THE EFFECTS OF HIGH INTENSITY INTERVAL TRAINING ON PULMONARY FUNCTION

by

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ABSTRACT

High-intensity interval training (HIT) has been utilized as a time-efficient strategy to induce numerous physiological adaptations and improve performance usually associated with "traditional" endurance training (ET). It is not known however, if HIT might lead to improvements in pulmonary function. Therefore we hypothesized that HIT would increase respiratory muscle strength and expiratory flow rates. Fifteen healthy subjects were randomly assigned to an ET group (n = 7) and a HIT group (n = 8). All subjects performed an incremental test to exhaustion (VO_2 max) on a cycle ergometer prior to and after training. Standard pulmonary function tests, maximum inspiratory pressure (PImax), maximum expiratory pressure (PEmax), and maximal flow volume loops, were performed pre training and after each week of training. HIT subjects performed a four week training program on a cycle ergometer at 90% of their VO_2 max final workload while the ET subjects performed exercise at 60-70% of their VO_2 max final workload. All subjects trained three days/ week. The HIT group performed five one-minute bouts with three minute recovery periods and the ET group cycled for 45 minutes continuously at a constant workload. A five-mile time trial was performed prior to training, after two weeks of training, and after four weeks of training. Both groups showed similar ($p<0.05$) increases in VO_2 max (~8-10%) and improvements in time trials following training (HIT 6.5 ± 1.3%, ET 4.4 ± 1.8%) with no difference ($p>0.05$) between groups. Both groups increased ($p<0.05$) PImax post training (ET ~25%, HIT ~43%) with values significantly higher for HIT than ET. There was no change ($p>0.05$) in

expiratory flow rates with training in either group. These data suggest that whole body exercise training is effective in increasing inspiratory muscle strength with HIT leading to greater improvements than ET. Also, HIT offers a time-efficient alternative to ET in improving aerobic capacity and performance.

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INTRODUCTION

Exercise training elicits numerous physiological adaptations which may lead to increased exercise performance. Improved metabolic, cardiovascular, and pulmonary adaptations delay the onset of fatigue, potentially allowing for improved exercise performance. Traditionally, endurance training (ET) has been the preferred method to elicit these changes. Alternatively, high intensity interval training (HIT) has long been employed by elite athletes but little attention has been given to this type of training in the scientific community until recent years. Research investigating HIT has found many surprisingly similar physiologic adaptations to those observed with ET. However, differences in HIT protocols has made comparing results difficult. The vast majority of research on HIT has focused on metabolic and cardiovascular adaptations and their effects on performance. With regard to the pulmonary system, it is believed that it does not adapt to exercise training. However, HIT may lead to pulmonary adaptations not commonly seen with ET. To our knowledge, this theory has not been empirically addressed. Therefore, the purpose of our study was to determine the effects of HIT on respiratory muscle strength and expiratory flow rates.

CHAPTER I

LITERATURE REVIEW

Exercise training is widely recognized as an effective method for improving fitness, health, and human performance. However, training induced benefits may be limited by several factors. These factors include metabolic rate, ATP production, oxygen delivery and extraction, blood flow, and respiratory muscle fatigue. Depending upon the type of training, performance can also be limited by exercise capacity, power, and muscular fatigue. This review will specifically focus on the difference between endurance training (ET) and high intensity interval training (HIT) and the adaptations associated with each. Additionally, the possible role of HIT on pulmonary function will be discussed.

I. ENDURANCE TRAINING

Traditionally, endurance training (ET), defined as exercise that is long in duration and primarily relies on aerobic energy metabolism, has long been the preferred form of training for most people (Gibala et al, 2008). In 1995 the American College of Sports Medicine (ACSM) made recommendations for health benefits that accrue from endurance training (ACSM, 2009). The most recent guidelines recommend 30 minutes of moderate intensity aerobic exercise most days of the week (ACSM, 2009). These recommendations are for the average healthy adult under 65 years of age. Moderate intensity, according to the ACSM, is defined as a level where you “break a sweat and

raise your heart rate but can still carry on a conversation" (ACSM, 2009). The 30 minute duration can be accumulated in bouts of no less than 10 minutes throughout the day. With this type of training, numerous physiological adaptations can occur.

A. *Metabolic adaptations*

Endurance training is well known to yield many metabolic adaptations. Most of these adaptations occur by meeting or exceeding the ACSM guidelines for physical activity (Burgomaster et al, 2008; Gibala et al, 2006; Rakobowchuk et al, 2008). Most metabolic adaptations lead to an increased amount of ATP available to perform work. In a recent study investigating muscle metabolism, the most likely sources of ATP appear to be derived from a higher glycolytic rate via greater pyruvate and/or intramuscular triglyceride oxidation and a larger recruitment of both type I and II muscle fibers (Harmer et al, 2000). Certain markers of ATP production, including creatine phosphate, creatine kinase, resting glycogen content, lactate dehydrogenase, and lactate have been investigated to determine whether an increase in ATP production occurs with training (Gibala et al, 2007). Lactate has long been known as a marker for these changes. Typically, the higher the lactate threshold the less lactate produced at a given work rate or intensity and the longer it will be before fatigue sets in (Juel et al, 2004). The release of Ca^{2+} from the sarcoplasmic reticulum activates pyruvate dehydrogenase, an enzyme that brings pyruvate from anaerobic glycolysis in to the mitochondria for aerobic glycolysis, or oxidative phosphorylation, to occur. If a greater release of Ca^{2+} occurs, there will be an increased shuttling of pyruvate increasing the

amount of ATP produced by glycolysis (Parolin et al, 1999). Theoretically, if the process of oxidative phosphorylation is consuming pyruvate-derived aceytl units, then an increase in pyruvate dehydrogenase activity will occur (Burgomaster et al, 2006; Hellsten et al, 1998). It has also been shown that Ca^{2+} release is primarily responsible for the stimulation of peroxisome proliferator-activated receptor coactivator (PGC-1α), which functions as a regulator of mitochondrial biogenesis (Terada et al, 2005). Studies have shown an enhanced expression of PGC-1α with endurance training (Burgomaster et al, 2008; Ortenblad et al, 2000; Terada et al, 2005).

It has been previously established that type I fibers typically have two thirds more mitochondria than type II fibers and when aerobic energy systems are the primary source of ATP during exercise, more type I fibers are recruited (Mogensen et al, 2006). After seven to ten days of endurance training performed for two hours daily, significant increases in mitochondrial activity occur as determined by increases in citrate synthase and 3-hydroxyacyl-CoA dehydrogenase (Burgomaster et al, 2008; Mogensen et al, 2006; Coyle et al, 2005). These increases suggest the possibility of training induced increases in mitochondrial content (Burgomaster et al, 2006; Burgomaster et al, 2008; Burgomaster et al, 2005). Together the possible increases in ATP production, mitochondrial content, and type I muscle fiber recruitment mentioned above can lead to an increase in muscle oxidative power and capacity and in the time to the onset of fatigue (Burgomaster et al, 2008; Gibala et al, 2008; Mogensen et al, 2006).

