Thesis

The Effects of a Reduced-Exertion High-Intensity Interval Training Protocol on Measures of Cardiovascular and Metabolic Health in Physically Inactive Individuals

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Table of Contents

List of Abbreviations

ANOVA – Analysis of variance

BP - Blood pressure

CSEP – Canadian Society for Exercise Physiology

CVD - Cardiovascular disease

ECG – Electrocardiogram

GLUT4 – Glucose transporter type 4

HIIT – High-intensity interval training

HR – Heart rate

HR_{max} – Age predicted maximal heart rate

HRV – Heart rate variability

ITT – Intent to treat

kp – Kilopond

MET - Metabolic equivalent

MICT – Moderate intensity continuous training

MS – Millisecond

RER – Respiratory exchange ratio

RMSRR – Root mean squared of successive differences between normal heartbeats

rpm – Revolutions per minute

SDNN – Standard deviation of normal to normal heartbeats

SIT - Sprint interval training

T2D – Type 2 diabetes

VO_{2max} – Maximal aerobic capacity or maximal oxygen consumption

WHO – World Health Organization

List of Tables

Table **Page**

List of Figures

Figure **Page**

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Abstract

Cardiometabolic diseases such as diabetes and cardiovascular disease are a growing threat to the quality of life of the population. Exercise is a frontline approach to treat and prevent cardiometabolic disease and its associated risk factors. The majority of individuals, however, are physically inactive and fail to meet weekly physical activity guidelines primarily due to time-constraints. High-intensity interval training (HIIT) is a time-efficient method of exercise for improving physical fitness and reducing cardiometabolic risk factors compared to moderate-intensity continuous training (MICT). Recent research indicates that the number of repetitions and duration of high-intensity intervals can be reduced without attenuation of health benefits. This study recruited nine physically inactive but otherwise healthy participants (6 female, 3 male) which engaged in a 6 week reduced-exertion HIIT protocol. Participants were assessed before and after a 2 week run-in period, and again upon completion of the exercise protocol to assess the effect on predicted aerobic capacity (VO_{2max}), resting heart rate (HR), resting blood pressure, heart rate variability (HRV), fasting blood glucose, peak power, mean power, body mass, and body fat %. Participants improved predicted VO_{2max} $F(2,16)=6.33$, $p=.009$, peak power $F(2,16)=10.84$, $p=.001$, and mean power $F(2,16)=20.87$, $p=.00006$, but no changes were observed in body mass, body fat %, resting HR, resting blood pressure, resting HRV, and fasting blood glucose. In conclusion, a reduced-exertion HIIT protocol with minimal time-commitment improved predicted VO_{2max}, peak power, and mean power and is a time-efficient alternative or adjunct method of exercise for eliciting health benefits in physically inactive individuals.

Chapter 1 - Introduction

In today's society, cardiometabolic diseases such as diabetes and cardiovascular disease (CVD) pose an extraordinary and growing threat to the quality of life of the population. In 2014, CVDs were the single leading cause of mortality in Canada at 26.8% (Statistics Canada, 2014) and they contributed to nearly half (48%) of the mortality from the noncommunicable disease worldwide (Mendis, Puska, & Norrving, 2011). The pathology of CVD is related to metabolic health as CVD is a commonly occurring complication in individuals with diabetes (Szuszkiewicz-Garcia & Davidson, 2014). Individuals with type 2 diabetes (T2D) are associated with a two- to three-fold higher risk of developing a secondary cardiovascular complication and mortality compared to a healthy population (Green, 2014; Huxley, Barzi, & Woodward, 2006; Peters, Huxley, & Woodward, 2014). In 2012, there were 1.96 million reported cases of diabetes in Canada; this figure is expected to more than double by 2022 (Bilandzic & Rosella, 2017). With these new cases comes an extraordinary financial burden adding an estimated $15 billion in healthcare costs by 2022 (Bilandzic & Rosella, 2017). If the current trend continues, the ongoing costs of diabetes treatment will soon become unaffordable. The risk of developing these diseases can be highly mitigated with modified lifestyle behaviors such as optimized diet and exercise (Chudyk & Petrella, 2011; Colberg et al., 2010; Warburton, Charlesworth, Ivey, & Nettleford, 2010).

An exercise as medicine approach is an effective option for the treatment and prevention of T2D and CVDs (Chudyk & Petrella, 2011; Warburton et al., 2010). The Canadian Society for Exercise Physiology (CSEP) recommends ≥150 minutes of moderate intensity or ≥75 minutes of vigorous physical activity training per week for individuals aged 18 to 64 years (Colberg et al., 2010; Colley et al., 2011; CSEP, 2013; Hallal et al., 2012; Haskell et al., 2007). Acquiring an adequate amount of physical activity is an essential component for maintaining health. Despite the apparent benefits, only 15 to 20% of Canadians achieve the recommended amount of exercise (Colley et al., 2011). Physical inactivity and prolonged sedentary behaviours are significant contributors to non-communicable disease around the world (Lee et al., 2012; World Health Organization (WHO), 2009). These behaviours are associated with a variety of poor health outcomes including all-cause mortality, T2D, incidence of breast and colon cancer, hypertension, low heart rate variability (HRV), and CVD risk (Warburton et al., 2010). Since the majority of the population is physically inactive, this poses a significant public health concern.

High-intensity interval training (HIIT) is a time-efficient means of producing similar or higher cardiometabolic benefits to moderate intensity continuous training (MICT) in a variety of populations (Batacan, Duncan, Dalbo, Tucker, & Fenning, 2017; Jelleymen et al., 2015; Weston, Wisloff, & Coombes, 2013). Many studies have demonstrated the efficacy of four to six repetitions of 30 second "all-out" cycle sprint protocols or sprint interval training (SIT) at improving insulin sensitivity and glucose disposal (Babraj et al., 2009; Burgomaster et al., 2005; Fisher et al., 2015; Racil et al., 2013). Moreover, comparative studies present evidence that high-volume aerobic HIIT protocols (4 repetitions of 4 minutes at 85-95% HR_{max}: active rest 3 minutes at 50-60% HR_{max}) are more efficacious than MICT for raising VO_{2max}, and reducing blood pressure (BP) and resting heart rate (HR; Moholdt et al., 2009; Molmen-Hansen et al., 2011; Rognmo et al., 2004; Tjonna et al., 2008; Wisloff et al., 2007). High-volume HIIT and

SIT protocols pose an entry barrier, as they require a high degree of motivation, are perceived as too intense, and require too much time to complete. Therefore, while it is essential to establish the health benefits of HIIT, a suitable protocol should be recommended, limiting the entry barriers, making time-efficient exercise more accessible.

Babraj et al. (2009) and Whyte et al. (2010) conjectured that the muscle glycogen depletion that occurs during a 30 second maximal effort plays a significant role in the physiological compensations following exercise. These findings are supported by the inverse relationship between muscle glycogen availability and cell-membrane GLUT4 content (Derave, Hansen, Lund, Kristiansen, & Richter, 2000), glycogen synthase activity (Jensen et al., 2006), and expression of GLUT4 mRNA (Steinberg et al., 2006). The upregulation of GLUT4 proteins in response to high-intensity exercise explains in part how exercise improves insulin dependent skeletal muscle glucose uptake (Steinberg et al., 2006). The relationship between muscle glycogen depletion and improved insulin action following exercise suggests that exercise which sufficiently depletes muscle glycogen will be more effective than those that do not.

The 30 second Wingate Anaerobic Power Test, commonly referred to as the Wingate Test, is a maximal exertion cycle ergometer test which measures the power output of the lower body (Bar-Or, 1987). A single bout of a 30 second Wingate Test is capable of depleting glycogen in the vastus lateralis muscle by 20% (Gibala et al., 2009). Work by Parolin et al. (1999) observed that glycogen breakdown during a Wingate Test is only highly active during the first 15 seconds, following which point glycogenolysis is severely attenuated. Glycogenolysis is also further attenuated in repeated bouts of the Wingate Test. Therefore, if an individual continues to perform additional 30 second maximal bouts, such as in a SIT protocol, the breakdown of glycogen would only be highly active in the first 15 seconds of the first one or two sprints (Parolin et al., 1999). Gibala et al. (2009) confirmed these observations, and muscle glycogen depletion was reduced by 20% following one bout but was only reduced by 30% following four bouts. These findings posit that the metabolic stimulus for the physiological adaptations that occur following SIT protocols may be accomplished with only one to two 15 second sprints.

New evidence has shown that the amount of exercise required to elicit a reduction in cardiometabolic risk factors can be achieved using a reduced-exertion HIIT protocol consisting of one to two short bouts of high-intensity exercise (Metcalfe et al., 2012; Metcalfe et al., 2016). Metcalfe et al. (2012) demonstrated an increase in aerobic capacity in healthy sedentary men and women by at least 1-metabolic equivalent (MET), an amount associated with a 10-25% reduction in CVD mortality risk (Kokkinos et al., 2004), and an increase in insulin sensitivity in men but not women. Metcalfe et al. (2016) also observed that 6 weeks of reduced-exertion HIIT elicited a significant improvement in aerobic capacity and a nonsignificant increase in insulin sensitivity equally in both men and women. The proposed research will mainly build on the work by Metcalfe et al. (2012) and Metcalfe et al. (2016) to establish if 6 weeks of a reduced-exertion HIIT intervention can elicit improvements in markers of cardiovascular and metabolic health in physically inactive individuals.

Therefore, the purpose of this research is to compare changes in anthropomorphic measures (body mass and body fat %), cardiovascular health (resting BP, resting HR, and HRV), metabolic health (fasting blood glucose), and physical fitness (predicted VO_{2max}, mean power, and peak power) between baseline, pre-, and post-test in healthy physically inactive individuals following 6 weeks of a reduced-exertion HIIT program.

Chapter 2 - Literature Review

Introduction

Exercise is the frontline approach for the treatment and prevention of cardiovascular and metabolic diseases; however, the majority of individuals increase their cardiometabolic disease risk by living a sedentary and physically inactive lifestyle (Lee et al., 2012). Commonly cited barriers to exercise are a lack of time, motivation, and/or enjoyment for not meeting weekly exercise recommendations (Colberg et al., 2011; Colley et al., 2011; Reichert et al., 2007; Stutts, 2002). Compared to MICT, research has shown that HIIT is a time-efficient way to reduce cardiometabolic risk factors (Batacan et al., 2017; Jelleymen et al., 2015; Weston et al., 2013). Many existing HIIT protocols are perceived as too intense for many populations to perform. Metcalfe et al. (2012) and Metcalfe et al. (2016) have demonstrated that minimal amounts of high-intensity exercise, no more than 2 minutes per week, can elicit cardiometabolic benefits. The reduction in exertion in this protocol is more suitable for a sedentary population that may not tolerate more intense forms of exercise. Furthermore, the amount of HIIT required to improve cardiometabolic health is much less than previously thought. Thus, a reduction in exertion may not result in a reduction in benefit (Vollaard & Metcalfe, 2017; Vollaard, Metcalfe, & Williams, 2017). In this section, literature pertinent to how exercise and HIIT affect markers of cardiovascular and metabolic health will be reviewed.

Research Problem

In Canada, diabetes poses an urgent threat to the health of the population. Currently, nearly 10% of all Canadians above the age of 12 years have either type I diabetes or T2D, 90% of which are T2D (Bilandzic & Rosella, 2017). In 2012, 1.96

million cases of diabetes were reported, yet by 2022 another 2.16 million cases are expected to be added (Bilandzic & Rosella, 2017). These new cases will add an estimated $15 billion to current healthcare costs over the next decade. If this trend continues, the costs of treating this disease will become unaffordable. It has been proposed that a mere 5% weight loss among individuals with diabetes would result in an estimated savings of $2 billion, and a 30% risk reduction in the most severe cases would save $1.5 billion (Bilandzic & Rosella, 2017).

Cardiovascular disease is the leading cause of mortality in Canada at 26.8% (Statistics Canada, 2014) and the cause of a third of the mortality worldwide (Mendis, Puska, & Norrving, 2011). The pathology of CVDs and diabetes are related as biomarkers used to assess risk in CVDs (e.g., hypertension, hyperglycemia, poor physical fitness) are also overlapping in those with T2D (Szuszkiewicz-Garcia & Davidson, 2014). This is concerning as individuals with T2D are associated with a two- to three-fold higher risk of developing a secondary cardiovascular complication and mortality compared to a healthy population (Green, 2014; Huxley et al., 2006; Peters et al., 2014).

The current recommendations for weekly exercise are at least 150 minutes of moderate exercise or at least 75 minutes of vigorous exercise for individuals 18 to 64 years of age (CSEP, 2013; Colberg et al., 2010; Haskell et al., 2007). Despite these recommendations, the majority (80-85%) of the population fails to meet weekly exercise guidelines (Colley et al., 2011; Hallal et al., 2012). The causes of any individual failing to meet exercise guidelines are complex. Among a host of reasons, the most commonly cited are a lack of time, motivation, and enjoyment (Colberg et al., 2011; Colley et al., 2011; Reichert et al., 2007; Stutts, 2002). The outcome of the failure to meet these guidelines is a population incurring the adverse health effects of physical inactivity. Physical inactivity has been identified as the fourth strongest predictor of mortality by the WHO and is one of the greatest contributors to major non-communicable diseases around the world (Lee et al., 2012; WHO, 2009). Moreover, physical inactivity and prolonged sedentary behaviours are associated with a number of poor health outcomes including diabetes and CVD (Lee et al., 2012; Warburton et al., 2010)

Exercise is accepted as an effective preventative and treatment intervention for metabolic dysfunctions and CVDs (Colberg et al., 2011; Colley et al., 2011; Hallal et al., 2012; Haskell et al., 2007). Due to a lack of time and enjoyment as prominent perceived barriers to exercise, a time-efficient method of exercising should be suggested as an alternative or adjunct to MICT. Previous research has shown HIIT to be a time-efficient means of eliciting health benefits compared to MICT (Batacan et al., 2017; Jelleymen et al., 2015; Weston et al., 2013) as well as being more enjoyable (Shepherd et al., 2015). Many existing HIIT protocols are perceived as too intense or requiring too much time to complete, although a reduction in the number of intervals and total duration of exercise may help remove these barriers. Metcalfe et al. (2012) and Metcalfe et al. (2016) demonstrated that a reduced-exertion HIIT protocol consisting of 2 minutes or less of high-intensity exercise per week can improve cardiometabolic risk factors (VO_{2max} and insulin sensitivity). Due to the interrelated nature of cardiometabolic pathologies, it is important to consider their effect on one another and how to target specific pathologies using clinically prescribed exercise. Further research on the merits of this protocol is needed to solidify its recommendation to a physically inactive population.

Cardiometabolic Risk Factors

The term cardiometabolic simply means both the heart and the metabolism. Cardiometabolic diseases are those that involve both metabolic and cardiovascular dysfunction (Chudyk & Petrella, 2011). These diseases include T2D, metabolic syndrome, coronary heart disease, cerebrovascular disease (stroke), peripheral artery disease, and CVD. Cardiometabolic risk factors are biomarkers or indices of health which have been associated with increasing risk of incurring the aforementioned diseases; these include poor aerobic exercise capacity (Kaminsky et al., 2013; Kokkinos & Myers, 2010), impaired glucose disposal (Sorkin, Muller, Fleg, & Andres, 2005), elevated resting BP (Lawes, Vander Hoorn, & Rodgers, 2008), elevated resting HR (Hanley et al., 2002), high adiposity (Verheggen et al., 2016), abnormal blood lipid profile (Depres & Lemieuz, 2006), and reduced HRV (da Silva et al., 2016; Kemp & Quintana, 2013).

Many of these risk factors arise from modifiable lifestyle behaviors, such as poor diet and lack of physical activity. The WHO has identified physical inactivity as the fourth strongest predictor of mortality, accounting for 6% of global mortality (WHO, 2009). This risk factor is only surpassed by elevated BP (13% of global mortality), tobacco use (9%), elevated blood glucose (6%), and with overweight and obesity not far behind (5%; WHO, 2009). Exercise is accepted as an effective preventative intervention for metabolic dysfunctions and CVDs (Haskell et al., 2007). Regularly engaging in exercise is an effective way of mitigating many cardiometabolic risk factors such as insulin resistance (Garber et al., 2011; Tjonna et al., 2008), elevated BP (Guimares et al., 2010), reduced aerobic capacity (Garber et al., 2011; Tjonna et al., 2008), and low HRV (Aubert, 2003; Hallman et al., 2017; Soares-Miranda et al., 2014).

Cardiometabolic Benefits of Exercise

The use of exercise as medicine is a frontline approach for treatment and prevention of cardiometabolic diseases. A vast amount of empirical evidence supports a causal link between positive health outcomes and physical activity and are the justification for weekly exercise recommendations by numerous organizations (Colberg et al., 2011; Colley et al., 2011; CSEP, 2013; Hallal et al., 2012). Regularly engaging in physical activity is strongly linked to a reduced risk of developing T2D (Gill & Cooper, 2008) and CVD mortality rate (Nocon et al., 2008). Furthermore, regular exercise is a proven means of mitigating individual risk factors associated with cardiometabolic diseases including improving insulin sensitivity (Garber et al., 2011; Holloszy, 2005; Kirwan, Solomon, Wojta, Staten, & Holloszy, 2009), improving glycemic control (Sigal, Kenny, Wasserman, & Castaneda-Sceppa, 2004), reducing BP (Guimaraes et al., 2010), and improving exercise capacity (Garber et al., 2011; Kaminsky et al., 2013; Kokkinos & Myers, 2010). As mentioned, there are barriers that prevent people from exercising. Designing an exercise protocol/recommendation that helps overcome these barriers is paramount to improving the health of the population.

High-Intensity Interval Training

Interval training can be defined broadly as short bouts of vigorous exertion interspersed with periods of low-intensity active recovery or passive rest. In a typical HIIT session, the intensity of the high intervals is 80-100% of a predicted HR_{max} or ≥70% of VO_{2max} (Weston et al., 2013) The duration of high-intensity intervals lasts from several seconds to 4 minutes (Weston et al., 2013) High-intensity intervals lasting from 2-3 and a total high-intensity time per session of ≥10 minutes are considering high-volume HIIT.

Variations of HIIT protocols that use high-intensity interval durations of 30 seconds to 1 minutes and complete high-intensity work per session of <10 minutes are classified as low-volume HIIT or SIT (Gibala, Little, Macdonald, & Hawley, 2012; Little et al., 2011). Another permutation of HIIT includes reduced-exertion HIIT, which further lowers high-intensity interval volume to 20 seconds and total high-intensity per session of only 2 minutes (Metcalfe et al., 2012; 2016).

Perhaps just as important as the high-intensity bouts are the active recovery periods that follow; active recovery periods must allow the HR to recover appropriately. In a review conducted by Weston et al. (2013), eligible studies controlled low-intensity active recovery periods at 50-75% of HR_{max} or 40-50% of VO_{2max}. The duration of the recovery periods varied based on the time at high intensity; typically work to rest ratios fell between 2:1 (Nybo et al., 2010) and 1:6 (Gibala et al., 2012). Currently, in the literature, the intensities of active recovery periods are often not stated. Controlling the intensity of the active recovery period is important to ensure that the participant is recovered enough to perform a subsequent maximal exertion bout without self-pacing due to fatigue. High-volume MICT has proven benefits for mitigating cardiometabolic health risk, but is not likely to be adhered to due to barriers to exercise such as a reported lack of time, boredom, and lack of enjoyment (Haskell et al., 2007; Reichert et al., 2007; Stutts, 2002). Previous research has shown that many indicators of cardiometabolic health can be influenced in much less time using short bouts of high-intensity exercise (Batacan et al., 2017; Jelleymen et al., 2015; Weston et al., 2013). In studies that compared equal energy expenditure, high-volume aerobic HIIT (four repetitions high-intensity, 4 minutes at 85-95% HR_{max}: active rest 3 minutes at 50-60% of HR_{max}) and

MICT protocols, those in the HIIT groups exercised approximately 10 minutes less per session and exhibited greater improvements in aerobic capacity (Karstoft et al., 2014; Moholdt et al., 2009; Molmen-Hansen et al., 2011; Rognmo, Hetland, Helgerud, Hoff, & Slørdahl, 2004; Schjerve et al., 2008; Tjonna et al., 2008; Wisloff et al., 2007). As well, equal or greater reduction in BP compared to MICT (Molmen-Hansen et al., 2011; Rognmo et al., 2004; Schjerve et al., 2008; Tjonna et al., 2008), improvements in glycemic control (Karstoft et al., 2014; Tjonna et al., 2008) and reduced adiposity (Karstoft et al., 2014; Schjerve et al., 2008; Tjonna et al., 2008) was reported.

Additional research examining HIIT protocols with even less time commitment and less energy expenditure compared to MICT also proved to be a more efficient way of improving cardiometabolic health. In HIIT protocols, which used bouts of high-intensity less than 1 minute and exercise volume was half as much as the MICT group, participants improved VO_{2max} (Fisher et al., 2015; Gibala et al., 2012; Mitranun et al., 2014; Shepherd et al., 2015), improved glycemic control (Fischer et al., 2015; Mitranun et al., 2014; Shepherd et al., 2015), reduced BP (Mitranun et al., 2014), and reduced adiposity (Mitranun et al., 2014; Shepherd et al., 2015). Metcalfe et al. (2012) demonstrated an improvement in aerobic capacity of greater than 1-MET as well as improved insulin sensitivity with as little as 1 minute of high-intensity exercise per week.

Reduced-exertion HIIT is a relatively new protocol for HIIT. It is an adapted form of low-volume HIIT, further abbreviating interval duration to make high-intensity exercise more palatable for populations averse to longer durations. Only two studies have measured changes in cardiometabolic risk factors using this protocol over a long enough duration to elicit meaningful changes (6 weeks; Metcalfe et al., 2012; 2016). Metcalfe et al. (2012) measured the effects of reduced-exertion HIIT on sedentary men and women, although a no exercise control was used in place of an MICT group. Both men and women demonstrated an increased VO_{2max} from baseline greater than 1-MET. In measures of glycemic control, men had improved insulin sensitivity but women did not (Metcalfe et al., 2012). Similar results were confirmed by Metcalfe et al. (2016), both men and women improved VO_{2max}, but only men improved insulin sensitivity. Reduced-exertion HIIT elicited a reduction in body mass index (BMI) in men, but no change was observed in women (Metcalfe et al., 2012). There were also no changes in BMI in men or women in Metcalfe et al., (2016). Skleryk et al. (2013) examined the effects of reduced-exertion HIIT on obese and sedentary men over a 2 week period. No statistically significant changes were observed from baseline in VO_{2max}, any measures of metabolic health, or in body composition (Skleryk et al., 2013).

Although not following the reduced-exertion HIIT protocol, other studies have observed beneficial health effects of brief bouts of high-intensity exercise with minimal time commitment. Studies such as Allison et al. (2016) and Jenkins et al. (2018) have investigated the effects of brief intense bouts of stair climbing (3 x 20 seconds). These exercise *snacks* have been shown to improve VO_{2max} in young physically inactive individuals (Jenkins et al., 2018) and in sedentary women (Allison et al., 2018).

Total exercise time and intervention duration should be considered when comparing HIIT protocols. Different intensities and interval durations will place varied metabolic load on energy-producing pathways, resulting in specific physiological and metabolic adaptations. Aerobic HIIT studies have reported a longer intervention duration and total exercise volume compared to low-volume HIIT and reduced-exertion HIIT protocols. For measures of glycemic control, aerobic HIIT and low-volume HIIT both improved glycemic control proportional to MICT (Batacan et al., 2017; Jelleymen et al., 2015; Weston et al., 2013). It has been demonstrated that low-volume HIIT caused rapid and long-lasting changes in the glucose delivery system (Babraj et al., 2009; Burgomaster et al., 2005) as well as stimulated mitochondrial biogenesis (Little et al., 2010). Improvements to the glucose delivery system may be more beneficial to an individual with T2D, while CVD patients may benefit more from increasing aerobic capacity. Further study is required to better inform exercise prescription for diseased populations.

