Title: Response

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We thank Chiappa et al. for commending our work (2,3,7), which we reciprocate in light of their thought-provoking research (4,5) that sparked the ensuing trans-Atlantic debate on the effects of inspiratory muscle loading on lactate clearance after exercise. Specifically, despite using similar methodologies, Chiappa et al. have twice shown accelerated lactate clearance with inspiratory loading (4,5), whereas we have twice shown no effect (2,7). We hypothesised that these discrepancies may be due to inter-study differences in participant endurance training status, as evidenced by higher $\dot{V}O_2$peak and faster blood lactate recovery kinetics in our participants. We were thus intrigued by the authors' unpublished data showing, in sedentary individuals, no effect of inspiratory loading on lactate clearance. In their accompanying figure the authors also present novel data showing a significant correlation between maximal inspiratory pressure (MIP) and changes in the area under the blood [La^-] curve with inspiratory loading. This observation informed their hypothesis that the efficacy of inspiratory loading is influenced by inspiratory muscle mass rather than training status.

We note three important observations from their data. Firstly, the authors report, for two similarly aged groups, MIP's of 166 cmH_2O (92% of predicted) and 106 cmH_2O (88% of predicted); these values seem discordant with commonly used predictive formulae and the similarity in % predicted values is curious given the large differences in MIP. Secondly, enhanced lactate clearance with inspiratory loading is evident in all participants irrespective of MIP; and thirdly, although their figure is, unfortunately, void of MIP measures within ~120-155 cmH_2O, substantial lactate clearance with inspiratory loading is evident when MIP exceeds ~155 cmH_2O. In light of these observations we also revisited our original data (2,7). Interestingly, and contrary to Chiappa et al., we did not observe a relationship between MIP and changes in the area under the blood [La^-] curve with inspiratory loading ($r = 0.22$) (Fig. 1), nor was greater lactate clearance evident in participants with MIP above 155 cmH_2O. Therefore, our data do not support the notion that the efficacy of inspiratory loading is influenced by inspiratory muscle mass. We also feel that MIP may not be a particularly strong indicator of inspiratory muscle mass: only 38% of the variance in MIP was explained by diaphragm cross-sectional area (8). It also seems unlikely that an increase in MIP from ~105 to 155 cmH_2O would correspond to an increase in inspiratory muscle mass sufficient to elicit an approximate four-fold increase in blood lactate clearance with inspiratory loading, as Chiappa et al.'s scattergram suggests. Thus after inspiratory muscle training greater lactate clearance with inspiratory loading is more likely explained by an increased inspiratory muscle oxidative capacity (1,2), rather than a modest increase in inspiratory muscle mass (6).

The lively debate on the efficacy of inspiratory muscle loading during recovery therefore continues. Whilst the role of inspiratory muscle mass remains uncertain, Chiappa et al.'s unpublished data in sedentary individuals have taken us a step closer to explaining the inter-study discrepancies. We envisage that further work will provide more definitive answers.

REFERENCES

1. Brown PI, Sharpe GR, Johnson MA. Inspiratory muscle training reduces blood lactate concentration during volitional hyperpnoea. *Eur J Appl Physiol*. 2008;104:111-7.
2. Brown PI, Sharpe GR, Johnson MA. Loading of trained inspiratory muscles speeds lactate recovery kinetics. *Med Sci Sports Exerc*. 2010;42:1103-12.
3. Brown PI, Sharpe GR, Johnson MA. Inspiratory muscle training abolishes the blood lactate increase associated with volitional hyperpnoea superimposed on exercise and accelerates lactate and oxygen uptake kinetics at the onset of exercise. *Eur J Appl Physiol*. 2012;112:2117-9.
4. Chiappa GR, Ribeiro JP, Alves CN, et al. Inspiratory resistive loading after all-out exercise improves subsequent performance. *Eur J Appl Physiol*. 2009;106:297-303.

5. Chiappa GR, Roseguini BT, Alves CN, Ferlin EL, Neder JA, Ribeiro JP. Blood lactate during recovery from intense exercise: impact of inspiratory loading. *Med Sci Sports Exerc*. 2008;40:111-6.
6. Downey AE, Chenoweth LM, Townsend DK, Ranum JD, Ferguson CS, Harms CA. Effects of inspiratory muscle training on exercise responses in normoxia and hypoxia. *Respir Physiol Neurobiol*. 2007;156:137-46.
7. Johnson MA, Mills DE, Brown DM, Bayfield KJ, Gonzalez JT, Sharpe GR. Inspiratory loading intensity does not influence lactate clearance during recovery. *Med Sci Sports Exerc*. 2012;44:863-71.
8. McCool FD, Conomos P, Benditt JO, Cohn D, Sherman CB, Hoppin FG. Maximal inspiratory pressures and dimensions of the diaphragm. *Am J Respir Crit Care Med*. 1997;155:1329-34.

Figure Caption

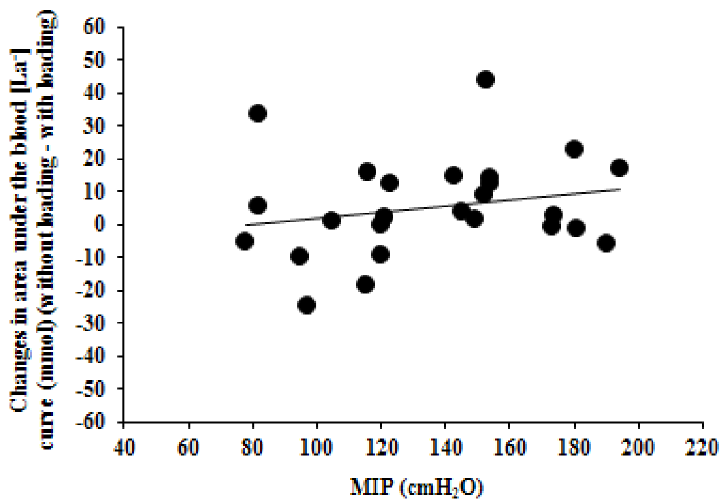

Figure 1. The association between maximal inspiratory pressure (MIP) and the change in the area under the blood [La$^-$] curve after exercise with inspiratory muscle loading.