ORIGINAL ARTICLE

Effects of high-intensity interval training on pulmonary function

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Abstract To determine whether high-intensity interval training (HIT) would increase respiratory muscle strength and expiratory flow rates more than endurance training (ET), 15 physically active, healthy subjects (untrained) were randomly assigned to an ET group $(n = 7)$ or a HIT group ($n = 8$). All subjects performed an incremental test to exhaustion ($VO₂max$) on a cycle ergometer before and after training. Standard pulmonary function tests, maximum inspiratory pressure (PImax), maximum expiratory pressure (PEmax), and maximal flow volume loops were performed pre training and after each week of training. HIT subjects performed a 4-week training program, 3 days a week, on a cycle ergometer at 90% of their $VO₂$ max final workload, while the ET subjects performed exercise at 60–70% $VO₂max$. The HIT group performed five 1-min bouts with 3-min recovery periods and the ET group cycled for 45 min continuously. A five-mile time trial (TT) was performed prior to, after 2 weeks, and after completion of training. Both groups showed improvements ($P < 0.05$) in $VO₂max$ (~8-10%) and TT (HIT 6.5 ± 1.3%, ET $4.4 \pm 1.8\%$) following training with no difference $(P > 0.05)$ between groups. Both groups increased $(P<0.05)$ PImax post training (ET $\sim 25\%$, HIT $\sim 43\%$) with values significantly higher for HIT than ET. There was no change ($P > 0.05$) in expiratory flow rates with training in either group. These data suggest that both whole-body exercise training and HIT are effective in increasing inspiratory muscle strength with HIT offering a

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time-efficient alternative to ET in improving aerobic capacity and performance.

Keywords Maximum inspiratory pressure - High-intensity interval training - Respiratory muscle strength - Pulmonary function tests - Expiratory flow rates

Introduction

Traditionally, endurance training (ET) has been the preferred method of training to elicit changes in performance, rehabilitation, and disease prevention. Alternatively, highintensity interval training (HIT) has long been employed by elite athletes but has been given little attention in the scientific community as an equally effective training method to improve aerobic performance measures until recent years. HIT leads to many similar physiologic adaptations as ET (Coyle [2005;](#page-6-0) Gibala and McGee [2008;](#page-7-0) Hellsten et al. [1998](#page-7-0); Parra et al. [2000](#page-7-0); Krustrup et al. [2004\)](#page-7-0). For example, the potential for delayed onset of fatigue has been found among HIT subjects in a number of different studies for various reasons, including an increased stimulus for ATP production (Hellsten et al. [1998\)](#page-7-0), an increased resting glycogen content (Parra et al. [2000\)](#page-7-0), a reduced rate of glycogen utilization (Parra et al. [2000\)](#page-7-0), and an enhanced expression of regulators of mitochondrial biogenesis (Coyle [2005;](#page-6-0) Krustrup et al. [2004](#page-7-0); MacDougall et al. [1998](#page-7-0); Mogensen et al. [2006](#page-7-0)). Studies have also observed an increased rate of oxygen delivery leading to an increased maximal oxygen uptake (Burgomaster et al. [2008](#page-6-0); Harmer et al. [2000;](#page-7-0) Krustrup et al. [2004](#page-7-0); MacDougall et al. [1998;](#page-7-0) McKenna et al. [1997](#page-7-0)). Adaptations have been similar to those found with ET.

While skeletal muscle adaptations to exercise training are well known, there is currently little information determining whether or not the effects of whole body exercise leads to increases in respiratory muscle strength. To date, it is believed that exercise training does not appreciably alter the pulmonary system. Eastwood et al. [\(2001](#page-6-0)) have shown no difference in inspiratory muscle strength with increased respiratory muscle endurance in endurance-trained athletes when compared with controls. Another study investigated airway responsiveness to see if the altered breathing patterns and hyperventilation from endurance exercise could reduce airway smooth muscle tone which would lead to increased expiratory flow rates through a reduction in airway hyperresponsiveness (Scichilone et al. [2005\)](#page-7-0). As airway diameter is reduced, increased airway resistance occurs. If smooth muscle tone is reduced through hyperventilation caused by HIT, a greater stretch in the airway could produce less resistance and ultimately increased expiratory flow rates.