High-intensity interval training protocols with shorter intervals (e.g., SIT) may be better suited for improving glycemic control due to the reliance on glucose for energy through glycolytic pathways (Babraj et al., 2009; Burgomaster et al., 2005; Little et al., 2010). Aerobic HIIT appears more beneficial for improving aerobic capacity and reducing BP and resting HR; although, study duration of aerobic HIIT is much longer than lower volume HIIT protocols. In the studies reviewed here, which measured both changes in aerobic capacity and a measurement of glycemic control, aerobic HIIT study duration ranged from 10 to 16 weeks (Rognmo et al., 2004; Tjonna et al., 2008); whereas, low-volume HIIT study duration ranged from 6 to 12 weeks (Gibala et al., 2012; Racil et al., 2013). For measures of adiposity, both aerobic HIIT and low-volume HIIT were comparable means of reducing body fat % and body fat mass to MICT.

High-volume HIIT such as aerobic HIIT appears to elicit greater improvements in VO_{2max} compared to low-volume HIIT, however, these observations were likely due to differences in exercise volume rather than other traits specific to the protocol. Regardless of the specific intensity, the number of intervals, and volume, HIIT is an effective therapy for T2D (Wormgoor, Dalleck, Zinn, & Harris, 2017). Despite reduced exercise volume, low-volume HIIT protocols are still effective at increasing VO_{2max} (Gist, Fedewa, Dishman, & Cureton, 2014; Sloth, Sloth, Overgaard, & Dalgas, 2013). Although, some authors conjectured that low-volume HIIT can be excessively fatiguing because the physiological events required to elicit physiological adaptations occur in the first one or two high-intensity intervals. These authors suggested that fewer intervals in a protocol may have equal or greater benefits for aerobic capacity (Vollaard & Metcalfe, 2017; Vollaard et al., 2017). In a comparison of HIIT protocols, those with four to six 30 second intervals (SIT) had little to no additional benefit in VO_{2max} compared to those with only two 20 seconds intervals (Vollaard & Metcalfe, 2017; Vollaard et al., 2017). These observations posit that the use of reduced-exertion HIIT may be a valuable and time-efficient means for improving aerobic capacity and glycemic control. High-intensity interval training protocols should be prescribed in low-volume with few intervals as an adjunct to other forms of exercise. Despite a marked reduction in energy expenditure and time commitment, various types of HIIT are effective means of reducing cardiometabolic risk factors and mortality.

High-intensity interval training summary. High-intensity interval training exercise consists of short bouts of vigorous exercise interspersed with periods of active rest. A HIIT protocol may include any number and duration of intervals; protocols with fewer intervals but with longer interval and total duration are defined as high-volume HIIT, while those with more frequent intervals with shorter, more intense intervals and shorter total duration are defined as low-volume HIIT. Both forms are effective for increasing exercise capacity, an important marker for assessing chronic disease risk.

High-volume HIIT elicits greater VO_{2max} improvements due to higher exercise volume and longer duration (Karstoft et al., 2014; Moholdt et al., 2009; Molmen-Hansen et al., 2011; Rognmo, Hetland, Helgerud, Hoff, & Slørdahl, 2004; Schjerve et al., 2008; Tjonna et al., 2008; Wisloff et al., 2007). High-volume HIIT may also be more useful for reducing adiposity (Karstoft et al., 2014; Schjerve et al., 2008; Tjonna et al., 2008) and improving cardiovascular health measures (Molmen-Hansen et al., 2011; Rognmo et al., 2004; Schjerve et al., 2008; Tjonna et al., 2008). Although, these observations may also be attributable to greater exercise volume and duration, rather than the type of protocol performed. Low-volume HIIT, due to the higher metabolic demand on anaerobic energy pathways appears to be more effective for improving metabolic health (Babraj et al., 2009; Burgomaster et al., 2005; Little et al., 2010). New research has posited that reduced-exertion HIIT, despite only using one to two high-intensity intervals, can elicit health benefits similar to low-volume HIIT (Metcalfe et al., 2012; 2016). A reduced-exertion form of HIIT may be a more appropriate form of exercise for physically inactive individuals and those with time constraints.

Mechanism of Action

The notion that physiological adaptations that occur as a result of exercise are due to an increased demand for energy in muscle cells is widely accepted. Exercise variables such as intensity and duration place specific burdens on different metabolic pathways. A more intense bout of exercise, such as that used in HIIT, will put more demands on anaerobic pathways (Babraj et al., 2009; Burgomaster, Heigenhauser, & Gibala, 2006). While this field of study has elaborated on the central and peripheral adaptations to MICT, a growing body of evidence is showing that equal or greater adaptations can occur following HIIT even when total exercise time and energy expenditure is reduced (Weston et al., 2013). It appears that intermittent high-intensity exertion within an exercise session is responsible for these physiological adaptations, yet the mechanisms of this action are not entirely understood.

More vigorous exercise utilizes larger muscle groups, which under substantial metabolic stress, results in a higher rate of glycogen breakdown-turnover and rapid degradation of muscle glycogen (Babraj et al., 2009; Burgomaster et al., 2006). This stress facilitates muscle glycogenolysis and translocation of GLUT4 to produce energy to a greater degree than lower intensity exercise (Gibala, Gillen, & Perceival, 2014). An increase in GLUT4 occurs to increase glucose extraction from the circulating blood for delivery to working muscles (Hughes et al., 1993). Burgomaster et al. (2006) demonstrated that after only 2 weeks of low-volume HIIT significant increases in GLUT4 concentrations occurred, which were sustained for 6 months after the termination of the protocol. Babraj et al. (2009) suggested that HIIT elicits remodeling of the glycogen molecule pool, making the mobilization of glucose easier during exercise. Both adaptations seem to improve insulin sensitivity via enhanced endogenous production of glucose (Babraj et al., 2009; Burgomaster et al., 2006).

The mechanism of action provides important physiological context for understanding why HIIT protocols are effective. Previous research has shown that HIIT based around a 30 second Wingate Test is effective at increasing aerobic capacity and glucose metabolism (Babraj et al., 2009; Fisher et al., 2015; Gibala et al., 2012; Racil et al., 2013). These effects are in part due to the interaction between muscle glycogen depletion and insulin-dependent muscle glucose uptake via GLUT4 upregulation (Graham et al., 2010). This method may be excessively fatiguing as glycogen breakdown via glycogenolysis is only predominantly active during the initial 15 seconds of high-intensity exercise, and is severely attenuated in the last 15 seconds and subsequent bouts (Parolin et al., 1999). Moreover, due to the excessively fatiguing nature, participants often subconsciously pace themselves by conserving energy at the beginning of the test (Zajac, Jarzabek, & Waskiewicz, 1999).

Metabolic Health Measures

The main pathway the body uses to move glucose from the blood into the cell is a protein located in the membrane of skeletal muscle and adipose cells called GLUT4 (James, Brown, Navarro, & Pilch, 1988). Insulin-stimulated glucose transport into the cell is a rate-limiting factor for glucose metabolism. When measuring the glucose disposal rate, insulin must signal GLUT4 to allow glucose into the cell and the cell must have enough transportation capacity to allow this to happen. As such, the disposal rate is determined by the efficiency of insulin signaling and glucose transport. An inability to move glucose into the cell can occur independently of insulin signaling when GLUT4 protein content is diminished and can result in elevated blood glucose levels.

Individuals with T2D have approximately 40% less total GLUT4 protein content than those without T2D (Kelley et al., 1992). A shortcoming in glucose transport is reflected in a decreased glucose disposal rate. In individuals with this pathology, insulin therapies may not meaningfully reduce circulating blood glucose, because the glucose disposal would be limited by glucose metabolism, not insulin signaling. Kelley et al. (1992), examined the difference in glucose disposal rates between noninsulin-dependent T2D and weight matched controls. They found that the glucose disposal rate was indeed lower in the T2D group, as well as glucose usage and storage capacity.

To improve insulin sensitivity in individuals with T2D, the whole-body glucose disposal rate must be increased via increasing concentrations of GLUT4. Individuals who are physically inactive experience a loss of GLUT4 concentrations and the glucose disposal rate (Kampmann et al., 2011). Kampmann et al. (2011) demonstrated that those with T2D had a lower expression of the GLUT4 gene than healthy individuals and gene expression is severely reduced if insulin is required. Exercise is an important stimulus for upregulation of GLUT4; with meaningful stimulation of this process occurring from relatively small amounts of exercise (Burgomaster et al., 2005). Hussey et al. (2012) evaluated the effects of a single bout of moderate intensity exercise on GLUT4 gene expression in T2D and healthy controls. After only 60 minutes of exercise, gene expression was significantly elevated immediately following exercise and 3 hours afterward. Also, the gene expression of GLUT4 at rest was lower in the T2D group (Hussey et al., 2012). O'Gorman et al. (2006) used a similar intensity 60 minute exercise session, with a group of obese T2D (n=8) and obese non-T2D participants (n=7). The GLUT4 protein content did not increase immediately following exercise, but protein content increased in the obese T2D group one day after exercise with only a subtle increase observed in the obese non-T2D. Seven days following exercise, both groups demonstrated a similar increase in GLUT4 concentration levels.

Christ-Roberts et al. (2004) examined the effects of moderate intensity exercise over a period of 8 weeks on a group of insulin resistant (n=16) and T2D (n=6) participants. To date, this is one of the few studies to measure adaptations in GLUT4 content and glucose storage with glucose disposal. Participants exercised three times per week for 20 minutes at 60% of VO_{2max} and progressed to four sessions per week for 45 minutes at 75% of VO_{2max}. The exercise protocol elicited meaningful changes in the glucose disposal rate in both groups; however, the glucose disposal rate was still lower in the T2D group. A similar change was observed in the total GLUT4 protein content; both groups experienced a significant increase, but this increase was more moderate in the T2D group.

While many studies typically gauge the effectiveness of a HIIT protocol by changes in aerobic capacity, it is more practical to look for improvements in glucose metabolism and insulin sensitivity when evaluating the efficacy of these protocols for T2D since skeletal muscle insulin resistance is the primary defect in T2D (DeFronzo & Tripathy, 2009). Little et al. (2010) is one of the few to measure GLUT4 adaptations in response to HIIT training. Seven healthy male participants were recruited for 2 weeks of HIIT consisting of eight to twelve, 60 second maximal bouts followed by 75 seconds of active rest. There was a statistically significant increase in GLUT4 protein concentration levels following the exercise protocol and a decrease in average 24 hour blood glucose levels. Burgomaster et al. (2005) also evaluated HIIT on glucose metabolism in eight healthy males over a period of 2 weeks. The exercise protocol followed a traditional Wingate Test format, using four to six, 30 second maximal intervals with 4 minutes of recovery. Participants were tested at baseline, 1 week, and 6 weeks into training and 1 week and 6 weeks post-training. They observed significant increases in GLUT4 from baseline at each of the measurement times. This evidence demonstrates that high-intensity exercise leads to meaningful improvement in the concentration of a much-needed protein for glucose transport. Importantly, GLUT4 concentrations remained elevated following 6 weeks of detraining, as long as the study period itself, indicating that HIIT training leads to long-term improvements in glucose metabolism.

Anaerobic power. It is rarely reported for a clinical trial of HIIT to include observation of the change in power output in a short maximal exertion effort. Anaerobic testing using a 30 second Wingate Test, is commonly used to assess the power output of the individual. Although a relationship between mean and peak power output and cardiometabolic health has not been identified, an individual's ability to expend energy is a strong predictor of better cardiovascular health (Kaminsky et al., 2013; Kokkinos & Myers, 2010). An increase of 1-MET has been associated with a reduction in CVD mortality (Kaminsky et al., 2013). Therefore, the anaerobic power output of an individual may be an indicator of cardiometabolic health, similar to aerobic capacity. Anaerobic tests are also usually less time consuming and fatiguing, with less risk of injury compared to a treadmill VO_{2max} test. Thus, the use of anaerobic power may be a less invasive and time-consuming method for assessing cardiometabolic risk in future research. Furthermore, an increase in power output is likely a favourable outcome for an individual following a fitness routine and for the reversal of glucose metabolism dysfunction.

Cardiovascular Health Measures

Heart rate variability. Heart rate variability is the change in time between adjacent heartbeats and is a product of interdependent regulatory systems which govern heart function (Shaffer, McCraty, & Zerr, 2014). A major cardiovascular center located in the medulla of the brainstem integrates sensory information from proprioceptors, chemoreceptors, and baroreceptors (mechanoreceptors), as well as higher brain input from the cerebral cortex and limbic system (Shaffer et al., 2014). The medulla responds to the information by increasing HR via the sympathetic nervous system (SNS) or decreasing HR via the parasympathetic nervous system (PNS). In a healthy individual, the HR at rest represents the dynamic balance of neural activity between the SNS and PNS (Shaffer & Venner, 2013). A high HRV indicates the efficacy of the cardiovascular control centers ability to adapt to a variety of stimuli to maintain homeostasis rapidly. Dysfunction of this regulatory system is apparent when the HRV is inappropriate for the stress on the body. As such, the HRV of an individual serve as a valuable biomarker for predicting health outcomes.

At rest, both the SNS and PNS are active in regulating HR; however, the vagal effects from the parasympathetic branch are dominant which slow the HR below intrinsic rhythm of the sinoatrial node (Opthof, 2000). An increase in sympathetic activity is a mechanism to raise HR above the inherent value and occurs in response to perceived stress; however, both nervous systems act on different timescales. Sympathetic activity can take up to 5 seconds before the HR increases and reaches a steady rate in 20-30 seconds if the stimulus is maintained (Nunan, Sandercock, & Brodie, 2010). In contrast, the PNS functions at a much quicker pace, with stimulation taking effect within 1 second (Nunan et al., 2010). As a result, instantaneous adaptations of HR are vagally mediated. An insufficient HRV is typically due to inadequate vagal stimulation and overstimulation of the SNS which is indicative of a dysfunctional autonomic nervous system (ANS).

The heart is regulated by a sophisticated neural network. Modern technology has made it possible to measure physiological markers associated with HR regulation. Through these observations, it is apparent that “a healthy heart is not a metronome” (Shaffer et al., 2014, p. 5), the dynamic interaction between many regulating mechanisms causes a highly variable HR even during steady-state conditions (Shaffer et al., 2014). Through a phenomenon known as respiratory sinus arrhythmia, we know that HR varies with respiration; during inhalation HR increases and decreases during expiration (Yasuma & Hayano, 2004). Heart rate variability biofeedback is a technique which uses respiratory sinus arrhythmia, where the participant breathes at a controlled rate between 6 and 10 breaths per minute (Gevirtz et al., 2013). Gevirtz et al. (2013) showed that it was possible to achieve higher HRV through these breathing techniques in populations with cardiovascular, metabolic, and cognitive diseases.

Heart rate variability is a key index of ANS functioning and is generally accepted as a diagnostic tool for identifying mental and physical illness (Thayer et al., 2012). The observation of an individual’s HRV to assess the health of the ANS has been a useful clinical tool for identifying cognitive and physiological pathology as well as prognosticating disease outcomes (da Silva et al., 2016; Kemp & Quintana, 2013). In addition, HRV is a valuable biomarker for determining stress, health, and cognitive performance (Thayer et al., 2009; Thayer et al., 2012). Research shows that lower HRV is strongly correlated to future health problems and all-cause mortality (Dekker et al., 1997; Tsuji et al., 1994). Low HRV is also commonly observed in metabolic diseases such as diabetes (Velcheva, Damianov, Mantarova, & Antonova, 2011) and metabolic syndrome (Stuckey, Tulppo, Kininiemi, & Petrella, 2014) compared to healthy individuals. Cardiovascular autonomic neuropathy, a complication common in individuals with diabetes, is commonly diagnosed using HRV and can be detected before symptoms manifest (da Silva et al., 2014; Ewing, Campbell, & Clarke, 1976). Further study has demonstrated that HRV is more severely reduced when hypertension is concurrent with diabetes (Istenes et al., 2014). A review conducted by Stuckey et al. (2014), found that generally HRV was reduced in those with metabolic syndrome although results are often inconsistent depending on methodology. A similar effect on HRV is observed in CVDs such as myocardial infarction, heart failure, and hypertension (Casolo et al., 1991; Kautzner & Camm, 1997). Heart rate variability is also a predictor of future cardiac events (Tsuji et al., 1996), hypertension (Mancia et al., 1983), and mortality following myocardial infarction (Bigger, Fleiss, Rolnitzky, & Steinman, 1993).

In its simplest form, HRV is operationalized by the inter beat interval (IBI) between adjacent heartbeats averaged over a given period. The IBI is defined as the time between the upward spike of an R wave using an electrocardiogram (ECG) or the peak in blood volume pulse signal using photoplethysmograph (PPG) sensors (Schafer & Vagedes, 2013). At rest, readings from both methods are highly correlated; however, ECG is more accurate during ambulatory and dynamic measurements because PPG is unable to accurately detect changes in HR from sympathetic stimulation (Schafer & Vagedes, 2013). In addition, because sympathetic stimulation causes physiological adaptations which make it harder to record a clear signal (e.g., stiffening of the arteries and changes in the elastic properties of heart/muscle tissue), a sampling rate of at least 200 Hz to 1000 Hz is recommended to capture accurate data (Bernston et al., 2007; Kuusela, 2013). The IBI data recorded using these methods can be interpreted as time domain measurement of HRV. Alternatively, HRV can be analyzed on a frequency domain using power spectral density (TFESCNASPE, 1996).

One of the advantages of analyzing HRV on a frequency domain is that the variance and amplitude of each of the heart’s component rhythms can be observed individually. The Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology (TFESCNASPE, 1996) divided HR oscillations on a frequency domain into four main bands. The high-frequency spectrum was between 0.15 and 0.4 Hz which reflects the parasympathetic or vagal activity and is tied to the sinus arrhythmia caused by the respiratory cycle (Shaffer et al., 2014; Yasuma & Hayano, 2004). The low-frequency band was between 0.04 and 0.15 Hz and reflects baroreceptor activity at rest (Malliani, 1995), and the very-low-frequency band was between 0.0033 and 0.04 Hz (TFESCNASPE, 1996). The physiological explanation is not clear; however, the very-low-frequency has been associated with a number of negative health outcomes including all-cause mortality (Tsuji et al., 1994), high inflammation (Lampert et al., 2008), and cardiac arrhythmia death (Bigger et al., 1992). The ultra-low-frequency band is below 0.0033 Hz and reveals patterns in HR which occur in timeframes of 5 minutes or greater (Kleiger, Stein, & Bigger, 2005). This frequency band is a strong predictor of future cardiac events (Tsuji et al., 1994; 1996).

Heart rate variability and physical activity. Regular participation in exercise promotes physiological adaptations in the central and peripheral mechanisms of the cardiovascular system (Martins-Pinge, 2011; Michelini & Stern, 2009). As mentioned previously, Gevirtz (2013) demonstrated the efficacy of a HRV biofeedback method to use the variations in HR while breathing to improve autonomic functioning by increasing HRV in a variety of pathologies. Some authors suggested that the effect was due to a strengthening of a baroreceptor reflex (Vaschillo et al., 2006; Lehrer et al., 2003), while others suggested improved vagal communication (Porges, 2011). Exercise is an effective means of increasing HRV in a variety of diseases (da Silva et al., 2016; Villafaina et al., 2017) and in a healthy population (Grant, Viljoen, Janse van Rensburg, & Wood, 2012). The deviation from homeostasis which occurs during exercise may be an important stimulus for regulating HR compensations via baroreceptors and vagal communication. Furthermore, athletes, some of the most physically active individuals in the population, have repeatedly demonstrated healthier HRV profiles than sedentary individuals (Aubert, 2003). Overall, it is the consensus that physical activity is associated with positive HRV outcomes (Aubert, 2003; Hallman et al., 2017; Soares-Miranda et al., 2014). Conversely, individuals who spend a prolonged amount of time sitting suffer reduced HRV (Hallman et al., 2015). Prolonged sedentary periods are themselves associated with an increased risk of CVD and mortality (Chrysant & Chrysant, 2015; Lee et al., 2012; Van der Ploeg, Chey, Korda, Banks, & Bauman, 2012).

Heart rate variability seems to improve with exercise, as those who participate in sports and physical activity have greater HRV than those who do not (Oliveria, Leicht, Bishop, Barbero-Alverez, & Nakamura, 2013). Too much exercise however, puts too much stress on the body resulting in reduced HRV (Kiviniemi et al., 2014). Although there is limited research on what kind of exercise is better for improving ANS health, a comparison of HRV in sprinters and endurance athletes yielded similar variability patterns despite the use of different training methods (Berkoff, Cairns, Sanchez, & Moorman III, 2007). In a comparison to high intensity interval exercise protocols varying in interval distance and intensity (2x6x250m @85% max velocity, 2x3x500m @85% max velocity, and 2x6x250m @105% max velocity) but equal in total work (distance), differences in HRV were able to distinguish between all three protocols (Kaikkonen et al., 2012). The HRV was most reduced following exercise with the longest duration highest intensity period (Kaikkonen et al., 2012). These findings illustrated how the method of exercise implementation is a variable for the training load on the individual and that higher intensity exercise does indeed place greater strain on cardiovascular regulation.

The effects of exercise on post-exercise HRV have led many authors to study the use of HRV as a measure of training load (Kaikkonen, Hynynen, Mann, Rusko, & Nummela, 2010; Kaikkonen et al., 2012). In addition to intensity, time at a given intensity is also a factor in determining training load. Individuals who performed exercise at a steady pace for longer periods of time had greater training loads as determined by the lower post-exercise HRV (Lepretre et al., 2012; Kaikkonen et al., 2010). Moreover, Kaikkonen et al. (2012) reported that HRV in the MO250 group was not as reduced as the MO500 group despite identical intensity. These observations suggested that longer interval duration with fewer repetitions within a protocol represents a higher training load than shorter intervals with greater repetitions. To demonstrate this concept, the second half of a 500 m race is more difficult than the first 250 m because the body is previously stressed from the first 250 m. Further study is required to determine the effects of long-term adaptations to autonomic cardiac regulation in different exercise protocols varying in time and intensity. These findings are pertinent to the effects of HIIT as protocols where the training load has been reduced, still have the same effects on VO_{2max} as higher training loads.

Nummela et al. (2010) explored the connection between physiological adaptations of exercise and the ANS. In the study, 24 sedentary individuals performed steady-state exercise at 75% of their HR_{max}. Following 4 weeks of exercise, the participants were then divided into responders and non-responders based on improvements in VO_{2max}. Interestingly, HRV frequencies associated with vagal activity were only improved in those who also increased their VO_{2max}. Based on these observations, it appears that exercise and physical activity are essential for maintaining healthy autonomic function.

Participation in physical activity which puts metabolic stress on the body appears to be an essential stimulus for healthy ANS function among countless other bodily functions. Individuals who do not participate in a sufficient amount of physical activity exhibit poorer indices of HRV, among numerous other negative health outcomes.

Heart rate variability summary. Heart rate variability is a valuable tool for assessing and identifying many health conditions. The value of this measure is derived from the interplay between the SNS and PNS within the ANS to govern the heart to meet the demands of the body (Shaffer et al., 2014). The resultant effect of many pathologies is dysfunctional ANS regulation of the heart which can be detected through HRV measurement. Pathologies generally tend to result in an individual having a lower HRV (Thayer et al., 2012). Research into the relationship between HRV and physical activity has demonstrated that physical activity plays a role in the regulation of the heart. Individuals who are physically active, such as athletes, tend to have more favourable HRV profiles (Oliveria et al., 2013). In contrast, individuals who are physically inactive tend to have poorer measures of HRV (Hallman et al., 2015). An unhealthy HRV has been associated with an increased risk for a number of diseases including CVDs and diabetes (Tsuji et al., 1996; Velcheva, Damianov, Mantarova, & Antonova, 2011). The measurement of HRV is not commonly used to measure changes in ANS heart regulation in HIIT studies or other exercise intervention research. The use of HRV following a reduced-exertion HIIT protocol would lend valuable information as to whether this form of exercise is sufficient to elicit healthier HRV measures.

