



**Manchester  
Metropolitan  
University**  
Institute of Sport

# MMU-Airofit collaboration

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Dr Christopher Morse, Institute of Sport, Manchester Metropolitan University

Dr Peter Turton, The University of Liverpool

Mr Cameron Stamp, Institute of Sport, Manchester Metropolitan University

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## 2 Executive summary

### 2.1 Aims

Efficacy testing was undertaken through collaboration with Airofit as part of a supervised MSc project within MMU institute of sport. Our aim was to determine whether 6 weeks of the proprietary “cross-fit” programme would improve diaphragm and lung function as determined through ultrasonography. As a secondary aim we undertook maximal exercise testing to determine whether there were any benefits to exercise performance or respiratory function during exercise. Our target was for completion in early September.

### 2.2 Recruitment

Of a target of 24 Participants, 19 completed Pre- and Post-intervention testing

### 2.3 Significant findings

There were significant benefits observed in “static” and “exercise” lung function measures following implementation of the Airofit intervention.

- Maximal inspiratory and expiratory pressures increased by 45% and 20%, respectively.
- An increase in diaphragm muscle thickness of about 30%.
- Power output per unit of oxygen improved by 7%, suggesting an increase in exercise efficiency.
- Greatest improvements in those with the weakest, thinnest diaphragms

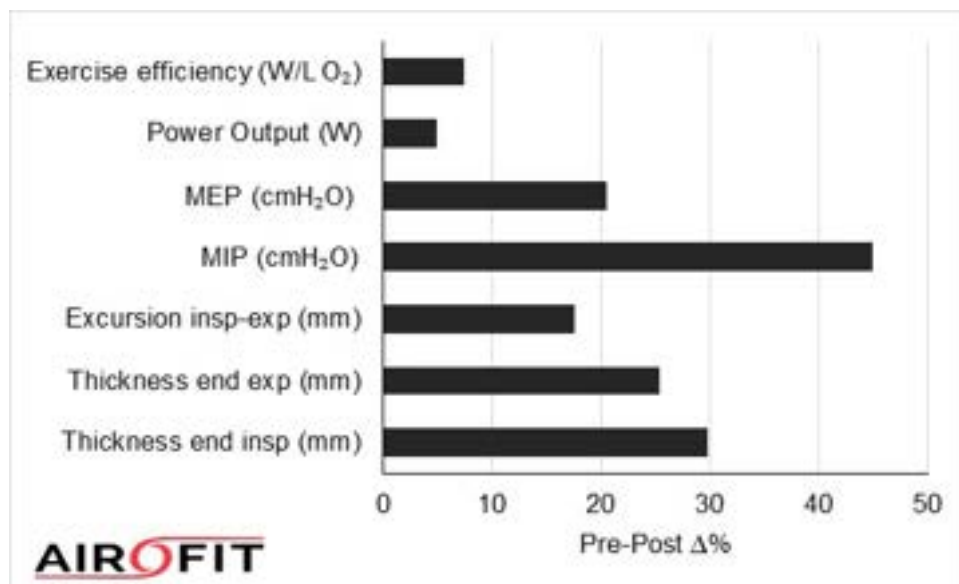


Figure 1 Significant outcomes of 6 weeks respiratory muscle training using the Airofit device.

### 3 Outcomes and results

#### 3.1 Participant Demographics

Table 1 Participant demographics and body composition

	PRE	POST	P value	Δ% <sup>1</sup>
Age	24.2 ± 7.8			
Height (cm)	174 ± 9			
Body mass (Kg)	74.1 ± 14.1	74.5 ± 12.9	0.51	0
Body fat (%)	20.7 ± 7.0	21.4 ± 6.6	0.43	3
Lean mass (%)	78.9 ± 7.0	78.6 ± 6.6	0.66	0
BMI	24.1 ± 2.4	24.5 ± 3.0	0.29	2
BMR (Kcal)	1830 ± 346	1727 ± 518	0.34	-6

Participant demographics for the  $n = 19$  who completed the pre and post testing. Data provided included body composition assessment through bioelectrical impedance including percentage body fat, lean mass, Body mass index (BMI) and Basal metabolic rate (BMR). There were no significant changes in demographics in response to the 6 week intervention. The P value denotes significance assessed through paired T-test or Wilcoxon test.