There is little research on the effects of whole-body exercise training on pulmonary function or the respiratory muscles. However, HIT may lead to pulmonary adaptations not commonly seen with ET. To our knowledge, this theory has not been empirically addressed. The rationale behind this postulate comes from research on inspiratory muscle training (IMT) which has shown positive effects on respiratory muscle strength(Downey et al. [2007](#page-6-0); Enright et al. [2006\)](#page-6-0). IMT uses increased breathing strategies only to train the respiratory muscles with intensities from 50 to 80% PImax in most studies. The greatest improvements have been found at 80% intensity (Downey et al. [2007](#page-6-0); Enright et al. [2006](#page-6-0); Gething et al. [2004](#page-7-0); Witt et al. [2007\)](#page-7-0). HIT, theoretically, is able to elicit hyperventilation from the subjects which should in turn increase respiratory muscle strength more than ET. Therefore, the purpose of our study was to determine the effects of HIT on the pulmonary system. We hypothesize that high-intensity interval training will increase respiratory muscle strength and expiratory flow rates.

Methods

Subjects

Twenty healthy men $(n = 11)$ and women $(n = 9)$ volunteered to participate. All subjects were active (but were not trained or regularly participating in competitive sports), free of heart and pulmonary disease, and non-smokers. After being informed of the risks, subjects completed a medical history questionnaire and signed an informed consent waiver. During the course of the study, five subjects dropped out due to schedule conflicts ($n = 2$), injuries $(n = 2)$, and illness $(n = 1)$. Fifteen subjects completed the protocol. Subjects maintained their normal activity level

throughout the training period. All procedures were approved by the Institutional Review Board at Kansas State University, Manhattan KS.

Protocol

Each subject reported to the lab three times prior to training, 12 times for training sessions, and twice post training. During the first visit, measurements included height, body mass, waist circumference, body mass index, and a five-mile time trial on a cycle ergometer. During the second visit, pulmonary function tests (PFT) and an incremental maximum oxygen uptake test $(VO₂max)$ were performed. 1–2 days later, subjects came to the lab to perform a second five-mile time trial to establish a baseline for performance measures. Subjects then completed 4 weeks of training after being randomly assigned to either a high-intensity interval training group (HIT: $n = 8$) or endurance group (ET: $n = 7$). After training was completed on the second to last visit, PFTs, body mass, body mass index, and a five-mile time trial were performed. On the last visit the $VO₂max$ test was repeated.

Exercise testing

The incremental maximal oxygen uptake test was performed on a cycle ergometer (Sensormedics 800). Metabolic and ventilatory data were collected and analyzed continuously breath-by-breath throughout exercise (Sensormedics 229 Metabolic Cart, Sensormedics Corp., YorbaLinda, CA, USA). A pulse oximeter (Datex-Ohmeda 3900P, Madison, WI, USA) was used to estimate arterial oxygen saturation $(SpO₂)$. Heart rate (HR) was continuously monitored via a four-lead ECG connected to the metabolic cart. Values from the modified Borg's rating of perceived exertion scale (RPE), measured 1–10, were recorded for each stage of exercise. After 4 min of rest, warm up exercise commenced at 20 W for another 4 min with a cadence of 60–70 rpm. Every minute beyond the warm up period the workload was increased by 25 W. Termination occurred when the subject was unable to maintain the pedal cadence of 60–70 rpm for five consecutive revolutions. Criteria for a successful test included an $RER \ge 1.10$, heart rate (HR) within 10% of predicted HR_{max} and/or a plateau in oxygen consumption (<150) ml/min) with an increase in workload.

Five-mile time trials were completed on an electronically braked cycle ergometer (Vision Fitness HRT E3600, Cottage Grove, WI, USA) at least twice pre training, (to familiarize the subject with the test and establish consistency in baseline measurements) after 2 weeks of training, and once posttraining. A HR monitor (Polar FS3C, Lake Success, NY, USA) was worn and HR was recorded every minute. The seat height was recorded and remained constant for all trials. Warm-up time, pedal cadence, and workload were determined by the subjects. Subjects were blinded to distance and given verbal cues each mile during the test. At every mile the time was recorded. All subjects were encouraged to complete the time trial as fast as possible.

