or cardiovascular disease (e.g. insulin resistance, blood pressure); SE, self-esteem; PRO, pro-social behaviour; AA, academic achievement.

sedentary time and weight status [24,85,137,183-204]. One study [131] reported an effect in boys but not girls and one showed an effect in girls but not boys [139]. One study showed that among boys, being underweight was associated with more screen time [111]. The level of evidence reporting on the relationship between sedentary behaviour and body composition was of moderate quality and was classified as Level 2 with a mean Downs and Black score of 20.6 (standard deviation: ± 1.9).

Fitness

Fifteen studies assessed the relationship between time spent engaging in sedentary behaviour and fitness (Table 4). Increased time spent being sedentary was associated with decreased scores for overall physical fitness, VO_2 max, cardiorespiratory fitness, and musculoskeletal fitness. An intervention reported that targeting decreased sedentary behaviour lead to increases in aerobic fitness [56]. This study (n = 13 boys and 26 girls, mean age =

Table 3 Summary table of results showing relation between sedentary behaviour and measures of body composition

Type of Study	Number of Studies	Number of participants	Narrative recommendation and main findings
RCT	8	1886	Reductions in sedentary behaviour are directly related to improved body composition.
Intervention	10	3547	TV watching and overweight/obesity were related in a dose-response manner (i.e. those who watched more TV were more likely to be overweight/obese).
Longitudinal	33	85753	TV watching and overweight/obesity were related in a dose-response manner (i.e. those who watched more TV were more likely to be overweight/obese).
Cross sectional	119	691759	> 2 hrs of sedentary behaviour related to increased risk of being overweight or obese.
Total of all studies	170	782884	Meta-analysis was performed on randomized controlled studies that looked at change in BMI. They found an effect of -0.89 kg/m^2 (95% CI of -1.67 to -0.11, p = 0.03) decrease in mean BMI in the intervention group. > 2 hrs of sedentary behaviour per day is associated with an increased risk for overweight/obesity. This risk increases in a dose-response manner. Each additional hour of TV viewing increased risk for obesity. > 2 hrs/day significantly increased risk for overweight/obesity. Mean Downs and Black score = 20.9 (± 1.9), Level 2 evidence.

10.5 years) showed that an intervention to decrease targeted sedentary behaviours (watching TV, playing computer games, talking on the telephone, or playing board games) led to increases in both physical activity and non-targeted sedentary behaviours. Longitudinal evidence was conflicting. One longitudinal study showed that > 2 hours per day of TV and computer use was associated with decreased musculoskeletal fitness [205]; while the second longitudinal study found no association between increased screen time and decreased fitness. Eight of 12 cross sectional studies showed that greater than 2 hours of screen time per day was associated with decreased VO_2max, lower cardiorespiratory fitness, and lower aerobic fitness [95,206-212]. Two studies showed weak relationships between television watching and fitness [197,213]. Two studies showed no consistent association between television viewing and aerobic and musculoskeletal fitness [184,214]. The level of evidence related to fitness was classified as Level 3 with a mean Downs and Black score of 20.9 (standard deviation: ± 2.1), indicating moderate quality of reporting.

Metabolic syndrome and risk for cardiovascular disease

Eleven studies assessed the relationship between time spent engaging in sedentary behaviour and risk factors for MS and CVD (Table 5). All of the studies reported that increased sedentary time was associated with increased risk for MS or CVD. However, the results of these studies should be viewed with caution as the proportion of children and youth who have measurable health risk factors for MS or CVD is quite low. Longitudinal studies found that those watching more than 2 hours of television per day had higher serum cholesterol levels [88] and were more likely to have high blood pressure [215] than their peers who watched less TV. Cross sectional studies reported that high levels of screen time and self-reported sedentary behaviour were associated with increased risk for high systolic and diastolic blood pressure [47,108,216,217], higher HbA1 c [218], fasting insulin [134,216], insulin resistance [48,219], and MS [220]. These risk factors increase in a dose response manner with increased screen time [216,220]. One cross sectional study reported a significant relationship between watching TV and increased cholesterol in adolescents, but not in younger children [185]. The level of evidence for MS and CVD risk factors was classified as Level 3 with a mean Downs and Black score of 21.7 (standard deviation: ± 2.1), indicating moderate quality of reporting.

Table 4 Summary table of results showing relation between sedentary behaviour and fitness

Type of Study	Number of Studies	Number of participants	Narrative recommendation and main findings
RCT	0		
Intervention	1	76	Reductions in sedentary behaviour lead to increased fitness.
Longitudinal	2	561	One study showed no association whereas one study showed higher musculoskeletal fitness in those watching < 2 hrs of TV per day.
Cross sectional	12	17227	> 2 hrs of screen time per day is associated with better VO_2max scores, better musculoskeletal and cardiorespiratory fitness scores.
Total of all studies	15	17864	Those watching less than 2 hours of TV a day showed higher results for fitness testing and more favourable bone health. Mean Downs and Black score = 20.6 (± 2.1), Level 3 evidence.

Table 5 Summary table of results showing relation between sedentary behaviour and markers for metabolic syndrome and cardiovascular disease

Type of Study	Number of Studies	Number of participants	Narrative recommendation and main findings
RCT	0		
Longitudinal	2	1675	> 2 hr of TV per day is associated with higher serum cholesterol levels. > 1.2 hrs of TV per day is associated with increased systolic blood pressure.
Cross sectional	9	17339	> 2 of screen time per day is associated with higher blood pressure and increased risk for metabolic syndrome.
Intervention	0		
Total of all studies	11	19014	Increased screen time is associated with increased risk for markers of metabolic syndrome and cardiovascular disease. Risk increases in a dose-response manner. Mean Downs and Black score = 21.7 (± 2.0), Level 3 evidence.

Self esteem

Fourteen studies assessed the relationship between time spent engaging in sedentary behaviour and self-esteem (Table 6). One RCT aimed to increase physical activity and decrease TV viewing [221], leading to a trend in improvements in self-esteem (P = 0.26) and concerns with body shape (p = 0.03). Intervention studies that targeted changes in sedentary behaviour produced inverse changes in physical self-worth and self-esteem [52,54]. Cross sectional studies showed that increased screen time was associated with higher depressive symptoms, low self-esteem, and decreased perceptions of self-worth [44,115,147,212, 221-223]. There was evidence for a dose-response relationship as each additional hour of screen time seemed to increase the risk for lower self-esteem [147]. Two studies [224,225] reported that increased TV viewing was associated with decreased self-esteem in boys but not girls, and increased aggression in girls but not boys. Two studies showed no significant relationship [226,227]. One study [228] showed a significant relationship between increased TV viewing and decreased self-esteem in adolescents but not in young children. The level of evidence for studies examining self-esteem was classified as Level 3 with a mean Downs and Black score of 21.0 (standard deviation: ± 2.4) indicating moderate quality of reporting.

Pro-social behaviour

Eighteen studies assessed the relationship between time spent engaging in sedentary behaviour and pro-social behaviour (Table 7). The one longitudinal study examining the relationship between sedentary behaviour and pro-social behaviour found that sustained TV exposure (i.e. ≥ 2 hours per day) was a significant risk factor for behavioural problems [229]. Cross sectional studies reported similar findings. Those who watched less TV were more emotionally stable, sensitive, imaginative, outgoing, self-controlled, intelligent, moralistic, college bound, and less likely to be aggressive or to engage in risky behaviour [42,115,230-235]. Two studies found a significant relationship between increased computer use and behaviour problems in boys [111,236] but not girls. One study showed that increased TV viewing was associated with aggression in girls but not boys [225]. The level of evidence for studies reporting on pro-social behaviour was classified as Level 3 with a mean Downs and Black score of 19.9 (standard deviation: ± 1.3) indicating moderate quality of reporting.

Academic achievement

Thirty five studies assessed the relation between time spent engaging in sedentary behaviour and academic achievement (Table 8). Academic achievement was measured in a variety of ways but included measures of I.Q., school grades, grade point average (GPA), performance on standardized tests, and self-report questionnaires (e.g. students rated their own level of academic achievement). The longitudinal studies included in this review found that children who watched higher amounts of TV had

Table 6 Summary table of results showing relation between sedentary behaviour and self-esteem

Type of Study	Number of Studies	Number of participants	Narrative recommendation and main findings
RCT	1	61	Girls who decreased sedentary behaviour had lower body dissatisfaction and showed a trend towards improved self-esteem.
Intervention	2	984	Decreases in sedentary behaviour lead to improved self worth and self-esteem.
Longitudinal	0		
Cross sectional	11	71068	Those with higher reported sedentary behaviour had poorer scores on self worth. This association seems to increase in a dose-response manner
Total of all studies	14	72113	Each additional hour of TV viewing was associated with decreases in self-worth and self-concept. Mean Downs and Black score = 21.0 (± 2.4), Level 3 evidence.

Table 7 Summary table of results showing relation between sedentary behaviour and pro-social behaviour

Type of Study	Number of Studies	Number of participants	Narrative recommendation and main findings
RCT	0		
Longitudinal	1	2707	Watching > 2 hrs of TV per day is a risk factor for social behaviour problems
Intervention	0		
Cross sectional	17	91934	Individuals watching > 3 hrs of TV per day are more likely to exhibit poor social behaviours and be more aggressive. Limited evidence to suggest this relationship is stronger in boys.
Total of all studies	18	94391	> 2 hrs of TV per day is associated with poor pro-social behaviour. Those watching less than 3 hrs of TV per day scored more positively in aspects of pro-social behaviour Mean Downs and Black score = 19.9 (± 1.34), Level 3 evidence.

greater difficulties with attention as teenagers [41], showed lower progression for reading level [237], and performed worse on cognitive tests [238] than those watching less than one hour of television per day. The majority of cross sectional studies (75%) reported that children and youth who watched higher levels of TV tended to spend less time doing homework, studying, and reading for leisure which may lead to a decrease in academic achievement [42,181,239-255]. This association increased in a dose response manner [181,244,248]. Ten of the cross sectional studies found no significant relationship [57,226,227,238,256-261]. One study [228] found that this relationship was significant in adolescents but not younger children. The evidence for academic achievement was classified as Level 3 with a mean Downs and Black score of 19.2 (standard deviation: ± 2.1) indicating moderate quality of reporting.

