Determinants of inspiratory muscle strength in healthy humans

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ABSTRACT

We investigated 1) the relationship between the baseline and inspiratory muscle training (IMT) induced increase in maximal inspiratory pressure ($P_{I,max}$) and 2) the relative contributions of the inspiratory chest wall muscles and the diaphragm (P_{oes}/P_{di}) to $P_{I,max}$ prior to and following-IMT. Experiment 1: $P_{I,max}$ was assessed during a Müeller manoeuvre before and after 4-wk IMT (n=30). Experiment 2: $P_{I,max}$ and the relative contribution of the inspiratory chest wall muscles to the diaphragm (P_{oes}/P_{di}) were assessed during a Müeller manoeuvre before and after 4-wk IMT (n=20). Experiment 1: $P_{I,max}$ increased 19% ($P<0.01$) post-IMT and was correlated with baseline $P_{I,max}$ ($r=-0.373$, $P<0.05$). Experiment 2: baseline $P_{I,max}$ was correlated with P_{oe}/P_{di} ($r=0.582$, $P<0.05$) and after IMT $P_{I,max}$ increased 22% and P_{oe}/P_{di} increased 5% ($P<0.05$). In conclusion, baseline $P_{I,max}$ and the contribution of the chest wall inspiratory muscles relative to the diaphragm affect, in part, baseline and IMT-induced $\Delta P_{I,max}$. Great care should be taken when designing future IMT studies to ensure parity in the between-subject baseline $P_{I,max}$.

Key words: Respiratory muscles, inspiratory muscle training, diaphragm, chest wall

1.0 Introduction

The maximal inspiratory pressure ($P_{I,max}$) generated during a Müeller manoeuvre reflects the volitional force output of the inspiratory muscles working in synergy and is an established and reliable measure of global inspiratory muscle strength in health (e.g., Romer and McConnell, 2004) and disease (e.g., Larson et al., 1993). Inspiratory muscle training (IMT) specifically targets and progressively overloads these muscles and the resulting change in $P_{I,max}$ may reflect morphological adaptation of these muscles (Downey et al., 2007) and/or changes in inspiratory muscle recruitment patterns. $P_{I,max}$ is frequently reported as an outcome measure used to quantify the efficacy of such interventions (Brown et al., 2012).

The between-participant improvements in $P_{I,max}$ following IMT is highly variable ranging from ~10% up to ~55% (Brown et al., 2012; Leith and Bradley, 1976; Romer et al., 2002b; Volianitis et al., 2001b). It has been postulated that the baseline (i.e. resting and untrained) $P_{I,max}$ may explain, in part, the variability in the relative increase in $P_{I,max}$ following IMT (Johnson et al., 2007) as the window for physiological adaptation is reduced in participants with a greater baseline strength (Kraemer et al., 1996). This notion has gained support from studies demonstrating a negative relationship between the baseline and $\Delta P_{I,max}$ following IMT in healthy and clinical populations (Brown et al., 2008; Winkler et al., 2000). Therefore, understanding this relationship may be important when designing IMT-based interventions in order to maximise confidence in the outcomes of the intervention. However, this hypothesis has yet to be systematically addressed using individuals with a wide range of baseline $P_{I,max}$ values and a range of outcome measures. Therefore, the first aim of this study was to investigate the relationship between baseline $P_{I,max}$ and the changes in $P_{I,max}$ and a wide range of outcome measures including inspiratory muscle endurance and dynamic inspiratory muscle function following a period of IMT (Experiment 1). These data aim to provide important

methodological guidelines for participant recruitment for future IMT based intervention studies which have the potential to influence a large number of research trials (c.f., Illi et al., 2013).

