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The Effect of Respiratory Muscle Training on 20km Cycling Time Trial Performance

Chad Butts
Ithaca College

THE EFFECT OF RESPIRATORY MUSCLE TRAINING ON 20 KM CYCLING TIME TRIAL PERFORMANCE

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Chad Butts

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CERTIFICATE OF APPROVAL

MASTER OF SCIENCE THESIS

This is to certify that the Thesis of

Chad J Butts

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Thesis Advisor:

Committee Member:

Candidate:

Chair,
Graduate Program:

Dean of Graduate Studies:

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ABSTRACT

The purpose of this study was to investigate the effects of concurrent inspiratory and expiratory muscle training (CRMT) on maximal and sub-maximal exercise performance. Ten trained cyclists were randomly assigned to an experimental (EG; $n = 5$) or control (CG; $n = 5$) group. The mean ± *SD* for age, weight, and VO_{2max} for all subjects was 27 ± 5.7 y, 76.3 ± 8.9 kg, and 57.8 ± 7 $ml \cdot kg^{-1} \cdot min^{-1}$. Both groups trained for 6 wk using a handheld breathing device. EG performed 3x10 repetitions twice a day, while CG performed 30 repetitions once a day with a sham device; both groups were blind to the study intent. Pulmonary, physiological, and perceptual responses to maximal, sub-maximal, and 20 km time-trial were assessed. CRMT significantly increased inspiratory muscle strength (PI_{max}) by 28% ($p = 0.026$) and lowered time-trial perceived exertion (TT_{RPE}) by 9.7% across time ($p = 0.017$). There were no other changes across time or differences between groups before or after training. Six wk of respiratory muscle training improves inspiratory muscle strength in endurance trained individuals; it also decreases the perception of effort during intense prolonged efforts lasting longer than 30 minutes. In conclusion, although CRMT improved PI_{max} and lowered TT_{RPE}, it did not affect maximal or sub-maximal exercise performance.

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Chapter 1

INTRODUCTION

The ventilatory muscles of untrained and trained individuals fatigue during maximal and sustained sub-maximal exercise (Boutilleur, Buchel, Kundert & Spengler, 1992; Dodd, Powers, Thompson, Landry, & Lawler, 1989; Fitting, 1991; Johnson, Aaron, Babcock, & Demsey, 1996; Romer, McConnell, & Jones, 2002b). Despite this fatigue, ventilatory homeostasis is usually maintained in healthy untrained individuals and consequently does not limit their exercise performance (Boutilleur et al., 1992). However, in some highly-trained athletes (VO_2max >55 $ml \cdot kg^{-1} \cdot min^{-1}$), the respiratory system may limit exercise performance as the cardiovascular and metabolic adaptations resulting from whole-body endurance training exceed those that occur in the pulmonary system (Dempsey, 1986). Since whole-body endurance training may not optimize ventilatory adaptations in some well trained athletes, researchers have augmented these athletes' training regimens with specific forms of exercise for the respiratory muscles (RM) to determine if such training enhances respiratory muscle function and physical performance (Boutilleur et al., 1992; Dempsey, 1986; Dodd et al., 1989; Fairbarn, Coutts, Pardy, & McKenzie, 1991; Fitting, 1991: Inbar, Weiner, Azgad, Rotstein, &Weinstein, 2000; Johnson et al., 1996; Morgan, Kohrt, Bates, & Skinner, 1987; Romer, McConnell, & Jones, 2002a; Volianitis, McConnell, Koutedakis, McNaughton, Backx & Jones, 2001).

Respiratory muscle training (RMT) methods vary greatly across these studies. Irrespective of the training methodologies, these RMT programs improved some aspect of respiratory muscle performance, such as RM strength, assessed by measuring maximal inspiratory and expiratory mouth pressures, which increased by as much as 50% and 11%, respectively, in well-trained athletes (Fairbarn et al., 1991; Romer et al., 2002a, 2002b; Volianitis et al., 2001). Even though RMT improves RM performance, it does not alter maximal exercise performance (> 85% of VO_2max) in well-trained individuals (Inbar et al., 2000; Morgan et al., 1987).

The effect of RMT on sub-maximal exercise performance, however, is less conclusive. Many studies show that RMT improves sub-maximal time-trial (TT) performance (Boutellier, 1998; Fairbarn et al., 1991; Inbar et al., 2000; Romer et al., 2002a; Spengler, Roos, Laube, & Boutellier, 1999; Voliatitis et al., 2001). Eleven weeks of RMT, for example, improved 5000-m rowing TT performance by 3.1% in well trained female rowers (Volianitis et al., 2001). Similarly, 6 wk of RMT increased simulated 20 and 40 km TT performance by 3.8 ± 1.7% and 4.6 ± 1.9%, respectively, in well trained male cyclists (Romer et al., 2002a). In contrast to the aforementioned studies that show RMT improves submaximal exercise performance, many other studies show that RMT does not (Fairbarn et al., 1991; Hanel & Secher, 1991; Morgan et al., 1987; Sonetti, Wetter, Pegelow, & Dempsey, 2001; Williams, Wongsathikun, Boon, & Acevedo, 2002).

There are myriad explanations for the inconsistencies in the literature, such as the duration of the RM training protocol and the type of exercise test used for sub-maximal evaluation. RMT programs that lasted more than 4 wk, for example, typically improved sub-maximal exercise performance, whereas those that lasted less than 4 wk did not (Fairbarn et al., 1991; Hanel & Secher, 1991; Morgan et al., 1987; Sonetti et al., 2001; Williams et al., 2002). More important than training duration may be the type of test used to assess sub-maximal performance. Many studies showing a lack of improvement in sub-maximal performance assessed changes using an open-ended or time-to-fatigue task. These tasks are completed at a certain percentage of VO_2max, usually 80 to 95%, and end when the subject reaches volitional exhaustion (Fairbarn et al., 1991; Hanel & Secher, 1991; Morgan et al., 1987). In contrast, studies showing improvements in sub-maximal performance generally assessed sub-maximal performance with a close-ended or fixed time or distance test, such as a time-trial (Boutellier et al., 1992; Romer et al., 2002a; Spengler et al., 1999; Voliantitis et al., 2001). Such close-ended tests are a more valid, reliable, and reproducible than time-to-fatigue tasks. Close ended tests also simulate real racing performance by more closely approximating the stress and motivation of actual competition (Jeukendrup, Saris, Brouns, & Kester, 1996). Hence, studies using close-ended tests or time-trial tasks rather than open ended tasks may be

more meaningful when assessing the literature on the effects of RMT on performance. Future studies should consider close-ended tests for performance assessment given the aforementioned strengths of such protocols.

Historically, most RMT research used protocols that trained only the inspiratory muscles, neglecting the expiratory musculature, which are also heavily recruited during intense exercise. Fatigue in this musculature, moreover, accelerates inspiratory muscle fatigue (Suzuki, Suzuki, & Okubo, 1991), which may further decrease exercise performance. Like the inspiratory muscles, RMT also improves expiratory muscle performance (Grinton, Powers, Lawler, Criswell, Dodd, & Edwards, 1992). The extent of expiratory muscle adaptations to RMT on exercise performance, however, are not known. It is possible that concurrently training the inspiratory and expiratory muscles will elicit greater changes in RM and sub-maximal exercise performance than just inspiratory muscle training.

The purpose of the proposed study, therefore, was to examine the effects of concurrent inspiratory and expiratory muscle training (CRMT) on RM function and maximal and sub-maximal exercise performance. A close-ended 20 km TT test will increase the reliability and external validity of sub-maximal performance.

Scope of the Problem

The purpose of this study was to determine the effects of respiratory muscle training and if these changes improve time-trial cycling performance among moderately trained cyclists.

Hypotheses

The hypotheses for this study were: (1) respiratory muscle strength and endurance will improve after 6 weeks of RMT; (2) sub-maximal blood lactate values would decrease following RMT; (3) there will be no change in maximal exercise performance and (4) 20 km TT performance will improve following RMT.

Delimitations

The delimitations of the study were as follows:

1. Cyclists were recruited from the local community to voluntarily serve as subjects and were currently training on their own.
2. The possibility that training and learning effects may have influenced the results was accepted; an attempt to minimize these effects was made by selecting an appropriate research design.
3. Even though all subjects had at least 2 years of competitive cycling experience, some were not experienced in time-trial racing.
4. Only one sub-maximal workload will be used to detect changes in performance.

Limitations

The limitations of the study were as follows:

1. These results may only apply to moderately trained cyclists and are not indicative of what might occur in non-trained or elite athletes.
2. The results only apply to short-term (6 wk) respiratory muscle training programs.
3. Respiratory muscle and physical training were not objectively measured during the 6 wk of study. Assumptions on these variables were based on voluntary journal entries.

Assumptions of the Study

For the purpose of this study, the following assumptions were made at the start of the investigation:

1. Testing equipment was calibrated and reliable enough to detect differences between conditions.
2. Subjects are representative of moderately trained cyclists who have been training and competing for several years.
3. Subjects adhered to all the regulations of the study and were honest when

answering all questions and reporting training habits.

4. Subjects continued to train on the bike during the 6 wk of the study without drastic increases or decreases in volume or intensity.
5. Subjects were adequately screened for fitness and conditions that may limit the applicability of their results in this study.
6. Subjects completed all the required RM training sessions.
7. Simulated 20 km time trials are valid indicators of changes in fitness and physiological function.

Definition of Terms

The following terms are operationally defined for the purpose of this investigation:

Respiratory Muscle Training (RMT). Using a device or training method to isolate and specifically train the respiratory muscles (e.g. diaphragm, intercostals, abdominals).

Time-Trial (TT). A competitive cycling event in which riders race one at a time over a selected course or distance and the fastest time wins.

Maximal Oxygen Consumption ($\dot{V}O_{2max}$). A GXT used to assess the maximal amount of oxygen one can utilize during aerobic exercise. It is measured in liters per minute ($l \cdot min^{-1}$) or millimeters per minute per kilogram of body weight ($ml \cdot kg^{-1} \cdot min^{-1}$)

Maximal Power Output (PO_{max}). The maximal amount of energy produced during the $\dot{V}O_{2max}$ test. It is measured in watts.

Watts (W). A measure of the power produced during an exercise test.

Maximal Voluntary Ventilation (MVV_{15}). A 15 s long test in which a subject blows into a device as fast as possible for 15 s to assess lung capacity.

Respiratory Muscle Endurance (RME). A test measuring how long the respiratory muscles can perform against a given workload.

Maximal Inspiratory Mouth Pressure (PI_{max}). A test that measures the maximal amount of pressure in cm H_2O the inspiratory muscles can produce.

Maximal Expiratory Mouth Pressure (PE_{max}). A test that measures the maximal

amount of pressure in cm H_2O the expiratory muscles can produce.

Computrainer™ Pro 8001. A stationary bicycle trainer, which provides resistance and allows one to measure workloads in watts.

Micro Medical Mouth Pressure Meter. A handheld device that a subject blows into; it measures the strength of the respiratory muscles.

PowerLung®. A handheld device used to train the respiratory muscles by providing adjustable resistance for each inhalation and exhalation.

