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Letter to the Editor

BREATHING 100% O_2 HAS NO EFFECT ON BLOOD LACTATE CONCENTRATION DURING A SHORT PASSIVE RECOVERY FROM EXHAUSTIVE EXERCISE

Dear Editor-in-Chief

Many researchers (e.g. Coffey et al., 2004) have indicated that recovery from acute exercise induced muscular fatigue could be expedited by increased rapidity of lactate (LA^-) clearance from the blood. This argument is based on the following logical progression: Firstly, increased intra-myocellular LA^- concentration has been proposed to exert various deleterious electrochemical influences over excitation/contraction coupling and metabolic function (e.g. Favero et al., 1997). Secondly, because LA^- is extruded from the muscle cells to the blood in a concentration gradient dependent fashion (Mengual et al., 2003); lowered blood LA^- concentration should therefore allow increased rapidity of myocellular LA^- export. Finally, LA^- accumulation is continually cited as having a causal relationship with exercise induced acidosis; and further that such acidosis is deleterious to muscular function (for review see Pedersen et al., 2004). Given that protons are co-transported out of the muscle cells with LA^- at a 1:1 ratio (Mengual et al., 2003); it may appear this is another reason for suggesting increased LA^- extrusion rate could be beneficial.

Several challenges to the above logic can be made: Firstly, the negative effects of increased LA^- concentration alluded to above are absent at physiological pH and temperature *in situ*: at concentrations as high as 30 $mMol \cdot L^{-1}$ (for review, see Allen and Westerblad, 2001). Secondly, at higher intra-myocellular LA^- concentrations, pyruvate is imported from the blood to rebalance redox and metabolic equilibria, including the ratio of NAD^+:$NADH + H^+$ (Mengual et al., 2003). This process therefore theoretically counteracts the proposed need to remove LA^- from the blood in order to facilitate continued myocellular LA^- efflux. Furthermore, LA^- accumulation is not causally linked to acidification (Robergs et al., 2004), and there is evidence that acidification is beneficial to muscular function in any case (Pedersen et al., 2004).

Nonetheless, past research has focussed on methods by which the clearance of LA^- from the blood could be expedited. As the clearance of LA^- from the blood occurs primarily due to import and oxidation by other cells (Mengual et al., 2003), methods trialled include breathing hyperoxic gas mixtures during recovery (Maeda and Yasukouchi, 1997; Murphy, 1986; Shell et al., 1986). It has been argued, however, that due to the near horizontal nature of the oxyhaemoglobin dissociation curve (i.e. 95%+ O_2 saturated) at normal alveolar PO_2 (~95 mmHg); it appears unlikely that the large increase in alveolar PO_2 caused by breathing 100% O_2 (667% % increase over ambient air) would be effective in raising actual oxygen delivery to the mitochondria by a useful margin. However, Haseler et al. (1999) have shown that increasing the inspired O_2 percentage to 100% during a passive recovery from exercise significantly reduced the time constant for phosphocreatine (PCr) repletion (20-s vs. 25-s, $p < 0.05$). Given that PCr repletion is dependent on ATP, these findings provide surety that O_2 delivery to, and uptake by the mitochondria is indeed usefully increased during passive recovery by breathing 100% O_2 as compared with ~21% O_2 (Haseler et al., 1999).

We therefore undertook this investigation because previous methodologies and results regarding hyperoxic breathing and blood LA^- concentration (Maeda and Yasukouchi, 1997; Murphy, 1986; Shell et al., 1986) are somewhat conflicting. Specifically; acute muscular exhaustion was not universally imposed, hyperoxia was often imposed during the exercise also, and subjects of differing aerobic or cardiovascular fitness showed differential responses. Given the above arguments, we intended to clarify the effect of breathing 100% O_2 on blood LA^- concentration during a brief period

of passive recovery from incremental exercise to exhaustion under controlled conditions. We hypothesised that breathing 100% O_2 during a 5-minute passive recovery from exhaustive incremental exercise would not affect the rate of blood LA^- clearance in a relatively fit and homogenous subject pool.