Pulmonary function tests

The maximum flow volume loop (Sensormedics 229 Metabolic Cart, Sensormedics Corp.) was used to determine peak expiratory flow (PEF), forced expiratory flow between 25 and 75% of vital capacity (FEF_{25–75}), forced expiratory volume at 1 s (FEV_1) , forced vital capacity (FVC), and the ratio of $FEV₁/FVC$. Inspiratory (PImax) and expiratory maximum pressure (PEmax) tests were used to measure respiratory muscle strength. PImax was measured from residual volume and PEmax was measured from total lung capactiy. All PFTs were performed a minimum of three times, with the average of the three closest values used for analysis.

Training

Endurance training subjects trained on an electronically braked cycle ergometer (Sensormedics 800) 3 days/week, alternating days, for 4 weeks. Seat height was determined by the subject and recorded to remain consistent throughout training. Changes in respiratory muscle strength with inspiratory muscle training have been reported to occur in this length of time (Downey et al. [2007](#page-6-0)). Training began with a 5-min warm-up period at 20 W. Workload was determined from their incremental $VO₂$ max test at 60–70% of each subject's final workload. A training session consisted of 45 min of constant load cycling. Pedal cadence was maintained between 50 and 80 rpm. HR was recorded each minute for the warm-up period and every 5 min during the training session.

High-intensity interval training subjects also trained on an electronically braked cycle ergometer (Sensormedics 800) 3 days/week, alternating days, for 4 weeks. Seat height was determined by the subject and recorded to remain consistent throughout training. Each individual

training session began with a 3-min warm-up at 20 W. After the warm-up period, the subjects performed a 1-min bout at a workload eliciting 90% of the final workload the subjects were cycling at from their $VO₂$ max test. Pedal rate was maintained between 60 and 100 rpm. When the interval was completed, subjects pedaled for a 3-min recovery period at 20 W. This was repeated a total of five times in one session totaling 20 min, excluding the warmup period. HR was recorded every minute of the warm-up and exercise. Workload values remained constant for the 4-week training period. Maximum flow volume loops, PImax,, and PEmax tests were performed at the end of each week of training for both groups.

Statistical analysis

SigmaStat statistical software (Jandel Scientific Software) was used for data analysis. Data are expressed as mean \pm standard deviation. A 2 \times 2 (group vs. time) mixed ANOVA was used to determine differences. Tukey post hoc tests were used to determine significant interaction effects. Significance was set at $P < 0.05$ for all analyses.

Results

Subjects

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

Training

Training adherence was 98% for all subjects across all training sessions. The total work per training session for ET was $408.86 \pm 109.04 \text{ kJ}$ and for HIT was $86.06 \pm 109.04 \text{ kJ}$

Table 1 Subject characteristics in the subject characteristics in t

13.44 kJ (including recovery) ($P \lt 0.05$). Average heart rate for both groups during training is shown in Fig. 1. HR values were significantly higher for HIT compared with ET. In the ET group, HR averaged $77.0 \pm 3.3\%$ of HR max, while the workload ranged from 110 to 225 W. For HIT, HR averaged $84.9 \pm 0.5\%$ of HRmax, while the workload ranged from 130 to 265 W. Average HR during the training sessions also significantly decreased from weeks 1 and 2 to weeks 3 and 4 with ET.

Pulmonary function tests

Table 2 shows ET and HIT pulmonary function values pre and post training. Groups were well matched prior to training with no significant differences between groups. Following training, there were no differences ($P > 0.05$) for any measured variable or between groups.

Respiratory muscle strength

Weekly PImax values are shown in Fig. [2.](#page-4-0) There were no differences ($P > 0.05$) between groups prior to training. Both groups significantly increased PImax with training

Fig. 1 Heart rate during training. Mean heart rate for HIT and ET during each week of training. Heart rate was significantly higher during HIT than ET during each week of training. *Significantly different from ET; $+$ Significantly different from weeks 1 and 2 of ET; $P < 0.05$

(ET \sim 25%; HIT \sim 43%), with values significantly higher for HIT than ET following 4 weeks of exercise training. Figure [3](#page-4-0)a and b shows individual PImax values pre and post training for ET and HIT, respectively. All subjects in both the ET and HIT group increased PImax with training. PEmax was not different $(P > 0.05)$ between groups prior to training. There was also no difference ($P > 0.05$) in PEmax post training or between groups (ET: 131.0 ± 20.4 cmH₂O, HIT: 123.1 ± 44.8 $cmH₂O$).