B. *Oxygen delivery adaptations*

Over time, beginning with at least two weeks of training, cardiovascular adaptations result from ET that include an increase in mean peak power output, VO_2 max, and an increase in the time to the onset of fatigue (Burgomaster et al, 2008; Gibala et al, 2006; Mogensen et al, 2006). Endurance training can also yield a decrease in the time to completion of time trials, respiratory exchange ratio, and a lowered resting heart rate (HR) (Burgomaster et al, 2008; Gibala et al, 2006). All of these adaptations result from the ability of the heart to delivery more oxygen either from an increased oxygen extraction or delivery. An increase in maximal oxygen uptake is well established with ET and results from an increased oxygen delivery through a greater cardiac output thus increasing the ability to do work (Burgomaster et al, 2008).

C. Pulmonary adaptations

Historically, ET has not been shown to produce pulmonary adaptations other than controversial improvements in respiratory muscle strength and endurance. As exercise intensity or duration increases greater workloads on the respiratory muscles, an increased work of breathing, and higher ventilation rates occur. There is a higher demand for blood flow due to the increasing work of the respiratory muscles (Guenette et al, 2007; Guenette et al, 2009; Harms et al, 2000; McClaran et al, 1999). It has been suggested that the respiratory muscles can demand as much as 10-15% of the blood flow during heavy exercise (Harms et al, 1998). This results in an earlier onset of respiratory muscle fatigue due to a recruitment of additional respiratory muscles,

especially with maximal exercise (Enright et al, 2006; Gibala et al, 2006; Harms et al, 1997; Harms et al, 1998). During these conditions, it has been demonstrated that the respiratory muscles, in a sense, "steal" oxygen and blood flow from the working locomotor muscles to meet the demand placed upon them (Harms et al, 1997; Harms et al, 1998).

Skeletal muscle adaptations to exercise training have been well documented, but there is currently little information on how respiratory muscle responds to whole body exercise training. One study has shown no difference in inspiratory muscle strength with increased respiratory muscle endurance in endurance trained athletes when compared to controls (Eastwood et al, 2001). In this study, changes in breathing pattern and active expiration through recruitment of additional respiratory muscles during exercise was suggested as the mechanism for greater respiratory muscle endurance (Eastwood et al, 2001). The majority of the current research using ET to alter respiratory muscle strength has been conducted using mainly rat models. Specifically, Powers et al (1994, 1997, 2002) have demonstrated that a workload of ~70% VO_2 max used with continuous exercise training (60 minutes, 4-5 days/wk for 10 weeks) increases the oxidative and antioxidant capacity of the rat diaphragm (20-30%), which results in an improvement in diaphragm endurance. They suggested that these changes may improve fat utilization, defend against free radical damage, and increase fatigue resistance (Powers et al, 1992). Another ten week rat treadmill training program, with a protocol similar to the one above, showed a 10% increase in citrate synthase activity in

the rat diaphragm. This change in oxidative capacity of the diaphragm appears to be dictated by the intensity and duration of exercise training (Vrabas et al, 1999). High intensity endurance training (75-80% VO_2 max) improved the oxidative capacity of the rat rectus abdominus and external oblique muscles. While the oxidative capacity of the rat diaphragm increased, it is possible that the diaphragm may not adapt like limb skeletal muscles in humans (Grinton et al, 1992; Metzger and Fitts, 1986). The research on humans that has been conducted has shown improved ventilatory performance due to respiratory muscle adaptations. These improvements have been shown by increases in maximal sustainable ventilations and maximal voluntary ventilation (O'Kroy et al, 1993). Researchers have found that athletes can sustain 80% of their maximal voluntary ventilation for a significantly longer time period than sedentary subjects (Martin and Stager, 1981). Another study demonstrated that after six weeks of ET in hypoxic conditions, no differences were found on any respiratory measure (Thomas et al, 1998).

It is possible that the altered breathing patterns and hyperventilation from endurance exercise could reduce airway smooth muscle tone which would lead to increased expiratory flow rates. To date, there has been little direct evidence that ET leads to these changes. However, a recent study by Scichilone et al (2005) has provided support for this premise. Comparing sedentary individuals and non-elite long distance runners, they found reduced tone in the smooth muscle of the airways of the runners (Scichilone et al, 2005). Repeated lung inflation-induced stretch of airway smooth muscle associated with exercise is also thought to reduce airway resistance. Decreasing

smooth muscle tone and contractility could be a mechanism for this reduction in airway resistance (Scichilone et al, 2005). By decreasing smooth muscle tone, airway resistance, and increasing lung volumes it is possible these mechanisms would lead to increased flow rates.

II. HIGH INTENSITY INTERVAL TRAINING

High intensity interval training (HIT) is defined as recurring sessions of exercise bouts at an "all out" effort level (greater than 90% of HR max or VO_2max) separated by periods of recovery (Gibala et al, 2008). Its primary use for many years has been with elite athletes. HIT has gained much attention in the scientific community in recent years as an alternative form of training to ET. This review will focus on the ability of HIT to be a time efficient but potent form of training that yields many similar, or even greater, adaptations as ET.

While training adaptations with ET are well known and understood, adaptations associated with HIT are not. This is partially due to a variety of training protocols that have been utilized to study the effects of HIT. For example, many studies have used the Wingate test in which a subject pedals on an electronically braked cycle ergometer at an "all out" effort level against a fixed resistance for 30 seconds, typically repeated 4-10 times within one session. In addition, most studies had subjects exercise three days a week for two weeks, but some studies lasted as long as seven weeks (Burgomaster et al, 2005; Burgomaster et al, 2006; Burgomaster et al, 2008; Gibala et al, 2006; Souissi et al, 2007). Some researchers increased the number of bouts per session as the weeks

progressed (Burgomaster et al, 2005; Burgomaster et al, 2006; Burgomaster et al, 2007; Burgomaster et al, 2008; Gibala et al, 2006; Harmer et al, 2000; Juel et al, 2004; MacDougall et al, 1998; McKenna et al, 1997; Rakobowchuk et al, 2008) and some kept it at a consistent number (McKenna et al, 1997). Exercise duration and frequency may lead to variability in training adaptations. The length of the bout, number of times repeated in one session, time of day the bout is executed, recovery time between bouts, and recovery time betweens sessions could also contribute to changes in metabolic, cardiovascular, and pulmonary adaptations.

A. Metabolic adaptations

Immediate energy is required in order to perform high intensity intervals, which requires large amounts of ATP. Some evidence suggests an increased stimulus for ATP production from the increases observed in ATP production markers such as creatine phosphate, creatine kinase, glycogen, lactate dehydrogenase, and lactate (Burgomaster et al, 2005; Burgomaster et al, 2008; Gibala et al, 2007; Hargreaves et al, 1998; Juel et al, 2004; McKenna et al, 2007; McKenna et al, 2007; Parra et al, 1999). Each of these has a role in the production of ATP through anaerobic energy systems. Researchers have found an increase in the production of each marker with HIT, although a reduction in creatine phosphate was observed during HIT sessions in other studies (Burgomaster et al, 2008; Parra et al, 1999).