Blood Lactate. An indirect measure of anaerobic metabolism taking place in the working muscle.

Rating of Perceived Exertion (RPE). A relative measure of how hard a subject is working during exercise based on a scale of 6-20.

Rating of Ventilatory Exertion (RVE). A subjective rating of how hard a subject feels he or she is breathing based on a scale of 1-10.

Inspiratory muscles. Diaphragm, external intercostals, scalene (anterior, middle, and posterior), and sternocleidomastoid.

Expiratory Muscles. Internal intercostals, latissimus dorsi, quadratus lumborum, and abdominals.

Chapter 2

LITERATURE REVIEW

Introduction

The lungs are suspended by pleural sacs, which never physically touch the lung tissue, although they are attracted to each other by small hydrostatic forces that are created within the fluid filled layers between them. This viscous layer also serves to decrease friction between the two during the movements of ventilation. The pleural sacs are also indirectly connected, by way of this hydrostatic cling, to the thoracic cage (ribs, sternum, spine, and diaphragm) causing them to conform to the same size and shape of this cavity (Wilmore & Costill, 1994).

Respiratory passageways transport air from the external environment to the gas exchange zones deep within the lungs (Brown, 2000). These passageways begin at the mouth and nose and travel through the neck and thorax continually branching into smaller and smaller airways, terminating at the alveolar sacs. These passageways include the nasal cavity, oral cavity, pharynx, larynx, trachea, primary bronchi, secondary bronchi, segmental bronchioles, terminal bronchioles, respiratory bronchioles, alveolar duct, and terminal alveolar sacs (type I and II alveolar cells) (Brown, 2000). These passageways eventually reach the alveoli, bulbous, one-cell layer thick sacs that when laid out flat have a combined surface area of a tennis court (60-80m^2) (Martini, 1992). This large surface area combined with the thinness of the alveolar cells maximizes gas exchange via diffusion (Martini, 1992).

The primary inspiratory muscle is the diaphragm, which contracts and flattens at the base of the thoracic cavity. This contraction increases the volume within the thoracic cavity, pulling and expanding the lungs with it. This expansion increases pulmonary volume, which decreases alveolar pressure relative to the atmosphere outside the lungs, allowing the bulk flow of air down this pressure gradient into the lungs (Rafoth, 2000).

During resting ventilation, the external intercostals muscles also contract, causing the ribs and sternum to swing up and out, further increasing thoracic volume and decreasing alveolar pressure (Wilmore & Costill, 1994). At rest, the process of expiration is a passive process whereby the relaxation of inspiratory muscles and elastic recoil of the lung tissue passively decrease the size of the thoracic cavity, elevating alveolar pressure above atmospheric pressure, which forces air out of the lungs via bulk flow (Wilmore & Costill, 1994).

Pulmonary ventilation ($l \cdot min^{-1}$) is the process of moving air in and out of the lungs via bulk flow, which is a mass movement of air in or out of the lungs that maintains the alveolar ventilation needed for blood gas homeostasis (Wilmore & Costill, 1994). Pulmonary ventilation is defined as the product of tidal volume (V_T), the amount of air moved in per breath, and breathing frequency (V_f) (Martini, 1992). Humans typically ventilate 12 to 20 times a minute and have a resting ventilation of approximately 9 $l \cdot min^{-1} \cdot breath^{-1}$ (Seiler, 1997). This ventilation can increase to 200 $l \cdot min^{-1}$ during maximal exercise (Seiler, 1997). A maximal inhalation followed by a maximal expiration is termed vital capacity (VC), which varies among individuals and is related to gender, age, and body size; VC typically ranges from 3000 to 6000 milliliters (Martini, 1992). No matter how much effort is put into expiration, however, there is a residual volume (V_{RV}) of air left in the lungs. Like VC, V_{RV} varies among individuals and is related to gender, age, and body size; it typically ranges from 800-1400 milliliters.

Regulation of Ventilation

The regulation of ventilation involves the coordination of many complex mechanisms. The respiratory centers (inspiratory and expiratory) located in the brainstem exhibit direct control over most of the ventilatory process (Wilmore & Costill, 1994). But these respiratory centers do not regulate ventilation alone. Sensitive areas in the brain, aortic arch (aortic bodies), and bifurcation of the common carotid artery

(carotid bodies) respond to changes in the chemical environment of the blood. These chemoreceptors are sensitive to changes in O_2, CO_2, and hydrogen ion [H^+] concentations, and stimulate the respiratory centers to change ventilation to maintain blood gas homeostasis (Wilmore & Costill, 1994). In addition, the bronchioles and alveoli also contain stretch receptors, which send direct signals to the respiratory centers in response to pressure or tension developed by the respiratory muscles. Although we have some voluntary control of ventilation, in the end this control is overridden by the involuntary control center (Wilmore & Costill, 1994).

Exercise Ventilation

The onset of exercise results in a dual phased increase in ventilation. The initial phase is characterized by an immediate rise in ventilation that is due to indirect stimulation of inspiratory centers by the motor cortex as it becomes more active during physical activity. No chemical stimulation of the respiratory centers occurs during this phase. In a sense, the nerve impulses traveling down the motor units of active muscles overflow within the brainstem and thereby stimulate the respiratory center (Wilmore & Costill, 1994). The second phase is characterized by a gradual rise in ventilation that results from elevated plasma CO_2, H^+, and temperature changes associated with prolonged exercise (Wilmore & Costill, 1994). Chemo- and thermo-receptors detect these changes and stimulate the inspiratory center to increase depth and rate of ventilation to an appropriate level (Wilmore & Costill, 1994).

Exercise induced changes in ventilation begin with an elevated V_T followed by an increase in V_f. The increase in V_f typically occurs when V_T equals 65% of vital capacity (VC). These changes in V_T require more forceful contractions of the diaphragm and external intercostals, the primary inspiratory muscles. At increasingly higher workloads, accessory inspiratory muscles are activated to maintain ventilatory homeostasis, such as the scalenes (anterior, middle, and posterior) and sternocleidomastoid, which work to

increase the volume and negative pressure with-in the alveoli, thereby increasing tidal volume (Wilmore & Costill, 1994). To sustain elevated V_f requires active expiration in contrast to the passive process that occurs during rest and low levels of physical activity. Expiratory muscles include the internal intercostals, latisimus dorsi, quadratus lumborum, and abdominals. Activity in these muscles expels air from the chest faster, which permits a greater V_f, and ultimately, rate of pulmonary ventilation (Wilmore & Costill, 1994).

Like all working muscles, the respiratory muscles (RM) need energy to perform their function. At rest, these muscles require about 2% of total body energy expenditure to maintain ventilatory homeostasis. As exercise intensity increases the rate of ventilation, the energy demands of the RM also increase. At maximal exercise, the cost of ventilation can reach 15% of total energy expenditure; it also demands a large percentage of energy, 9-12%, during recovery (Wilmore & Costill, 1994).

Pulmonary Adaptations

Research shows that endurance athletes have greater RM endurance than non-athletes (Eastwood, Hillman, & Finucane, 2001; Martin & Stager, 1981). Athletes, for instance, reach a higher percentage of their 12 s maximal voluntary ventilation (MVV) during a progressive breathing test then non-athletes. They can also sustain 80% of 12 s MVV for a significantly longer period of time than non-athletes. Based on these cross-sectional data, it appears that whole body endurance training induces changes in the RM that make them more resistant to fatigue (Coast, Clifford, Henrich, Stray-Gundersen, & Johnson, 1990). These RM adaptations include increased oxidative enzyme activity and capillary density (Grinton et al., 1992; Powers & Criswell, 1996). Endurance training also decreases the size of the muscle fibers, which further increases the relative concentration of the oxidative enzymes and muscle capillaries (Powers & Williams, 1987; Tamaki, 1987).

These RM adaptations may allow athletes to adopt a different breathing strategy than non-athletes, which may further aid RM performance. Athletes typically use a larger tidal volume and a lower breathing frequency than non-athletes at any given respiratory load (Eastwood et al., 2001). The shallow rapid breathing pattern used by non-athletes to increase ventilation is less efficient, which requires more RM work, and hence, earlier RM fatigue (Eastwood et al., 2001; Martin & Stager, 1981).

Respiratory Performance Limitations

In healthy to moderately fit individuals, the pulmonary system does not appear to limit exercise at sea level, as arterial oxygen content is relatively stable (i.e., 94-98%) even during maximal exercise (Dempsey, 1986). Indeed, the traditional view is that the lungs are ideally suited for the demands of exercise and consequently, do not limit performance in most individuals. In some elite athletes (VO_{2max} > 68 $ml \cdot kg^{-1} \cdot min^{-1}$) however, the pulmonary system may limit performance (Powers & Williams, 1987). This limitation may result from a mismatch in the relative adaptations among the pulmonary, cardiovascular, and metabolic systems to chronic endurance exercise. When compared to the magnitude of adaptation in the cardiovascular and metabolic systems, the changes in the RM are small (Dempsey, 1986). This limited RM adaptability or plasticity may eventually hinder athletic performance in some elite athletes by causing arterial O_2 desaturation or exercise induced hypoxemia during sub-maximal and maximal exercise (Powers & Williams, 1987).

Exercise-induced hypoxemia (EIH) is typically defined as a drop in arterial blood oxygen saturation levels below ninety-one percent, resulting in blood that is 10% less saturated with oxygen than normal. Individuals typically remain at or near a resting saturation level of 98%. Most of the time oxygen saturation levels remain within 2 to 3% of this resting level, even during maximal exercise. In some elite athletes, however, arterial oxygen saturation drops below 91% of normal levels (Dempsey, 1986; Powers,

Dodd, Woodyard, Beadle, & Church, 1984). EIH may affect 52% of elite athletes and occurs at work rates as low as 75% of VO_{2max} (Powers & Williams, 1987). The effects of EIH are significant. Ameliorating EIH by having athletes breathe gas with a higher concentration of oxygen than normal (a hyperoxic gas), for example, improves VO_{2max} during intense exercise (Powers & Williams, 1987). Although the exact mechanism for EIH is unknown, there are several possible factors that may contribute to the phenomenon, such as inadequate red blood cell transit time through the pulmonary capillaries, hypoventilation, mechanical and diffusion limitations at the capillary-alveolar interface, and competition for blood flow (Powers & Williams, 1987).

Inadequate red blood cell transit time through the pulmonary capillaries may contribute to EIH because it does not allow for full gas exchange between the pulmonary and circulatory systems. It has been reported that 0.35 s is the minimum time required for complete blood-gas equilibration between the two systems (Powers & Williams, 1987). Untrained subjects, even during maximal exercise, routinely show red blood cell transit times of 0.5 s, more than enough time for complete blood-gas equilibration (Powers & Williams, 1987). In many highly trained individuals exercising at maximal intensity, however, red blood cell transit time may decrease to approximately 0.4 to 0.3 s. These transit times are the result of high blood volumes and cardiac outputs maintained by highly trained endurance athletes. In short, high blood volumes and cardiac outputs may produce a pulmonary capillary blood flow that reaches the morphological limits of these capillaries to permit full equilibration with alveolar gas, thereby resulting in EIH (Powers & Williams, 1987).