Quantitative data synthesis

Data for each of the outcomes were assessed to determine if they were sufficiently homogeneous to make meta-analysis appropriate. The only outcome for which data were consistently collected and reported and for which the characteristics of the studies were similar enough to undertake a meta-analysis was body composition. However, this was only for the RCTs; the longitudinal, cross sectional and intervention studies that examined body composition had too many inconsistencies to allow for a quantitative synthesis of results.

Change in mean BMI before and after the intervention (at the longest point of follow-up for each study) was used as the point estimate for the meta-analysis of the RCT data. Of the 8 RCTs, only 6 had data that could be used to calculate the change in BMI after the intervention [50,58,221,262-264] (the other two reported on prevalence of overweight and obesity) [57,265]. Of the remaining six studies, one [50] examined standardized estimates of BMI only and one [262] presented only median change in BMI and not a mean change. Study authors were contacted for missing information, but no additional data was made available and thus these studies were excluded from the meta-analysis. Meta-analysis of the 4 RCTs that remained revealed an overall significant effect of -0.89 kg/m^2 (95% CI of -1.67 to -0.11, p = 0.03) indicating an overall decrease in mean BMI associated with the interventions (Figure 2). The Chi square test for heterogeneity was not significant but the I^2 was 46% indicating that there was low to moderate heterogeneity in the data. The funnel plot showed no indication of publication bias (data not shown).

Meta-analyses were not undertaken for other outcomes or study designs because there was substantial heterogeneity in the units of measures and type of reporting of sedentary behaviour, as well as the specific measures of each outcome. For example, when reporting on the relation between time spent watching TV and overweight and obesity, one study may report the relation between

Table 8 Summary table of results showing relation between sedentary behaviour and academic achievement

Type of Study	Number of Studies	Number of participants	Narrative recommendation and main findings
RCT	0		
Longitudinal	3	3530	Watching > 1 hr of TV per day is associated with attention difficulties.
Intervention	0		
Cross sectional	32	157637	> 2 hrs of screen time per day resulted in lower academic achievement.
Intervention	0		
Total of all studies	35	161167	> 2 hrs of screen time per day is negatively associated with academic achievement. Dose-response relation between time spent playing video games, watching TV and using the computer (for non-academic purposes). > 3 hrs/day associated with poor school performance and lower I.Q. scores. Mean Downs and Black score = 19.1 (± 2.1), Level 3 evidence.

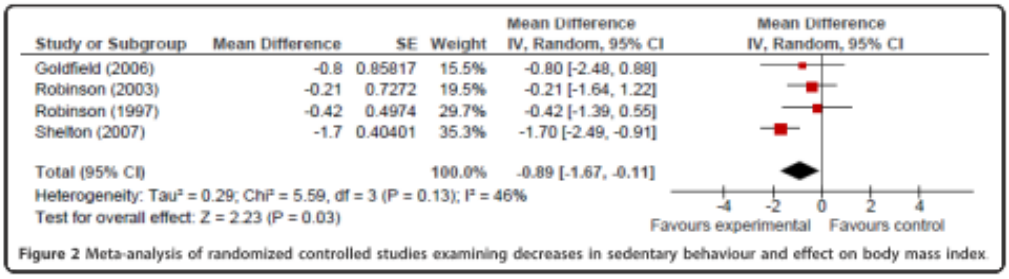

Figure 2 Meta-analysis of randomized controlled studies examining decreases in sedentary behaviour and effect on body mass index.

the frequency of TV watching and skin fold thickness, whereas another may examine the relation of daily volume of TV watching and BMI. Even for studies that examined the same outcome, for instance BMI, some would report the proportion overweight or obese, while others would report mean BMI. In addition, some studies reported on data for males or females only, while others reported only overall estimates and many were missing key information about participant characteristics or study design. As a result, we were unable to determine common point estimates and associated measures of errors for many of the studies. Due to the scope of the review, it was not feasible to contact every author for individual data to re-run the analyses. Developing reporting standards for primary studies examining the relationship between sedentary behaviour and health would help to ensure that appropriate data are available for future meta-analyses.

Discussion

Based on this systematic review of 232 studies, sedentary behaviour (assessed primarily through increased TV viewing) for more than 2 hours per day was associated with unfavourable body composition, decreased fitness, lowered scores for self-esteem and pro-social behaviour and decreased academic achievement in school-aged children and youth (5-17 years). This was true for all study designs, across all countries, using both direct and indirect measurements, and regardless of participant sample size. All studies examining risk factors for MS and CVD disease reported that increased sedentary time was associated with increased health risk; however, the included studies examined a wide range of risk factors, and thus there was insufficient evidence to draw conclusions on the relationship for metabolic risk as a whole.

High heterogeneity of the included studies limited meta-analysis to RCTs examining the relationship between television viewing and BMI. This revealed a trend to support the hypothesis that decreased time spent sedentary is associated with decreases in BMI. This result should be interpreted cautiously, given that it is only based on a small number of RCTs and that only half of the RCTs included in the review were included in the meta-analysis. Nonetheless, this meta-analysis of RCTs, which are considered to be the highest quality of research evidence, coupled with the qualitative syntheses of data from the other study designs, provides consistent evidence of the inverse relationship between sedentary behaviour and health outcomes, and that reducing sedentary behaviour can improve body composition. Furthermore, this finding was consistent with the results of observational studies and previous reviews [19-21,23,25].

Studies included in this review used primarily indirect measures (i.e. parent, teacher, and self-report questionnaires) to assess time spent engaging in sedentary behaviour. Those studies that did use direct (i.e. accelerometer) measures found that children and youth are spending a large proportion of their day (up to 9 hours) being sedentary [24,27,29,39-47,49,178]. Therefore, for some children and youth, a viable approach to improving health may be to work towards a reduction of at least *some* of their sedentary behaviours either through smaller, micro-interventions (e.g. interrupting prolonged sedentary time), or lager macro-interventions (e.g. population-based interventions and public health initiatives). Decreasing sedentary time is important for all children and youth, but it may be may be especially important to promote gradual decreases in the *most* sedentary group as a stepping stone to meeting sedentary behaviour guidelines [266].

Strengths and limitations

Strengths of this review included a comprehensive search strategy, *a-priori* inclusion and exclusion criteria and analyses, and inclusion of non-English language articles. We included direct and indirect measures of sedentary behaviour and focused on 6 diverse health indicators in children and youth. Although efforts were made to include grey literature (e.g. by contacting key informants and reviewing government documents), we did not include conference proceedings and other types of grey literature because it was impractical and unfeasible to sift through all unpublished work, and also because of limitations in

the quality of reporting in conference abstracts [267,268]. We do not anticipate that additional, unpublished work would change the results.

Our study has limitations, including the types of outcome measurements and analyses reported in the primary studies and primary study quality. The scope of this review was large and included a great deal of health indicators and measurement tools. A more detailed meta-analysis would have allowed us to estimate the overall effect sizes for each outcome. However, due to the heterogeneity of the data, it was impossible to complete such analysis. Furthermore, some studies had missing information on participant characteristics making it impossible to determine if basic demographics act as a confounder for the relationship between sedentary behaviour and health. Many studies also grouped their variables into tertiles, or groups that also took into account physical activity level. Although it was still possible to ascertain information regarding the association between level of sedentary behaviour and health indicators, it made it very difficult to compare the information across studies. Similarly, very few studies measured time spent being sedentary directly (i.e. with direct observation or accelerometry). Previous work [269,270] has shown significant differences between direct and indirect measures of physical activity; similar work needs to be completed with respect to sedentary behaviour to gain a better understanding of possible biases in previous studies. Indirect measurements of sedentary behaviour often lead to grouping for analyses. This may lead to bias in the results of the systematic review as many studies arbitrarily grouped their participants as ''high users'' if they watched more than 2 hours of television per day. This could perhaps be falsely leading us to conclude that 2 hours is the critical cut-point or threshold. Further work using direct (i.e. accelerometer) measures of sedentary behaviour and screen time as continuous variables will help to clarify if a cut-point of 2 hours is in fact biased.

The final important limitation of this review was the type of primary studies that were available for analysis. Studies with small sample sizes were excluded; however we do not believe that this had a significant impact upon the strength or direction of associations observed in this review. The majority of studies (78.4%) included in this review were cross sectional, observational studies, using indirect (i.e. parent-, teacher, or self-report) measurements of sedentary behaviour. Cross sectional data make it impossible to infer causation and results should therefore be interpreted with caution. However, it should be noted that due to ethical considerations, it may be impossible to conduct a RCT on the effects of long periods of sedentary behaviours in children and youth. Due to the large and diverse sample sizes available in population-based cross sectional research, and given that this information demonstrates similar trends as those seen in RCTs and intervention studies, we believe that the evidence presented in this review provides important insights into the relationship between sedentary behaviour and health outcomes in school-aged children and youth.

Future work

The purpose of this review was to provide an evidence base to inform clinical practice sedentary behaviour guidelines for children and youth [266]. Future work is needed to translate this information into clinical practice guidelines and disseminate this information to health care providers and the general public. While this review was limited to children and youth, similar work is needed to inform sedentary guidelines for young children aged 0-5 years, adults, and older adults.

As the accessibility and popularity of multiple forms of screen-based technology increases among the pediatric population, future work needs to continue to focus on media engagement. Specifically, with increasing popularity for hand-held, portable devices, 'sedentary multitasking' is becoming increasingly common. Children and youth are able to watch television, talk on the phone, and use the computer at the same time. This is a relatively new phenomenon and we are currently unaware what, if any, are the health effects associated with this high level of 'multi-screen' time. This is also true for the effect of advancements in technology and their associated health effects. For example, 'active video gaming' (e.g., Nintendo Wii™, Microsoft Kinect™, Sony's Playstation Move™) is advertised as an effective mode of physical activity. Although it is true that some games can require sufficient energy expenditure for health benefits [271], the socio-cognitive and physiological aspects of remaining indoors for long periods are unknown. Furthermore, children and youth can learn quite quickly how to use minimal gestures (e.g., using wrist movement only) to play the game thereby substantially reducing energy expenditure.

