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The Respiratory System during Intermittent-Sprint Work: Respiratory Muscle Work and the Critical Distribution of Oxygen

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Abstract

In healthy individuals at rest and while performing moderate-intensity exercise, systemic blood flow is distributed to tissues relative to their metabolic oxygen demands. During sustained high-intensity exercise, competition for oxygen delivery arises between locomotor and respiratory muscles, and the heightened metabolic work of breathing, therefore, contributes to limited skeletal muscle oxygenation and contractility. Intriguingly, this does not appear to be the case for intermittent-sprint work. This chapter presents new evidence, based on inspiratory muscle mechanical loading and hypoxic gas breathing, to support that the respiratory system of healthy men is capable of accommodating the oxygen needs of both locomotor and respiratory muscles when work is interspersed with short recovery periods. Only when moderate hypoxemia is induced, substantial oxygen competition arises in favour of the respiratory muscles. These findings extend our understanding of the relationship between mechanical and metabolic limits of varied exercise modes.

Keywords: blood flow, hyperpnoea, metaboreflex, oxygen uptake, hyperventilation, muscle fatigue

1. Introduction

Blood flow to contracting skeletal muscles closely matches their metabolic rate [1, 2]. In humans, it has been robustly demonstrated that there is a positive linear relationship between the rate of oxygen uptake (VO_2) in the quadriceps muscles and blood flow through the femoral artery [1], which ensures there is a match between oxygen (O_2) supply and demand for the exercising muscles. Blood flow is directed to areas in need by adjusting vasoconstriction in the relatively inactive regions and vasodilatation in the active locomotor muscles [2–4]. During high-intensity and maximal exercise, the accompanying increase in cardiac output is almost exclusively devoted to the working skeletal muscle [5], whereas blood flow to the splanchnic, renal and inactive skeletal muscle tissue beds can fall by $\approx 70\%$ from resting values [6, 7]. It is likely that multiple biological factors contribute to biological redundancy in the system [8]. However, there does appear to be a limit to systemic vasodilation, a proactive mechanism to maintain arterial blood pressure and ensure adequate

O₂ supply to vital organs [4, 9–11]. Additionally, when the metabolic demands of multiple muscle groups are high, and cardiac output is nearing maximal flow rates, competition for available blood flow can arise between muscle groups. One such example is the interplay between limb locomotor musculature and the respiratory muscles.

The respiratory muscles perform work to overcome the elastic recoil of the lungs and chest, resistance from turbulent and viscous airflow through the respiratory tract and tissue deformation [12]. As pulmonary ventilation (V_E) rises, there is an exponential increase in the work being performed by the respiratory muscles [12, 13]. This ventilation-induced rise in work of breathing is caused by two factors; (1) dynamic hyperinflation to accommodate greater expiratory flow rates [14], and (2) progressive increase in the contribution of the expiratory muscles to breathing [15]. As the lungs and chest are progressively stretched to accommodate the increasing volume of inhaled air and end-expiratory lung volume is reduced, the contribution of elasticity in these tissues to the work of breathing increases [16, 17]. Accompanying the changes in work of breathing with V_E , there is a certain O₂ cost of exercise hyperpnoea [13, 18]. By mimicking the ventilation pattern (respiratory frequency and tidal volume) obtained during exercise while at rest, it is possible to estimate the proportion of whole-body VO₂ that is devoted to the respiratory muscles. During moderate exercise, the O₂ cost of breathing accounts for 3–6% of the total whole-body VO₂. During high-intensity exercise, the relative contribution of exercise hyperpnoea to whole-body VO₂ is estimated increases to 10–15% and can become a limiting factor of exercise capacity [2, 9, 19–21].

2. Consequences of sustained respiratory muscle work during continuous exercise

The work of breathing associated with high-intensity and maximal exercise is responsible for stealing a considerable portion of whole-body VO₂, which creates an environment where the locomotor and respiratory muscles compete for O₂ delivery [2, 3]. As such, respiratory muscle work, fatigue and metaboreflex are interrelated and suggested to contribute to the development of locomotor muscle fatigue, limiting one's capacity to sustain high-intensity exercise [9, 22].

