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Original Research Article

# Respiratory muscle strength is decreased after maximal incremental exercise in trained runners and cyclists☆

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ABSTRACT

The respiratory muscle fatigue seems to be able to limit exercise performance and may influence the determination of maximal oxygen uptake ($\dot{V}O_{2max}$) or maximum aerobic work rate during maximal incremental test. The aim of this study was therefore to investigate whether maximal incremental exercise decreases respiratory muscle strength. We hypothesized that respiratory muscle strength (maximal pressure) will decrease after maximal incremental exercise to exhaustion. 36 runners and 23 cyclists completed a maximal incremental test on a treadmill or a cycle ergometer with continuous monitoring of expired gases. Maximal inspiratory (MIP) and expiratory (MEP) pressure measurements were taken at rest and post- exercise. At rest, the MIP and MEP were 140 ± 25 and 172 ± 27 in runners *vs.* 115 ± 26 and 146 ± 33 in cyclists ($p < 0.05$ between groups, respectively). The rest values of MIP and MEP were correlated to the $\dot{V}O_{2peak}$ in all athletes, $r = 0.34$, $p < 0.01$ and $r = 0.36$, $p < 0.01$, respectively. At exhaustion, the MIP and MEP decreased significantly post- test by 13 ± 7% and 13 ± 5% in runners *vs.* 17 ± 11% and 15 ± 10% in cyclists ($p > 0.05$), respectively. Our results suggest that respiratory muscle strength is decreased following maximal incremental exercise in trained runners and cyclists.

## 1. Introduction

Respiratory muscle fatigue can be defined as a loss in the capacity for developing force and/or velocity resulting from muscle activity under load which reverses by rest ("ATS/ERS" 2002). The development of such fatigue is well documented not only in pathological patients (Hardiman, 2011; Klimathianaki et al., 2011) but also in trained subjects during high exercise intensities (Romer et al., 2006). In this context, several studies found a significant respiratory muscle fatigue in healthy subjects (Janssens et al., 2013) performing in a range of sports including running (Loke et al., 1982; Ross et al., 2008), cycling (Romer et al., 2006), swimming (Jakovljevic and McConnell, 2009; Lomax and Castle, 2011) and triathlon (Hill et al., 1991). In this context, it has been shown that respiratory muscle fatigue can cause a sympathetically mediated vasoconstriction at the locomotor muscles by means of the respiratory muscle metaboreflex. This metaboreflex comprises limb blood flow and thus $O_2$ delivery in favor of the respiratory muscles (Borghi-Silva et al., 2008; Dominelli et al., 2017), which might stimulate the development of locomotor muscle fatigue (Derchak et al., 2002; Shadgan et al., 2011; Vogiatzis et al., 2009), and thus limit exercise tolerance. The observation that respiratory muscle training can enhance time to exhaustion during constant load exercise > 80% $VO_{2max}$ (Bailey et al., 2010), supports the notion that respiratory muscle function should be considered as a possible determinant of exercise performance.

Although in laboratory conditions, the observation of respiratory muscle fatigue is well established during constant-load exercise (Gonzales and Williams, 2010; Kabitz et al., 2008; Oueslati et al., 2017), the occurrence of this potentially exercise-limiting phenomenon during maximal incremental exercise testing is unclear. Sugiura et al. (2013) and Segizbaeva et al. (2013), assessing maximal inspiratory and expiratory pressures, could not support the occurrence of respiratory muscle fatigue, although in this latter study electromyographic activity of the sternocleidomastoid and scalene muscles was reduced. Also Romer et al. (2007) observed that unloading of the respiratory muscles had no impact on incremental exercise duration, suggesting that the