In addition to the between-participant variability in $\Delta P_{I,max}$ following IMT, baseline measures of inspiratory muscle strength are also highly variable between individuals. For example, in motivated, healthy participants fully familiarised with the Müeller manoeuvre and using the same predictive equation (Wilson et al., 1984), some studies report $P_{I,max}$ values ~137% of predicted (Johnson et al., 2007) while others, despite the same sex and similar age are considerably lower ~90% of predicted (Romer et al., 2002a). The mechanism(s) explaining this phenomenon are unknown but may be accounted for by the degree of relative activation of the diaphragm and the accessory chest wall inspiratory muscles during inspiratory efforts (Hershenson et al., 1989). During maximal inspiratory efforts at greater muscles lengths, the weakest inspiratory muscles (i.e., the chest wall muscles) are maximally activated and the strongest inspiratory muscle (the diaphragm) is sub-maximally activated (Hershenson et al., 1988; Nava et al., 1993). However, despite the markedly different intrathoracic pressures generated and activation patterns, the relative strengths of these muscles must be equal. If the neural activation of the diaphragm was maximal during these efforts, the thoracoabdominal configuration would be distorted, thereby reducing respiratory system compliance (Kenyon et al., 1997) and increasing the potential for shearing injuries (Hershenson et al., 1988). Consequently, increasing the strength of the weaker chest wall inspiratory muscles through targeted training should increase their neural activation and maximal force generating capacity, resulting in greater activation of the diaphragm and thus increased $P_{I,max}$ (Hershenson et al., 1988). Therefore, the second aim of this study was to evaluate the relationship between the relative contributions of the chest wall inspiratory muscles and the

diaphragm to global inspiratory muscle strength before and after IMT (Experiment 2) in attempt to explain the variability in $P_{I,max}$ at baseline and following specific training.

2.0 Materials and methods

2.1 Participants

Following ethics approval and written informed consent, 50 non-smoking, recreationally active individuals volunteered for this study. Participants abstained from alcohol, caffeine and exercise in the 24 h prior to testing and arrived at the laboratory 2 h post-prandial. All laboratory visits were separated by at least 48 h and performed at a similar time of day.

2.2 Experiment 1

Participants (n=30; age 22.8 ± 6.6 years, body mass 69.9 ± 12.0 kg, stature 1.72 ± 0.07 m) were initially familiarised with all testing procedures and subsequently attended the laboratory on two occasions prior to and following a 4 wk control period and then following a 4 wk IMT period; in total visiting the laboratory on 9 occasions (of which two were for inspiratory muscle strength measurements during the intervention periods; see *Intervention*, below). In this repeated measures design, the post-control data served as the pre-IMT baseline data. During the first visit, participants completed pulmonary and maximal inspiratory muscle function tests. In the second visit maximal dynamic inspiratory muscle function and inspiratory muscle endurance were assessed.

2.3 Visit 1: pulmonary and maximal inspiratory muscle function

Pulmonary function was assessed in accordance with published guidelines (ATS/ERS, 2005) using a pneumotachograph (ZAN 600USB, Nspire Health, Oberthulba, Germany). The pneumotachograph was calibrated prior to all trials with a 3 L syringe according to the manufacturer guidelines. $P_{I,max}$ was measured as an index of global inspiratory muscle strength using a hand-held mouth pressure meter fitted with a flanged mouthpiece

(MicroRPM, Micro Medical, Kent, UK) calibrated over the physiological range using a digital pressure meter (Pirani strain gauge, MKS Barathon, MKS Instruments, MA, USA). The mouthpiece assembly incorporated a 1 mm orifice to prevent glottic closure and minimise the contribution of the buccal muscles during inspiratory efforts. Manoeuvres were performed standing, initiated from residual volume (RV), and sustained for at least 1 s. A minimum of 3 and maximum of 8 manoeuvres were performed every 30 s, and the maximum value of 3 measures that varied by <5% was used for subsequent analysis (ATS/ERS, 2002). In addition, the $P_{I,max}$ data was also combined with that of our previous studies for further analyses (Brown et al., 2008, 2010, 2012; Johnson et al., 2007) which was collected using identical equipment and the procedures stated above.

