

RESPIRATORY MUSCLE TRAINING AS AN ERGOGENIC AID

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Most sport scientists still do not consider breathing to be a limiting factor for exercise performance. However, the past decade has seen evidence emerge showing unequivocally that breathing not only limits exercise performance, but that removal of this limitation improves performance. This review will describe the mechanisms by which respiratory muscle work limits exercise performance, as well as the evidence that specific training of the respiratory pump muscles improves performance in the context of both endurance and repeated sprint sports. Finally, the mechanisms underlying this ergogenic effect will be considered, as well as their implications for the practical application of respiratory muscle training. [*J Exerc Sci Fit* • Vol 7 • No 2 (Suppl) • S18–S27 • 2009]

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Introduction

Exercise physiologists have conventionally viewed the respiratory system as not imposing any limits on human exercise performance in healthy people, much less, athletes. This preconception is predicated on three main assumptions: (1) at sea level, oxygen transport is not limited by the diffusion of oxygen in the lung; (2) human beings appear to have considerable breathing reserve, even at maximal exercise; (3) the respiratory muscles are highly evolved and developed for their function and do not show fatigue during exercise. These preconceptions argue against the existence of any benefit from specific training of the respiratory pump. The purpose of this review is to challenge assumptions regarding the role of breathing in exercise limitation, and, more importantly, to present evidence for an ergogenic effect of respiratory muscle training (RMT).

An obvious place to alight on this journey is to address the three points above, which have led directly to the assumption that the respiratory system is not limiting to performance. Points 1 and 2 are factually

indisputable, but their widely assumed implications are based in fallacy. The implication of point 1 is that $\dot{V}O_{2\max}$ is the single determinant of exercise performance, which is untrue. The implication of point 2 is that maximal ventilation can be sustained indefinitely, which is also untrue. With respect to point 3, research has now shown that inspiratory muscles do exhibit fatigue following exercise (see below). Accordingly, the assumption that breathing does not limit exercise performance is built on shaky foundations.

The past decade has witnessed a considerable advancement in our understanding of the limitations imposed by respiratory muscle work, as well as providing insights into the mechanisms by which specific training of the respiratory pump muscles elicits improvements in exercise performance. The following sections summarize this knowledge.

The Evidence for Respiratory-related Limitations to Performance

In the context of this article, discussion of respiratory limitations to performance will be confined to those originating from the mechanical work of breathing. What has been largely overlooked is the fact that the mechanical work of pumping air in and out of the lungs has both sensory and metabolic repercussions. In other



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words, the work of breathing is perceived, and contributes to how hard exercise feels; the work of breathing also places demands on the circulatory system for blood flow to sustain muscle contraction. Both of these factors contribute to exercise limitation and will be discussed in greater detail later.

An important premise in an argument that respiratory muscles do limit exercise tolerance/performance is that they function at, or close to, the limits of their capacity, and that this is manifest as specific fatigue. Without such evidence, it is hard to justify a rationale for specific training of any muscle. The earliest reports of inspiratory muscle fatigue (IMF) following competitive events appeared in the early 1980s after marathon running (Loke et al. 1982). Later research confirmed these findings following marathon running (Ross et al. 2008; Chevolet et al. 1993), but also provided data suggesting that ultra-marathon (Ker & Schultz 1996) and triathlon (Hill et al. 1991) competition were fatiguing to the respiratory pump muscles.

Under laboratory and field-based research conditions, my own research group has also demonstrated IMF following rowing (Griffiths & McConnell 2007; Volianitis et al. 2001), cycling (Romer et al. 2002c) and swimming (Lomax & McConnell 2003), as well as sprint triathlon (Sharpe et al. 1996) and treadmill marathon running (Ross et al. 2008).