Aerobic capacity. The measure of an individual’s maximal oxygen uptake (VO_{2max}) during a cardiopulmonary exercise test is the gold standard for evaluating cardiovascular function in research, athletic, and clinical settings (Palange et al., 2007). These tests are also referred to as a VO_{2max} test and use the upper limit of the body's capacity to use oxygen during exercise to determine the degree of aerobic fitness, identify cardiopulmonary pathology, and assess disease risk (Laukkanen et al., 2001). The most common form of the VO_{2max} test is an incremental exercise test (IET) to volitional exhaustion where oxygen extraction of the participant is measured using a gas analyzer and spirometer (American Thoracic Society, 2003). Maximal oxygen consumption may also be measured using constant high-intensity exercise tests and indirectly use submaximal predictive tests (Evans et al., 2015; Noonan & Dean, 2000). The VO_{2max} test is prevalent across many fields of study and is near universally accepted (Palange et al., 2007).

The term VO_{2max} was first coined by Hill and Lupton (1923) and is defined as "the oxygen intake during an exercise intensity at which actual oxygen intake reaches a maximum beyond which no increase in effort can raise it" (p. 135). Subsequently, the VO_{2max} test is a measure of the upper limit of the cardiovascular, respiratory, and musculoskeletal system to extract and use oxygen for energy production (Astrand & Saltin, 1963). As such, the operationalization of cardiovascular fitness by VO_{2max} produces a measurable and reproducible parameter of oxygen usage by the body to fuel exercise. Maximal oxygen consumption is expressed in absolute terms (L/min) or relative terms (mL/min/kg). The direct measurement of VO_{2max} in a laboratory setting requires special equipment to measure the ratio of oxygen and carbon dioxide in expired gases. To conduct these tests, a great deal of physical exertion is required, as the participant must perform exercise to exhaustion. To yield the most accurate results, the participant should continue to exercise past maximal workload, producing a plateau in VO_2.

A predictive test is a less invasive method of estimating an individual's VO_{2max}. Predictive tests are intended for use in populations who are not capable of completing maximal exercise tests to exhaustion such as the elderly, cardiac patients, and those with abnormal gait patterns (Arena et al., 2007). Alternatively, predictive tests can be a useful tool when lab equipment is unavailable or to minimize risk to participants. An example of a predictive test is the Cooper's run, where a participant must run as far as possible within a 12 minute time-limit. A predicted VO_{2max} is then calculated based on distance covered and post-exercise HR. Another minimally invasive method of conducting a predictive test is the CAFT step test where VO_{2max} is computed based on the percentage of age predicted HR at a certain step rate (workload). Many studies have established that predictive tests are moderately to strongly accurate when compared to laboratory tests (Evan et al., 2015).

The Ekblom-Bak cycle ergometer test (EB-test) is a novel predictive VO_{2max} test which estimates aerobic capacity based on changes in HR between two different workloads (Ekblom-Bak, Bjorkman, Hellenius, & Ekblom, 2014). To conduct the EB-test, the participant's HR is recorded during the final minute of a 4 minute cycling bout at two standard workloads, and one higher self-selected workload (Ekblom-Bak et al., 2014). The changes in HR relative to the change in workload between the two trials is used to generate an age- and sex-specific VO_{2max} value. Ekblom-Bak et al. (2014) conducted a 1 week test-retest reliability analysis, using a sample (n=143) of varying gender, age, and physical activity. The results indicated that the EB-test exhibited high test-retest reliability and no statistically significant mean differences in VO_{2max} and HR between testing time points (Ekblom-Bak et al., 2014). The EB-test demonstrated superior test-retest reliability compared to the commonly used Astrand cycle ergometer test, with a 6.2% coefficient of variation (CV) and 9.8% CV, respectively. The study also observed a strong correlation (*r*=.91) and a moderate correlation (*r*=.68) for the EB-test and Astrand test, respectively, to an actual measurement of VO_{2max} (Ekblom-Bak et al., 2014). The overall CV for the EB-test was 9.3% compared to 18.1% for the Astrand test (Ekblom-bak et al., 2014). These findings indicate that the EB-test is a reliable and valid instrument for predicting VO_{2max} and demonstrates higher reliability and validity than the commonly used Astrand test (Ekblom-Bak et al., 2014).

Upon analysis of the EB-test prediction equation, a strong correlation between the actual VO_{2max} and the prediction residuals was discovered (ρ=.41 total, ρ=.51 in women, ρ=.81 in men), indicating that those who achieved a higher VO_{2max} were underestimated and those achieved the lowest values were overestimated. The EB-test prediction equation was revised by Bjorkman, Ekblom-Bak, Ekblom, & Ekblom (2016), who used a larger sample size (n=217) to create the equation and a cross-validation group (n=115). A new equation using forward multiple regression was used based on the larger sample, which was internally validated with an actual VO_{2max} test and subsequently, externally validated by the cross-validation group (Bjorkman et al., 2016). A comparison group consisting of only those from the cross-validation and those who also met the prerequisites for the Astrand test completed the Astrand test, and both the original and revised EB-test. The revised EB-test prediction equation demonstrated less error than the previous equation, with an 8.4% CV in men and 9.2% CV in women, accounting for 91%

of the adjusted variance in the overall sample (Bjorkman et al., 2016). The revised EB-test equation in the cross-validation sample accounted for 90% of the variance, and the CV was 8.3% for men and 10% for women (Bjorkman et al., 2016). In the comparison group, the revised EB-test equation had a lower error than the previous equation and the Astrand test.

Aerobic capacity as a measure of health. Aerobic capacity, or exercise capacity, is a strong predictor of overall health and risk aversion for cardiometabolic diseases. Myers et al. (2002) examined men with an abnormal cardiovascular exercise test and/or a history of CVD (n=3,679) against men who had normal test results and/or no history of CVD (n=2,534). The longitudinal study evaluated participants after a mean follow up time of approximately six years. During this time, there were 1,256 deaths, and on average the mortality group was older, had lower maximal HRs, greater systolic BP, greater diastolic BP, and lower exercise capacity (Myers et al., 2002). When adjusting for age, the exercise capacity was the strongest predictor of mortality in both the abnormal and healthy groups (Myers et al., 2002). The regression analysis revealed a 12% mortality risk reduction for each 1-MET, equivalent to a VO_{2max} of 3.5 ml/kg/min, in both groups (Myers et al., 2002).

These results were confirmed by Kokkinos (2004) who discovered an asymptotic relationship between exercise capacity and mortality following a seven year longitudinal study. A sample of 15,660 male veterans were divided into an abnormal test results and/or CVD history and a normal result void of CVD history group. Again, it was reported that exercise capacity was the strongest predictor of mortality; a logistic regression analysis revealed a 13% mortality reduction for every 1-MET increase

(Kokkino et al., 2004). Similar conclusions were reached by Kaminsky et al. (2013) and Kokkinos and Myers (2010) which helped solidify the inverse relationship between energy expenditure and mortality. Kokkinos et al. (2004) and Myers et al. (2002) reported that exercise capacity was equally as strong at predicting mortality regardless of CVD history. Although exercise capacity has been independently associated with CVD risk (Kodama et al., 2009), interestingly, additional CVD risk factors or a history of CVD does not increase the risk of mortality, indicating that the consideration of an individual's specific CVD risk factors may not be better at predicting mortality than exercise capacity alone. Thus, the relationship between exercise capacity and mortality is independent of CVD history.

Resting heart rate. Simple measures of cardiovascular function such as resting HR, resting BP, and HRV are among those considered to be simple yet strong indicators of cardiometabolic health (Hanley et al., 2002). Resting HR is a method, like HRV, of determining ANS function and balance of the CNS (Lahiri, Kannankeril, & Goldberger, 2008). An elevated resting HR is associated with increased SNS activity and decreased PNS activity (Lahiri et al., 2008). A high RHR has also been shown to be independently associated with developing metabolic syndrome (Hanley et al., 2002; Rogowski et al., 2009). Furthermore, increases in resting HR over a 2 year span were able to predict the incidence of diabetes 10 years later (Kim et al., 2017). Liu et al. (2016) found a linear relationship between resting HR and the risk of metabolic syndrome; for every 10 beats-per-minute increment, the risk of metabolic syndrome increased by 28%. Due to the interrelatedness of cardiometabolic diseases, measures of cardiovascular function which indicate dysfunction of the ANS, should be taken into consideration when assessing the risk of cardiometabolic disease.

Resting blood pressure. Resting BP is another simple measure, which can be used to evaluate ANS function and predict cardiometabolic disease. Elevated SNS activity induced by obesity is one proposed cause of hypertension (Elser et al., 2006; Landsberg, 2004). Although there are many reasons for increasing BP, the association between adiposity and hypertension has been well documented (Kotsis et al., 2005; Rahmouni, Correia, Haynes, & Mark, 2005). Comorbidities, obesity, and hypertension have been shown to increase the risk of a cardiovascular event (Landsberg et al., 2012). New evidence has shown that insulin sensitivity is a potential moderator for hypertension (Zhang et al., 2016). Obese individuals with insulin resistance are more likely to develop hypertension than those who are more insulin sensitive (Zhang et al., 2016).

Literature Review Summary

Despite the necessity of physical activity for regulation of a variety of essential bodily functions, preventing disease, and staving off mortality, the majority of individuals fail to meet weekly exercise recommendations due to a lack time (Colberg et al., 2011; Colley et al., 2011; CSEP, 2013; Hallal et al., 2012; Haskell et al., 2007; Reichert et al., 2007; Stutts, 2002). Physical inactivity and sedentary behaviours resulting in a diminished exercise capacity are a major public health concern (Lee et al., 2011). High-intensity interval training is a time-efficient alternative or adjunct to MICT (Batacan et al., 2017; Jelleymen et al., 2015; Weston et al., 2013); although, many existing protocols are perceived as too intense and time-consuming for a physically, inactive populations to perform (Gibala & Howley, 2009). Additionally, these protocols are not based in any physiological merits; shorter and less frequent intervals provide a comparable or better increase in VO_{2max} than traditionally used HIIT (Vollaard & Metcalfe, 2017; Vollaard et al., 2017). Therefore, the effects of a reduced-exertion HIIT on cardiovascular and metabolic risk factors should be made evident so as to provide justification for the recommendation of this protocol.

Research Question

The following research question was used to guide this study:

What are the effects of 6 weeks of reduced-exertion HIIT on anthropomorphic measures (body mass, body fat %), cardiovascular health (resting HR, HRV, resting BP), metabolic health (fasting blood glucose), and physical fitness (predicted VO_{2max}, peak power, mean power) in healthy but physically inactive individuals?

Chapter 3 - Methodology

Participants

Participants were defined as physically inactive by scoring in the *low* category on the International Physical Activity Questionnaire (IPAQ; Craig et al., 2003) and were deemed as healthy to participate in exercise by the Get Active questionnaire (Appendix A). Participants were excluded if they had a personal history of cardiovascular, metabolic, or neurological disease (e.g., previous cardiac event, T2D, metabolic syndrome, Parkinsons Disease, Alzheimer's Disease). Participants were also be excluded if they had hypertension (>140/90 mm/Hg), were a current smoker, taking medications for hormone replacement therapy, glycemic control, or cardiovascular conditions, or for any other reason that would prevent them from completing the testing procedures or intervention (e.g., musculoskeletal injury).

Participant Recruitment

Ethical approval was obtained from the Lakehead University and Thunder Bay Regional Health Sciences Centre Research Ethics Board. A combination of convenience, purposive, and snowball sampling was used to recruit 9 participants (6 women, 3 men) for this study. Potential participants were recruited through postings on Facebook or were informed of the study by directly speaking to individuals currently attending Lakehead University, or residents of Thunder Bay, Ontario. Posters and information handouts were also distributed at Lakehead University and at several workplaces around the city (Appendix F and G). These workplaces were selected by direct communication with potential participants. The researcher briefly explained the purpose of the study to prospective participants and how it benefited present and future research. Individuals who decided to participate were given an information letter and eligibility questionnaire. Upon completion of the questionnaire, eligible participants were asked to fill out an informed consent form and the IPAQ (Appendix C), Get Active Questionnaire (Appendix A), and a maximal effort test form (Appendix B) before the commencement of any testing procedures.

Experimental Design

This study used a single group pre-test post-test repeated measures quasi-experimental design with a 2 week run-in period. All participants were assessed at three time points (baseline, pre-test, and post-test) and were completed within 1 week at approximately the same time of the day. A run-in period consisting of 2 weeks before pre-testing in which participants were instructed not to deviate from their normal lifestyle was used to establish baseline physiological values to be used as a control. During the run-in and treatment periods, participants were sent biweekly e-mails to remind them of the influence of confounding variables such as changing their diet and physical activity level. During each of the testing times, participants were assessed for body mass, body fat %, resting HR, resting BP, resting HRV, predicted VO_{2max}, lower body power, and fasting blood glucose. Participants who exhibited significant lifestyle changes were excluded from the primary data analysis on a case by case basis although all participants were included in the ITT analysis. Post-test was conducted at least 24 hours to up to 1 week following the completion of the intervention. Changes in group means between the pre-test and post-test as well as the baseline and pre-test compared to post-test time points were used to evaluate the effect of the treatment.

Run-in period. The 2 week run-in period allowed participants to be their own controls foregoing a control group to allow greater recruitment to the treatment group. During this time, participants did not receive any treatment and were given the same instructions concerning the influence of confounding variables as a result of significant lifestyle modifications. Participants were sent bi-weekly e-mails to check in with participants to record any protocol deviating behaviours and to gently remind them to maintain their normal lifestyle from before the start of the study. A protocol deviating behaviour was defined as any behaviour that could potentially influence the results of any of the current testing measures or a behaviour that considerably deviated from normal lifestyle behaviour. Protocol deviating behaviours included engaging in an amount of exercise that would no longer define the participant as physically inactive or sedentary (for more than 1 week), a change in diet which would significantly increase or decrease sugar intake, and starting a weight loss program. Protocol deviation was monitored through e-mail and direct correspondence with the participants during the data collection period.

Testing Procedures

All testing and exercise was completed in the School of Kinesiology exercise physiology lab (SB-1025) at room temperature (19º C) and at approximately the same time of day. Participants were instructed to refrain from consuming caffeine 6 hours before testing (Bell & McLellan, 2002; Ryu et al., 2001), and were verbally instructed on potential dietary caffeine sources to avoid. Participants were asked to avoid vigorous physical activity 24 hours before testing and refrain from food or drink at least 1 hour prior. Vigorous intensity physical activity was defined as physical activity in which the

HR substantially increased, body temperature increased, and the individual was not able to say more than a few words before taking a breath (CSEP, 2013). This degree of exercise intensity corresponded to a perceived exertion of 7 to 9 out of 10 on the original Borg scale (Borg, 1998; CSEP, 2013). Participants who failed to comply with these standards were rescheduled to a later date less than seven days after the completion of the treatment.

Body mass. For this study, body mass was measured at all three time points (baseline, pre-, and post-test) to observe changes in participants that could indicate a deviation from normal lifestyle behavior. If a participant significantly changed his/her body mass between time points, it may have been a reason for exclusion from the data analysis as he/she may have engaged in a weight loss program. Significant body mass changes were red flags for protocol deviation because it was not consistent in the literature for this volume of exercise to result in significant anthropomorphic changes. All testing was conducted at approximately the same time of day for each participant to control for daily body mass fluctuations. When the participant arrived at the lab (SB-1025), he/she was instructed to remove his/her shoes and any excess clothing and then to step on the same calibrated digital scale to have his/her body mass recorded (kg).

Body fat percentage. Body fat % was estimated by using bioelectrical impedance with an OMRON HBF-306CAN fat loss monitor (Burlington, ON). The researcher inputted the participant's height (cm), body mass (kg), age, and body type into the bioelectrical impedance device. The participant then removed any additional clothing and any large metal jewelry (e.g., metal band watches). He/she then held the device ensuring adequate skin coverage of the metal surfaces; the researcher then began the measurement and recorded the results.

Heart rate variability. A time-domain measurement of HRV was assessed by three lead ECG and processed by ADInstruments© HRV module using the LabChart© 9 interface. Heart rate variability was operationalized by the standard deviation of the average R to R interval (ms) of normal heartbeats (SDRR) and, additionally, as the root mean squared of successive differences between normal heartbeats (RMSRR). Ectopic beats were excluded. Once the ECG was connected, the participant then laid on a massage table in a supine position for 5 minutes then the 10 minute data collection period began. The participant was instructed to relax and breathe at a consistent and comfortable rate for the data collection period and to avoid any unnecessary movement while the researcher left the room.

Resting heart rate. Resting HR was operationalized by the mean HR during a 10 minute resting period in a supine position. This data for resting HR was extracted using the same procedures for HRV measurement.

Resting blood pressure. An auscultatory measurement of resting BP was taken using the Korotkoff method with an aneroid sphygmomanometer (AMG Medical Inc., Montreal, QC) and a stethoscope (AMG Medical Inc., Montreal, QC). Guidelines by Pickering et al. (2005) were modified to have the participant lying in a supine position. Some factors have been identified as influencing BP measurement including room temperature, exercise, alcohol, and nicotine, arm/body position, muscle tone, bladder distension, talking, and background noise (Pickering et al., 2005). Participants were instructed not to consume alcohol or nicotine as well as to avoid exercise before testing to control for potential confounding variables. Participants were also instructed to remove any clothing that covered the location of the cuff placement and to remain supine with the legs uncrossed for at least 10 minutes before the first reading was taken (Pickering et al., 2005). Participants remained in a supine position for at least 15 minutes before their BP was recorded as this took place following the HRV measurement. Additionally, the researcher instructed the participant not to talk during the measurement as this could cause measurement error. A second reading was taken 1 minute apart to confirm the results of the first reading.

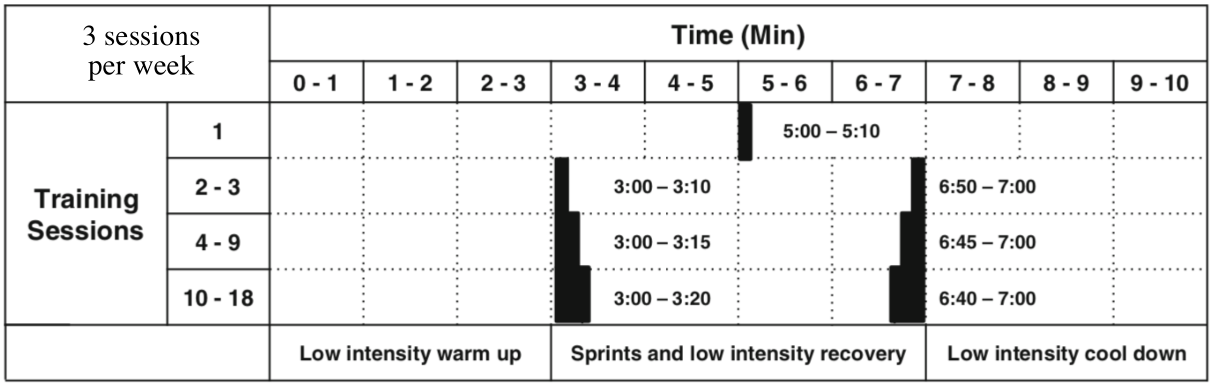
Predicted aerobic capacity. The predicted VO_{2max} of each participant was determined by the EB test (Ekblom-Bak et al., 2016), using a Monark 828E mechanically braked cycle ergometer (Vansbro, Sweden). The EB-test began by the participant cycling for 4 minutes at a standard work rate of 0.5 kp (the same for all participants) and pedaling at 60 rpm. During the final minute, HR was recorded every 15 seconds (four total) to produce a mean HR for that workload. After a 2 minute rest period, the participant was asked to self-select a higher work rate which corresponded to an RPE of 14 (Borg, 1998). Then a second 4 minute bout was performed at the higher work rate repeating the same procedure for recording the HR. The predicted VO_{2max} was then calculated based on age, sex, and relative changes in HR and work rate. The revised Ekblom-Bak equation is the natural logarithm of [VO_{2max} men = 2.04900 - 0.0058*(age) - 0.90742*(ΔHR/ΔWorkload) + 0.00178*(ΔWorkload) - 0.00290*(HR at standard workload); VO_{2max} women = 1.84390 - 0.00673*(age) - 0.62578*(ΔHR/ΔWorkload) + 0.00175*(ΔWorkload) - 0.00471*(HR at standard workload)] (Bjorkman et al., 2016).

Anaerobic power. Participants had their peak and mean power assessed at baseline, pre-, and post-test as an additional parameter to identify if the protocol elicited a change in lower body power. As a secondary research purpose, anaerobic power was measured to determine if a relationship existed between anaerobic power and aerobic capacity. Anaerobic power was assessed using a 15 second maximal exertion cycle ergometer sprint using 7.5% resistance of body mass (kg) on a mechanically braked Monark 894e cycle ergometer (Vansbro, Sweden). The seat height was adjusted for each individual so that when seated there was a slight bend in the knee when the pedal was in the lowest position. The participant was also instructed to remain seated for the duration of the test. Testing commenced 5 minutes after the predicted VO_{2max} test, although participants could take more time, if required. Before the first test each participant performed a familiarization trial to mitigate the learning effect of the weight basket drop which was particular to the type of cycle ergometer used.

Fasting blood glucose. Fasting blood glucose was assessed at baseline, pre-, and post-test. The participant arrived at the lab in the morning, following a 10 hour fast, during which time he/she was instructed not to ingest anything by mouth except for water. For example, the fasting period consisted of a 10 hour window following the participant’s last meal of the day and before their first meal the following morning (e.g. bedtime snack 10 p.m., fasting BG measurement after 8 a.m. the next day). Upon arrival at the lab, the compliance of the 10 hour fast was confirmed. In the event of noncompliance, the test was rescheduled within 7 days of the original test day. To obtain the sample, the participant was positioned in a seated position and then a fingertip of his/her choosing was sanitized. The participant then self-administered a finger prick to collect a capillary blood sample. The lancet containing the blood sample was then inserted into a Bayer Contour® Next glucometer (Mishawaka, IN) to record the fasting blood glucose (mmol/L).

Intervention

All participants completed 6 weeks of a supervised reduced-exertion HIIT protocol using the protocol as described by Metcalfe et al. (2012) and Metcalfe et al. (2016; Table 1), following a 2 week run-in period. The protocol was performed on either a Monark 894E or 828E cycle ergometer (Vansbro, Sweden). Participants were required to complete three 10 minutes sessions per week (18 sessions), with an adherence rate of at least 75% (14 of 18 sessions) for inclusion into the secondary data analysis. All participants were included in the primary analysis, using the intent to treat (ITT) principle. The participant wore a HR monitor to provide feedback on the level of intensity (FT2, Polar® Electro Canada, Lacine, QC). Each participant's HR_{max} was based on an age predicted maximum using the Tanaka method (HR_{max}=208–0.7 *age; Tanaka, Monahan, & Seals, 2001). During high-intensity sprints, the participant was encouraged to achieve a HR of at least 85% of HR_{max} and maintain an intensity between 50% and 75% of HR_{max} during active recovery. Each participant was given a card with written and visual instructions with the target HR ranges (Appendix D). Due to the latency period between the initiation of activity and compensatory action of the heart, feedback based on HR for sprints was only given as a guideline. The participant was verbally instructed to elicit an intense or all-out effort and manually increase the pedal resistance between 5-7.5% body weight resistance (Metcalfe et al., 2012; 2016). During active rest, the participant was instructed to maintain a comfortable pace with the intent to sufficiently recover within the allotted time to perform the subsequent sprint at maximal effort without pacing due to fatigue. The participant was permitted to exercise below the controlled intensities during the recovery periods should they experience any discomfort, nausea, or dizziness. Each training session consisted of a 3 minutes low-intensity (50-70% of HR_{max}) warm-up and cool-down and one (1st session only) or two (all other sessions) high-intensity cycling sprints (>85% of HR_{max}).The duration of the sprints was 10 seconds in week one, 15 seconds in weeks two and three, and 20 seconds in weeks four to six. There was 3 minutes and 40 seconds of active rest between the first and second 10 seconds interval. For subsequent weeks, when the interval time increased to 15 and 20 seconds, active rest time reduced to 3 minutes and 30 seconds, and 3 minutes and 20 seconds, respectively, to allow all sessions to be equal in duration (Table 1).