Finally, as described above, the vast majority of the current evidence has been based on self-report questionnaires focused on TV viewing and body composition. It is now clear that these two variables are related. Future work needs to move beyond this relationship and focus on other modes of sedentarism (e.g., prolonged sitting, passive transport) and other associated health indicators. To do this, objective measures of the time, type and context of sedentary pursuits will be needed in combination with robust and standardized measures of health indicators.

Conclusions

Physical inactivity and sedentary behaviour are pervasive and persistent public health challenges to overcome. This

review demonstrates that there is a need to advocate for increases in physical activity AND decreases in sedentary behaviour. It is believed that a multi-level, multi-sectoral approach is required for this to be successful [11]. Ultimately, resolving the problem of inactivity requires a sustained change in individual daily activity and sedentary patterns. From a public health perspective, a reduction in sedentary behaviour may be easier than increasing physical activity *per se* because there are fewer restrictions (i.e. no need to change clothing or use special equipment), and can be easily attained with minimal burden to a person's time or financial resources.

This systematic review summarizes the current evidence examining the relationship between sedentary behaviours and a series of health indicators. It was determined that increased sedentary time was associated with negative health outcomes in both boys and girls; this was true across all study designs with the majority of studies (85.8%) reporting similar relationships. The majority of current work has focused on television viewing and body composition and suggests that children and youth should watch less than 2 hours of TV per day during their discretionary time. Furthermore, children and youth should try to minimize the time they spend engaging in other sedentary pursuits throughout the day (e.g. playing video games, using the computer for non-school work or prolonged sitting). This work can be used to inform the development of evidence-based sedentary behaviour recommendations for children and youth.

Additional material

Additional file 1: Search strategy.

Additional file 2: Search strategy.

List of Abbreviations

BMI: Body Mass Index; CVD: Cardiovascular disease; DXA or DEXA: Dual-energy x-ray absorptiometry; MS: Metabolic syndrome; RCT: Randomized controlled trial; TV: Television.

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Authors' contributions

MT was responsible for the initiation, conceptualization and design of the systematic review; oversaw the data collection and extraction, analysis, and interpretation of data and was responsible for revising the manuscript critically for important intellectual content. AL was responsible for conducting the search, data collection and extraction, the risk of bias assessment, analysis and interpretation of data, and drafting the manuscript. MEK was responsible for the design and methodology of the review and revising the manuscript critically for important intellectual content. SCG was responsible for the design and methodology of the manuscript, conducting the meta-analysis, and revising the manuscript critically for important intellectual content. RC, GG, TS and RL were responsible for data collection and extraction, risk of bias assessment, and were responsible for revising the manuscript critically for important intellectual content. JM was responsible for the generation of systematic review search terms. MS was responsible for methodology of the review. All authors have read and approved the final manuscript. MT is the guarantor of the paper.

Competing interests

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Video Game Playing Is Independently Associated with Blood Pressure and Lipids in Overweight and Obese Adolescents

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Abstract

Objective: To examine the association between duration and type of screen time (TV, video games, computer time) and blood pressure (BP) and lipids in overweight and obese adolescents.

Design: This is a cross-sectional study of 282 overweight or obese adolescents aged 14–18 years (86 males, 196 females) assessed at baseline prior to beginning a lifestyle intervention study for weight control. Sedentary behaviours, defined as hours per day spent watching TV, playing video games, recreational computer use and total screen time were measured by self-report. We examined the associations between sedentary behaviours and BP and lipids using multiple linear regression.

Results: Seated video gaming was the only sedentary behaviour associated with elevated BP and lipids before and after adjustment for age, sex, pubertal stage, parental education, body mass index (BMI), caloric intake, percent intake in dietary fat, physical activity (PA) duration, and PA intensity. Specifically, video gaming remained positively associated with systolic BP (adjusted $r = 0.13$, $\beta = 1.1$, $p < 0.05$) and total cholesterol/HDL ratio (adjusted $r = 0.12$, $\beta = 0.14$, $p < 0.05$).

Conclusions: Playing video games was the only form of sedentary behaviour that was independently associated with increased BP and lipids. Our findings provide support for reducing time spent playing seated video games as a possible means to promote health and prevent the incidence of cardiovascular disease (CVD) risk factors in this high risk group of overweight and obese adolescents. Future research is needed to first replicate these findings and subsequently aim to elucidate the mechanisms linking seated video gaming and elevated BP and lipids in this high risk population.

Trial Registration: Clinicaltrials.gov NCT00195858

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Introduction

Most adolescents living in Western countries spend excessive amounts of time being sedentary, mainly in the form of screen time behaviours such as TV viewing, seated video gaming, and recreational computer use [1–3]. This is concerning because sedentary behaviours track throughout adolescence and into adulthood [4], and sedentariness via screen time in adulthood is associated with increased morbidity and mortality, making the relationship between sedentary behaviour and health indicators an important area of study [5]. Many of the studies conducted in the pediatric population have focused primarily on glucose-related measures with relatively little attention to traditional cardiovascular disease (CVD) risk factors such as high blood pressure (BP) and dyslipidemia. However, there is evidence that dyslipidemia and elevated BP are being increasingly observed in youth, especially overweight and obese youth [6,7], and that these CVD risk factors in adolescence predict the development of CVD and mortality in adulthood [8,9]. Moreover, animal research has shown that extended bouts of sedentary behaviour result in dramatic

reductions in lipoprotein lipase activity leading to reductions in high-density lipoprotein cholesterol (HDL-C) in rat skeletal muscle [10]. Similarly, excessive sedentary behaviour is associated with increases in low density lipoprotein cholesterol (LDL-C) and triglycerides in adult humans [11].

In the studies focusing on BP and lipids as an indicator of CVD risk in youth, screen time was independently associated with increased CVD risk after adjusting for moderate-to-vigorous intensity physical activity (PA) and other confounders in some studies [12–14] but not others [15–17]. However, the studies demonstrating null findings have design limitations including limited statistical power [16], TV viewing as the only index of sedentary behaviour [15], a limited number of measured CVD risk factors [15], and the assessment of screen time not including video gaming [17]. Including seated video gaming as part of screen time measurement is important because it is appealing to adolescents [18] and is associated with obesity in youth [19–21], possibly due to increased food intake [22,23]. Moreover, recent research found that different types of screen behaviour may have different and independent effects on chronic disease risk in youth [24,25] but again these studies failed to include video gaming as part of screen time. Thus, measurement of time spent playing video games in association with the full spectrum of BP and lipid measurements as indicators of CVD risk factors remains unclear.

The purpose of this study was to examine the independent relationships between the volume and types of sedentary screen time behaviours (i.e., TV viewing, seated video games, computer time) with systolic and diastolic BP and lipids in overweight and obese adolescents, controlling for a wide range of confounders.

Materials and Methods

Participants

The sample consisted of 282 adolescents who were either obese (≥95 body mass index (BMI) percentile for age and sex) or overweight (85th to 94th BMI percentile for age and sex) based on cut-off values from the Centers for Disease Control (CDC) growth charts [26], recruited as potential participants in a diet and exercise trial. Those participants who were overweight had to have at least one comorbid CVD or diabetes risk factor, including family history, to be included in the study. The sample was comprised of 86 males and 196 female aged 14 to 18 years, with a mean age of 15.5±1.4 years. All participants were Tanner stage IV or V with respect to pubertal development, and the mean BMI was 34.4±4.3 kg/m^2. The majority (70%) of the sample was Caucasian, 12% were African Canadian, 2% Asian, 3% Hispanic, 2% First Nations, 4% Arabic, 4% mixed race, and 3% categorized as other. Parents of participants were well educated, with 75% of mothers and 68% of fathers having completed some university or community college program. Participants were recruited by posters and advertisements in the Children's Hospital of Eastern Ontario's endocrine/obesity clinic, by radio and bus advertisements, and flyers in community physicians' offices. Bus advertisements were the most effective recruitment strategy, accounting for 48% of recruitment. Data for this analysis come from the Healthy Eating and Aerobic and Resistance Training in Youth (HEARTY) trial, a randomized controlled exercise intervention aimed at reducing adiposity in obese adolescents. The data reported herein represent baseline data collected before the intervention from 2005 to 2010. Of the 358 adolescents, 282 (79%) had complete data for analysis from the baseline data collection of the HEARTY trial. All participants completed the testing individually (i.e. one on one) with the research coordinator.

Ethics Statement

Informed written consent from each participant was obtained in accordance with the principles expressed in the Declaration of Helsinki guidelines for human subjects. In the case of minors, written informed consent was also obtained by parents or legal guardians. This study was reviewed and approved by the Research Ethics Boards of the Children's Hospital of Eastern Ontario (CHEO) and The Ottawa Hospital.

Screen Time Behaviour and Physical Activity

Time (hours per day) spent watching TV, playing seated (inactive) video games (excluding computer games), and recreational computer time (excluding school work) was assessed by self-report questionnaire. Total screen time was calculated by aggregating the three types of screen behaviours. Self-reported PA duration was calculated based on the question "On average, how long do you participate in some sort of physical activity each day – with physical activity being cumulative not consecutive." Likert-type rating scales included 6 response ratings, whereby 1 = less than 5 minutes, 2 = 5 to 15 minutes, 3 = 15 to 30 minutes, 4 = 30 to 45 minutes, 5 = 45 to 60 minutes, and 6 = greater than 60 minutes. Intensity of PA was assessed by the question "on average, how would you describe the intensity of most of your physical activity?" Response ratings included 1 = light, 2 = moderate, and 3 = vigorous. Both PA duration and PA intensity were used as covariates in our statistical analyses.

Lipids

Overnight-fasting blood samples of approximately 20 mL of venous blood were taken in the morning, from a forearm or antecubital vein and were transported directly to the laboratory at the Ottawa Hospital for analysis. The lipid profile measurements included triglycerides (mmol/L), total cholesterol (mmol/L), and HDL-C (mmol/L) which were measured by using enzymatic methods on a Beckman-Coulter LX20 analyzer (Beckman instruments, Brea, California), while LDL-C concentrations were calculated by using the Friedewald equation [27]. Total cholesterol/HDL-C ratio was derived from measured values.