VO₂max data

Data recorded during the $VO₂max$ test are shown in Table [3](#page-5-0). Following training, both groups significantly increased VO_2 max by $\sim 8-10\%$ with no difference $(P > 0.05)$ between groups. Post training, there were no differences ($P > 0.05$) between groups on any measure.

Time trials

Figure [4](#page-5-0) shows the pre and post training time trial values for ET and HIT. There was no difference $(P > 0.05)$ between groups prior to training $(1,072 \pm 198 \text{ s for ET})$ and $1,089 \pm 136$ s for HIT) or after 2 weeks of training. Following 4 weeks of training, both groups significantly decreased time to completion of the five miles to $1,024 \pm 177$ s in endurance ($\sim 4.4\%$) and $1,003 \pm 104$ s in HIT (\sim 6.5%) with no difference (P > 0.05) between ET and HIT.

Discussion

The major findings of this study demonstrated significant increases in inspiratory muscle strength with significantly greater gains shown by the HIT over the ET. However, in disagreement with our hypothesis, no differences were observed in expiratory flow rates with training or between groups. Interestingly, similar increases in $VO₂max$ and decreases in the time to completion of time trials were found in both groups despite the fact that the total amount of work per training session was significantly less for HIT.

Fig. 2 Maximal inspiratory pressure (PImax) before and after each week of training. PImax was significantly increased from baseline and significantly higher with HIT ($P < 0.05$). HIT increased $\sim 43\%$ by week 4 and ET increased \sim 25%. *Significantly different between groups; $P \lt 0.05$, ⁺Significantly different from pre; $P \lt 0.05$

Respiratory muscle strength

Our findings have demonstrated that 4 weeks of either HIT or ET resulted in \sim 25–43% increases in inspiratory muscle strength. While no studies to our knowledge have directly assessed the effects of whole-body exercise training on respiratory muscle strength, the improvements in inspiratory muscle strength that we have demonstrated are similar to studies utilizing inspiratory muscle training (IMT). IMT typically uses high ventilation rates and generates large pressures that are similar to those experienced during HIT. Research suggests that most inspiratory muscle strength gains occur when intensity is at or above 60–80% of maximum inspiratory pressure (PImax) (Enright et al. [2006](#page-6-0)). Using IMT, one study observed an increase in inspiratory muscle strength of \sim 41% in healthy subjects (Enright et al. [2006\)](#page-6-0). Other studies using healthy subjects found smaller increases in inspiratory muscle strength of \sim 7–17% (Downey et al. [2007](#page-6-0); Gething et al. [2004;](#page-7-0) Holm et al. [2004;](#page-7-0) Witt et al. [2007\)](#page-7-0). The difference in these studies appears to be the intensity at which the IMT was performed, varying from 50 to 80%, with the largest increases observed at about 80% (Downey et al. [2007](#page-6-0); Enright et al. [2006;](#page-6-0) Gething et al. [2004;](#page-7-0) Witt et al. [2007](#page-7-0)). Effects observed with high-intensity IMT are consistent with those observed in this study, demonstrating that the stimulus for inducing respiratory muscle adaptations requires high-intensity work. This suggests that the greater inspiratory muscle strength we observed with HIT compared with ET was due to greater demand placed on the respiratory muscles with HIT. We believe this is the first study to demonstrate substantially greater increases in inspiratory muscle strength with HIT.