All of the studies cited above have evaluated IMF using maximal inspiratory pressure (MIP) measured at the mouth, which is a holistic, voluntary surrogate of inspiratory muscle force production. Whilst MIP has its merits (noninvasive, portable, quick and easy to administer, reliable, holistic), it can also be criticized for being susceptible to the influence of changes in effort. In other words, immediately after exercise, a lower MIP might be the result of reduced effort, and not due to physiological factors. However, diaphragm fatigue has also been confirmed following heavy exercise using electrical stimulation of the phrenic nerves (Babcock et al. 1998, 1997; Johnson et al. 1996; Mador et al. 1993). Thus, not only is there evidence of IMF following real world sports participation and laboratory simulations of competition, rigorous laboratory trials also demonstrate specific contractile fatigue of the diaphragm after heavy exercise.

But what of the expiratory muscles? The effect of real world sports activities upon expiratory muscle function has been much less extensively studied, and the data that exist are currently contradictory. Following marathon running that induced a fall in MIP, no change in maximal expiratory pressure (MEP) was observed

post-exercise (Ross et al. 2008; Chevolet et al. 1993). Similarly, following a triathlon that elicited a significant fall in MIP, there was no change in MEP (Hill et al. 1991). In contrast, following a rowing time trial that simulated a 2000-m rowing race in the laboratory, a significant decline in MEP was observed (Griffiths & McConnell 2007). Similarly, under laboratory conditions where cycling exercise was performed to the limit of tolerance (Tlim), MEP was shown to decline (Cordain et al. 1994). In contrast, some authors observed no change in MEP following maximal cycle ergometer exercise, but did observe a fall in MIP (Coast et al. 1999). Non-volitional assessment of expiratory muscle fatigue (EMF) using magnetic stimulation of abdominal muscles has recently demonstrated EMF following high intensity cycle ergometer exercise to Tlim (Taylor et al. 2006; Verges et al. 2006). Thus, it appears that EMF may be specific to certain exercise modalities and/or intensities of exercise. These conditions appear to be characterized by exercise at maximal intensity, and/or situations in which the expiratory muscles have a key role in propulsive force transmission, such as rowing.

Collectively, the literature points to IMF occurring in response to a wider range of activities than EMF, and possibly also at lower intensities and/or following shorter durations of activity.

The next important question is, "What are the functional repercussions of respiratory muscle fatigue (RMF)?" This has been studied using a variety of experimental designs, but principally by using two basic approaches. First, by inducing RMF and studying its influence upon subsequent exercise; second, by manipulating the work of breathing during exercise to accelerate RMF (by adding a resistance to breathing), or to delay the time to RMF (by using a ventilator to undertake the work of breathing).

The effects of prior IMF and EMF are to increase the intensity of breathing effort during subsequent whole body exercise, and to lead to a shorter time to Tlim during constant power output exercise. For example, in one of the first studies to examine the effect of prior IMF on exercise tolerance, Mador and Acevedo (1991) observed a 23% reduction in Tlim during cycling at 90% of $\dot{V}O_{2max}$. They also noted an increase in the sensation of effort during exercise, i.e. exercise after IMF felt harder. Using a slightly different design, my own group has also studied the effects of prior IMF on subsequent exercise (McConnell & Lomax 2006). In this case, isolated plantar flexion was used as the index of exercise performance. We observed a more rapid fatigue of the plantar flexor muscles after IMF (the reasons for this

are explained later). More recently, the effects of prior EMF on cycling performance were assessed (Taylor & Romer 2008). These authors also observed a decrease in $\dot{V}_{E_{lim}}$ during exercise (33%) that followed EMF, as well as increases in the perceptions of both breathing and leg effort. Leg fatigue was also more severe after EMF. However, a note of caution is required in the interpretation of studies that have pre-fatigued the expiratory muscles. Taylor and Romer (2009) have also shown that expiratory loading induces simultaneous IMF and EMF. In other words, the effect of EMF on subsequent exercise performance is “contaminated” by accompanying IMF. Thus, any effects on performance cannot be ascribed solely to the expiratory muscles.

On the face of it, the observations that RMF exacerbates limb muscle fatigue defy logical explanation; after all, why should fatiguing the respiratory muscles exacerbate fatigue in the limb muscles? The answer lies in the findings of a series of studies that have examined the influence of manipulating the work of breathing during exercise.