2.4 Visit 2: Dynamic inspiratory muscle function and inspiratory muscle endurance

Maximal dynamic inspiratory muscle function was assessed to determine the pressure-flow relationship of the inspiratory muscles using a pressure threshold arrangement (POWERbreathe®, HaB Ltd, UK) as described previously (Romer and McConnell, 2004). Inspiratory mouth pressure was measured by a differential pressure transducer (± 400 cmH_2O; TSD104A, BIOPAC systems Inc., California, USA), calibrated over the physiological range (Pirani strain gauge, MKS Barathon, MKS Instruments, MA, USA), inserted in to the ceiling of the device. Inspiratory airflow was measured using a calibrated pneumotachograph (TSD160A Fleisch number 3 Pneumotachograph, BIOPAC systems Inc., California, USA) connected distally to the inspiratory port of the device. The pressure and flow signals were digitised at 200 Hz and recorded using bespoke software (Acq*knowledge* version 3.7.3, BIOPAC systems Inc., California, USA). Inspiratory pressure at zero flow (P_0) was measured by closing the inspiratory port of the device and exposing a 1 mm leak to prevent glottic closure. Participants performed in random order 3 maximal inspiratory efforts

from RV at ~0, 20, 25, 35, 50 and 65% P_0 separated by 30 s. The product of inspiratory pressure (P_I) and flow ($\dot{V}_I$) at each %P_0 defined inspiratory muscle power ($\dot{W}_I$). Maximal inspiratory flow ($\dot{V}_I max$) and power ($\dot{W}_I max$) were calculated from extrapolation of the linear pressure-flow relationship and identification of the asymptote of the power-flow relationship, respectively. Optimal flow ($\dot{V}_{opt}$, $L \cdot s^{-1}$ and $\%\dot{V}_I max$) and optimal pressure ($\dot{P}_{opt}$, cmH_2O and $\%P_0$) were subsequently calculated. The maximal rate of inspiratory pressure development (MRPD) was assessed during inspiratory efforts at P_0 and was defined as the positive peak of the pressure derivative as a function of time.

Incremental threshold loading (ITL) assessed inspiratory muscle endurance using a weighted plunger inspiratory pressure threshold device as described previously (Johnson et al., 1996, 1997). The initial threshold pressure was 10 cmH_2O and increased by 5 $cmH_2O \cdot min^{-1}$ until task failure. Task failure (endurance time) was defined as the inability to maintain tidal volume or the target pressure for three consecutive breaths despite verbal encouragement (ATS/ERS, 2002). Participants performed the test seated and were required to maintain tidal volume at resting levels while breathing frequency and duty cycle were paced by an audio metronome (breathing frequency = 15 $breaths \cdot min^{-1}$, duty cycle = 0.5) (Johnson et al., 1997). Online integration of inspiratory flow measured using a calibrated Fleisch number 3 pneumotachograph (TSD160A, BIOPAC systems Inc., California, USA) attached to the inspiratory port of the device provided continual visual feedback of the target tidal volume. Inspiratory mouth pressure was measured using a differential pressure transducer (± 400 cmH_2O; TSD104A, BIOPAC systems Inc., California, USA), calibrated over the physiological range, inserted into the ceiling of the device.

2.5 Intervention

Throughout the 4 wk control period participants performed no IMT. During the 4 wk intervention period 30 consecutive maximal dynamic inspiratory efforts were performed twice daily over a 4 wk period using a pressure-threshold device (POWERbreathe®, HaB Ltd, UK) with a training load of 50% $P_{I,max}$. This protocol is known to be effective in eliciting an adaptive response (Brown et al., 2008, 2010, 2012). Each inspiratory effort was initiated from RV and participants strove to maximise tidal volume such that task failure was reached at around the 30th inspiratory effort. Measurement of $P_{I,max}$ following 2 wk of the intervention period permitted the resistance of the device to be adjusted to ensure the appropriate relative training load. Participants were instructed to record IMT adherence in a training diary. Post-intervention trials were conducted at least 48 h following the cessation of the intervention period.

