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**THE EFFECTS OF RESPIRATORY MUSCLE TRAINING ON VO₂ MAX, THE
VENTILATORY THRESHOLD AND PULMONARY FUNCTION**

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ABSTRACT

THE EFFECTS OF RESPIRATORY MUSCLE TRAINING ON VO₂ MAX, THE VENTILATORY THRESHOLD AND PULMONARY FUNCTION. **William E. Amonette, Terry L. Dupler. JEPonline.** 2002;5(2):29-35. This study evaluated the effect of inspiratory and expiratory muscle training on pulmonary function and maximal exercise performance in competitive triathletes and marathon runners. The participants in this study (N=12) had a mean weekly aerobic training time of 7.5 hours per week of swimming, cycling, or running. Eight subjects were assigned to a pulmonary resistance treatment group and four control subjects were given a sham device that allowed no greater than 15% resistance on inspiration or expiration. The subjects performed 30 maximal inhalation/exhalation maneuvers on their respective devices two times per day for four weeks. The subjects were tested for forced vital capacity (FVC), forced expiratory volume in one second (FEV₁), FEV₁/FVC ratio, forced inspiratory vital capacity (FIVC), peak inspiratory flow rate (PIFR), and peak expiratory flow rate (PEFR). Each subject was also tested for peak exhalation force (P_{EF}) as well as a maximal oxygen consumption (VO₂ max), carbon dioxide production (VCO₂), tidal volume (V_T), ventilation (V_E), ventilatory threshold (VT), and respiration rate (RR). The data revealed that training using the pulmonary resistance device produced significant increases in maximal V_E and maximal V_T while decreasing RR (although not statistically significant) at maximum exercise. However, no significant changes were seen in VO₂ or any pulmonary function variables measured.

Key Words: Lungs, Resistance, Breathing, Triathletes

INTRODUCTION

The effects of resistance training on skeletal muscle are well documented (1). When performed with the correct repetition scheme and load assignment, resistance training can produce skeletal muscle hypertrophy, strength, or local muscle endurance. Traditionally, strength and power athletes have used resistance training to augment sport performance while endurance athletes have avoided resistance exercise believing that increased muscle would decrease aerobic performance. However, skeletal muscle also controls many crucial elements of aerobic

conditioning including lung ventilation. The diaphragm, external and internal intercostals, scalene, and abdominal muscles (i.e. respiratory muscles) help to facilitate the increased ventilation needed to sustain blood oxygenation during exercise (2). If these muscles play such a crucial role in exercise, logically one would think we should train them just as if we would any other skeletal muscle.

Devices have been manufactured that offer resistance on inhalation and exhalation and thus cause an increased strain on the respiratory muscles. This resistance has been shown to facilitate positive pulmonary function changes in people with chronic obstructed pulmonary disease (COPD). Villafranca et al. (3) showed increases in maximal inspiratory pressure (P_Imax) after ten weeks of inspiratory muscle training using a threshold inspiratory trainer allowing 30% resistance. Likewise, Larson and Kim (4) observed increases in P_Imax after one month of inspiratory muscle training in people with COPD.

Pulmonary resistance training has also been shown to change enzymatic profiles in sheep. Akabas et al.(5) assigned nine adult sheep to a training group and seven to a control group. The experimental group trained for twenty minutes using inspiratory flow resistance (50-100 cmH₂O) five to six times per week for three weeks. In the end, biochemical changes were observed between the experimental and control group. Compared to the control group, the experimental group diaphragm muscle had a 26% increase in citrate synthase (CS), a 29% increase in β -hydroxyacyl-CoA dehydrogenase (β HAD), and a 36% increase in cytochrome oxidase (COX). It can be concluded from this study that the aerobic enzymatic profiles increased significantly in sheep when put under inspiratory stress. An increase in aerobic enzymes during exercise in humans would equate to more efficient energy utilization of the respiratory muscles and lower fatigueability.

