


PERSPECTIVES

The misuse of respiratory resistive loading during aerobic exercises: revisiting mechanisms of “standalone” inspiratory muscle training

 **Gabriel Dias Rodrigues^{1,2} and Alison K. McConnell³**

¹Department of Clinical Sciences and Community Health, University of Milan, Milan, Italy; ²Federal Fluminense University, Niterói, Rio de Janeiro, Brazil; and ³Christchurch, United Kingdom

Abstract

Systematic reviews and meta-analyses support the benefits of inspiratory muscle training (IMT) for sports and clinical populations. A typical application of “standalone” IMT intervention consists of breathing against an inspiratory load (IRL), twice daily, for 5–7 days/wk, for 4–12 wk. However, the application of IRL during aerobic exercise is often seen in a training routine of sports and rehabilitation centers with no evidence-based guide. In this Perspective, we will revisit putative mechanisms underlying the established benefits of “standalone” IMT to support our contention that IMT need not and should not be used during aerobic exercise.

COPD; respiratory muscle training; respiratory muscles; respiratory muscles metaboreflex

INTRODUCTION

The application of “standalone” respiratory muscle training (RMT) to clinical and athletic populations is now supported by a plethora of systematic reviews and meta-analyses supporting its benefits (1). The most commonly applied method of RMT adopts the principles of strength training, whereby brief bouts of moderate-intensity respiratory loading (40%–60% of maximal strength) are applied, resulting variously in improvements in respiratory muscle strength, power, shortening velocity, and endurance (2). Among RMT modalities, inspiratory muscle training (IMT) is the most commonly employed method for healthy (1, 3) and clinical (4, 5) populations. A typical IMT intervention comprises breathing against an inspiratory load (IRL), twice daily, for 5–7 days/wk, for 4–12 wk (3–5). Recently, a high-intensity inspiratory muscle strength training (IMST) protocol that required 30 breaths (~5 min) per session, for 5–7 days/wk (25–35 total min) for 6 wk, reduced resting blood pressure in young healthy adults (6), midlife/older adults (7), and older adults with obstructive sleep apnea (8). Behind its mechanisms, IMST improved endothelial function, nitric oxide bioavailability, and oxidative stress (7) and reduced muscle sympathetic nerve activity (8).

IMT has been included as a component of exercise-based cardiopulmonary rehabilitation programs. Indeed, randomized controlled trials (RCTs) suggested an additive effect of inspiratory muscle training (IMT) and aerobic exercise (AE) training in chronic heart failure (CHF) and chronic obstructive pulmonary disease (COPD) if conducted in independent sessions (9, 10). IMT plus AE training improved cardiorespiratory responses to exercise in CHF (9), while providing additional gains in endurance time and reduced dyspnea in COPD (10).

However, no study supports the inspiratory loading use during AE in either sporting or clinical populations. In this Perspective, we revisit putative mechanisms underlying the established benefits of “standalone” IMT to support our contention that IMT need not and should not be used during AE sessions.

MECHANISM 1: THE RESPIRATORY MUSCLE METABOREFLEX

During heavy-intensity exercise, respiratory muscle work requires an average of 14%–21% of the cardiac output, potentially “competing” for blood flow (e.g., oxygen and nutrients) with locomotor muscles. The mechanism by which blood flow competition arises is suggested to occur via the so-called “respiratory muscle metaboreflex” (11), whereby the fatiguing activity of the inspiratory muscles leads to accumulation of metabolites, which stimulate unmyelinated afferents. This stimulation induces a sympathetically mediated vasoconstriction within limb locomotor muscles (12), which hastens locomotor muscle fatigue and exercise limitation (12) and intensifies effort perceptions. IMT has been shown to increase the threshold for respiratory metaboreflex activation in healthy individuals (13) and patients with heart failure (5), thereby improving exercise tolerance.

MECHANISM 2: REDUCED RESPIRATORY AND LOCOMOTOR EFFORT PERCEPTIONS

The hypothesis that IMT improves exercise performance may be explained, in part, by the reduced respiratory and whole body effort perceptions in athletes (14) and clinical

populations (4) after IMT. The reduced dyspnea seems to be associated with improvements in the force-generating capacity of inspiratory muscles after IMT, which decreases the relative tension for a given level of ventilation (4). During conditions where elevated ventilation is needed, such as exercise, this adaptation from inspiratory muscles most likely underpins the diminished respiratory effort (4). IMT also reduces peripheral effort sensations (14), probably via a reduction in respiratory muscle blood flow needs and boosting of oxygen delivery to and metabolite removal from the limbs.