2.6 *Experiment 2*

Participants were initially familiarized with all testing procedures, divided into a control (n=10; age 27.0 ± 4.5 years, body mass 75.0 ± 8.2 kg, stature 1.80 ± 0.08 m) or an IMT group (n=10; age 21.3 ± 2.9 years, body mass 72.4 ± 10.1 kg, stature 1.76 ± 0.06 m) and completed pulmonary and inspiratory muscle function tests (for protocol see *visit 1*: Experiment 1). Subsequently, prior to and following a 4 wk control period (no IMT) or a 4 wk IMT intervention (see *Intervention*, Experiment 1), $P_{I,max}$, oesophageal (P_{oe}), gastric (P_{ga}) and the transdiaphragmatic pressure (P_{di}) were assessed during repeated Müeller manoeuvres. Volitional manoeuvres were favoured above non-volitional techniques due to their superior between-day (i.e., pre to post intervention) reliability (Hart et al., 2001; Romer and McConnell, 2004). Every 30 s, 8 efforts were performed from RV and following a 5 min break from functional residual capacity (FRC) in order to minimise the effects of the elastic

recoil pressure of the lung and chest wall upon $P_{I,max}$ (ATS/ERS, 2002). During efforts from FRC an end-expiratory P_{oe} of approximately -2.0 to -5.0 cmH_2O ensured a constant end-expiratory lung volume (Romer et al., 2007). All efforts were performed while standing to minimise the compressive effects of the mediastinal compartment on P_{oe} (Baydur et al., 1982) and efforts were performed against a calibrated mouth pressure meter. The device was fitted with a flanged mouthpiece (MicroRPM, Micro Medical, Kent, UK) aligned at the mouth using a table-mounted clamp. Data were obtained from the maximum P_{di} of 3 measures that varied by <5% (ATS/ERS, 2002).

2.7 Intrathoracic pressure measurements

P_{oe} and P_{ga} were measured via two latex nasopharyngeal balloons sealed over a single catheter (Milic-Emili et al., 1964) (Nspire health, Oberthulba, Germany). The oesophageal and gastric balloons were passed in to the stomach and filled with 1 and 2 ml of air, respectively, according to their optimal pressure-volume characteristics. The oesophageal balloon was withdrawn until a negative pressure deflection was observed during inspiration and then withdrawn a further 10 cm to ensure correct placement within the oesophagus; positioning was confirmed using the occlusion technique (Baydur et al., 1982). Participants were instrumented with the same catheter during their experimental trials and the internal length of the catheter passed in to the participant was recorded on the first trial and repeated in all subsequent trials. Each catheter was connected to a differential pressure transducer (± 400 cmH_2O; TSD104A, BIOPAC systems Inc., California, USA) calibrated across the physiological range using a digital pressure meter (Pirani strain gauge, MKS Barathon, MKS Instruments, MA, USA). The pressure signal was digitised at 200 Hz and recorded using bespoke software (Acq*knowledge* version 3.7.3, BIOPAC systems Inc., California, USA). P_{di}

was calculated by online subtraction of P_{oe} from P_{ga}. The pattern of relative chest wall muscle recruitment was expressed by the P_{oe}/P_{di} ratio (Nava et al., 1993).

2.8 *Statistical analyses*

Differences between variables were assessed using a paired or independent samples *t*-test. Pearson's product moment correlation assessed the relationships between selected variables. Statistical significance was set a-priori at $P \leq 0.05$. Data are presented as mean ± SD unless stated otherwise.

3.0 Results

3.1 Experiment 1

Pulmonary, static and dynamic inspiratory muscle function and inspiratory muscle endurance prior to and following the control and intervention periods are shown in Table 1. One participant failed to complete the post-IMT measures and their data were omitted from the analyses. All variables were unchanged following the control period. Baseline median $P_{I,max}$ (% predicted: according to the equation of Wilson et al., 1984) was 156 cmH_2O (147%) and ranged from 82 (66%) to 278 cmH_2O (227%). Throughout the intervention, IMT compliance was 87 ± 11% which is similar to previous training studies (Illi et al., 2013). $P_{I,max}$ increased 19 ± 10% following the intervention (P<0.001, range 6 to 45%) and was negatively correlated with the baseline $P_{I,max}$ (n=29; r = -0.373, P<0.05: medium effect; Figure 1A). When results were combined with data previously collected within our laboratory the relationship improved further (n=67; r = -0.48, P<0.01: large effect; Figure 1B). Pulmonary function remained unchanged following IMT and as expected, with the exception of $\%\dot{V}_I max$ and $\%P_0$, all measures of dynamic inspiratory muscle function were improved (P<0.05; Table 1). Baseline $P_{I,max}$ was negatively correlated with the relative increase in $\dot{W}_I max$ (r = -0.458, P<0.05) and $\dot{V}_I max$ (r = -0.383, P<0.05). Inspiratory muscle endurance increased by 27% following IMT (P<0.05).