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duration of heavy intensity cycling is too restricted to induce the respiratory muscle metaboreflex. However, in contrast to the above studies, we recently found reduced maximal inspiratory and expiratory pressures following maximal incremental exercise testing in highly trained cyclists (Oueslati et al., 2016; Oueslati et al., 2017), suggesting the presence of respiratory muscle fatigue. Additionally, given that the lungs and airways are less prone to structural and functional adaptations compared to the cardiovascular and muscle system, it has been reported that exercise-induced arterial hypoxemia occurs in some endurance trained athletes. This exercise-induced arterial hypoxemia can affect arterial $O_2$ content and might limit locomotor muscle $O_2$ delivery as well. Therefore, it is currently unclear whether a respiratory system limitation might be able to limit exercise performance during maximal incremental exercise by reducing locomotor muscle $O_2$ delivery and consequently could influence the determination of maximal oxygen uptake ($\dot{V}O_{2max}$) or maximum aerobic work rate, especially, when the $\dot{V}O_2$ does not develop a $\dot{V}O_2$ plateau (Poole and Jones, 2017).

Additionally, it should be noted that the impact of the respiratory system might differ between exercise test modalities, and more specifically between cycling and running. In this context, Hue et al. (2000). (2000) reported a higher respiratory rate (57 ± 11 breath $min^{-1}$) and a lower tidal volume (2.60 ± 0.41 L) during incremental running compared to cycling exercise (respiratory rate, 48 ± 7 breath $min^{-1}$ and tidal volume, 3.00 ± 0.55 L) for the same subject. However, Gavin and Stager (1999) observed a greater minute ventilation and ventilator equivalents as well as respiratory exchange ratios during maximal cycling compared to running exercise in healthy men. Recently, Tanner et al. (2014) stated that ventilator patterns differ between maximal running and cycling, by recording the tidal flow-volume loops during the exercise. It seems plausible that the changes in the ventilatory patterns due to the exercise modalities could affect the occurrence of a decrease in respiratory muscle strength.

Therefore, the purpose of the present study is twofold: First, we wanted to investigate whether respiratory muscle fatigue, assessed from maximal inspiratory and expiratory pressures, would occur during incremental ramp exercise in trained subjects. Second, we wanted to assess whether the influence of the respiratory system on exercise tolerance to maximal incremental tests would differ between cycling and running. We hypothesized that respiratory muscle pressure will decrease post maximal incremental exercise in runners and cyclists, with no difference between the test modalities.

## 2. Methods

To verify our hypothesis, participants visited the laboratory on two occasions. During the first visit, dynamic pulmonary function was assessed and subjects were familiarized with the respiratory muscle strength tests. During the second visit, cyclists and runners performed either a maximal cycling or running test, respectively, followed by the same respiratory muscle strength tests, from which the change in maximal inspiratory and expiratory pressures (MIP and MEP, respectively) from rest to post exercise test was assessed.

### 2.1. Subjects

59 trained men volunteered to participate in this study (thirty-six runners and 23 cyclists) (Table. 1). They were involved in a minimum 6 sessions of aerobic training (running and cycling for runners and cyclists participant, respectively). All participants were non-smokers and did not report heart and lung diseases, and had normal resting pulmonary function, as assessed by spirometry tests. Prior to any data collection, they were informed of the risks and benefits of the study and then gave written informed consent to participate (from their parents for minors). This study was approved by the Ethics Committee of UHC Farhat-Hached Hospital and in accordance with the Declaration of Helsinki (1964).

**Table 1**
Participants' characteristics.

| | Runners | Cyclists |
|---|---|---|
| Subjects, No | 36 | 23 |
| Age, years | 23 ± 3 | 16 ± 2* |
| Body mass, kg | 73 ± 8 | 63 ± 10* |
| Height, cm | 177 ± 5 | 174 ± 7 |
| VC, l | 5 ± 0.5 | 5 ± 0.6 |
| FVC, l | 5.6 ± 0.6 | 5.6 ± 0.8 |
| $FEV_1$, l | 4.4 ± 0.3 | 4.6 ± 0.5 |
| MVV, $l.min^{-1}$ | 184 ± 23 | 166 ± 16* |

VC, vital capacity; FVC, forced vital capacity; $FEV_1$, forced expiratory volume second; MVV, maximum voluntary ventilation. Data are expressed as mean ± SD.
* $p < 0.05$ between groups.

