

November 2013

A six week modified sprint interval training program incorporating extended exercise bouts does not increase maximal cardiac output

Alan R. Smith

The University of Western Ontario

Supervisor

Dr. Peter W.R. Lemon

The University of Western Ontario

Graduate Program in Kinesiology

A thesis submitted in partial fulfillment of the requirements for the degree in Master of Science

© Alan R. Smith 2013

Follow this and additional works at: <https://ir.lib.uwo.ca/etd>



Part of the [Exercise Science Commons](#)

Recommended Citation

Smith, Alan R., "A six week modified sprint interval training program incorporating extended exercise bouts does not increase maximal cardiac output" (2013). *Electronic Thesis and Dissertation Repository*. 1707.

<https://ir.lib.uwo.ca/etd/1707>

A SIX WEEK MODIFIED SPRINT INTERVAL TRAINING PROGRAM
INCORPORATING EXTENDED EXERCISE BOUTS DOES NOT INCREASE
MAXIMAL CARDIAC OUTPUT

(Thesis format: Monograph)

by

Alan Robert Smith

Graduate Program in Kinesiology

A thesis submitted in partial fulfillment
of the requirements for the degree of
Master of Science

The School of Graduate and Postdoctoral Studies
The University of Western Ontario
London, Ontario, Canada

© Alan Smith 2013

Abstract

Sprint interval training (SIT) improves maximal oxygen uptake ($\dot{V}O_{2\max}$) and exercise performance but not maximal cardiac output (Q_{\max}). The brevity of typical SIT bouts (30-seconds) might hinder improvements in Q_{\max} . The purpose of this study was to determine whether extended duration SIT (up to 45 second bouts) improves Q_{\max} . Pre-/Post-SIT (or control) $\dot{V}O_{2\max}$, Q_{\max} , maximum stroke volume (SV_{\max}), maximum heart rate, arterial-mixed venous oxygen difference, and 5-minute run distance were measured. SIT progressed from 4x30s to 7x45s “all-out” efforts (4 min recovery) over 6 wk (3x/wk) on a manually driven treadmill. Following SIT, $\dot{V}O_{2\max}$ improved (pre= 3.6 ± 0.8 vs post= 3.8 ± 0.8 L·min⁻¹; $p=0.012$). Increases in Q_{\max} (pre= 26.13 ± 5.09 vs post= 27.10 ± 4.82 L·min⁻¹; $p=0.166$) and SV_{\max} (pre= 138 ± 27 vs post= 144 ± 28 mL·beat⁻¹; $p=0.095$) were not significant. These data suggest 6 wk of extended SIT bouts (up to 45 s) does not increase Q_{\max} significantly and the observed increase in $\dot{V}O_{2\max}$ was due primarily to non-significant improvements in Q_{\max} and SV_{\max} .

Keywords

Interval training, oxygen uptake, exercise performance

Acknowledgments

I am very grateful to the many people who have helped me during my time at the University of Western Ontario. In particular, I must thank my supervisor, Dr. Peter Lemon. I have learned a great deal from him; his knowledge, expertise, and approach to research and teaching have greatly influenced me. I would like to thank Drs. Don Paterson and John Kowalchuk for allowing us to use their lab space to collect data. Thank you to the students in the Canadian Centre for Activity and Aging for accommodating this project and providing assistance when needed.

I must also thank my friends, colleagues, and lab mates in kinesiology. I am especially indebted to Stephanie and Dylan; without their suggestions, hard work, and involvement this thesis wouldn't have been possible.

Finally, I would like to thank my family for their constant encouragement, support, and patience.

Table of Contents

Abstract	ii
Acknowledgments.....	iii
Table of Contents	iv
List of Tables	vi
List of Figures.....	vii
List of Abbreviations	viii
List of Appendices	x
Chapter 1	1
1 Introduction	1
1.1 Exercise performance, maximal oxygen uptake, and exercise training.....	2
1.2 Endurance training	3
1.3 Interval training.....	4
1.4 Central adaptations.....	5
1.5 Peripheral adaptations	7
1.6 Summary, purpose, hypothesis	10
Chapter 2.....	12
2 Methods.....	12
2.1 Participants.....	12
2.2 Study design.....	12
2.3 Tests	14
2.4 Measurements	15
2.5 Statistical analysis.....	17
2.6 Excluded data points.....	17
Chapter 3.....	18

3	Results	18
3.1	Maximal oxygen uptake ($V\dot{O}_{2max}$; mL·kg lean mass ⁻¹ ·min ⁻¹)	18
3.2	Maximal oxygen uptake ($V\dot{O}_{2max}$; L·min ⁻¹)	19
3.3	Maximal cardiac output ($Q\dot{O}_{max}$; mL·kg lean mass ⁻¹ ·min ⁻¹)	19
3.4	Maximal cardiac output ($Q\dot{O}_{max}$; L·min ⁻¹)	20
3.5	Maximum heart rate (HR_{max} ; beats·min ⁻¹)	21
3.6	Maximum stroke volume (SV_{max} ; mL·kg lean mass ⁻¹ ·beat ⁻¹)	21
3.7	Maximum stroke volume (SV_{max} ; mL·beat ⁻¹)	21
3.8	Arterial-mixed venous oxygen difference ($a-v\dot{O}_{2diff}$; mL O ₂ ·100mL blood ⁻¹ ·kg lean mass ⁻¹)	22
3.9	Arterial-mixed venous oxygen difference ($a-v\dot{O}_{2diff}$; mL O ₂ ·100mL blood ⁻¹)...	22
3.10	Five minute run: total distance (m)	23
3.11	Five minute run: change in distance (Δd ; m)	24
	Chapter 4.....	25
4	Discussion	25
4.1	Effect on $V\dot{O}_{2max}$	25
4.2	Effect on $Q\dot{O}_{max}$	26
4.3	Effect on $a-v\dot{O}_{2diff}$	27
4.4	Effect on 5-minute run	29
4.5	Summary	30
4.6	Future directions	31
4.7	Conclusion	31
	References	32
	Appendices.....	44
	Curriculum Vitae	50

List of Tables

Table 1. Participant characteristics.	12
Table 2. Sprint interval training program.	13

List of Figures

Figure 1. Maximal oxygen uptake ($V\dot{O}_{2\max}$; mL·kg lean mass ⁻¹ ·min ⁻¹).....	18
Figure 2. Maximal cardiac output ($Q\dot{O}_{\max}$; L·min ⁻¹).....	20
Figure 3. Maximum stroke volume (SV_{\max} ; mL·beat ⁻¹).....	22
Figure 4. Arterial-mixed venous oxygen difference (a-v $\dot{O}_{2\text{diff}}$; mL O ₂ ·100mL blood ⁻¹)	23
Figure 5. Total distance run (m)	24
Figure 6. Individual changes in distance run (Δd).....	30

List of Abbreviations

Δd – change in distance

a-v \square O_{2diff} – arterial-mixed venous oxygen difference

bFbF – basic fibroblast growth factor

CON – control

ET – endurance training

FABPpm – plasma membrane fatty acid-binding protein

FAT/CD36 – fatty acid translocase/cluster of differentiation 36

GLUT4 – glucose transporter type 4

HIIT – high intensity interval training

HR – heart rate

HR_{max} – maximum heart rate

IT – interval training

LV – left ventricle

MCT4 – monocarboxylate transporter 4

MIT – moderate intensity interval training

NHE1 – sodium-hydrogen antiporter 1

NO – nitric oxide

PAR-Q – physical activity readiness questionnaire

Q \square – cardiac output

Q_{\max} – maximal cardiac output

SET – speed endurance training

SIT – sprint interval training

SV – stroke volume

SV_{\max} – maximum stroke volume

VEGF – vascular endothelial growth factor

V_{O_2} – oxygen uptake

$V_{O_{2\max}}$ – maximal oxygen uptake

List of Appendices

Appendix A: Ethics approval notice.....	44
Appendix B: Letter of information.	45
Appendix C: Informed consent statement form.....	49

Chapter 1

1 Introduction

Sprint interval training (SIT) is a low-volume, time efficient, exercise training method capable of rapidly improving exercise performance (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005). Although there are a variety of formats, typically, SIT consists of 4 to 6 bouts of 30-second “all-out” exercise bouts separated by 4 minutes of rest/recovery. SIT is vastly different from traditional high-volume endurance training (ET; constant-load, submaximal exercise maintained for an extended period of time), as SIT requires ~14 to 23 minutes (of which only 2 to 3 is actual exercise) while ET usually requires 30 to 60 minutes (of which all is exercise). SIT is performed at supramaximal intensity (~150-200% maximal aerobic power) and ET is typically performed between 60-80% of maximal aerobic power. Despite these obvious differences, SIT and ET result in similar $\dot{V}\text{O}_{2\text{max}}$ and exercise performance improvements (Gibala et al., 2006; MacPherson, Hazell, Olver, Paterson, & Lemon, 2011).

Several types of exercise training elicit key central and peripheral physiological adaptations (Blomqvist & Saltin, 1983; Holloszy & Booth, 1976). Central adaptations occur within the cardiovascular system (resulting in increased oxygen delivery) and peripheral adaptations occur at the level of the muscle (resulting in greater oxygen extraction and utilization). The improved delivery, extraction, and utilization of oxygen alter energy substrate utilization and metabolite production resulting in both improved capacities and exercise performance.

Sprint interval training and ET both cause peripheral adaptations, although greater with SIT, but differ most with regards to central adaptations (Gibala et al., 2006; MacPherson et al., 2011). Maximal cardiac output (Q_{max}), an important factor in oxygen delivery, is improved with ET but not SIT (MacPherson et al., 2011). Improvements in Q_{max} appear to be related to training volume (volume = intensity x duration) (Daussin et al., 2007; Helgerud et al., 2007). It is possible that SIT does not improve Q_{max} because exercise

interval duration is insufficient (MacPherson et al., 2011). Time efficiency and potency of SIT make it an attractive training method, but unfortunately the adaptations are not all encompassing (only peripheral adaptations are obtained). Thus, in order to obtain both central and peripheral adaptations, SIT may complement, but not replace ET.

Interestingly, other, less intense forms of interval training utilizing longer duration intervals have been shown to improve $\dot{V}\text{O}_{2\text{max}}$ (Daussin et al., 2007). This suggests that supramaximal intensity is not a requirement for central adaptations but some exercise duration is. However, neither the necessary intensity nor the duration is clear at the present time. Previous studies have not used 45-second intervals. It is possible that extending SIT bouts to 45-seconds while maintaining an “all-out” intensity would provide a time-efficient method of training that might affect $\dot{V}\text{O}_{2\text{max}}$.

The purpose of the current study was to determine if 6 weeks of modified SIT (duration of sprint intervals was lengthened from 30 up to 45 seconds) improves exercise performance and maximal cardiac output ($\dot{V}\text{O}_{2\text{max}}$). Maximal oxygen uptake, $\dot{V}\text{O}_{2\text{max}}$, and 5-minute run performance were assessed pre- and post-training, in healthy, young, male and female participants. It was hypothesized that the modified SIT protocol would generate improvements in $\dot{V}\text{O}_{2\text{max}}$, resulting in improved maximal oxygen uptake and exercise performance.

1.1 Exercise performance, maximal oxygen uptake, and exercise training

Exercise performance is determined by the integration of various body systems (respiratory, cardiovascular, and neuromuscular) (Bangsbo, Mohr, Poulsen, Perez-Gomez, & Krstrup, 2006). A progressive exercise training program providing sufficient frequency, volume, intensity, and duration will lead to improved exercise performance (Baar, 2006). Manipulating these 4 variables determines the degree to which each system is influenced and where adaptive responses occur (Jones & Carter, 2000). Maximal oxygen uptake ($\dot{V}\text{O}_{2\text{max}}$) is a general indicator of fitness and is often used to assess the effectiveness of a training program (Poole, Wilkerson, & Jones, 2008).

Maximal oxygen uptake is the greatest amount of oxygen that can be taken up, delivered to, and utilized by muscle tissue to perform exercise (measured as an absolute value: $\text{L}\cdot\text{min}^{-1}$; or, measured relative to body mass: $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) (Åstrand, 1956; Jones & Carter, 2000). Oxygen uptake ($V\dot{O}_2$) is the product of cardiac output ($Q\dot{}$; surrogate for total oxygen delivery) and arterio-venous oxygen difference (i.e. oxygen extraction/utilization) (Bevegård, 1962; González-Alonso, 2008).

Cardiac output is the volume of blood ejected from the heart per minute (measured in $\text{L}\cdot\text{min}^{-1}$). It is the product of stroke volume (SV) and heart rate (HR), and is considered to be the “central” component of any training response because the respiratory system is seldom limiting (Bassett & Howley, 2000).

Arterio-venous oxygen difference ($a\text{-}vO_{2\text{diff}}$) is a measure of oxygen extraction by the muscle. Oxygen content of blood is measured at the arterial and venous levels (i.e. before and after the capillary bed in the muscle where oxygen extraction takes place) (Clausen, Klausen, Rasmussen, & Trap-Jensen, 1973). The difference between arterial and venous oxygen content provides a measure of oxygen extraction. Changes in $a\text{-}vO_{2\text{diff}}$ following exercise training reflect adaptations that occur at the muscle (i.e. improved extraction or utilization) (Coyle et al., 1984; Ekblom, Åstrand, Saltin, Stenberg, & Wallström, 1968). These changes are considered to be “peripheral” changes (Daussin et al., 2008).