3.2 Experiment 2

Baseline $P_{I,max}$ at RV and FRC and pulmonary function for the control and IMT groups are shown in Table 2. Two participants from the IMT group failed to complete the post-intervention trials and their data were omitted from the analyses. All variables were unchanged following the intervention period in the control group. Throughout the intervention, IMT compliance was 92 ± 9%. $P_{I,max}$ increased in the IMT group 22 ± 24% at

RV (pre: 170 ± 50 cmH_2O vs. post: 196 ± 55 cmH_2O; P<0.05) and 20 ± 21% at FRC (pre: 137 ± 30 vs. post: 156 ± 24; P<0.05). Intrathoracic pressures at RV and FRC for both groups prior to and following the intervention period are shown in Table 3. Following IMT, P_{oe} decreased (i.e. became more negative) (RV: 14 ± 11%, FRC: 18 ± 13%; P<0.01), whereas increases were observed in P_{di} (RV: 9 ± 9%, FRC: 15 ± 14%; P<0.05) and P_{oe}/P_{di} (RV: 5 ± 5%, RV 3 ± 3%; P<0.05).

Pooled baseline $P_{I,max}$ was positively correlated with pooled baseline P_{oe}/P_{di} at RV (r = 0.582, P<0.05) and FRC (r = 0.523, P<0.05). Pooled baseline $P_{I,max}$ was also correlated with P_{di} at both RV (r = 0.561, P<0.05) and FRC (r = 0.515, P<0.05). Following IMT the absolute (r = 0.707, P<0.05) and relative (r = 0.759, P<0.05) increase in P_{di} was correlated with the absolute increase in P_{oe} at RV. No relationship was observed however between $\%\Delta P_{I,max}$ and $\Delta P_{oe}/P_{di}$ following IMT at RV (r = 0.16, P>0.05) and FRC (r = -0.25, P>0.05).

4. Discussion

4.1 Main findings

The aim of this study was to investigate the determinants of $P_{I,max}$ before and after IMT. In Experiment 1, baseline $P_{I,max}$ was negatively correlated with IMT-mediated increases in $P_{I,max}$, $\dot{W}_I max$ and $\dot{V}_I max$. In Experiment 2, although baseline $P_{I,max}$ was positively correlated with P_{oe}/P_{di} and Pdi, IMT-mediated increases in these measures were not correlated.

4.2 Experiment 1

The negative relationship observed between baseline $P_{I,max}$ and the IMT-mediated increase in $P_{I,max}$ (Figures 1A and 1B) suggests that care must be taken to ensure parity in baseline $P_{I,max}$ between participants/experimental groups when designing IMT-based interventions. We have identified for the first time that the baseline strength of these muscles may affect the efficacy (when based on $P_{I,max}$) of the IMT intervention. This relationship confirms and extends the suggestions of previous studies in healthy (Brown et al., 2008) and clinical (Winkler et al., 2000) populations and may explain the differentiated IMT-induced increase in $P_{I,max}$ observed in previous studies (range: 10% to 55%) (Brown et al., 2008, 2010, 2012; Leith and Bradley, 1976; Romer et al., 2002b; Volianitis et al., 2001b). The large range of $\%\Delta P_{I,max}$ after IMT (>45%) demonstrates the great plasticity of the inspiratory muscles and importantly, that these muscles behave similarly to other non-respiratory skeletal muscles during strength training. For example, in limb skeletal muscles the physiological potential for adaptation following strength training has been shown to be inversely related to the baseline strength; therefore, the closer the muscles are to their physiological ceiling, the smaller the potential for physiological adaptation (Häkkinen, 1994; Kraemer et al., 1996). However, since baseline $P_{I,max}$ explained 23% of the variance in $\%\Delta P_{I,max}$ (Figure 1B), other factors must also

influence the inspiratory muscle training response and this presents an interesting avenue for future investigation.