MECHANISM 3: REDUCED OXYGEN COST OF BREATHING

The oxygen cost of breathing is related directly to the energy requirement of the respiratory muscles. IMT reduces the oxygen cost of breathing for a given ventilatory requirement during voluntary hyperpnea post-IMT (15). Specifically, in highly trained cyclists, IMT reduced the oxygen cost of breathing at ventilations above 50% of the maximal oxygen uptake (15). It suggests that IMT, at least in this population, reduces the energy demand from respiratory muscles during hyperpnea.

Thus, in trained individuals, a reduced oxygen cost of breathing most likely contributes to exercise performance improvements following IMT by reducing their demand for oxygen, thereby liberating oxygen for use by locomotor muscles. To our knowledge, this mechanism has yet to be evaluated in clinical populations.

MECHANISM 4: ATTENUATION OF CENTRAL FATIGUE

Finally, we proffer an as-yet-untested mechanism underpinning the ergogenic effect of IMT. Exercise intolerance is elicited by a number of interrelated peripheral factors but is also affected by so-called “central fatigue,” which is driven by feedback from muscle afferents (16). To date, the focus of attention has been on feedback originating from locomotor muscles; however, feedback from the respiratory muscles almost certainly contributes to the ensemble of inputs influencing central motor drive. Accordingly, we suggest that attenuation of respiratory muscle afferent feedback following

IMT might delay, or attenuate, central fatigue, thereby improving exercise tolerance. To our knowledge, this has yet to be evaluated directly.

DOES INSPIRATORY LOADING DURING EXERCISE HAVE AN ERGOGENIC EFFECT?

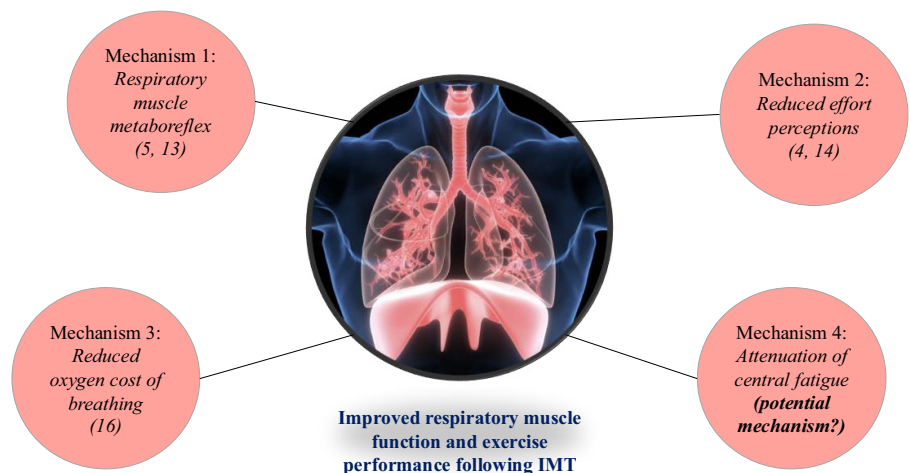
Finally, we close by considering how knowledge of putative mechanisms described above should influence the way in which IMT is implemented in practice; specifically, is there any rationale for loading to be applied during exercise?

A recent systematic review (17) of 19 studies concluded that inspiratory loading during AE impairs exercise tolerance due to an inadequate ventilatory (\dot{V}_E) response. Among the 12 studies that reported peripheral oxygen saturation (SpO_2), 7 showed a decrease in SpO_2 . On the contrary, when inspiratory loading was applied during recovery from high-intensity interval training, a positive effect was found upon clearance of lactate. The psychophysiological effects of imposing respiratory loads during exercise are negative and well established (17), including increased breathing discomfort, anxiety, and intensification of effort during AE.