Data involving healthy humans using respiratory muscle training with exercise is limited. In two separate studies, Suzuki et al. (6,7) observed changes in the rate of perceived exertion (RPE) after inspiratory and expiratory muscle training in healthy adults. Suzuki concluded that expiratory muscle training did not decrease RPE at a given work load while inspiratory muscle training did decrease RPE.

Data involving athletes using respiratory muscle training is almost non-existent. It is recognized that the respiratory muscles will adapt to aerobic training (9,10). Most aerobic athletes have very well trained respiratory muscles from their sport alone. However, it is not known if additional respiratory muscle training could elicit positive adaptations within the aerobically trained athlete that would make the ventilatory process more efficient. During competition athletes will take thousands of breaths. Like all other skeletal muscles, the pulmonary muscles when engaging in aerobic metabolism require oxygen. The fatigue resistance of this process is related to the training status of the muscle. If the muscle is more endurance trained, then it will be less likely to constrain ventilation and exercise performance.

The purpose of our research was to investigate the effects of respiratory muscle training using a Powerlung device on pulmonary function and exercise performance in triathletes and marathon runners. We hypothesized that the treatment group will increase pulmonary function during exercise and increase dynamic lung functions. We did not believe that the treatment group or control group would increase VO₂ max or the ventilatory threshold.

METHODS

Subjects

A total of twelve subjects (9 male, 3 female) were recruited from area triathlon and running clubs. The subjects had a mean aerobic training time of 7.5 hrs/week (range; 5-10 hrs). Each subject was informed of the risks and benefits of the study, made aware of their right to withdraw from the study at any time, and then signed an institutional approved consent form. Eight subjects (6 male; 2 female) were assigned to a Powerlung treatment

group (PLT) and four subjects (3 male; 1 female) were assigned to a control group (CON). The experimental and control groups physical characteristics are presented in Table 1.

Table 1: Mean±standard deviation (SD) data of group characteristics

Group	N	Age y(rs)	Ht (cm)	Wt (Kg)
<i>PLT</i>	8	36.75±8.83	174.5±9.41	72.94±7.22
<i>CON</i>	4	34.75±7.05	172.8±4.22	71.23±3.64

Maximal Exercise Testing

Each subject was given a treadmill maximal test before and after Powerlung training. The VO_2max protocol consisted of 1.5 min stages of work, each made successively more difficult by increasing the grade by 1%. The testing speed was constant, and was selected based on the 5 km time of the subjects. The protocol had a 2-stage ramp-up to the target speed to ease the transition to the test speed. In addition to the 2-stage ramp-up built into the protocol, the subject was allowed to warm-up on the treadmill as long as needed before beginning the test. The protocol continued to increase by 1% grade until the subject reached maximal exertion (i.e. asked for the treadmill to be stopped or grabbed the handrail). Metabolic data was acquired using a Quinton metabolic cart (Model QMC). The QMC sampled from a mixing chamber every 15 seconds and provided oxygen consumption (VO_2), ventilation (V_E), respiratory rate (RR), volume of carbon dioxide produced (VCO_2), and ventilatory threshold (VT). Ventilatory threshold was derived via the v-slope method by the Quinton metabolic software version 3.3 (8). Heart rate (HR) was measured using a five lead electrocardiogram from a Quinton Q-4500 stress-test system.

Pulmonary Function Testing

Each subject was given a spirometry test using a ChestTest spirometer (Vaccumed, Ventura, CA) before the VO_2max test both pre- and post-training. Each subject stood facing the spirometer and performed a maximal inhalation followed by a forceful exhalation into the tube until all air was expelled. The subject then performed a maximal inhalation to complete the maneuver. The ChestTest spirometer provided forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), FEV_1/FVC ratio, Peak expiratory flow rate (PEFR), and peak inspiratory flow rate (PIFR).

Each subject also performed a peak exhalation force (P_{EF}) test to determine the strength of the expiratory muscles. Force was measured using a standing mercury sphygmomanometer. The subject performed a maximal inhalation then exhaled forcefully against a closed valve. Maximal force in mmHg was recorded over a maximum of three trials. The P_{EX} test was performed pre- and post-training after the spirometry test but before the VO_2max test. Test-retest reliability was performed on the apparatus using 8 graduate students ($r=0.92$).