Blood Pressure

BP was measured manually using a mercury sphygmomanometer on the left arm after 4 minutes of rest, with subjects sitting with their back supported. Three BP measurements were taken at 1-minute intervals; the mean of the final two measures of BP was used for the analysis.

Covariates

Weight and height were measured using a Health O Meter manual scale (Health O Meter, Continental Scale Corp, Bridgeview, ILL) while participants wore light clothing, without shoes, using standard techniques. BMI was calculated as weight (kg)/height in metres^2. Children were classified as overweight or obese according to age and sex specific cut-points as described above in the participants section [26]. Sexual maturity was assessed using the 5 stage scale for breast development in females and testicular volume in males, according to Tanner [28].

Participants completed questionnaires on the same date as the physical measurements were taken. They were asked to report their age, sex, race and whether their mother and father completed elementary school, high school, community college or university. Highest level of maternal and paternal education was assessed as a proxy for socio-economic status.

Participant dietary intake, including total kilocalories consumed, kilocalories (and percent kilocalories) from fat, protein, and carbohydrates were assessed using 3-day, 24 hour food diaries under the supervision of a registered dietitian. The research coordinator provided instruction on food recording prior to evaluation of energy intake, and participants were provided with tools to aid in measurement. Food records were completed for three days, two weekdays and one weekend day, and the mean intake was averaged across the three days using food composition analysis software (The Food processor SQL 2006, ESHA Research, Salem, OR). A registered dietitian met with each participant shortly after completing the 3-day food records to clarify recording and gain more accurate measurement of energy intake.

Statistical Analyses

The distributions for BP and lipid outcomes were examined to determine if they were normally distributed. All distributions were normally distributed except for triglycerides, which was positively skewed but normalized when it was logarithmically transformed, and this transformed triglycerides variable was used in the analyses. Sex differences were examined by independent t-tests and chi-squares for continuous and categorical variables, respectively (Table 1). The relationships between the covariates and the individual BP and lipid outcomes were assessed using Pearson Correlations for continuous variables and Spearman Rho (non-parametric) correlations for categorical variables (Table 2). The independent associations between the types of sedentary behaviour (seated video games, TV, recreational computer time, total screen time) and PA duration and PA intensity with BP and lipid measures (dependent variables) were tested by multiple linear regression, adjusting for sex, age, highest level of maternal and paternal education, sexual maturity, BMI, total caloric intake, percent of calories from dietary fat, PA intensity and PA duration (Table 3). Each regression for the associations between sedentary behaviours and BP and lipid outcomes were also assessed for a sex×sedentary behaviour interaction. Regarding the association between PA duration and PA intensity and BP and lipid outcomes, total screen time was used as the sedentary behaviour in all regressions because it represents an aggregate measure of the time spent in all screen time behaviours. For regressions involving PA duration and PA intensity, sex×PA interactions were also assessed for each BP and lipid outcome. Statistics were analyzed with SPSS for Windows (Version 18) and a p-value≤0.05 denoted statistical significance.

Results

The descriptive characteristics of the sample are shown in Table 1. On average, males had a higher BMI, reported more time spent playing seated video games, greater total screen time, and greater caloric intake compared to females. Males were significantly more likely to be obese than females (100% vs 88%, $p<0.001$). Males also had higher systolic BP, triglycerides, and total cholesterol/HDL ratio, while females were more sexually mature and had higher HDL-C. There were no sex differences on self-reported PA.

Unadjusted correlations are shown in Table 2. None of the sedentary behaviours was significantly correlated with BP or lipids except for time spent playing video games, which was positively associated with systolic BP ($r=0.20$, $p<0.001$), triglycerides ($r=0.10$, $p<0.001$), total cholesterol/HDL-C ratio ($r=0.19$, $p<0.001$), and negatively associated with HDL-C ($r=-0.19$, $p<0.001$). Although PA duration was not associated with any BP or lipid measures, PA intensity was inversely correlated with systolic BP ($r=-0.12$, $p<0.05$) and total cholesterol/HDL-C ratio ($r=-0.11$, $p<0.05$). There were no significant associations between total caloric intake (kcals) or percent fat intake and BP and lipids, as shown in Table 2. Similarly, there was no significant relationship between percent protein or carbohydrate intake and BP and lipids (data not shown). BMI correlated with BP and lipids more strongly than any other of the covariates.

Table 1. Descriptive characteristics of the sample.

	Males (N = 86)	Females (N = 197)
Age (years)	15.4±1.3	15.6±1.4
Height (CM)***	174.5±7.6	165.4±5.9
Weight (kg)***	108.3±16.1	93.1±14.7
BMI (kg/m^2)**	35.5±4.0	33.9±4.4
Tanner (pubertal) Stage***	4.5±0.5	4.8±0.4
TV viewing (hours/day)	2.7±1.7	2.8±2.1
Computer time (hours/day)	2.5±1.9	2.2±2.0
Video games (hours/day)***	1.5±1.9	0.2±0.6
Screen time (hours/day)***	6.7±2.9	5.2±2.8
Intake in Dietary Fat (%)	34.4±6.4	34.0±5.6
Total caloric intake (kcals)**	2322±601	2086±590
Physical activity score	3.5±1.7	3.4±1.7
PA intensity-light (%)	68.6	64.3
PA intensity-moderate (%)	29.1	33.2
PA intensity-vigorous (%)	2.3	2.5
Systolic BP (mm Hg)***	119.0±10.9	111.3±9.1
Diastolic BP (mm Hg)	75.6±6.8	74.4±7.3
Triglycerides (mmol/L)*	1.4±0.6	1.2±0.6
HDL-C (mmol/L)***	1.0±0.2	1.1±0.3
LDL-C (mmol/L)	2.6±0.7	2.6±0.7
Total cholesterol (mmol/L)	4.2±0.8	4.3±0.8
Cholesterol/HDL-C Ratio***	4.4±1.2	3.9±1.0

Data are presented as means ± standard deviations except for PA intensity which are in percentages: PA = Physical activity score of 1 = less than 5 minutes, 2 = 5 to 15 minutes, 3 = 15 to 30 minutes, 4 = 30 to 45 minutes, 5 = 45 to 60 minutes, and 6 = Greater than 60 minutes. Sex differences determined by independent t-tests for continuous data and chi-square for categorical data;
*p<.05,
**p<.01,
***p<.001.
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Table 3 shows the independent associations between the types of sedentary behaviour and intensity of PA and BP and lipids after adjustment for potential confounders. The adjusted sex×sedentary behaviour interaction was not significant for any of the health outcomes. Similarly, the sex×PA duration and sex×PA intensity interactions were not significant (data not shown). However, after adjusting for age, sex, parental education, BMI, sexual maturity, total caloric intake, percent of energy intake derived from dietary fat, duration of PA, and intensity of PA, the positive associations between video gaming and systolic BP (adjusted $r=0.13$, $\beta=1.1$, $p<0.05$) and total cholesterol/HDL ratio (adjusted $r=0.12$, $\beta=0.10$, $p<0.05$) remained significant. The relationship between video gaming and triglycerides reached borderline significance (adjusted $r=0.11$, $\beta=0.04$, $p=.07$). No other types of sedentary behaviour, including total screen time, were associated with BP or lipid measures after adjusting for covariates. After adjustment, PA

Table 2. Correlations between Various Sedentary Behaviours, Physical Activity and Blood Pressure and Lipids.

	TV	CT	VG	ST	BMI	Age	Sex	PE	ME	TS	%Fat	Cal	PA-I	PA-D
Systolic BP	0.06	−0.08	0.20**	0.07	0.25**	0.15**	−0.33**	0.06	0.01	0.01	0.10	0.10	0.12	0.03
Diastolic BP	0.00	−0.04	0.03	−0.02	0.25**	0.15**	−0.09	0.05	−0.01	0.01	0.08	0.06	−0.02	0.01
Total Cholesterol	0.03	−0.01	0.00	0.01	−0.05	0.07	0.04	−0.04	0.09	0.01	0.03	0.00	−0.05	−0.04
HDL-C	−0.03	0.08	−0.19**	−0.05	−0.25**	0.14**	0.27**	0.04	0.08	0.19**	−0.04	−0.08	0.10	0.04
Triglycerides	0.08	−0.09	0.10*	0.03	−0.23**	0.10*	−0.12*	−0.04	−0.01	−0.12*	0.07	0.04	−0.03	−0.05
LDL-C	0.02	−0.01	0.03	0.02	0.06	0.08	−0.01	−0.07	−0.01	−0.12*	0.03	0.02	−0.09	−0.04
Total Chol/HDL-C	0.04	−0.04	0.19**	0.08	0.25**	−0.07	−0.23**	−0.09	−0.01	0.15**	0.06	0.07	−0.11*	0.09

TV = television viewing in hours/day; CT = recreational computer time in hours/day; VG = video games in hours/day; ST = screen time in hours/day; Sex: 0 = male, 1 = females; ME = Maternal education; PE = paternal education; TS = tanner pubertal stage score; %Fat = %intake in dietary fat; Cal = total caloric intake(kcals); PA-I; Physical Activity Intensity, 1 = light, 2 = moderate, 3 = vigorous; PA-D = Physical Activity Duration score (hours/day). Pearson correlations used to assess continuous variables and Spearman Rho correlations used for categorical variables.

*p<.05;

**p<.001.

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intensity was inversely associated with systolic BP (adjusted $r = -0.13$, $\beta = -2.3$, $p<0.05$), LDL-C (adjusted $r = -0.12$, $\beta = -0.15$, $p<0.05$), and total Cholesterol/HDL ratio (adjusted $r = -0.14$, $\beta = -0.28$, $p<0.05$), and positively associated with HDL-C (adjusted $r = 0.11$, $\beta = 0.05$, $p<0.05$). After adjustment, PA duration was only inversely associated with total cholesterol/HDL-C ratio (adjusted $r = -0.11$, $\beta = -0.08$, $p<0.05$).