Anaerobic glycolysis is a system in which glycogen is converted to ATP for immediate use, typically less than 2-3 minutes (Parra et al, 1999). Changes in glycogen,

such as increased resting glycogen content and a reduced rate of glycogen utilization during matched-work exercise, were found with HIT (Burgomaster et al, 2005; Gibala et al, 2007). The ability to sustain a given work rate is dependent upon the amount of glycogen stored in the muscle prior to exercise. An increased resting glycogen content should result in a delay in the onset of fatigue. Subjects trained every day for two weeks in one study which led to a significantly lowered rate of glycogenolysis, increased creatine kinase, pyruvate kinase, and lactate dehydrogenase activity (Parra et al, 1999). As lactate production increases, it accumulates in the muscles due to an inefficient system of transportation out of the cell. Lactate threshold is defined as the point where blood lactate accumulates in an increasing non-linear fashion, when heavy or severe exercise occurs. The higher the lactate threshold, the less lactate produced at a given work rate or intensity (Gibala et al, 2007). From these results it was assumed that a high production of lactate following repeated bouts of exercise could possibly induce an aerobic adaptation by improving metabolism of pyruvate (Hargreaves et al, 1998; Juel et al, 2004; McKenna et al, 2007; McKenna et al, 2007; Parra et al, 1999).

High intensity exercise bouts less than 10 seconds are considered mainly anaerobic but if a bout lasts longer than 10 seconds a greater demand for energy will occur and cause an increased involvement from the aerobic energy systems (Parra et al, 1999). If a 30 second bout were to be repeated three more times, by the fourth bout, the energy would primarily be derived from oxidative metabolism. This could possibly be attributed to an increased rate of oxygen transport and utilization (Gibala et al,

2008). Studies using sprint-training bouts of 10 seconds with 50-second rest periods found an increase in the peak rate of the sarcoplasmic reticulum Ca^{2+} release following training (Ortenblad et al, 2000; Hargreaves et al, 1998).

Increases in the activity of pyruvate dehydrogenase, Ca^{2+}, citrate synthase, and peroxisome proliferator-activated receptor coactivator (PGC-1α) have been observed with HIT (Burgomaster et al, 2005; Burgomaster et al, 2006; Burgomaster et al, 2007; Hellsten et al, 1998; Krustrup et al, 2004; MacDougall et al, 1998). A few studies have seen an enhanced expression of PGC-1α. This protein functions as a regulator of mitochondrial biogenesis (Coyle, 2005). Burgomaster et al (2008) was the first to investigate this protein with low volume sprint interval training and find a similar increase as that observed with endurance training. The subjects performed one session of four maximal effort 30-second exercise bouts. Thus, HIT could have the ability to stimulate the signaling pathways responsible for mitochondrial biogenesis. Due to the larger motor unit recruitment with HIT, more mitochondria are being utilized, potentially leading to an increased ATP production (Mogensen et al, 2006; Coyle, 2005). It has been proposed that these changes stem from the high muscle fiber recruitment and have been shown to lead to an increased exercise capacity (Burgomaster et al, 2008; Gibala et al, 2009; Gibala et al, 2008; Gibala et al, 2006; Terada et al, 2005).

B. Oxygen delivery

High intensity interval training has also shown improved mechanisms contributing to oxygen delivery. According to one study, as a result of sprint interval

training, greater vasodilation of the blood vessels occurs during exercise which could be a mechanism for a reduction in resistance and a possible increased blood flow. Researchers found enhanced endothelial function and endothelial nitric oxide synthase (eNOS) content of arteries in rats white gastrocnemius muscle (Tereda et al, 2005) when performing swimming HIT. The increased dilation caused a decrease in vascular resistance which was greatest in the white region, the fast twitch skeletal muscle, of the trained rats consequently leading to a proposed overall increase in blood flow capacity (Laughlin et al, 2004). Improved endothelial function usually relates to reduced oxidative stress, an improved antioxidant defense, or upregulation of eNOS gene expression causing an improved NO bioavailability which can cause smooth muscle relaxation (Rakobowchuk et al, 2008).

An increase in blood flow through a decreased vessel resistance allows for a greater oxygen delivery to the working muscles which increases the oxygen available for extraction which in turn may increase VO_2 max, and also the time to exhaustion. While studies using a two week six session HIT protocol did not see an increase in maximal oxygen uptake, protocols lasting four to eight weeks did find an increase in maximal oxygen uptake with HIT (Burgomaster et al, 2008; Harmer et al, 2000; Krustrup et al, 2004; MacDougall et al, 1998; McKenna et al, 1997). All of the studies mentioned above trained with cycling exercise but one study using running HIT found no change in maximal oxygen uptake over the course of six weeks of training (Marles et al, 2007). The studies performing six to seven sessions of HIT over two weeks were sufficient to

double time to fatigue, from 26 minutes to 51 minutes, in an aerobic exercise test to exhaustion performed post HIT (Burgomaster et al, 2005). Other researchers have also found an increased time to exhaustion after HIT (Burgomaster et al, 2006; Harmer et al, 2000; McKenna et al, 1997). Additionally, HR has been found to be lower at a given work rate during exercise tests to exhaustion after HIT (Burgomaster et al, 2008; Harmer et al, 2000).

C. Pulmonary adaptations

To date, it is believed that exercise training does not appreciably alter the pulmonary system. However, HIT may lead to changes in respiratory muscle strength and expiratory flow rates not commonly observed with ET although this has not yet been determined. The mechanisms responsible for these proposed adaptations may be the high ventilations and pressures associated with HIT. Although not directly tested with exercise training, the effects of increased respiratory muscle strength have been observed with studies using inspiratory muscle training (IMT). Specific IMT is typically performed at high ventilation rates at a fixed resistance (~50-70% PImax) similar to rates observed during high intensity exercise (Downey et al, 2007). Researchers have observed increases in inspiratory muscle strength with IMT ranging from 7-41% (Downey et al, 2007; Enright et al, 2006; Mickelborough et al, 2008). A strong relationship exists between cross sectional area and strength of a muscle. Downey et al (2007) has shown an 8-12% increase in diaphragm thickness after four weeks of IMT

supporting the thought that increased cross sectional area increases the strength of a muscle (Downey et al, 2007).