An inverse relationship exists between the work of breathing and leg O₂ uptake during maximal exercise [3]. To reduce the work of breathing, proportional assist ventilation (PAV) can be used to generate inspiratory pressure proportional to the effort of the patient/subject. Conversely, to elevate inspiratory muscle work, a mesh screen can be placed over the inspiratory line, or the aperture of an inspiratory port can be reduced. In one such study employing these techniques, subjects exercised at a workload sustainable for 2.5–3 min at, or near a work rate corresponding to the attainment of VO_{2max}. The work of breathing was attenuated by 60% with PAV and increased by 95% with inspiratory loading, compared to control during the exercise bout. Elevating the work of breathing had a negligible effect on whole-body VO₂. Moreover, both leg blood flow and VO₂ fell compared to control exercise which coincided with an increase in leg vascular resistance. These data suggest that cardiac output did not increase to accommodate the additional muscular work [2]. It is likely that blood flow was redistributed to the respiratory muscles to support the heightened metabolic activity at the expense of the locomotor muscles [3]. When the respiratory muscles were unloaded with PAV, there was a slight increase in limb blood flow which corresponded with an increase in leg VO₂. Thus, by reducing the metabolic demands of the respiratory muscles, O₂ delivery to the lower limbs can be improved. Taken together, these data indicate that the

'normal' work of breathing incurred during high-intensity exercise may actually be a limiting factor of O_2 transport to the locomotor muscle during high-intensity exercise [3].

Exercise intensity also plays an important role in the competition between the locomotor and respiratory muscles for available O_2 . While exercising at submaximal work rates (50–75% VO_{2max}), there is a small but significant increase in whole-body VO_2 in response to an elevated work of breathing [23]. Since VO_2 responded proportionally to the changes in inspiratory muscle work, it has been concluded that there is enough capacity in cardiac output to increase and meet the demands of additional muscular work during submaximal exercise [3]. It is only during high-intensity exercise when cardiac output approaches maximal flow rates that competition for available blood flow begins to develop [3].

Since the high work of breathing during high-intensity exercise ($>80\%$ VO_{2max}) seems to have a limiting effect on locomotor muscle blood flow, the rate of development of peripheral fatigue is likely affected too. To examine exercise-induced quadriceps muscle fatigue, supra-maximal femoral nerve stimulation can be used to provide an objective measure of muscle force-generating capacity [24]. In one such example, peripheral muscle fatigue was assessed after exercise at a work rate corresponding to the attainment of 92% of VO_{2max} [25]. On one occasion, subjects exercised to volitional exhaustion (13.2 min). On a separate visit, exercise at the same work rate and duration was repeated while the respiratory muscles were unloaded using PAV (56% reduction of inspiratory muscle work). Following the completion of exercise, quadriceps muscle fatigue was 8% greater when subjects were not using breathing assistance. To examine how a heightened work of breathing affects peripheral fatigue, exercise was repeated with inspiratory loading (80% increase of inspiratory muscle work) to exhaustion (7.9 min). Following the termination of exercise, the force-generating capacity of the quadriceps was 8% lower when performed with inspiratory loading compared to control [25]. These data robustly demonstrate that peripheral fatigue can be manipulated by altering the work for breathing, which suggests that respiratory muscle work is a limiting factor of high-intensity exercise [2, 3].