Hypoventilation, or the inability to increase ventilation to the proper level, is also a potential cause of EIH. Hypoventilation during heavy exercise may limit the O_2 diffusion gradient within the alveoli by lowering alveolar PO_2 to insufficient levels (Dempsey et al., 1963). One possible explanation for the inadequate ventilation in some individuals is that their peripheral chemoreceptors lose the sensitivity needed to help the

respiratory center increase ventilation sufficiently to maintain blood gas homeostasis (Powers & Williams, 1987). This hypothesis is supported by the fact that these individuals do not increase maximal ventilation (V_{Emax}) during exercise while breathing hypercapnic (low CO_2) or hypoxic (low O_2) gases (Dempsey, 1986). Chemoreceptor response to intense exercise, however, is thought to play only a minor role in maintaining arterial O_2 tension or blood saturation (Powers & Williams, 1987).

A more likely explanation for hypoventilation is respiratory muscle fatigue. Respiratory muscle fatigue occurs after prolonged sub-maximal and maximal exercise in normal trained individuals (Fitting, 1991; Loke & Virgulto, 1982). This fatigue is inferred to occur when respiratory frequency increases and tidal volume decreases during the latter stages of steady-state prolonged endurance exercise (Gallagher, Hof, & Younes, 1985; Mador & Acevedo, 1991). In addition, inducing respiratory muscle fatigue prior to endurance exercise, decreases exercise time and increases tachypnic breathing (Mador & Acevedo, 1991). Highly trained athletes also exhibit signs of respiratory muscle fatigue following short bouts of intense exercise and long bouts of endurance exercise (>75% of VO_{2Max}) (Coast et al., 1990). One muscle particularly susceptible to fatigue is the diaphragm. Diaphragmatic fatigue, for example, occurs in normal subjects hyperventilating above 70% of MVV, ventilatory levels commonly seen during intense sub-maximal exercise performance (Fitting, 1991). Diaphragmatic fatigue has been measured directly via electromyography (EMG) during exercise produced via phrenic nerve stimulation (Johnson et al., 1996; Mador & Acevedo, 1991).

Another potential cause of EIH is that some individuals may reach the mechanical limits of their pulmonary system during intense exercise. One such mechanical limit involves airflow into and out of the chest. In highly fit humans, those with a $VO_{2max} > 70$ $ml \cdot kg^{-1} \cdot min^{-1}$ at exercise ventilations > 160 $l \cdot min^{-1}$, V_{Emax} increases when a Helium (He)-O_2 gas mixture is breathed during the exercise bout (Dempsey, 1986). A He-O_2 gas mixture is lighter than atmospheric gas and thereby increases the mechanical reserve of

the lung by making it easier to ventilate. As a result, the athlete can sustain greater breathing frequencies and tidal volumes, which forestall the onset of EIH (Dempsey, 1986).

Available diffusion surface area is another potential mechanical limitation of the lung. Diffusion surface area does not change with endurance training, whereas cardiac output and plasma volume do. An inability to increase the alveolar surface area relative to changes in the cardiovascular system may lead to insufficient O_2 exchange (Dempsey, 1986).

Inadequate ventilatory muscle blood flow and elevated perceptions of respiratory effort are other pulmonary related variables that may hinder exercise performance directly or indirectly by exacerbating EIH (Boutellier, 1998). Another possible mechanism whereby the pulmonary system may limit exercise performance involves competition between the pulmonary and skeletal muscle systems for blood flow during exercise. Blood flow distribution to the body's systems during exercise is proportional to the metabolic activity of the systems (Harms, 2000). During maximal cycle ergometry, 80-85% of cardiac output (CO) is sent to the active leg musculature. The heart, brain, skin, and organs other than the lungs need at least 7-10% of CO for normal functioning, which leaves 7 to 15% of CO for pulmonary blood flow. This amount of blood flow may not be sufficient to met the metabolic demands of the lungs during maximal cycle ergometry, which require 14-16% of total body VO_2 (Harms, 2000). Consequently, the pulmonary and skeletal muscle systems may compete for blood flow.

Effects of RMT on Respiratory Muscle Performance

Given that the pulmonary system can limit performance in trained individuals and fatigues in sedentary people during exercise, many researchers have examined the effects of RMT on pulmonary function. It is quite clear that RMT improves RM performance in both respiratory patients and healthy individuals, including trained athletes (Boutellier et

al., 1992; Fairbarn et al., 1991; Holm, Sattler, & Fergosi, 2004; Morgan et al., 1987; Romer et al., 2002a, 2002b; Sonetti et al., 2001; Spengler et al., 1999; Voliantis et al., 2001; Williams et al., 2002). In one of the first studies to examine the effects of RMT on RM function, eight sedentary subjects completed 5 wk of either maximal static inspiratory and expiratory maneuvers (45 min per day, 5 days per wk) against obstructed airways or normocapnic hyperpnea to exhaustion (Leith & Bradley, 1976). Respiratory muscle strength increased 55% in the subjects that completed the pulmonary resistance training, but vital capacity and total lung volume increased by only 4%. Experimental subjects training for endurance via normocapcnic hyperpnea improved maximal voluntary ventilation (MVV) by 14%, while breathing endurance, as defined by the percentage of MVV sustained for 15 min, increased from 81% to 96% of MVV (Leith & Bradley, 1976).

Aside from this early work with sedentary individuals, the majority of the RMT research has focused on its effects on athletes. Three wk of RMT, for example, increased 15s MVV_{15} by 14% and endurance breathing time (time at 100% of MVV_{15}) by 17-fold in 4 moderately trained cyclists (VO_{2max} = 50.7 $\pm$ 2 ml·kg^{-1}·min^{-1}) that trained once per day for 5 d·wk^{1} through a specially designed breathing apparatus designed to increase inspiratory resistance (Morgan et al., 1987). Simiarily, maximal sustainable ventilatory capacity (MSVC) increased significantly in 10 trained male subjects (VO_{2max} > 60 ml·kg^{-1}·min^{-1}) after 4 wk of isocapnic hyperpnea training (Fairbarn et al., 1991). Likewise, 4 wk of voluntary hyperpnea (85-160 l·min^{-1} for 30 min·d^{-1}, 5 d·wk^{-1}) increased RM endurance, defined as the length of time spent at 90% of MVV_{15}, from 6.1 min to 40 min in eight endurance trained (VO_{2max} = 4.4 $\pm$ 0.5 l·min^{-1}) subjects (Boutellier et al., 1992). Respiratory muscle endurance and maximal voluntary ventilation also improved significantly in 20 trained individuals (VO_{2max} = 61.5 $\pm$ 6.7 ml·kg^{-1}·min^{-1}) after 30 min of voluntary isocapnic hyperpnea 5 d·wk^{-1} for 4 wk (Spengler et al., 1999). Similarly, inspiratory muscle strength and endurance increased by about 30% in 10

trained subjects (VO_{2max} >55 mL·kg^{-1}·min^{-1}) who completed inspiratory muscle training (IMT) with a handheld threshold inspiratory muscle trainer 30 min·d^{-1}, 6 d·wk^{-1} for 10 wk (Inbar et al., 2000). There were no changes in these variables in the matched control group (n=10), who completed IMT with a sham device (Inbar et al., 2000).

In addition, 11 wk of RMT using a handheld pressure device significantly reduced inspiratory muscle fatigue after 6 min of maximal rowing performance in 14 highly trained (VO_{2max} = 3.56 ± 0.17 L·min^{-1}) female rowers (Volianitis et al., 2001). The RMT protocol also improved inspiratory muscle strength 45.3 ± 29.7% compared to the 5.3 ± 9.8% change across time in the control group. Similarly, RM strength and breathing endurance improved by 31% and 128%, respectively, in eight trained collegiate runners (VO_{2max} = 59.9 ± 11.7 ml·kg^{-1}·min^{-1}) after 4 wk of RMT, which was completed 25 min·d^{-1}, 4-5d·wk^{-1} with a handheld Threshold Breathing device that was set at >80% of individual maximal inspiratory mouth pressure (Williams et al., 2002). Last, RM strength was significantly improved by 49% in 16 male cyclists (VO_{2max} = 64 ± 2 ml·kg^{-1}·min^{-1}) after 6 wk of RMT that involved 60 dynamic inspiratory efforts every day with a handheld pressure-threshold device (Romer et al., 2002a, 2002b).

In short, RMT improves RM performance across a spectrum of individuals, from sedentary people to highly trained athletes. In most of these studies, moreover, the training focused on enhancing the functionality of the inspiratory muscles. Since the expiratory muscles also aid ventilation, particularly during high intensity exercise, perhaps training them in addition to the inspiratory muscles will improve RM performance more than only inspiratory muscle training. Irrespective of its effects on RM performance, the more germane question to an athlete is whether or not RMT improves exercise performance.

Does RMT Enhance Exercise Performance?

The majority of the literature shows that RMT improves RM and exercise performance (Amonette & Dupler, 2001; Boutellier, 1998; Boutellier et al., 1992; Holm

et al., 2004; Romer et al., 2002a, 2002b; Spengler et al., 1999; Volianitis et al., 2001). Four weeks of voluntary hyperpnea (85-160 $l \cdot min^{-1}$ for 30 $min \cdot d^{-1}$ and 5 $d \cdot wk^{-1}$), for example, increased RM endurance and cycling performance at the anaerobic threshold (AT) by 550% and 38%, respectively, in 8 endurance trained subjects (Boutellier et al., 1992). The training regimen did not, however, improve VO_2max or the AT. In another study, 30 min of voluntary isocapnic hyperpnea, completed 5 $d \cdot wk^{-1}$ for 4 wk, improved respiratory muscle endurance, MVV, and cycling time to exhaustion at 85% of maximum mechanical power output (W_{max}) by 532%, 19%, and 27%, respectively, in 20 trained subjects (VO_{2max} = 61.5 $\pm$ 6.7 $ml \cdot kg^{-1} \cdot min^{-1}$) (Spengler et al., 1999). End-test blood lactate levels (Hla) were also lower for all cycling tests in this study. This training program did not, however, alter VO_{2max} or W_{max}. Collectively, these studies clearly show that RMT that involves voluntary isocapnic hyperpnea improves exercise performance in trained individuals.

Respiratory muscle training via handheld pressure devices also improves RM and exercise performance. Eleven wk of RM resistance training at a load equivalent to 50% of peak inspiratory mouth pressure performed 30 times twice daily, for example, significantly improved 6 min maximal and 5000 m ergometer rowing performance by 3.5 $\pm$ 1.2 and 3.1 $\pm$ 0.8%, respectively, in seven highly trained (VO_{2max} = 3.56 $\pm$ 0.17 $l \cdot min^{-1}$) female rowers (Volianitis et al., 2001). In contrast, 6 min maximal and 5000 m ergometer rowing time only improved by 1.6 $\pm$ 1.0 and 0.9 $\pm$ 0.6%, respectively, in seven matched controls. The RMT program also reduced respiratory muscle fatigue following maximal exercise and improved inspiratory muscle strength by 45.3 $\pm$ 29.7% in the experimental group compared to a 5.3 $\pm$ 9.8% increase in the controls, as previously mentioned. Similarly, a 6 wk RMT program that involved 30 dynamic inspiratory efforts completed $2 \cdot d^{-1}$ with a handheld pressure device set at a load equal to approximately 50% of each individual's maximal inspiratory pressure improved simulated 20 and 40 km TT performance by 66 $\pm$ 30 s and 115 $\pm$ 38 s in 16 highly trained cyclists (VO_{2max} = 64 $\pm$ 2

$ml \cdot kg^{-1} \cdot min^{-1}$) (Romer et al., 2002a). The RMT also reduced perceived respiratory and peripheral exertion by 16 ± 4% and 18 ± 4%, respectively, during an incremental VO_{2max} test (Romer et al., 2002a).