In this series of very elegant studies undertaken at the University of Wisconsin, Professor Jerome Dempsey and colleagues have painstakingly and systematically studied the role of the inspiratory muscles in exercise tolerance. An important tool in their research has been a proportional assist ventilator that is able to undertake the work of the inspiratory muscles during exercise.

In their first study exploring the impact of the work of breathing on exercise tolerance, Professor Dempsey and colleagues examined the influence of changes in the work of breathing on blood flow to the legs during maximal cycle ergometer exercise (Harms et al. 1997). They observed a reciprocal relationship between leg blood flow and the work of breathing, such that when the inspiratory work of breathing was undertaken by a ventilator, there was a 4.3% increase in leg blood flow. In contrast, when the work of inspiration was increased by breathing against a resistance, leg blood flow decreased by 7%. The changes in leg blood flow were mediated by changes in the extent of limb vasoconstriction. In a series of subsequent studies, Professor Dempsey and colleagues showed that the stimulus for limb vasoconstriction was a cardiovascular reflex originating within the inspiratory muscles (Sheel et al. 2002, 2001; St Croix et al. 2000).

The “inspiratory muscle metaboreflex”, as it has become known, is activated when metabolite accumulation within the inspiratory muscles stimulates afferent nerve fibers (type III and IV afferents) to increase their

firing frequency. Stimulation of these fibers precipitates an increase in the strength of sympathetic neural outflow, which induces a generalized vasoconstriction. In a recent study, Professor Dempsey’s group have shown that the changes in leg blood flow elicited by increasing and reducing the work of inspiration are correlated with changes in the magnitude of exercise-induced leg fatigue. In other words, increasing the work of inspiration reduces leg blood flow and exacerbates leg fatigue (Romer et al. 2006). These findings complete the circle that links RMF with leg fatigue, i.e. metaboreflex activation precipitates limb vasoconstriction, reducing limb blood flow and accelerating limb fatigue.

Earlier, we touched briefly on the effect of RMF on breathing and limb effort, i.e. RMF intensifies these sensations during exercise (Taylor & Romer 2008; Mador & Acevedo 1991). The influence on limb effort should be clear from the preceding discussion, i.e. RMF reduces limb blood flow, accelerates limb fatigue and increases limb effort perception. Before closing this discussion, I want to mention briefly the effect of IMF on the perception of breathing effort, because breathing effort is a potent but overlooked factor that influences whole body perceived exertion and performance.

Whilst it might seem self-evident that weak or fatigued muscles generate greater perception of effort than fresh or stronger muscles, the neurophysiological mechanism underpinning this reality merits description. The human brain is able to judge the size of the outgoing neural drive to muscles (McCloskey et al. 1983). As the muscle responds to this drive, it returns information to the brain about the tension that is being generated within it, as well as the speed and distance of muscle and joint movement (Cafarelli 1982). Tension, speed and distance are all proportional to the resistance that the muscle is overcoming. The sensory area of the brain compares the size of the neural drive to the muscles, with the sensory information from the muscles. In doing so, it formulates a perception of effort (Cafarelli 1982). For example, if the neural drive to the biceps during an arm curl is high (because the muscle is weak or fatigued), and the sensory feedback suggests that there is a high tension within the muscles, and that the bar is moving slowly and through a limited range, the perception of effort is high, i.e. the weight feels heavy (Gandevia & McCloskey 1978).

The size of the neural drive required to generate a given external force is influenced by a number of factors, but principally by the force-generating capacity of the muscle. A strong muscle requires a lower neural

drive to generate a given force, because the force represents a smaller proportion of its maximum capacity. Similarly, a fatigued muscle requires a higher neural drive to generate a given force, because the force represents a higher proportion of its maximum capacity.