Accordingly, we argue that imposing an ergogenic intervention, such as inspiratory loading, during exercise has the same pitfalls as training at high altitude; specifically, it impairs the quality of the training that can be accomplished, while not providing any additional benefits to sea-level performance (18). Altitude researchers realized quickly that the benefits of altitude training could be optimized by adopting the so-called “live-high-train-low” paradigm, whereby the erythropoietic benefits of altitude exposure were achieved without compromising athletes’ training quality (18).

Our understanding of the mechanisms that underpin the ergogenic effects of RMT (see Fig. 1), as well as the conclusions of the systematic review by López-Pérez (17), led to the inevitable conclusion that implementing RMT via respiratory loading during AE sessions is a mistake. Moreover, this combination may lead to harmful psychophysiological effects, while also being ineffective (17). In contrast, there is ample evidence that the implementation of “standalone” IMT using resistance-training principles is well tolerated and highly effective for a wide range of individuals (19).

Figure 1. Putative mechanisms underlying improved respiratory muscle function and exercise performance following “standalone” inspiratory muscle training (IMT). “?” indicates that this mechanism is yet to be investigated.



The results of RCTs suggest that IMT is a key part of exercise-based cardiac and pulmonary rehabilitation programs. However, in these studies, IMT protocols were conducted in separate sessions, and they provided benefits in cardiorespiratory responses to exercise and reduced dyspnea in patients with CHF (9) and COPD (10), respectively. Based on the current literature, it is recommended to combine IMT with AE in separate sessions. In summary, using inspiratory loading during AE may have a negative impact, whereas performing IMT and AE in separate sessions allows for sufficient recovery and may lead to additional physiological enhancements compared with just doing IMT or AE training alone.

CONCLUSIONS AND FUTURE DIRECTIONS

Finally, there is one context in which we recommend further research into the effects of combining RMT with other forms of exercise. A single study has investigated the combination of IMT with whole body, “functional” strength training (19). The data suggested that a standalone IMT provided a foundation for improved inspiratory muscle strength and core muscle functions. Furthermore, a follow-on program of IMT, combined with simultaneous core muscle training exercises, enhanced core muscle function further, as well as providing cumulative benefits to running performance. The mechanistic rationale for this approach is the role of the respiratory musculature in trunk stabilization (2) and in balance (20). To underscore, a strong foundation of standalone IMT was the first step of the aforementioned study and, in our opinion, is crucial to support the combination of IMT with other forms of strength training.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

G.D.R. prepared figures; G.D.R. drafted manuscript; G.D.R. and A.K.M. edited and revised manuscript; G.D.R. and A.K.M. approved final version of manuscript.