3 sessions per week		Time (Min)									
		0 - 1	1 - 2	2 - 3	3 - 4	4 - 5	5 - 6	6 - 7	7 - 8	8 - 9	9 - 10
Training Sessions	1						▮ 5:00 – 5:10				
	2 - 3				▮ 3:00 – 3:10			▮	6:50 – 7:00		
	4 - 9				▮ 3:00 – 3:15			▮	6:45 – 7:00		
	10 - 18				▮ 3:00 – 3:20			▮	6:40 – 7:00		
		Low intensity warm up			Sprints and low intensity recovery				Low intensity cool down		

▮ = *High intensity sprints*

Table 1. This table shows the progression of sprint times over the duration of the exercise protocol.

Lifestyle management. To aid in controlling for the potential confounding variables which arise from individuals deviating from their normal lifestyle, e-mail reminders, and short questionnaires were sent out bi-weekly to participants. This correspondence was meant to encourage participants to maintain a normal lifestyle and refrain from making lifestyle changes that could potentially influence the outcome of the study. Participants were informed of the potential influence deviant behaviours could have on the results of the study in hopes of encouraging cooperation. Such changes included adopting a new diet, adding a new supplement/ergogenic aid into a diet, or starting a new exercise routine. The correspondence between the researcher and participants was used to determine if the participants had engaged in any behaviour deviating from the protocol.

Data Analysis

The primary data analysis followed the ITT principle. The concept of the ITT principle, is to perform the analysis based on the group the participant was randomized to irrespective of noncompliance, protocol deviation, or withdrawal (Gupta, 2011). Although the study was not a randomized controlled trial, the purpose of the use of the ITT concept was to replicate real world conditions, improving the external validity. All data were analysed using the Statistical Package for the Social Sciences® Version 24 (SPSS Inc., Chicago, IL). Measures of centrality and spread were presented as means ± standard deviations.

All dependent variable data were assessed for normality, sphericity, and significant outliers. The differences in group means between assessments (baseline, pre-, and post-treatment) periods for body mass, body fat %, resting HR, resting systolic BP, resting diastolic BP, SDNN, RMSSD, predicted VO_{2max}, peak power, mean power, and fasting blood glucose (Table 2), were analyzed using a one-way repeated measure analysis of variance (ANOVA). A planned comparison post-hoc test was conducted to analyze the individual group mean differences which achieve a significance of $p<.05$.

The planned comparisons included comparisons between baseline and pre-test, pre-test and post-test, and baseline and post-test. A two-tailed alpha level of .05 was split to .01667 for the three planned comparisons.

Table 2. This table shows the variables and units used to measure the effects of the exercise protocol.

Category	**Variable**	**Units**
Anthropomorphic measures	Body mass	kg
	Body fat %	%
Cardiovascular health	Resting HR	bpm
	Resting HRV - SDNN	ms
	Resting HRV - RMSNN	ms
	Resting BP - Systolic	mm/Hg
	Resting BP - Diastolic	mm/Hg
Physical fitness	Predicted VO_{2max}	L/min
	Peak power	W/kg
	Mean power	W/kg
Metabolic health	Fasting blood glucose	mmol/L

Chapter 4 - Results

Nine participants (3 males and 6 females) with an average age of 27.8±6.6 years participated in the study. All participants met the minimum amount of training sessions (14 out of 18); only one participant completed all 18 training sessions, with a mean adherence rate of 16 out of 18 sessions (89%). All participants maintained similar physical activity habits and did not engage in any meaningful deviations from the study protocol, as indicated by the responses to the bi-weekly surveys.

What are the effects of a reduced-exertion HIIT protocol on anthropomorphic measures, cardiovascular health, metabolic health, and physical fitness in physically inactive individuals?

A repeated measures ANOVA was used to detect changes in participants before and after a 2 week run-in period (baseline and pre-test) and again following 6 weeks of exercise (post-test). All data were evaluated against the assumptions of the repeated measures ANOVA test. The assumptions of sphericity and normality were evaluated by Mauchly's Test of Sphericity and the Shapiro-Wilk Test, respectively. The presence of outliers at each time point was evaluated using boxplots; data points greater than 1.5 standard deviations away from the mean were considered outliers. Outliers were left in the data unless they were legitimate measurement errors. All data met the assumptions of the repeated measures ANOVA at a level of α=.05, unless subsequently noted.

The data for body mass and body fat % contained one outlier at each time point, which was not removed as these outliers were considered to be genuinely unusual data points considering the small sample size. The resting HR data had an outlier at the pre-test time point, which was removed to meet the assumption of normality as it would not have a meaningful effect on the result of the test. The data for mean power had two outliers. The same participant had an outlier at the baseline and the post-test time point, respectively; the outliers were removed because the participant did not sufficiently adhere to the test protocol.

Anthropomorphic Measures

The assumption of sphericity was not met for measurements of body mass (kg), as assessed by Mauchly's Test of Sphericity, $\chi2(2)=7.19$, *p=.027*. Epsilon (ε) was .61, as calculated according to Greenhouse and Geisser (1959), and was used to correct the one-way repeated measures ANOVA. The reduced-exertion HIIT protocol did not elicit statistically significant changes in body mass over time, *F*(1.22, 9.74)=1.27, *p=.30*, with body mass increasing from baseline (M=83.46, SD=9.21 kg) to pre-test (M=83.78, SD=9.21 kg) to post-test (M=84.04, SD=9.45 kg).

The assumption of sphericity was not met for the body fat % data, as assessed by Mauchly's Test of Sphericity, $\chi2(2)=7.55$, *p=.023*. Epsilon was .60, as such a Greenhouse and Geisser (1959) correction was applied to the data. The reduced-exertion HIIT protocol did not elicit statistically significant changes in body mass over time, *F*(1.20, 9.64)=.91, *p=.38*, with body fat % decreasing from baseline (M=28.94, SD=3.27%) to pre-test (M=28.06, SD=3.63%) to post-test (M=27.51, SD=3.08%).

Cardiovascular Measures

The exercise intervention did not elicit statistically significant changes in SDNN time, *F*(2, 14)=.24, *p=.79*, with SDNN slightly decreasing from 58.95 ± 6.18 ms at baseline to 57.67 ± 8.04 ms at pre-test, and increased to 64.37 ± 5.52 ms at post-test.

The exercise intervention did not elicit statistically significant changes in RMSSD over time, *F*(2, 14)=1.02, *p=.39*, with RMSSD increasing from 50.07 ± 7.91 ms at baseline to 69.08 ± 16.83 ms at pre-test and increased to 69.98 ± 13.12 ms at post-test.

The reduced-exertion HIIT protocol did not elicit statistically significant changes in RHR over time, *F*(2, 12)=.74, *p=.50*, with RHR decreasing from 70.58 ± 3.51 bpm and 70.73 ± 2.47 bpm at baseline and pre-test, respectively, to 66.04 ± 1.51 bpm at post-test.

Resting systolic blood pressure decreased over time, *F*(2, 16)=7.85, *p=.004*, partial η2=.50, decreasing from baseline (M=121, SD=2 mm/Hg) and pre-test (M=117, SD=3 mm/Hg) to post-test (M=115, SD=2 mm/Hg) as shown Figure 1. Planned contrasts showed that there was a statistically significant decrease from baseline to post-test (*p=.004*) with a mean difference of -5, 95% CI [-10, 1] mm/Hg. Planned contrasts between baseline and pre-test and pre-test and post-test revealed no statistically significant differences. There was a mean difference of -4, 95% CI [-10, 1] mm/Hg (*p=.05)* between baseline and pre-test and a mean difference of -1, 95% CI [-5, 2] mm/Hg (*p=.24)* between pre-test and post-test.

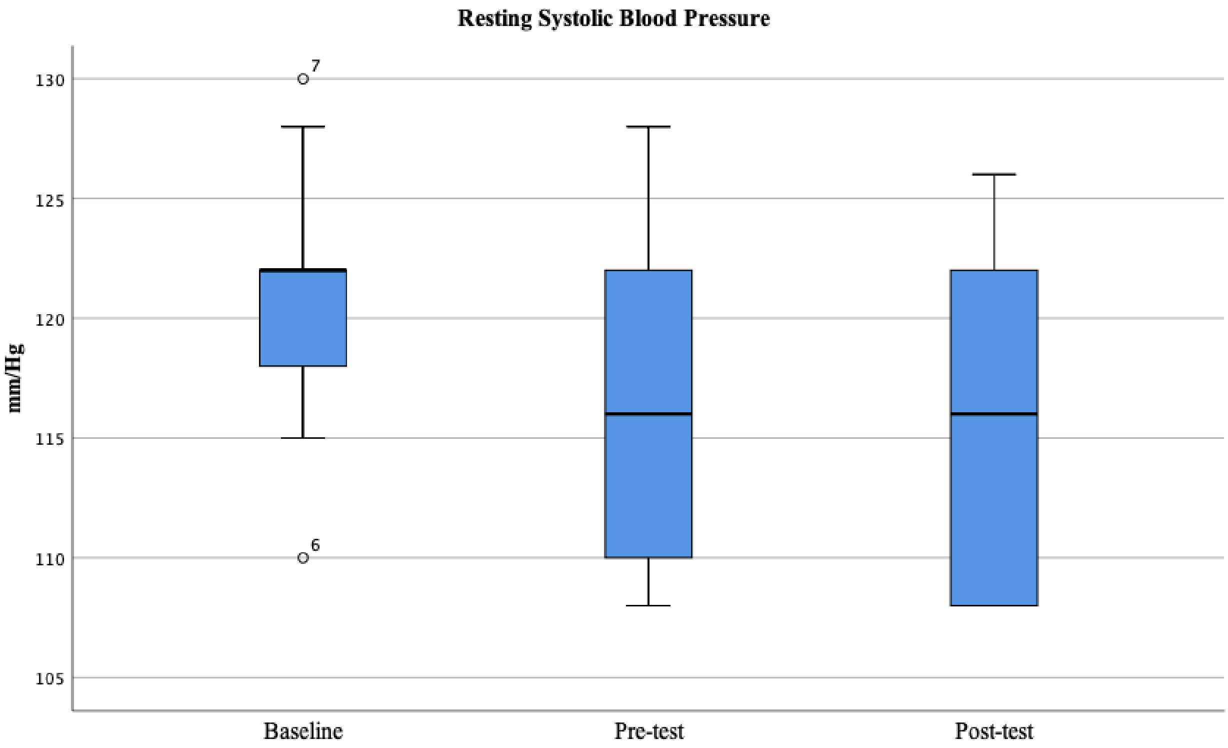


Figure 1. Boxplot, including outliers of resting systolic blood pressure. This figure shows the marginal mean estimates of resting systolic blood pressure over time.

The exercise intervention did not elicit statistically significant changes in diastolic blood pressure over time, *F*(2, 16)=1.65, *p=.22*, with diastolic blood pressure decreasing from 72 ± 2 mm/Hg at baseline to 70 ± 2 mm/Hg at pre-test and decreased further to 69 ± 2 mm/Hg at post-test.

Physical Fitness

The reduced-exertion HIIT protocol elicited statistically significant changes in predicted VO_{2max} over time, *F*(2, 16)=6.33, *p=.009*, partial η2=0.44, increasing from baseline (M=2.67, SD=.21 L/min) and pre-test (M=2.69, SD=.22 L/min) to post-test (M=2.84, SD=.18 L/min) as shown in Figure 2. Planned contrasts showed there was no statistically significant difference in VO_{2max} between baseline and pre-test (*p=.49*) with a mean difference of .02, 95% CI [-.06, .10] L/min. There was also no statistically significant difference between pre-test and post-test (*p=.03*) with a mean difference of .15, 95% CI [-.03, .33] L/min. There was also no statistically significant mean difference between baseline and post-test (*p=.03*) with a mean difference of .17, 95% CI [-.02, .36] L/min.

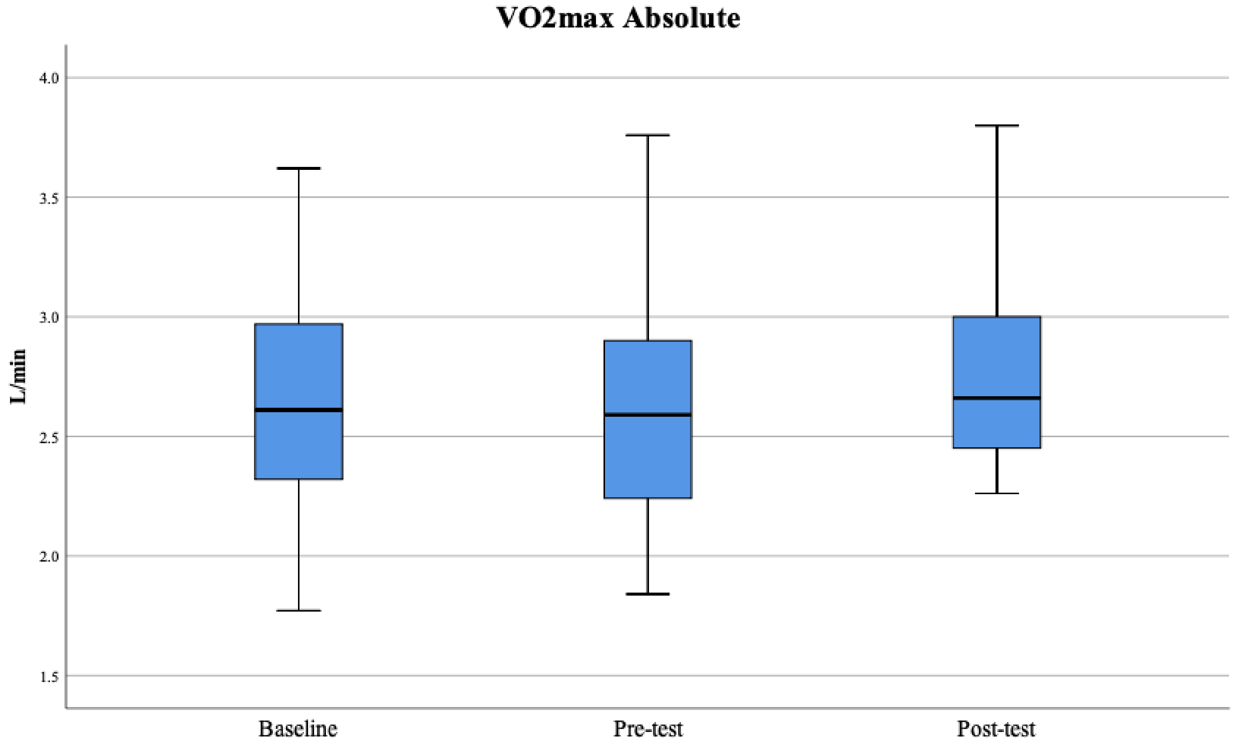


Figure 2. Boxplot of the estimated marginal means of predicted VO_{2max}.

Peak power statistically significantly increased over time, *F*(2, 16)=10.84, *p=.001*, partial η2=.58, with peak power increasing from baseline (M=6.92, SD=.40 W/kg) and pre-test (M=7.02, SD=.40 W/kg) to post-test (M=7.71, SD=.43 W/kg) as shown in Figure 3. Planned contrasts revealed that there was no difference in means of peak power between baseline and pre-test (*p=.64*) with a mean difference of .10, 95% CI

[-.54, .75] W/kg. There was a statistically significant mean difference between pre-test and post-test (*p=.002)* of .68, 95% CI [.22, 1.14] W/kg. There was also a statistically significant mean difference in planned comparison between baseline and post-test (*p=.002*) of .79, 95% CI [.25, 1.32] W/kg.

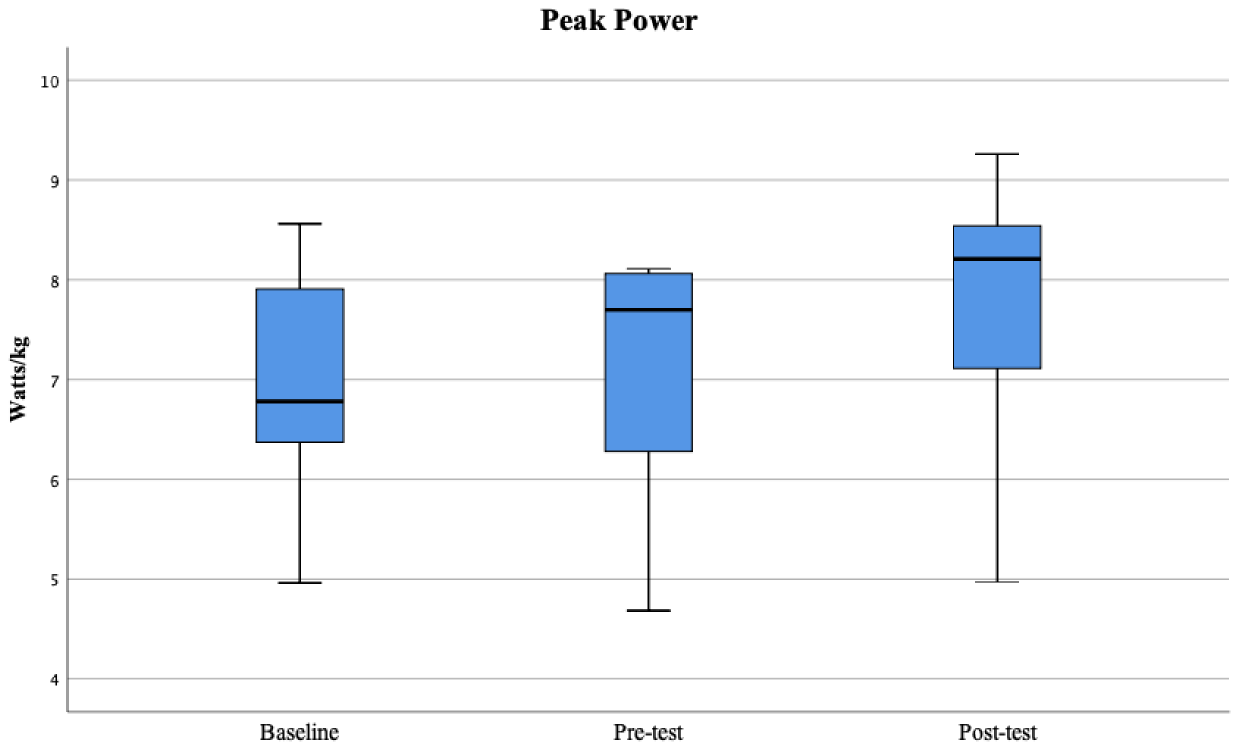


Figure 3. Boxplot of estimated marginal means of peak lower body power over time.

The reduced-exertion HIIT protocol elicited a statistically significant increase in mean power over time, *F*(2, 14)=20.89, *p=.00006*, partial η2=.75, with peak power increasing from baseline (M=5.73, SD=.27 W/kg) and pre-test (M=5.78, SD=.30 W/kg) to post-test (M=6.60, SD=.27 W/kg) as shown in Figure 4. Planned contrasts showed that there was no difference in mean power between baseline and pre-test (*p=.80*) with a mean difference of .04, 95% CI [-.44, .52] W/kg. There was a statistically significant mean difference between pre-test and post-test (*p=.0004*) of .82, 95% CI [.41, 1.23]

W/kg. There was also a statistically significant mean different planned comparison between baseline and post-test (*p=.001*) of .86, 95% CI [.35, 1.38] W/kg.

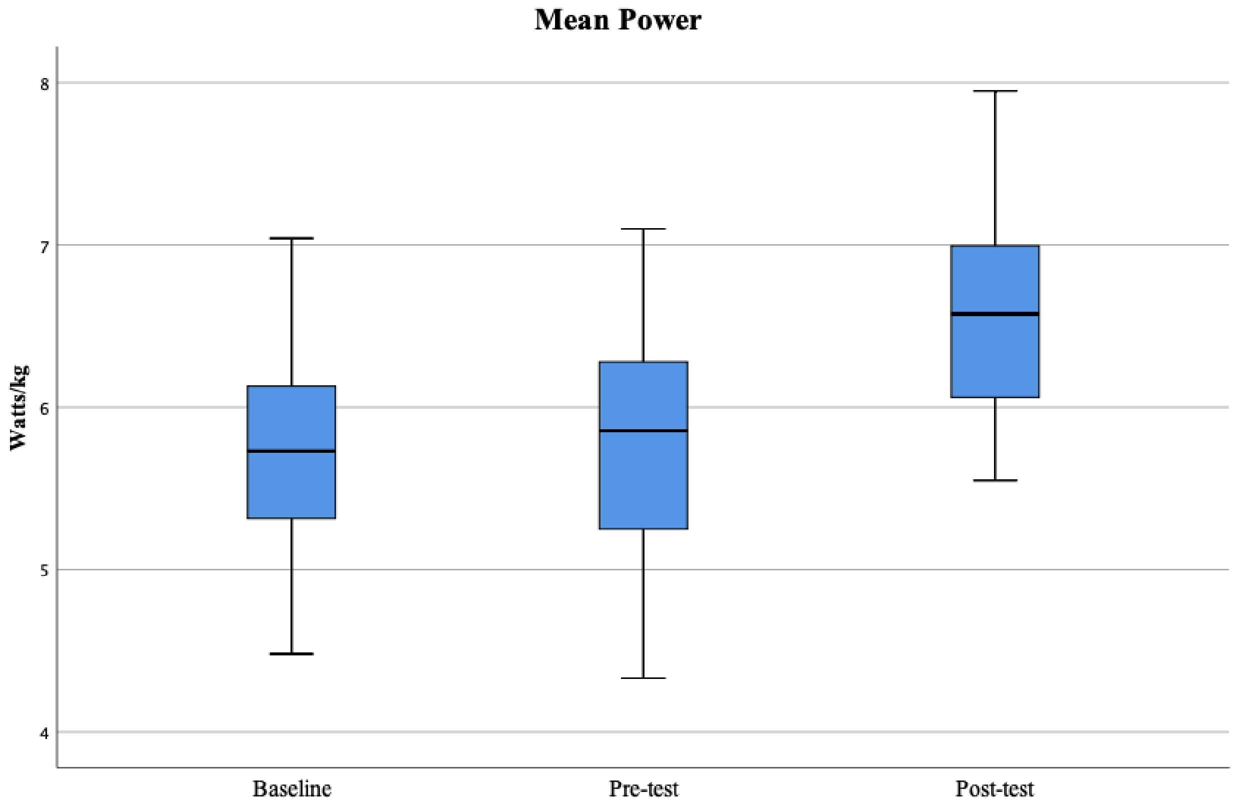


Figure 4. Boxplot of the estimated marginal means of mean lower body power over time.