As previously discussed, changes in breathing pattern adopted by ET and potentially even more so with HIT could also lead to increased flow rates through a number of different mechanisms within the pulmonary system. As proposed by Scichilone et al (2005), the hyperventilation associated with heavy exercise has been theorized to cause reductions in smooth muscle tone and contractility leading to a decreased airway resistance and ultimately increased flow rates (Scichilone et al, 2005). Recent studies have demonstrated that the active length-tension relationship in airway smooth muscle is dynamic and adapts to length changes over a period of time (Speich et al, 2007). The properties of mechanical adaptations in airway smooth muscle are proposed to result from dynamic cytoskeletal processes outside of the actomyosin interaction (Zhang and Gunst, 2008). Repeated airway smooth muscle stretch over time may therefore help reduce its tension, leading to increased airflow. By increasing the expiratory flow rates, there could be a potential reduction on the pulmonary limitations discussed above. While this has been indirectly shown with endurance trained subjects, from hyperventilation and adoption of differing breathing patterns, the effects could potentially be even greater with HIT (Eastwood et al, 2001; Holm et al, 2004).

III. Summary

While the effects of HIT on metabolic and cardiovascular adaptations are fairly well know, the effects of HIT on pulmonary function are currently unknown. Although

ET may not lead to appreciable changes in respiratory muscle strength and expiratory flow rates, HIT, due to greater demands placed upon the pulmonary system, potentially could lead to significant improvements in these measures. If an increase in respiratory muscle strength occurs with HIT, it could result in a possible reduction in respiratory muscle fatigue and improved performance. The increased ventilation associated with HIT may also lead to greater airway smooth muscle stretch, decreased smooth muscle tone, and possible increases in expiratory flow rates. Therefore, we hypothesized that high intensity interval training will increase: 1) respiratory muscle strength, and 2) expiratory flow rates to a greater extent than "traditional" endurance training.

CHAPTER II

METHODS

I. SUBJECTS

Twenty healthy men (n = 11) and women (n = 9) volunteered to participate. All subjects were active, but not highly trained, free of heart and pulmonary disease, and non-smokers. After being informed of the risks, subjects completed a medical history questionnaire and signed an informed consent waiver. During the course of the study, five subjects dropped out due to schedule conflicts (n = 2), injuries (n = 2), and illness (n = 1). Fifteen subjects completed the protocol. Subjects maintained their normal activity level throughout the training period. All procedures were approved by the Institutional Review Board at Kansas State University, Manhattan KS.

II. PROTOCOL

Each subject reported to the lab three times prior to training, 12 times for training sessions, and twice post training. During the first visit, measurements included height, weight, waist circumference, body mass index, and a five-mile time trial on a cycle ergometer. During the second visit, pulmonary function tests (PFT) and an incremental maximum oxygen uptake test (VO_2max) were performed. One to two days later they came to the lab to perform a second five-mile time trial to establish a baseline for performance measures. Subjects then completed four weeks of training after being randomly assigned to either a high intensity interval training group (HIT: n = 8) or

endurance group (ET: n = 7). After training was completed on the second to last visit, PFT's, height, weight, body mass index, and a five-mile time trial were performed. On the last visit the VO_2 max test was repeated.

III. EXERCISE TESTING

The incremental maximal oxygen uptake test was performed on a cycle ergometer (Sensormedics 800). Metabolic and ventilatory data were collected and analyzed continuously breath-by-breath throughout exercise (Sensormedics 229 Metabolic Cart, Sensormedics Corp., YorbaLinda, CA). A pulse oximeter (Datex-Ohmeda 3900P, Madison, WI) was used to estimate arterial oxygen saturation (SpO_2). Heart rate (HR) was continuously monitored via a four-lead ECG connected to the metabolic cart. Values from the modified Borg's rating of perceived exertion scale (RPE), measured 1-10, were recorded for each stage of exercise. After four minutes of rest, warm up exercise commenced at 20W for another four minutes with a cadence of 60-70 rpm. Every minute beyond the warm up period the workload was increased by 25W. Termination occurred when the subject was unable to maintain the pedal cadence of 60-70 rpm for five consecutive revolutions.

Five-mile time trials were completed on an electronically braked cycle ergometer (Vision Fitness HRT E3600, Cottage Grove, WI) at least twice pre training, (to familiarize the subject with the test and establish a baseline) after two weeks of training, and once post-training. A HR monitor (Polar FS3C, Lake Success, NY) was worn and HR was recorded every minute. The seat height was recorded and remained constant for all

trials. Subjects were blinded to distance and given verbal cues each mile during the test. At every mile the time was recorded. All subjects were encouraged to complete the time trial as fast as possible.

IV. PULMONARY FUNCTION TESTS

The maximum flow volume loop (Sensormedics 229 Metabolic Cart, Sensormedics Corp., YorbaLinda, CA) was used to determine peak expiratory flow (PEF), forced expiratory flow (FEF_{25-75}), forced expiratory volume at 1 second (FEV_1), forced vital capacity (FVC) and the ratio of FEV_1/FVC. Inspiratory (PImax) and expiratory maximum pressure (PEmax) tests were used to measure respiratory muscle strength. PImax was measured from residual volume and PEmax was measured from total lung capactiy. All PFT's were performed a minimum of three times, with the average of the three closest values used for analysis.

V. TRAINING

Endurance training subjects trained on an electronically braked cycle ergometer (Sensormedics 800) three days/week, alternating days, for four weeks. Training began with a five-minute warm-up period at 20W. Workload was determined from their incremental VO_2 max test at 60-70% of their final workload. A training session consisted of 45 minutes of constant load cycling. Pedal cadence was maintained between 50-80 rpm. HR was recorded each minute for the warm-up period and every five minutes during the training session.

High intensity interval training subjects also trained on an electronically braked cycle ergometer (Sensormedics 800) three days/week, alternating days, for four weeks. Seat height was determined by the subject and recorded to remain consistent throughout training. Each individual training session began with a three-minute warm-up at 20 W. After the warm-up period, the subjects performed a one-minute bout at a workload eliciting 90% of their VO_2 max test's final workload. Pedal rate was maintained between 60-100 rpm. When the interval was completed, subjects pedaled for a three-minute recovery period at 20W. This was repeated a total of five times in one session totaling 20 minutes, excluding the warm-up period. HR was recorded every minute of the warm-up and exercise. Workload values remained constant for the four-week training period. Maximum flow volume loops, PImax, and PEmax tests were performed at the end of each week of training for both groups.

VI. *STATISTICAL ANALYSIS*

SigmaStat statistical software (Jandel Scientific Software) was used for data analysis. Data is expressed as mean ± standard deviation. A 2x2 (group vs time) mixed ANOVA was used to determine differences. Significance was set at $p < 0.05$ for all analyses.

CHAPTER III

RESULTS

I. SUBJECTS

Subject characteristics are shown in Table 1. The male-to-female ratio was similar between groups, ET (m = 4, f = 3) HIT (m = 5, f = 3). No significant differences were detected between sexes, therefore men and women were grouped together for analysis. Groups were well matched ($p>0.05$) for age, height, weight, body mass index, and waist circumference. Body weight, body mass index, and waist circumference did not change ($p>0.05$) with training.