### 2.2. Procedures

#### 2.2.1. Maximal incremental cycling test

After a 2-min resting period (sitting on the bike) followed by 5-min warm-up at 60 W (pedaling rate 60–70 rpm), a maximal incremental exercise (+30 W $min^{-1}$ with fixed pedaling rate at ~90 rpm, ± 5 rpm at exhaustion) was completed until exhaustion. The test was terminated when the subjects could no longer maintain a pedaling rate > 60 rpm despite strong verbal encouragement.

#### 2.2.2. Maximal incremental running test

The test was performed on a motorized treadmill (Johnson fitness T8000, USA) for the runners' participants. After a 3 min warm-up at 8 km $h^{-1}$, the exercise began at an initial speed of 10 km $h^{-1}$ and then the speed was increased by 1 km $h^{-1}$ every two minutes until exhaustion despite strong verbal encouragement.

#### 2.2.3. Pulmonary function procedure

Spirometry test was performed with the participant in the sitting position while breathing room air, with the nose being occluded by a clip to assess the slow vital capacity (VC), forced vital capacity (FVC), forced expiratory volume in 1 s ($FEV_1$) and maximum voluntary ventilation over 12 s (MVV). A breathing tube was inserted into the subject's mouth with the lips sealed around the mouthpiece. All testing was completed using a calibrated computerized spirometer Spirolab III (Medical International Research, Rome, Italy) by the same technician. The subjects were familiarized with the device and the procedure for each test demonstrated by the technician. The flow and volume measurement sensor is a digital turbine, based on the infrared interruption principal. This principal ensures the accuracy and the reproducibility of the measurements without requiring a periodic calibration. For an accurate and reliable calibration, the syringe volume used was 3 L. Each subject performed tests five times. Respiratory maneuvers were performed in accordance with the standards established by (Miller et al., 2005).

#### 2.2.4. Respiratory muscle strength

Maximal inspiratory (MIP) and expiratory pressures (MEP) were measured before and after the maximal incremental test (~2 min and at rest's minute ventilation) in order to evaluate exercise-induced changes in the respiratory muscle strength. MIP and MEP were assessed with portable manometer (MicroRPM, MicroMedical Ltd, Kent, UK) according to published guidelines (Evans and Whitelaw, 2009). All measurements were made while the participant sat with their nose occluded. Briefly, each participant was instructed to exhale to residual volume and then to inhale to total lung capacity (pressure sustained for at least 1.5 s) when assessing MIP and MEP, respectively. The maneuver was repeated five times (coefficient of variation less than 5%) and maximum values were retained for final analysis.

### 2.3. Measurements

#### 2.3.1. Cardiopulmonary responses

Expired gases were collected by a mask enclosing both the mouth and nose and recorded by a breath by breath portable analyzer (Cosmed $K_4b^2$, Rome, Italy) for calculation of $\dot{V}O_2$, ventilation ($\dot{V}_E$), carbon dioxide production ($\dot{V}CO_2$), tidal volume and respiratory rate. The gas analyzer and the pneumotachograph were calibrated prior to each test following manufacturer's recommendations using a reference gas mixture (16% $O_2$ and 5% $CO_2$) and a 3 L syringe (Cosmed, Rome, Italy), respectively. HR was measured with a chest strap polar device (RS300, Polar Electro, Kempele, Finland) and expressed as a percentage of theoretical HR (=210-0.65* age).

The obtained breath-by-breath values were averaged into 30 s intervals and these values were used for further analysis. Attainment of maximum oxygen uptake ($\dot{V}O_{2peak}$), defined as the highest averaged value of $\dot{V}O_2$ over 30 s was determined and used to assess the corresponding velocity ($v\dot{V}O_{2p}$) and power output ($p\dot{V}O_{2p}$). Then the $\dot{V}O_{2peak}$ values were expressed in percentage of predicted $\dot{V}O_{2max}$ established from the equation of Larsen et al. (2002) for runners and Jones et al. (1985) for cyclists. The $\dot{V}_E$ values were calculated at rest and at 20, 40, 60, 80 and 100% (average values over 30 s) of the test duration for each subject.