Exercise training can elicit adaptations that occur centrally and/or peripherally (Coffey & Hawley, 2007; Daussin et al., 2008). These adaptations increase $V\dot{O}_{2\text{max}}$ as well as exercise performance (Ekblom et al., 1968; Gunnarsson & Bangsbo, 2012; Jones & Carter, 2000). Endurance training (ET) and interval training (IT) are training methods used to elicit these changes. The degree of influence exerted on central and peripheral aspects is determined by the frequency, volume, intensity, and duration of the training method used (Baar, 2006; Buchheit & Laursen, 2013; Laursen, 2010).

1.2 Endurance training

As mentioned above, endurance training is exercise which is performed at a constant, submaximal intensity for an extended period of time (Jones & Carter, 2000). Endurance

training is aerobic and relies mainly on a mix of lipid and carbohydrate oxidation via the oxidative system (relatively little energy is contributed from ATP-PCr and glycolytic systems) (Jeukendrup, 2002; Spriet, 2002). Endurance training needs to be performed several times per week (at least 2-3) in order to adequately stress the body and promote positive adaptations (Laursen & Jenkins, 2002). Typically, high-volume (long duration) training results in both peripheral and central adaptations, such as increased Q_{max} and $a\text{-}v\text{O}_{2\text{diff}}$, and produces improvements in both $V\text{O}_{2\text{max}}$ and exercise performance (Coffey & Hawley, 2007; Holloszy & Booth, 1976)

1.3 Interval training

Interval training differs from traditional ET in that it is a discontinuous workload. Interval training is characterized by repeated blocks (bouts) of vigorous exercise separated by periods of rest or lower intensity exercise utilized for recovery in preparation for the next interval of vigorous exercise (Laursen & Jenkins, 2002). The combinations of exercise intensities, work to “rest” ratios, and number of intervals are endless and many IT protocols exist (Buchheit & Laursen, 2013). As a result, the frequency, volume, intensity, and duration of each protocol may differ, which makes comparisons between training methods challenging (Laursen, 2010). Additionally, identifying which factor is responsible for driving certain adaptive responses is often unclear (Laursen, 2010). Various terms for types of IT exist, including moderate intensity interval training (MIT), high intensity interval training (HIIT), speed-endurance training (SET), and sprint interval training (SIT) (Burgomaster et al., 2005; Iaia & Bangsbo, 2010; Keteyian, 2012; Talanian, Galloway, Heigenhauser, Bonen, & Spriet, 2007). Recently, SIT, a specific interval training method, has gained significant popularity with both researchers and athletes. Typically, SIT is characterized by 4-6 bouts of 30-second “all-out” exercise separated by 4 minutes of rest/recovery (Burgomaster et al., 2005; MacPherson et al., 2011). Research studies have utilized Wingate cycling tests or “manual” mode treadmill sprinting as an exercise mode primarily but any exercise mode will work. Energy during the 30-second bouts is produced primarily from anaerobic sources but as the number of bout repeats increases there is a shift towards a greater aerobic contribution (Bogdanis, Nevill, Boobis, & Lakomy, 1996). Additionally, the 4 minute recovery period relies

heavily on oxidative metabolism. Similarly to ET, SIT results in comparable improvements in exercise performance; however, these improvements in exercise performance are likely caused by peripheral adaptations, as 6 weeks of SIT does not increase $Q_{\square_{\max}}$ (Gibala et al., 2006; MacPherson et al., 2011). Sprint interval training is a low-volume, time efficient, intense training method that increases exercise performance and $V_{\square}O_{2\max}$ (via greater $a-vO_{2\text{diff}}$, but not $Q_{\square_{\max}}$) (MacPherson et al., 2011).

1.4 Central adaptations

According to the Fick principle, an increase in $Q_{\square_{\max}}$ will increase $V_{\square}O_{2\max}$ unless extraction decreases (Stray-Gundersen et al., 1986). Cardiac output is the product of SV and HR (so $Q_{\square_{\max}}$ is the product of SV_{\max} and HR_{\max}) (Stray-Gundersen et al., 1986). In response to exercise training, HR_{\max} does not increase; instead HR_{\max} decreases or is unchanged (Blomqvist & Saltin, 1983). Therefore, any increase in $Q_{\square_{\max}}$ must be the result of increased SV_{\max} (Ekblom & Hermansen, 1968).

Stroke volume is directly related to end-systolic and end-diastolic myocardial fibre length. In order to increase SV, end-diastolic fibre length must be greater and/or end-systolic fibre length must be lesser (Kiil, 1978). Stroke volume can be modulated by intrinsic (e.g. cardiac dimensions, response to inotropic agents) and extrinsic (e.g. blood volume, peripheral resistance) elements which impact upon factors affecting myocardial fibre length (Blomqvist & Saltin, 1983; Kiil, 1978). The main factors that affect myocardial fibre length are: 1) preload; 2) afterload; 3) inotropy; and 4) myocardial dimensions and performance.

Preload refers to factors that affect myocardial fibre length prior to contraction; end-diastolic volume is the principal measure of preload. The importance of preload in relation to SV can be seen at rest and during exercise in a supine position and during acute blood volume expansion. A supine position imposes a large preload on the heart due to reduced gravitational effects (more of the body is at or above heart level) and results in greater SV when compared to an upright position at both rest or during exercise for a given workload (Hopper, Coggan, & Coyle, 1988; Poliner et al., 1980). Acute expansion of blood volume, with whole blood or saline solution, leads to greater end-

diastolic volumes and improves SV (Convertino, 1991; Kanstrup & Ekblom, 1982; Robinson, Epstein, Kahler, & Braunwald, 1966). In response to training there is an increase in blood volume so preload also increases (Kjellberg, Rudhe, & Sjöstrand, 1949; Sawka, Convertino, Eichner, Schnieder, & Young, 2000). Increased preload improves SV through the Frank-Starling mechanism whereby increasing left-ventricular end-diastolic volume leads to myocardial stretching which improves contractility, producing an increased SV (Ferguson, Gledhill, Jamnik, Wiebe, & Payne, 2001; Konhilas, Irving, & de Tombe, 2002).

Afterload refers to the external forces which oppose the shortening of myocardial fibres (Kiil, 1978). If afterload is decreased, SV will increase (because end-systolic fibre length will decrease) (Blomqvist & Saltin, 1983). Afterload is mainly determined by peripheral resistance (Jaski, Fifer, Wright, Braunwald, & Colucci, 1985). Exercise training also leads to increased capillarization and improvements in the ability to vasodilate (Jensen, Bangsbo, & Hellsten, 2004; Rakobowchuk et al., 2008). These adaptations have a number of beneficial effects, among them, is a decrease in peripheral resistance (Booth & Thomason, 1991; Laughlin & Roseguini, 2008).

Inotropy refers to the response of the myocardium to substances which affect contractility. Inotropic agents include calcium, catecholamines, and glucagon (Kiil, 1978). Increased inotropy leads to increased force and velocity of contraction (Ross, Covell, Sonnenblick, & Braunwald, 1966). Stroke volume will increase in response to increased inotropy (Blomqvist & Saltin, 1983). Exercise training can enhance myocardial inotropy (Ehsani, Ogawa, Miller, Spina, & Jilka, 1991).

Myocardial performance is determined by structural or functional changes within the myocardium itself that affect contractile force (Blomqvist & Saltin, 1983). Intrinsic contractile performance is difficult to measure but studies in rats have shown contractile performance, calcium handling, and myosin ATPase improvements with exercise training (Baldwin, Cooke, & Cheadle, 1977; Penpargkul, Repke, Katz, & Scheuer, 1977). Exercise training also leads to myocardial hypertrophy and an increased left ventricle mass to body mass ratio (Kingwell, Arnold, Jennings, & Dart, 1998; Wisløff, Helgerud,

Kemi, & Ellingsen, 2001). Myocardial hypertrophy is beneficial, and not pathological, as long as left ventricular (LV) volume is not reduced simultaneously (Pluim, Zwinderman, van der Laarse, & van der Wall, 2000; Vinereanu et al., 2001). Endurance and interval training lead to myocardial hypertrophy which does not impede LV volume (Maron & Pelliccia, 2006; Pelliccia et al., 2002). Weightlifters and people suffering from hypertension also develop myocardial hypertrophy but this occurs with heart wall thickening which reduces LV volume and hampers cardiac performance and function (Haykowsky, Dressendorfer, Taylor, Mandic, & Humen, 2002; Vinereanu et al., 2001).

There are several ways of measuring adaptations to the central system. It is often difficult to determine exactly which factor is responsible for increases in SV (Blomqvist & Saltin, 1983). Most studies have used animals or isolated muscle fibres and attempted to distinguish between the different factors and how they affect SV (Glower et al., 1985; Sagawa, 1978; Sonnenblick & Downing, 1963). Measuring the independent contribution of preload, afterload, inotropy, and myocardial performance is difficult, while measuring Q_{\square} is more practical. Cardiac output can be measured invasively (direct Fick method, dye-dilution, thermodilution) and non-invasively (acetylene breathing methods, carbon dioxide rebreath, Doppler ultrasound, impedance). Invasive techniques pose greater risks, require medical and technical assistance, and are not always practical during maximal exercise (Warburton, Haykowsky, Quinney, Humen, & Teo, 1999). Less invasive techniques, particularly open-circuit acetylene methods, are reliable, reproducible, correlate well with values produced from invasive methods, and are applicable to conditions of maximal exercise. Acetylene is a gas which is soluble in blood and inert (does not bind with haemoglobin). Typically, inspired and expired acetylene concentrations are measured by a mass spectrometer over a number of breaths and rate of disappearance is calculated. As cardiac output increases, the rate of acetylene disappearance increases. An open circuit acetylene method is an effective and reliable method for measuring maximal cardiac output during exercise (Johnson et al., 2000).

1.5 Peripheral adaptations

Peripheral adaptations are those that occur at the muscle and allow oxygen to be extracted/utilized to a greater extent (ultimately increasing $a-vO_{2\text{diff}}$). Changes in muscle

metabolism (glycolytic capacity, oxidative capacity, substrate utilization), glycogen storage, ion transporters, fibre type, haemoglobin/myoglobin concentration, and capillary density reflect peripheral adaptations (Coffey & Hawley, 2007; Holloszy & Coyle, 1984; Iaia & Bangsbo, 2010).

Improvements in muscle metabolism are related to the energy system stressed by the exercise task. Peripherally, endurance training results mainly in improvements to the oxidative capacity of the muscle whereas interval training augments glycolytic and/or oxidative capacity of muscle (depending on which interval training method is used) (Gollnick & Saltin, 1982; Hawley, 2002; Kubukeli, Noakes, & Dennis, 2002; MacDougall et al., 1998).

Glycolysis is an energy liberating process that involves the breakdown of glucose/glycogen. Glycolysis takes place in the cell cytoplasm and does not require oxygen to be present. The net result of glycolysis alone is the production of 2 mol of ATP per mol of glucose (Alberts et al., 2004). Glycolytic capacity is often assessed by measuring the concentration and activity of various glycolytic enzymes. Most commonly, phosphofructokinase, pyruvate kinase, lactate dehydrogenase, glyceraldehyde-3-phosphate dehydrogenase, or aldolase are measured (MacDougall et al., 1998; Mujika & Padilla, 2001; Parra, Cadefau, Rodas, Amigó, & Cussó, 2000). Generally, glycolytic capacity is unaffected by ET but increases in response to interval training (Gollnick et al., 1973; MacDougall et al., 1998).

The oxidative energy system includes the citric acid cycle and electron transport chain. This system relies on a special organelle, called the mitochondria, and oxygen to produce energy. Fat, carbohydrate, and protein can be used as energy sources (fat and carbohydrate being used most often during exercise) (Brooks & Mercier, 1994). Energy production from oxidation is very efficient (oxidation of 1 mol of glucose produces ~30 mol of ATP) but the rate of production is slower in comparison to other energy systems because of the limitation of oxygen delivery (Alberts et al., 2004; Flück & Hoppeler, 2003). Different enzymes from the citric acid cycle or electron transport system as well as the number, size, and function of muscle mitochondria are used often as measures of

oxidative capacity. Specifically, studies often investigate the activity and/or concentration of citrate synthase, 3-hydroxyacyl-CoA dehydrogenase, succinate dehydrogenase, pyruvate dehydrogenase, malate dehydrogenase, and cytochrome c oxidase (Dudley, Abraham, & Terjung, 1982; Irrcher, Adihetty, Joseph, Ljubicic, & Hood, 2003; Mujika & Padilla, 2001). Oxidative capacity increases in response to both ET and SIT (Gibala et al., 2006; Ingjer, 1979; MacDougall et al., 1998).

In addition to affecting muscle glycolytic and oxidative capacity, exercise training can influence substrate utilization. Following training, glucose (GLUT4) and fatty acid (FAT/CD36 and FABPpm) transporter contents increase, muscle glycogen content increases, intramuscular triglyceride content increases, and there is a shift in substrate utilization with an increase in the relative contribution of fat and a decrease in the relative contribution of carbohydrate at a given workload (Burgomaster et al., 2005; Hawley, 2002; Holloszy & Booth, 1976; Juel, 2006).

In addition to GLUT4 and fatty acid transporters, there are other sarcolemmal transport proteins that are affected by exercise. Ion transporters, such as Na^+/K^+ -ATPase, NKCC1, NHE1, MCT1, MCT4, La^+/H^+ , and $\text{Na}^+/\text{HCO}_3^-$, exist to prevent disruptions in cellular homeostasis (Juel, 2006). The transition from rest to exercise imposes an immediate increased energy demand. The resulting metabolic products and processes cause ionic imbalances which disrupt cellular homeostasis and have a tremendous influence on muscle function (Cairns & Lindinger, 2008). Changes in muscular ionic concentrations, muscle pH, and metabolite concentrations affect muscle membrane potential, muscle excitability, muscle contractility, and contribute to muscular fatigue (Allen, Lamb, & Westerblad, 2008). Exercise training can increase the muscle content of ion transporter proteins which improves exercise performance by delaying or sequestering ion imbalance related fatigue (Iaia et al., 2008; Juel, 2006; Juel et al., 2004). Endurance training and interval training promote increased protein content of most ion transporters (Juel & Halestrap, 1999; Juel, 2006; McKenna, 1995).