Table 1: Subject Characteristics

	Pre-training		**Post-training**	
	Endurance (n = 7)	**HIT (n = 8)**	**Endurance (n = 7)**	**HIT (n = 8)**
Age (yrs)	21.3 ± 2.3	20.2 ± 2.1		
Height (cm)	170.2 ± 10.9	170.7 ± 6.7		
Weight (kg)	67.1 ± 11.8	71.4 ± 10.6	67.1 ± 11.3	71.0 ± 10.2
BMI (kg/m^2)	23.2 ± 2.6	24.2 ± 2.2	23.2 ± 2.6	24.0 ± 2.0
Waist circumference (cm)	75.0 ± 5.8	79.1 ± 6.9	75.7 ± 6.5	78.6 ± 6.9

Values are presented as mean ± SD
No differences ($p > 0.05$) were observed in either group with training

II. TRAINING

Training adherence was 98% for all subjects across all training sessions. The total work per training session for ET was 408.86 ± 109.04 kJ and for HIT was 86.06 ± 13.44kJ (including recovery) ($p<0.05$). Average heart rate for both groups during training is shown in Table 2. HR values were significantly higher for HIT compared to ET. In the ET group, HR averaged 77.0 ± 3.3% of HR max, while the workload ranged from 110-225W. For HIT, HR averaged 84.9 ± 0.5% of HR max, while the workload ranged from 130-265W. HR also decreased from weeks one and two to weeks three and four with ET.

Table 2: HR During Training

Endurance	**Monday**	**Wednesday**	**Friday**	**Average**
Week 1	164.1 ± 11.0	160.3 ± 9.8	158.0 ± 10.9	160.8 ± 3.1
Week 2	156.3 ± 6.5	156.3 ± 9.4	158.3 ± 12.3	157.0 ± 1.1
Week 3	150.7 ± 9.8	152.0 ± 18.3	150.3 ± 17.0	151.0 ± 0.9ł
Week 4	145.3 ± 14.5	149.6 ± 14.4	149.8 ± 19.1	148.2 ± 2.5ł
HIT				
Week 1	170.7 ± 7.9	170.7 ± 12.5	167.4 ± 9.7	169.6 ± 1.9*
Week 2	167.9 ± 7.4	168.7 ± 10.1	167.4 ± 11.8	168.0 ± 0.7*
Week 3	168.9 ± 10.6	169.7 ± 8.4	170.1 ± 9.9	169.6 ± 0.6*
Week 4	168.7 ± 9.4	170.1 ± 9.9	169.0 ± 13.7	169.3 ± 0.7*

Values are presented as mean ± SD
*significantly different from ET; $p < 0.05$
Ł significantly different from weeks 1 & 2 of ET; $p < 0.05$
Final column is an average of all values from the week

III. PULMONARY FUNCTION TESTS

Table 3 shows ET and HIT pulmonary function values pre and post training. Groups were well matched prior to training with no significant differences between groups. The middle column includes the percentage of the predicted values for the subjects. Following training there were no differences ($p>0.05$) for any measured variable or between groups.

Table 3: Pulmonary Function Tests

	Pre-training			**Post-training**	
	Endurance (n = 7)	**HIT** (n = 8)	**Predicted Values** (%)	**Endurance** (n = 7)	**HIT** (n = 8)
PEF (L/sec)	6.27 ± 2.13	6.14 ± 1.61	75.5 ± 14.7	6.81 ± 2.58	7.87 ± 2.83
$FEF_{25-75\%}$ (L/sec)	3.19 ± 0.83	3.66 ± 0.55	79.3 ± 12.5	3.11 ± 1.18	3.84 ± 0.51
FVC (L)	3.44 ± 0.95	3.52 ± 0.71	90.7 ± 9.4	3.4 ±1.27	3.63 ± 0.86
FEV_1 (L)	4.39 ± 1.27	4.24 ± 0.96	97.1 ± 10.5	4.31 ± 1.61	4.02 ± 0.88
FEV_1/FVC (%)	78.7 ± 2.5	83.7 ± 5.1	81.2 ± 4.8	78.9 ± 4.7	91.0 ± 13.8

Values are presented as mean ± SD
Predicted values from West (2008)

IV. RESPIRATORY MUSCLE STRENGTH

Weekly PImax values are shown in Figure 1. There were no differences ($p>0.05$) between groups prior to training. Both groups significantly increased PImax with training (ET ~25%; HIT ~43%), with values significantly higher for HIT than ET following four weeks of exercise training. Figures 2A and 2B show individual PImax values pre and post training for ET and HIT respectively. All subjects in both the ET and HIT group increased PImax with training. PEmax was not different ($p>0.05$) between groups prior to training. There was also no difference ($p>0.05$) in PEmax post training or between groups (ET: 131.0 ± 20.4 cmH_2O, HIT: 123.1 ± 44.8 cmH_2O).