### 2.4. Statistical analyses

Descriptive statistics (mean ± SD) were calculated for all variables. The data was processed using SigmaPlot (version 11, Systat software, Germany). Assumptions of normality were analyzed by the Kolmogrov-Smirnov test. The differences between MIP and MEP pre- and post- exercise were analyzed using the Paired *t*-test. The differences in values between runners and cyclists group were assessed using the Mann-Whitney Rank Sum test. The differences in the $\dot{V}_E$ between groups during were tested with the ANOVA of Fried-man test. The relationships between selected parameters were investigated by Pearson correlation coefficients. The threshold for statistical significance was set at $p < 0.05$

## 3. Results

### 3.1. Cardiopulmonary responses

The data collected from both running and cycling incremental tests are presented in Table. 2. The running and cycling tests lasted 1135 ± 201 s and 508 ± 84 s ($p < 0.05$), respectively. Runners sustained a $\dot{V}_E > 100$ L min$^{-1}$ for 522 ± 184 s while cyclists maintained a $\dot{V}_E > 100$ L min$^{-1}$ for 142 ± 53 s, ($p < 0.05$). The $\dot{V}O_{2peak}$ was only correlated with the MVV ($r = 0.44$, $p = 0.001$) but not with the VC ($r = 0.27$, $p = 0.06$), FEV ($r = 0.14$, $p = 0.34$), FVC ($r = 0.27$, $p = 0.57$). The $\dot{V}_E$ increased significantly and was higher in runners compared to cyclists from 20 to 80% of time to exhaustion (Fig. 1).

**Table 2**
Cardiorespiratory values at the end of the maximal incremental test.

| | Runners | Cyclists |
|---|---|---|
| $\dot{V}O_{2peak}$, ml kg$^{-1}$ min$^{-1}$ | 55 ± 6 | 51 ± 8 |
| $\dot{V}O_{2peak}$, %predicted | 120 ± 18 | 95 ± 14 |
| $\dot{V}_{Epeak}$, l min$^{-1}$ | 141 ± 16 | 132 ± 25 |
| $\dot{V}_{Epeak}$/MVV, % | 78 ± 10 | 78 ± 11 |
| $HR_{peak}$, | 187 ± 5 | 195 ± 4 |
| $HR_{peak}$, %theoretical | 95 | 97 |
| RER | 1.2 ± 0.1 | 1.1 ± 0.1 |
| $v\dot{V}O_2p$,km h$^{-1}$ | 17 ± 1.7 | – |
| $p\dot{V}O_2p$, W | – | 314 ± 42 |

$\dot{V}O_{2peak}$, peak of oxygen uptake; $\dot{V}_{Epeak}$, peak of minute ventilation; MVV, maximum voluntary ventilation; HR, heart rate; RER, respiratory exchange rate; $v\dot{V}O_2p$, velocity at $\dot{V}O_{2peak}$; $p\dot{V}O_{2p}$, power output at $\dot{V}O_{2peak}$.