An important determinant of oxygen uptake at the muscle is the oxygen carrying capacity of the blood. In blood, 98% of oxygen is bound to haemoglobin (oxyhaemoglobin), and

the remainder is dissolved. Haemoglobin binds oxygen at the lung and transports it to the muscle where it is off-loaded to muscle (Wilmore & Costill, 2004a). Conditions during exercise leads to the Bohr effect where the affinity of haemoglobin for oxygen is decreased in the presence lower pH, increased temperature, and the increase in certain exercise-related metabolites (Krustrup, Hellsten, & Bangsbo, 2004). The Bohr effect promotes oxygen off-loading at the muscle without having much effect on loading in the lung due to the shape of the oxygen-haemoglobin dissociation curve (Thomson, Dempsey, Chosy, Shahidi, & Reddan, 1974). Increases in production of red blood cells and haemoglobin are induced by the hormone erythropoietin (whether endogenous or exogenous) (Silverthorn, 2004). Increases in haemoglobin, irrespective of changes in blood volume, improve exercise performance and oxygen uptake (Kanstrup & Ekblom, 1984). A molecule similar to haemoglobin, myoglobin, is found in muscle tissue. Oxygen binds to myoglobin (oxymyoglobin) facilitating oxygen transport to the mitochondria (myoglobin also acts as an oxygen store) (Wilmore & Costill, 2004b). Theoretically, an increase in myoglobin could improve $\dot{V}O_{2\max}$ but studies have produced equivocal results thus far (Coyle et al., 1984; Jürgens, Papadopoulos, Peters, & Gros, 2000).

The formation of new blood vessels is called angiogenesis. Angiogenesis occurs in response to mechanical (e.g. shear stress, muscle stretch) and chemical (e.g. VEGF, bFbF, NO, MMPs, integrins) stimuli that are produced during exercise (Egginton, 2009). Of particular importance to the exercise response is the proliferation of new muscle capillaries. Increased muscle capillary density improves gas and metabolite exchange between blood and muscle by 1) increasing surface area of capillary in muscle, 2) increasing blood transit time in capillary beds, and 3) reducing the diffusion distance between blood and muscle cells (Iaia et al., 2011; Saltin, 2009).

1.6 Summary, purpose, hypothesis

Recently, SIT has received much attention because it is a time-efficient method of training and, despite its low volume, results in similar oxidative and exercise performance improvements as ET (Gibala et al., 2006; MacPherson et al., 2011). However, SIT does not elicit improvements in \dot{Q}_{\max} (MacPherson et al., 2011). The purpose of this study was to determine if 6 weeks of a modified SIT protocol, that utilizes longer work periods

(45 instead of 30 seconds), would elicit improvements in Q_{\max} . We hypothesized that 45-second intervals would be a sufficient duration to improve Q_{\max} .

Chapter 2

2 Methods

2.1 Participants

Twenty men and 18 women volunteered for this study. Twenty-eight (14 men; 14 women) performed sprint interval training and 10 (6 men; 4 women) acted as sedentary controls (Table 1). Participants were recreationally active and had not performed interval training for at least 3 months prior to the start of the study. All were given a letter of information regarding this study. Several control subjects were not able to complete the post testing due to noise complaints during the treadmill testing. As a result, the control group is small than planned. Experimental procedures and potential risks were outlined before participants each gave his/her written consent to participate. In addition, participants completed a PAR-Q to screen for any potential contraindications to exercise. This study was approved by the University of Western Ontario ethics board.

Table 1. Participant characteristics.

	Age (y)	Height (cm)	Body Mass (kg)	$\dot{V}\text{O}_{2\text{max}}$ ($\text{L}\cdot\text{min}^{-1}$)
SIT (n=28)	23.0 \pm 4.1	170.3 \pm 7.6	71.7 \pm 11.1	3.6 \pm 0.8
CON (n=10)	23.8 \pm 2.3	170.3 \pm 7.8	69.9 \pm 12.8	3.8 \pm 0.9

***P<0.05; SIT = sprint interval training; CON = control.**

2.2 Study design

Participants: Participants were placed randomly into either the sprint interval training (SIT) or the control (CON) group. All were familiarized with the testing procedures prior to the study. Pre- and post-testing consisted of a maximal incremental treadmill test, a constant load verification test, a 5-minute manual treadmill performance run, and body composition analyses. These exercise tests were used to assess $\dot{V}\text{O}_{2\text{max}}$, \dot{Q}_{max} , $a\text{-}\dot{v}\text{O}_{2\text{diff}}$, SV_{max} , HR_{max} and exercise performance. Body composition was measured via air displacement plethysmography (BodPod®) and used to normalize $\dot{V}\text{O}_{2\text{max}}$, \dot{Q}_{max} , $a\text{-}\dot{v}\text{O}_{2\text{diff}}$, and SV_{max} relative to lean mass. Post-training testing began between 48 and

72 hours after the final training session. Prior to testing, participants refrained from alcohol, nicotine, and caffeine for 12 hours and fasted for 3 hours.

Training: Participants assigned to the training group performed 18 sessions of modified SIT over 6 weeks (Table 2). Specifically, training was performed 3 times per week and sessions were separated by at least 48 hours. Training sessions consisted of “all out” sprints on a manual treadmill (Desmo Pro; Woodway, Waukesha, WI), progressing from 4 x 30-second sprints to 7 x 45-second sprints. Sprints were separated by 4 minutes of active rest/recovery. Training took place on a treadmill set in manual mode. Manual mode is an un-motorized mode in which the tread is propelled solely by the muscular actions of the exerciser.

Table 2. Sprint interval training program.

Day	# of sprints	Sprint duration (s)
1,2	4	30
3,4	4	35
5,6	4	40
7,8,9	4	45
10,11,12	5	45
13,14,15	6	45
16,17,18	7	45

s = seconds

Familiarization: Prior to the initial testing, participants visited the lab to complete a PAR-Q questionnaire (Thomas, Reading, & Shephard, 1992). During this visit participants were introduced to the lab, the testing equipment, and all procedures to be used.

Controls: Individuals assigned to the control group did not partake in any exercise training. Controls performed the battery of tests twice. These testing periods were separated by 6 weeks.

2.3 Tests

Maximal Incremental Exercise Test: This test was performed on a motorized treadmill (Model TM310, Trackmaster; Jas Fitness System, Newton, KS). Following 5 minutes of seated rest, participants were familiarized with our open-circuit acetylene cardiac output measurement and 4 resting cardiac output measurements were taken (2 seated followed by 2 standing measurements). Resting measurements provided submaximal reference values and served as practice for participants. Next, participants performed a 5-minute warm-up jog at 9.7 (for men) or 8.0 km·h⁻¹ (for women). Immediately following the warm-up, participants began the incremental exercise test. The test started at a speed of 10.5 (for men) or 8.9 km·h⁻¹ (for women) and increased 0.8 km·h⁻¹ every minute until participants reached volitional exhaustion. $\dot{V}\text{O}_2$ (mass spectrometry) and heart rate (ECG) were measured continuously throughout the test. A cardiac output measurement was taken when participants indicated they were near exhaustion (approximately 30 seconds remaining).

Constant Load Verification Test: Following the maximal incremental exercise test, participants were given 5 minutes of active rest/recovery. Then, they began running at a speed equal to 90% of the final speed achieved during the incremental test. Speed remained constant throughout the test and participants were instructed to run as long as possible. Another cardiac output measurement was taken 2 minute into the verification test and used as a submaximal reference value and a third cardiac output measurement was taken when participants indicated they were nearing exhaustion (approximately 30 seconds remaining). $\dot{V}\text{O}_2$ (mass spectrometry) and HR (ECG) were measured continuously throughout the test. This test was used to confirm that the $\dot{V}\text{O}_2$ and Q_{max} values from the incremental test were in fact maximal.

Five Minute Performance Run: Participants completed a 5-minute run for distance on a treadmill (Desmo Pro; Woodway, Waukesha, WI) in manual mode. They were instructed to run as far as possible in 5 minutes. Feedback on the remaining time was provided. Heart rate, speed, and distance were recorded continuously (MED-PRO; Woodway, Waukesha, WI). After the test was explained a short demonstration of running in manual

mode was given. Two 5-minute runs were completed (separated by at least 24 hours) and the greatest distance covered was recorded to minimize any learning effect.

Body Composition: Air displacement plethysmography (BodPod®) was used to assess body density. Participants were tested following a 3 hour fast. They wore approved clothing (compression shorts for men, bathing suit or compression shorts plus sports bra for women), placed a lycra cap on their head to minimize hair volume, and removed all jewelry. Thoracic air volume was predicted using an equation from the BodPod® software. Body density was used in the Siri equation to calculate body composition (% fat mass and % lean mass) (Siri, 1961).

2.4 Measurements

Oxygen uptake, heart rate, and cardiac output were measured. Stroke volume and arterial-mixed venous oxygen difference were calculated.

Oxygen Uptake ($\dot{V}O_2$): $\dot{V}O_2$ was measured continuously during the maximal incremental test and verification test. A low dead space (90 mL) bidirectional turbine (VMM-110; Alpha Technologies, Laguna Beach, CA), calibrated prior to testing with a 3.003 L syringe, was used to measure inspired and expired flow rates. The mass spectrometer (1100 Spectrometer; Perkin-Elmer, Ontario, Canada) used was calibrated with precision-analyzed gas mixtures. Inspired and expired gases were sampled continuously (50 Hz) and analyzed for concentrations of O_2 , CO_2 , and N_2 . As reported previously, changes in gas concentrations were aligned with gas volumes by measuring the time delay for a square-wave bolus of gas passing the turbine to the resulting changes in fractional gas concentrations as measured by the mass spectrometer. Breath-by-breath alveolar gas exchange was calculated by using algorithms of Beaver et al. (Beaver, Lamarra, & Wasserman, 1981). $\dot{V}O_2$ values were averaged over 20 seconds. The greatest averaged value was taken as $\dot{V}O_{2max}$. During pre-testing, 5 out of 38 participants showed a plateau in $\dot{V}O_2$ (oxygen uptake increased <50% of the estimated oxygen requirement), 20 out of 38 reached an RER greater than 1.15, and 31 out of 38 achieved 95% of their age-predicted HR_{max} . During post-testing, 12 out of 38 participants showed a plateau in $\dot{V}O_2$ (oxygen uptake increased <50% of oxygen requirement), 20

out of 38 reached an RER greater than 1.15, and 28 out of 38 achieved 95% of their age-predicted HR_{max} .

Heart Rate (HR): HR was monitored continuously by electrocardiogram (three-lead arrangement) using PowerLab L132/ML880 (ADInstruments, Colorado Springs, CO, USA) and recorded using LabChart v7.1 (ADInstruments, Colorado Springs, CO, USA). HR was calculated using a human ECG macro that was included with the software.

Cardiac Output (Q_{CO}): An acetylene (C_2H_2) non-rebreathing technique (breathing a C_2H_2 gas mixture in an open-circuit system) was used to measure Q_{CO} at rest and during exercise.

For acetylene non-rebreathing, participants were connected to a one-way valve assembly with a two-way valve on the inspired side. One input was room air and the other a gas mixture containing known concentrations of acetylene (C_2H_2) (0.698%), helium (He) (8.99%), O_2 (21%), and N_2 (BAL)). The gas mixture was released from the gas tank into a plastic bag (Douglas bag; Hans Rudolph, Inc.). This plastic bag was subsequently attached to the valve leading to the mouth piece. At the end of expiration the valve was turned and inspired air changed from room air to the acetylene mixture. Concentrations of C_2H_2 and He were then measured continuously for 10 breaths by mass spectrometry. Digitized signals were interpreted by a commercially available software program (BIPS). After 10 breaths, the valve was switched from acetylene mixture back to room air at the end of expiration. Q_{CO} was calculated based on computer analysis of the rate of disappearance of acetylene (Johnson et al., 2000).

Stroke Volume (SV): Using HR and Q_{CO} values, SV was calculated as

$$SV = Q_{CO} \div HR$$

Arterial-mixed venous oxygen difference ($a-vO_{2diff}$): Using V_{O_2} and Q_{CO} values, $a-vO_{2diff}$ was calculated as:

$$a-vO_{2diff} = V_{O_2} \div Q_{CO}$$

2.5 Statistical analysis

Statistical analyses were performed using SigmaPlot for Windows (Version 12.0). All data, except 5-minute run Δd , were analyzed using a two-way repeated measures ANOVA. Tukey's HSD test was used for post-hoc analysis of any significant interactions or main effects. A two-tailed, paired t-test was used to analyze 5-minute run Δd . Significance was set at a $P < 0.05$. Data are presented as means \pm standard deviation (SD). All results are presented as group data, but when applicable (findings diverge from group results), sex differences are reported.

2.6 Excluded data points

During post-testing, 2 subjects (a male SIT participant and a male CON participant) had non-physiological $Q_{\square_{\max}}$ values. During post-testing for another subject (a female SIT participant), we had technical issues while measuring $Q_{\square_{\max}}$. An error occurred with the computer calculations and a Q_{\square} value was never produced. Maximal oxygen uptake and HR_{\max} data had an n of 38 but, due to the issues mentioned above, Q_{\max} , SV_{\max} , and $a-v_{\square}O_{2\text{diff}}$ had an n of 35. Additionally, one male control subject who participated in the fall/winter went home for winter holidays before performing his post-testing 5-minute runs. As a result, the 5-minute run data had an n of 37.