These principles apply equally to the respiratory muscles (Campbell 1966) as they do to other skeletal muscles, so it is easy to see why and how weakness, fatigue, or indeed strengthening, of the respiratory muscles can modulate the perception of breathing effort. How might this impact upon performance? During whole body exercise, athletes use their perceived effort as a pacing cue; intolerable effort for a given speed will lead to a reduction in pace. Similarly, if the tolerable threshold is raised by muscle training, pace is also raised. There is now overwhelming evidence that strengthening of the inspiratory muscles reduces breathing effort perception and/or inspiratory motor drive (Huang et al. 2009; Edwards et al. 2008; Tong et al. 2008; Griffiths & McConnell 2007; Huang et al. 2003; Romer et al. 2002a, 2002b; Volianitis et al. 2001; Kellerman et al. 2000; Redline et al. 1991). This being the case, strengthening of the inspiratory muscles has the potential to improve performance as a direct result of the effect on breathing and whole body effort perception.

Thus, the respiratory muscles can contribute to exercise limitation through their influence on cardiovascular reflex control, i.e. fatiguing respiratory muscle work elicits reflex responses that reduce limb blood flow. The respiratory muscles also make a potent contribution to the perception of effort during exercise. When respiratory muscle work is relatively high (for the capacity of the muscles), there is a greater perception of effort. Similarly, fatigue of the respiratory and limb muscles exacerbates effort perception still further.

RMT

Research on RMT predates the insights discussed above by almost two decades, having its origins in the 1970s, when early studies sought to demonstrate nothing more than that the respiratory muscles exhibited adaptation to specific training (Leith & Bradley 1976). Later research sought to evaluate the potential benefits of such training with respect to exercise tolerance in both healthy people and patients with respiratory disease. Up until the late 1990s, the literature on RMT in healthy young adults was contradictory to say the least (see McConnell & Romer 2004). Poor research designs and inappropriate

outcome measures had created a very “mixed bag” of data that only served to confirm the received wisdom that breathing did not limit exercise performance. A turning point in RMT research came during the late 1990s, when more reliable methods of training became available and were used more widely (specifically pressure threshold training). In addition, with the benefit of hindsight, a more rigorous approach to research design was also adopted.

The following section is divided into three subsections. The first addresses the responses to RMT during endurance exercise; the second examines the responses during repeated sprint exercise; and the third addresses the unique aquatic environment.

Endurance exercise

The majority of studies examining the influence of RMT on exercise performance have done so using performance tests that would be considered endurance based. Two types of exercise test have been assessed: (1) fixed intensity exercise undertaken to Tlim; (2) time trials.

As an outcome measure, Tlim tests are extremely sensitive to small physiological improvements (Amann et al. 2008), yield large changes (>30%), and allow physiological and perceptual responses to be studied under identical conditions before and after the intervention. In contrast, the obvious advantage of using a time trial to assess performance is that it simulates a race. However, for this very reason, the magnitude of the changes that are typically observed following ergogenic interventions is extremely small (<5%). In addition, it is impossible to compare physiological responses during time trials in a meaningful way before and after the intervention because the exercise conditions are not identical pre- and post-intervention.

Typically, Tlim tests are conducted at intensities that are just above the lactate threshold, and previous studies examining RMT have used exercise intensities that elicit Tlim within 20–40 minutes. In contrast, time trials have varied considerably, depending on the modality of exercise that has been studied (as little as 6 minutes, or as much as 1 hour).

There are principally two methods for training the respiratory muscle: (1) resistance training using imposed external loads at the mouth; (2) endurance training using voluntary hyperpnea. Studies that have employed resistance training of the respiratory muscles have assessed performance during cycling, rowing, and running. In the case of cycling, this has been undertaken using both Tlim tests and time trials, whilst rowing and running have used time trials only. Table 1 summarizes

Table 1. Placebo controlled studies of the influence of resistance respiratory muscle training upon endurance performance