Whilst the existence of a physiological ceiling may explain some of the $\%\Delta P_{I,max}$, $P_{I,max}$ is also prone to a learning effect (Volianitis et al., 2001a; Wen et al., 1997) probably because of the volitional, effort-dependent nature of the Müeller manoeuvre. Thus, as cautioned previously (Polkey et al., 2011) participants with lower baseline $P_{I,max}$ may also develop greater aptitude with the Müeller manoeuvre during IMT, which is technically very similar. Although inspiratory muscle recruitment patterns during IMT have not been examined, participants performing repeated inspiratory pressure-threshold loading tests adjust their breathing, and thus presumably inspiratory muscle recruitment, pattern in order to optimise inspiratory muscle endurance (Eastwood et al., 1998; Roussos et al., 1979). Therefore, some of the $\%\Delta P_{I,max}$ after IMT may also reflect a change in inspiratory muscle recruitment to "maximise" $P_{I,max}$, and this may occur to a greater extent in those with lower baseline $P_{I,max}$.

4.3 Experiment 2

Baseline $P_{I,max}$ was positively correlated with both P_{di} and P_{oe}/P_{di} indicating that diaphragm and relative chest wall muscle recruitment are important determinants of $P_{I,max}$. Diaphragm and inspiratory intercostal muscle hypertrophy has been reported after IMT (Downey et al., 2007; Enright et al., 2006; Ramirez-Sarmiento et al., 2002) and such changes may have contributed to the IMT-mediated improvements in inspiratory muscle function observed in the present study. Furthermore, the increases in P_{di} (in the absence of a change in P_{ga}) and P_{oe}/P_{di} during the Müeller manoeuvre after IMT also indicates greater diaphragm activation and relative inspiratory chest wall muscle recruitment, respectively. Understanding the nature of these increases is, however, complicated due to the complex synergism between the

diaphragm and the inspiratory intercostals during inspiration (De Troyer et al., 2005; Roussos et al., 1979). Specifically, voluntary activation of the diaphragm during a Müeller manoeuvre is dependent on lung volume, such that activation is lowest (~80%, although inter-individual variability exists) at RV (McKenzie et al., 1996) and increases with increasing lung volume, with full activation being achieved at and above FRC (Gandevia et al., 1990; McKenzie et al., 1996). The submaximal activation of the diaphragm during a Müeller manoeuvre at RV may result from reflex inhibition of the phrenic motoneurones (McKenzie et al., 1996) and serve to minimise chest wall distortion (De Troyer et al., 2005). Given these observations, the increased P_{di} measured at RV after IMT in the present study may have been permitted because of greater chest wall muscle activation and subsequently less reflex inhibition of the phrenic motoneurones.

Reasons for the increased P_{oe}/P_{di} after IMT remain somewhat less clear, as does the functional significance of this change given the absence of a relationship between $\%\Delta P_{oe}/P_{di}$ and $\%\Delta P_{I,max}$. The length-tension relationships of the diaphragm and inspiratory intercostals are not matched over the vital capacity range (De Troyer et al., 2005) and thus the relative loads placed on these muscles during IMT may differ. Indeed, McConnell et al. (2002) speculate that IMT imposes a greater relative training load on the inspiratory chest wall muscles compared to the diaphragm (McConnell et al., 2002), which might explain, in part, our observed increase in P_{oe}/P_{di}. However, this suggestion is based on there being submaximal diaphragm activation, and greater chest wall muscle recruitment, during a Müeller manoeuvre that evokes $P_{I,max}$, whereas it seems unlikely that such inhibition would be seen during IMT at 50% $P_{I,max}$. Indeed, during submaximal inspiratory loading the diaphragm and inspiratory chest wall muscles undergo periodic recruitment and de-recruitment, which may limit/delay fatigue of these muscles (Roussos et al., 1979). Thus,

rather than IMT evoking preferential loading of inspiratory chest wall muscles, an alternative explanation is that repeated IMT simply enhanced the participants ability to recruit the inspiratory chest wall muscles during loaded inspiratory efforts. This notion could be examined in future studies using periodic measures of inspiratory muscle recruitment throughout an IMT intervention.