Discussion

This study examined the independent association between duration of screen time and time spent in types of sedentary screen behaviours with BP and lipids in overweight and obese adolescents. Interestingly, total screen time was not associated with BP or lipids before or after adjusting for confounders. However, to our knowledge, we are the first to show that time spent playing video games was the only type of sedentary behaviour associated with increased BP and lipids before and after adjusting for multiple confounders in a large sample of overweight and obese adolescents. Although males spent significantly more time playing video games than females, the sex×video game interaction was not significant, suggesting that the relationship between video games and BP and lipid outcomes did not differ by sex.

Only a few studies have examined the relationship between types of screen time behaviours and chronic disease risk factors in youth. In a large, nationally representative sample of over 2500 children and adolescents from the 2003/4 and 2005/6 National Health and Nutrition Examination Survey (NHANES), Carson and Janssen [24] found that time spent watching TV was predictive of a higher score of an aggregated or clustered measure of cardio-metabolic risk, but recreational computer time was not. Similarly, Martinez-Gomez et al. [25] found that TV viewing but not computer time was associated with increased BP in pre-pubertal children. However, neither study assessed time spent playing seated video games, so it is uncertain how this form of sedentary behaviour would have predicted chronic disease risk compared to other screen behaviours. In contrast to these findings, neither TV viewing nor recreational computer time was associated with BP or lipids in the current study, whether considering unadjusted or adjusted relationships. These discrepant findings are somewhat surprising since many studies have documented

Table 3. Independent Associations of Various Sedentary Behaviours, Physical Activity Intensity and Blood Pressure and Lipids.

	TV	CT	VG	ST	PA- Intensity
Systolic BP	0.48 (−0.72 to 1.7)	−0.77 (−1.8 to 0.28)	1.1 (0.11 to 2.2)*	0.34 (−0.36 to 1.0)	−2.3 (−0.3 to −4.3)*
Diastolic BP	−0.09 (−0.21 to 0.03)	−0.02 (−0.12 to 0.09)	−0.01 (−0.12 to 0.09)	−0.04 (−0.12 to 0.03)	−0.58 (−2.1 to 0.96)
Total Cholesterol	−0.01 (−0.11 to 0.09)	−0.07 (−0.02 to 0.16)	0.03 (−0.06 to 0.17)	0.04 (−0.02 to 0.10)	−0.12 (−0.23 to 0.05)
HDL-C	0.00 (−0.03 to 0.03)	0.01 (−0.02 to 0.04)	−0.02 (−0.04 to 0.01)	0.00 (−0.02 to 0.02)	0.05 (0.00 to 1.0)*
Triglycerides	0.05 (−0.03 to 0.13)	−0.04 (−0.11 to 0.03)	0.04 (−0.03 to 0.11)	0.02 (−0.03 to 0.07)	−0.05 (−0.19 to 0.08)
LDL-C	0.03 (−0.12 to 0.05)	0.07 (−0.01 to 0.15)	0.02 (−0.05 to 0.10)	0.03 (−0.02 to 0.08)	−0.15 (−0.29 to −0.01)*
Total Chol/HDL-C	−0.03 (−0.16 to 0.11)	0.05 (−0.06 to 0.17)	0.10 (0.00 to 0.21)*	0.07 (−0.01 to 0.14)	−0.28 (−0.50 to −0.06)*

Data are presented as unstandardized βeta –Coefficients (95% confidence interval). βeta –Coefficients assess the relationship between the sedentary and physical activity variables (IVs) with the BP and lipid outcomes (DVs) adjusting for the covariates, thus the higher the beta weight, the stronger the independent relationship between the IVs and DVs; TV = television viewing in hours/day; CT = recreational computer time in hours/day; VG = video gameplaying in hours/day; ST = screen time in hours/day; PA-Intensity = Physical Activity Intensity score, with 1- light, 2 = moderate, 3 = vigorous.

*p<.05; Each linear regression assessing sedentary behaviours and PA intensity (primary IV's) on BP and lipids controlled for sex, mother and father's highest level of education, sexual maturity, age, BMI, total caloric intake (kcals) and % calories in dietary fat, physical activity duration and intensity and a sex×sedentary behaviour interaction. For the case of PA intensity, screen time was chosen as the sedentary behaviour because it is an aggregate measure of types of sedentary behaviour, and sex×PA intensity interaction was included in the regression equation. Results of regressions for PA duration and BP and lipids are presented in text of results section.

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independent relationships between TV viewing and biochemical markers of metabolic and CVD risk in children and adolescents [12–14,24], though not all have found these relationships [15]. The discrepant findings are unlikely to be due to differences in absolute time spent in either type of sedentary behaviour or variation since the mean values for time spent in TV and seated video gaming in the current study was similar, as was the variability. However, our sample was comprised of overweight and obese adolescents rather than a nationally representative sample [24], so it is possible these differences in sample characteristics could explain, in part, the discrepant findings.

Given the novelty of the findings, it is difficult to speculate what mechanisms may link video game use to high BP and more adverse lipid profiles; however, a few possible explanations are offered. Similar to TV viewing, observational studies showed an association between video game playing and obesity in youth [19–21]. Well-controlled crossover laboratory research indicates that video game playing was associated with an increase in spontaneous food intake of energy dense snack foods compared to resting conditions [22,23] which may have an adverse impact on obesity, BP and lipid profiles. Even though various dietary measures (percent of calories from fat and total caloric intake) were adjusted for in this study, residual confounding may have still been present, although unlikely given we found no association between food intake and the BP and lipid measures. In addition, laboratory studies have shown that seated video game playing acutely causes increased heart rate, elevated systolic and diastolic BP, increased sympathetic tone and mental workload compared to rest [22], perhaps due to the excitement, stress and concentration required for effective gaming. Given that these associations between seated video gaming and BP occurred in laboratory settings, it is possible that these effects become exacerbated and more chronic when video games are played frequently over time as observed in our study. It is also possible that the self-reporting of gaming was more accurate than other screen behaviours since gaming may be played in discrete bouts that are more distinct and memorable, whereas it may be more difficult to accurately quantify time spent watching TV or computer use because they may be more susceptible to periodic interruptions.

Similar to previous studies [15,16,24,29], PA intensity in this study, primarily that performed at moderate-to-vigorous-intensity, was associated with lower BP and more favourable lipid profile before and after adjustment for confounders, including sedentary behaviour. However, duration of PA, defined as time spent in PA per day, was not associated with BP or lipids before adjustment, and was only associated with total cholesterol/HDL-C ratio after adjustment, indicating that PA intensity rather than duration of PA may be more closely related to a lower BP and more favourable lipid profile in this population. In addition, these findings highlight the notion that sedentary behaviour and PA are distinct constructs that may have different mechanisms in how they relate to health outcomes [30].

Limitations and Strengths

This study has several strengths and limitations. We utilized a sample of overweight and obese adolescents volunteering for exercise intervention, thus it is uncertain whether our findings can be generalized to overweight and obese adolescents in the community. In addition, time spent in screen time behaviours and PA duration and intensity were measured by self-report which may introduce inaccuracies and bias in youth, whereby PA is generally over reported and sedentary behaviour is under reported [31], and it is possible that objective measures of these behaviours may have resulted in a different pattern of results. Another limitation is the cross-sectional design, which limits the ability to make causal inferences about the relationships observed. Also, males spent more time video gaming than females, but males only comprised about 30% of the sample, thus it is possible that sex differences between seated video games and BP and lipid profiles may have been detected if the sample was more balanced on sex, thus future research is needed to test the veracity of this hypothesis. Regarding the imbalance in sex in the current study, it is possible that males perceive obesity to be a problem warranting intervention only at greater degrees of obesity, perhaps explaining, in part, that males tended to have more adverse BP and lipid profiles than females.

Strengths of this study include an assessment of the three primary forms of sedentary screen time behaviours (seated video games, TV and recreational computer time), whereas previous studies in the pediatric population examining the relationship between types of sedentary behaviour and risk factors of chronic disease only included TV and computer time [17,24,25]. Our study highlights the importance of measuring seated video gaming, which large surveys show has mass appeal to teenagers [18], especially males, and our data also reflect the popularity of video games given time spent in gaming was comparable to TV viewing. Our study is also the first to examine the associations between sedentary screen time behaviours and BP and lipid profiles in an overweight and obese adolescent sample, who is at increased risk of CVD and premature mortality in adulthood compared to their normal weight peers [8,9]. Additionally, the present study included the broadest spectrum of BP and lipid measures as a proxy for assessing CVD risk factors compared to other studies [12–16]. Assessing the full spectrum of BP and lipids is important because these CVD risk factors in adolescence track into adulthood [7], and heart disease and stroke are still leading causes of morbidity and premature mortality [32]. Lastly, the current study included a comprehensive set of covariates that statistically controlled for several confounding variables, strengthening the internal validity of the findings.

Conclusions

To the best of our knowledge, the present study is the first to demonstrate that seated video gaming is associated with increased BP and lipids in a sample of overweight and obese adolescents, after controlling for adiposity, caloric intake, dietary fat intake, PA duration and intensity and several other important confounders. Our results provide further support for the public health guidelines recommending that children and youth limit their sedentary behaviour, especially screen time behaviours [33,34]. Our findings provide support for reducing time spent playing seated video games as a possible means to promote health and prevent CVD in this high risk group of overweight and obese adolescents. Future research is needed to first replicate these novel findings and subsequently aim to elucidate the mechanisms linking seated video gaming and elevated BP and lipids in a high risk sample of overweight and obese youth.

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Author Contributions

Conceived and designed the experiments: GSG GPK SH JM DP RG TJS MST RJS. Performed the experiments: PP ASA. Analyzed the data: GSG. Wrote the paper: GSG GPK SH PP ASA TJS MST JM DP RG RJS.

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The association between accelerometer-measured patterns of sedentary time and health risk in children and youth: results from the Canadian Health Measures Survey

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Abstract

Background: Self-reported screen time is associated with elevated health risk in children and youth; however, research examining the relationship between accelerometer-measured sedentary time and health risk has reported mixed findings. The purpose of this study was to examine the association between accelerometer-measured patterns of sedentary time and health risk in children and youth.