It is usually unlikely for healthy humans to experience inspiratory loading during exercise. However, exposure to (simulated) altitude is a more common environmental condition that will increase the work of breathing compared to normoxia via stimulation of pulmonary ventilation [26, 27]. To examine the relationship between hypoxia-induced elevated work of breathing and peripheral fatigue, subjects exercised at a constant work rate (≈ 273 W) corresponding to 82% of VO_{2max} in simulated altitude (fraction of inspired oxygen, $F_{IO_2} = 0.15$) to exhaustion [28]. Exercise was then repeated at the same work rate for an identical duration in normoxia (≈ 273 W for 8.6 min). Compared to hypoxia, inspiratory muscle work was 36% less when exercising in normoxia and induced a lesser reduction in quadriceps force generation (normoxia –16% vs., hypoxia –30%). To isolate the effects of the work of breathing on peripheral muscle fatigue, subjects repeated both exercise trials (normoxia and hypoxia) using PAV. Inspiratory muscle work was nearly identical during exercise between normoxia and hypoxia with PAV, and the reduction in hypoxia-induced peripheral fatigue was attenuated relative to normoxia (normoxia –15% vs., hypoxia –22%). Combined, these data demonstrate that the development of quadriceps fatigue is accelerated in hypoxia in part due to heightened inspiratory muscle work. Moreover, this occurs at a work rate and exercise duration at which inspiratory muscle work usually does not affect quadriceps fatigue [28]. Sustained exercise $\geq 90\%$ VO_{2max} and the accompanying work of breathing may have to reach a given threshold to elicit meaningful changes in quadriceps fatigue [25].

3. Factors influencing respiratory muscle work during repeated-sprint exercise

Repeated-sprints are characterised by brief 'all-out' exercise bouts of 4–15 s, separated by incomplete recovery periods of 14–30 s [29, 30]. Performance in a repeat-sprint context is therefore represented as the ability to reproduce power output after a previous bout of maximal exercise [31]. Over the course of a repeated-sprint series, there is a progressive decline in total mechanical work performed in each successive sprint. The rate of performance decline is also typically accelerated in low O₂ environments [32]. Initial sprint performance is largely determined by muscular strength and power production [33], whereas the ability to resist fatigue and maintain performance is underpinned by aerobic capacity and the ability to deliver O₂ to the locomotor muscles in the recovery periods between sprint recovery periods [34, 35]. Below, we outline the work load-induced physiological factors known to the work of breathing during intense intermittent exercise.

3.1 Metabolic determinates of repeated-sprint exercise

Resting intramuscular stores of ATP are limited to $\approx 20\text{--}25\text{ mmol}\cdot\text{kg}^{-1}$ of dry muscle weight, which during a sprint, can only provide energy for 1–2 s [36, 37]. As resting ATP stores become depleted, three major energy systems are responsible for ATP resynthesis. Rapid resynthesis is achieved through phosphocreatine (PCr) degradation [36]. Anaerobic glycolysis also has a large involvement in sprint metabolism [36]. Though as sprints are repeated, the relative contribution of anaerobic glycolysis towards ATP resynthesis declines [36, 37]. Conversely, aerobic metabolism has a very small role in isolated sprint performance ($\approx 10\%$ of total ATP production), which increases as sprints are repeated [37, 38].

Intramuscular PCr is especially important for the rapid resynthesis of ATP during explosive activities via the reversible PCr-creatine kinase pathway [39–41]. In the presence of the enzyme creatine kinase, adenosine diphosphate (ADP) is converted to ATP through the dephosphorylation of PCr to form creatine (Cr). It is estimated that during a single 6-s sprint, 50% of anaerobic ATP production is derived predominantly through PCr degradation [36]. The remaining anaerobic energy contribution during an isolated sprint is supported mainly by glycolysis (44%), and in minority by intramuscular ATP stores (6%). When sprints are repeated, the relative contribution of PCr to anaerobic ATP resynthesis increases. By the tenth 6-s sprint (each separated by 30 s passive rest), PCr degradation is estimated to account for 80% of the total anaerobic energy contribution [36]. However, intramuscular PCr stores are limited to $\approx 80\text{ mmol}\cdot\text{kg}^{-1}$ of dry muscle weight, and after only a single 6-s sprint, stores are reduced $\approx 50\%$ from baseline [36, 42]. When multiple sprints are performed, PCr depletion can be up to 75% after 5 repetitions [42], and 84% after 10 [36]. Since PCr degradation has such a large contribution to ATP resynthesis, the recovery of intramuscular stores PCr is critically important to the restoration of power output [43].