Type of training	Exercise modality	Type of exercise test	Duration/intensity of test	Training duration	Performance change	Physiological changes in training group	Comments	Authors
Resistance IMT	Cycling	Tlim	21 min	4 wk	33%	Attenuation of [La] _b and RPE		Caine & McConnell 1998
Resistance IMT	Cycling	TT	20 km & 40 km (~30 & ~60 min)	6 wk	3.8% & 4.6%	Attenuation of breathing & leg effort	IMF also attenuated	Romer et al. 2002a
Resistance IMT	Cycling	Tlim	75% $\dot{V}O_{2max}$	10 wk	36%	Attenuated fc, V_E & perception of effort		Gething et al. 2004
Resistance IMT	Cycling	TT	25 km (36 min)	6 wk	2.6%		IMF also attenuated	Johnson et al. 2007
Resistance IMT	Running	Tlim	3.8 min	4 wk	4%			Edwards & Cooke 2004
Resistance IMT	Running	TT	5000 m (~20 min)	4 wk	2%	Attenuation of RPE		Edwards et al. 2008
Resistance IMT	Rowing	TT	6 min & ~20 min (~2 km & 5 km)	4 wk & 11 wk	1.9% & 2.2%	Attenuation of [La] _b & breathing effort & increased V_T	Performance improved at 4 & 11 wk. IMF also attenuated	Volianitis et al. 2001
Resistance IMT + EMT	Rowing	TT	6 min (~2 km)	4 wk & 10 wk	2.7%	Attenuated fc, [La] _b & perception of breathing effort	Performance & other outcomes only improved in response to IMT. IMF also attenuated	Griffiths & McConnell 2007

IMT = inspiratory muscle training; EMT = expiratory muscle training; Tlim = fixed intensity to the limit of tolerance; TT = time trial; $\dot{V}O_{2max}$ = maximal oxygen uptake; [La]_b = blood lactate concentration; RPE = rating of perceived exertion; fc = heart rate; V_E = minute ventilation; V_T = tidal volume; IMF = inspiratory muscle fatigue.

the findings of placebo-controlled studies of resistance RMT. Only studies that have included a placebo group that undertook “sham” training are included in this summary, since the results of studies not employing this level of scientific rigor are highly questionable.

It is apparent from Table 1 that there are improvements in performance following inspiratory muscle training (IMT) [but not expiratory muscle training (EMT)] when assessed using both time trials and Tlim tests. In the case of time trials, the improvements range from 1.9% to 4.6% (above placebo) in trials ranging in duration from 6 minutes (rowing) to 60 minutes (cycling). Improvements in Tlim trials are larger in magnitude, typically >30% for tests lasting around 20 minutes. For higher intensity, shorter duration Tlim tests, the improvements are correspondingly smaller (4% for a test lasting <4 minutes). This difference is most likely due to the fact that during very high intensity exercise, an escalating metabolic acidosis, and associated contractile failure of locomotor muscles, is the principal source of limitation. In contrast, for lower intensity tests, unwillingness to tolerate unpleasant respiratory and locomotor muscle discomfort is the limiting factor.

Table 2 summarizes the controlled and placebo-controlled trials of respiratory muscle endurance training. Non-placebo-controlled trials have been included, because to do otherwise would reduce the number of studies to just two. Furthermore, the controlled trials in question are of high quality. A similar pattern emerges from these data, in that Tlim trials typically show a post-RMT improvement of 20–50%, whilst the only time trial following respiratory muscle endurance training showed a 4% improvement.

Respiratory muscle endurance training involves hyperventilation at high levels for prolonged periods, and its effects on the inspiratory and expiratory muscles are therefore impossible to separate. In contrast, inspiratory and expiratory muscle resistance training are easy to separate, and doing so provides an indication of the role of the two groups of muscles in performance changes. The only study to undertake a crossover trial of both IMT and EMT found no evidence of an effect of EMT on performance (Griffiths & McConnell 2007). Indeed, adding EMT to IMT during the same breath cycle seemed to impair inspiratory muscle responses to IMT.