In contrast to these positive findings, other RMT programs have not improved exercise performance despite increased RM functionality (Fairbarn et al., 1991; Hanel & Secher, 1991; Inbar et al., 2000; Morgan et al., 1987; Sonetti et al., 2001; Williams et al., 2002). Three weeks of voluntary isocapnic hyperpnea, for example, did not improve cycling endurance time at 95% of VO_{2max} in three trained subjects despite increasing RM endurance 17 fold (Morgan et al., 1987). Likewise, 25 sessions of combined inspiratory muscle resistance strength (3-5 min per session) and hyperpnea endurance training (30 min per session) over 5 wk did not alter 8 km TT performance in nine highly trained (VO_{2max} >60 $ml \cdot kg^{-1} \cdot min^{-1}$) cyclists despite improving RM strength and endurance by 64% and 19%, respectively (Sonetti et al., 2001). Similarly, 4 wk of inspiratory muscle resistance strength training did not alter VO_{2max} or endurance capacity at 85% of VO_{2max} in 8 trained (VO_{2max} >59.9 $ml \cdot kg^{-1} \cdot min^{-1}$) distance runners despite significant improvements in all RM performance tests (Williams et al., 2002). Further, 4 wk of isocapnic hyperpnea training did improve performance in 10 trained male subjects (VO_{2max} > 60 $ml \cdot kg^{-1} \cdot min^{-1}$) despite increasing MSVC (Fairbarn et al., 1991). Last, 10 wk of threshold inspiratory muscle training that was completed 30 $min \cdot d^{-1}$, 6 $d \cdot wk^{-1}$ with a handheld device did not increase aerobic capacity in 10 trained subjects (VO_{2max} >55 $mL \cdot kg^{-1} \cdot min^{-1}$), although it enhanced pulmonary function (Inbar et al., 2000).

At first glance it is difficult to reconcile the different exercise performance outcomes among the studies. Closer examination reveals that the primary difference among the studies that could account for the divergent outcomes was methodology. One methodological difference is training program duration. Most programs lasting ≥4 wk improved endurance exercise performance (Boutellier, 1998; Boutellier et al., 1992; Holm et al., 2004; Romer et al., 2002a, 2002b; Spengler et al., 1999; Volianitis et al.,

2001;), whereas shorter training protocols (< 4 wk) did not (Fairbarn et al., 1991; Hanel & Secher, 1991; Morgan et al., 1987; Sonetti et al., 2001; Williams et al., 2002).

Another important methodological difference between those studies that showed improved performance and those that did not is the type of tests used to assess changes in performance across time. Those studies that used open-ended or volitional time-to-fatigue tests, in which subject's exercise at a certain percentage of VO_{2max} for as long as possible, frequently found no improvement in performance (Fairbarn et al., 1991; Morgan et al., 1987; Sonetti et al., 2001; Williams et al., 2002). In contrast, those studies that assessed performance with closed-end tests in which the amount of work is pre-determined, frequently found that RMT improved performance (Holm et al., 2004; Romer et al., 2002a; Volianitis et al., 2001). Closed-ended tests are preferable to open-ended tests because they are less subjective, more reliable, and better simulate actual competitive performance (Jeukendrup et al., 1996).

The intensity of the performance tests also may have affected the outcomes. Many of the aforementioned studies that showed that RMT did not improve exercise performance used very intense, short-duration testing protocols that were ≥ 85 of VO_{2max} (Fairbarn et al., 1991; Hanel & Secher, 1991; Inbar et al., 2000; Morgan et al., 1987). In contrast, those studies that found that RMT improved exercise performance used longer duration test at < 85% of VO_{2max} (Holm et al, 2004; Boutellier, 1998; Boutellier et al., 1992; Romer et al., 2002a; Spengler et al., 1999; Volianitis et al., 2001).

The evidence of respiratory muscle fatigue following intense and prolonged exercise is growing, as is the body of literature showing the RMT improves RM functionality. Although the data on the effects of RMT on exercise are equivocal, it is clear that the methodological differences among the studies may account for the divergent findings. Nevertheless, data show that RMT improves intense sub-maximal (< 85% of VO_{2max}) exercise performance among trained athletes (Fairbarn et al., 1991; Hanel & Secher, 1991; Morgan et al., 1987; Sonetti et al., 2001; Williams et al., 2002).

The exact mechanism for the change is not clear, but since RMT improves respiratory muscle function, it is logical to assume that the better trained respiratory muscles are working at lower relative load for a given work rate after RMT. Consequently, RMT may reduce RM fatigue, while also decreasing the shunting of blood flow from the locomotor muscles and renal-splanchnic region to maintain ventilation. Collectively, these changes may reduce ventilatory and locomotor lactate acid production, while increasing lactate clearance. These changes also may lead to reduced peripheral and ventilatory perceived exertion, which may enhance exercise performance psychologically, by allowing the athlete to maintain focus on important aspects of the task.

Chapter 3

METHODS

Subjects

Ten trained cyclists (VO_{2max}= 4.39 ± 0.59 l·min^{-1}) volunteered from the local cycling community. The Mean ± *SD* for age, height, weight, and maximal power output (PO_{max}) were 27 ± 5.7 y, 182 ± 5.3 cm, 76.3 ± 8.9 kg, and 288 ± 30.8 W, respectively. Prior to testing, all subjects were fully briefed on the nature of the experiment and signed an informed consent form that was approved by the college's review board for research on human subjects (Appendix B). Data collection took place in late April through early May, which typically marks the end of the first major phase of preparation for a cyclist's competitive season.

Experimental Outline

Subjects reported to the testing lab a total of four times over the course of the study. In the initial visit, pulmonary function, sub-maximal, and VO_{2max} tests were completed. Within one week, subjects returned to complete a 20 km time-trial. Following the second lab visit, subjects were randomly assigned to either an experimental or control group. The experimental group trained with the PowerLung® (Houston, TX), a device that exercises both the inspiratory and expiratory muscles concurrently, while the control group trained with a sham version of the device. After 6 weeks of respiratory muscle training, subjects returned to the lab for two more visits, repeating the aforementioned protocol. Subjects were instructed to refrain from alcoholic or caffeinated beverages for at least 24 hours before each lab test and to maintain current training volume throughout the study. To assess compliance with pre-testing instructions, subjects completed a 24-hour history prior to each lab visit. All lab testing was conducted at 19-22°C and a constant relative humidity. For each maximal, sub-maximal, and TT test, subjects used their own bike attached to the Computrainer™ Pro 8001 (Seattle, WA) resistance unit, which provided resistance and recorded work and power in watts (W). All subjects were given a training journal and instructed to log

training activity (both cycling and respiratory) so that experimental protocol adherence could be monitored.

Experimental Procedures

In the subject's initial visit to the lab, they first completed respiratory muscle tests, including maximal expiratory (PE_{max}) and inspiratory (PI_{max}) pressure tests using a Micro Medical MPM hand held pressure meter (Micro Medical Mouth Pressure Meter, Rochester, England), which digitally displays mouth pressure in cmH_20. From the standing position, subjects blew into the pressure meter via a disposable mouthpiece connected to a one-way valve that could be switched for inspiratory or expiratory maneuvers. The PE_{max} test began at total lung capacity followed by a maximal exhalation, whereas the PI_{max} test started from maximal voluntary exhalation followed by a maximal inspiration. Subjects performed each test three times and the highest value was recorded. These tests reflect respiratory muscle strength.

After the strength tests, respiratory muscle endurance (RME) was assessed with the PowerLung®. The device contains three ports: one that subjects breathe through and two others with adjustable apertures, used to alter inspiratory and expiratory resistance. The inspiratory aperture has six settings, while the expiratory aperture has three settings. Number one, the first setting, is the easiest whereas the last respective setting is the hardest. Subjects performed RME test in a seated position and they were required to breathe through a disposable mouthpiece into the PowerLung® forcefully for 3-4 s on each inhalation and exhalation. Subjects continued until they were no longer able to maintain adequate ventilation and had to remove the mouthpiece to take a breath. Respiratory muscle load was set at 1.5 and 2 for the inspiratory and expiratory resistance settings, respectively.

The final respiratory measurement was a 15 s maximal voluntary ventilation test (MVV_{15}) using a CDX Spiro 110 plus automated spirometer (Aurora, CO). From the standing position, subjects breathed as deeply and forcefully as possible though a mouthpiece connected to the spirometer, subjects were coached throughout the test to alter breathing pattern to maximize MVV_{15} volume. Subjects completed three trials (one

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practice and two experimental trials). The highest score of the experimental trials was MVV_{15} ($l \cdot min^{-1}$).

After the pulmonary function testing, subjects performed a 10 min sub-maximal cycling test (Sub_{10}). Subjects warmed up at 150 W for 10 min prior to starting the test, and subsequently maintained a workload of 300 W for 10 min. Blood lactate was measured via a YSI 1500 Sport Lactate Analyzer (Yellow Springs, OH) from 25µl blood samples drawn from a fingertip at three intervals (min 0, 5, and 10). Heart rate (HR_{Sub}) via telemetry (Polar, Lake Success, NY), rating of perceived exertion (RPE_{Sub}) via Borg scale (6-20), and rating of ventilatory exertion (Sub_{RVE}) via modified Borg scale (1-10) were measured every three minutes. Oxygen consumption (Sub_{VO_2}), expired ventilation (V_{ESub}), tidal volume (VT_{Sub}), and respiratory rate (RR_{Sub}) were recorded continuously with a ParvoMedics TrueMax 2400 (Salt Lake City, UT) metabolic cart and averaged for 15sec. for later analysis.

After Sub_{10}, subjects recovered at 150 W for 10 min before completing a maximal graded exercise test (VO_{2max}). The test began with an initial workload of 200 W, which was increased 40 W every 2 min until volitional exhaustion or subjects could no longer maintain a cadence of at least 60 rpm. HR, V_E, and VO_2 were measured continuously as previously described. Following each stage, RPE and RVE were recorded as previously described. To assess blood lactate, blood samples were taken prior to and immediately following the test, as previously mentioned.

Within five days of the first test, subjects returned to the lab for a 20 km time-trial test (TT), which was preceded by a 10 min warm-up at 150 watts. Heart rate and PO were measured continuously, while V_E, VO_2, RPE, RVE, and blood lactate were measured every 4 km as previously described.