Metabolic Health

The exercise intervention did not elicit statistically significant changes in fasting blood glucose over time, *F*(2, 12)=1.28, *p=.31*, with fasting blood glucose decreasing from 4.84 ± .15 mmol/L at baseline to 4.59 ± .18 mmol/L at pre-test and increased to 4.71 ± .14 mmol/L at post-test.

Chapter 5 - Discussion

In the present study, a 6 week high-intensity training protocol designed to minimize time commitment (30 minutes per week) improved peak power, mean power, and predicted VO_{2max} in physically inactive individuals. This study extended the previous literature showing the beneficial effects of HIIT despite a reduction in the number of sprints, and overall volume while still maintaining positive health effects. The current weekly exercise guidelines adopted almost universally by health organizations included 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity exercise (in bouts of 10 minutes or more). According to accelerometer data, only 15% of Canadians meet weekly exercise guidelines, most commonly due to a lack of time. This protocol elicits health benefits despite an 80% reduction in time commitment compared to the moderate-intensity recommendation, yet has the same perceived exertion on the modified Borg scale (*somewhat hard, or 14*; Borg, 1998) as moderate intensity exercise (50-75% VO_{2max}). From an epidemiological perspective, those who are the least physically active are disproportionately at greater risk for chronic disease. In terms of public health benefit, the greatest gain in chronic disease avoidance, would be to get those who are sedentary to do a minimal amount of exercise.

It would be difficult to argue that exercise does not have a positive health effect; however, a major societal health concern are chronic diseases fostered by a lack of physical activity. In general, more exercise within reason, is better, but based on observations of societal behaviours, a recommendation for more exercise is not likely to be adhered. One goal this research aimed to accomplish was to determine a minimal amount of exercise required to elicit health benefits in a physically inactive population.

The effects of minimal amounts of high-intensity in a reduced-exertion HIIT protocol in this study and in other works, help posit a minimal exercise recommendation. This may improve adherence to exercise if small amounts of exercise are perceived as beneficial rather than a waste of time.

In previous works by Metcalfe et al. (2012; 2016) which used a reduced-exertion HIIT protocol, sedentary men and women improved VO_{2max} despite no changes in anthropomorphic measurements such as body mass, BMI, and body fat %. One key difference is this study is that in place of an incremental exercise test to exhaustion, a predictive test was used due to equipment failure. This study observed a statistically significant mean increase in predicted VO_{2max} over time. Predicted VO_{2max} increased 6.8% from pre-test to post-test and 7.5% from baseline to post-test, although it did not reach statistical significance in the post hoc analysis despite having a large effect size (partial $\eta 2$=.442). This result can be attributed to the added variance from using a predictive VO_{2max} test and the statistical test being underpowered due to the small sample size. There is a higher margin for error inherent to any predictive test, which is the product of the means of prediction rather than not observing the measurement in its purest form. The improvement in predicted VO_{2max} due to the intervention was likely diluted by the added variance from the predictive test and was therefore not able to detect significant differences in the post hoc.

Metcalfe et al. (2016) observed a 15% and 12% increase in VO_{2max} in men and women, respectively. While similar observations were made in Metcalfe et al., (2012) who showed an ~10% increase and Cuddy et al., (2019) who saw a 12% increase in both men and women. Other studies which have examined the effects of similar amounts of exercise have also observed comparable improvements in VO_{2max} (Allison et al., 2017; Gillen et al., 2016; Jenkins et al., 2019). In all studies which implemented reduced-exertion HIIT the IPAQ was used to determine if the participant was physically inactive. As a result, this tool was able to allow the recruitment of participants with similar VO_{2max} compared to previous works. The mean predicted VO_{2max} of participants recruited here at baseline (2.7 L/min or 33.6 ml/kg/min) was similar to those recruited in Metcalfe et al. (2016) which observed 2.5 L/min and Metcalfe et al. (2012) which observed 36.3 ml/kg/min and 32.5 ml/kg/min in men and women at pre-test in the HIIT group, respectively. Although the participants recruited here appear to be in similar aerobic physical fitness to those recruited in previous works, it is possible that the method of prediction was not accurate for this sample and the participants' physical fitness was in fact higher than what was measured. Therefore, this protocol was not sufficient to elicit changes in VO_{2max}.

Another possible explanation for the lack of significant change in VO_{2max} is due to individual differences resulting in non-responders in the sample. Of the nine participants, all but one improved VO_{2max}, who exhibited a 1.6% decrease. Previous literature demonstrated that a certain percentage of a population will not respond to specific physiological adaptations following exercise (Vollaard et al., 2009). In a sample of this size (n=9), it would not take many non-responders to have an impact on the results of the data. The participant who decreased their VO_{2max} started with the highest VO_{2max} relative to body mass but was physically inactive and was an admitted *non-exerciser*. Perhaps for this individual, the prescribed exercise protocol was not sufficiently demanding to elicit a change in VO_{2max}, and the decrease we observed was likely due to measurement error (fluctuations in the prediction). One study described an interesting phenomenon in which only participants who increased VO_{2max} also improved high frequency power measurement of HRV despite identical training load as determined by HRV post-exercise (Nummela et al., 2010). Physiological adaptations are complex and are not uniform across the population. It is possible that those with different biological makeups (e.g., muscle fibre type or anatomical variations) will respond differently to metabolic stimulus.

Glycemic control adaptations to exercise are important physiological changes that may not be uniform across the population. Up to 40% of the population has been reported to be a non-responder for changes in insulin sensitivity following exercise (Vollaard et al., 2009). Although our sample did not exhibit elevated fasting blood glucose, it is unlikely we would not have detected any change with our sample size if it contained 40% non-responders. Esbjornsson-Liljedahl et al. (2002) described a phenomenon where women break down 50% less muscle glycogen compared to men during a Wingate Test. These sexual dimorphisms in glycogen breakdown may be responsible for the lack of change in insulin sensitivity observed here due to the sample being predominantly women (n=6) and in other similar studies (Jenkins et al., 2019; Metcalfe et al., 2012; 2016). A review by Jelleyman et al. (2015) concluded that HIIT improved insulin sensitivity, although the total amount of exercise volume performed in those studies exceeded a reduced-exertion protocol. It is difficult, especially with glycemic control, to reliably detect these adaptations even with the most methodologically sound design. Godkin et al. (2018) examined the effect of minimal amounts of high-intensity stair climbing on metabolic health in a population with T2D. Despite the participants being diabetic, physically inactive, and controlling dietary intake, no change in 24 hour blood glucose level was observed. This protocol had a duration of 6 weeks which is a similar duration to many others, but may not be long enough for these adaptations to develop. Only Gillen et al. (2016) examined a protocol similar to reduced-exertion HIIT over a long enough duration (12 weeks) to detect improvements in insulin sensitivity.

Participants recruited in this study on average did not exhibit an elevated fasting blood glucose. Only one participant on one occasion showed an elevated fasting blood glucose (>5.6 mmol/L). Therefore, changes in these levels may not necessarily represent an improvement in metabolic health. The glucose clamp technique is a more appropriate test for measuring changes in glucose and insulin functioning in individuals who do not exhibit unhealthy fasting blood glucose levels. The glucose clamp technique is a more dynamic test that can evaluate insulin sensitivity or glucose uptake of the whole body (DeFronzo, Tobin, & Andres, 1979). In contrast, the fasting blood glucose test only provided a single snapshot of blood glucose levels, which may have not provided a detailed enough account of an individual’s metabolic health (primarily represented the hepatic output of glucose). It would have been more appropriate to use a glucose clamp test, as these are the gold standard in glycemic function; however, it was not feasible to use the glucose clamp in the current study. Using this test in the present study would have been cost prohibitive for a masters project as there was no funding to pay for blood work. In addition, using a measurement that requires a phlebotomist would also incur further scheduling and recruitment problems.

The 30 second Wingate Test is considered the gold standard for measuring anaerobic fitness (Bar-Or, 1987). To measure the change in anaerobic fitness, a 15 second maximal cycle sprint was used, which replicated the sprint duration and resistance used during weeks two and three of the exercise protocol. A significant increase in peak power and mean power was observed with large effect sizes (partial $\eta 2$=.575 and .749), respectively. Based on the principle of specificity, these adaptations are to be expected. Since the gold standard VO_{2max} was not able to be used here, perhaps the best way to indicate the effectiveness of the treatment is to look at the most specific adaptation attributable to the protocol. This form of power measurement is not commonly used in any exercise intervention studies. If lower body power is measured it is usually the highest workload achieved during a cycle ergometer VO_{2max} test which does not accurately assess maximal power output as the participant is fatigued from previous workloads.

There was a statistically significant decrease in resting systolic BP over time; however, the planned comparison reveals that this change over time is due to the added variance from the elevated baseline measurement. The resting systolic BP baseline assessment (M=121, SD=2 mm/Hg) was higher than both other time points; considering that these time point had mean systolic BP of <120 mm/Hg, it is likely that the elevated baseline measurement is a result of anxiety or nervousness due to the participants having a novel experience. A similar phenomenon was observed in diastolic blood pressure, the baseline assessment was the highest (72 ± 2 mm/Hg), and the two preceding time points dropped slightly. Although the difference in the diastolic BP was not enough to achieve significance, this trend is likely due to the same reason as the systolic BP. Considering that the highest means of resting systolic and diastolic blood pressure we observed would combine to make a blood pressure of 121/72 mm/Hg, this is of minimal concern as is it only a few points away from a healthy blood pressure range. In the present study a decrease in blood pressure as a result of the exercise was not observed because, although participants were sedentary, they did not have elevated blood pressure. Therefore, decreases in resting blood pressure would be minimal at best as there would be no reason to expect a deviation from already healthy blood pressure. The reason we detected significance in the repeated measures ANOVA was likely due to the variance added by the elevated BP measurements at the baseline assessment.

Cardiovascular changes in the BP or resting HR take longer time or more exercise volume to manifest compared to changes in the peripheral skeletal system. Metcalfe et al. (2012; 2016) did not observe changes in resting blood pressure over the 6 week protocol. Cuddy et al. (2019) lengthened the protocol duration to 8 weeks, this was sufficient to detect a modest 5% reduction in systolic BP. When comparing HIIT studies of low-volume and high-volume HIIT, studies which used low-volume HIIT were usually 6 weeks in length did not show changes in cardiovascular health (Fisher et al., 2015) almost all studies which used longer durations (e.g., 12-16 weeks) showed reductions in blood pressure (Mitranum et al., 2014, Nybo et al., 2010; Tjonna et al., 2008). Interestingly, in studies which compared HIIT to MICT following a longer protocol duration it was difficult to determine which method of exercise was more beneficial. These findings showed that comparable changes in the cardiovascular system may take longer to occur in HIIT.

No changes in either SDRR or RMSSD measurements of HRV were observed, which is likely because HRV is affected by multiple confounding variables that are difficult to control. Previous literature on HRV typically has not included it in HIIT studies. Since HRV is derived from the balance of the PNS and SNS, it is easily affected by a copious amount of external factors. Simply put, HRV is highly sensitive to an individual's stress, what he/she ate, and even his/her body position that it would not be feasible to control for all of the external factors, making this a difficult measure to use in this context. A review by Villafaina et al. (2017) concluded that exercise interventions improved HRV in individuals with T2D. These studies, however, all used greater exercise volumes compared to reduced-exertion HIIT and sometimes combined resistance training (Villafaina et al., 2017). This sensitivity to change can be valuable to diagnose illness (Tsuji et al., 1996) or to assess training load (Kaikkonen et al., 2010) in appropriate settings, but this same sensitivity makes it hard to detect any change due to the intervention. Heart rate variability adaptations may take longer to occur, as such with other cardiovascular health markers, therefore, the study duration may not have been long enough to elicit change. While there may be a reason to study chronic changes in HRV following exercise interventions, future studies will need to make methodological changes or only use HRV when the sample size is large enough.

Limitations

A major limitation of the current research was the small sample size (n=9). A larger sample size may have more likely led to a statistically significant finding in the predictive VO_{2max} post hoc comparison. The added variance from a predictive test would require a larger sample to detect the same change in mean. Another limitation due to the small sample size there was there were not enough participants of each sex to analyze sex differences. One of the challenges of achieving a large enough sample is recruitment. Perhaps unsurprisingly, it is challenging to recruit a physically inactive population to participate in an exercise intervention study. While only 9 participants were recruited, this was disappointing considering the number of individuals who were contacted and expressed interest in the study. Recruitment efforts were made in the kinesiology, psychology, and sociology programs at Lakehead University, although only two students from kinesiology participated. Several other groups across Thunder Bay were also contacted such as the bowling and curling community, although this may not have been an effective strategy as these individuals may not perceive themselves as physically inactive given that they participated in recreational sports.

Future research

To confirm that a reduced-exertion HIIT protocol can cause chronic adaptations, future studies should measure the effects post-treatment several weeks after the completion of the exercise protocol to see if the adaptations were maintained. Currently, only Burgomaster et al. (2005) has demonstrated that physiological adaptations can be maintained several weeks post-exercise cessation following a SIT protocol. Therefore, it is promising that a reduced-exertion HIIT and other HIIT protocol will elicit similar chronic adaptations.

The current study protocol is based on the notion that a 20-30% glycogen depletion is primarily responsible for the physiological adaptations observed following high-intensity exercise. Studies that use muscle biopsies could confirm if the current protocol can meet this threshold for glycogen depletion. While a glycogen mechanism of action is suspected to be the primary driver of physiological adaptations, studies should confirm and detail its function using the reduced-exertion HIIT protocol. Of particular interest should be to compare sex difference in glycogen breakdown, as women have been observed to breakdown 50% less muscle glycogen during a Wingate Test (Esbjornsson-Liljedahl et al., 2002). If there are meaningful differences in the rate of glycogen breakdown between sexes yet no difference in physical fitness adaptations, then this does not support the glycogen breakdown threshold as a mechanism of action.

Certain types of HIIT, such as aerobic interval training, which have higher exercise volume, also use much longer protocol durations compared to reduced-exertion or SIT. Aerobic interval training has been shown to improve cardiovascular risk factors such as resting HR and resting blood pressure, which are not commonly observed in reduced-exertion HIIT or SIT. Future research should observe lower volume HIIT protocols over greater periods (>6 weeks) to determine its effect on cardiovascular risk factors. It is possible that the effects of these protocols on the cardiovascular system have not been appropriately measured because those physiological changes take longer to occur.

Aerobic HIIT is typically studied in a population with CVDs, conversely, SIT, which is based on the Wingate Test, has its roots in performance and is typically only studied in a healthy population. Performing HIIT in a population with CVDs has been shown to have minimal additional risk compared to MICT (Wewege, Ahn, Yu, Liou, & Keech, 2018). Future research should examine the effects of HIIT protocol in a population with CVD, which use shorter, more intense high-intensity periods such as SIT or reduced-exertion HIIT over longer durations (>6 weeks) to determine the effects on cardiovascular health in this population.

Conclusion

A reduced-exertion HIIT protocol of only 30 minutes of exercise per week is a vast reduction from the 150 minutes of moderate intensity and 75 minutes of vigorous intensity of exercise per week recommended by major public health organizations. Reducing the time of moderate and vigorous exercise intensity has the potential to overcome barriers to exercise and, thus, improve activity uptake by more of the population. Moreover, reduced-exertion HIIT has no more than 2 minutes of high-intensity exercise per week, yet despite only a minimal amount of exercise this protocol has demonstrated improved physical fitness in a physically inactive population. The observed effects of the reduced-exertion HIIT protocol in the present study concur with previous observations about the health benefits of minimal amounts of high-intensity exercise. Reduced-exertion HIIT protocol is an effective method of exercise for improving physical fitness (predicted VO_{2max}, peak power, and mean power) with minimal time-commitment and exercise volume. These findings suggest that reduced exertion HIIT is a valuable adjunct or alternative method of exercise for those who are physically inactive and has the potential to mitigate chronic disease risk. From a public health perspective, it would be most beneficial for those who are least physically inactive to do at least a minimal amount of exercise. Public health organizations should recommend this form of exercise as this could have meaningful benefits in a physically inactive population.

References

Allison, M. K., Baglole, J. H., Martin, B. J., Macinnis, M. J., Gurd, B. J., & Gibala, M. J (2017). Brief intense stair climbing improves cardiorespiratory fitness. *Medicine & Science in Sports & Exercise*, *49*(2), 298-307. doi:10.1249/MSS.0000000000001188

American Thoracic Society. (2003). ATS/ACCP statement on cardiopulmonary exercise testing. *American Journal of Respiratory and Critical Care Medicine*, *167*(2), doi:211.10.1164/rccm.167.2.211

Babraj, J. A., Vollaard, N. B., Keast, C., Guppy, F. M., Cottrell, G., & Timmons, J. A. (2009). Extremely short duration high intensity interval training substantially improves insulin action in young healthy males. *BioMed Central Endocrine Disorders, 9*(1), 8. doi:10.1186/1472-6823-9-3

Bar-Or, O. (1987). The Wingate anaerobic test an update on methodology, reliability, and validity. *Sports Medicine*, *4*(6), 381-394.

Batacan, R. B., Duncan, M. J., Dalbo, V. J., Tucker, P. S., & Fenning, A. S. (2017). Effects of high-intensity interval training on cardiometabolic health: A systematic review and meta-analysis of intervention studies. *British Journal of Sports Medicine*, *51*(6), 494-503. doi:10.1136/bjsports-2015-095841

Bell, D., & Mclellan, T. (2002). Exercise endurance 1, 3, and 6 h after caffeine ingestion in caffeine users and nonusers. *Journal of Applied Physiology, 93*(4), 1227-1234. doi:10.1152/japplphysiol.00187.2002

Bigger, J. T., Fleiss, J. L., Rolnitzky, L. M., & Steinman, R. C. (1993). The ability of several short-term measures of RR variability to predict mortality after myocardial infarction. *Circulation*, *88*(3), 927-934. doi:10.1161/01.CIR.88.3.927

Bilandzic, A., & Rosella, L. (2017). The cost of diabetes in Canada over 10 years: Applying attributable healthcare costs to a diabetes incidence prediction model. *Chronic Diseases and Injuries in Canada*, *37*(2). Retrieved from http://search.proquest.com

Björkman, F., Ekblom-Bak, E., Ekblom, Ö., & Ekblom, B. (2016). Validity of the revised Ekblom Bak cycle ergometer test in adults. *European Journal of Applied Physiology*, *116*(9), 1627-1638. doi:10.1007/s00421-016-3412-0

Borg, G.A.V. (1998). *Borg's Perceived Exertion and Pain Scales*. Champaign, IL: Human Kinetics

Burgomaster, K. A., Heigenhauser, G. J., & Gibala, M. J. (2006). Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *Journal of Applied Physiology*, *100*(6), 2041-2047. doi:10.1152/japplphysiol.01220.2005

Burgomaster, K. A., Hughes, S. C., Heigenhauser, G. J., Bradwell, S. N., & Gibala, M. J. (2005). Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal of Applied Physiology*, *98*(6), 1985-1990. doi:10.1152/japplphysiol.01095.2004

Canadian Society for Exercise Physiology. (2013). CSEP-PATH: Physical activity training for health. Ottawa, ON: Canadian Society of Exercise Physiology

Casolo, G., Balli, E., Fazi, A., Gori, C., Freni, A., & Gensini, G. (1991). Twenty-four-hour spectral analysis of heart rate variability in congestive heart failure secondary to coronary artery disease. *American Journal of Cardiology*, *67*(13), 1154-1158. doi:10.1016/0002-9149(91)90887-Q

Christ-Roberts, C. Y., Pratipanawatr, T., Pratipanawatr, W., Berria, R., Belfort, R., Kashyap, S., & Mandarino, L. J. (2004). Exercise training increases glycogen synthase activity and GLUT4 expression but not insulin signaling in overweight nondiabetic and type 2 diabetic subjects. *Metabolism*, *53*(9), 1233-1242. doi:10.1016/j.metabol.2004.03.022

Chrysant, S. G., & Chrysant, G. S. (2015). The cardiovascular consequences of excess sitting time. *Journal of Clinical Hypertension*, *17*(7), 528-531. doi:10.1111/jch.12519.

Chudyk, A., & Petrella, R. J. (2011). Effects of exercise on cardiovascular risk factors in type 2 diabetes. *Diabetes Care*, *34*(5), 1228-1237. doi:10.2337/dc10-1881

Colberg, S. R., Albright, A. L., Blissmer, B. J., Braun, B., Chasan-Taber, L., Fernhall, B., ... & Sigal, R. J. (2010). Exercise and type 2 diabetes: American College of sports medicine and the American diabetes association: Joint position statement. Exercise and type 2 diabetes. *Medicine and Science in Sports and Exercise*, *42*(12), 2282-2303. doi:10.2337/dc10-9990

Colley, R. C., Garriguet, D., Janssen, I., Craig, C. L., Clarke, J., & Tremblay, M. S. (2011). Physical activity of Canadian adults: Accelerometer results from the 2007 to 2009 Canadian Health Measures Survey. *Health Reports*, *22*(1), 7. Retrieved from http://search.proquest.com

Craig, C. L., Marshall, A. L., Sjostrom, M., Bauman, A. E., Booth, M. L., Ainsworth, B. E., … & Oja, P. (2003). International physical activity questionnaire: 12-country reliability and validity. *Medicine & Science in Sports & Exercise*, *35*(8), 1381-1395. doi:10.1249/01.MSS.0000078924.61453.FB

da Silva, F., Kastelianne, A., de Rezende Barbosa, P. D. C., Marques Vanderlei, F., Destro Christofaro, D. G., Vanderlei, M., & Carlos, L. (2016). Application of heart rate variability in diagnosis and prognosis of individuals with diabetes mellitus: Systematic review. *Annals of Noninvasive Electrocardiology*, *21*(3), 223-235. doi:10.1111/anec.12372

Esbjornsson-Liljedahl, M., Bodin, K., & Jansson, E. (2002). Smaller muscle ATP reduction in women than in men by repeated bouts of sprint exercise. *Journal of Applied Physiology*, *93*(3), 1075-1083.doi:10.1152/japplphysiol.00732.1999

Ewing, D. J., Campbell, I. W., and Clarke, B. F. (1976). Mortality in diabetic autonomic neuropathy. *Lancet,* 1, 601–603. doi: 10.1016/S0140-6736(76)90413-X

DeFronzo, R. A., Tobin, J. D., & Andres, R. (1979). Glucose clamp technique: A method for quantifying insulin secretion and resistance. *The American Journal of Physiology*, *237*(3), E214–E223. doi:10.1152/ajpendo.1979.237.3.E214

DeFronzo, R. A., & Tripathy, D. (2009). Skeletal muscle insulin resistance is the primary defect in type 2 diabetes. *Diabetes Care*, *32*(2), S157–S163. doi:10.2337/dc09-S302

Dekker, J. M., Schouten, E. G., Klootwijk, P., Pool, J., Swenne, C. A., & Kromhout, D. (1997). Heart rate variability from short electrocardiographic recordings predicts mortality from all causes in middle-aged and elderly men: The Zutphen Study. *American Journal of Epidemiology*, *145*(10), 899-908. doi:10.1093/oxfordjournals.aje.a009049

Derave, W., Hansen, B. F., Lund, S., Kristiansen, S., & Richter, E. A., (2000) Muscle glycogen content affects insulin-stimulated glucose transport and protein kinase B activity. *American Journal Physiology Endocrinology Metabolism, 279*(5), E947-955

Després, J. P., & Lemieux, I. (2006). Abdominal obesity and metabolic syndrome. *Nature*, *444*(7121), 881-887. doi:10.1038/nature05488

Eckel, R. H., Kahn, R., Robertson, R. M., & Rizza, R. A. (2006). Preventing cardiovascular disease and diabetes a call to action from the American Diabetes Association and the American Heart Association. *Circulation*, *113*(25), 2943-2946. doi:10.1161/CIRCULATIONAHA.106.176583

Ekblom-Bak, E., Björkman, F., Hellenius, M. L., & Ekblom, B. (2014). A new submaximal cycle ergometer test for prediction of VO_{2max}. *Scandinavian Journal of Medicine & Science in Sports*, *24*(2), 319-326. doi:10.1111/sms.12014

Esler, M., Straznicky, N., Eikelis, N., Masuo, K., Lambert, G., & Lambert, E. (2006). Mechanisms of sympathetic activation in obesity-related hypertension. *Hypertension*, *48*(5), 787-796. doi:10.1161/01.HYP.0000242642.42177.49

Evans, H. J., Ferrar, K. E., Smith, A. E., Parfitt, G., & Eston, R. G. (2015). A systematic review of methods to predict maximal oxygen uptake from submaximal, open circuit spirometry in healthy adults. *Journal of Science and Medicine in Sport*, *18*(2), 183-188. doi:10.1016/j.jsams.2014.03.006

Fisher, G., Brown, A. W., Brown, M. M. B., Alcorn, A., Noles, C., Winwood, L., ... & Allison, D. B. (2015). High intensity interval-vs moderate intensity-training for improving cardiometabolic health in overweight or obese males: A randomized controlled trial. *PloS One*, *10*(10), e0138853. doi:10.1371/journal. pone.0138853

Garber, C. E., Blissmer, B., Deschenes, M. R., Franklin, B. A., Lamonte, M. J., Lee, I. M., ... & Swain, D. P. (2011). American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: Guidance for prescribing exercise. *Medicine and Science in Sports and Exercise*, *43*(7), 1334-1359. doi:10.1249/MSS.0b013e318213fefb

Gibala, M. J., Gillen, J. B., & Percival, M. E. (2014). Physiological and health-related adaptations to low-volume interval training: Influences of nutrition and sex. *Sports Medicine*, *44*(2), 127-137. doi:10.1007/s40279-014-0259-6

Gibala, M. J., Little, J. P., MacDonald, M. J., & Hawley, J. A. (2012). Physiological adaptations to low-volume, high-intensity interval training in health and disease. *Journal of Physiology*, *590*(5), 1077-1084. DOI: 10.1113/jphysiol.2011.224725

Gibala, M. J., Little, J. P., Van Essen, M., Wilkin, G. P., Burgomaster, K. A., Safdar, A., ... & Tarnopolsky, M. A. (2006). Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *Journal of Physiology*, *575*(3), 901-911. doi: 10.1113/jphysiol.2006.112094

Gibala, M. J., McGee, S. L., Garnham, A. P., Howlett, K. F., Snow, R. J., & Hargreaves, M. (2009). Brief intense interval exercise activates AMPK and p38 MAPK signaling and increases the expression of PGC-1α in human skeletal muscle. *Journal of Applied Physiology*, *106*(3), 929-934. doi:10.1152/japplphysiol.90880.2008

Gill, J. M. R., & Cooper, A. R. (2008). Physical activity and prevention of type 2 diabetes mellitus. *Sports Medicine, 38*(10), 807-24.