Methods: The results are based on 1,608 children and youth aged 6 to 19 years from the Canadian Health Measures Survey (2007–2009). Sedentary time was measured using the Actical accelerometer. Breaks in sedentary time and prolonged bouts of sedentary time lasting 20 to 120 minutes were derived for all days, weekend days and during the after-school period (i.e., after 3 pm on weekdays). Regression analyses were used to examine the association between patterns of sedentary time and body mass index (BMI), waist circumference, blood pressure and non-HDL cholesterol.

Results: Boys accumulated more sedentary time on weekdays after 3 pm and had a higher number of breaks in sedentary time compared to girls. Overweight/obese boys (aged 6–19 years) accumulated more sedentary time after 3 pm on weekdays (282 vs. 259 min, $p < .05$) and as prolonged bouts lasting at least 80 minutes (171 vs. 133 min, $p < .05$) compared to boys who were neither overweight nor obese. Prolonged bouts of sedentary time lasting at least 80 minutes accumulated after 3 pm on weekdays were positively associated with BMI and waist circumference in boys aged 11–14 years ($p < .006$). Each additional 60 min of sedentary time after 3 pm on weekdays was associated with a 1.4 $kg{\cdot}m^{-2}$ higher BMI and a 3.4 cm higher waist circumference in 11–14 year old boys. No sedentary pattern variables differed between girls who were not overweight or obese and those who were overweight/obese and none of the sedentary pattern variables were associated with any health markers in girls.

Conclusions: The findings confirm results of other studies that reported accelerometer-measured sedentary time was not associated with health risk in children and youth. Even when the pattern and timing of sedentary time was examined relative to health markers, few associations emerged and were limited to boys aged 11–14 years.

Keywords: Behaviour, Breaks, Bouts, Physical activity, Pediatric

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Background

The association between sedentary behaviour and health risk in children appears to be influenced by how the sedentary behaviour is measured, defined and categorized. Several studies have reported significant associations between self-reported screen time and increased risk of obesity and cardio-metabolic disease risk in children [1-4]. However, screen time provides a limited perspective on total sedentary time because it is only a sub-component of a behaviour that is defined as encompassing "any waking behaviour characterized by an energy expenditure ≤1.5 METs while in a sitting or reclining position" [5]. Further, self-reported data relating to lifestyle habits may be limited by bias and recall difficulties [6,7].

Accelerometers are now commonly used to objectively measure total sedentary time, and have the capacity to also derive the pattern and timing in which it is accumulated. In contrast to self-report, associations between accelerometer-measured sedentary time and health risk in children have been mixed with some studies reporting significant associations [8-10] and others not [1,11-16]. Studies that collected both questionnaire and accelerometer data on sedentary behaviour/time found an association between self- or parent-reported screen time and health risk but no association between accelerometer-measured sedentary time and health risk [1,14,15]. The only studies reporting significant associations between accelerometer-measured sedentary time and health risk did not adjust for moderate-to-vigorous physical activity (MVPA) [8-10] or reported that the significant associations existed in unadjusted models but were attenuated when MVPA was controlled for [13,16]. Some engagement in sedentary behaviour is inevitable in the day (e.g., eating, relaxing, homework, school etc.); however, it is presently unknown how much sedentary time is too much. Inevitably, there is variation between people in the length of time they engage in sedentary behaviour bouts (i.e., prolonged periods of sedentary time) and how often these bouts are interrupted by activity. Research in adults suggests that the pattern of accumulation of sedentary time is important to consider in relation to health risk.

The inconsistent findings between accelerometer-measured sedentary time and health risk among children and youth have led to the examination of more sophisticated sedentary time variables. For example, it has been proposed that the pattern in which sedentary time is accumulated may provide insight beyond what has been observed to-date using the total volume of sedentary time [17]. Others have attempted this approach in adults and children; however, the sedentary pattern variables have been limited to breaks or interruptions in sedentary time [17] and engagement in prolonged bouts of sedentary time lasting up to 30 minutes [1]. Further, studies examining how the pattern of sedentary time relates to health risk in children have not considered the importance of periods of discretionary free time separate from the whole day in children [18-20]. The present study sought to build upon this work by extending the length of the prolonged bouts up to 2 hours and by examining these variables during periods when children and youth typically have free time. In other words, this study sought to identify novel sedentary pattern variables that were more representative of how children and youth typically engage in sedentary behaviour.

The purpose of this study was to examine the association between accelerometer-measured patterns of sedentary time and health risk in children and youth. Specifically, this study examines whether breaks in sedentary time and sedentary time accumulated as prolonged bouts during periods of discretionary free time in children (i.e., after-school and weekends) have stronger associations with health risk in children when compared to average daily sedentary time across the week. We hypothesize that sedentary time accumulated during periods of discretionary free time will better discriminate between children engaging in healthy and unhealthy levels of sedentary behaviour when compared to simply examining overall sedentary time.

Methods

Data source

The Canadian Health Measures Survey (CHMS) collected data from a nationally representative sample of the population aged 6 to 79 years living in private households at the time of the survey. Data were collected at 15 sites across Canada from March 2007 through February 2009. Ethics approval was obtained from Health Canada's Research Ethics Board [21]. For children aged 6–13 years, written informed consent was obtained from a parent or legal guardian, in addition to written informed assent from the child; youth aged ≥14 years provided independent consent. Of the households selected, 69.6% agreed to participate. Of that group, 88.5% of the selected 6–19 year olds completed a questionnaire and 86.9% of this group participated in the mobile examination centre component. Of the children and youth who agreed to wear the accelerometer and returned the device, 87.4% had at least one valid day of data, and 76.3% had at least four valid days. These multiple stages of response can be multiplied together (69.6% × 88.5% × 86.9% × 76.3%) to provide an overall response rate of 40.8%. Adjustments were made at each stage to manage any potential non-response bias. The data were then weighted to be representative of the Canadian population. More extensive details of the CHMS [22] and direct measurement of physical activity in the CHMS [23,24] are available elsewhere.

Study procedures

Upon completion of the mobile examination centre visit, ambulatory respondents were asked to wear an Actical accelerometer (Phillips – Respironics, Oregon, USA) over their right hip on an elasticized belt during their waking hours for seven consecutive days, except when the device could get wet. The Actical measures and records time-stamped acceleration in all directions, providing an index of physical activity intensity. The Actical has been validated to measure physical activity in children [25] and cut-points for sedentary intensity have been proposed for children [26]. The accelerometers were initialized to collect data in 60-sec epochs.

Accelerometer data reduction

Participants aged 6 to 19 years with four or more valid days [24], one of which was a weekend day, were included in this analysis (Table 1). A valid day was defined as having 10 or more hours of wear time [24]. Wear time was determined by subtracting non-wear time from 24 hours. Non-wear time was defined as at least 60 consecutive minutes of zero counts, with allowance for two minutes of counts between zero and 100 [24]. For each minute, the level of movement intensity was based on cut-points corresponding to intensity level: sedentary = < 100 counts per minute (cpm) [26]; MVPA = ≥ 1,500 cpm [25]. Minutes of MVPA and sedentary time were summed for each day for each participant.

Sedentary time variables

Sedentary time was calculated for all days, weekdays and weekend days. The total number of breaks in sedentary time was summed for each valid day and then averaged across the week, weekdays and weekend days. A break was considered as an interruption in sedentary time (lasting a minimum of one minute) in which there was a transition in accelerometer count from <100 cpm to ≥ 100 cpm.

To be defined as a prolonged sedentary bout, there had to be ≥80% of minutes below the 100 cpm cut-point (e.g., 16 out of 20 minutes or 32 out of 40 minutes) [1]. The bout stopped when <80% was below the 100 cpm cut-point or when there were ≥3 consecutive minutes ≥100 cpm or any observations ≥1500 cpm (cut-point for moderate intensity). Sedentary bouts lasting at least 20, 40, 60, 80, 100, 120 minutes were derived using this approach. Multiple lengths of sedentary bouts were derived to reflect a range of different sedentary behaviours such as watching a television show (30 minutes), watching a movie (1.5-2 hours), or playing video games (anywhere between 20 minutes and 2 hours). The choice of 80% as the criteria for sedentary minutes within a bout was purposeful to mimic real-world situations where largely sedentary pursuits (e.g., watching TV, doing homework) are often occasionally interrupted with light activity (e.g., to go to washroom, answer the phone, get a snack etc.).

Body mass index and waist circumference

Height was measured to the nearest 0.1 cm using a ProScale M150 digital stadiometer (Accurate Technology Inc., Fletcher, USA) and weight was measured to the nearest 0.1 kg with a Mettler Toledo VLC with Panther Plus terminal scale (Mettler Toledo Canada, Mississauga, Canada). BMI was calculated as weight (kg) divided by height squared (m^2). Children were categorized as not overweight/obese (which includes underweight and healthy weight) or overweight/obese according to age- and sex-specific cut-points [27]. Waist circumference was measured with a stretch-resistant anthropometric tape at the end of a normal expiration to the nearest 0.1 cm at the mid-point between the last rib and the top of the iliac crest [28].

Blood pressure

Systolic and diastolic blood pressure were measured with the BpTRU™ BP-300 device (BpTRU Medical Devices Ltd., Coquitlam, British Columbia); an automated and validated [29,30] electronic monitor that uses an upper arm cuff. Six measurements were taken at 1-min intervals with the last 5 measurements used to calculate

Table 1 Descriptive characteristics of the sample (mean ± standard deviation)

	Boys			Girls		
	6 to 10 years	11 to 14 years	15 to 19 years	6 to 10 years	11 to 14 years	15 to 19 years
Total sample (n)	369	256	184	340	248	211
Age (years)	8.2 ± 1.4	12.5 ± 1.0	17.0 ± 1.5	8.1 ± 1.3	12.3 ± 1.1	16.9 ± 0.1
Height (cm)	133.9 ± 10.4	158.9 ± 11.1	175.6 ± 7.6	131.6 ± 10.3	156.9 ± 7.8	166.2 ± 6.7
Weight (kg)	32.5 ± 9.4	52.1 ± 14.7	72.4 ± 18.1	29.9 ± 8.9	50.6 ± 11.6	62.5 ± 13.8
BMI (kg/m^2)	17.8 ± 3.1	20.3 ± 3.9	23.4 ± 5.0	17.0 ± 3.1	20.4 ± 3.8	22.6 ± 4.4
Waist circumference (cm)	61.1 ± 9.8	70.6 ± 10.9	80.1 ± 12.9	57.9 ± 8.5	70.1 ± 10.0	75.4 ± 10.9
MVPA (average $min{\cdot}d^{-1}$)	69.4 ± 29.1	59.5 ± 29.4	53.1 ± 25.9	58.1 ± 22.6	47.2 ± 24.6	39.1 ± 23.0

BMI body mass index.
MVPA moderate-to-vigorous physical activity.

average blood pressure and heart rate [29]. The device automatically inflates and deflates the cuff and uses an oscillometric technique to calculate systolic and diastolic blood pressure.