Chapter 3

3 Results

3.1 Maximal oxygen uptake ($V\dot{O}_{2\max}$; mL·kg lean mass⁻¹·min⁻¹)

Group Data $V\dot{O}_{2\max}$: There was a significant group by time interaction ($p=0.032$). There was a 2.3 mL·kg lean mass⁻¹·min⁻¹ increase in $V\dot{O}_{2\max}$ following SIT (pre= 64 ± 6.9 post= 66 ± 6.1 mL·kg lean mass⁻¹·min⁻¹; $p=0.024$) and $V\dot{O}_{2\max}$ did not change following control (pre= 65 ± 6.9 post= 63 ± 7.4 mL·kg lean mass⁻¹·min⁻¹; $p=0.244$) conditions.

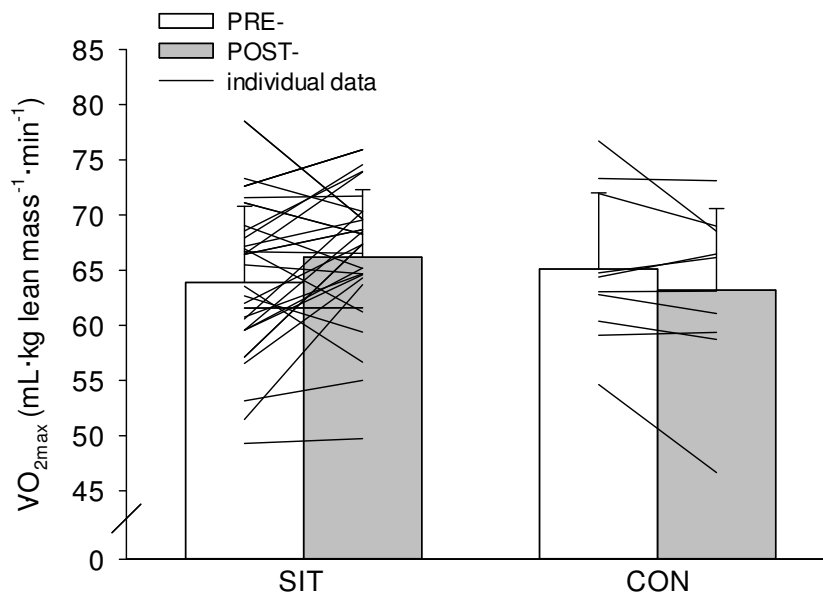


Figure 1. Maximal oxygen uptake ($V\dot{O}_{2\max}$; mL·kg lean mass⁻¹·min⁻¹) before (PRE) and after (POST) 6 weeks of SIT and CON conditions. Group data. Values are means \pm SD. * $P < 0.05$ (POST significantly greater than PRE). SIT = sprint interval training; CON = control; lines are individual data.

Sex Differences $V\dot{O}_{2\max}$: There was a significant group by time interaction for $V\dot{O}_{2\max}$ ($p=0.013$). There was a 3.3 mL·kg lean mass⁻¹·min⁻¹ increase in $V\dot{O}_{2\max}$ following SIT (pre= 64 ± 6.3 post= 67 ± 7.2 mL·kg lean mass⁻¹·min⁻¹; $p=0.015$) and $V\dot{O}_{2\max}$ did not

change following control (pre= 67 ± 8.7 post= 64 ± 9.6 mL·kg lean mass⁻¹·min⁻¹; p=0.150) conditions in men.

There were no main or interaction effects for $V\dot{O}_{2\max}$ in women ($p \geq 0.427$). There was no change in $V\dot{O}_{2\max}$ following SIT (pre= 64 ± 7.6 post= 66 ± 4.9 mL·kg lean mass⁻¹·min⁻¹; p=0.604) or control (pre= 63 ± 1.8 post= 62 ± 3.2 mL·kg lean mass⁻¹·min⁻¹; p=0.604) conditions in women.

3.2 Maximal oxygen uptake ($V\dot{O}_{2\max}$; L·min⁻¹)

Group Data $V\dot{O}_{2\max}$: There was a significant group by time interaction ($p=0.014$). There was a 0.14 L·min⁻¹ increase in $V\dot{O}_{2\max}$ following SIT (pre= 3.62 ± 0.78 vs post= 3.76 ± 0.80 L·min⁻¹; p=0.012) and $V\dot{O}_{2\max}$ did not change following control (pre= 3.82 ± 0.92 vs post= 3.69 ± 0.84 L·min⁻¹; p=0.160) conditions.

Sex Differences $V\dot{O}_{2\max}$: There was a significant group by time interaction for $V\dot{O}_{2\max}$ in men ($p=0.004$). There was a 0.18 L·min⁻¹ improvement in $V\dot{O}_{2\max}$ following SIT (pre= 4.22 ± 0.59 vs post= 4.40 ± 0.61 L·min⁻¹; p=0.018) in men. There was a 0.24 L·min⁻¹ decline in $V\dot{O}_{2\max}$ following control (pre= 4.38 ± 0.71 vs post= 4.13 ± 0.76 L·min⁻¹; p=0.035) conditions in men.

There were no main or interaction effects for $V\dot{O}_{2\max}$ in women ($p \geq 0.363$). There was no significant change in $V\dot{O}_{2\max}$ following SIT (pre= 3.03 ± 0.40 vs post= 3.13 ± 0.30 L·min⁻¹; p=0.788) or control (pre= 2.98 ± 0.35 vs post= 3.04 ± 0.44 L·min⁻¹; p=0.788) conditions in women.

3.3 Maximal cardiac output ($Q\dot{O}_{\max}$; mL·kg lean mass⁻¹·min⁻¹)

Group Data $Q\dot{O}_{\max}$: There were no main or interaction effects for $Q\dot{O}_{\max}$ ($p \geq 0.136$). There was no change in $Q\dot{O}_{\max}$ following SIT (pre= 466 ± 56.9 vs post= 484 ± 58.4 mL·kg lean mass⁻¹·min⁻¹; p=0.181) or control (pre= 447 ± 65.2 vs post= 439 ± 62.5 mL·kg lean mass⁻¹·min⁻¹; p=0.181) conditions.

Sex Differences \dot{Q}_{\max} : There were no main or interaction effects for \dot{Q}_{\max} ($p \geq 0.066$). There was no change in \dot{Q}_{\max} following SIT (pre= 467 ± 54 vs post= 477 ± 49 mL·kg lean mass⁻¹·min⁻¹; $p=0.245$) or control (pre= 426 ± 71 vs post= 409 ± 66 mL·kg lean mass⁻¹·min⁻¹; $p=0.245$) conditions in men.

There were no main or interaction effects for \dot{Q}_{\max} ($p \geq 0.436$). There was no change in \dot{Q}_{\max} following SIT (pre= 464 ± 61 vs post= 490 ± 68 mL·kg lean mass⁻¹·min⁻¹; $p=0.484$) or control (pre= 474 ± 54 vs post= 475 ± 37 mL·kg lean mass⁻¹·min⁻¹; $p=0.484$) conditions in women.

3.4 Maximal cardiac output (\dot{Q}_{\max} ; L·min⁻¹)

Group Data \dot{Q}_{\max} : There were no main or interaction effects for \dot{Q}_{\max} ($p \geq 0.166$). There was no significant change in \dot{Q}_{\max} following SIT (pre= 26.13 ± 5.09 vs post= 27.10 ± 4.82 L·min⁻¹; $p=0.166$) or control (pre= 25.23 ± 4.53 vs post= 24.68 ± 2.92 L·min⁻¹; $p=0.166$) conditions.

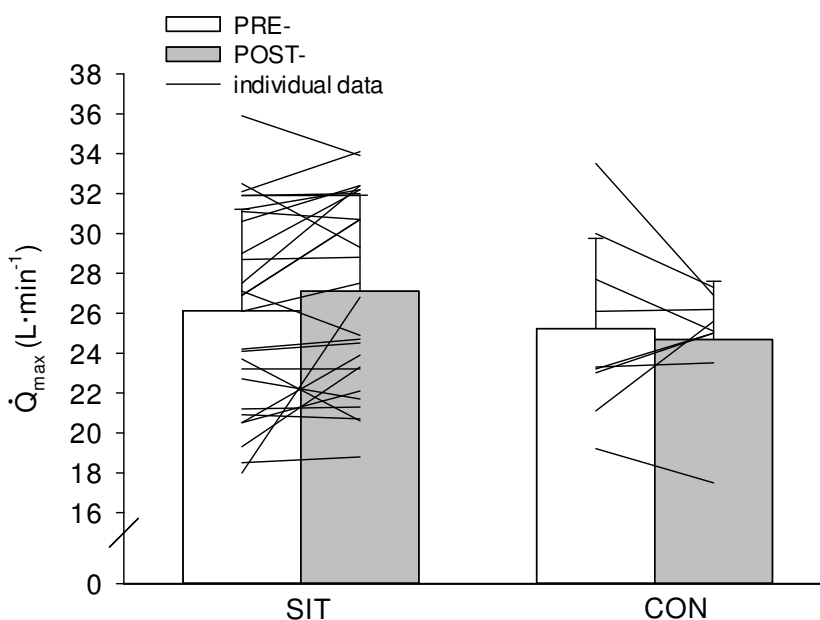


Figure 2. Maximal cardiac output (\dot{Q}_{\max} ; L·min⁻¹) before (PRE) and after (POST) 6 weeks of SIT and CON conditions. Group data. Values are means \pm SD. SIT = sprint interval training; CON = control; lines are individual data.

Sex Differences $Q_{\square_{\max}}$: There was a main effect of group on $Q_{\square_{\max}}$ (SIT=31±2.6 vs CON=27±3.2 L·min⁻¹; p=0.010) in men. There was no change in $Q_{\square_{\max}}$ following SIT (pre= 30±2.7 vs post=31±2.6 L·min⁻¹; p=0.145) or control (pre= 28±4.5 vs post=26±1.1 L·min⁻¹; p=0.145) conditions in men.

There were no main or interaction effects for $Q_{\square_{\max}}$ (p≥0.200). There was no change in $Q_{\square_{\max}}$ following SIT (pre= 22±2.7 vs post=23±3.4 L·min⁻¹; p=0.671) or control (pre= 22±3.0 vs post= 23±4.0 L·min⁻¹; p=0.671) conditions in women.

3.5 Maximum heart rate (HR_{\max} ; beats·min⁻¹)

Group Data HR_{\max} : There were no main or interaction effects for HR_{\max} (p≥0.135). There was no change in HR_{\max} following SIT (pre= 190±8 vs post= 189±8 beats·min⁻¹; p=0.243) or control (pre= 185±7 vs post= 186±6 beats·min⁻¹; p= 0.243) conditions.

3.6 Maximum stroke volume (SV_{\max} ; mL·kg lean mass⁻¹·beat⁻¹)

Group Data SV_{\max} : There were no main or interaction effects for SV_{\max} (p≥0.109). There was no significant change in SV_{\max} following SIT (pre=2.5±0.3 vs post=2.6±0.3 mL·kg lean mass⁻¹·beat⁻¹; p=0.109) or control (pre=2.4±0.4 vs post=2.4±0.3 mL·kg lean mass⁻¹·beat⁻¹; p=0.109) conditions.

3.7 Maximum stroke volume (SV_{\max} ; mL·beat⁻¹)

Group Data SV_{\max} : There were no main or interaction effects for SV_{\max} (p≥0.095). There was no significant change in SV_{\max} following SIT (pre= 138±27 vs post=144±28 mL·beat⁻¹; p=0.095) or control (pre=137±27 vs post=134±17 mL·beat⁻¹; p=0.095) conditions.

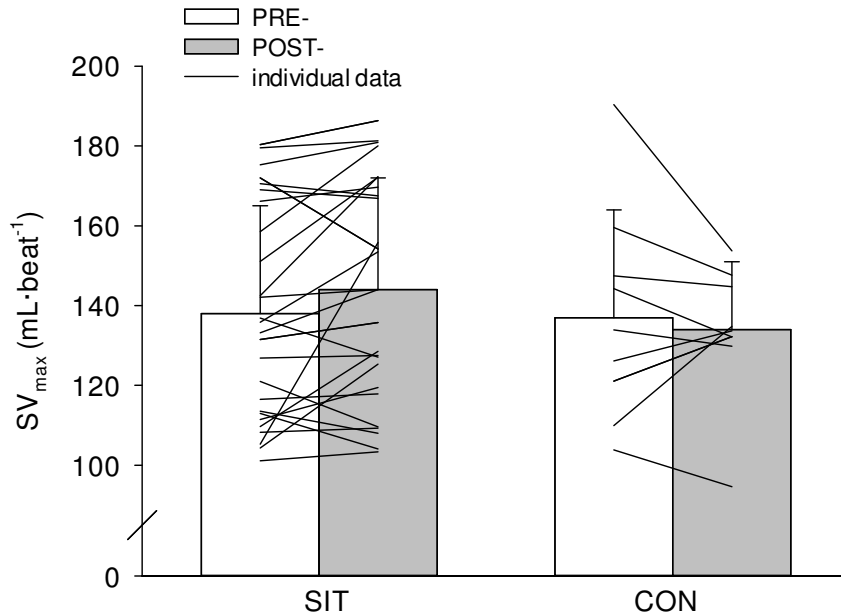


Figure 3. Maximum stroke volume (SV_{\max} ; $\text{mL}\cdot\text{beat}^{-1}$) before (PRE) and after (POST) 6 weeks of SIT and CON conditions. Group data. Values are means \pm SD. SIT = sprint interval training; CON = control; lines are individual data.