Training

For the duration of the study, the subjects were asked to maintain their present aerobic training and not to increase or decrease their training time in order to eliminate the possibility of adaptations from aerobic exercise (9,10). Both groups performed thirty maximal inhalation/exhalation cycles on the Powerlung training device two times per day for four weeks. The device allows for varying resistance on inhalation and exhalation via hand adjusted knobs. After the maximal tests, the treatment group was given the Powerlung device and instructed to perform the above-mentioned protocol as assigned by Powerlung manufacturers. The subjects were instructed to adjust the resistance to allow for the 30th breath on both inhalation and exhalation to be close to a maximal effort (i.e. 30 RM). The subjects controlled the resistance via trial and error.

In the same way, the subjects assigned to the control group were given a sham device after their maximal tests. The sham device looked identical to the Powerlung device, but the chambers inside the sham device were constructed to allow a resistance no greater than 15%. The control subjects were given the same instructions as

the experimental group. Neither group was informed as to which group they were assigned to until the completion of their last maximal test.

Statistical Analyses

All data was analyzed using analysis of variance (ANOVA) with repeated measures. The data was copied from a summary spreadsheet in Microsoft Excel to SPSS statistical software and the software derived all F-ratios. All significant F-ratios were analyzed using Tukey's post hoc test and p-values represent Greenhouse-Geisser adjustment for sphericity. Anthropometric data is expressed as mean±standard deviation (SD), while all exercising and pulmonary dependent variables are expressed as mean±standard error of the mean (SEM). Percent change was calculated using Microsoft Excel with the formula: % change = $[(x_2 - x_1) / x_1] * 100$.

RESULTS

Results are provided in Tables 2 and 3 for all maximal exercising and pulmonary function data, respectively.

Table 2: Descriptive statistics (mean±standard error of the mean (SEM)) for data at maximal exercise intensity.

<i>Variable</i>	<i>Pre Mean</i>	<i>Post Mean</i>	<i>% Change</i>	<i>P/b Value</i>
<i>PLT VO₂max (ml/kg/min)</i>	55.35±2.70	55.86±2.54	0.92%	β=.14
<i>CON VO₂max (ml/kg/min)</i>	53.45±3.95	52.53±3.85	-1.72%	P=0.36
<i>PLT V_Emax (L/min)</i>	138.16±7.37	142.71±8.29	3.29%	β=.57
<i>CON V_Emax (L/min)</i>	125.60±8.69	125.60±9.69	0.00%	P=0.04
<i>PLT V_Tmax (L/breath)</i>	3.03±0.16	3.84±0.26	26.73%	β=.55
<i>CON V_Tmax (L/breath)</i>	3.30±0.44	3.05±0.45	-7.58%	P=0.04
<i>PLT RRmax (breath/min)</i>	52.38±2.40	50.50±1.60	-3.59%	β=.84
<i>CON RRmax (breath/min)</i>	47.00±5.48	47.25±5.84	0.53%	P=0.57
<i>PLT VT (%VO₂max)</i>	77.13±7.38	74.50±1.54	-3.41%	β=.13
<i>CON VT (%VO₂max)</i>	73.25±5.65	75.00±2.35	2.39%	P=0.34

PLT=Treatment; CON=Control; VO₂max=Maximal oxygen consumption ; V_E=Ventilation in L/min; V_T=Tidal volume ; R=Respiratory rate ; VT=Ventilatory threshold.

DISCUSSION

Exercise Variables

The purpose of this study was to determine if pulmonary function or exercise performance could be changed by specifically training the respiratory muscles using a Powerlung resistance device. Respiration is only one component of aerobic capacity. Cardiac output is generally thought to be the major limiting factor to aerobic exercise (8). At maximal exercise, the ventilation to perfusion ratio reaches 4.0, indicating that we are ventilating four times the volume of air then is provided to the alveoli by pulmonary blood flow (10). Research shows runners with the lower ventilation responses to high exercise are both hypoxemic and more acidotic (11). This could be attributed to an inadequate hyperventilation, reducing CO₂ exhalation and the removal of blood protons.