Gist, N. H., Fedewa, M. V., Dishman, R. K., & Cureton, K. J. (2014). Sprint interval training effects on aerobic capacity: A systematic review and meta-analysis. *Sports Medicine*, *44*(2), 269-279. doi:10.1007/s40279-013-0115-0

Godin, G., Desharnais, R., Valois, P., Lepage, L., Jobin, J., & Bradet, R. (1994). Differences in perceived barriers to exercise between high and low intenders: Observations among different populations. *American Journal of Health Promotion*, *8*(4), 279-385. doi:10.4278/0890-1171-8.4.279

Green, J. B. (2014). Understanding the type 2 diabetes mellitus and cardiovascular disease risk paradox. *Postgraduate Medicine*, *126*(3), 190-204.doi:10.3810/pgm.2014.05.2767

Guimaraes, G. V., Ciolac, E. G., Carvalho, V. O., D'Avila, V. M., Bortolotto, L. A., & Bocchi, E. A. (2010). Effects of continuous vs. interval exercise training on blood pressure and arterial stiffness in treated hypertension. *Hypertension Research*, *33*(6), 627-632. doi:10.1038/hr.2010.42

Hallal, P. C., Andersen, L. B., Bull, F. C., Guthold, R., Haskell, W., Ekelund, U., & Lancet Physical Activity Series Working Group. (2012). Global physical activity levels: Surveillance progress, pitfalls, and prospects. *Lancet*, *380*(9838), 247-257. doi:10.1016/S0140-6736(12)60646-1

Haskell, W. L., Lee, I. M., Pate, R. R., Powell, K. E., Blair, S. N., Franklin, B. A., ... & Bauman, A. (2007). Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation*, *116*(9), 1081. doi:10.1161/CIRCULATION.107.185649

Hanley, A. J., Karter, A. J., Festa, A., D'Agostino, R., Wagenknecht, L. E., Savage, P., ... & Haffner, S. (2002). Factor analysis of metabolic syndrome using directly measured insulin sensitivity the insulin resistance atherosclerosis study. *Diabetes*, *51*(8), 2642-2647. doi:10.2337/diabetes.51.8.2642

Hawley, J. A., & Gibala, M. J. (2009). Exercise intensity and insulin sensitivity: How low can you go? *Diabetologia*, *52*(9), 1709-1713. doi:10.1007/s00125-009-1425-5

Hill, A. V., & Lupton, H. (1923). Muscular exercise, lactic acid, and the supply and utilization of oxygen. *QJM: Quarterly Journal of Medicine*, (62), 135-171. doi:10.1093/qjmed/os-16.62.135

Holloszy, J. O. (2005). Exercise-induced increase in muscle insulin sensitivity. *Journal of Applied Physiology*, *99*(1), 338-343. doi: 10.1152/japplphysiol.00123.2005

Hughes, V. A., Fiatarone, M. A., Fielding, R. A., Kahn, B. B., Ferrara, C. M., Shepherd, P. ... & Evans, W. J. (1993). Exercise increases muscle GLUT-4 levels and insulin action in subjects with impaired glucose tolerance. *American Journal of Physiology-Endocrinology and Metabolism*, *264*(6), E855-E862. Retrieved from http://ajpendo.physiology.org

Hussey, S. E., McGee, S. L., Garnham, A., McConell, G. K., & Hargreaves, M. (2012). Exercise increases skeletal muscle GLUT4 gene expression in patients with type 2 diabetes. *Diabetes, Obesity and Metabolism*, *14*(8), 768-771. doi:10.1111/j.1463-1326.2012.01585

Huxley, R., Barzi, F., & Woodward, M. (2006). Excess risk of fatal coronary heart disease associated with diabetes in men and women: meta-analysis of 37 prospective cohort studies. *BMJ*, *332*(7533), 73-78. doi:10.1136%2Fbmj.38678.389583.7C

Istenes, I., Körei, A. E., Putz, Z., Németh, N., Martos, T., Keresztes, K., ... & Kempler, P. (2014). Heart rate variability is severely impaired among type 2 diabetic patients with hypertension. *Diabetes/Metabolism Research and Reviews*, *30*(4), 305-312. doi: 10.1002/dmrr.2496

James, D. E., Brown, R., Navarro, J., & Pilch, P. F. (1988). Insulin-regulatable tissues express a unique insulin-sensitive glucose transport protein. *333,* 183-185, doi:10.1038/333183a0

Jelleyman, C., Yates, T., O'Donovan, G., Gray, L. J., King, J. A., Khunti, K., & Davies, M. J. (2015). The effects of high-intensity interval training on glucose regulation and insulin resistance: a meta-analysis. *Obesity Reviews, 16*(11), 942-961. doi:10.1111/obr.12317

Jenkins, E. M., Nairn, L. N., Skelly, L. E., Little, J. P., & Gibala, M. J. (2019). Do stair climbing exercise “snacks” improve cardiorespiratory fitness? *Applied Physiology, Nutrition, and Metabolism*, *44*(6), 681-684. doi:10.1139/apnm-2018-0675

Jensen, J., Jebens, E., Brennesvik, E. O., Ruzzin, J., Soos, M. A., Engebretsen, E. M., ... & Whitehead, J. P. (2006). Muscle glycogen inharmoniously regulates glycogen synthase activity, glucose uptake, and proximal insulin signaling. *American*

Journal of Physiology-Endocrinology and Metabolism, *290*(1), E154-E162. doi:10.1152/ajpendo.00330.2005

Kaikkonen, P., Hynynen, E., Mann, T., Rusko, H., & Nummela, A. (2010). Can HRV be used to evaluate training load in constant load exercises?. *European Journal of Applied Physiology*, *108*(3), 435-442. doi:10.1007/s00421-009-1240-1

Kaikkonen, P., Hynynen, E., Mann, T., Rusko, H., & Nummela, A. (2012). Heart rate variability is related to training load variables in interval running exercises. *European Journal of Applied Physiology*, *112*(3), 829-838.doi:10.1007/s00421-011-2031-z

Kaminsky, L. A., Arena, R., Beckie, T. M., Brubaker, P. H., Church, T. S., Forman, D. E., ... & Patel, M. J. (2013). The importance of cardiorespiratory fitness in the United States: The need for a national registry a policy statement from the American Heart Association. *Circulation*, *127*(5), 652-662. doi:10.1161/CIR.0b013e31827ee100

Karstoft, K., Winding, K., Knudsen, S. H., James, N. G., Scheel, M. M., Olesen, J., ... & Solomon, T. P. (2014). Mechanisms behind the superior effects of interval vs continuous training on glycaemic control in individuals with type 2 diabetes: A randomised controlled trial. *Diabetologia*, *57*(10), 2081-2093. doi:10.1007/s00125-014-3334-5

Kautzner, J., & Camm, A. (1997). Clinical relevance of heart rate variability. *Clinical Cardiology*, *20*(2), 162-168. doi:10.1002/clc.4960200214

Kelley, D. E., Mokan, M., & Mandarino, L. J. (1992). Intracellular defects in glucose metabolism in obese patients with NIDDM. *Diabetes*, *41*(6), 698-706. doi:10.2337/diab.41.6.698

Kemp, A. H., & Quintana, D. S. (2013). The relationship between mental and physical health: Insights from the study of heart rate variability. *International Journal of Psychophysiology*, *89*(3), 288-296. doi:10.1016/j.ijpsycho.2013.06.018

Kim, G., Lee, Y. H., Jeon, J. Y., Bang, H., Lee, B. W., Kang, E. S., ... & Kim, C. S. (2017). Increase in resting heart rate over 2 years predicts incidence of diabetes: A 10-year prospective study. *Diabetes & Metabolism*, *43*(1), 25-32.

Kirwan, J. P., Solomon, T. P., Wojta, D. M., Staten, M. A., & Holloszy, J. O. (2009). Effects of 7 days of exercise training on insulin sensitivity and responsiveness in type 2 diabetes mellitus. *American Journal of Physiology-Endocrinology and Metabolism*, *297*(1), E151-E156. doi: 10.1152/ajpendo.00210.2009

Kleiger, R. E., Stein, P. K., & Bigger, J. T. (2005). Heart rate variability: Measurement and clinical utility. *Annals of Noninvasive Electrocardiology*, *10*(1), 88-101. doi: 10.1111/j.1542-474X.2005.10101.x

Kodama, S., Saito, K., Tanaka, S., Maki, M., Yachi, Y., Asumi, M., ... & Yamada, N. (2009). Cardiorespiratory fitness as a quantitative predictor of all-cause mortality and cardiovascular events in healthy men and women: A meta-analysis. *JAMA*, *301*(19), 2024-2035. doi:10.1001/jama.2009.681

Kokkinos, P., & Myers, J. (2010). Exercise and physical activity clinical outcomes and applications. *Circulation*, *122*(16), 1637-1648. doi:10.1161/CIRCULATIONAHA.110.948349

Kokkinos, P., Myers, J., Kokkinos, J. P., Pittaras, A., Narayan, P., Manolis, A., ... & Singh, S. (2008). Exercise capacity and mortality in black and white men. *Circulation*, *117*(5), 614-622. doi:10.1161/CIRCULATIONAHA.107.734764

Kotsis, V., Stabouli, S., Bouldin, M., Low, A., Toumanidis, S., & Zakopoulos, N. (2005). Impact of obesity on 24-hour ambulatory blood pressure and hypertension. *Hypertension*, *45*(4), 602-607. doi:10.1161/01.HYP.0000158261.86674.8e

Lahiri, M. K., Kannankeril, P. J., & Goldberger, J. J. (2008). Assessment of autonomic function in cardiovascular disease: Physiological basis and prognostic implications. *Journal of the American College of Cardiology*, *51*(18), 1725-1733. doi:10.1016/j.jacc.2008.01.038

Lampert, R., Bremner, J. D., Su, S., Miller, A., Lee, F., Cheema, F., ... & Vaccarino, V. (2008). Decreased heart rate variability is associated with higher levels of inflammation in middle-aged men. *American Heart Journal*, *156*(4), 759.e1–759.e7. doi:10.1016/j.ahj.2008.07.009.

Landsberg, L. (2001). Insulin-mediated sympathetic stimulation: role in the pathogenesis of obesity-related hypertension (or, how insulin affects blood pressure, and why). *Journal of Hypertension*, *19*(3), 523-528.

Landsberg, L., Aronne, L. J., Beilin, L. J., Burke, V., Igel, L. I., Lloyd-Jones, D., & Sowers, J. (2013). Obesity-related hypertension: Pathogenesis, cardiovascular risk, and treatment—A position paper of the Obesity Society and the American Society of Hypertension. *Obesity*, *21*(1), 8-24. doi:10.1002/oby.20181

Laukkanen, J. A., Lakka, T. A., Rauramaa, R., Kuhanen, R., Venäläinen, J. M., Salonen, R., & Salonen, J. T. (2001). Cardiovascular fitness as a predictor of mortality in men. *Archives of Internal Medicine*, *161*(6), 825-831. doi: 10.1001/archinte.161.6.825

Lawes, C. M., Vander Hoorn, S., & Rodgers, A. (2008). Global burden of blood-pressure-related disease, 2001. *Lancet*, *371*(9623), 1513-1518. doi:10.1016/S0140-6736(08)60655-8

Lee, I. M., Shiroma, E. J., Lobelo, F., Puska, P., Blair, S. N., Katzmarzyk, P. T., & Lancet Physical Activity Series Working Group. (2012). Effect of physical inactivity on major non-communicable diseases worldwide: An analysis of burden of disease and life expectancy. *Lancet*, *380*(9838), 219-229. doi:10.1016/S0140-6736(12)61031-9

Little, J. P., Safdar, A., Wilkin, G. P., Tarnopolsky, M. A., & Gibala, M. J. (2010). A practical model of low-volume high-intensity interval training induces mitochondrial biogenesis in human skeletal muscle: Potential mechanisms. *Journal of Physiology*, *588*(6), 1011-1022. doi: 10.1113/jphysiol.2009.181743

Little, J. P., Gillen, J. B., Percival, M. E., Safdar, A., Tarnopolsky, M. A., Punthakee, Z., ... & Gibala, M. J. (2011). Low-volume high-intensity interval training reduces hyperglycemia and increases muscle mitochondrial capacity in patients with type 2 diabetes. *Journal of Applied Physiology*, *111*(6), 1554-1560. doi:10.1152/japplphysiol.00921.2011

Low, P. A., Opfer-Gehrking, T. L., McPhee, B. R., Fealey, R. D., Benarroch, E. E., Willner, C. L., ... & Corfits, J. L. (1995). Prospective evaluation of clinical characteristics of orthostatic hypotension. *Mayo Clinic Proceedings* 7(70), 617-622. doi:10.4065/70.7.617

Mancia, G., Ferrari, A., Gregorini, L., Parati, G., Pomidossi, G., Bertinieri, G., ... & Zanchetti, A. (1983). Blood pressure and heart rate variabilities in normotensive and hypertensive human beings. *Circulation Research*, *53*(1), 96-104. doi:10.1161/01.RES.53.1.96

Mendis, S., Puska, P., & Norrving, B. (2011). *Global atlas on cardiovascular disease prevention and control*. Geneva, Switzerland: World Health Organization. Retrieved from http://www.who.int

Metcalfe, R. S., Babraj, J. A., Fawkner, S. G., & Vollaard, N. B. (2012). Towards the minimal amount of exercise for improving metabolic health: Beneficial effects of reduced-exertion high-intensity interval training. *European Journal of Applied Physiology*, *112*(7), 2767-2775. doi:10.1007/s00421-011-2254-z

Metcalfe, R. S., Tardif, N., Thompson, D., & Vollaard, N. B. (2016). Changes in aerobic capacity and glycaemic control in response to reduced-exertion high-intensity interval training (REHIT) are not different between sedentary men and women. *Applied Physiology, Nutrition, and Metabolism*, *41*(11), 1117-1123. doi:10.1139/apnm-2016-0253

Michelini, L. C., & Stern, J. E. (2009). Exercise-induced neuronal plasticity in central autonomic networks: role in cardiovascular control. *Experimental physiology*, *94*(9), 947-960. doi:10.1113/expphysiol.2009.047449

Mitranun, W., Deerochanawong, C., Tanaka, H., & Suksom, D. (2014). Continuous vs interval training on glycemic control and macro-and microvascular reactivity in type 2 diabetic patients. *Scandinavian Journal of Medicine & Science in Sports*, *24*(2), e69-e76. doi:10.1111/sms.12112

Moholdt, T. T., Amundsen, B. H., Rustad, L. A., Wahba, A., Løvø, K. T., Gullikstad, L. R., ... & Slørdahl, S. A. (2009). Aerobic interval training versus continuous moderate exercise after coronary artery bypass surgery: A randomized study of cardiovascular effects and quality of life. *American Heart Journal*, *158*(6), 1031-1037. doi:10.1016/j.ahj.2009.10.003

Molmen-Hansen, H. E., Stolen, T., Tjonna, A. E., Aamot, I. L., Ekeberg, I. S., Tyldum, G. A., ... & Stoylen, A. (2012). Aerobic interval training reduces blood pressure and improves myocardial function in hypertensive patients. *European Journal of Preventive Cardiology*, *19*(2), 151-160. doi:10.1177/17418267

Mootha, V. K., Lindgren, C. M., Eriksson, K. F., Subramanian, A., Sihag, S., Lehar, J., ... & Houstis, N. (2003). PGC-1α-responsive genes involved in oxidative phosphorylation are coordinately downregulated in human diabetes. *Nature Genetics*, *34*(3), 267-273. doi:10.1038/ng1180

Myers, J., Prakash, M., Froelicher, V., Do, D., Partington, S., & Atwood, J. E. (2002). Exercise capacity and mortality among men referred for exercise testing. *New England Journal of Medicine*, *346*(11), 793-801. doi:10.1056/NEJMoa011858

Nocon, M., Hiemann, T., Müller-Riemenschneider, F., Thalau, F., Roll, S., & Willich, S. N. (2008). Association of physical activity with all-cause and cardiovascular mortality: A systematic review and meta-analysis. *European Journal of Cardiovascular Prevention & Rehabilitation*, *15*(3), 239-246. doi: 10.1097/HJR.0b013e3282f55e09

Noonan, V., & Dean, E. (2000). Submaximal exercise testing: Clinical application and interpretation. *Physical Therapy*, *80*(8), 782-807. doi: 10.1093/ptj/80.8.782

Nunan, D., Sandercock, G. R., & Brodie, D. A. (2010). A quantitative systematic review of normal values for short-term heart rate variability in healthy adults. *Pacing and Clinical Electrophysiology*, *33*(11), 1407-1417. doi: 10.1111/j.1540-8159.2010.02841.x

Nybo, L., Sundstrup, E., Jakobsen, M. D., Mohr, M., Hornstrup, T., Simonsen, L., ... & Krustrup, P. (2010). High-intensity training versus traditional exercise interventions for promoting health. *Medicine & Science in Sports and Exercise*, *42*(10), 1951-8. doi:10.1249/MSS.0b013e3181d99203

Palange, P., Ward, S. A., Carlsen, K. H., Casaburi, R., Gallagher, C. G., Gosselink, R., ... & Whipp, B. J. (2007). Recommendations on the use of exercise testing in clinical practice. *European Respiratory Journal*, *29*(1), 185-209. doi: 10.1183/09031936.00046906

Parolin, M. L., Chesley, A., Matsos, M. P., Spriet, L. L., Jones, N. L., & Heigenhauser, G. J. (1999). Regulation of skeletal muscle glycogen phosphorylase and PDH during maximal intermittent exercise. *American Journal of Physiology-Endocrinology and Metabolism*, *277*(5), E890-E900. Retrieved from http://ajpendo.physiology.org

Peters, S. A., Huxley, R. R., & Woodward, M. (2014). Diabetes as risk factor for incident coronary heart disease in women compared with men: A systematic review and meta-analysis of 64 cohorts including 858,507 individuals and 28,203 coronary events. *Diabetologia, 57*(8), 1542-1551. doi:10.1007/s00125-014-3260-6

Pickering, T. G., Hall, J. E., Appel, L. J., Falkner, B. E., Graves, J., Hill, M. N., ... & Roccella, E. J. (2005). Recommendations for blood pressure measurement in humans and experimental animals part 1: Blood pressure measurement in humans: A statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Hypertension*, *45*(1), 142-161. doi:10.1161/01.HYP.0000150859.47929.8e

Porges, S. W. (2011). *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation (Norton Series on Interpersonal Neurobiology)*. New York, NY: WW Norton & Company. Retrieved from https://books.google.ca

Racil, G., Ounis, O. B., Hammouda, O., Kallel, A., Zouhal, H., Chamari, K., & Amri, M. (2013). Effects of high vs. moderate exercise intensity during interval training on lipids and adiponectin levels in obese young females. *European Journal of Applied Physiology*, *113*(10), 2531-2540. doi:10.1007/s00421-013-2689-5

Rahmouni, K., Correia, M. L., Haynes, W. G., & Mark, A. L. (2005). Obesity-associated hypertension new insights into mechanisms. *Hypertension*, *45*(1), 9-14. doi: 10.1161/01.HYP.0000151325.83008.b4

Reichert, F. F., Barros, A. J., Domingues, M. R., & Hallal, P. C. (2007). The role of perceived personal barriers to engagement in leisure-time physical activity. *American Journal of Public Health*, *97*(3), 515-519. doi:10.2105/AJPH.2005.070144

Ritov, V. B., Menshikova, E. V., Azuma, K., Wood, R., Toledo, F. G., Goodpaster, B. H., ... & Kelley, D. E. (2010). Deficiency of electron transport chain in human skeletal muscle mitochondria in type 2 diabetes mellitus and obesity. *American Journal of Physiology-Endocrinology and Metabolism*, *298*(1), E49-E58. doi:10.1152/ajpendo.00317.2009

Rognmo, Ø., Hetland, E., Helgerud, J., Hoff, J., & Slørdahl, S. A. (2004). High intensity aerobic interval exercise is superior to moderate intensity exercise for increasing aerobic capacity in patients with coronary artery disease. *European Journal of Cardiovascular Prevention & Rehabilitation*, *11*(3), 216-222. doi:10.1097/01.hjr.0000131677.96762.0c

Ryu, S., Choi, S. K., Joung, S. S., Suh, H., Cha, Y. S., Lee, S., & Lim, K. (2001). Caffeine as a lipolytic food component increases endurance performance in rats and athletes. *Journal of Nutritional Science and Vitaminology*, *47*(2), 139-146. doi:10.3177/jnsv.47.139

Saltin, B., & Calbet, J. A. (2006). Point: in health and in a normoxic environment, VO2 max is limited primarily by cardiac output and locomotor muscle blood flow. *Journal of Applied Physiology*, *100*(2), 744-748.Doi: 10.1152/japplphysiol.01395.2005