After initial testing, subjects were randomly assigned to either the experimental (EG) or control groups (CG). To ensure naivety, subjects were blinded to the study intent and believed they were completing strength (EG) or endurance (RM) training. A debriefing statement was read to each control subject upon completion of the study to inform him of this deception (Appendix B). EG completed 30 (3 x 10 repetitions) breaths

twice daily, once in the AM and once in the PM, using the PowerLung® at a resistance level ≥ 60% of their peak inspiratory mouth pressure. CG completed 30 (1 x 30 repetitions) breaths once a day at a resistance ≤15% of their peak inspiratory mouth pressure, a level shown to produce negligible training effects (Caine & McConnell, 2000). Both groups trained daily for 6 wk. EG subjects were instructed to periodically increase the load 1/8 to 1/4 of a turn on the PowerLung® whenever all repetitions at the assigned load could be easily completed to ensure a progressive overload of the respiratory muscles. If subjects increased the load but could not complete 3 x 10 repetitions they returned to the previous setting and trained for another week. After 6 wk of training, subjects returned to the lab to repeat all tests. Subjects were instructed to stop RMT at least 48 hours before returning to the lab.

A 2 x 2 ANOVA (Group x Time) with repeated measures on the 2nd factor was completed for all variables. The first (between subjects) factor, Group, consisted of the training group (EG) and control group (CG). The 2nd factor, Time, represents the pre- and post-training variables both before and after 6 wk of respiratory muscle training. For all statistical tests alpha was set at 0.05. A Pairwise Comparisons post hoc test was used to determine differences for Group and Time.

Chapter 4

RESULTS

Initial group and selected performance data are shown in Table 1. Despite random group assignment, the experimental group (EG) was significantly larger ($F_{(1,8)}$ = 5.6; p=0.05) in mass than CG. Irrespective of this initial difference in mass, pre-training relative VO_{2max} scores were similar between groups.

Table 1.
Initial descriptive characteristics (Mean ± *SD*) of experimental (EG) and control (CG) groups.

	CG (n = 5)		EG (n = 5)	
Anthropometry				
Age (y)	26.2	(7.6)	27.8	(3.6)
Height (cm)	180.0	(7.4)	183.4	(2.1)
Mass (kg)	70.5	(4.1)*	82.1	(8.7)*
Maximal Incremental Exercise				
PO_{max} (W)	278.2	(28.4)	297.8	(32.9)
VO_{2max} ($l \cdot min^{-1}$)	4.2	(0.65)	4.6	(0.49)
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	58.9	(6.4)	56.6	(8.2)

Note. PO_{max}: maximal power output; VO_{2max}: maximal oxygen uptake. * = significant difference between groups ($p < 0.05$).

Table 2 shows the changes in mass, VO_{2max}, and PO_{max} across time. Body mass decreased significantly in EG by 2.5% over the 6 wk training period ($F_{(1,8)}$ = 14.9; p=0.01). As a result of this drop in body mass, EG was no longer significantly larger than CG after training.

Table 2.
Changes in descriptive characteristics (Mean ± *SD*) pre- to post- respiratory muscle training.

	CG (n = 5)		EG (n = 5)	
	Pre	Post	Pre	Post
Mass (kg)	70.5 (4.1)*	71.3 (4.7)	82.1 (8.7)*	80.0 (8.4)#
VO_{2max} ($l \cdot min^{-1}$)	4.2 (0.65)	4.1 (0.58)	4.6 (0.49)	4.6 (0.4)
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	58.9 (6.4)	57.6 (5.1)	56.6 (8.2)	58.0 (6.2)
PO_{max} (W)	278.2 (28.4)	284.8 (22.9)	297.8 (32.9)	303.2 (13.1)

Note. VO_{2max}: maximal oxygen uptake; PO_{max}: maximal power output. * = significant difference between groups ($p<0.05$) # =significant difference across time ($p<0.05$).

Changes in respiratory muscle function pre- to post-training are shown in Table 3. Inspiratory muscle strength (PI_{max}) increased by 27.6% across time in EG ($F_{(1,8)} = 17.9$; $p=0.00$), as shown in Table 3. There was also a significant interaction between groups for PI_{max} ($F_{(1,8)} = 7.48$; $p=0.03$) There were no other differences between groups or across time for any other RM function variable

Table 3.
Changes in respiratory muscle function (Mean ± *SD*) pre- to post- respiratory muscle training.

	CG (n = 5)		EG (n = 5)	
	Pre	Post	Pre	Post
RME (s)	51.4 (18.6)	89.4 (56.9)	100.4 (81.4)	232.2 (92.9)
PI_{max} (cm H_20)	96.3 (25.3)	102.6 (34.3)	106.3 (20.3)	135.7 (8.1)#
PE_{max} (cm H_20)	141.4 (46.2)	157.9 (32.8)	185.2 (44.2)	188.3 (49.9)
MVV_{15} ($l \cdot min^{-1}$)	169.4 (22.4)	171.0 (36.5)	196.5 (13.0)	198.3 (14.8)

Note. RME: respiratory muscle endurance; PI_{max}: inspiratory muscle strength; PE_{max}: expiratory muscle strength; MVV_{15}: maximal voluntary ventilation. # = significant difference across time ($p<0.05$).

Tables 4 and 5 show sub-maximal and TT performance data pre- and post- RMT. There were no significant differences in any sub-maximal variable across time or between groups. In contrast, time-trial rating of perceived exertion (RPE_{TT}) decreased

9.7% across time in EG ($F_{(1,8)} = 8.96$; $p = 0.02$). There was, however, no difference between the groups after training. There were no other differences between groups or across time in any other TT performance variable.

Training log data showed that endurance training remained constant throughout the 6 wk study and that both groups strictly adhered to their lung training regimens. Entries showed that EG and CG completed 72.8 ± 9.4 of 84 and 34.6 ± 8.4 of 45 training sessions, respectively, yielding compliance rates of 86% and 82%.

ANOVA summary tables can be found in Appendix E.

Table 4.
Changes in sub-maximal exercise performance variables (Mean ± *SD*) pre- to post-respiratory muscle training.

	CG (n = 5)		EG (n = 5)	
	Pre	Post	Pre	Post
Hla_{Sub} (mM)	8.9 (3.1)	8.2 (2.8)	6.1 (3.9)	4.8 (2.3)
HR_{Sub} (bpm)	172.0 (5.6)	176.0 (6.0)	64.2 (13.0)	161.0 (10.0)
RVE_{Sub}	3.7 (0.89)	4.6 (1.8)	3.6 (2.5)	2.4 (0.77)
RPE_{Sub}	14.1 (1.3)	14.9 (1.3)	14.1 (2.2)	12.8 (1.2)
PO_{Sub}	289.4 (16.8)	288.6 (14.8)	293.6 (13.2)	299.6 (0.9)
VO_{2sub} ($l \cdot min^{-1}$)	3.7 (0.35)	3.6 (0.36)	3.8 (32.0)	3.9 (0.18)
VO_{2sub} ($ml \cdot kg^{-1} \cdot min^{-1}$)	51.9 (2.26)	50.0 (2.2)	47.1 (6.5)	48.8 (5.4)

Note. Hla_{Sub}: sub-maximal lactate values; HR_{Sub}: sub-maximal heart rate; RVE_{Sub}: sub-maximal rating of ventilatory exertion; RPE_{Sub}: sub-maximal rating of perceived exertion; VO_{2sub}: sub-maximal oxygen consumption.

Table 5.
Changes in time-trial exercise performance variables (Mean ± *SD*) pre- to post-respiratory muscle training.

	CG (n = 5)		EG (n = 5)	
	Pre	Post	Pre	Post
PO_{TT} (W)	256.8 (19.3)	255.6 (20.0)	289.0 (38.4)	298.0 (23.0)
RVE_{TT} (1-10)	4.4 (0.80)	5.3 (1.5)	4.4 (1.5)	4.8 (1.1)
RPE_{TT} (6-20)	15.3 (0.87)	15.7 (1.1)	16.4 (0.86)	14.8 (1.6) #
TV_{TT} ($l \cdot breath^{-1}$)	2.3 (0.30)	2.3 (0.29)	2.9 (0.41)	3.0 (0.40)
TM_{TT} (s)	1982.0 (49.0)	1985.0 (47.0)	1923.0 (100.0)	1897.0 (52.0)
AVS_{TT} (kph)	36.7 (0.97)	36.7 (0.97)	38.0 (1.9)	38.3 (1.1)
VO_{2TT} ($l \cdot min^{-1}$)	3.5 (0.38)	3.5 (0.35)	4.1 (0.58)	4.0 (0.34)
HR_{TT} (bpm)	175.0 (8.0)	174.0 (10.0)	177.0 (3.0)	174.0 (7.0)
LA_{TT} (mM)	8.8 (2.0)	7.9 (2.4)	9.4 (2.6)	8.8 (3.1)

Note. PO_{TT}: TT power output; RVE_{TT}: TT rating of ventilatory exertion; RPE_{TT}: TT rating of perceived exertion, TV_{TT}: TT tidal volume; TM_{TT}: TT time; AVS_{TT}: TT average speed; VO_{2TT}: TT oxygen uptake; HR_{TT}: TT heart rate, Hla_{TT}: TT lactate values. # = significant across time ($p<0.05$)

Chapter 5

DISCUSSION

It is well established that respiratory muscle training (RMT) improves respiratory muscle (RM) performance (Amonette & Dupler, 2001; Boutellier 1998; Boutellier et al., 1992; Fairbarn et al., 1991; Hanel & Secher, 1991; Holm et al., 2004; Inbar et al., 2000; Morgan et al., 1987; Romer et al., 2002a, 2002b; Spengler et al., 1999; Volianitis et al., 2001). In most of these studies, however, subjects trained their inspiratory musculature. Since both inspiratory and expiratory muscles are highly active during intense exercise, it is possible that concurrent inspiratory and expiratory muscle training may improve respiratory muscle performance more than inspiratory muscle training (IMT) alone. Therefore, the primary purpose of this study was to determine the effects of CRMT on respiratory muscle performance. The secondary purpose was to determine if the putative training effects of concurrent inspiratory and expiratory muscle training would enhance maximal, sub-maximal, and simulated 20 km time-trial cycling performance.

As shown in Figure 1, 6 wk of CRMT, as performed in this study, improved inspiratory muscle strength (PI_{max}) by 27.6% ($p=0.03$) across time in the experimental group (EG). This finding concurs with the literature, which shows that RMT improves PI_{max} by 25-45.3% (Hanel & Secher, 1991; Inbar et al., 2000; Romer et al., 2002a; Volianitis et al., 2001). However, in contrast to the changes in PI_{max}, CRMT did not change expiratory muscle strength (PE_{max}). Since the effects of CRMT on PE_{max} have not been tested previously, a comparison with the literature is not possible. PE_{max} was expected to improve but it is possible that the expiratory muscles are sufficiently trained already by endurance cycling and activities of daily living; therefore, additional expiratory muscle training may not be necessary. Consistent with other pressure threshold training studies, CRMT did not elicit an increase in 15 s maximum voluntary ventilation performance (MVV_{15}) (Romer et al., 2002a). Collectively, CRMT significantly increased PI_{max}, but it did not alter PE_{max} or MVV_{15}. Given the similarity in findings between CRMT and inspiratory muscle training, adding expiratory muscle

training appears to be superfluous for improving the aforementioned variables. Therefore, respiratory muscle training protocols, as previously reported, do not require expiratory muscle training to be effective (Amonette & Dupler, 2001; Boutellier, 1998; Boutellier et al., 1992; Fairbarn et al., 1991; Hanel & Secher, 1991; Holm et al., 2004; Inbar et al., 2000; Morgan et al., 1987; Romer et al., 2002a, 2002b; Spengler et al., 1999; Volianitis et al., 2001).