In a study by Boutellier et al. (2) on trained aerobic athletes using a respiratory resistance device different from the Powerlung, the researchers found no significant increases in either VO₂max or LT during a cycle ergometer test. However, they did see an increase of sub-maximal cycling time. Like Boutellier's group, we did not see an increase in VO₂ max or VT. The subjects that were tested in our study were highly trained aerobic athletes.

Significant changes in VO_2max with high caliber aerobic athletes are rarely seen in short durations, and are usually a function of high levels of aerobic training over a long period of time. For this reason we did not expect changes in VO_2 during a four- week study.

Table 3: Descriptive statistics (mean±standard error of the mean (SEM)) for pulmonary variables

<i>Variable</i>	<i>Pre Mean</i>	<i>Post Mean</i>	<i>% Change</i>	<i>P/b Value</i>
<i>PLT FVC (L)</i>	4.98±0.30	5.02±0.27	0.80%	$\beta= .06$
<i>CON FVC (L)</i>	4.69±0.34	4.67±0.39	-0.43%	P=0.77
<i>PLT FEV₁ (L)</i>	3.77±0.16	3.77±0.15	0.00%	$\beta= .06$
<i>CON FEV₁ (L)</i>	3.68±0.28	3.71±0.30	0.82%	P=0.74
<i>PLT P_{EF} (mmHg)</i>	121.0±7.78	149.25±9.97	23.35%	$\beta= .28$
<i>CON P_{EF} (mmHg)</i>	143±29.03	145.5±25.33	1.75%	P=0.16
<i>PLT Ratio</i>	0.77±0.00	0.76±0.00	-1.30%	$\beta= .13$
<i>CON Ratio</i>	0.79±0.00	0.8±0.00	1.27%	P=0.38
<i>PLT PEFR (L/s)</i>	9.38±0.56	9.35±0.53	-0.32%	$\beta= .10$
<i>CON PEFR (L/s)</i>	7.95±0.46	7.87±0.50	-1.01%	P=0.49
<i>PLT FIVC (L)</i>	4.52±0.30	4.51±0.23	-0.22%	$\beta= .07$
<i>CON FIVC (L)</i>	4.44±0.30	4.31±0.38	-2.93%	P=0.64

PLT=Treatment ; CON=Control ; FVC=Forced vital capacity ; FEV₁=Forced expiratory volume in one second ; P_{EF}=Peak exhalation force ; Ratio is FVC/FEV₁ ratio ; PEFR=Peak expiratory flow rate ; FIVC=Forced expiratory flow rate.

However, using highly conditioned aerobic athletes did create an ideal situation to control the test the Powerlung. Because our subjects had likely maximized the pulmonary adaptations elicited by aerobic conditioning alone, any changed observed in oxygen consumption or pulmonary function could be attributed to the Powerlung training.

We did expect to see changes in pulmonary function during exercise. Because the subjects had trained their pulmonary muscles, they were able to increase ventilation. The increase in V_E and decrease in RR in the training group indicated that the Powerlung device increased the strength of the respiratory muscles. The increased strength of the respiratory muscles allowed the subjects to perform more work (i.e. move more air) while breathing fewer times.

Earlier work by Hanel and Secher (12) showed similar results. These investigators studied inspiratory muscle training on 20 physical education students. The students trained using a device similar to the Powerlung, but only allowed resistance on inspiration. The students trained on the device for 10 min twice per day at a progressively increasing resistance for 27.5 days. VO_2max was measured via treadmill test pre- and post-training and revealed a 3 breath/min decrease at max exercise in the training group and no change in the control group. A small increase in VO_2 (~2 L/min) was observed in both the training and the control group from pre to post testing. Likewise, our study revealed changes in RR with a 1.88 breath/min decrease at max exercise (Table 2). However, Hanel and Secher (12) saw a 2 L/min decrease in the treatment group after 27.5 days of inspiratory muscle training where as we saw a 4.53 L/min increase in V_E post Powerlung training.