#### 3.2 Lung sonography

Table 2 Diaphragm and abdominal muscle outcomes determined from ultrasound.

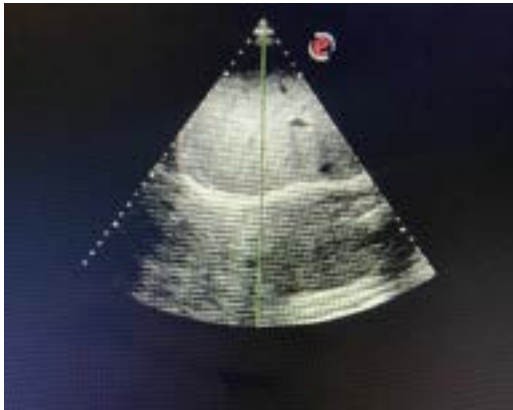
	PRE	POST	P value	Δ%
Thickness end insp (mm)	3.23 ± 1.11	4.19 ± 0.89	0.00	30
Thickness end exp (mm)	1.79 ± 0.39	2.24 ± 0.52	0.00	25
Dia thickening fraction Insp-Exp (%)	85.7 ± 69.8	92.0 ± 37.9	0.34	7
Excursion insp-exp (mm)	62.0 ± 29.1	72.9 ± 22.9	0.02	18
Ab thickness (mm)	11.8 ± 1.9	12.4 ± 2.5	0.09	5

The above data were collected through sonography using three main approaches. The first was to measure the thickness of the diaphragm in B-mode which is used for identifying anatomical structures, here we identify diaphragm thickness at the end of inspiration (End insp) and expiration (End exp) of a vital capacity breath. Both measures of thickness at end inspiration and expiration increased by 25-30%, representing significant hypertrophy of the diaphragm as a result of the 6-week intervention. In the second sonography method, we used M-mode ultrasound to monitor the excursion of the diaphragm during a series of vital capacity breaths. Here we saw significant increases in diaphragm excursion from inspiration to expiration, and in each respiratory phase. Finally, to determine whether there were any benefits to the accessory muscles, we measured abdominal muscle thickness, showing no change from Pre- to Post intervention. Abdominal muscle thickness was however strongly correlated with both FEV1 ( $r=0.641$ ,  $p=0.002$ ) and FVC ( $r=0.691$ ,  $p=0.001$ ) pre training, with similar significant observations made post training. This would fit with spirometric measurements being forced expiratory manoeuvres, dependent on expiratory muscle usage.

<sup>1</sup> Δ is used to denote a change from pre to post, here presented as a % of the starting value.

Maximal diaphragmatic excursion was significantly increased, with the diaphragm descending a further 1.9cm on average after training. In keeping with other known data, there was a moderate correlation between diaphragm excursion and forced vital capacity, in both pre ( $r=0.556$ ,  $p=0.017$ ) and post training ( $r=0.616$ ,  $p=0.007$ ). However, the increase in diaphragm excursion was not accompanied by an increase in indices of lung volume. This does however fit with previous data on purely inspiratory-only muscle training, where increases in MIP were not accompanied by changes in lung volume.

There were some interesting associations within our measures such that those individuals with the thinnest diaphragms showed the greatest development during the intervention ( $R^2 = 0.58$ ,  $P<0.001$ ). This is notable as it suggests, as with skeletal muscle that there is a greater potential for development in those with the thinnest diaphragm muscle. Similarly, there was an inverse relationship between diaphragm excursion pre training, and the percentage change in excursion post training ( $r=-0.609$ ,  $p=0.006$ ). This suggests that people who started the study generating smaller depths of diaphragm movement on inspiration showed the greatest increases after training.