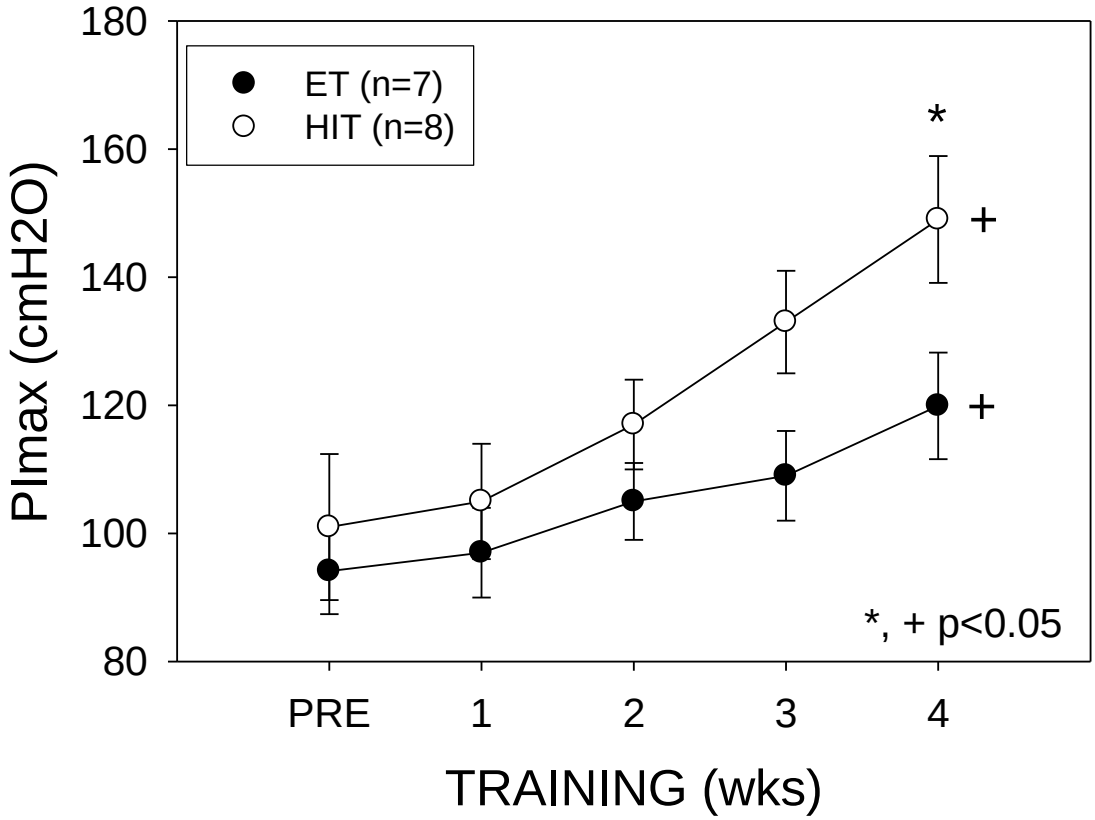

Figure 1. Maximal inspiratory pressure (PImax) before and after each week of training. PImax was significantly increased from baseline and significantly higher with HIT ($p < 0.05$). HIT increased ~43% by week 4 and ET increased ~25%.

* significantly different between groups; $p < 0.05$

+ significantly different from pre; $p < 0.05$

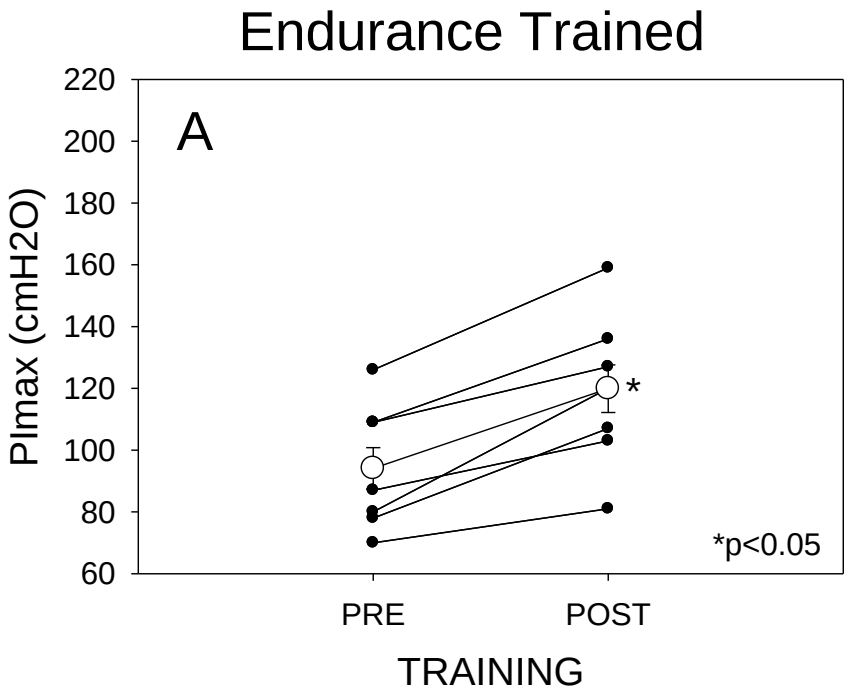

Figure 2A. Individual and mean data pre and post training for the ET group. The open circles are the mean for the ET group pre and post training. The filled circles are the individual subject data. *significant at $p < 0.05$

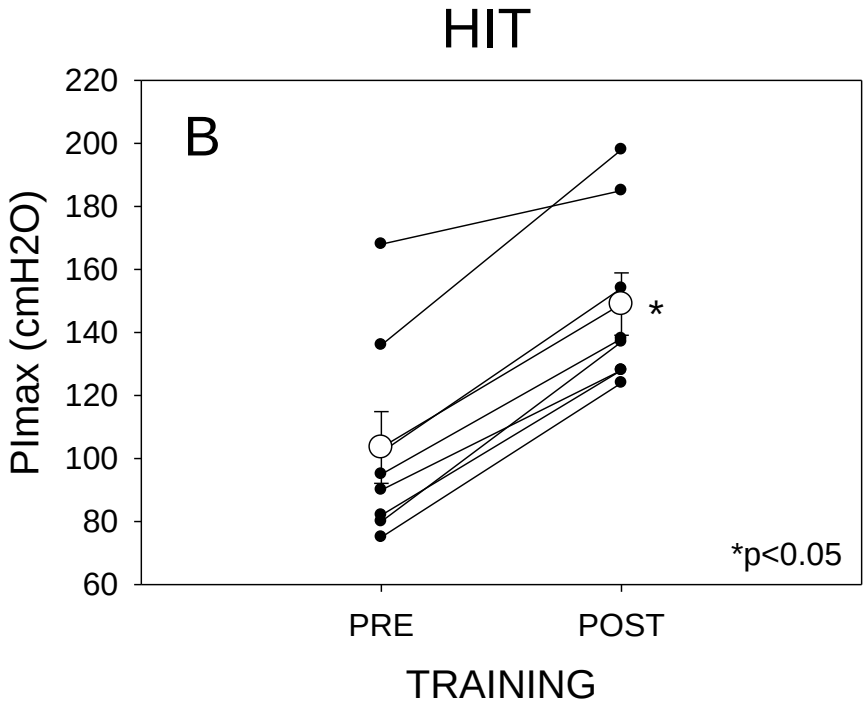

Figure 2B. Individual and mean data pre and post training for the HIT group. The open circles are the mean for the HIT group pre and post training. The filled circles are the individual subject data. *significant at $p < 0.05$

IV. VO_2 MAX DATA

Data recorded during the VO_2 max test are shown in Table 4. Following training, both groups significantly increased VO_2 max by ~8-10% with no difference ($p>0.05$) between groups. Post training there were no differences ($p>0.05$) between groups on any measure.

Table 4: VO_2 max Data

	Pre-training		**Post-training**	
	Endurance (n = 7)	**HIT** (n = 8)	**Endurance** (n = 7)	**HIT** (n = 8)
VO_2max (L /min)	2.24 ± 0.86	2.36 ± 0.56	2.37 ± 0.80*	2.58 ± 0.58*
VO_2max (ml/kg/min)	32.5 ± 7.9	33.3 ± 5.7	34.3 ± 6.7*	36.5 ± 6.7*
VE (L/min)	74.8 ± 18.8	82.6 ± 17.8	83.5 ± 14.6	95.9 ± 17.8
RER	1.15 ± 0.11	1.12 ± 0.06	1.20 ± 0.11	1.15 ± 0.05
VE/VO_2	35.1 ± 6.9	35.9 ± 6.0	38.4 ± 7.3	37.9 ± 3.7
VE/VCO_2	30.6 ± 4.1	32.0 ± 4.2	31.8 ± 4.8	32.9 ± 2.4
HR (bpm)	176.3 ± 4.0	176.7 ± 10.4	179.1 ± 1.3	182 ± 11.8
S_pO_2 (%)	97.3 ± 2.5	97.5 ± 1.5	97.7 ± 1.0	95.1 ± 2.6

Values are presented as mean ± SD
* significant at $p < 0.05$

VI. TIME TRIALS

Figure 3 shows the pre and post training time trial values for ET and HIT. There was no difference ($p>0.05$) between groups prior to training (1072 ± 198 seconds for ET and 1089 ± 136 seconds for HIT) or after two weeks of training. Following four weeks of training, both groups significantly decreased time to completion of the five miles to 1024 ±177 seconds in endurance (~4.4%) and 1003 ±104 seconds in HIT (~6.5%) post training with no difference ($p>0.05$) between ET and HIT.