While increased pulmonary muscle function did not result in an increase in VO_2 , it could have benefit to longer duration exercise. The maximal treadmill test lasted on average 9-12 minutes. The test is designed to end in a relatively short period of time so that central and peripheral oxygen delivery are the limiting factors and not leg

fatigue. If there are to be any changes due to pulmonary resistance training, they might be more likely to occur over a longer period than 9-12 minutes. Over the duration of a marathon or triathlon, the athletes will take thousands of breaths. This equates to a large amount of air flow and related delivery of oxygen that must be distributed to the pulmonary muscles to facilitate the mechanics of ventilation. If RR can be decreased by 2 breaths/min as seen in our study, and V_E could increase or remain constant, an athlete would take significantly fewer breaths over the period of their competition. This O_2 that was being distributed to the pulmonary muscles could be used by other working muscles to increase or prolong exercising performance.

Pulmonary Variables

The spirometry readings did not change significantly with Powerlung training. O’Kroy and Coast (13) examined nineteen untrained students and randomly assigned them to either a control group, an exercising group, an inspiratory loading group, an expiratory loading group, or a hyperventilating group. The subjects trained four days/week, 20 min/day for four weeks with their assigned groups. The subjects training with inspiratory loading showed increases in MIP and MIF, while the expiratory loading group showed increases in maximal expiratory pressure (M_{EP}) and expiratory force (M_{EF}). The only other group showing significant changes was the hyperventilating group, which exhibited increased MEP, maximum voluntary ventilation (MVV), maximal sustained ventilation for 4 minutes (MSVV), and MIF. This research suggests resistance training offers some benefit to fatigue resistance in untrained students. The research also suggests that inspiratory muscles, like all other skeletal muscles, adapt according to the stress placed on them (2).

Tzelpis et al. (14,15) showed similar results in a study with nineteen untrained students using flow specific training. Tzelpis assigned the students to three specific groups: a low, medium, and high pressure groups. The high pressure group performed 30 maximal static inspiratory contractions against an occluded valve. The low pressure group performed of thirty maximal inspiratory contractions against an unoccluded valve, and the medium pressure group performed 30 maximal contractions through a 7 mm resistor tube. The results were specific to the type of training. The high pressure training groups had the highest increases in peak esophageal pressure (P_{ESmax}) and maximum inspiratory flow rate (V_{Imax}). Tzelpis et al. (14,15) concluded that the groups that trained using high pressure resistance had the greatest increases in P_{ESmax} while the groups training with higher flow rates` (i.e. low pressure group) had higher increases in V_{Imax} as compared to the other two higher pressure groups.

It is known that pulmonary muscles will adapt to aerobic exercise (9,10), so we would expect to see smaller changes in aerobic athletes as their pulmonary muscles are already developed. Hanel and Sechler (12) showed no change in FEV_1 , FVC, FEV_1/FVC , and peak expiratory flow rate after 27.5 days of training. In the same way, Boutellier et al. (2) did not find changes in either peak expiratory flow rate or FEV_1 .

Conclusion

Overall, it can be concluded from our research that the Powerlung device does elicit positive changes in the pulmonary muscles. The Powerlung device increased the strength of the respiratory muscles as seen by the increase in P_{EX} , V_E and V_T while decreasing RR at maximal exercise. The device did not produce a significant change in VO_{2max} . It could be deduced that a longer training period (i.e., 8-12 weeks) might reveal more significant changes. Research with intermediate level athletes might also produce larger changes.

When doing further research with highly trained athletes, one might also consider trying different training protocols. In all forms of resistance training, not only is the intensity increased, but the overall volume and duration is also varied. Perhaps training at a higher volume (i.e. more reps) might produce more favorable metabolic changes in the respiratory muscles.

As long as athletes and scientist strive to push the limits of human performance every avenue of training will be explored. Pulmonary resistance training with athletes is still relatively new, and has seen only minimal

research. As records continue to fall in the athletic world, research should continue on pulmonary resistance training along with all other aspects of exercise performance.

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