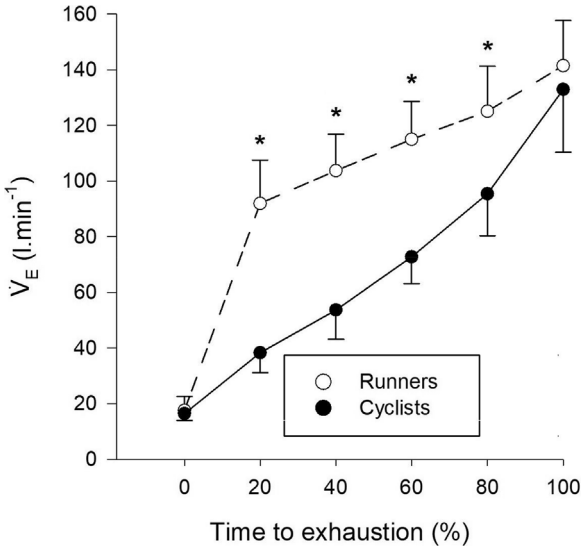


**Fig. 1.** Minute ventilation in runners and cyclists during the maximal ramp exercises. *, $p < 0.05$ between groups.

### 3.2. Respiratory muscle strength

The $\dot{V}O_{2peak}$ was correlated to the MIP and MEP at rest, $r = 0.34$, $p < 0.01$ and $r = 0.36$, $p < 0.01$ (Fig. 2), and post the exercise, $r = 0.32$, $p < 0.01$ and $r = 0.36$, $p < 0.01$, respectively. However, a lack of a significant correlation was observed between the decrease of MIP and MEP and the $\dot{V}O_{2peak}$. The MIP values were only correlated to the body mass ($r = 0.46$, $p < 0.001$) (Fig. 3). The MIP and MEP prior to the maximal incremental exercise were respectively higher ($p < 0.05$) in runners compared to cyclists (Table. 2). A lack of difference in MIP of rest between groups was observed when MIP was corrected by body mass (Runners: 1.9 ± 0.31 *vs.* Cyclists: 1.84 ± 0.57, $p > 0.05$). The MIP and MEP decreased significantly after the maximal incremental exercise by 13 ± 7% and 13 ± 5% in runners, *vs.* 17 ± 11% and 15 ± 10% in cyclists, respectively ($p > 0.05$) (Table 3).

## 4. Discussion

The aim of this study was to assess respiratory muscle strength pre- and post- maximal incremental exercise in trained runners and cyclists. In line with our hypothesis it was observed that a maximal incremental exercise test induced a decrease in maximal inspiratory and expiratory pressure. Additionally, it was found that the decrease in MIP and MEP did not differ between cyclists and runners.

Muscle fatigue can be defined as a loss in the capacity for developing force and/or velocity resulting from muscle activity under load which reverses by rest (ATS/ERS, 2002). Respiratory muscle fatigue can be detected invasively using phrenic nerve stimulation (magnitic or electrical) to measure transdiaphragmatic pressure, oesphageal pressure or gastric pressure (Guleria et al., 2002; Kabitz et al., 2008; Laghi et al., 1998; Tomczak et al., 2011), or non-invasively by assessing mouth pressure (Brown and Kilding, 2011; Hill et al., 1991; Loke et al., 1982; McConnell and Lomax, 2006; Perret et al., 1999; Ross et al., 2008) (see Janssens et al. (2013)). In the present study, maximal inspiratory and expiratory pressures were assessed non-invasively to determine exercise-induced alterations in respiratory muscles strength at exhaustion.

It is clear that the present results confirm that respiratory muscle strength decreases significantly after maximal incremental exercise.