4.4 Conclusions

This study demonstrates that baseline $P_{I,max}$ is an important, though not the only, determinant of the IMT-mediated increase in $P_{I,max}$ and that great care must therefore be taken in standardising $P_{I,max}$ when recruiting participants for IMT-based interventions. The IMT-mediated increases in P_{di} and P_{oe}/P_{di} during the Müeller manoeuvre indicates that all inspiratory muscles are targeted by IMT. Furthermore, the increase in P_{di} at RV during the Müeller manoeuvre may have been permitted due to greater recruitment of the inspiratory chest wall muscles after IMT. Whether IMT-mediated increases in P_{oe}/P_{di} reflect a greater relative training load placed on the inspiratory chest wall muscles or a shift in recruitment strategy remains unknown.

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Table 1 Experiment 1: Inspiratory muscle strength, pulmonary function, dynamic inspiratory muscle function and inspiratory muscle endurance prior to (Baseline) and following the 4 wk control period (Post-control/pre-IMT) and following 4 wk inspiratory muscle training (Post-IMT).

	Baseline	Post-Control / pre-IMT	Post-IMT
Maximal inspiratory pressure and pulmonary function			
$P_{I,max}$ (cmH_2O)	147 ± 48	149 ± 45	174 ± 48*
FVC (L)	4.67 ± 0.86	4.68 ± 0.89	4.70 ± 0.89
FEV_1 (L)	3.89 ± 0.69	3.84 ± 0.74	3.84 ± 0.74
FEV_1/FVC (%)	83.6 ± 6.5	82.3 ± 6.9	82.2 ± 6.7
PEF ($L \cdot s^{-1}$)	8.39 ± 1.76	8.38 ± 1.77	8.46 ± 1.87
MVV_{10} ($L \cdot min^{-1}$)	152.5 ± 36.3	154.4 ± 37.1	158.7 ± 38.4
Dynamic inspiratory muscle function and inspiratory muscle endurance			
P_0 (cmH_2O)	143 ± 41	150 ± 43	172 ± 49*
$\dot{V}_I max$ ($L \cdot s^{-1}$)	7.26 ± 1.44	7.16 ± 1.43	7.55 ± 1.35*
$\dot{W}_I max$ ($cmH_2O \cdot L^{-1} \cdot s^{-1}$)	246.7 ± 94.8	244.1 ± 89.4	328.8 ± 109.0*
$\dot{V}_{opt}$ ($L \cdot s^{-1}$)	3.70 ± 0.70	3.64 ± 0.73	3.82 ± 0.78*
$\dot{P}_{opt}$ (cmH_2O)	64.1 ± 18.3	66.5 ± 16.5	83.6 ± 22.0*
% $\dot{V}_I max$ (%)	50.5 ± 5.3	50.9 ± 3.6	50.5 ± 4.6
% P_0 max (%)	44.9 ± 7.0	48.6 ± 5.3	49.2 ± 6.1
MRPD ($cmH_2O \cdot ms^{-1}$)	0.51 ± 0.19	0.47 ± 0.17	0.76 ± 0.69*
ITL (min)	13.58 ± 4.98	13.43 ± 5.30	16.03 ± 4.76*

Values are expressed as means ± SD. * $P<0.05$ vs. post-control. For abbreviations see methodology, Experiment 1.

Table 2 Experiment 2: Baseline inspiratory muscle strength and pulmonary function of the control and inspiratory muscle training (IMT) groups.

	Control	IMT
$P_{I,max}$ RV (cmH_2O)	155 ± 44	170 ± 50
$P_{I,max}$ FRC (cmH_2O)	148 ± 408	137 ± 30
FVC (L)	5.43 ± 0.92	4.92 ± 0.66
FEV_1 (L)	4.22 ± 0.78	3.92 ± 0.77
FEV_1/FVC (%)	77.7 ± 7.4	79.3 ± 6.4
PEF ($L \cdot s^{-1}$)	10.04 ± 1.81	8.43 ± 1.64
MVV_{10} ($L \cdot min^{-1}$)	186.1 ± 36.4	172.4 ± 41.0

* $P<0.05$ between groups. For abbreviations see methodology, Experiment 1.

Table 3 Experiment 2: Intrathoracic pressures during a Müeller manoeuvre in the control and IMT groups prior to and following the intervention period.