Schäfer, A., & Vagedes, J. (2013). How accurate is pulse rate variability as an estimate of heart rate variability? A review on studies comparing photoplethysmographic technology with an electrocardiogram. *International Journal of Cardiology*, *166*(1), 15-29. doi:10.1016/j.ijcard.2012.03.119

Schiaffino, S., & Reggiani, C. (2011). Fiber types in mammalian skeletal muscles. *Physiological Reviews*, *91*(4), 1447-1531.doi:10.1152/physrev.00031.2010

Schjerve, I. E., Tyldum, G. A., Tjønna, A. E., Stølen, T., Loennechen, J. P., Hansen, H. E., ... & Smith, G. L. (2008). Both aerobic endurance and strength training programmes improve cardiovascular health in obese adults. *Clinical Science*, *115*(9), 283-293. doi:10.1042/CS20070332

Shaffer, F., McCraty, R., & Zerr, C. L. (2014). A healthy heart is not a metronome: An integrative review of the heart's anatomy and heart rate variability. *Frontiers in Psychology*, *5*. doi:10.3389/fpsyg.2014.01040

Shaffer, F., & Venner, J. (2013). Heart rate variability anatomy and physiology. *Biofeedback*, *41*(1), 13-25. doi:10.5298/1081-5937-41.1.05

Shepherd, S. O., Wilson, O. J., Taylor, A. S., Thøgersen-Ntoumani, C., Adlan, A. M., Wagenmakers, A. J., & Shaw, C. S. (2015). Low-volume high-intensity interval training in a gym setting improves cardio-metabolic and psychological health. *PloS One*, *10*(9), e0139056. doi:10.1371/journal.pone.0139056

Sigal, R. J., Kenny, G. P., Wasserman, D. H., & Castaneda-Sceppa, C. (2004). Physical activity/exercise and type 2 diabetes. *Diabetes Care*, *27*(10), 2518-2539. doi:10.2337/diacare.27.10.2518

Sloth, M., Sloth, D., Overgaard, K., & Dalgas, U. (2013). Effects of sprint interval training on VO_{2max} and aerobic exercise performance: A systematic review and meta-analysis. *Scandinavian Journal of Medicine & Science in Sports*, *23*(6). doi:10.1111/sms.12092

Soares-Miranda, L., Sattelmair, J., Chaves, P., Duncan, G., Siscovick, D. S., Stein, P. K., & Mozaffarian, D. (2014). Physical activity and heart rate variability in older adults: The Cardiovascular Health Study. *Circulation*, doi:10.1161/CIRCULATIONAHA.113.005361

Sorkin, J. D., Muller, D. C., Fleg, J. L., & Andres, R. (2005). The relation of fasting and 2-h postchallenge plasma glucose concentrations to mortality data from the Baltimore longitudinal study of aging with a critical review of the literature. *Diabetes Care*, *28*(11), 2626-2632. doi:10.2337/diacare.28.11.2626

Steinberg, G. R., Watt, M. J., McGee, S. L., Chan, S., Hargreaves, M., Febbraio, M. A., ... & Kemp, B. E. (2006). Reduced glycogen availability is associated with increased AMPKα2 activity, nuclear AMPKα2 protein abundance, and GLUT4 mRNA expression in contracting human skeletal muscle. *Applied Physiology, Nutrition, and Metabolism*, *31*(3), 302-312. doi:10.1139/h06-003

Stuckey, M. I., Tulppo, M. P., Kiviniemi, A. M., & Petrella, R. J. (2014). Heart rate variability and the metabolic syndrome: A systematic review of the literature. *Diabetes/Metabolism Research and Reviews*, *30*(8), 784-793. doi:10.1002/dmrr.2555

Stutts, W. C. (2002). Physical activity determinants in adults. Perceived benefits, barriers, and self-efficacy. *American Association of Occupational Health Nurses*, *50*(11), 499-507. Retrieved from http://search.proquest.com

Sullivan, P. W., Morrato, E. H., Ghushchyan, V., Wyatt, H. R., & Hill, J. O. (2005). Obesity, inactivity, and the prevalence of diabetes and diabetes-related cardiovascular comorbidities in the US, 2000–2002. *Diabetes Care*, *28*(7), 1599-1603. doi:10.2337/diacare.28.7.1599

Szuszkiewicz-Garcia, M. M., & Davidson, J. A. (2014). Cardiovascular disease in diabetes mellitus: Risk factors and medical therapy. *Endocrinology and metabolism clinics of North America*, *43*(1), 25-40. doi:10.1016/j.ecl.2013.09.001

Tanaka, H., Monahan, K. D., & Seals, D. R. (2001). Age-predicted maximal heart rate revisited. *Journal of the American College of Cardiology*, *37*(1), 153-156. doi:10.1016/S0735-1097(00)01054-8

Task Force of the European Society of Cardiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *Circulation*, *93*, 1043-1065.

Thayer, J. F., Hansen, A. L., Saus-Rose, E., & Johnsen, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: The neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of Behavioral Medicine*, *37*(2), 141-153. doi: 10.1007/s12160-009-9101-z

Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*, *61*(3), 201-216. doi:10.1016/S0165-0327(00)00338-4

Tjonna, A. E., Lee, S. J., Rognmo, O., Stolen, T. O., Bye, A., Haram, P. M., ... & Kemi, O. J. (2008). Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome: A pilot study. *Circulation*, *118*(4), 346-354. doi:10.1161/CIRCULATIONAHA.108.772822

Tokmakidis, S. P., Léger, L. A., & Pilianidis, T. C. (1998). Failure to obtain a unique threshold on the blood lactate concentration curve during exercise. *European Journal of Applied Physiology and Occupational Physiology*, *77*(4), 333-342. doi:10.1007/s004210050342

Tsuji, H., Larson, M. G., Venditti, F. J., Manders, E. S., Evans, J. C., Feldman, C. L., & Levy, D. (1996). Impact of reduced heart rate variability on risk for cardiac events. *Circulation*, *94*(11), 2850-2855. doi:10.1161/01.CIR.94.11.2850

Tsuji, H., Venditti, F. J., Manders, E. S., Evans, J. C., Larson, M. G., Feldman, C. L., & Levy, D. (1994). Reduced heart rate variability and mortality risk in an elderly cohort. The Framingham Heart Study. *Circulation*, *90*(2), 878-883. doi:10.1161/01.CIR.90.2.878

Velcheva, I., Damianov, P., Mantarova, S., & Antonova, N. (2011). Hemorheology and heart rate variability in patients with diabetes mellitus type 2. *Clinical Hemorheology and Microcirculation*, *49*(1-4), 513-518. doi: 10.3233/CH-2011-1500

Verheggen, R. J. H. M., Maessen, M. F. H., Green, D. J., Hermus, A. R. M. M., Hopman, M. T. E., & Thijssen, D. H. T. (2016). A systematic review and meta-analysis on the effects of exercise training versus hypocaloric diet: Distinct effects on body weight and visceral adipose tissue. *Obesity Reviews, 17*(8). doi: 10.1111/obr.12406

Vollaard, N. B., Constantin-Teodosiu, D., Fredriksson, K., Rooyackers, O., Jansson, E., Greenhaff, P. L., ... & Sundberg, C. J. (2009). Systematic analysis of adaptations in aerobic capacity and submaximal energy metabolism provides a unique insight into determinants of human aerobic performance. *Journal of Applied Physiology*, *106*(5), 1479-1486.doi:10.1152/japplphysiol.91453.2008.

Vollaard, N. B., & Metcalfe, R. S. (2017). Research into the health benefits of sprint interval training should focus on protocols with fewer and shorter sprints. *Sports Medicine*, 1-9. doi:10.1007/s40279-017-0727-x

Vollaard, N. B., Metcalfe, R. S., & Williams, S. (2017). Effect of number of sprints in an SIT session on change in VO_{2max}: A meta-analysis. *Medicine and Science in Sports and Exercise*, *49*(6), 1147-1156. doi:10.1249/MSS.0000000000001204

Vollenweider, P., Randin, D., Tappy, L., Jequier, E., Nicod, P., & Scherrer, U. (1994). Impaired insulin-induced sympathetic neural activation and vasodilation in skeletal muscle in obese humans. *Journal of Clinical Investigation*, *93*(6), 2365. doi:10.1172%2FJCI117242

Warburton, D. E., Charlesworth, S., Ivey, A., Nettlefold, L., & Bredin, S. S. (2010). A systematic review of the evidence for Canada's Physical Activity Guidelines for Adults. *International Journal of Behavioral Nutrition and Physical Activity*, *7*(1), 39. doi:10.1186/1479-5868-7-39

Wewege, M. A., Ahn, D., Yu, J., Liou, K., & Keech, A. (2018). High-intensity interval training for patients with cardiovascular disease—Is it safe? A systematic review. *Journal of the American Heart Association*, *7*(21), e009305. doi:10.1161/JAHA.118.009305

Weston, K. S., Wisløff, U., & Coombes, J. S. (2013). High-intensity interval training in patients with lifestyle-induced cardiometabolic disease: A systematic review and meta-analysis. *British Journal of Sports Medicine*, *48*(16), 1227-1234. doi:10.1136/bjsports-2013-092576

Wisløff, U., Støylen, A., Loennechen, J. P., Bruvold, M., Rognmo, Ø., Haram, P. M., ... & Videm, V. (2007). Superior cardiovascular effect of aerobic interval training versus moderate continuous training in heart failure patients a randomized study. *Circulation*, *115*(24), 3086-3094. doi:10.1161/CIRCULATIONAHA.106.675041

World Health Organization. (2009). *Global health risks: Mortality and burden of disease attributable to selected major risks*. Switzerland: WHO Press. Retrieved from https://books.google.ca

Wormgoor, S. G., Dalleck, L. C., Zinn, C., & Harris, N. K. (2017). Effects of high-intensity interval training on people living with type 2 diabetes: A narrative review. *Canadian Journal of Diabetes*. *41*, 536-547. doi: 10.1016/j.jcjd.2016.12.004

Whyte, L. J., Gill, J. M., & Cathcart, A. J., (2010) Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. *Metabolism, 59*(10), 1421-1428. doi:10.1016/j.metabol.2010.01.002

Yasuma, F., & Hayano, J. I. (2004). Respiratory sinus arrhythmia: why does the heartbeat synchronize with respiratory rhythm? *Chest Journal*, *125*(2), 683-690. Retrieved from https://journal.chestnet.org

Zajac, A., Jarzabek, R., & Waskiewicz, Z. (1999). The diagnostic value of the 10-and 30-second Wingate test for competitive athletes. *Journal of Strength & Conditioning Research*, *13*(1), 16-19. Retrieved from http://journals.lww.com/nsca-jscr/pages/default.aspx

Zhang, T., Zhang, H., Li, S., Li, Y., Liu, Y., Fernandez, C., ... & Chen, W. (2016). Impact of adiposity on incident hypertension is modified by insulin resistance in adults longitudinal observation from the Bogalusa heart study. *Hypertension*, *67*(1), 56-62. doi: 10.1161/HYPERTENSIONAHA.115.06509

Appendix A

Get Active Questionnaire

CSEP | SCPE
THE GOLD STANDARD IN EXERCISE SCIENCE AND PERSONAL TRAINING

Get Active Questionnaire

CANADIAN SOCIETY FOR EXERCISE PHYSIOLOGY –
PHYSICAL ACTIVITY TRAINING FOR HEALTH (CSEP-PATH®)

Physical activity improves your physical and mental health. Even small amounts of physical activity are good, and more is better.

For almost everyone, the benefits of physical activity far outweigh any risks. For some individuals, specific advice from a Qualified Exercise Professional (QEP – has post-secondary education in exercise sciences and an advanced certification in the area – see csep.ca/certifications) or health care provider is advisable. **This questionnaire is intended for all ages – to help move you along the path to becoming more physically active.**

☐ I am completing this questionnaire for myself.
☐ I am completing this questionnaire for my child/dependent as parent/guardian.

PREPARE TO BECOME MORE ACTIVE

The following questions will help to ensure that you have a safe physical activity experience. Please answer **YES** or **NO** to each question before you become more physically active. If you are unsure about any question, answer **YES**.

YES	NO	
		1 Have you experienced **ANY** of the following (A to F) **within the past six months**?
○	○	**A** A diagnosis of/treatment for heart disease or stroke, or pain/discomfort/pressure in your chest during activities of daily living or during physical activity?
○	○	**B** A diagnosis of/treatment for high blood pressure (BP), or a resting BP of 160/90 mmHg or higher?
○	○	**C** Dizziness or lightheadedness during physical activity?
○	○	**D** Shortness of breath at rest?
○	○	**E** Loss of consciousness/fainting for any reason?
○	○	**F** Concussion?
○	○	**2** Do you currently have pain or swelling in any part of your body (such as from an injury, acute flare-up of arthritis, or back pain) that affects your ability to be physically active?
○	○	**3** Has a health care provider told you that you should avoid or modify certain types of physical activity?
○	○	**4** Do you have any other medical or physical condition (such as diabetes, cancer, osteoporosis, asthma, spinal cord injury) that may affect your ability to be physically active?

NO to all questions: go to Page 2 – ASSESS YOUR CURRENT PHYSICAL ACTIVITY ➤

YES to any question: go to Reference Document – ADVICE ON WHAT TO DO IF YOU HAVE A YES RESPONSE ➤➤

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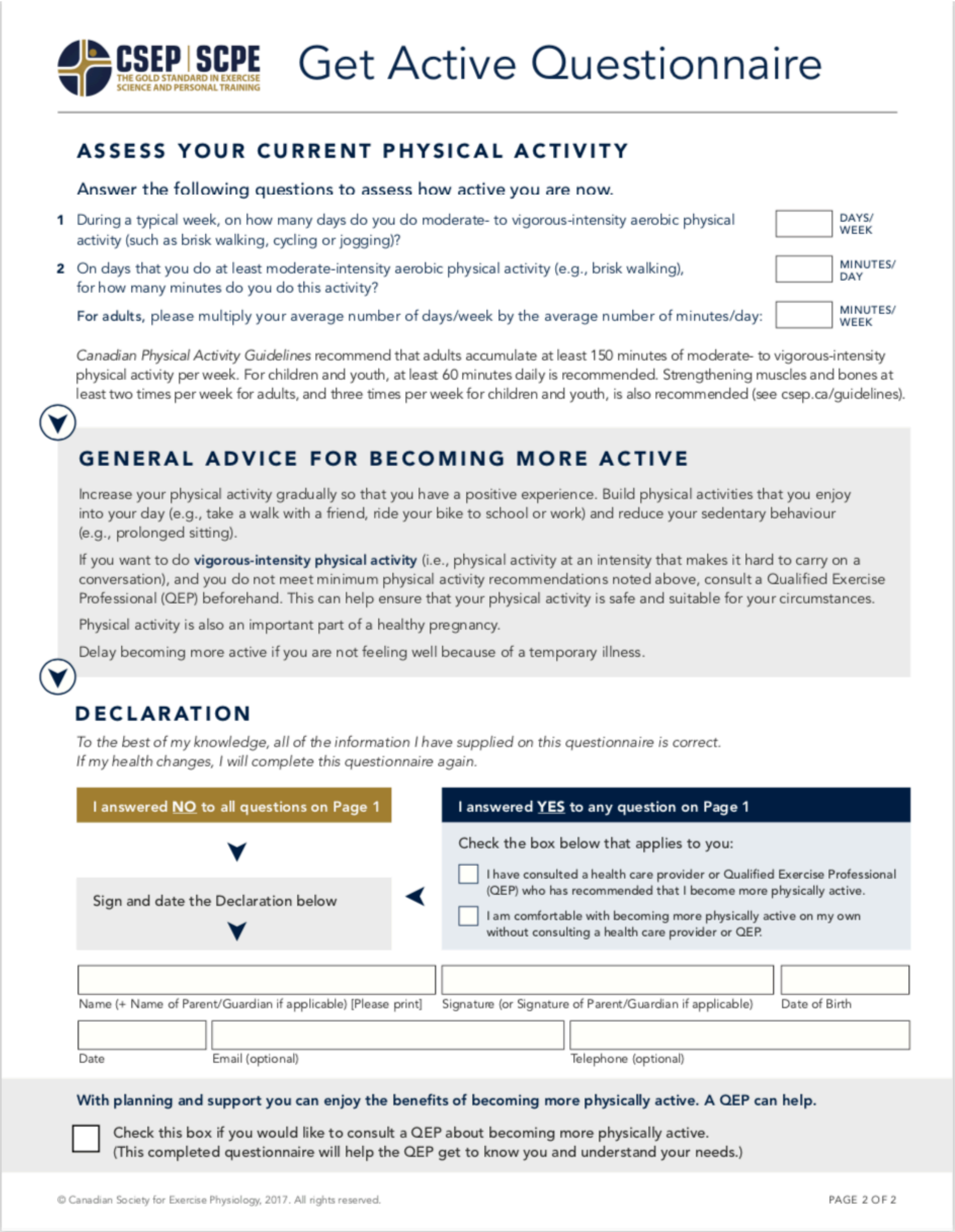
PAGE 1 OF 2



CSEP | SCPE
THE GOLD STANDARD IN EXERCISE SCIENCE AND PERSONAL TRAINING

Get Active Questionnaire

ASSESS YOUR CURRENT PHYSICAL ACTIVITY

Answer the following questions to assess how active you are now.

1 During a typical week, on how many days do you do moderate- to vigorous-intensity aerobic physical activity (such as brisk walking, cycling or jogging)? ☐ DAYS/WEEK

2 On days that you do at least moderate-intensity aerobic physical activity (e.g., brisk walking), for how many minutes do you do this activity? ☐ MINUTES/DAY

For adults, please multiply your average number of days/week by the average number of minutes/day: ☐ MINUTES/WEEK

Canadian Physical Activity Guidelines recommend that adults accumulate at least 150 minutes of moderate- to vigorous-intensity physical activity per week. For children and youth, at least 60 minutes daily is recommended. Strengthening muscles and bones at least two times per week for adults, and three times per week for children and youth, is also recommended (see csep.ca/guidelines).

GENERAL ADVICE FOR BECOMING MORE ACTIVE

Increase your physical activity gradually so that you have a positive experience. Build physical activities that you enjoy into your day (e.g., take a walk with a friend, ride your bike to school or work) and reduce your sedentary behaviour (e.g., prolonged sitting).

If you want to do **vigorous-intensity physical activity** (i.e., physical activity at an intensity that makes it hard to carry on a conversation), and you do not meet minimum physical activity recommendations noted above, consult a Qualified Exercise Professional (QEP) beforehand. This can help ensure that your physical activity is safe and suitable for your circumstances.

Physical activity is also an important part of a healthy pregnancy.

Delay becoming more active if you are not feeling well because of a temporary illness.

DECLARATION

To the best of my knowledge, all of the information I have supplied on this questionnaire is correct.
If my health changes, I will complete this questionnaire again.

I answered NO to all questions on Page 1

Sign and date the Declaration below

I answered YES to any question on Page 1

Check the box below that applies to you:

☐ I have consulted a health care provider or Qualified Exercise Professional (QEP) who has recommended that I become more physically active.

☐ I am comfortable with becoming more physically active on my own without consulting a health care provider or QEP.

Name (+ Name of Parent/Guardian if applicable) [Please print]

Signature (or Signature of Parent/Guardian if applicable)

Date of Birth

Date

Email (optional)

Telephone (optional)

With planning and support you can enjoy the benefits of becoming more physically active. A QEP can help.

☐ Check this box if you would like to consult a QEP about becoming more physically active.
(This completed questionnaire will help the QEP get to know you and understand your needs.)

PAGE 2 OF 2

Appendix B

Maximal Exercise Assessment Form

Maximal Exercise Assessment Checklist *(Nov. 2007 – updated Feb. 2011)*

(Complete for all Max. Assessments – in-class demonstrations, labs and research – file with ParQ)

Preparation	√
1. Protocol presented to the Risk Management Committee for review & recommendation (includes termination guidelines).	
2. Supervisor is recognized by the Risk Management Committee as having competence with maximal testing.	
3. Supervisor certified with Standard First Aid and CPR.	
4. Supervisor will be on-site for all max. assessment activity.	
5. Approved physician on-site for max. assessment with **high risk participants.**	
6. Equipment and facility prepared and inspected for safety of operation.	
7. Exercise protocol and termination guidelines discussed by the supervisor with all those involved in the actual assessment prior to commencing.	
Screening & Risk Stratification	
1. Par-Q & Screening Stratification Questionnaire reviewed and completed with low risk populations.	
2. Par-MedX completed by physician for moderate to high risk populations.	
3. All screening records to be maintained in the School of Kinesiology for seven years.	
Assessment	
1. Exercise protocol verbally reviewed with participant and posted in clear view.	
2. Exercise termination guidelines reviewed and posted in clear view.	
3. Informed consent signed by participant.	

4. Post exercise vital signs monitored and recorded.	
5. Completed screening tools and assessment records filed in the Kinesiology office.	
6. Incidents/accidents promptly addressed, report form completed and filed with the Kinesiology office.	

Name of Participant:____(print)

____(sign)

Date of Assessment: ____

Supervisor: ____(print)

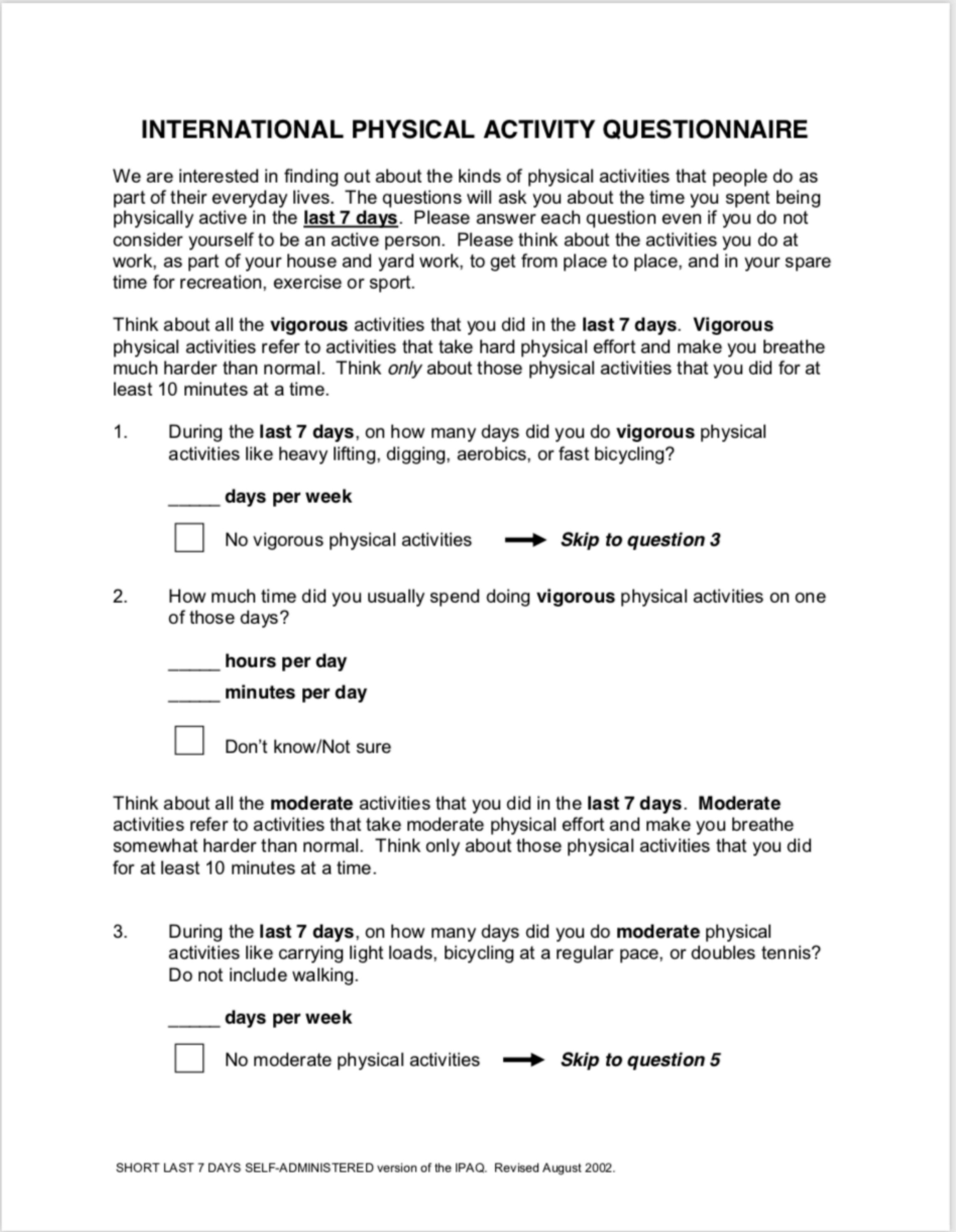
____(sign)

Witness: ____(print)

____(sign)

Appendix C

International Physical Activity Readiness Questionnaire



INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the **vigorous** activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

1. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, digging, aerobics, or fast bicycling?