Fig. 3 a Individual and mean data pre and post training for the ET group. The open circles are the mean for the ET group pre and post training. The filled circles are the individual subject data. *Significant at $P < 0.05$. **b** Individual and mean data pre and post training for the HIT group. The open circles are the mean for the HIT group pre and post training. The filled circles are the individual subject data. *Significant at $P < 0.05$

Expiratory flow rates

We were mildly surprised that expiratory flow rates did not change with HIT. While there has been no direct evidence of increased expiratory flow rates with whole-body exercise training, research has suggested that repeated lung inflation-induced airway stretch may reduce airway resistance through decreased smooth muscle tone and contractility (Scichilone et al. [2005\)](#page-7-0). A reduction in resistance could also cause a greater dilation in the airways. However, despite large amounts of airway stretch, lung inflation, and high ventilation rates that are associated with HIT, it apparently was not sufficient to lead to expansion of the maximal flow volume loop or an increase expiratory flow rates. Also, it is possible that 4 weeks is not a sufficient

Table 3 VO₂max data

Values are mean \pm SD RER respiratory exchange ratio, S_pO_2 arterial oxygen saturation * Significantly different from pre-training at $P < 0.05$

Fig. 4 Five-mile time trial in seconds to completion from pre training, after 2 weeks, and after 4 weeks. Time to completion was significantly different at 4 weeks than pre training and 2 weeks. There was no difference between groups ($P < 0.05$)

length of time to induce these adaptations. Our results are consistent with prior research which has found unaltered airway resistance with an increase in inspiratory muscle strength, endurance, and lung volumes in endurance athletes (Guenette et al. [2009](#page-7-0)).

Work and training volume

Similar physiological adaptations have been reported to occur in metabolic and cardiovascular measures with ET and HIT, as well as similar improvements in performance, which agrees with our study (Burgomaster et al. [2008](#page-6-0); Gibala et al. [2006](#page-7-0); Krustrup et al. [2004;](#page-7-0) Rakobowchuk et al. [2008](#page-7-0)). In these previous reports, similar increases were observed with $VO₂max (~3-4 ml/kg min⁻¹), time$ trials (\sim 4–10%), and power output (\sim 7–17%). Surprisingly, these changes occurred despite the volume of training performed by ET being much greater than HIT. In these studies, the amount of work ranged from 225 to 315 kJ/week for HIT and 2,250–3,250 kJ/week for ET, which is similar to our study. These reports also showed increases in mean power output, and the time to the onset of fatigue with no differences between ET and HIT (Burgomaster et al. [2006](#page-6-0), [2008](#page-6-0); Gibala et al. [2006](#page-7-0); Mogensen et al. [2006;](#page-7-0) Rakobowchuk et al. [2008\)](#page-7-0). Researchers have also found a decrease in the time to completion of time trials and a reduction in HR at a given workload and resting HR (Burgomaster et al. [2008](#page-6-0); Gibala et al. [2006](#page-7-0)). We found the reduction in HR at a given workload to be true for the ET group in our study as well. Yet, the vast difference in volume and work elicited similar changes $(VO₂)$ max, time trials) over only a 4-week training program. Our study, therefore, agrees with and provides additional support to many studies that have utilized similar protocols with similar training volume differences (Burgomaster et al. [2006](#page-6-0), [2008;](#page-6-0) Gibala et al. [2006](#page-7-0); Rakobowchuk et al. [2008](#page-7-0)).

Implications

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

As exercise intensity or duration increases, so does the demand for blood flow to all working muscles due to an increased demand for oxygen (Dempsey et al. [2006](#page-6-0); Enright et al. [2006](#page-6-0); Witt et al. [2007](#page-7-0)). Expiration becomes active which requires larger muscle recruitment, and a larger amount of oxygen demanding an increase in blood flow (Babcock et al. [2002;](#page-6-0) Johnson et al. [1993](#page-7-0); Witt et al. [2007](#page-7-0)). Evidence suggests that the demand for an increase in blood flow to the respiratory muscles during maximal or heavy exercise "steals" blood flow from the other exercising muscles through sympathetically mediated vasoconstriction (Harms et al. [1998;](#page-7-0) St. Croix et al. [2000](#page-7-0)). As metabolites accumulate due to this vasoconstriction, the

muscle metaboreflex is activated to restore blood flow. It is thought that the need for increased blood flow to the diaphragm is the potential reason for vasoconstriction to the working muscles (Dempsey et al. 2006; Enright et al. 2006; Harms et al. [1998\)](#page-7-0). During high-intensity exercise (i.e. $>90\%$ VO₂max), proportional assist ventilation, reducing the work of breathing, has been shown to help prevent or delay diaphragmatic fatigue and increase performance (Harms et al. [2000](#page-7-0)). This suggests that respiratory muscle fatigue can be reduced with exercise training possibly resulting in an improved performance.