3.8 Arterial-mixed venous oxygen difference ($a-v\Box O_{2\text{diff}}$; $\text{mL O}_2\cdot 100\text{mL blood}^{-1}\cdot\text{kg lean mass}^{-1}$)

Group Data $a-v\Box O_{2\text{diff}}$: There were no main or interaction effects for $a-v\Box O_{2\text{diff}}$ ($p\geq 0.683$). There was no change in $a-v\Box O_{2\text{diff}}$ following SIT (pre= 0.25 ± 0.05 vs post= 0.25 ± 0.05 $\text{mL O}_2\cdot 100\text{mL blood}^{-1}\cdot\text{kg lean mass}^{-1}$; $p=0.843$) or control (pre= 0.26 ± 0.04 vs post= 0.25 ± 0.04 $\text{mL O}_2\cdot 100\text{mL blood}^{-1}\cdot\text{kg lean mass}^{-1}$; $p=0.843$) conditions.

3.9 Arterial-mixed venous oxygen difference ($a-v\Box O_{2\text{diff}}$; $\text{mL O}_2\cdot 100\text{mL blood}^{-1}$)

Group Data $a-v\Box O_{2\text{diff}}$: There were no main or interaction effects for $a-v\Box O_{2\text{diff}}$ ($p\geq 0.292$). There was no change in $a-v\Box O_{2\text{diff}}$ following SIT (pre= 13.8 ± 1.6 vs post= 13.8 ± 1.9 $\text{mL O}_2\cdot 100\text{mL blood}^{-1}$; $p=0.803$) or control (pre= 14.5 ± 1.5 vs post= 14.4 ± 2.2 $\text{mL O}_2\cdot 100\text{mL blood}^{-1}$; $p=0.803$) conditions.

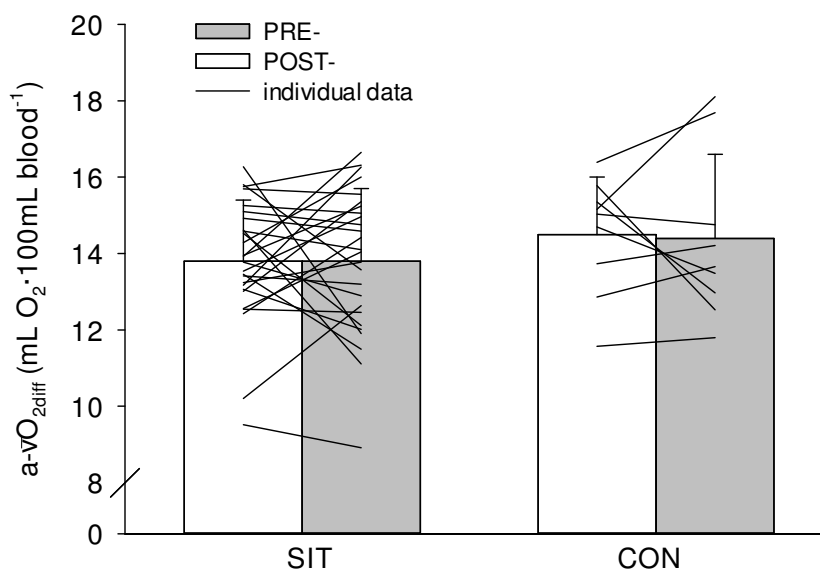


Figure 4. Arterial-mixed venous oxygen difference ($a-v\text{O}_{2\text{diff}}$; mL O₂·100mL blood⁻¹) before (PRE) and after (POST) 6 weeks of SIT and CON conditions. Group data. Values are means \pm SD. SIT = sprint interval training; CON = control; lines are individual data.

3.10 Five minute run: total distance (m)

Group Data Total Distance Run: There was a significant group by time interaction ($p < 0.001$) for total distance run (m). There was no improvement in total distance run following SIT (pre= 701.1 ± 146.2 vs post= 717.0 ± 151.5 m; $p = 0.087$) and there was a 60.3 m decrease in total distance run following control (pre= 777.8 ± 181.3 vs post= 717.5 ± 149.8 m; $p < 0.001$) conditions.

Sex Differences Total Distance Run: There were no main or interaction effects for total distance run in women ($p \geq 0.370$). There was no change in total distance run following SIT (pre= 583.2 ± 67.7 vs post= 595.0 ± 62.8 m; $p = 0.370$) or control (pre= 626.8 ± 75.0 vs post= 616.8 ± 122.6 m; $p = 0.370$) conditions in women.

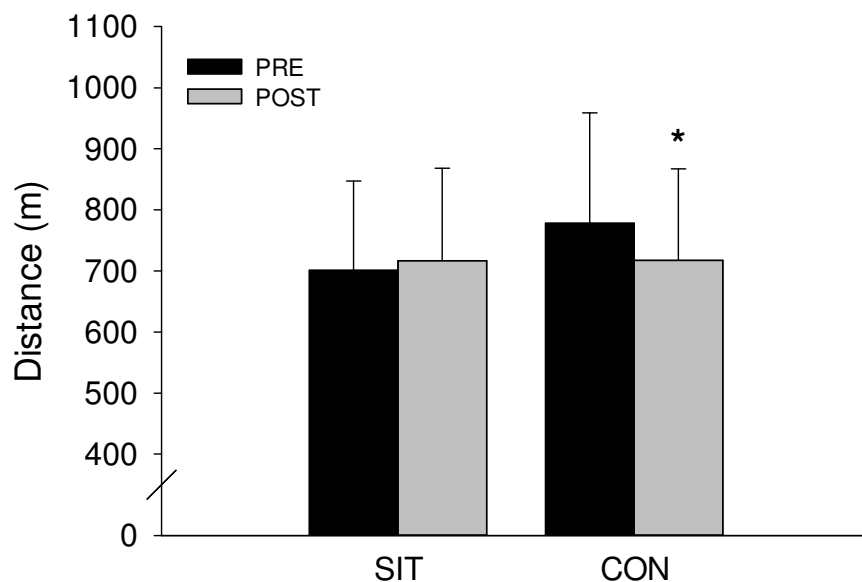


Figure 5. Total distance run (m) during 5-minute run before (PRE) and after (POST) 6 weeks of SIT and CON conditions. Group data. Values are means \pm SD. * $P < 0.05$ (POST significantly less than PRE). SIT = sprint interval training; CON = control.

3.11 Five minute run: change in distance (Δd ; m)

Group Data Δd : There was a significant Δd achieved during the 5 min run following SIT compared with control conditions (SIT= 15.94 ± 42.39 vs CON= -60.27 ± 62.76 m; $p=0.0002$).

Sex Differences Δd : There was no change in Δd achieved during the 5 min run following SIT compared with control conditions in women (SIT= 11.71 ± 36.74 vs CON= -10.00 ± 58.31 m; $p=0.370$).

Chapter 4

4 Discussion

The major findings from the present study were that increasing sprint interval exercise bout duration from 30 to 45 seconds resulted in an improved $\dot{V}\text{O}_{2\text{max}}$. Also, there was a 4% non-significant increase in \dot{Q}_{max} whereas $a\text{-}\dot{v}\text{O}_{2\text{diff}}$ was essentially unchanged following training. Distance completed in the 5-minute run was not improved but there was likely a real improvement in exercise performance with SIT that was masked by the nature of that particular test (see below for details).

4.1 Effect on $\dot{V}\text{O}_{2\text{max}}$

As in other SIT studies, $\dot{V}\text{O}_{2\text{max}}$ was increased following training. However, in the present study, $\dot{V}\text{O}_{2\text{max}}$ was only increased by 3% which is smaller than the 7 to 12% increases seen by others (Burgomaster et al., 2008; MacDougall et al., 1998; MacPherson et al., 2011). Participants' baseline $\dot{V}\text{O}_{2\text{max}}$ in this study was $3.6 \text{ L}\cdot\text{min}^{-1}$, compared to 3.6, 3.7, and $2.8 \text{ L}\cdot\text{min}^{-1}$ found in similar studies by MacPherson, MacDougall, and Burgomaster, respectively. The sex of participants affects the absolute $\dot{V}\text{O}_{2\text{max}}$ measured; due to a smaller blood volume and greater proportion of fat mass, typically females have lower $\dot{V}\text{O}_{2\text{max}}$ values than males. While the baseline values between studies appear similar, the values may actually represent different training statuses based on the composition of each population. The present study included 14 men and 14 women trainees. Burgomaster studied men and women but baseline $\dot{V}\text{O}_{2\text{max}}$ was only $2.8 \text{ L}\cdot\text{min}^{-1}$, MacPherson included more men than women, and MacDougall studied men exclusively. Our baseline values appear similar to these related studies despite the greater proportion of female participants, which may be expected to reduce the mean $\dot{V}\text{O}_{2\text{max}}$. The fact that baseline $\dot{V}\text{O}_{2\text{max}}$ was not lower than similar studies involving more males might indicate that our participants were actually more trained (for example, men in the SIT group had a baseline $\dot{V}\text{O}_{2\text{max}}$ of $4.2 \text{ L}\cdot\text{min}^{-1}$) before the SIT intervention. Changes in $\dot{V}\text{O}_{2\text{max}}$ in response to training are smaller in trained individuals.

In young, trained, healthy humans, breathing normoxic air, cardiac output is often cited as the factor which imposes the greatest limit to $\dot{V}O_{2\max}$ (Bergh, Ekblom, & Åstrand, 2000). Increasing cardiac output (via blood volume expansion, pericardectomy) leads to an instantaneous increase in $\dot{V}O_{2\max}$ (Kanstrup & Ekblom, 1982; Stray-Gundersen et al., 1986). If subjects in the present study had a relatively high training status, it is possible that the training stimulus to peripheral systems is diminished, and that training improvements are driven mainly by changes in Q_{\max} . This would explain the small observed increase in $\dot{V}O_{2\max}$, and agrees with the higher than expected baseline $\dot{V}O_{2\max}$.

4.2 Effect on Q_{\max}

Following SIT, participants showed a trend toward increased Q_{\max} (4%) which did not reach statistical significance ($p=0.166$). In agreement with previous research, HR_{\max} did not change following training (Blomqvist & Saltin, 1983). As Q_{\max} is the product of HR_{\max} and SV_{\max} , the unchanged HR_{\max} indicates that the trend for Q_{\max} to increase was likely related to increased SV_{\max} . In support of this, there was a 5% non-significant increase in SV_{\max} following SIT ($p=0.095$). While neither change reached significance, there was considerable variability about each mean (SIT Q_{\max} : pre= 26.13 ± 5.09 vs post= 27.10 ± 4.82 L \cdot min $^{-1}$; SIT SV_{\max} : pre= 138 ± 27 vs post= 144 ± 28 mL \cdot beat $^{-1}$). It is possible that a real change occurred but it is undetectable due to the variability.

Additionally, the other components that might contribute to an increase in $\dot{V}O_{2\max}$ (HR_{\max} and a- $\dot{V}O_{2\text{diff}}$) were not even approaching significant p values (SIT HR_{\max} $p=0.243$; SIT a- $\dot{V}O_{2\text{diff}}$ $p=0.803$). Thus, our conclusion is that Q_{\max} and SV_{\max} likely increased in response to SIT and were the driving forces in producing a greater $\dot{V}O_{2\max}$. More subjects and/or a more homogeneous response would be needed to determine this definitively.

An exercise training program must impose a certain level of volume load (increased cardiac output during training) in order to elicit improvements in Q_{\max} (Abergel et al., 2004; Ehsani et al., 1991). MacPherson et al. (2011) showed that, during a 30-second SIT bout, heart rate increases from 140 to 185 beats \cdot min $^{-1}$. Hazell observed that during an SIT session, using 30-second bouts with 4 minutes of rest, average HR was 149 beats \cdot min $^{-1}$

for the entirety of the training session including recovery periods (Hazell, Olver, Hamilton, & Lemon, 2012). So apparently SIT drives heart rate to near maximal values during the exercise bout and remains elevated during the “rest/recovery” periods between bouts. By extending the exercise bout by 15 seconds, there would be a great increase in volume load on the heart because HR would be near maximal during the entirety of the added 15 seconds. Additionally, the exercise was performed at supramaximal intensities so metabolic products as well as increased neural outflow could result in a further elevated HR during “rest/recovery” in comparison to 30-second SIT. We did not alter the length of the “rest” period (4 minutes) which means the ratio of work:rest was greater compared to 30-second bouts. A greater work:rest ratio will be another factor contributing to an increased average HR (and cardiac volume load) during each training session.

Sprint interval training is known to be a potent stimulus for a number of other adaptations. For example, after 1 training session there is elevated PGC-1 α and evidence of mitochondrial biogenesis occurring (Little, Safdar, Bishop, Tarnopolsky, & Gibala, 2011). However as mentioned, SIT, using 30-second bouts does not appear to result in improvements in Q_{\max} . The current study suggests that using 45-second bouts could provide a minimal stimulus because there is a trend for an increased Q_{\max} . It is possible that a longer program (e.g. 8 weeks) would result in significant changes to Q_{\max} because the stimulus, if real, appears to be weak. Perhaps slightly longer exercise bouts (e.g. 60-80 seconds) could provide a sufficient stimulus.