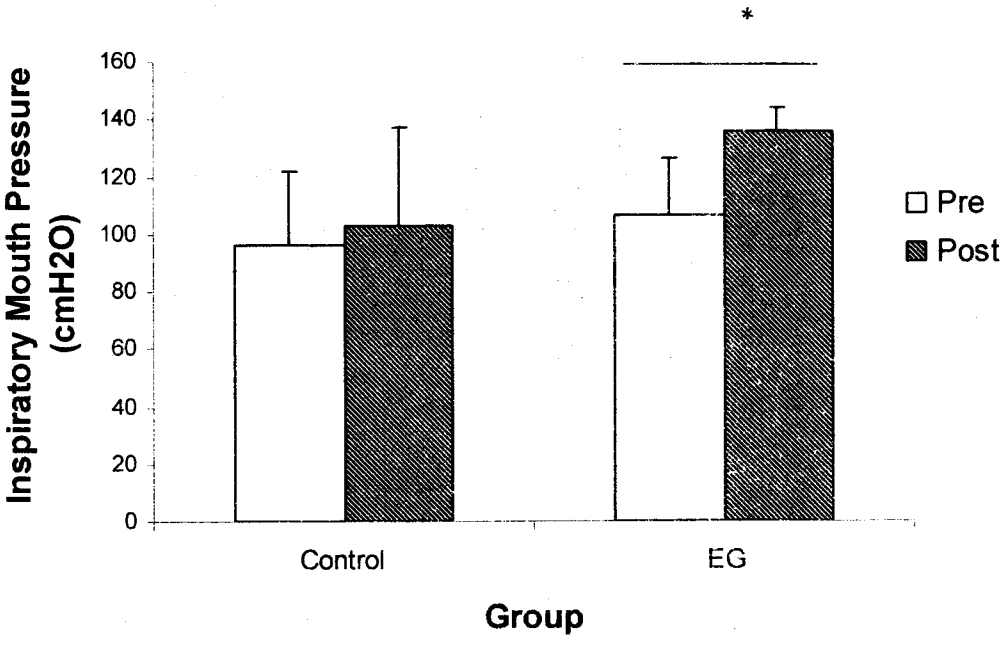


Figure 1. Inspiratory mouth pressure (Mean ± *SD*) pre- to post- respiratory muscle training. Control (n = 5), EG (n = 5). * = significantly different across time ($p<0.05$).

Respiratory muscle training has been shown to increase respiratory muscle endurance (RME) 12-128% across time (Fairbarn et al., 1991; Inbar et al., 2000; Morgan et al., 1987; Romer et al., 2002a, 2002b; Sonetti et al., 2001; Volianitis et al., 2001; Williams et al., 2002). As shown in Figure 2, CRMT increased RME by 132% in EG; RME in EG was also 159% greater than CG post-training.

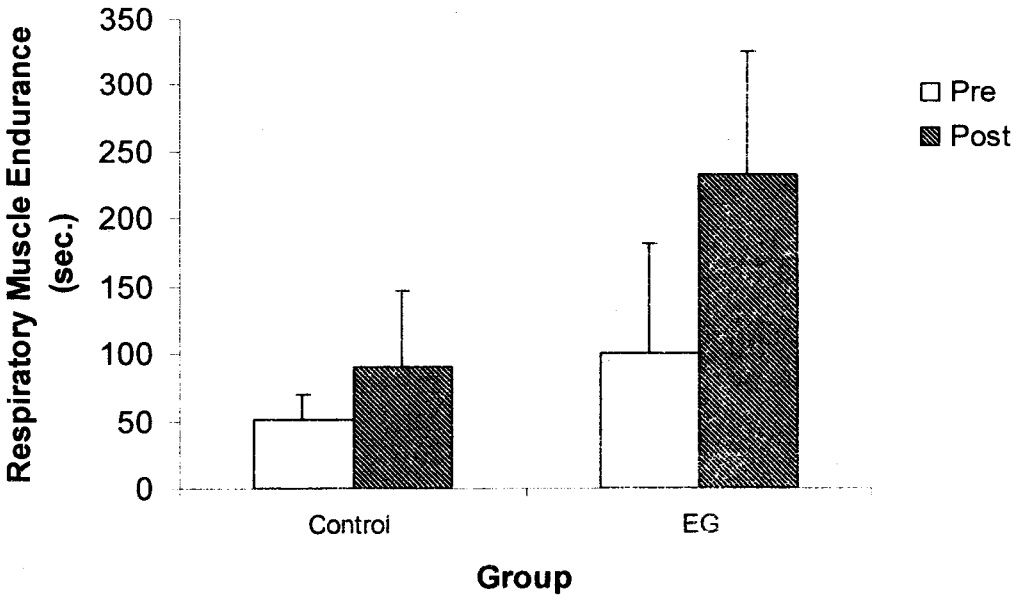


Figure 2. Respiratory muscle endurance (Mean ± *SD*) pre- to post- respiratory muscle training. Control (n = 5), EG (n = 5).

Irrespective of these large improvements in RME, the changes were not statistically significant. One reason for the lack of statistical significance was methodological error. There was an *a priori* testing limit of 300 s for RME, for example, which prevented three of the five EG subjects from reaching true fatigue during post tests. Note in Figure 3 that all EG subjects reached at least 120 s after CRMT, whereas only one of the CG subjects reached this limit. Other reasons for the lack of statistical significance were the small sample size and large variances. A power analysis revealed that the addition of 5 more subjects would have resulted in a significant difference in RME across time and between groups.

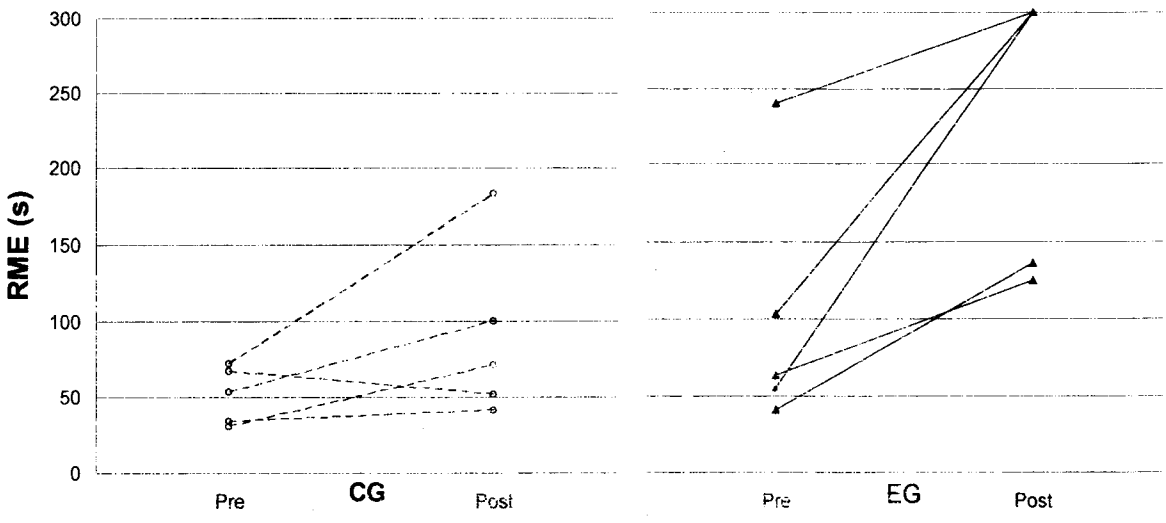


Figure 3. Individual respiratory muscle endurance results for each subject pre- to post-respiratory muscle training. Control (n = 5), EG (n = 5).

RMT has not been shown to improve maximal exercise performance. The effects of CRMT on performance are not known. In this study, however, CRMT did not alter VO_{2max} or maximal power output. These data are consistent with the literature and support the proposition that the respiratory system sufficiently maintains gas homeostasis during maximal exercise, and hence, does not limit maximal performance.

To assess changes in sub-maximal exercise performance, VO_2, HR, RPE, and blood lactate concentration (Hla) were assessed during a 10 min cycling test at an absolute workload of 300 W. This workload was assumed to be equivalent to 75-80% of VO_{2max} and 5-10% below the workload that could be sustained during the 20 km TT. However, the sub-max data showed that CG and EG exercised at 88 and 83% of VO_{2max}, respectively, which indicates that the sub-max workload was too high. In CG, moreover, sub-maximal power output (PO_{Sub}) and VO_{2Sub} values, which should have been lower than corresponding TT values, were actually 14.4% and 4.6% higher. Furthermore, blood lactate levels (Hla_{Sub}), which should have been lower than TT values (Hla_{TT}), were similar in both venues. Even though EG VO_{2Sub}, PO_{Sub}, Hla_{Sub} values were 2.8%, 3.6%, and 3.9% lower, respectively, than the corresponding TT measurements, they were still

higher than expected. Clearly, the sub-maximal workload was too hard for both groups, a hypothesis further substantiated by the fact that three out of the ten subjects could not maintain 300 W for 10 minutes.

The primary factor that accounted for the unexpected sub-maximal testing data was that the subjects were not as fit as expected. Although all subjects met the study's pre-testing VO_{2max} requirements, mean peak power output (PO_{max}) was only 288 W. Consequently, a 300 W absolute sub-maximal workload represented too high a percentage of PO_{max} to be properly considered a sub-maximal for most subjects. In hindsight, it was a mistake to assign an absolute sub-max workload for all subjects. Instead, a relative workload based on each subject's PO_{max} should have been assigned.

Although statistically insignificant, it is notable that EG sub-maximal blood lactate concentration (Hla_{Sub}) was 21% lower across time (6.09 ± 3.9 to 4.82 ± 2.3) compared to 7.4% in CG (8.86 ± 3.1 to 8.20 ± 2.8); it was also 41% lower than corresponding CG values after RMT. A plot of subject time-trial lactate levels pre- to post-training is shown in Figure 4. These data, like many other data in this study, were not significantly different because of the study's small sample size and the large standard deviations measured for each variable. However, the magnitude and direction of these Hla_{Sub} trends concur with data from other studies, and had statistical power been better with this study a significant difference may have been demonstrated (Boutellier et al., 1992; Kohl, Koller, Brandenberger, Cardenas, & Boutellier, 1997; Romer et al., 2002a; Spengler et al., 1999). Data from these studies provide evidence that RMT reduces plasma lactate levels during absolute sub-maximal exercise, perhaps by altering local blood flow dynamics. Theoretically, CRMT should lower the relative intensity of RM work at any pre-training absolute workload, thereby decreasing metabolic by-product formation within these muscles and the associated RM vasodilation. As a consequence, less blood would be shunted away from the locomotor muscles at each pre-training absolute workload, thereby allowing more oxidative work by these muscles (Harms, 2000). These changes should result in lower lactate levels at each sub-maximal

workload; they may also lead to improved exercise performance by increasing the amount of locomotor muscle oxidative work.