Non-HDL-Cholesterol

Non-HDL-cholesterol was calculated by subtracting HDL cholesterol, measured using a non-HDL precipitation method on the Vitros 5,1FS (Ortho Clinical Diagnostics), from total cholesterol [31]. Non-HDL cholesterol consists of very low density, low density, and intermediate density lipoprotein cholesterol and therefore reflects the cholesterol content of all apo B containing lipoproteins. Non-HDL cholesterol was chosen as the lipid marker because it is an important indicator of cardiovascular and diabetes risk among children and adolescents and is not reliant upon a fasted blood sample [32]. Blood samples were taken by a certified phlebotomist and were analyzed at the Health Canada Laboratory (Bureau of Nutritional Sciences, Nutrition Research Division). Other blood markers are available in the CHMS; however, the fasting requirement for some of these measures resulted in a marked reduction in the sample size when they are included. To ensure we had appropriate power for the primary purpose of this analysis, we included non-HDL-cholesterol as the sole blood marker.

Statistical analysis

Differences between sex, age groups and BMI status were assessed using t-tests. Statistical significance was set at a p value of 0.05. It is important to note that the values presented from this analysis in Figures 1 and 2 represent the mean across the week for sedentary time accumulated in prolonged bouts. In other words, there are zeros included in the averaging (because not all individuals had bouts of each length on all days) which in the case of 120 minutes bouts, brings the mean time accumulated below 120 minutes.

Associations between sedentary time variables and BMI and waist circumference were assessed using regression analyses. BMI and waist circumference vary by sex and change with normal growth and maturation [27,33]. Age and sex were both significantly correlated with average daily minutes of MVPA and age was significantly correlated with average daily minutes of sedentary time. Therefore, all regression analyses were completed separately by sex and the following age categories: 6–10, 11–14 and 15 to 19 years. The choice of age categories was based on the sampling design of the CHMS. Linear regression models were run separately for each sex and age grouping and were adjusted for age, average daily minutes of MVPA on valid days, and accelerometer wear time. The wear time adjustment was specific to the time period being examined. For example, the models for weekdays after 3 pm where adjusted for wear time on weekdays after 3 pm. Eight separate regression models (sedentary time, breaks in sedentary time, prolonged bouts lasting at least 20, 40, 60, 80, 100, 120 minutes) were run for each time period: overall, weekdays after 3 pm and weekends. The p-value to reach statistical significance in the linear regression analyses was adjusted for the number of models run. In other words, to reach statistical significance, the regression p values had to be less than .006 (i.e., 0.05/8 = 0.006).

All statistical analyses were performed using SAS v9.1 (SAS Institute, Cary, NC) and were based on weighted data (to be representative of the Canadian population and to account for non-response bias) for respondents with at least four valid days. To account for survey design effects of the CHMS, standard errors, coefficients of

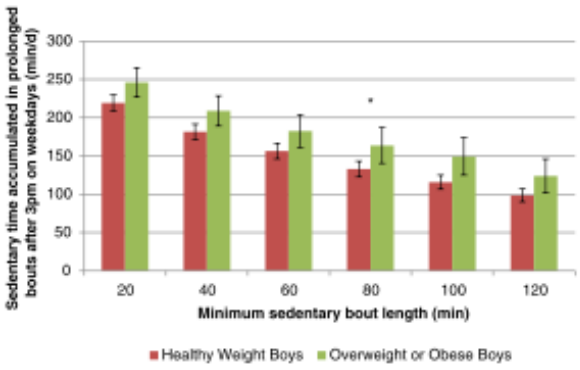

Figure 1 Sedentary time accumulated in prolonged bouts after 3 pm on weekdays in boys. *significant difference between healthy weight and overweight or obese.

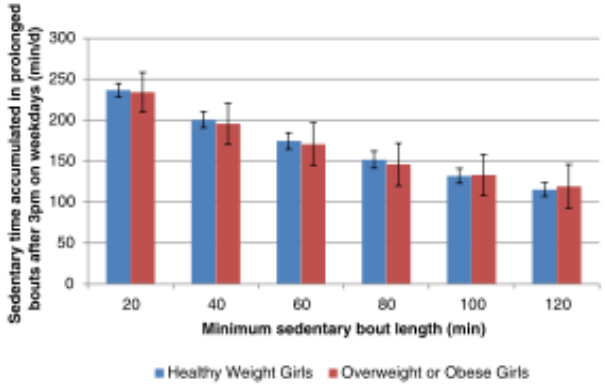

Figure 2 Sedentary time accumulated in prolonged bouts after 3 pm on weekdays in girls.

variation, and 95% confidence intervals were estimated using the bootstrap technique [34-36]. Overweight and obese were collapsed into one category because we lacked statistical power to compare not overweight/obese (includes healthy weight and underweight), overweight and obese as 3 separate categories.

Results

Descriptive characteristics of the sample are provided in Table 1. The analysis is based on 1,608 children and youth between the ages of 6 and 19 years. The sex split was even between boys (n = 809, 50.3%) and girls (n = 799).

Sex and Age differences

Sex and age differences are presented in Table 2. On average, boys accumulated 508 minutes per day of sedentary time while girls accumulated 524 minutes per day. Boys accumulated more sedentary time on weekdays after 3 pm compared to girls, while boys had a lower number of breaks in sedentary time per day compared to girls. Sedentary time was higher in 11–14 year olds and 15–19 year olds compared to children aged 6–10 years. Girls aged 15–19 years accumulated more sedentary time overall and after school compared to boys of the same age.

Body mass index differences

Differences by BMI status are represented graphically in Figure 1 for boys and in Figure 2 for girls. Overweight and obese boys accumulated more sedentary time after 3 pm on weekdays when compared to boys who are not overweight/obese (Table 2). Overweight and obese boys accumulated more sedentary time after 3 pm on weekdays as prolonged bouts lasting at least 80 minutes when compared to boys who are not overweight/obese (171 vs. 133 min·d^{-1}) (Figure 1). No sedentary time variables differed between girls who are overweight/obese and those who are not overweight or obese (Table 2; Figure 2).

Regression analysis results

Prolonged bouts of sedentary time lasting at least 40 - minutes, after 3pm on weekdays, were positively associated with waist circumference (ß = 2.23, $p < .006$) while prolonged bouts of sedentary time lasting at least 80 - minutes was positively associated with both BMI (ß = 0.72, $p < .006$) and waist circumference (ß =1.76, $p < .006$) in boys aged 11–14 years. Each additional 60 minutes of sedentary time accumulated during the after school period was associated with a 1.4 kg·m^{-2} higher BMI and a 3.4 cm higher waist circumference in 11–14 year old boys. Number of breaks in sedentary time, after 3pm on weekdays, was negatively associated with waist circumference (ß = –4.04, $p < .006$) in boys aged 11–14 years. No sedentary time variables were significantly associated with BMI or waist circumference in girls of any age or in boys aged 6–10 or 15–19 years. No sedentary time variables were associated with blood pressure or non-HDL cholesterol in boys or girls. The full results from the regression analyses are available as Additional file 1: Tables S1, Additional file 2: Tables S2, Additional file 3: Tables S3, Additional file 4: Tables S4, Additional file 5: Tables S5.

Discussion

The objective of this study was to examine the association between accelerometer-measured sedentary time and health risk in children. Our analysis supports previous studies that found few or no significant associations between accelerometer-measured sedentary time and health risk in children [1,11-16]. This study is novel because it

Table 2 Descriptive sedentary time results (mean ± standard deviation), by age, sex and obesity status

	Sedentary time (min/d)	Sedentary time on weekdays after 3 pm (min/d)	Sedentary time on weekends (min/d)	Breaks in sedentary time per day (number/d)
Boys	**507.5 ± 90.8**	**265.5 ± 65.8***	**490.9 ± 114.5**	**81.2 ± 11.6***
Age				
6 to 10 years	445.5 ± 79.5	212.8 ± 52.0	440.5 ± 101.1	84.4 ± 10.3*
11 to 14 years	524.1 ± 76.9**	273.9 ± 58.0**	503.2 ± 103.3**	79.2 ± 11.2*,**
15 to 19 years	553.9 ± 77.0*,**	310.7 ± 44.7*,**	533.8 ± 126.4**	80.1 ± 13.6**
BMI				
Not overweight or obese	499.9 ± 88.4	259.4 ± 63.7	483.7 ± 115.0	81.2 ± 11.3
Overweight or obese	527.8 ± 94.6	281.7 ± 69.2***	509.7 ± 220.0	81.1 ± 12.6
Girls	**523.8 ± 91.6**	**277.1 ± 67.5**	**493.6 ± 106.7**	**85.4 ± 11.7**
Age				
6 to 10 years	446.1 ± 72.9	215.4 ± 52.1	428.7 ± 96.0	89.6 ± 10.4
11 to 14 years	526.8 ± 63.5**	275.0 ± 49.3**	503.2 ± 87.5**	84.7 ± 10.5**
15 to 19 years	582.1 ± 81.4**	326.3 ± 47.5**	538.4 ± 106.1**	82.6 ± 13.6**
BMI				
Not overweight or obese	523.5 ± 91.3	277.1 ± 68.3	495.4 ± 107.2	85.2 ± 11.5
Overweight or obese	524.6 ± 93.1	277.0 ± 64.2	487.1 ± 105.0	86.2 ± 12.5

* significantly different to estimate for girls ($p < .05$).
** significantly different to estimate for 6–10 year olds of same sex ($p < .05$).
*** significantly different to estimate for not overweight or obese ($p < .05$).
BMI, body mass index.

included a wider range of sedentary time variables than what has been previously considered that characterize the timing and patterning of how the sedentary time is accumulated. Further, the sedentary pattern variables were designed to be more reflective of real-world sedentary behaviour. For example, a limited number of short transitions into light activity were allowed to reflect real-life situations where individuals are sedentary for long periods but move around occasionally (i.e., to go to the washroom or answer the phone). Despite the inclusion of more comprehensive sedentary pattern variables, this study found few significant relationships with health risk and the associations observed were limited to boys aged 11–14 years.