	RV pre	RV post	FRC pre	FRC post
Control Group				
P_{oe} (cmH_2O)	-129.3 ± 46.0	-129.7 ± 52.3	-132.0 ± 36.4	-133.2 ± 39.2
P_{ga} (cmH_2O)	20.8 ± 24.8	23.8 ± 23.6	29.7 ± 22.0	27.6 ± 18.5
P_{di} (cmH_2O)	150.0 ± 40.8	153.6 ± 36.9	161.8 ± 41.8	160.8 ± 43.8
P_{oe}/P_{di} (%)	85.2 ± 85.1	84.8 ± 18.2	82.2 ± 13.3	83.4 ± 11.8
IMT Group				
P_{oe} (cmH_2O)	-126.3 ± 20.0	-144.6 ± 29.9**	-117.2 ± 26.9	-136.8 ± 31.5**
P_{ga} (cmH_2O)	28.8 ± 27.1	24.6 ± 23.9	35.6 ± 24.9	31.2 ± 28.3
P_{di} (cmH_2O)	152.1 ± 32.7	166.7 ± 39.9*	147.8 ± 33.3	168.0 ± 35.2*
P_{oe}/P_{di} (%)	84.4 ± 10.1	88.3 ± 12.2*	80.4 ± 14.0	82.6 ± 14.5*

* $P < 0.05$ and ** $P < 0.01$ vs. pre. P_{oe} = oesophageal pressure; P_{ga} = gastric pressure; P_{di} = transdiaphragmatic pressure.

Figure captions

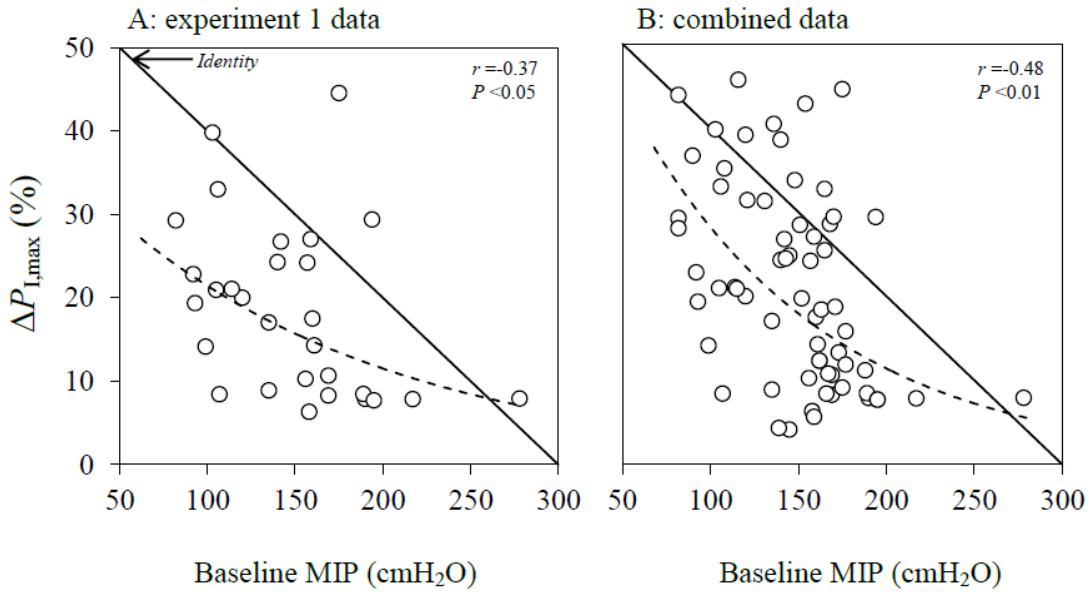

Figure 1 Relationship between baseline maximal inspiratory pressure (Baseline $P_{I,max}$) and the relative change in $P_{I,max}$ ($\Delta P_{I,max}$) following 4 wk inspiratory muscle training with (A) data from Experiment 1 [n=29] and (B) data from Experiment 1 combined with data from our previous studies [n=67] (Brown et al., 2008, 2010, 2012; Johnson et al., 2007). An exponential model fit was used in both (A) and (B).