_____ **days per week**

☐ No vigorous physical activities → ***Skip to question 3***

2. How much time did you usually spend doing **vigorous** physical activities on one of those days?

_____ **hours per day**

_____ **minutes per day**

☐ Don't know/Not sure

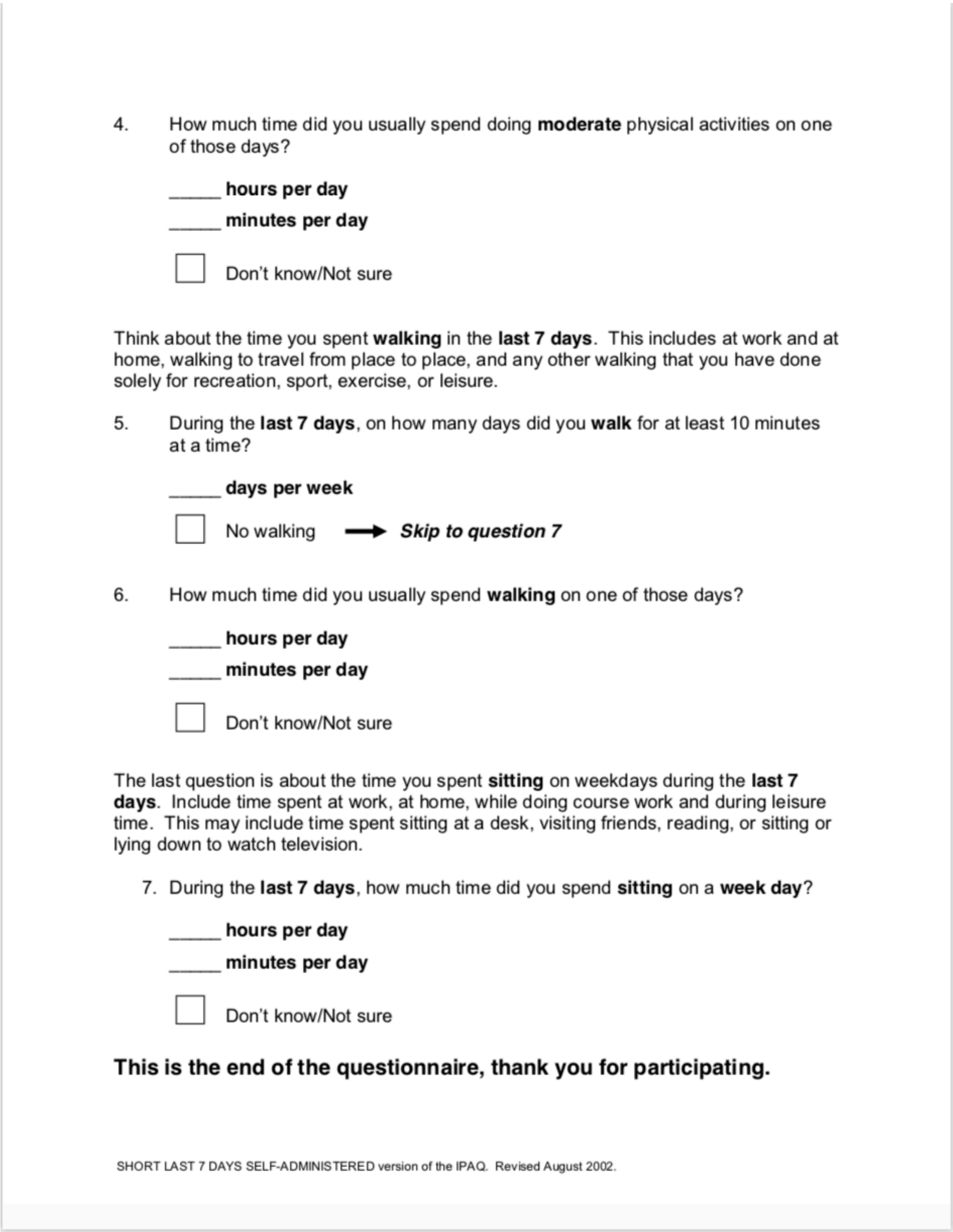
Think about all the **moderate** activities that you did in the **last 7 days**. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

3. During the **last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

_____ **days per week**

☐ No moderate physical activities → ***Skip to question 5***

SHORT LAST 7 DAYS SELF-ADMINISTERED version of the IPAQ. Revised August 2002.



4. How much time did you usually spend doing **moderate** physical activities on one of those days?

_____ **hours per day**

_____ **minutes per day**

☐ Don't know/Not sure

Think about the time you spent **walking** in the **last 7 days**. This includes at work and at home, walking to travel from place to place, and any other walking that you have done solely for recreation, sport, exercise, or leisure.

5. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time?

_____ **days per week**

☐ No walking → ***Skip to question 7***

6. How much time did you usually spend **walking** on one of those days?

_____ **hours per day**

_____ **minutes per day**

☐ Don't know/Not sure

The last question is about the time you spent **sitting** on weekdays during the **last 7 days**. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

7. During the **last 7 days**, how much time did you spend **sitting** on a **week day**?

_____ **hours per day**

_____ **minutes per day**

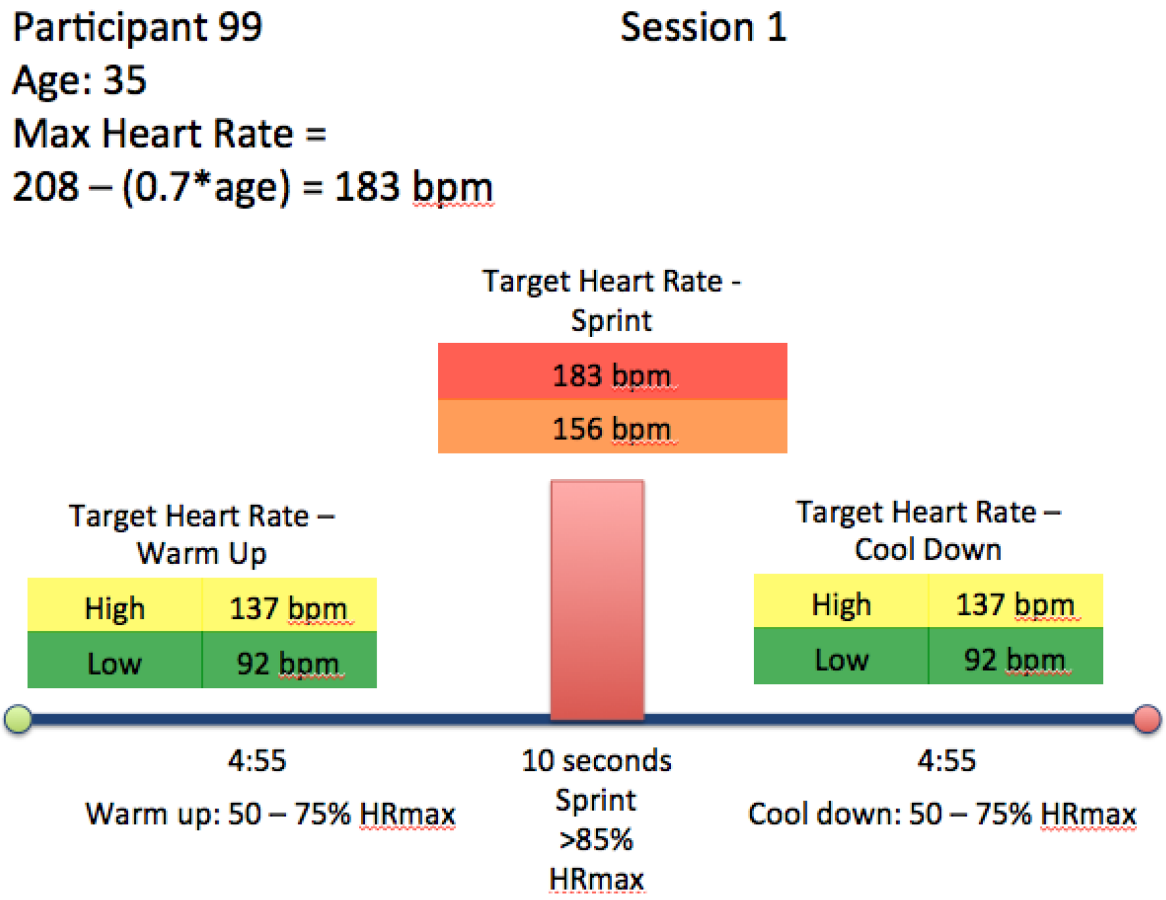
☐ Don't know/Not sure

This is the end of the questionnaire, thank you for participating.

SHORT LAST 7 DAYS SELF-ADMINISTERED version of the IPAQ. Revised August 2002.

Appendix D

Participant Heart Rate Card



Participant 99
Age: 35
Max Heart Rate =
208 – (0.7*age) = 183 bpm

Session 1

Target Heart Rate - Sprint

183 bpm
156 bpm

Target Heart Rate – Warm Up

High	137 bpm
Low	92 bpm

Target Heart Rate – Cool Down

High	137 bpm
Low	92 bpm

4:55
Warm up: 50 – 75% HRmax

10 seconds
Sprint
>85%
HRmax

4:55
Cool down: 50 – 75% HRmax

Appendix E

Bi-Weekly E-mail Survey

1. Are you currently maintaining weekly physical activity levels from before the start of this study, (not including exercise performed as part of the intervention)?

Yes / No

2. Are you currently maintaining similar eating habits from before the start of this study?

Yes / No

3. Have you started a weight loss program or made any other significant lifestyle changes

Yes / No If yes identify the change __________________

Appendix F

Recruitment Poster

Lakehead
UNIVERSITY

RESEARCH PARTICIPANTS WANTED!!!

CAN 2 MINUTES OF HIGH-INTENSITY EXERCISE PER WEEK IMPROVE YOUR HEALTH?

Looking for healthy males and females 18-45 years of age who exercise less than 150-minutes of moderate exercise or 75-minutes of vigorous exercise per week

3x10 minute exercise sessions per week on a stationary bike
Each session includes 1-2x 10-20 second sprints
3 testing periods - 1 hour each
8 weeks total - 2 weeks rest, 6 weeks of exercise

To participate contact Michael Makela,
mmakela@lakeheadu.ca or 807-251-9413

Research conducted by Michael Makela (MSc kinesiology student),
Supervised by Dr. Ian Newhouse

Version 1 - March 2019

Appendix G

Information Handout

Lakehead
UNIVERSITY

CAN ONLY 2-MINUTES OF HIGH-INTENSITY EXERCISE PER WEEK IMPROVE YOUR HEALTH?

WE NEED YOUR HELP TO FIND OUT!

Looking for healthy males and females, ages 18 - 45, who exercise less than 150 minutes of moderate exercise or 75 minutes of vigorous exercise per week

To participate please contact Michael Makela, mmakela@lakeheadu.ca or 807-251-9413

What is required?

- 3x 10 minute exercise sessions per week on a stationary bike
- Each 10-minute session contains 1-2 short 10-20 second sprints
- 3 testing periods - 1 hour each
- 8 weeks total - 2 weeks of rest followed by 6 weeks of exercise

How can you benefit?

- Participation in exercise improves overall well-being
- Learn something new - exercise that includes short bursts of high-intensity is more effective
- Access to a variety of health tests
- Use the results of your tests to inform future health decisions - discuss the results with your doctor

Where?

- Lakehead University Fieldhouse
- Parking passes will be provided

Version 1 - March 2019

Appendix H

Information Letter

Dear Potential Participant,

You have been invited to take part in a study titled “Can 2 Minutes of Intense Exercise Per Week Improve Health Outcome Measures - The Effects of a Reduced-Exertion High-Intensity Interval Training (HIIT) Protocol on Measures of Cardiovascular, Metabolic, and Physical Health in Physically Inactive Individuals.” The researcher performing this study is a third-year Master’s Kinesiology student: Michael Makela, under the supervision of Dr. Ian Newhouse. The purpose of this study is to examine the effects of minimal amounts (less than two minutes per week) of high-intensity exercise on various health measures in individuals who do not meet weekly exercise guidelines. The results of this study may demonstrate that minimal amounts of high-intensity exercise can improve your health.

The present study is seeking 18 healthy male or female participants between the ages of 18 to 45 with physical activity not exceeding 150 minutes of moderate intensity or 75 minutes of vigorous intensity exercise per week. Participants must not have a history of cardiovascular, metabolic, or neurological disease, and be free of any injuries or illnesses that may impair their ability to complete the testing procedures or the exercise program. Participants will also be excluded if they are a current smoker, take hormone, steroid, diabetic, or blood pressure medication, or have hypertension. The student researcher will determine the eligibility of participants. If you are interested in taking part in this study, feel free to bring this letter home and take your time to review the study’s guidelines. If you would like to participate in this study, please contact the student researcher at mmakela@lakeheadu.ca or (807) 251-9413.

What Happens During This Study?

The present study will consist of three identical testing periods requiring two visits to the lab: one lasting 45 minutes to one hour and a second lasting five minutes following a 10-hour fast. You will be assessed before the start of the study (baseline), after a two-week no-exercise phase (pre-testing), and again following six-weeks of exercise (post-testing). During the no-exercise phase, you will be asked to maintain your regular daily routine as best as possible and not make any significant lifestyle changes (e.g., start a weight loss program). During the exercise phase, you will be required to participate in three 10 minute exercise sessions per week for six weeks. The total duration of the study is expected to be eight to nine weeks and will take place in one of the multi-purpose labs in the School of Kinesiology at Lakehead University in the Sanders Building.

What Are The Study Procedures?

At the testing periods, once you have provided consent, your height and weight will be measured. You will then be asked to hold onto a bioelectrical impedance device, which will estimate your body’s fat percentage. Next, a single lead electrocardiogram will be strapped around your chest, and you will be asked to lie on your back and relax for five minutes to assess resting heart rate (HR), heart rate variability (HRV), and resting blood pressure (BP). To test your physical fitness, you will be required to perform two exercise tests. The first is a 15-second bike sprint against 7.5% of your bodyweight resistance to measure lower body power. Next, to measure your aerobic capacity, a measure of physical fitness, you will be required to perform an incremental exercise test on a stationary bike, while a gas analysis system measures the air you breath out (VO2max). You will start off pedaling on a stationary bike at a light resistance which will increase every two minutes until you are no longer able to continue. Finally, on a separate occasion, although, part of the same testing period will be a fasting blood sugar test. A capillary blood sample will be obtained from a finger prick which you will administer yourself. This will be following a 10 hour fast, during which time you will not be permitted to consume anything by mouth except for water. For example, for an 8:00 am test before work, you would not be allowed to drink anything but water after 10:00 pm the night before. The collection of this sample is not expected to take more than five minutes. The time and date of the testing periods will be arranged at approximately the same time of day on the same day of the week at least 24 hours after the completion of the no-exercise and exercise phase. You will be instructed to refrain from consuming caffeine 6 hours before testing, avoid vigorous physical activity 24 hours prior.

Due to the nature of this study, some of the health measures during the testing periods are subject to change based on things you do in your everyday life. As such, changes in diet, exercise, and even sleep habits could potentially influence the results of the study. For this reason, we ask that you maintain your regular routine as best as possible throughout the study. The purpose of this instruction is to reduce the influence of lifestyle changes on the health measures being recorded. To monitor changes in your lifestyle, you will receive a short e-mail survey every second week throughout this study. This survey is expected to take one to two minutes to complete and will ask if you are maintaining similar daily habits before the start of the study.

What Is The Study Intervention?

The intervention in this study is the three weekly exercise sessions for six weeks following the two week no exercise period. Each 10 minute exercise session will consist of one 10 second sprint in the first session only, all other sessions will consist of two sprints starting at 10 seconds which will progress to two 15 second sprints, in week two, and to two 20 second sprints in week four. All sessions will be supervised in a multi-purpose lab at the School of Kinesiology in the Saunders Fieldhouse. If desired you may exercise in a group of one to three other individuals. Each session is expected to last no more than 20 minutes, totaling no more than one hour per week. Exercise time slots will be created based on your availability and the availability of the researcher and equipment. Complimentary parking passes will be provided to avoid any parking expenses: however, participants will be responsible for their own transportation. There are no other foreseeable expenses.

What Are The Potential Risks?

There are some minimal risks associated with participating in this study; the researcher has taken precautions to minimize risks to participants. To participate in this study, you will be required to fill out several short surveys that may make you feel uncomfortable and the finger prick needed to obtain a blood sample may cause slight pain and tenderness to the finger. There is also some social risk as some personal information will be collected and potential privacy loss from other individuals recognizing you in the lab. You also must perform several short bouts of maximal physical exertion during the exercise sessions and the lower body power test and exercise to exhaustion during the incremental exercise test. Both of these forms of exercise have some potential harm/risk. You may experience temporary shortness of breath, light-headedness, dizziness, muscle fatigue, and delayed onset muscle soreness. If you experience any adverse effects such as chest pain, unusual shortness of breath, worsening joint pain, persistent light-headedness or any other signs of injury, exercise will be stopped immediately, and medical assistance will be provided if necessary. In the event of an injury or adverse effect, your participation may be terminated. At each testing period, your blood pressure will be measured and recorded before exercise testing. You will not be allowed to continue if your blood pressure is above 140/90 mm Hg and upon completion, your blood pressure will be monitored until it returns below 140/90 mm Hg. It is impossible to know if apparently healthy individuals may have a previously undetected cardiovascular condition or may experience arrhythmia (irregular heartbeat) during maximal exercise. If a clinically relevant finding is discovered such as elevated fasting blood sugar or irregular heartbeat you will be informed immediately.

To reduce risk, before performing exercise sessions and exercise tests you will complete a warm up to prepare the body for exercise. Following exercise, a cool down will aid your body in removing waste products and reduce the risk of dizziness or fainting caused by the pooling of blood in the extremities. It is recommended that you bring a bottle of water to maintain adequate hydration throughout the testing protocol. There is no planned injury response, as Lakehead University does not compensate research participants in the event of an injury. If you require post-injury treatment, you will be required to pay out of pocket or seek compensation through private health insurance. If you experience an injury or adverse effect, please do not hesitate to contact the student research, contact information will be provided at the end of this letter.

What Are The Potential Benefits?

There is some benefit to participating in this study, previous research has shown that the amount of exercise performed in this study can increase physical fitness, which is related to lower chronic disease risk. You will gain the ability to compare your performance results to normative data. Student participants will receive experience and knowledge in regards to conducting exercise testing procedures. You will also be entitled to your results on several health measures which may yield insight into their well-being, which they may not otherwise have access. For example, you may report the results of fasting blood glucose, resting heart rate, heart rate variability, aerobic capacity, and resting blood pressure to your preferred health care provider.

What Are My Rights As a Participant?

Participating in this study is voluntary, and you may drop out at any time. Your decision to participate will not have any effect on your employment or academic status. If any questions, demands, or scenarios result in distress or discomfort, you may refuse further participation without penalty. If you choose to drop out of the study and you wish to withdraw your data you will be able to do so; however, once the study is completed, you can no longer withdraw data. You also have the right to withdraw any human biological materials; however, in this study, biological samples will be discarded once data has been collected. To withdraw from this study contact the student researcher, Michael Makela. If you have any questions regarding the conduct of this study or your rights as a research participant contact information can be found at the end of this letter.

How Will My Confidentiality Be Maintained?

All personal information and recorded data will remain completely confidential. To protect your identity, your name will not be used on documents throughout the study. Instead, you will be randomly assigned a number that will be used to identify you. Only the student researcher, Michael Makela, and the supervising professor Dr. Ian Newhouse will have access to the recorded data and personal information. During this study, digital data will be secured on a password-protected computer, and physical documentation will be stored with the supervising professor in a locked filing cabinet at Lakehead University for at least five years. When the study is finished, you will be able to view your data and will be emailed a summary, on request.

What Will My Data Be Used For?

The data collected from your participation in this study will help determine if the exercise program was effective. The results from this study will be presented in a paper and oral presentation as part of the student researcher's requirements for the completion of the Masters of Science degree in the Kinesiology program, which is expected to be completed by the end of year. Also, this study may be submitted for consideration for conferences and/or academic journals. In any case, your data will remain fully anonymous, your name and personal information will not be used to identify you in any way.

Contact Information

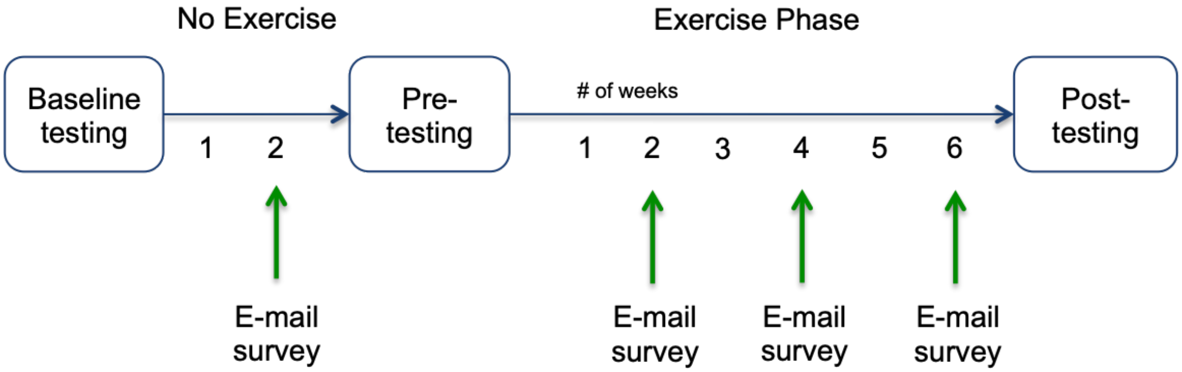
This research study has been reviewed and approved by the Thunder Bay Regional Health Sciences Centre Research Ethics Board. At any given point before, during, or after the testing periods, you may feel free to contact the researcher for inquiries. If you have any concerns regarding your rights as a research participant or wish to speak to someone other than a research team member about this research project, you are welcome to contact:

Chair, Research Ethics Board
Thunder Bay Regional Health Sciences Centre
Phone: 807-684-6422 Fax: 807-684-5904
Email: ResearchEthics_Chair@tbh.net

If you wish to participate or if you have any further question, please contact the student researcher Michael Makela at mmakela@lakeheadu.ca or (807) 251-9413.

Thank you for your time and consideration in this study.

Study Summary



Tests include:

~10 minute incremental exercise test on a stationary bike, 15 second maximal bike sprint, fasting blood sugar, resting heart rate, resting blood pressure, resting heart rate

Study requirements:

- **3** assessment periods
- **2** week no exercise phase
- **6** week exercise phase
- **Exercise** involves **3** 10-minute cycling sessions per week