Repeated sprint exercise

Perhaps because of the underlying mechanisms and the assumptions regarding the factors that limit exercise during sprinting, i.e. contractile failure due to

Table 2. Controlled studies of the influence of endurance respiratory muscle training on endurance performance

Type of training	Exercise modality	Type of exercise test	Duration/intensity of test	Training duration	Performance change	Physiological changes in training group	Comments	Authors
Endurance RMT	Cycling	Tlim	70% peak power	15 wk	24%		No placebo group, just non-RMT control group	Markov et al. 2001
Endurance RMT	Cycling	Tlim	70% peak power	15 wk	24%		No placebo group, just non-RMT control group	Stuessi et al. 2001
Endurance RMT	Cycling	Tlim	85% peak power	4–6 wk	~20%		No placebo group, just non-RMT control group	McMahon et al. 2002
Endurance RMT	Cycling	Tlim	40 min	6 wk	4.7%			Holm et al. 2004
Endurance RMT	Running	TT & Tlim	TT = 4 miles Tlim = 80% $\dot{V}O_{2max}$	4 wk	TT = 4% Tlim = 50%	Attenuated V_E , $\dot{V}O_{2}$, $[La]_b$		Leddy et al. 2007

RMT = respiratory muscle training; Tlim = fixed intensity to the limit of tolerance; TT = time trial; $\dot{V}O_{2max}$ = maximal oxygen uptake; V_E = minute ventilation; $\dot{V}O_{2}$ = oxygen uptake; $[La]_b$ = blood lactate concentration.

metabolic acidosis, there have been relatively few studies of IMT in the context of sprint sports.

My own group was the first to study this, and we did so based on the premise that IMT attenuated breathing effort perception, and that it might therefore improve the perceived rate of recovery during repeated sprinting (Romer et al. 2002b). A repeated sprint test was devised, consisting of a series of 20-m sprints, with a self-selected rest period of up to 30 seconds between each sprint. The performance outcomes for the test were the total recovery time and total sprint time. We hypothesized that after IMT, the associated reduction in breathlessness and slower rate of IMF development would be manifested in a reduction in recovery time. However, we did not anticipate sprint performance to improve, as these were maximal, very brief (3.2 seconds), and limited by metabolic pathways that are largely independent of blood flow. The hypothesis was confirmed, as we observed a 6% reduction in the rate of recovery during a repeated sprint test. In separate 20-minute fixed speed shuttle running tests, we also observed a reduction in blood lactate concentration ($[La]_b$), as well as breathing and whole body effort perception. There was also a correlation between the change in recovery time, and the changes in $[La]_b$ and effort perceptions. The latter observation suggests that the improvement in recovery time was related to the amelioration of effort perceptions generated by IMT.

More recently, two controlled studies have explored a slightly different aspect of repeated sprint performance by employing a test that was developed originally as a sport-specific performance assessment for soccer (Bangsbo 1994). In common with our original repeated sprint test (Romer et al. 2002b), the so-called Yo-Yo intermittent recovery test drives breathing and metabolism to maximal levels, but unlike our test, performance in the Yo-Yo test is influenced strongly by the capacity of the aerobic energy system (lasting approximately 20 minutes for a well-trained individual) (Bangsbo et al. 2008). Performance in the Yo-Yo test is therefore influenced by both effort perception (Impellizzeri et al. 2008) and blood flow related factors such as oxygen delivery and metabolite removal.

Two studies employing the level 1 Yo-Yo test (level 1 is designed to emphasize the aerobic contribution to repeated sprinting) have demonstrated improvements in the number of repetitions that can be achieved after IMT (5–6 weeks) of 16% and 17% (Nicks et al. 2009; Tong et al. 2008). Accompanying the improvement in performance were reductions in breathing and whole

body effort perception, as well as markers of metabolic stress (Tong et al. 2008). None of these changes were present in the placebo or control groups (Tong et al. 2008).

Aquatic exercise

The aquatic environment is one of the most challenging for the respiratory muscles, and competitive swimming presents one of the ultimate challenges for breathing. High intensity front crawl swimming is associated with the most severe (29% fall in MIP) and fastest developing (2.5 minutes) states in IMF of any sport thus far studied (Lomax & McConnell 2003).