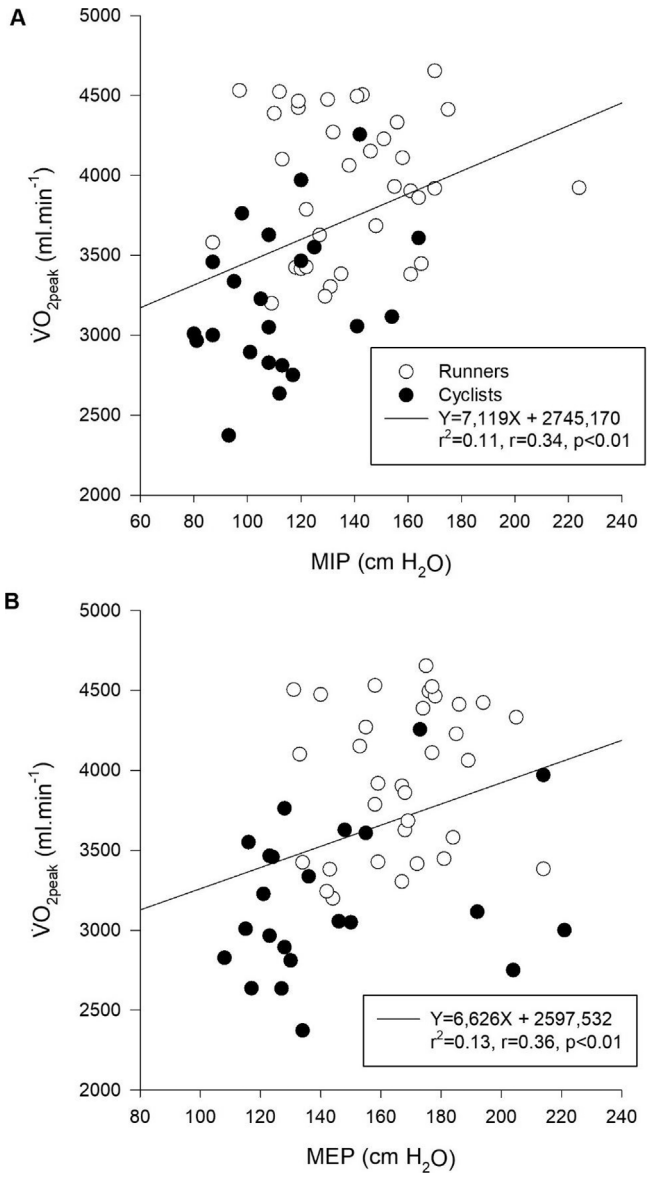


**Fig. 2.** (A) Maximal inspiratory pressure and (B) maximal expiratory pressure relationships with the peak of oxygen uptake.

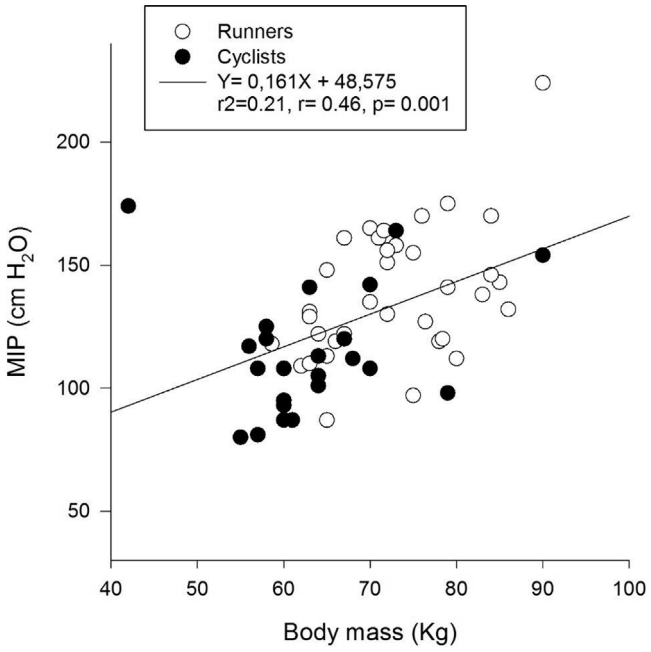


**Fig. 3.** Maximal inspiratory pressure and body mass relationships in runners and cyclists.

**Table 3**
Changes in maximal respiratory pressure after the maximal incremental test.

| | Runners | | Cyclists | |
|---|---|---|---|---|
| | Pre- | Post- | Pre- | Post- |
| MIP, cm $H_2O$ | 140 ± 25 | 120 ± 25* | 115 ± 26‡ | 103 ± 27‡,* |
| MEP, cm $H_2O$ | 172 ± 27 | 148 ± 25* | 146 ± 33‡ | 125 ± 37‡,* |

MIP, maximal inspiratory pressure; MEP, maximal expiratory pressure. Data are expressed as mean ± SD.

* $p < 0.05$ between pre- post- test.

‡ $p < 0.05$ between group.

However, it is less obvious to conclude that the decline in respiratory muscle strength corresponded to the occurrence respiratory muscle fatigue. Janssens et al. (2013) have demonstrated in a systemic review that the decrease in respiratory muscle function was wide-ranging between studies from 5 to 67%. Generally, the significant decrease from pre- exercise values was sufficient to define respiratory muscle fatigue (Lomax and Castle, 2011; Lomax and McConnell, 2003). Sometimes, a critical threshold of 10% (Luo et al., 2001; Mador et al., 1996) and 15% (Mador et al., 2002) for the decrease in respiratory muscle strength were used to conclude respiratory muscle fatigue. In general the 13% reduction in MIP and MEP might be indicative for a decrease in respiratory muscle function as a result of peripheral respiratory muscle fatigue per se and/or decrease in central command.