4.3 Effect on $a-v\dot{V}O_{2\text{diff}}$

In contrast to the findings of MacPherson et al. (2011), who showed that 30-second SIT bouts caused an increase in $a-v\dot{V}O_{2\text{diff}}$, there was no change in $a-v\dot{V}O_{2\text{diff}}$ when 45-second exercise bouts were used. Our baseline $a-v\dot{V}O_{2\text{diff}}$ values were similar to those of MacPherson ($\sim 14 \text{ mL O}_2 \cdot 100\text{mL blood}^{-1}$). However, like $V\dot{V}O_{2\text{max}}$, $a-v\dot{V}O_{2\text{diff}}$ tends to be smaller in women. Our study involved a greater proportion of women than MacPherson. It is possible that, as with $V\dot{V}O_{2\text{max}}$, our $a-v\dot{V}O_{2\text{diff}}$ values were great initially due to a greater training status.

Improvements in $a-v \dot{V}O_{2\text{diff}}$ reflect increased extraction/utilization at the muscle. Sprint interval training using 30-second bouts increase oxidative and glycolytic enzyme concentrations and as a result increases $a-v \dot{V}O_{2\text{diff}}$ (Burgomaster, Heigenhauser, & Gibala, 2006; MacPherson et al., 2011). If our subjects were indeed more trained as suggested they would have already experienced increases in enzyme concentrations/activity prior to the study and therefore would be expected to experience a reduced training adaptation. Importantly, it is also possible that 45-second bouts provide a stimulus that differs from 30-second bouts. Sprint interval training is unique in that the effort is constant (“all-out” or supramaximal) but power output (or running speed in our case) varies throughout the exercise bout. The first 10 seconds are characterized by a large power output and then power output is considerably lower for the remaining 20 to 35 seconds. Hazell et al. (2010) showed that 10-second SIT bouts produced improvements in aerobic and anaerobic exercise performance that were similar to improvements seen with 30-second bouts. Perhaps SIT is a potent and effective training method largely due to the great power outputs produced during the initial 10 seconds of each bout while the remaining 20 seconds have a negligible effect on training adaptations. Helgerud et al. (2007) had subjects perform 1 of 4 training programs (Submaximal endurance training, endurance training at maximal lactate steady state, 1:1 short interval training bouts at 90-95% $\dot{V}O_{2\text{max}}$, and 1:1 long interval training bouts at 90-95% $\dot{V}O_{2\text{max}}$). Training frequency and total work were matched, the greatest improvements in $\dot{V}O_{2\text{max}}$ were associated with higher exercise intensities (interval training was greater than endurance training). SIT is performed at “all-out” intensities and that could be the key to its effectiveness. Because there is an inverse relationship between exercise volume and intensity, by increasing the duration of SIT bouts, we increased the volume and decreased intensity. It is possible that this minimized improvements in $\dot{V}O_{2\text{max}}$ via a decreased effect on peripheral adaptations. Interestingly, the greater exercise volume vs 30-second bouts may stimulate Q_{max} but, based on our data, insufficiently so to observe a significant increase. Apparently, longer bouts are necessary. Consequently, our choice of 45-second sprint bouts and their effect on both exercise intensity and duration may have contributed to the smaller than expected increase in $\dot{V}O_{2\text{max}}$ observed.

4.4 Effect on 5-minute run

The 5-minute run was used to assess the effect of training on exercise performance. Total distance run during the 5 minutes did not change with training and there was a decrease following control conditions.

However, following completion of the study, a number of participants reported that they had modified their pacing strategy and/or effort for the 5-minute run because of the knowledge that it was extremely challenging gained during the initial testing. The tread poses greater than normal resistance so running in manual mode is more difficult than running freely on a road or track. Also, participants must use their upper body to push on the treadmill's handles in order to properly propel the tread. Running in manual mode is more similar to pushing a sled than running freely. This combination of resistance on the tread and upper body effort made the test quite difficult as indicated. Consistent with this possibility is the observed decreased performance in the control group on the post test. This might explain why the 5-minute run total distance did not improve significantly in the SIT group. As mentioned, there was a main effect of time (pre>post) on total distance run which supports participants' reports of changing their pacing strategy due to the extreme demands/effort required for the test. During their first run they did not know how hard the test would be and gave a maximal effort. After having completed the test once, participants may have attempted to minimize the associated discomfort by altering their strategy (i.e. pacing themselves), resulting in a poorer performance.

To clarify any coexisting effects of training improvements and voluntary pacing, the post study distance run was compared to the pre-training performance (post-training distance – pre-training distance; Δd in metres run) to obtain a better representation of performance changes following SIT (Figure 6). Consistent with pacing affecting the outcome, the majority of control participants actually decreased their performance (Δd was decreased) while SIT participants exhibited a greater range of responses (with most participants showing improvement or little to no change). If altered pacing strategy (which participants in both groups reported) is responsible for the control group's reduction in Δd , then the SIT group was likely exposed to this effect as well. With this in mind, the preservation of running performance (and even a trend for improvement), despite the

negative effect of pacing, may represent a significant improvement in training status without a significant improvement in absolute distance run.

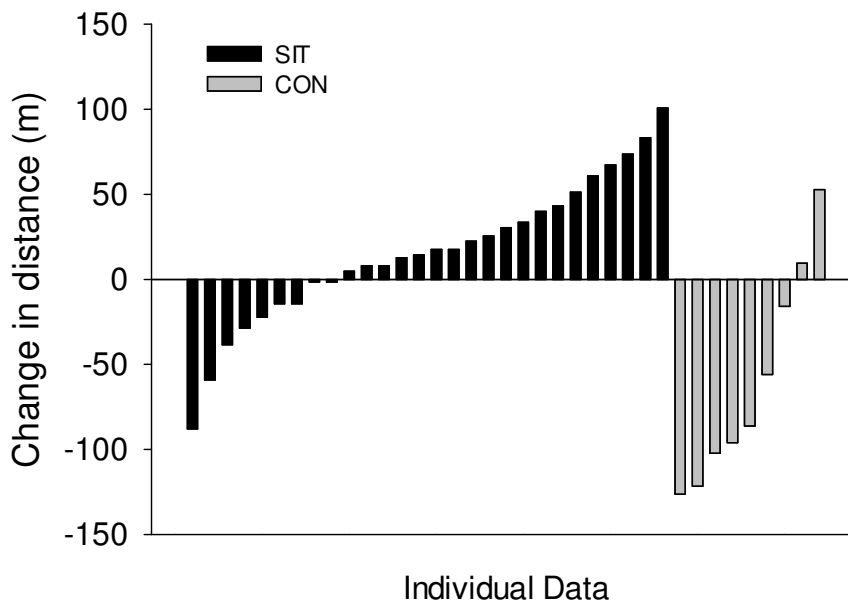


Figure 6. Individual changes in distance run (Δd) during 5-minute run. SIT = sprint interval training; CON = control. Each bar represents one participant.

In order to assess exercise performance, a 5-minute run on this type of manually driven treadmill is likely a poor test due to the effort it requires and the discomfort it produces, which may obscure real physiological changes. Tests which have successfully been used to assess exercise performance, such as the Yo-Yo intermittent recovery test, cycling time trials, and running time trials, should be used in future studies to measure exercise performance (Burgomaster et al., 2006; Iaia et al., 2008; MacPherson et al., 2011).

4.5 Summary

Thirty-eight recreationally active men and women performed 6 weeks of SIT, progressing from 4x30s to 7x45s bouts, (n=28) or acted as controls (n=10). Pre- and post-training (or post-control conditions), testing assessed $\dot{V}O_{2max}$, $Q_{O_{2max}}$, HR_{max} , SV_{max} , $a-vO_{2diff}$, and exercise performance (5-minute run). The primary finding from this study was that longer SIT bouts (up to 45-seconds) do not significantly increase $Q_{O_{2max}}$. Maximal oxygen

uptake increased significantly as a result of insignificant increases in \dot{Q}_{\max} and SV_{\max} with no effect on $a-v\dot{O}_{2\text{diff}}$. The five minute run Δd following SIT suggests a positive effect from training, despite an overall non-significant difference in absolute distance. Limitations of the treadmill running task may have obscured any underlying training effect, and a future studies should employ a different task.

4.6 Future directions

Based on the findings from this study, longer duration sprint interval training (up to 45-seconds) should not be a replacement for ET. Rather, athletes should use a combination of SIT and ET in order to obtain the gamut of adaptations. Future studies should use untrained individuals in hopes of uncovering real changes in $V\dot{O}_{2\max}$, \dot{Q}_{\max} , and $a-v\dot{O}_{2\text{diff}}$ that may have been obscured by the training status of participants in the current study. Studying the effects of an SIT program which uses a combination of various exercise bout lengths (e.g. 10-second, 30-second, and 60-second bouts) would be of interest. This would help determine if sprint interval exercise could effectively elicit central and peripheral adaptations concurrently. Additionally, due to the unique power profile of SIT exercise bouts, it would be of interest to compare time- and work-matched exercise training using constant-load exercise bouts to SIT exercise bouts.

4.7 Conclusion

Lengthening the duration of SIT bouts, from 30 to 45 seconds over 6 weeks of training (3 times/week) promotes non-significant central adaptations (increase in \dot{Q}_{\max} and SV_{\max}) which result in a modest increase in $V\dot{O}_{2\max}$. Peripheral adaptations were minimal. In addition, it is likely that exercise performance is improved with 45-second exercise bouts; however, 5-minute treadmill running test for distance, at least on the manual treadmill used here, is not a reliable measurement of exercise performance.

References

- Abergel, E., Chatellier, G., Hagege, A. A., Oblak, A., Linhart, A., Ducardonnet, A., & Menard, J. (2004). Serial left ventricular adaptations in world-class professional cyclists: implications for disease screening and follow-up. *Journal of the American College of Cardiology*, *44*(1), 144–149.
- Alberts, B., Bray, D., Hopkin, K., Johnson, A., Lewis, J., Raff, M., ... Walter, P. (2004). How cells obtain energy from food. In *Essential Cell Biology* (Second., pp. 427–452). New York: Garland Science.
- Allen, D. G., Lamb, G. D., & Westerblad, H. (2008). Skeletal Muscle Fatigue: Cellular Mechanisms. *Physiological Reviews*, *88*, 287–332.
- Åstrand, P. O. (1956). Human physical fitness with special reference to sex and age. *Physiological Reviews*, *36*(3), 307–335.
- Baar, K. (2006). To perform your best: work hard not long. *The Journal of Physiology*, *575*(3), 690.
- Baldwin, K. M., Cooke, D. A., & Cheadle, W. G. (1977). Time course adaptations in cardiac and skeletal muscle to different running programs. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, *42*(2), 267–272.
- Bangsbo, J., Mohr, M., Poulsen, A., Perez-Gomez, J., & Krstrup, P. (2006). Training and Testing the Elite Athlete. *Journal of Exercise Science and Fitness*, *4*(1), 1–14.
- Bassett, D. R., & Howley, E. T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Medicine and Science in Sports and Exercise*, *32*(1), 70–84.
- Beaver, W. L., Lamarra, N., & Wasserman, K. (1981). Breath-by-breath measurement of true alveolar gas exchange. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, *51*(6), 1662–1675.

- Bergh, U., Ekblom, B., & Åstrand, P. O. (2000). Maximal oxygen uptake “classical” versus “contemporary” viewpoints. *Medicine and Science in Sports and Exercise*, 32(1), 85–88.
- Bevegård, S. (1962). Studies on the regulation of the circulation in man. *Acta Physiologica Scandinavica*, 57(S200), 1–36.
- Blomqvist, C. G., & Saltin, B. (1983). Cardiovascular adaptations to physical training. *Annual Review of Physiology*, 45(46), 169–189.
- Bogdanis, G. C., Nevill, M. E., Boobis, L. H., & Lakomy, H. K. (1996). Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *Journal of Applied Physiology*, 80(3), 876–884.
- Booth, F. W., & Thomason, D. B. (1991). Molecular and cellular adaptation of muscle in response to exercise: perspectives of various models. *Physiological Reviews*, 71(2), 541–85.
- Brooks, G. A., & Mercier, J. (1994). Balance of carbohydrate and lipid utilization during exercise: the “crossover” concept. *Journal of Applied Physiology*, 76(6), 2253–2261.
- Buchheit, M., & Laursen, P. B. (2013). High-intensity interval training, solutions to the programming puzzle. Part I: cardiopulmonary emphasis. *Sports Medicine*, 43(5), 313–338.
- Burgomaster, K. A., Heigenhauser, G. J. F., & 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, 2041–2047.
- Burgomaster, K. A., Howarth, K. R., Phillips, S. M., Rakobowchuk, M., MacDonald, M. J., McGee, S. L., & Gibala, M. J. (2008). Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *The Journal of Physiology*, 586(1), 151–160.

- Burgomaster, K. A., Hughes, S. C., Heigenhauser, G. J. F., 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*, 1985–1990.
- Cairns, S. P., & Lindinger, M. I. (2008). Do multiple ionic interactions contribute to skeletal muscle fatigue? *The Journal of Physiology*, *586*(17), 4039–4054.
- Clausen, J. P., Klausen, K., Rasmussen, B., & Trap-Jensen, J. (1973). Central training and peripheral circulatory changes after training of the arms or legs. *American Journal of Physiology*, *225*(3), 675–682.
- Coffey, V. G., & Hawley, J. A. (2007). The molecular bases of training adaptation. *Sports Medicine*, *37*(9), 737–763.
- Convertino, V. A. (1991). Blood volume: its adaptation to endurance training. *Medicine and Science in Sports and Exercise*, *23*(12), 1338–1348.
- Coyle, E. F., Martin, W. H., Sinacore, D. R., Joyner, M. J., Hagberg, J. M., & Holloszy, J. O. (1984). Time course of loss of adaptations after stopping prolonged intense endurance training. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, *57*(6), 1857–1864.
- Daussin, F. N., Ponsot, E., Dufour, S. P., Lonsdorfer-Wolf, E., Doutreleau, S., Geny, B., ... Richard, R. (2007). Improvement of VO₂max by cardiac output and oxygen extraction adaptation during intermittent versus continuous endurance training. *European Journal of Applied Physiology*, *101*, 377–383.
- Daussin, F. N., Zoll, J., Dufour, S. P., Ponsot, E., Lonsdorfer-Wolf, E., Doutreleau, S., ... Richard, R. (2008). Effect of interval versus continuous training on cardiorespiratory and mitochondrial functions: relationship to aerobic performance improvements in sedentary subjects. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, *295*, R264–R272.