Bearing this in mind, and the obvious “respiratory gymnastics” that are such an integral part of swimming, it is surprising that there have been so few studies of IMT in the context of swimming performance. At the time of writing, only two studies have examined the influence of RMT on surface swimming performance. The first employed simultaneous IMT and EMT, and failed to elicit significant improvements (above the similarly modest changes of the placebo group) in either respiratory muscle strength or swim performance (Wells et al. 2005). The latter finding is undoubtedly related to the former, since breathing effort perception and the inspiratory muscle metaboreflex are unlikely to be modified if RMT fails to improve respiratory muscle function. Most recently, Kilding and colleagues (2009) examined the influence of IMT on 100 m, 200 m and 400 m front crawl swim performance. The placebo-controlled study involved 16 club-level swimmers (average age, 19 years). After 6 weeks of IMT, MIP increased significantly (9%) and rating of perceived exertion during incremental swimming was reduced. There were also improvements in 100 m and 200 m swim performance (by 1.7% and 1.5% compared to placebo, respectively), but no change in 400 m performance. Maximal swim velocity and velocity at the lactate threshold also remained unchanged, which is consistent with results from studies of IMT during terrestrial exercise. It is presently unclear why IMT did not improve 400 m swim performance, and it is also noteworthy that the increase in MIP after IMT was lower than that seen in terrestrial athletes. This may be an artefact of swim training *per se*, which has been shown to provide a training stimulus to the respiratory system (Clanton et al. 1987).

There have also been a series of studies of underwater swimming performance in experienced divers following RMT, and these have demonstrated improvements in both respiratory muscle function and underwater swim performance (Ray et al. 2008; Lindholm

et al. 2007; Wylegala et al. 2007). In two of these studies, both underwater and surface fin swimming endurance (T_{lim}) were assessed at 75% of maximum heart rate. Underwater swimming employed SCUBA, whilst surface swimming employed a snorkel. Following RMT, T_{lim} improved in surface and underwater swimming by 33–50% and 38–88%, respectively (Lindholm et al. 2007; Wylegala et al. 2007). There was also a small reduction in the oxygen uptake requirement of the underwater swim (7.8%), and heart rate was 5 beats \cdot min⁻¹ lower post-RMT (Wylegala et al. 2007). In common with terrestrial exercise, tidal volume also increased (12%) and breathing frequency was lower (19%) after RMT (Wylegala et al. 2007).

These data from fin swimming divers are not directly comparable to those of surface swimmers using front crawl, breast stroke, etc., but they provide strong supportive evidence that surface swimmers may experience meaningful performance enhancements when IMT is undertaken with the correct training equipment and regimen.

Underlying Mechanisms

No study of RMT has ever demonstrated a change in maximal oxygen uptake ($\dot{V}O_{2max}$). Some exercise physiologists interpreted this finding as a weakness of the studies; after all, how could training the respiratory pump increase exercise performance without increasing maximal oxygen uptake? Of course, a change in $\dot{V}O_{2max}$ would completely contradict our understanding of the factors that limit oxygen transport, since most human beings are not diffusion limited. For a time, it was thought that the answer may lie in a change in the lactate threshold, which is the other key mechanism by which exercise performance improvements are normally achieved following whole-body training. However, in a carefully conducted study, we showed that IMT did not improve the lactate threshold (McConnell & Sharpe 2005). Furthermore, a recent study has also demonstrated that IMT does not enhance critical power, but does enhance “anaerobic work capacity” (Johnson et al. 2007). The latter is thought to represent a finite energy store that can be used when exercise exceeds “critical power”, and the size of this store may be influenced by factors such as muscle metabolite accumulation. In turn, this accumulation is influenced by muscle blood flow.