Given that we found a weak but significant relationship between MIP/MEP at rest and $VO_{2peak}$ (MIP: $r = 0.34$, $p < 0.01$ and MEP: $r = 0.36$, $p < 0.01$), it appears that respiratory muscle function might be a limiting factor in the performance of an individual during maximal incremental testing. It is well known that ventilation strongly (from $\sim 6\ L\ min^{-1}$ to 150–200 $L\ min^{-1}$ in trained athletes) increases during incremental exercise to meet the increasing metabolic demand (Aaron et al., 1992). The hyperpnoea accompanying rhythmic exercise of increasing intensity is driven by strong feedforward central command influences in combination with feedback contributions originating from the carotid chemoreceptors, respiratory muscle metaboreceptors and locomotor muscle group III–IV afferents (Dempsey, 2012). In this way, minute ventilation shows two distinct disproportionate increases (i.e., the first and second ventilatory thresholds), not related to the increase in metabolic demand per se, which also result in additional recruitment of respiratory muscles (Segizbaeva et al., 2013). This exercise hyperpnoea accounts for 8–12% of $VO_{2peak}$ values, with values of > 15% in trained individuals (Aaron et al., 1992; Vogiatzis et al., 2008). In this context it has been shown that the oxygenation level of the respiratory muscles starts to decrease disproportionally in close correspondence with the second ventilatory threshold (Legrand et al., 2007). If the increased respiratory muscle recruitment is sustained for several minutes it can induce respiratory muscle fatigue (Babcock et al., 1998; Johnson et al., 1993). It is well documented that respiratory muscle fatigue occurs during heavy exercise (> 80% $VO_{2max}$) sustained for 8–10 min (Oueslati et al., 2017; Smith et al., 2014), but also during high exercise intensity of shorter duration (Ohya et al., 2015). In this context, this is the first study to investigate the respiratory muscle fatigue (assessed from MIP and MEP) occurrence non-invasively during a maximal incremental exercise in a large population of trained cyclists (n = 23) and runners (n = 36).

There are several possible mechanisms underpinning the limiting effect of respiratory muscle function on exercise tolerance. First, it has been shown that respiratory muscle fatigue can induce a respiratory

steal phenomenon, by which a sympathetically mediated vasoconstriction limits blood flow and $QO_2$ to the locomotor muscles in favor of the respiratory muscles, i.e., the respiratory metaboreflex (Dominelli et al., 2017; Smith et al., 2016, 2017; Vogiatzis et al., 2009). This phenomenon might therefore, stimulate locomotor muscle fatigue and limit exercise tolerance. It should be noted however, that Romer et al. (2007) strongly question the occurrence of the respiratory metaboreflex to incremental exercise since the duration of exercise test, and especially the part of the test at heavy intensities, is too limited to induce respiratory muscle fatigue. In the present study the runners performed ~9 min of exercise at a VE > 100 L min$^{-1}$, whereas this was only 2–3 min in the cyclists. Surprisingly the decrease in MIP and MEP was similar between the runners (13 ± 7% and 13 ± 5%) and cyclists (17 ± 11% and 15 ± 10%) indicating the duration alone might only partly explain the occurrence of respiratory muscle fatigue. It is possible that the exercise modality, and more specifically body posture, might induce a different recruitment pattern of the respiratory muscles between running and cycling. In this context, Hue et al. (2000) observed that ventilator pattern, at exhaustion, was different between maximal running (respiratory rate: 57 ± 11 breath min$^{-1}$; tidal volume: 2.60 ± 0.41 L) and cycling (respiratory rate: 48 ± 7 breath min$^{-1}$; tidal volume: 3.00 ± 0.55 L). Additionally, Tanner et al. (2014) reported greater end-expiratory (3.40 ± 0.54 *vs.* 3.21 ± 0.55 L) and end-inspiratory (6.24 ± 0.88 *vs.* 5.90 ± 0.74 L) lung volumes during maximal cycling compared to running test for the same participants. Assuming that the subjects had a similar training program with an equal intensity distribution, the findings is not surprising, and in line with the study of Ohya et al. (2016), in which middle-distance runners (142.7 cm $H_2O$; 24.1 ± 3.2 years) had higher MIP values compared to road cyclists (109 cm $H_2O$; 20.8 ± 0.9 years). Interestingly, Ohya et al. (2016) observed a correlation between MIP and body mass ($r = 0.59$, $p < 0.001$). Similarly, a significant correlation was observed in the present study between MIP and body mass ($r = 0.46$, $p < 0.001$). On the other hand, the differences in anthropometric characteristics (i.e., the runners have a higher body weight) and age (the runners are old) might also be responsible for the differences in MIP and MEP, and thus hinder a valid comparison of MIP and MEP between the groups. In this context, when the MIP was corrected by body mass, a lack of difference in athlete's MIP rest value between groups was observed.