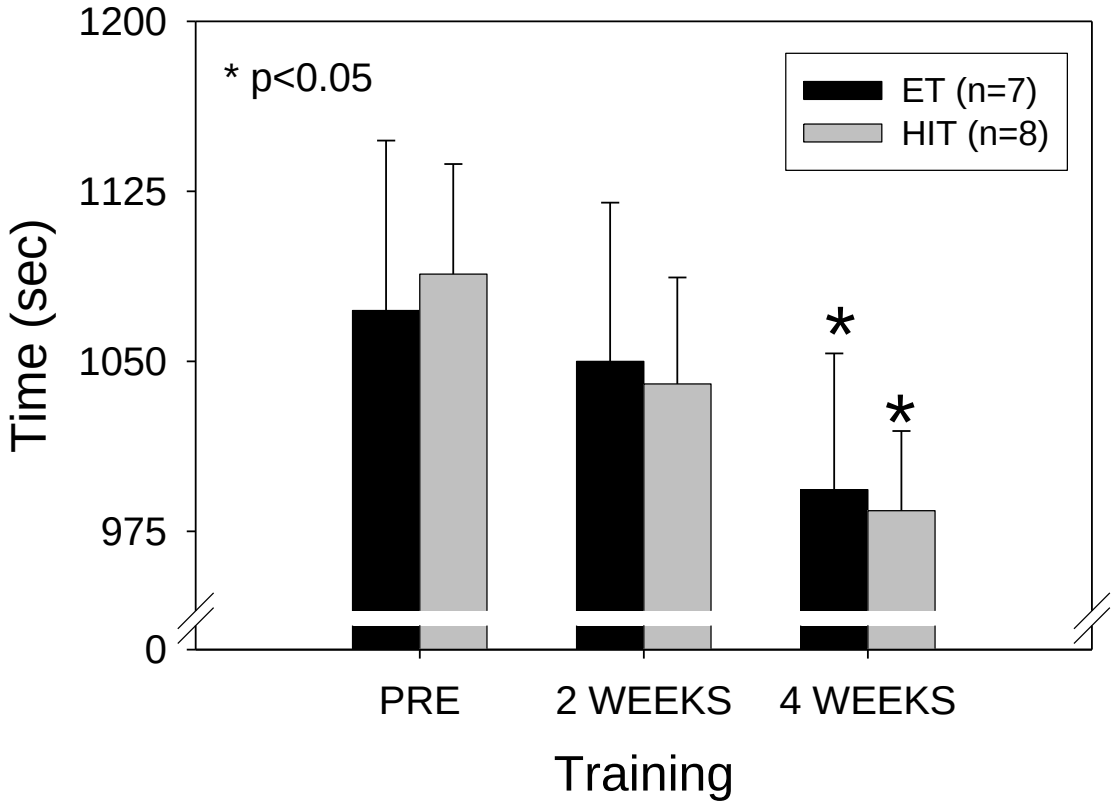

Figure 3. Five mile time trial in seconds to completion from pre training, after two weeks, and after four weeks. Time to completion was significantly different at four weeks than pre training and 2 weeks. There was no difference between groups ($p < 0.05$)

CHAPTER IV

DISCUSSION

The purpose of this study was to determine the effects of high intensity interval training (HIT) on pulmonary function. Major findings were that both ET and HIT led to significant increases in inspiratory muscle strength with significantly greater increases with HIT. However, no differences were observed in expiratory flow rates with training or between groups. Interestingly, similar increases in VO_2 max and decreases in the time to completion of time trials were found in both groups despite the fact that the total amount of work per training session was substantially less for HIT.

I. RESPIRATORY MUSCLE STRENGTH

Our findings have demonstrated that four weeks of either HIT or ET resulted in ~25-43% increases in inspiratory muscle strength. While no studies to our knowledge have directly assessed the effects of whole body exercise training on respiratory muscle strength, the improvements in inspiratory muscle strength that we have demonstrated are similar to studies utilizing inspiratory muscle training (IMT). Inspiratory muscle training typically uses high ventilation rates and large pressures that are similar to those experienced during HIT. Research suggests that most inspiratory muscle strength gains occur when intensity is at or above 60-80% of maximum inspiratory pressure (PImax) (Enright et al, 2006). Using IMT, one study observed an increase in inspiratory muscle strength of ~41% in healthy subjects (Enright et al, 2006). Other studies using healthy

subjects found smaller increases in inspiratory muscle strength of ~7-17% (Downey et al, 2007; Gething et al, 2004; Holm et al, 2004; Witt et al, 2007). The difference in these studies appears to be the intensity at which the IMT was performed, varying from 50-80%, with the largest increases observed at 80% (Downey et al, 2007; Enright et al, 2006; Gething et al, 2004; Witt et al, 2007). Effects observed with high intensity IMT are consistent with those observed in this study, suggesting that the greater inspiratory muscle strength we observed with HIT compared to ET, was due to greater demand placed on the respiratory muscle with HIT. We believe this is the first study to demonstrate substantially greater increases in inspiratory muscle strength with HIT.

II. EXPIRATORY FLOW RATES

We are mildly surprised that expiratory flow rates did not change with HIT. While there has been no direct evidence of increased expiratory flow rates with training, research has suggested that repeated lung inflation induced airway stretch may reduce airway resistance through decreased smooth muscle tone and contractility (Scichilone et al, 2005). A reduction in resistance could also cause a greater dilation in the airways. However, despite large amounts of airway stretch, lung inflation, and high ventilation rates that are associated with HIT, it apparently was not sufficient to lead to expansion of the maximal flow volume loop or an increase expiratory flow rates. Also, it is possible that four weeks is not a sufficient length of time to induce these adaptations. Our results are consistent with prior research which has found unaltered airway resistance with an increase in inspiratory muscle strength, endurance, and lung volumes in

endurance athletes, typically marathoners or road cyclists (Guenette et al, 2009). Together, these results suggest that training simply may not lead to increased airway smooth muscle stretch.