Our study demonstrated improvements in performance through decreased time to completion of time trials. However, the magnitude of decrease was not significantly different between groups. While theories discussed above would suggest that increases in performance would be larger with HIT, it is possible that the main source of adaptation is simply exercise training, regardless of the type of training. This theory is supported by Iaia et al. [\(2009](#page-7-0)) who investigated endurance-trained runners and divided them into two groups, one that performed moderate intensity exercise and the other implementing a speed endurance training program. Improvements in performance, as measured through time trials, were similar between groups. Additionally, in our study we did not see a difference between groups in $VO₂$ max post training. Thus, it is not unexpected that time trials were not different between groups.

Limitations

There are several potential limitations that we have identified which could have affected our results. One limitation is the pedal rates during training. While most subjects pedaled within the same range (ET 45–80 rpm, HIT 60–100 rpm) each subject was free to pedal at their preferred rate. Previous reports have shown that power output and endurance exercise time increased when pedal rates were freely chosen or were slightly below the freely chosen rate by trained and untrained subjects (Nielsen et al. [2004](#page-7-0)). When pedal rate was slightly above the freely chosen rate, the endurance capacity was reduced (Nielsen et al. [2004](#page-7-0)). In our study all subjects were supervised for the entirety of each training session and were encouraged to work at the same intensity throughout the session. Subject effort was consistent as evidenced by the small range of HR deviation throughout each session. Second, the use of maximal inspiratory and expiratory pressure tests indirectly estimates respiratory muscle strength. While these are commonly and widely accepted methods for measuring respiratory muscle strength, these tests are effort dependent. However, multiple tests were performed prior to, during, and after training by the same investigator to ensure consistency. Therefore, due to the similarity in obtained values within each session, we believe these values are accurate. Third, we did not use a measure of respiratory muscle fatigue in our study. Respiratory muscle fatigue may be an important component of performance. A measure of respiratory muscle fatigue would have helped in part to determine a mechanistic basis for improvements in performance with training and should be measured in future studies.

Conclusions

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

References

- Babcock MA, Pegelow DF, Harms CA, Dempsey JA (2002) Effects of respiratory muscle unloading on exercise-induced diaphragm fatigue. J Appl Physiol 93(1):201–206
- Burgomaster KA, Heigenhauser GJ, Gibala MJ (2006) Effect of shortterm sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. J Appl Physiol 100(6):2041–2047
- Burgomaster KA, Howarth KR, Phillips SM, Rakobowchuk M, MacDonald MJ, McGee SL et al (2008) Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. J Physiol 586(1):151–160
- Coyle EF (2005) Very intense exercise-training is extremely potent and time efficient: a reminder. J Appl Physiol 98(6):1983–1984
- Dempsey JA, Romer L, Rodman J, Miller J, Smith C (2006) Consequences of exercise-induced respiratory muscle work. Respir Physiol Neurobiol 151(2–3):242–250
- Downey AE, Chenoweth LM, Townsend DK, Ranum JD, Ferguson CS, Harms CA (2007) Effects of inspiratory muscle training on exercise responses in normoxia and hypoxia. Respir Physiol Neurobiol 156(2007):137–146
- Eastwood PR, Hillman DR, Finucane KE (2001) Inspiratory muscle performance in endurance athletes and sedentary subjects. Respirology 6(2):95–104
- Enright SJ, Unnithan VB, Heward C, Withnall L, Davies DH (2006) Effect of high-intensity inspiratory muscle training on lung

volumes, diaphragm thickness, and exercise capacity in subjects who are healthy. Phys Ther $86(3):345-354$