Seven men aged 21 ± 0 years, body mass 83 ± 19 kg [means ± SD]: peak incremental cycling power output 410 ± 19 W, [mean ± SE]) were recruited on the basis of heterogeneity of maximum power output and time to exhaustion (coefficients of variation: 0.12 and 0.11 respectively). On two occasions separated by 7-days, subjects' resting blood LA^- concentrations were determined at the same time of day (YSI-1500 Sport, USA) following 20-minutes of postural stasis and a 12-h fast. An identical maximal-incremental power output cycle ergometer (Lode, Netherlands) protocol was then used on each occasion to elicit both acute muscular exhaustion, and an accumulation blood LA^-. The exercise comprised a fixed cadence of 90 RPM, starting at 50 W and increasing by 50 $W \cdot min^{-1}$ until exhaustion (or until the same period of exercise time had elapsed on the second occasion). Subjects breathed ambient air during both exercise trials. Immediately following the exercise, subjects were assisted to a chair beside the ergometer, where they remained for the next 5-minutes. During one trial, subjects breathed either ambient air or 100% O_2 during the recovery period. The order of trials was randomised. Blood LA^- was analyzed every minute during the 5-minute recovery periods; beginning at time 'zero' (immediately upon being seated post-exercise i.e. six samples per trial).

Figure 1 illustrates the blood LA^- concentration (mean ± SE) for each trial at each reading. Alpha was set at 0.05. The data were subjected to three-way (treatment, time, and treatment x time) ANOVA with repeated measures. The results indicate no significant effect of the treatment ($p = 0.22$), a significant effect of time ($p = 0.0004$), however no interaction was observed ($p = 0.41$). The current results therefore support our hypothesis. We conclude that if expedited LA^- clearance from the blood provides any benefit to recovery from acute muscular exhaustion in relatively fit young men (as elicited by maximal incremental cycle exercise), the current intervention has not assisted in this respect within the recovery time monitored. A longer exposure to 100% O_2 during recovery was not imposed, as we were concerned about the potential for oxygen toxicity. Given that minute ventilation at exhaustion routinely exceeds 150L, as opposed to ~30L at rest (and at ~21% O_2) O_2 toxicity (which appears over several hours at rest while breathing 100% O_2) would be expected to develop much more rapidly.

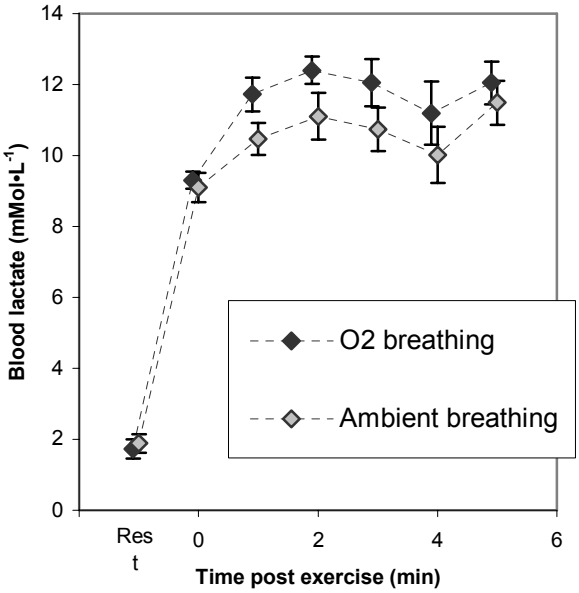

Figure 1. Blood lactate concentration (n = 7, mean ± SE) during a five minute passive recovery from standardised exhaustive exercise; a) when 100% O_2 was inspired, and b) when 21% O_2 (balance N_2) was inspired. Trials proceeded in random counterbalanced order.

The practical utility of breathing 100% O_2 for up to 5-minutes immediately following incrementally elicited acute muscular exhaustion is yet to be determined with respect to repeat exercise performance. If a performance increment is apparent from future investigation, it appears unlikely to be attributable to improved rates of blood LA^- clearance. Longer exposures to 100% O_2 and/or different exercise modalities could be investigated in future if sport specificity justifies it; however the safety of this should be determined first.

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