III. WORK AND TRAINING VOLUME

Similar physiological adaptations have been reported to occur in metabolic and cardiovascular measures with ET and HIT, as well as similar improvements in performance, which agrees with our study (Burgomaster et al, 2008; Gibala et al, 2006; Krustrup et al, 2004; Rakobowchuk et al, 2004). In these previous reports, similar increases were observed with VO_2 max (~3-4ml/kg/min), time trials (~4-10%), and power output (~7-17%). Surprisingly these changes occurred despite the volume of training performed by ET being much greater than HIT. In these studies, the amount of work ranged from 225-315 kJ/week for HIT and 2250-3250 kJ/week for ET, which is similar to our study. These reports also showed increases in mean power output, and the time to the onset of fatigue with no differences between ET and HIT (Burgomaster et al, 2006; Burgomaster et al, 2008; Gibala et al, 2006; Mogensen et al, 2006; Rakobowchuk et al, 2008). Researchers have also found a decrease in the time to completion of time trials and a reduction in HR at a given workload and resting HR (Burgomaster et al, 2008; Gibala et al, 2006). We found the reduction in HR at a given workload to be true for the ET group in our study as well. Yet, the vast difference in volume and work elicited similar changes (VO_2 max, time trials) over only a four week training program. Our study therefore agrees with and provides additional support to

many studies that have utilized similar protocols with similar training volume differences (Burgomaster et al, 2006; Burgomaster et al, 2008; Gibala et al, 2006; Rakobowchuk et al, 2008).

IV. *IMPLICATIONS*

The greater inspiratory muscle strength that occurred with HIT, as well as that observed with ET in our study, may lead to improved exercise performance through decreased respiratory muscle fatigue. Although we did not directly assess respiratory muscle fatigue in our study, we can speculate that the increase in inspiratory muscle strength delayed the onset of fatigue as evidenced though the decreased time to completion of time trials.

As exercise intensity or duration increases, so does the demand for blood flow to all working muscles due to an increased demand for oxygen (Dempsey et al, 2006; Enright et al, 2006; Witt et al, 2007). Expiration becomes active which requires larger muscle recruitment, and a larger amount of oxygen demanding an increase in blood flow (Babcock et al, 2002; Johnson et al, 1993; Witt et al, 2007). Evidence suggests that the demand for an increase in blood flow to the respiratory muscles during maximal or heavy exercise "steals" blood flow from the other exercising muscles through sympathetically mediated vasoconstriction (Harms et al, 1998; St. Croix et al, 2000). As metabolites accumulate due to this vasoconstriction, the muscle metaboreflex is activated to restore blood flow. It is thought that the diaphragm is potentially the reason for vasoconstriction to the working muscles (Dempsey et al, 2006; Enright et al,

2006; Harms et al, 1998). During high-intensity exercise (ie >90% VO_2 max), proportional assist ventilation, reducing the work of breathing, has been shown to help prevent or delay diaphragmatic fatigue and increase performance (Harms et al, 2000). This suggests that respiratory muscle fatigue can be reduced with exercise training possibly resulting in an improved performance.

Our study observed improvements in performance through decreased time to completion of time trials. However, the magnitude of decrease was not significantly different between groups. While theories discussed above would suggest that the increases in performance would be larger with HIT, it is possible that the main source of adaptation is exercise training in itself regardless of the type of training. This theory is supported by Iaia et al (2008) who investigated endurance trained runners and divided them into two groups, one that performed moderate intensity exercise and the other implementing a speed endurance training program. Improvements in performance, as measured through time trials, were similar between groups. Additionally, in our study we did not see a difference between groups in VO_2 max post training. Thus, it is not unexpected that time trials were not different between groups.

V. LIMITATIONS

There are several potential limitations that we have identified which could have affected our results. One limitation is the pedal rates during training. While most subjects pedaled within the same range (ET 45-80rpm, HIT 60-100rpm) each subject was free to pedal at their preferred rate. Previous reports have shown that power output

and endurance exercise time increased when pedal rates were freely chosen or slightly below the freely chosen rate by trained and untrained subjects (Nielsen et al, 2004). When pedal rate was slightly above the freely chosen rate, the endurance capacity was reduced (Nielsen et al, 2004). In our study all subjects were supervised for the entirety of each training session and were encouraged to work at the same intensity throughout the session. Subject effort was consistent as evidenced by the small range of HR deviation throughout each session. Second, the use of maximal inspiratory and expiratory pressure tests are indirect estimates of respiratory muscle strength. While these are commonly and widely accepted methods for measuring respiratory muscle strength, these tests are effort dependent. However, multiple tests were performed prior to, during, and after training by the same investigator to ensure consistency. Therefore, due to the similarity in obtained values within each session, we believe these values are accurate. Thirdly, we did not use a measure of respiratory muscle fatigue in our study. Respiratory muscle fatigue may be an important component of performance. A measure of respiratory muscle fatigue would have helped in part to determine a mechanistic basis for improvements in performance with training.

VI. FUTURE DIRECTIONS

This is the first study, to our knowledge, to investigate the effects of HIT on pulmonary function. We found a greater increase in inspiratory muscle strength and a range of adaptations similar to that seen with traditional endurance training but with a much smaller volume and total work. While this suggests that HIT is a useful alternative

form of training, it leaves many questions yet unanswered. For example, to better understand how increased inspiratory muscle strength contributes to endurance and fatigue, measurements of respiratory muscle fatigue should be determined. To establish if the adaptations we observed were a result of the exercise itself or the increased ventilation rates, future studies should determine the effects of hyperventilation only on changes in inspiratory muscle strength and expiratory flow rates. Also, much more research is needed to understand the long term training effects of HIT. To our knowledge, no studies have utilitzed a protocol longer than eight weeks of training. We do not know whether the adaptations seen in this short period of time are sustainable or able to be improved upon beyond eight weeks. Investigating the ability to maintain these adaptations, could provide us with an answer to whether or not there would be additional changes. Finally, our study used a population of young, healthy individuals. This does not necessarily imply that this type of training is not beneficial for other populations. People with chronic obstructive pulmonary disease (COPD) and children are two populations that could potentially benefit from HIT. It has been suggested that with COPD patients, the advantages of rehabilitation depend on the training intensity used (Hsieh et al, 2007). Thus far, most studies utilize inspiratory muscle training to rehabilitate COPD patients (Hill et al, 2007). Potentially, HIT may be a useful alternative training approach. Children reach higher ventilations during high intensity exercise than adults, partially because they are more susceptible to developmental changes such as increased lung volumes, than adults depending upon

the stage of maturation (Nourry et al, 2005). It is possible the adaptations could be even greater with children.

VII. CONCLUSION

Training induced adaptations are known to improve exercise performance. New and varied forms of training are being developed and utilized routinely. Yet, despite much understanding involving the effects of exercise training to overall health and fitness, most people do not get the recommended amount necessary to induce these adaptations. High intensity interval training provides a time efficient alternative form of training to traditional endurance training that yields similar benefits. Results from our study suggest benefits in inspiratory muscle strength as well. This may act to reduce pulmonary limitations to exercise performance. Further research is needed to determine the mechanistic bases for these improvements and their implications to respiratory muscle fatigue and exercise performance.

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