



# 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 Müller 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 Müller 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  $P_{oes}/P_{di}$  ( $r = 0.582$ ,  $P < 0.05$ ) and after IMT  $P_{i,max}$  increased 22% and  $P_{oes}/P_{di}$  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  $P_{i,max}$ .

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## 1. Introduction

The maximal inspiratory pressure ( $P_{i,max}$ ) generated during a Müller 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 Müller 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

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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. 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  $22.8 \pm 6.6$  years, body mass  $69.9 \pm 12.0$  kg, stature  $1.72 \pm 0.07$  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 Section 2.5, 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 metre fitted with a flanged mouthpiece (MicroRPM, Micro Medical, Kent, UK) calibrated over the physiological range using a digital pressure metre (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 ( $\pm 400$  cmH<sub>2</sub>O; TSD104A, BIOPAC systems Inc., CA, 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., CA, 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 (Acqknowledge 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  $\sim 0, 20, 25, 35, 50$  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 ( $\dot{V}_I \text{ max}$ ) and power ( $\dot{W}_I \text{ max}$ ) were calculated from extrapolation of the linear pressure–flow relationship and identification of the asymptote of the power–flow relationship, respectively. Optimal flow ( $V_{opt}$ , L s<sup>-1</sup> and  $\% \dot{V}_I \text{ max}$ ) and optimal pressure ( $\dot{P}_{opt}$ , cmH<sub>2</sub>O 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<sub>2</sub>O and increased by 5 cmH<sub>2</sub>O min<sup>-1</sup> 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 min<sup>-1</sup>, 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., CA, 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 ( $\pm 400$  cmH<sub>2</sub>O; TSD104A, BIOPAC systems Inc., CA, 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

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