REFERENCES

1. Illi SK, Held U, Frank I, Spengler CM. Effect of respiratory muscle training on exercise performance in healthy individuals: a systematic review and meta-analysis. *Sports Med* 42: 707–724, 2012. doi:10.1007/BF03262290.
2. McConnell AK. *Respiratory Muscle Training: Theory and Practice* (1st ed.). Oxford: Churchill Livingstone, 2013, p. 256.
3. Rodrigues GD, Lago PD, da Silva Soares PP. Time-dependent effects of inspiratory muscle training and detraining on cardiac autonomic control in older women. *Exp Gerontol* 150: 111357, 2021. doi:10.1016/j.exger.2021.111357.
4. Langer D, Ciavaglia C, Faisal A, Webb KA, Neder JA, Gosselink R, Dacha S, Topalovic M, Ivanova A, O'Donnell DE. Inspiratory muscle training reduces diaphragm activation and dyspnea during exercise in COPD. *J Appl Physiol* (1985) 125: 381–392, 2018. doi:10.1152/japplphysiol.01078.2017.
5. Moreno AM, Toledo-Arruda AC, Lima JS, Duarte CS, Villacorta H, Nóbrega ACL. Inspiratory muscle training improves intercostal and forearm muscle oxygenation in patients with chronic heart failure: evidence of the origin of the respiratory metaboreflex. *J Card Fail* 23: 672–679, 2017. doi:10.1016/j.cardfail.2017.05.003.
6. DeLucia CM, De Asis RM, Bailey EF. Daily inspiratory muscle training lowers blood pressure and vascular resistance in healthy men and women. *Exp Physiol* 103: 201–211, 2018. doi:10.1113/EP086641.
7. Craighead DH, Heinbockel TC, Freeberg KA, Rossman MJ, Jackman RA, Jankowski LR, Hamilton MN, Ziemba BP, Reisz JA, D'Alessandro A, Brewster LM, DeSouza CA, You Z, Chonchol M, Bailey EF, Seals DR. Time-efficient inspiratory muscle strength training lowers blood pressure and improves endothelial function, NO bioavailability, and oxidative stress in midlife/older adults with above-normal blood pressure. *J Am Heart Assoc* 10: e020980, 2021. doi:10.1161/JAHA.121.020980.
8. Ramos-Barrera GE, DeLucia CM, Bailey EF. Inspiratory muscle strength training lowers blood pressure and sympathetic activity in older adults with OSA: a randomized controlled pilot trial. *J Appl Physiol* (1985) 129: 449–458, 2020 [Erratum in *J Appl Physiol* (1985) 132: 1591, 2022]. doi:10.1152/japplphysiol.00024.2020_cor.
9. Winkelmann ER, Chiappa GR, Lima CO, Viecili PR, Stein R, Ribeiro JP. Addition of inspiratory muscle training to aerobic training improves cardiorespiratory responses to exercise in patients with heart failure and inspiratory muscle weakness. *Am Heart J* 158: 768.e1–768.e7, 2009. doi:10.1016/j.ahj.2009.09.005.
10. Charusisin N, Gosselink R, Decramer M, Demeyer H, McConnell A, Saey D, Maltais F, Derom E, Vermeersch S, Heijdra YF, van Helvoort H, Garms L, Schneeberger T, Kenn K, Gloeckl R, Langer D. Randomised controlled trial of adjunctive inspiratory muscle training for patients with COPD. *Thorax* 73: 942–950, 2018. doi:10.1136/thoraxjnl-2017-211417.
11. Dempsey JA, Romer L, Rodman J, Miller J, Smith C. Consequences of exercise-induced respiratory muscle work. *Respir Physiol Neurobiol* 151: 242–250, 2006. doi:10.1016/j.resp.2005.12.015.
12. McConnell AK, Lomax M. The influence of inspiratory muscle work history and specific inspiratory muscle training upon human limb muscle fatigue. *J Physiol* 577: 445–457, 2006. doi:10.1113/jphysiol.2006.117614.
13. Witt JD, Guenette JA, Rupert JL, McKenzie DC, Sheel AW. Inspiratory muscle training attenuates the human respiratory muscle metaboreflex. *J Physiol* 584: 1019–1028, 2007. doi:10.1113/jphysiol.2007.140855.
14. Romer LM, McConnell AJ, Jones DA. Effects of inspiratory muscle training upon time trial performance in trained cyclists. *J Sports Sci* 20: 547–562, 2002. doi:10.1080/026404102760000053.
15. Turner LA, Tecklenburg-Lund SL, Chapman RF, Stager JM, Wilhite DP, Mickleborough TD. Inspiratory muscle training lowers the oxygen cost of voluntary hyperpnea. *J Appl Physiol* (1985) 112: 127–134, 2012. doi:10.1152/japplphysiol.00954.2011.
16. Amann M. Significance of Group III and IV muscle afferents for the endurance exercising human. *Clin Exp Pharmacol Physiol* 39: 831–835, 2012. doi:10.1111/j.1440-1681.2012.05681.x.
17. López-Pérez ME, Romero-Arenas S, Giráldez-García MA, Colomer-Poveda D, Márquez G. Acute psychophysiological responses during exercise while using resistive respiratory devices: A systematic review. *Physiol Behav* 256: 113968, 2022. doi:10.1016/j.physbeh.2022.113968.
18. Beijder J, Nordsborg NB. Specificity of “Live High-Train Low” altitude training on exercise performance. *Exerc Sport Sci Rev* 46: 129–136, 2018. doi:10.1249/JES.0000000000000144.
19. Tong TK, McConnell AK, Lin H, Nie J, Zhang H, Wang J. “Functional” inspiratory and core muscle training enhances running performance and economy. *J Strength Cond Res* 30: 2942–2951, 2016. doi:10.1519/JSC.0000000000000656.
20. Ferraro FV, Gavin JP, Wainwright TW, McConnell AK. Association between inspiratory muscle function and balance ability in older people: a pooled data analysis before and after inspiratory muscle training. *J Aging Phys Act* 30: 421–433, 2022. doi:10.1123/japa.2020-0507.