- Dudley, G. A., Abraham, W. M., & Terjung, R. L. (1982). Influence of exercise intensity and duration on biochemical adaptations in skeletal muscle. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, 53(4), 844–850.
- Egginton, S. (2009). Invited review: activity-induced angiogenesis. *European Journal of Physiology*, 457, 963–977.
- Ehsani, A. A., Ogawa, T., Miller, T. R., Spina, R. J., & Jilka, S. M. (1991). Exercise training improves left ventricular systolic function in older men. *Circulation*, 83, 96–103.
- Ekblom, B., Åstrand, P. O., Saltin, B., Stenberg, J., & Wallström, B. (1968). Effect of training on circulatory response to exercise. *Journal of Applied Physiology*, 24, 518–528.
- Ekblom, B., & Hermansen, L. (1968). Cardiac output in athletes. *Journal of Applied Physiology*, 25(5), 619–625.
- Ferguson, S., Gledhill, N., Jamnik, V. K., Wiebe, C., & Payne, N. (2001). Cardiac performance in endurance-trained and moderately active young women. *Medicine and Science in Sports and Exercise*, 33(7), 1114–1119.
- Flück, M., & Hoppeler, H. (2003). Molecular basis of skeletal muscle plasticity--from gene to form and function. *Reviews of Physiology, Biochemistry and Pharmacology*, 146, 159–216.
- 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. *The Journal of Physiology*, 575(3), 901–911.
- Glower, D. D., Spratt, J. A., Snow, N. D., Kabas, J. S., Davis, J. W., Olsen, C. O., ... Rankin, J. S. (1985). Linearity of the Frank-Starling relationship in the intact heart: the concept of preload recruitable stroke work. *Circulation*, 71(5), 994–1009.

- Gollnick, P. D., Armstrong, R. B., Saltin, B., Saubert IV, C. W., Sembrowich, W. L., & Shepherd, R. E. (1973). Effect of training composition on enzyme activity and fiber of human skeletal muscle. *Journal of Applied Physiology*, *34*(1), 107–111.
- Gollnick, P. D., & Saltin, B. (1982). Significance of skeletal muscle oxidative enzyme enhancement with endurance training. *Clinical Physiology*, *2*, 1–12.
- González-Alonso, J. (2008). Point: Stroke volume does/does not decline during exercise at maximal effort in healthy individuals. *Journal of Applied Physiology*, *104*(1), 275–280.
- Gunnarsson, T. P., & Bangsbo, J. (2012). The 10-20-30 training concept improves performance and health profile in moderately trained runners. *Journal of Applied Physiology*, *113*(1), 16–24.
- Hawley, J. A. (2002). Adaptations of skeletal muscle to prolonged, intense endurance training. *Clinical and Experimental Pharmacology and Physiology*, *29*, 218–222.
- Haykowsky, M. J., Dressendorfer, R., Taylor, D., Mandic, S., & Humen, D. (2002). Resistance Training and Cardiac Hypertrophy: Unravelling the Training Effect. *Sports Medicine*, *32*(13), 837–849.
- Hazell, T. J., MacPherson, R. E. K., Gravelle, B. M. R., & Lemon, P. W. R. (2010). 10 or 30-S Sprint Interval Training Bouts Enhance Both Aerobic and Anaerobic Performance. *European Journal of Applied Physiology*, *110*, 153–160.
- Hazell, T. J., Olver, T. D., Hamilton, C. D., & Lemon, P. W. R. (2012). Two Minutes of Sprint-Interval Exercise Elicits 24-hr Oxygen Consumption Similar to That of 30 min of Continuous Endurance Exercise. *International Journal of Sport Nutrition and Exercise Metabolism*, *22*(4), 276–283.
- Helgerud, J., Høydal, K., Wang, E., Karlsen, T., Berg, P., Bjerkaas, M., ... Hoff, J. (2007). Aerobic high-intensity intervals improve VO₂max more than moderate training. *Medicine and Science in Sports and Exercise*, *39*(4), 665–671.

- Holloszy, J. O., & Booth, F. W. (1976). Biochemical Adaptations to Endurance Exercise in Muscle. *Annual Review of Physiology*, 38, 273–291.
- Holloszy, J. O., & Coyle, E. F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, 56(4), 831–838.
- Hopper, M. K., Coggan, A. R., & Coyle, E. F. (1988). Exercise stroke volume relative to plasma-volume expansion. *Journal of Applied Physiology*, 64, 404–408.
- Iaia, F. M., & Bangsbo, J. (2010). Speed endurance training is a powerful stimulus for physiological adaptations and performance improvements of athletes. *Scandinavian Journal of Medicine and Science in Sports*, 20(S2), 11–23.
- Iaia, F. M., Perez-Gomez, J., Thomassen, M., Nordsborg, N. B., Hellsten, Y., & Bangsbo, J. (2011). Relationship between performance at different exercise intensities and skeletal muscle characteristics. *Journal of Applied Physiology*, 110, 1555–1563.
- Iaia, F. M., Thomassen, M., Kolding, H., Gunnarsson, T., Wendell, J., Rostgaard, T., ... Bangsbo, J. (2008). Reduced volume but increased training intensity elevates muscle Na⁺-K⁺ pump alpha-1-subunit and NHE1 expression as well as short-term work capacity in humans. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 294, R966–R974.
- Ingjer, F. (1979). Effects of endurance training on muscle fibre ATP-ase activity, capillary supply and mitochondrial content in man. *The Journal of Physiology*, 294, 419–432.
- Irrcher, I., Adhietty, P. J., Joseph, A., Ljubicic, V., & Hood, D. A. (2003). Regulation of mitochondrial biogenesis in muscle by endurance exercise. *Sports Medicine*, 33(11), 783–793.
- Jaski, B. E., Fifer, M. A., Wright, R. F., Braunwald, E., & Colucci, W. S. (1985). Positive Inotropic and Vasodilator Actions of Milrinone in Patients with Severe Congestive Heart Failure. *Journal of Clinical Investigation*, 75, 643–649.

- Jensen, L., Bangsbo, J., & Hellsten, Y. (2004). Effect of high intensity training on capillarization and presence of angiogenic factors in human skeletal muscle. *The Journal of Physiology*, 557(2), 571–582.
- Jeukendrup, A. E. (2002). Regulation of fat metabolism in skeletal muscle. *Annals New York Academy of Sciences*, 967, 217–235.
- Johnson, B. D., Beck, K. C., Proctor, D. N., Miller, J., Dietz, N. M., & Joyner, M. J. (2000). Cardiac output during exercise by the open circuit acetylene washin method: comparison with direct Fick. *Journal of Applied Physiology*, 88, 1650–1658.
- Jones, A. M., & Carter, H. (2000). The effect of endurance training on parameters of aerobic fitness. *Sports Medicine*, 29(6), 373–386.
- Juel, C. (2006). Training-induced changes in membrane transport proteins of human skeletal muscle. *European Journal of Applied Physiology*, 96(6), 627–635.
- Juel, C., & Halestrap, A. P. (1999). Lactate transport in skeletal muscle - role and regulation of the monocarboxylate transporter. *Journal of Physiology*, 517(3), 633–642.
- Juel, C., Klarskov, C., Nielsen, J. J., Krstrup, P., Mohr, M., & Bangsbo, J. (2004). Effect of high-intensity intermittent training on lactate and H⁺ release from human skeletal muscle. *American Journal of Physiology: Endocrinology and Metabolism*, 286, E245–E251.
- Jürgens, K. D., Papadopoulos, S., Peters, T., & Gros, G. (2000). Myoglobin: Just an Oxygen Store or Also an Oxygen Transporter? *Physiology*, 15, 269–274.
- Kanstrup, I. L., & Ekblom, B. (1982). Acute hypervolemia, cardiac performance, and aerobic power during exercise. *Journal of Applied Physiology*, 52, 1186–1191.

- Kanstrup, I. L., & Ekblom, B. (1984). Blood volume and hemoglobin concentration as determinants of maximal aerobic power. *Medicine and Science in Sports and Exercise*, *16*(3), 256–262.
- Keteyian, S. J. (2012). Swing and a miss or inside-the-park home run: which fate awaits high-intensity exercise training? *Circulation*, *126*, 1431–1433.
- Kiil, F. (1978). Principles of cardiac output regulation. *Scandinavian Journal of Clinical and Laboratory Investigation*, *38*, 401–406.
- Kingwell, B. A., Arnold, P. J., Jennings, G. L., & Dart, A. M. (1998). The effects of voluntary running on cardiac mass and aortic compliance in Wistar-Kyoto and spontaneously hypertensive rats. *Journal of Hypertension*, *16*(2), 181–185.
- Kjellberg, S. R., Rudhe, U., & Sjöstrand, T. (1949). Increase of the Amount of Hemoglobin and Blood Volume in Connection with Physical Training. *Acta Physiologica Scandinavica*, *19*, 146–151.
- Konhilas, J. P., Irving, T. C., & de Tombe, P. P. (2002). Frank-Starling law of the heart and the cellular mechanisms of length-dependent activation. *European Journal of Physiology*, *445*(3), 305–310.
- Krustrup, P., Hellsten, Y., & Bangsbo, J. (2004). Intense interval training enhances human skeletal muscle oxygen uptake in the initial phase of dynamic exercise at high but not at low intensities. *The Journal of Physiology*, *559*(1), 335–345.
- Kubukeli, Z. N., Noakes, T. D., & Dennis, S. C. (2002). Training techniques to improve endurance exercise performances. *Sports Medicine*, *32*(8), 489–509.
- Laughlin, M. H., & Roseguini, B. (2008). Mechanisms for exercise training-induced increases in skeletal muscle blood flow capacity: differences with interval sprint training versus aerobic endurance training. *Journal of Physiological Pharmacology*, *59*(S7), 71–88.

- Laursen, P. B. (2010). Training for intense exercise performance: high-intensity or high-volume training? *Scandinavian Journal of Medicine and Science in Sports*, 20(S2), 1–10.
- Laursen, P. B., & Jenkins, D. G. (2002). The scientific basis for high-intensity interval training: optimising training programmes and maximising performance in highly trained endurance athletes. *Sports Medicine*, 32(1), 53–73.
- Little, J. P., Safdar, A., Bishop, D., Tarnopolsky, M. A., & Gibala, M. J. (2011). An acute bout of high-intensity interval training increases the nuclear abundance of PGC-1 α and activates mitochondrial biogenesis in human skeletal muscle. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 300, R1303–R1310.
- MacDougall, J. D., Hicks, A. L., MacDonald, J. R., McKelvie, R. S., Green, H. J., & Smith, K. M. (1998). Muscle performance and enzymatic adaptations to sprint interval training. *Journal of Applied Physiology*, 84, 2138–2142.
- MacPherson, R. E. K., Hazell, T. J., Olver, T. D., Paterson, D. H., & Lemon, P. W. R. (2011). Run sprint interval training improves aerobic performance but not maximal cardiac output. *Medicine and Science in Sports and Exercise*, 43(1), 115–122.
- Maron, B. J., & Pelliccia, A. (2006). The heart of trained athletes: cardiac remodeling and the risks of sports, including sudden death. *Circulation*, 114, 1633–1644.
- McKenna, M. J. (1995). Effects of training on potassium homeostasis during exercise. *Journal of Molecular and Cellular Cardiology*, 27, 941–949.
- Mujika, I., & Padilla, S. (2001). Muscular characteristics of detraining in humans. *Medicine and Science in Sports and Exercise*, 33(8), 1297–1303.
- Parra, J., Cadefau, J. A., Rodas, G., Amigó, N., & Cussó, R. (2000). The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle. *Acta Physiologica Scandinavica*, 169(2), 157–165.

- Pelliccia, A., Maron, B. J., De Luca, R., Di Paolo, F. M., Spataro, A., & Culasso, F. (2002). Remodeling of Left Ventricular Hypertrophy in Elite Athletes After Long-Term Deconditioning. *Circulation*, *105*(8), 944–949.
- Penpargkul, S., Repke, D. I., Katz, A. M., & Scheuer, J. (1977). Effect of physical training on calcium transport by rat cardiac sarcoplasmic reticulum. *Circulation Research*, *40*(2), 134–138.
- Pluim, B. M., Zwinderman, A. H., van der Laarse, A., & van der Wall, E. E. (2000). The Athlete's Heart: A Meta-Analysis of Cardiac Structure and Function. *Circulation*, *101*, 336–344.
- Poliner, L. R., Dehmer, G. J., Lewis, S. E., Parkey, R. W., Blomqvist, C. G., & Willerson, J. T. (1980). Left ventricular performance in normal subjects: a comparison of the responses to exercise in the upright and supine positions. *Circulation*, *62*, 528–534.
- Poole, D. C., Wilkerson, D. P., & Jones, A. M. (2008). Validity of criteria for establishing maximal O₂ uptake during ramp exercise tests. *European Journal of Applied Physiology*, *102*(4), 403–410.
- Rakobowchuk, M., Tanguay, S., Burgomaster, K. A., Howarth, K. R., Gibala, M. J., & MacDonald, M. J. (2008). Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow-mediated dilation in healthy humans. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, *295*, R236–R242.
- Robinson, B. F., Epstein, S. E., Kahler, R. L., & Braunwald, E. (1966). Circulatory Effects of Acute Expansion of Blood Volume: Studies During Maximal Exercise and at Rest. *Circulation Research*, *19*, 26–32.
- Ross, J., Covell, J. W., Sonnenblick, E. H., & Braunwald, E. (1966). Contractile State of the Heart Characterized by Force-Velocity Relations in Variably Afterloaded and Isovolumic Beats. *Circulation Research*, *18*(2), 149–163.