Second, it is also possible that the decrease in MIP and MEP is related to a decrease in the central command to the respiratory and/or locomotor muscles. It has been shown from measurements during incremental exercise that brain oxygenation (i.e., at the site of the motor cortex) starts to decrease at an intensity corresponding to the second ventilatory threshold (Boone et al., 2016; Vandekerckhove et al., 2016). At this point, the hyperventilatory response induced a reduction in arterial $CO_2$ pressure (i.e. hypocapnia) to minimize the disturbance of the acid-base balance. Cerebral blood flow and thus $QO_2$ to the brain will be negatively affected by this hypocapnia which might in turn reduce central command to the respiratory and/or locomotor muscles. This can induce both a decrease in MIP and MEP following maximal exercise as well as set a limitation to exercise tolerance in general by reducing the motor command to the locomotor muscles. It should be noted that, in this case, the exercise tolerance is not related to respiratory muscle fatigue per se, but rather to the impact of hypocapnia inducing hyperventilation.

A third possible mechanism by which respiratory muscle function can affect exercise tolerance to maximal incremental ramp exercise, is by an exercise-induced arterial hypoxemia. In some highly trained subjects it has been shown that the transit time of the blood through the capillaries surrounding the alveoli is too short to fully saturate the blood (Constantini et al., 2017; Dominelli et al., 2014; Kyparos et al., 2012). This will affect the $O_2$ delivery to the respiratory and/or locomotor muscles and might in this way set a limitation to the maximal effort of an incremental exercise test by enhance locomotor muscle fatigue. Additionally, it can also stimulate respiratory muscle fatigue and thus underpin the decrease in MIP and MEP following incremental exercise. Again, it should be noted that this possible limitation to incremental exercise is not driven by respiratory muscle fatigue per se, although the limitation is strongly relation to the functioning of ventilation.

Given the potential role of the respiratory muscle function as a factor limiting exercise performance even during incremental exercise tests, it can be argued that inspiratory muscle training might have beneficial effects even during this type of exercise. It has been shown that inspiratory muscle training can reduce the metabolic cost of hyperpnea during high intensity exercise and in this way delay respiratory muscle fatigue (and the occurrence of the respiratory metaboreflex). As such the blood flow to the locomotor muscles can be maintained, which will have beneficial effects on exercise tolerance (Witt et al., 2007). However, a systematic review of Illi et al. (Illi et al., 2012) showed that exercise tolerance only increased during high intensity constant load tests (> 80% $VO_{2max}$) and time trials but not during incremental exercise tests. As suggested above, the duration of the high intensity exercise during incremental exercise could be too limited to induce respiratory muscle fatigue (Romer et al., 2007). Taking the results of the present study into account with a large variability in the decrease in MIP and MEP, it seems possible that some but not all subjects could benefit from inspiratory muscle training as it can be suggested that respiratory muscle fatigue limited exercise tolerance only in some subjects.

## 5. Conclusion

This study demonstrates that respiratory muscle strength decreases after maximal incremental exercise in trained runners and cyclists. Based on these findings, coaches and sports scientists would be wise to prescribe specific inspiratory muscle training or warm-up in an effort to improve respiratory muscle strength and thereby reduce the inevitable respiratory muscle fatigue associated with maximal efforts running and cycling.

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