- Gething AD, Williams M, Davies B (2004) Inspiratory resistive loading improves cycling capacity: a placebo controlled trial. Br J Sports Med 38(6):730–736
- Gibala MJ, McGee SL (2008) Metabolic adaptations to short-term high-intensity interval training: a little pain for a lot of gain? Exerc Sport Sci Rev 36(2):58–63
- Gibala MJ, Little JP, van Essen M, Wilkin GP, Burgomaster KA, Safdar A et al (2006) Short-term sprint interval versus traditional endurance training: Similar initial adaptations in human skeletal muscle and exercise performance. J Physiol 575(Pt 3):901–911
- Guenette JA, Querido JS, Eves ND, Chua R, Sheel AW (2009) Sex differences in the resistive and elastic work of breathing during exercise in endurance-trained athletes. Am J Physiol Regul Integr Comp Physiol 297(1):R166–R175
- Harms CA, Wetter TJ, McClaran SR, Pegelow DF, Nickele GA, Nelson WB et al (1998) Effects of respiratory muscle work on cardiac output and its distribution during maximal exercise. J Appl Physiol 85(2):609–618
- Harms CA, Wetter TJ, St Croix CM, Pegelow DF, Dempsey JA (2000) Effects of respiratory muscle work on exercise performance. J Appl Physiol 89(1):131–138
- Harmer AR, McKenna MJ, Sutton JR, Snow RJ, Ruell PA, Booth J et al (2000) Skeletal muscle metabolic and ionic adaptations during intense exercise following sprint training in humans. J Appl Physiol (Bethesda, Md.: 1985) 89(5):1793–1803
- Hellsten Y, Sjodin B, Richter EA, Bangsbo J (1998) Urate uptake and lowered ATP levels in human muscle after high-intensity intermittent exercise. Am J Physiol 274(4 Pt 1):E600–E606
- Holm P, Sattler A, Fregosi RF (2004) Endurance training of respiratory muscles improves cycling performance in fit young cyclists. BMC Physiol 4:9
- Iaia FM, Hellsten Y, Nielsen JJ, Fernstrom M, Sahlin K, Bangsbo J (2009) Four weeks of speed endurance training reduces energy expenditure during exercise and maintains muscle oxidative capacity despite a reduction in training volume. J Appl Physiol 106(1):73–80
- Johnson BD, Babcock MA, Suman OE, Dempsey JA (1993) Exerciseinduced diaphragmatic fatigue in healthy humans. J Physiol 460:385–405
- Krustrup P, Hellsten Y, Bangsbo J (2004) Intense interval training enhances human skeletal muscle oxygen uptake in the initial phase of dynamic exercise at high but not at low intensities. J Appl Physiol 559(1):335–345
- MacDougall JD, Hicks AL, MacDonald JR, McKelvie RS, Green HJ, Smith KM (1998) Muscle performance and enzymatic adaptations to sprint interval training. J Appl Physiol (Bethesda, Md.: 1985) 84(6):2138–2142
- McKenna MJ, Heigenhauser GJ, McKelvie RS, Obminski G, MacDougall JD, Jones NL (1997) Enhanced pulmonary and active skeletal muscle gas exchange during intense exercise after sprint training in men. J Physiol 501(Pt 3):703–716
- Mogensen M, Bagger M, Pedersen PK, Fernstrom M, Sahlin K (2006) Cycling efficiency in humans is related to low UCP3 content and to type I fibres but not to mitochondrial efficiency. J Physiol 571(Pt 3):669–681
- Nielsen JS, Hansen EA, Sjogaard G (2004) Pedalling rate affects endurance performance during high-intensity cycling. Eur J Physiol 92(1-2):114–120
- Parra J, Cadefau JA, Rodas G, Amigo N, Cusso R (2000) The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle. Acta Physiol Scand 169(2):157–165
- Rakobowchuk M, Tanguay S, Burgomaster KA, Howarth KR, Gibala MJ, MacDonald MJ (2008) Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow-mediated dilation in healthy humans. Am J Physiol Regul Integr Comp Physiol 295(1):R236–R242
- Scichilone N, Morici G, Marchese R, Bonanno A, Profita M, Togias A, Bonsignore MR (2005) Reduced airway responsiveness in nonelite runners. Med Sci Sports Exerc 37(12):2019–2025
- St Croix CM, Morgan BJ, Wetter TJ, Dempsey JA (2000) Fatiguing inspiratory muscle work causes reflex sympathetic activation in humans. J Physiol 529(Pt 2):493–504
- Witt JD, Guenette JA, Rupert JL, McKenzie DC, Sheel AW (2007) Inspiratory muscle training attenuates the human respiratory muscle metaboreflex. J Physiol 584(Pt 3):1019–1028