*Figure 2 diaphragm excursion recorded using ultrasonography (left), maximal exercise testing for determining  $VO_2max$  (right)*

### 3.3 Lung function

*Table 3 Lung function and muscle strength outcomes*

	PRE	POST	P value	Δ%
<b>MIP (cmH<sub>2</sub>O)</b>	87 ± 31	127 ± 34	0.00	45
<b>MEP (cmH<sub>2</sub>O)</b>	98.4 ± 29.0	118.6 ± 29.8	0.00	20
<b>FEV<sub>1</sub>max (L)</b>	3.82 ± 0.92	3.78 ± 0.97	0.77	-1
<b>FVC (L)</b>	4.87 ± 1.10	4.68 ± 1.10	0.06	-4
<b>FEV<sub>1</sub>/FVC</b>	0.79 ± 0.11	0.81 ± 0.12	0.24	3
<b>Mid thigh pull max (Kg)</b>	137 ± 42	138 ± 36	0.12	0

The above data represent the improvements to “static” lung function and muscle strength. Maximal inspiratory and expiratory pressure showed significant improvements, with 45% and 20% increases in the maximal strength of maximal inspiratory and expiratory force. As these improvements were not seen in the volumes of the lung (Forced expiratory volume in 1s, or forced vital capacity) it can be considered with the data in table 2 above to correspond to an increase in strength of the respiratory muscles.

At baseline (PRE), inspiratory muscle thickness (i.e. diaphragm) was positively correlated with both MIP ( $r=0.641$ ,  $p=0.002$ ) and MEP ( $r=0.559$ ,  $p=0.008$ ), fitting with known data on the relationship between diaphragm thickness and strength.

The finding that MEP increased *without* an increase in expiratory muscle thickness (e.g. the abdominal muscle) can be explained in a number of ways. First, several studies looking at inspiratory-only muscle training have demonstrated increases in MEP. Second, the abdominal muscle thickness was measured at rest. *Rectus abdominis* thickness changes through the respiratory cycle, and also increases during active contraction, further measurement throughout the respiratory cycle may show changes in recruitment or adaptation. Thirdly, there are other muscles which contribute to performance of forced expiratory manoeuvres, such as the oblique muscles.

Similar to our observations from the sonography, those individuals with the weakest inspiratory muscles showed the greatest change over the 6-Wk intervention ( $R^2 = 0.601$ ,  $P < 0.001$ ), with the same observation from those with the weakest expiratory muscles showing the greatest adaptation over the 6-Wk intervention ( $R^2 = 0.475$ ,  $P < 0.002$ ).

To determine whether there was any benefit of these improvements in diaphragmatic strength to skeletal muscle performance we assessed whether an isometric back extension maneuverer, the “mid thigh pull” would benefit, we saw no change.

### 3.4 Exercise outcomes

Table 4 Outcomes determined from incremental exercise testing

	PRE		POST		P value	Δ%
VO <sub>2</sub> (mL/Kg/min)	40.9	± 9.8	40.3	± 8.7	0.32	-2
Power (Watts)	228	± 50	239	± 57	0.04	5
Test duration (s)	684	± 151	718	± 171	0.04	5
Lactate (mMOL/L)	6.3	± 3.2	5.7	± 3.6	0.24	-10
Breathing Freq (Breaths/min)	41.1	± 9.1	40.2	± 9.2	0.26	-2
Heart Rate (b/min)	179	± 15	180	± 9	0.28	1
PetCO <sub>2</sub> (mmHg)	36.3	± 4.9	37.9	± 4.6	0.02	4
PetO <sub>2</sub> (mmHg)	115	± 6	117	± 4	0.09	1
RER	1.04	± 0.08	1.10	± 0.06	0.00	5
VCO <sub>2</sub> (L)	3.23	± 0.78	3.25	± 0.71	0.41	1
Ventilation, VE (L)	107	± 29	102	± 28	0.13	-5
VE/VCO <sub>2</sub>	31.3	± 4.4	29.3	± 4.2	0.01	-6
VE/VO <sub>2</sub>	33.3	± 5.1	32.0	± 4.5	0.13	-4
VO <sub>2</sub> (L)	3.08	± 0.77	2.98	± 0.68	0.13	-3
Tidal Volume (L)	2.72	± 0.95	2.64	± 0.82	0.15	-3
Exercise Efficiency (W/L O <sub>2</sub> )	75.1	± 9.4	80.7	± 8.5	0.01	7