In addition to max and sub-max tests, subjects completed a simulated 20 km time-trial test (TT). A simulated TT is a close-ended test in which subjects compete to finish a predetermined quantity of work; such tests are highly reliable (1-4%) and reproducible (Jeukendrup et al., 1996). Simulated TT tests are also sensitive to changes in endurance performance and correlate well with actual TT performance in the field (Coyle et al., 1991). Time-trial performance was unchanged following 6 wk of CRMT, although the rating of perceived exertion during the TT (RPE_{TT}) was reduced in EG across time ($p=0.02$), as shown in Figure 5.

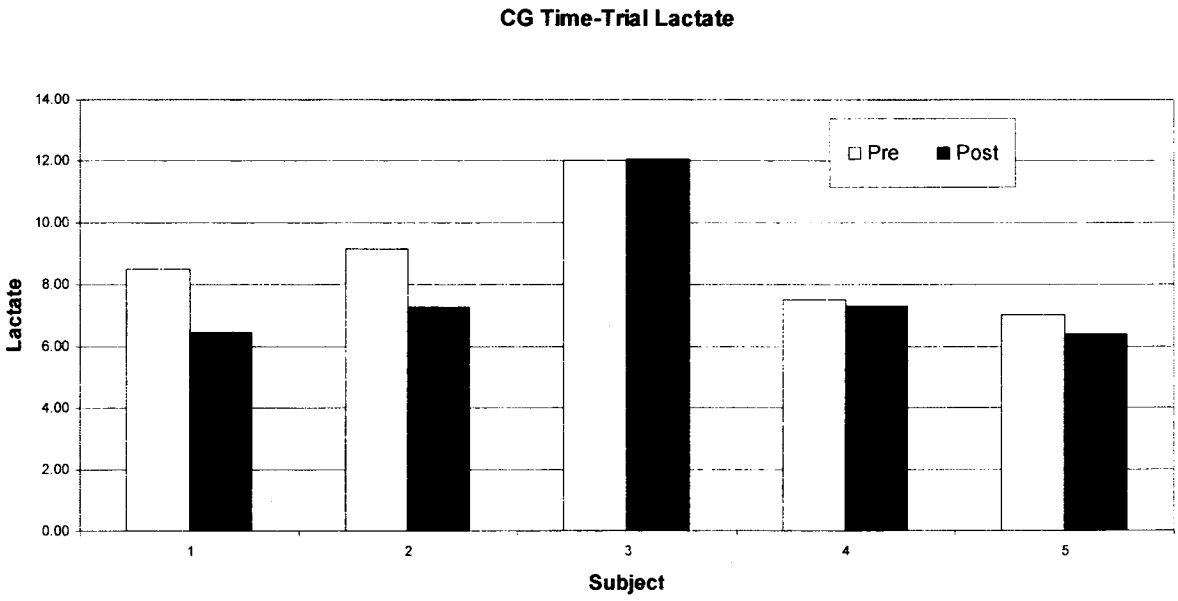
These data indicate that the EG subjects thought that the post-training TT required less whole body effort than the pre-training TT. Since RPE is an index of whole body effort it reflects a combination of muscular and respiratory effort. Given that there was no change across time or between groups in the rating of perceived ventilatory exertion (RVE) in this study, the drop in RPE across time in EG may mean that the subjects in this group perceived a decrease in leg muscle effort during the post-training TT. This reduced leg effort may have resulted from increased leg blood flow, a putative consequence of CRMT. Measures of RPE during sub-maximal tests did not reach statistical significance, but were notable ($p = 0.062$). Perhaps CRMT has the most potential influence on the psychological variables of intense exercise. If so, it may improve performance by decreasing an athletes attention placed on feelings of discomfort, thereby allowing increased focus on relevant performance tasks.

Despite the change in RPE_{TT}, TT performance did not significantly improve in EG, although their TT time decreased by 26 s or 1.3%. This statistically insignificant change is, however, similar with the data from other studies that show RMT improves simulated time-trial performance by as much as 3.8% (Holm et al., 2004; Romer et al., 2002a; Voliantitis et al., 2001). Typical winning margins in national class time-trial events is less than 3%, therefore the 1.3% improvement found in this study may be biologically advantageous for an athlete. As with the other variables, the lack of

significance in TT performance was due to the small sample size and large variability in the data.

One shortcoming of this study is the lack of training observation. To determine if changes in exercise performance following a bout of RMT are in fact the result of adaptations gained via RMT, training volume and intensity throughout the study needs to be strictly controlled. None of the studies that show that RMT improves exercise performance strictly controlled exercise training volume and intensity (Holm et al., 2004; Romer et al., 2002a; Voliantitis et al., 2001). Consequently, the improved exercise performance noted in these studies may have been due to changes in training or RMT or some combination of the two. It is also crucial to objectively quantify the total RMT training volume. Researchers in several studies did so by placing temperature and humidity counters within the RM training devices (Romer et al., 2002a). Such equipment avoids the subjective and potentially misleading data given in voluntary training diaries. In short, relying on subjective journal entry always limits studies of this type.

A.



B.

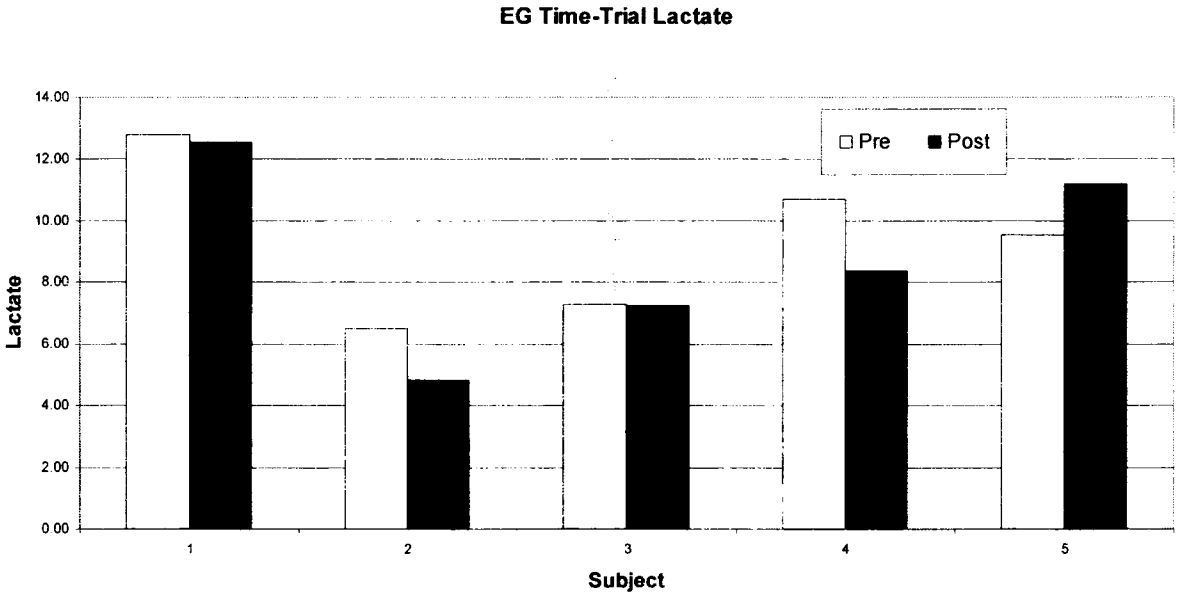


Figure 4. Individual 20 km time-trial blood lactate data pre- to post- respiratory muscle training. A shows the data for the CG (n = 5) group and B shows the data for the EG (n = 5) group.

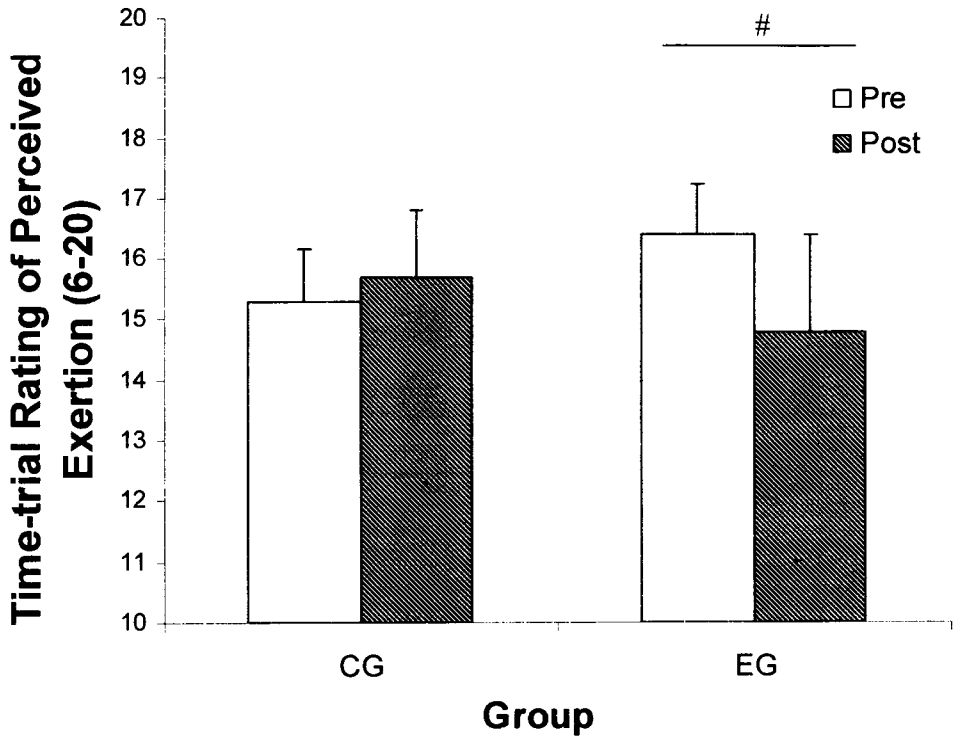


Figure 5. Time-trail rating of perceived exertion (Mean ± *SD*) pre- to post- respiratory muscle training. CG (n = 5). EG (n = 5). # = significantly different across time ($p<0.05$).

Chapter 6

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

This study examined the effects of concurrently training both inspiratory and expiratory muscles on maximal and sub-maximal exercise performance. Ten male cyclists volunteered to participate in the study. Subjects were randomly assigned to an experimental and control group and following initial testing of respiratory muscle endurance exercise performance commenced a 6 week training protocol. Several methodological errors were made with performance tests that likely prevented showing significant differences in several variables. A small subject pool and large individual variation also reduced the power of the study. Nevertheless, CRMT improved respiratory muscle performance and reduced perceived effort during a 20 km time-trial.

These changes were no greater than those from inspiratory muscle training alone. Hence, an athlete may wish to just train just the inspiratory muscles. Ultimately, RMT may be most beneficial for trained cyclists by reducing the perception of peripheral effort during intense prolonged exercise. This reduced perception of peripheral effort may allow more attention to relevant performance tasks during the event while reducing the amount of focus wasted on sensations of pain or discomfort.