These observations point to underlying mechanisms for the ergogenic effect of RMT that lie outside

our conventional view of training-induced improvements in performance, i.e. improvements are not due to increases in $\dot{V}O_{2max}$ and/or the lactate threshold. As was described in a previous section, there is now convincing evidence for the existence of an inspiratory muscle mediated reflex reduction in limb blood flow. This provides a convenient potential mechanism by which RMT might improve performance, but is there any direct evidence linking IMT to changes that delay or attenuate this reflex?

The majority of studies examining the inspiratory muscle metaboreflex have used models that evaluated vascular responses to the metaboreflex in either the resting limb (St Croix et al. 2000) or during whole body cycling at maximal exercise (Harms et al. 1998, 1997). These studies demonstrated that fatiguing inspiratory muscle work was capable of inducing vasoconstriction in the resting limb, and under conditions where cardiac output was maximal. However, they were unable to shed light on whether the metaboreflex could override the functional hyperemia of exercise under conditions where there was cardiac output reserve. Furthermore, even if the metaboreflex did operate under such conditions, it was far from clear whether IMT could modulate its operation.

In a recent study, we examined these questions using an isolated human lower limb model (McConnell & Lomax 2006). Our basic premise was that if the inspiratory muscle metaboreflex induced a functionally meaningful reduction in limb vascular conductance, then the resulting impairment of limb blood flow should accelerate limb fatigue. Furthermore, if IMT modulated the reflex, then the fatigue profile of the limb should also be modulated after IMT. To validate our limb fatigue model (plantar flexion), we first assessed its sensitivity to mechanical restriction of blood flow. In addition, we confirmed the ability of a fatiguing inspiratory muscle loading protocol (IMF) to activate the inspiratory muscle metaboreflex. Following this, a number of manipulations of the pre-plantar flexion conditions were implemented: (1) a bout of IMF immediately prior to the plantar flexion; and (2) an identical bout of IMF followed by a 30-minute period of rest (to allow the metaboreflex to dissipate) before plantar flexion. After a 4-week period of IMT, condition 1 was repeated. In addition, we implemented a final condition in which the intensity of the inspiratory muscle loading was increased to account for the training-induced improvement in inspiratory muscle strength.

When plantar flexion was preceded immediately by IMF, the rate of plantar flexor fatigue was accelerated.

When a 30-minute period of rest was given between the IMF and plantar flexion, the fatigue profile was not significantly different to control. Similarly, after IMT, the same bout of IMF failed to induce any change in the rate of plantar flexion fatigue. In contrast, when the intensity of IMF was increased to take account of the training-induced improvement in strength, the rate of plantar flexion fatigue was once again accelerated. These data support the notion that the inspiratory muscle metaboreflex operates at exercise intensities where there is cardiac output reserve. Furthermore, they suggest that IMT changes the threshold of inspiratory muscle work required to elicit the vasomotor response to activation of this metaboreflex. Thus, the most important determinant of the functional repercussions of inspiratory muscle work appears to be the relative intensity of that inspiratory muscle work.

In a more recent study, these presumptions have been confirmed. Witt and colleagues (2007) demonstrated that IMT attenuated the cardiovascular reflex responses induced by loaded breathing. In other words, their data confirmed that the threshold for activation of the metaboreflex was increased after IMT.

Summary

In summary, RMT research has been hampered by the results of early studies that generated contradictory data, fueling skepticism. In addition, there has been a very reasonable skepticism about an intervention that appeared to fly in the face of exercise physiologists' understanding of the role of the respiratory pump in oxygen transport. However, research conducted within the past decade has demonstrated that respiratory muscle work has far wider repercussions than was previously appreciated, and it is now known that these muscles contribute to both the metabolic demand and sensory experience of exercise. Recent studies suggest that IMT generates improvements in exercise tolerance through two main mechanisms, which are probably also interlinked: (1) attenuation of effort perception (exercise feels easier after IMT); and (2) modulation of the inspiratory muscle metaboreflex leading to a preservation of limb blood flow during exercise.

The role of RMT in enhancing exercise performance in athletes, as well as exercise tolerance in patients, has been made considerably more credible by newly acquired insights into the wider consequences of exercise-induced inspiratory muscle work.

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