- Sagawa, K. (1978). The ventricular pressure-volume diagram revisited. *Circulation Research*, 43(5), 677–687.
- Saltin, B. (2009). Physiological Adaptation to Physical Conditioning. *Acta Medica Scandinavica*, 220(S711), 11–24.
- Sawka, M. N., Convertino, V. A., Eichner, E. R., Schnieder, S. M., & Young, A. J. (2000). Blood volume: importance and adaptations to exercise training, environmental stresses, and trauma/sickness. *Medicine and Science in Sports and Exercise*, 32(2), 332–348.
- Silverthorn, D. U. (2004). Blood. In *Human Physiology: An Integrated Approach* (Third., pp. 523–545). San Francisco: Pearson Education Inc.
- Siri, W. E. (1961). Body composition from fluid spaces and density: analysis of methods. *Techniques for Measuring Body Composition*, 223–244.
- Sonnenblick, E. H., & Downing, S. E. (1963). Afterload as a primary determinant of ventricular performance. *American Journal of Physiology*, 204(4), 604–610.
- Spriet, L. L. (2002). Regulation of skeletal muscle fat oxidation during exercise in humans. *Medicine and Science in Sports and Exercise*, 34(9), 1477–1484.
- Stray-Gundersen, J., Musch, T. I., Haidet, G. C., Swain, D. P., Ordway, G. A., & Mitchell, J. H. (1986). The effect of pericardiectomy on maximal oxygen consumption and maximal cardiac output in untrained dogs. *Circulation Research*, 58(4), 523–530.
- Talanian, J. L., Galloway, S. D. R., Heigenhauser, G. J. F., Bonen, A., & Spriet, L. L. (2007). Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women. *Journal of Applied Physiology*, 102(4), 1439–1447.

- Thomas, S., Reading, J., & Shephard, R. J. (1992). Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Canadian Journal of Sport Sciences, 17*(4), 338–345.
- Thomson, J. M., Dempsey, J. A., Chosy, L. W., Shahidi, N. T., & Reddan, W. G. (1974). Oxygen transport and oxyhemoglobin dissociation during prolonged muscular work. *Journal of Applied Physiology, 37*, 658–664.
- Vinereanu, D., Florescu, N., Sculthorpe, N., Tweddel, A. C., Stephens, M. R., & Fraser, A. G. (2001). Differentiation between pathologic and physiologic left ventricular hypertrophy by tissue Doppler assessment of long-axis function in patients with hypertrophic cardiomyopathy or systemic hypertension and in athletes. *The American Journal of Cardiology, 88*(1), 53–58.
- Warburton, D. E., Haykowsky, M. J. F., Quinney, H. A., Humen, D. P., & Teo, K. K. (1999). Reliability and validity of measures of cardiac output during incremental to maximal aerobic exercise. Part I: Conventional techniques. *Sports Medicine, 27*(1), 23–41.
- Wilmore, J. H., & Costill, D. L. (2004a). Respiratory regulation during exercise. In *Physiology of Sport and Exercise* (Third., pp. 242–269). Windsor: Human Kinetics.
- Wilmore, J. H., & Costill, D. L. (2004b). Metabolic adaptations to training. In *Physiology of Sport and Exercise* (Third., pp. 184–204). Windsor: Human Kinetics.
- Wisløff, U., Helgerud, J., Kemi, O. J., & Ellingsen, Ø. (2001). Intensity-controlled treadmill running in rats □: VO₂max and cardiac hypertrophy. *American Journal of Physiology: Heart and Circulatory Physiology, 280*, H1301–H1310.

Appendices

Appendix A: Ethics approval notice.



Use of Human Participants - Ethics Approval Notice

Principal Investigator: Dr. Peter Lemon
Review Number: 17996E
Review Level: Delegated
Approved Local Adult Participants: 30
Approved Local Minor Participants: 0
Protocol Title: The effect of sprint interval training (SIT) on selected cardiovascular measures and exercise performance in men and women.
Department & Institution: Kinesiology, University of Western Ontario
Sponsor:
Ethics Approval Date: June 06, 2011 **Expiry Date:** July 31, 2012
Documents Reviewed & Approved & Documents Received for Information:

Document Name	Comments	Version Date
UWO Protocol		
Letter of Information & Consent		
Advertisement		

This is to notify you that The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced revision(s) or amendment(s) on the approval date noted above. The membership of this REB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the UWO Updated Approval Request Form.

Members of the HSREB who are named as investigators in research studies, or declare a conflict of interest, do not participate in discussion related to, nor vote on, such studies when they are presented to the HSREB.

The Chair of the HSREB is Dr. Joseph Gilbert. The UWO HSREB is registered with the U.S. Department of Health & Human Services under the IRB registration number IRB 0000940.



Ethics Officer to Contact for Further Information



This is an official document. Please retain the original in your files.



Appendix B: Letter of information.



LETTER OF INFORMATION REGARDING RESEARCH

Title of Study: The effect of sprint interval training on selected cardiovascular measures and exercise performance in men and women

You are being invited to participate in a research study conducted by P.W.R. Lemon (PhD), Stephanie Reid (BSc), Alan Smith (BSc), Terry Olver (MSc), Alex Stevens (BHK), and Craig Hamilton (BSc), from the Exercise Nutrition Research Laboratory in the School of Kinesiology at the University of Western Ontario.

If you have any questions or concerns about the research, please feel free to contact [REDACTED]

PURPOSE OF THE STUDY

The purpose of this research experiment is to determine if six weeks of sprint interval training (SIT) will improve selected cardiovascular measures and exercise performance in men and women.

INCLUSION/EXCLUSION CRITERIA

In order to be eligible to participate in this study you must be a healthy, 18 to 35 year old male or female.

You will be excluded from this study if you are: injured, diabetic, currently undergoing a similar training program or have any contraindications to this type of exercise.

PROCEDURES

If you volunteer to participate in this study, we will ask you to do the following things:

1. Complete a health survey (PAR-Q) to assess your current physical capability.
2. Record a 3-day dietary record for the weeks that pre- and post-training testing are completed.

3. Undergo an assessment of various cardiovascular and exercise measures, before and after training, that will include: determination of body composition (BodPod® - involves sitting comfortably in a chamber for about 5 minutes while the space your body takes up is measured), a multi-stage exercise test on a treadmill (maximal oxygen uptake test), blood pressure, femoral blood flow, cardiac output, pulse wave velocity, blood velocity, arterial compliance, femoral artery diameter, heart rate, a 2km run for time on a track.
4. Complete 6 weeks of sprint interval training (3 days/week). Each session will consist of 4 (wk 1 and 2), 5 (wk 3 and 4), or 6 (wk 5 and 6) 1 minute “all out” running efforts on an outdoor track, with each effort separated by 4 minutes of no exercise.

Testing will be conducted in the Exercise Nutrition Research Laboratory, the Neurovascular Research Laboratory, and the Canadian Centre for Activity and Aging. Training will be conducted on the outdoor track at the TD Waterhouse Stadium.

TIME INVOLVED

The study will take place over eight weeks. There will be six weeks of training and 2 weeks of testing (pre- and post-training).

Pre- and post-training testing will take place during the first and eighth week of the study and will take place over 4 days. Day one will involve being familiarized with the procedures that will be done and completion of a dietary record. On day two, body composition and maximal oxygen uptake tests will be completed. On day three, resting cardiovascular measures will be taken (blood pressure, femoral blood flow, cardiac output, pulse wave velocity, blood velocity, arterial compliance, and femoral artery diameter). On day four, a 2 km timed run will be completed.

Training will take place from week 2 to week 7. It will occur 3 days per week. The number of intervals per session will increase over the course of the training program. Subjects will complete 4 intervals per session during weeks 2 and 3, 5 intervals during weeks 4 and 5, and 6 intervals during weeks 6 and 7. At the end of weeks 3 and 5 subjects will be reassessed for changes in resting cardiovascular measures (blood pressure, femoral blood flow, and cardiac output).

POTENTIAL RISK AND DISCOMFORTS

All exercise involves some health risk (primarily cardiovascular or hydration-related) but lack of activity has been shown to more hazardous to one's health. Further, these concerns are much reduced in young, healthy individuals. This type of exercise is completed by many Western students in kinesiology classes,

in intramural sports, and by Mustang athletes. Participants will be encouraged to adequately hydrate. There are no risks associated with the finger cuff method for measuring blood pressure. When the cuff is inflated subjects may experience numbness but this is reversed immediately upon deflation of the cuff. Adhesive electrodes will be applied to the skin for ECG. Some individuals may experience a slight skin reaction in response to the adhesive. This typically clears up in a few days. There are no known risks associated with ultrasound.

POTENTIAL BENEFITS

Your aerobic fitness will likely improve using this training protocol.

COMPENSATION

You will not be compensated for your participation in this study.

CONFIDENTIALITY

Any information that is obtained in connection with this study that can identify you will remain confidential and will be disclosed only with your permission. This information will be collected on a master list that will be kept in a password protected file with access to only the investigators in this study. All data will be collapsed before results are printed (only group averages and variability). All participants will be assigned an arbitrary number to ensure anonymity. Mean data will be stored in a password protected file for comparison with future studies. Raw data will not be released to any other parties.

PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you are a student and you volunteer, you may withdraw at any time without any effect on your status at UWO. If you are not a student at UWO, you may withdraw from the study at any time. You may also refuse to answer any questions you feel are inappropriate and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so.

FEEDBACK OF THE RESULTS OF THIS STUDY TO THE SUBJECTS

We plan to publish this study in a reputable academic journal upon the completion of the research. The information published in a journal or subsequent studies will not identify you in any way. Copies of such articles will be available upon request.

SUBSEQUENT USE OF DATA

These data may be used in subsequent studies but the data will have no personal identifiers.

You will receive a copy of the consent form after it has been signed. You do not waive any legal rights by signing the consent form.

This letter is for you to keep. If you have any questions about this research project, feel free to call us [REDACTED] for clarification. Further, if you have any questions about the conduct of this study or your rights as a research subject you may contact [REDACTED].

Sincerely,

[REDACTED]

Appendix C: Informed consent statement form.

INFORMED CONSENT STATEMENT

The effect of sprint interval training on selected cardiovascular measures and exercise performance in men and women

Investigator: P.W.R. Lemon (Stephanie Reid and Alan Smith)

I have read the accompanying "Letter of Information", have had the nature of the study explained to me and I agree to participate. All questions have been answered to my satisfaction.

By signing below, I agree to participate in this study.

Name of Participant (please print):

Signature of Participant:

Date: _____

Name of Person Obtaining Informed Consent:

Signature of Person Obtaining Informed Consent:

Date: _____

RE-RECRUITMENT IN FUTURE STUDIES

If you wish to participate in future studies in the Exercise Nutrition Research Lab, please include your current contact information below.

I wish to be contacted for future studies in the Exercise Nutrition Research Laboratory.

Yes _____ (check mark), No _____ (check mark)

If yes, email _____

Date: _____

Curriculum Vitae

Name: Alan Robert Smith

Post-secondary Education and Degrees: The University of Western Ontario
London, Ontario, Canada
2005-2009 B.Sc.

The University of Western Ontario
London, Ontario, Canada
2010-2013 M.Sc.

Honours and Awards: Western Graduate Research Scholarship
2011-2012

Kinesiology Graduate Student Travel Award
2011

Related Work Experience Teaching Assistant
The University of Western Ontario
2011-2012

Published Abstracts:

A.R. Smith, S.M. Reid, T.D. Olver, D.H. Paterson, and P.W.R. Lemon (2013). Forty-five second sprint interval training bouts do not increase maximal cardiac output. *Applied Physiology, Nutrition, Metabolism* (in Press).

S.M. Reid, **A.R. Smith**, T.D. Olver, M. Zamir, P.W.R. Lemon, and J.K. Shoemaker (2012). Sprint interval training decreases viscoelastic and inertial effects in forearm vasculature in young men. *Applied Physiology, Nutrition, Metabolism* 37(S1), S30.

S.M. Reid, **A.R. Smith**, T.D. Olver, J.K. Shoemaker, P.W.R. Lemon (2012). Six weeks of sprint interval training speeds flow-mediated dilation in women. *Medicine & Science in Sports & Exercise*. 44(5S), 588.

A.R. Smith, T.D. Olver, S.M. Reid, J.K. Shoemaker, and P.W.R. Lemon (2011). Sprint interval exercise decreases pulse wave velocity and increases superficial femoral artery vascular conductance. *Applied Physiology, Nutrition, and Metabolism* 36(S2), S351.

S.M. Reid, T.D. Olver, **A.R. Smith**, M. Zamir, J.K. Shoemaker and P.W.R. Lemon (2011). Increases in forearm vascular compliance persist 2 h after sprint interval exercise in young men. *Applied Physiology, Nutrition, and Metabolism* 36(S2), S348.