Conclusion

The results of the study yielded the following conclusions:

1. Respiratory muscle training (RMT) using a handheld pressure device does increases respiratory muscle performance defined by an increase in maximal inspiratory mouth pressures.
2. CRMT did not improve maximal or sub-maximal endurance exercise performance in well trained cyclists.
3. CRMT decreased perceived effort during a 20 km time-trial.

Recommendations

The following recommendations for further study were made after the completion of this investigation:

1. A test of sub-maximal exercise performance workloads should be based on individual exercise performance and not an absolute arbitrary number.
2. Subject RMT adherence needs to be carefully tracked and objectively quantified as much as possible.
3. Further investigation into the differences between high resistance-low flow rates and low resistance-high flow rates as a respiratory muscle training protocol seems advisable.

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APPENDIX A

Informed Consent Form

Effect of respiratory muscle training on 20 km cycling time trial performance.

1. **Purpose of the study:** The purpose of the proposed project is to determine whether training the respiratory muscles improves intense sub-maximal exercise performance.
2. **Benefits of the study:** By participating in this study you will obtain measurements of your lactate threshold and maximum aerobic power output, both of which are valuable training tools. These tests cost approximately $250 in the open market. You will also have the opportunity to try an interesting new training method, which may improve your cycling performance. Upon completion of all test you will also be given your own PowerLung® respiratory muscle trainer and T-shirt. Your results will be accompanied by a complete explanation, including ways to incorporate them into your training program.
3. **Your Participation Requires:** To participate in this project you must be a male between the ages of 18 and 40 who has trained extensively the last 3 years, and have a VO_{2Max} greater than 55 ml/kg/min. You must complete a total of four lab testing sessions, each taking approximately one-hour. In the first testing session you will report to the lab and perform several tests. The first of which is a set of pulmonary measurements that include a maximal inspiratory and expiratory strength maneuver, a 15 sec maximal voluntary ventilation test (15s MVV), and a respiratory endurance test. The pulmonary strength test requires maximally inhaling and exhaling through a mouthpiece attached to a handheld device. This device will measure the total pressure generated by your lung muscles during each maneuver and is considered respiratory muscle strength. After a brief recovery period you will complete the second capacity test, a breathing endurance test, in which you will breath through the PowerLung® device at a frequency of 8 breaths per minute. The third respiratory test is 15-second maximal voluntary ventilation in which you will breath as much as possibly through a mouthpiece for a period of 15 seconds. After these tests you will warm-up of 15 min at 150 watts with your bike attached to a Computrainer™ resistance unit. Then you will complete a 10 min sub-maximal test at 300 watts. Small blood samples will be taken from a fingertip prick to determine blood lactate concentration 5 min before, during (5 and 10-minute mark), and 5 min after the test. Following the sub-maximal test and a 10 min recovery period, we will measure your maximum aerobic power output or oxygen consumption with a graded exercise test. In this test, also completed on your bike, we will begin at a workload of 200 watts and increase the intensity of exercise every 2 min until you become fatigued. While you pedal your bike, we will collect your expired air, which requires breathing through a mouthpiece attached to headgear. We will also measure your heart rate and power output throughout the test using the Computrainer™. At the end of each 2 min stage you will report your perceived level of whole body and respiratory exertion by rating from a chart numbered 1 through 10. One is easy and ten is max. Before the test begins and 5 min after, we will take a small blood sample from your fingertip so that we can determine your blood lactate concentration. Less than a week later you will return to the lab for one more test. You will start with a warm up of 15 min at 150 watts while your bike is attached to the Computrainer™ followed by a 20 km time-trial test. You will be instructed to finish the time-trial distance in the least amount of time possible. Verbal encouragement will be given and time checks

Initial__

given every 4 km. We will collect expired gases, blood samples, and ratings of perceived whole body and respiratory muscle exertion every 4 km throughout the test as described above. Blood samples will also be taken before the warm-up and 5-min after the time trial. Heart rate, power output, velocity, and cadence will be measured continuously during the time trial with the Computrainer™. After completing these initial tests you will be assigned to a respiratory muscle strength or endurance training group. Subsequently, you will be issued a PowerLung® respiratory muscle trainer to use for the next 6-weeks. The strength group will complete 30 breaths 2 times a day at a resistance equal to 80% peak inspiratory mouth pressure. The endurance group will take 120 breaths once a day at a resistance equal to 15% peak inspiratory mouth pressure. These tests will take about 8 min. You will keep a brief record of all cycling and respiratory training in the journal provided. Following the 6-week training period you will be contacted to come back to the lab to repeat all tests. Your participation will require approximately 4 hours of lab testing and 8 minutes a day for in-home respiratory training, which is about 1 hour per week. Total time, therefore, is approximately 10 hours.

4. **Risks of Participation:** You may feel some muscle soreness, particularly 24 to 48 hours after an exercise test. There is also a small chance of musculo-skeletal injury associated with vigorous exercise. In addition, you may experience some soreness in your fingertip(s) where the blood was drawn. There is the remote chance that a heart attack or other serious injury, even death, may occur.

If you would like more information about this study at anytime prior to, during, or following the data collection, you may contact either Chad Butts (257-2805) or Tom Swensen (274-3114)

6. **Withdrawal from the study:** You are free to withdraw from this study at anytime without prejudice. Please inform us of your decision should you decide to do so.

7. **Confidentiality:** All data collected in this study will be coded to insure your confidentiality. You name will not appear in any reports from this study.

I have read and understood the above document. I agree to participate in this study and realize that I can withdraw at anytime. I also understand that I can and should address questions related to this study at any time to the researchers involved. I also verify that I am at least 18 years of age.

_____
Name of Subject (please print)

_____ _____
Signature of Subject Date

_____ _____
Witness Date

APPENDIX B

Debriefing Statement

At the beginning of this study you were instructed that you would be participating in respiratory muscle endurance vs. respiratory muscle strength training study using the Powerlung^{TM} respiratory training device. However, deception was used in this study in order to maintain valid and rigorous results. Subjects participating in the endurance protocol were actually a control group, and given a training protocol that would produce negligible training results. This statement is to inform you of the deception in accordance with the regulations set forth by the Internal Review Board.

APPENDIX C

Training Diary

Week of: HR Zones: ( ) ( ) ( ) ( )

Weeks Goals (check as achieved):

Planned Hours_____

1._____

2._____

3._____

Monday ____ Warnings: Rate first four below on scale of 1-7. 1=best 7=worst. 5+ is a warning.

Sleep Quality: Fatigue: Stress: Soreness: RHR: Weight:

Wrkt 1: Plan: Actual: Distance: AVS: Avg. HR:____

Comments:

_____

Rating:

Tuesday ____ Warnings: Rate first four below on scale of 1-7. 1=best 7=worst. 5+ is a warning

Sleep Quality: Fatigue: Stress: Soreness: RHR: Weight:

Wrkt 1 Plan: Actual: Distance: AVS: Avg. HR:___

Comments:

_____

Rating:

Wednesday ____ Warnings: Rate first four below on scale of 1-7. 1=best 7=worst. 5+ is a warning.

Sleep Quality: Fatigue: Stress: Soreness: RHR: Weight:

Wrkt 1 Plan: Actual: Distance: AVS: Avg. HR:_____

Comments:

_____

Rating:

Thursday ____ Warnings: Rate first four below on scale of 1-7. 1=best 7=worst. 5+ is a warning

Sleep Quality: Fatigue: Stress: Soreness: RHR: Weight:

Wrkt 1 Plan: Actual: Distance: AVS: Avg. HR:_____

Comments:

_____

Week Of:

Friday ____ Warnings: Rate first four below on scale of 1-7. 1=best 7=worst. 5+ is a warning.
Sleep Quality: Fatigue: Stress: Soreness: RHR: Weight:
Wrkt 1 Plan: Actual: Distance: AVS: Avg. HR:___
Comments:
_____

Rating:

Saturday ____ Warnings: Rate first four below on scale of 1-7. 1=best 7=worst. 5+ is a warning
Sleep Quality: Fatigue: Stress: Soreness: RHR: Weight:
Wrkt 1 Plan: Actual: Distance: AVS: Avg. HR:___
Comments:
_____

Rating:

Sunday ___ Warnings: Rate first four below on scale of 1-7. 1=best 7=worst. 5+ is a warning
Sleep Quality: Fatigue: Stress: Soreness: RHR: Weight:
Wrkt 1 Plan: Actual: Distance: AVS: Avg. HR:___
Comments:
_____

Rating:

Weekly Summary: _____

_____

Total Bike Time: **Notes:**
Miles:
Strength Time:
I had soreness:

APPENDIX D

Rating of Ventilatory Exertion

0	No Breathlessness
0.5	Very, Very Slight (Just Noticeable)
1	Very Slight
2	Slight Breathlessness
3	Moderate
4	Some What Severe
5	Severe Breathlessness
6	
7	Very Severe Breathlessness
8	
9	Very, Very Severe (Almost Maximum)
10	Maximum

APPENDIX E

ANOVA Tables

Table A1.

Sub-maximal Test Summaries of F Score and p Values From the Analysis of Each Dependent Variable Using Separate 2 x 2 ANOVA (Group x Time) with Repeated Measures

	Source		
Variable	Group (df = 1,8)	Time (df = 1,8)	Group x Time (df = 1,8)
Mass			
F	5.59	3.19	14.9
p	0.05	0.11	0.00
PI_{max}			
F	2.19	17.9	7.48
p	0.18	0.00	0.03
PE_{max}			
F	1.92	2.03	0.95
p	0.20	0.19	0.36
RME			
F	6.51	15.3	4.68
p	0.03	0.00	0.06
MVV			
F	3.89	0.09	0.00
p	0.09	0.77	0.98
RPE_{Sub}			
F	1.49	0.29	4.70
p	0.26	0.60	0.06
RPE_{TT}			
F	0.05	3.36	8.96
p	0.83	0.10	0.02

Note. PI_{max}, inspiratory muscle strength; RME, respiratory muscle endurance; RPE_{Sub}, sub-maximal rating of perceived exertion; RPE_{TT}, TT rating of perceived exertion.

Table A2.

Respiratory Muscle Function Summaries of F Score and p Values From the Analysis of Each Dependent Variable Using Separate 2 x 2 ANOVA (Group x Time) with Repeated Measures

		Source		
Variable		Group (df = 1,8)	Time (df = 1,8)	Group x Time (df = 1,8)
PI_{max}	F	2.19	17.9	7.48
	p	0.18	0.00	0.03
RME	F	6.51	15.3	4.68
	p	0.03	0.00	0.06
RPE_{sub}	F	1.49	0.29	4.70
	p	0.26	0.60	0.06
RPE_{TT}	F	0.05	3.36	8.96
	p	0.83	0.10	0.02

Note. PI_{max}, inspiratory muscle strength; RME, respiratory muscle endurance; RPE_{Sub}, sub-maximal rating of perceived exertion; RPE_{TT}, TT rating of perceived exertion.