In theory, excessive sedentary time is associated with negative health outcomes [4,37] and self-reported screen time is associated with elevated health risk in children [1-3]; however, the way we currently measure sedentary time with accelerometers does not consistently support this link. To date, the research linking accelerometer-measured sedentary time with health risk among children and youth has been mixed. It is therefore unclear whether a relationship exists only in some populations or if differences in analytical approaches explain the inconsistencies observed. There appears to be more evidence supporting a lack of relationship between accelerometer-measured sedentary time and health risk in children and youth [1,11-16] than there is supporting a relationship [8-10]. Adjustment for MVPA appears to attenuate significant associations between accelerometer-measured sedentary time and health risk [13,16], suggesting that MVPA is more powerful than total sedentary time at explaining the variance in health risk in children and youth. In our unadjusted regression models, sedentary time was associated with BMI and waist circumference in boys aged 6 to 14 and girls aged 6 to 10 years; however, after adjustment for MVPA, these associations remained significant only in 11–14 year old boys.

In 2008, Healy and colleagues published a paper that reported a significant association between number of daily breaks in accelerometer-measured sedentary time and lower metabolic risk in adults [17]. This work led researchers to question whether it is the pattern of how sedentary time is accumulated, rather than simply the total volume of sedentary time, which matters for health. Do frequent interruptions in sedentary time attenuate the health risk that sedentary time imposes? Does this relationship apply in both children and adults? Carson and Janssen found no significant associations between breaks in sedentary time or prolonged bouts of sedentary time lasting 30 minutes with cardio-metabolic risk

factors in a large sample of American children [1]. Number of breaks in sedentary time was only associated with waist circumference in 11–14 year old boys in the present analysis. We included an additional layer of complexity by examining sedentary time, breaks and prolonged bouts of sedentary time during periods of discretionary free time: weekends and after school. We hypothesized that sedentary time accumulated during these periods would better discriminate between children engaging in healthy and unhealthy levels of sedentary behaviour when compared to simply examining overall sedentary time. We observed no significant associations between the patterns of sedentary time accumulated on weekends and health risk in children; however, some relationships emerged when we examined sedentary time accumulated during the after school period. Interestingly, we only observed significant associations in boys aged 11 to 14 years of age when the regression models were adjusted for age, MVPA and accelerometer wear time.

It is difficult to speculate why we observed significant findings in boys and not girls. It is possible that more overweight and obese boys in this sample were engaging in prolonged bouts of sedentary time after school, a finding consistent with previous research that has found that boys spend considerably more time in specific sedentary behaviours such as video game playing [38-40]. Average daily sedentary time and weekend sedentary time did not differ between boys and girls while sedentary time accumulated after 3 pm on weekdays was higher in boys compared to girls (277 vs. 266 minutes). Significant differences between boys who are not overweight/obese and overweight/obese boys were observed in the sedentary variables; however, no such differences were observed in girls. For example, there was virtually no difference in average daily sedentary time between girls who are not overweight/obese versus those who are (524 vs. 525 min·d^{-1}) while a more marked difference existed between boys who are not overweight/obese versus those who are (500 vs. 528 min·d^{-1}). In Figure 1, a distinction between boys who are not overweight/obese and those who are can be observed across all bout lengths; however the difference is only statistically significant when the bout length is at least 80 minutes long. By comparison, no difference is noticeable by overweight/obesity status in girls and there is more crossover in the error bars in girls (Figure 2). Similarly, no significant associations emerged in girls in the regression analyses while in 11–14 year old boys, prolonged bouts of sedentary time lasting at least 80 minutes, accumulated during the after school period were associated with both BMI and waist circumference. Explaining why significant associations were observed in 11–14 year olds boys but not those who were 6–10 or 15–19 years is not easy. In a large sample of US children, Sisson and colleagues observed an increase in screen time with age from 2 to 15 years [41]. In the Health Behaviour and School Aged Children Survey, the Canadian data show that screen time increases from age 11 to 15 years [42] with the peak occurring in grade 9 (approximately 14 - years) [43]. These large data sets suggest that the 11–14 year old age group may be an age range where screen time habits change significantly, thus increasing the amount of variation (and likelihood to find significant associations) in this variable.

The lack of evidence linking accelerometer-measured sedentary time with health risk in children is counter-intuitive given the consistent observation that screen time, a key contributor to total sedentary time, is associated with health risk [1-4]. One of the fundamental differences between self-reported screen time and objectively measured sedentary time is that the former is capturing one specific activity while the latter is capturing screen time in addition to many other sedentary behaviours. Much of the time accumulated as "sedentary" represents normal aspects of day-to-day life therefore capturing every minute in a day that is sedentary, as accelerometers do, may dilute the associations between specific sedentary behaviours (e.g., watching television) and health risk. It is possible that some forms of sedentary behavior (e.g., screen time, long car or bus travel) are associated with negative health outcomes while other forms of sedentary behavior (e.g., eating, reading, resting, socializing etc.) are not. Similarly, data reduction procedures used in accelerometry analysis (e.g., 10 hour wear time criteria) were developed to accurately capture MVPA and whether they are appropriate for sedentary behaviour research questions is unknown. For example, it has been suggested that wear time has a disproportionate impact upon estimates of sedentary time compared with MVPA [44]. Teasing out which sedentary behaviours beyond screen time are associated with negative health outcomes represents an important area for future research.

The sedentary behaviours that are of known public health concern in children and youth (e.g., excessive levels of screen time) typically last for extended periods of time (i.e., up to several hours at once). This reality was the motivation behind the way prolonged bouts of sedentary time were defined in the present analysis. Had we used a strict definition of what ended about (i.e., any transition out of sedentary) then our longest bout length would have been very short (e.g., 10 minutes) and thus not representative of one of the key sedentary behaviours that we were interested in capturing. The allowance of a modest amount of light intensity movement within the prolonged sedentary bouts was therefore purposeful and allowed much longer bout lengths to be

examined (up to 2 hours). Number of breaks per day, also assessed in this analysis, is an important aspect of sedentary behaviour patterns. Given that we and others [1] have not consistently observed significant associations between number of breaks per day and health risk, it is important to look at alternative pattern variables such as prolonged bouts. Further, the extension of bout length in the present analysis was important to build off the only other published work that examined prolonged bouts up to 30 minutes in children and youth [1].

It is possible that the true health effect of sedentary time is attenuated by limitations with the data and analysis. Possible limitations that dilute the possibility of observing a true relationship include: i) the cross-sectional nature of the data, ii) non-response bias, iii) the possibility that the findings in 11–14 year olds boys reflect Type 1 error. As described in the methodology, the non-response bias is adjusted for in the data. We attempted to minimize the likelihood of Type 1 errors in the regression analyses by adjusting the p-value for significance from .05 to .006. Other limitations include the lack of ability to confirm precisely when children were finished school. We examined the period after 3 pm on weekdays [45] based on the assumption that most kids would finish school sometime between 2-4 pm. Accelerometers are limited in their ability to capture postural changes (i.e., cannot differentiate between sitting and standing) and are therefore limited in their ability to measure sedentary before as well as other tools which encompass an inclinometer in addition to an accelerometer. No significant associations were observed between sedentary time variables and blood pressure or non-HDL cholesterol and this may be due to it likely being more difficult to detect meaningful differences in biomarkers in children and youth than adults because younger people are more distal to pathophysiological developments. A similar examination on a population of high-risk children (e.g., overweight or obese or with a family history of cardio-metabolic disease) may lead to different findings as these children would be more likely to exhibit abnormalities in blood markers and blood pressure. Finally, examination of interaction and confounding effects was limited because the number of variables (including interaction terms) that can be tested within the CHMS data set is limited by the available degrees of freedom.

Conclusions

Sedentary time accumulated during the after school period was associated with BMI and waist circumference, independent of MVPA, in boys aged 11 to 14 years. No sedentary behaviour variables were independently associated with any health markers in older or younger boys or in girls of any age. Future studies should consider examining more comprehensive sedentary time pattern variables when attempting to elucidate the relationships between sedentary time and health risk in children and youth.

Additional files

Additional file 1: Table S1. Associations between sedentary time variables and body mass index, presented by sex and age groups.

Additional file 2: Table S2. Associations between sedentary time variables and waist circumference, presented by sex and age groups.

Additional file 3: Table S3. Associations between sedentary time variables and systolic blood pressure, presented by sex and age groups.

Additional file 4: Table S4. Associations between sedentary time variables and diastolic blood pressure, presented by sex and age groups.

Additional file 5: Table S5. Associations between sedentary time variables and non-HDL cholesterol, presented by sex and age groups.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

RCC conceived the manuscript, formed the research team, directed the analysis, led the writing. DG was involved in the conception the manuscript, completed the analysis and contributed to the writing. IJ contributed to the writing and provided critical review of the analysis. SLW was involved in the conception of the manuscript, contributed to the writing and provided critical review of the analysis and writing. TJS, VC and MST contributed to the writing and provided critical review of the writing. All authors read and approved the final manuscript.

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Appendix D: Publications during PhD Studies

9.4 Published and Accepted Manuscripts

1. **Saunders TJ**, Chaput JP, Goldfield GS, Colley RC, Kenny GP, Doucet E, Tremblay MS. Prolonged sitting and markers of cardiometabolic disease risk in children and youth: A randomized crossover study. *Metabolism*, In Press. IF=2.772.

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9.5 Published Abstracts

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3. **Saunders T.J.**, Palombella A., McGuire, K.A. Janiszewski, P.M., Després, J.P., Ross, R. Acute exercise increases adiponectin levels in abdominally obese men.*Applied Physiology, Nutrition and Metabolism,* 36: S350, 2011.

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