Above are the data collected from an incremental exercise test to exhaustion. All values represent the maximal value obtained at maximal oxygen uptake (VO<sub>2</sub>max). There is a 5% increase in cycle power at the point of volitional exhaustion, with a corresponding increase in the time to reach VO<sub>2</sub>max. There should be some caution in the emphasis given to some of the significant findings where marginal differences are deemed significant in statistical terms. Here for example, there seems to be a decrease in the ventilatory response to an increase in end tidal CO<sub>2</sub> (PetCO<sub>2</sub>) suggesting that there is a small increase in the CO<sub>2</sub> accumulating in the venous blood, this could be due to an unchanged ventilation (VE) despite greater power output. Of the exercise outcomes above, the most meaningful is the 7% increase in exercise efficiency. The alternative presentation of this outcome would reveal a reduction in the Oxygen used per Watt of power output. Either of these interpretations, as with most improvements in exercise efficiency tends to be linked to a reduction in the work of breathing.

Of those outcomes above that are unchanged, the VE/VO<sub>2</sub> would be one we would have expected to improve. As a broad measure of respiratory efficiency the Ventilation for each L of oxygen, has been shown to decrease with improvements in exercise economy. It is unclear from the data presented why this was not the case, particularly given the much improved diaphragm strength. It is possible that because VO<sub>2</sub>max is not limited by maximal respiratory volumes, but cardiac output, the respiratory measures at maximum would remain unchanged despite the 6 week intervention.

## **4 Challenges and implementation**

### *4.1.1 Attrition*

With 5 dropouts, 20% of the starting population, there is the potential that some outcomes may have reached significance with further recruitment e.g. abdominal thickness where  $P = 0.09$ . Our aim however was to describe the results based on the external validity of the Airofit protocol, and to that end, future studies should account for an attrition of at least 20% when considering sample size.

### *4.1.2 Protocol compliance*

Of those who completed Post testing the compliance with the Airofit protocol ranged from 20-355mins, with an average of 202 minutes over the 6 week period. It is of significance to note that there was no “dose” response in our participants for our main outcome measures of Diaphragm thickness, Thickening ratio, Diaphragm Excursion or MIP and MEP.

### *4.1.3 Statistical Rigor*

All results are presented with the caveat that the implemented Pre-Post design would be more rigorously assessed through a 2-way analysis with the inclusion of a control population.

## **5 Conclusion**

Numerous data from previous studies show respiratory training has limited improvement in respiratory or exercise measures in healthy adults. Here we have described significant improvements in diaphragm muscle thickness and strength, with a small impact on exercise outcomes. The participants were healthy, with a range of fitness levels from sedentary to trained. All showed improvements in diaphragm muscle strength and mass, with the greatest improvements seen in those with the lowest starting points. This is a promising application of the Airofit device, particularly considering the possibilities for those with compromised respiratory function and weak diaphragms.





**Institute of Sport  
Manchester Metropolitan University  
99 Oxford Road  
Manchester  
M1 7EL**

For more information on the Faculty, please contact [instituteofsport@mmu.ac.uk](mailto:instituteofsport@mmu.ac.uk)  
Website: [www.mmu.ac.uk/institute-of-sport](http://www.mmu.ac.uk/institute-of-sport)  
Twitter: @McrlnstSport