The capacity to recover PCr is limited in a multiple sprint series, largely constrained by the short recovery periods between sprints. The rate of PCr resynthesis follows an initial fast phase, followed by a second longer slow component [44]. After a single 6-s sprint, approximately 70% of PCr replenishment is achieved in the first 30 s of passive rest [42]. But as sprints are repeated and muscle stores are further depleted, PCr can only recover to 50% of resting stores after just five repetitions. When rest is extended post a repeat-sprint series, only 80% of PCr is recovered after 3 min [42], and 85% after 6 min of passive rest [45]. Though PCr degradation is an anaerobic process, PCr resynthesis is an aerobic process and is sensitive to O₂ availability [43, 44, 46, 47]. When breathing a hypoxic gas mixture

($F_{I}O_2 = 0.10$), the rate of PCr resynthesis has been demonstrated to be attenuated by 23% [46]. While breathing a hyperoxic gas ($F_{I}O_2 = 1.00$) enhances recovery by 20% compared with normoxia, which suggests that under normal exercise conditions PCr resynthesis is limited by O_2 availability [46]. Therefore, if the work of breathing is high enough to limit locomotor muscle O_2 delivery, PCr resynthesis in repeated-sprint exercise may be impaired.

The energy debt created by the rapid decrease in muscle PCr during a single sprint is met by a sizable contribution of anaerobic glycolysis to ATP resynthesis. Approximately 44% of ATP resynthesis is derived from anaerobic glycolysis during a single 6-s sprint [36]. However, the relative contribution of anaerobic metabolism declines as sprints are repeated [48]. By the tenth sprint, Gaitanos, Williams [36] estimated that glycolysis was only responsible for 16% of total anaerobic ATP production. Moreover, in four of the seven subjects, it was estimated to be zero (range 0–23.1 mmol ATP·kg⁻¹ of dry muscle weight). Many mechanisms play a role in the relative decrease in anaerobic glycolysis during multiple-sprint work. The most likely being the progressive depletion of muscle glycogen that is associated with high-intensity activity [49].

The aerobic contribution to an isolated sprint is minimal since the maximal rate of ATP resynthesis is far below the requirements of maximal sprint work [39]. In an isolated sprint, aerobic metabolism is responsible for ≈10% of total energy production [37, 48]. But as sprints are repeated, the relative increase in aerobic metabolism to total ATP turnover rate rises to compensate for reduced energy supply from anaerobic pathways [38]. Following five 6-s sprints, it is estimated the aerobic energy contribution rises to ≈40% of total ATP production [48]. The remaining 60% is derived from anaerobic pathways, predominantly PCr degradation [36, 42]. Pulmonary VO_2 can fluctuate between 70 and 100% of VO_{2max} from sprint to recovery periods in the latter stages of a repeat-sprint series [50]. When no external work is being performed (i.e., passive rest) during the recovery period between sprints, the elevated VO_2 above baseline is representative of lactate metabolism, removal of inorganic phosphate, and most importantly PCr resynthesis [40, 51].

Aerobic metabolism may have a limited role in ATP formation during multiple sprint work [38, 48], but is fundamental to PCr resynthesis between sprints. Compartment specific creatine kinase isozymes are located in the cytosol and mitochondrial intermembrane space, and are associated with either the ATP-consuming or -delivering process, respectively [40, 41]. In the PCr shuttle system, mitochondrial creatine kinase mediates the reaction between creatine and ATP formed by oxidative metabolism, to generate PCr and ADP [40]. Therefore, the rate at which the mitochondria can generate ATP through oxidative phosphorylation, will dictate PCr resynthesis. A positive correlation between aerobic fitness and maintaining repeat-sprint performance exists [31, 34, 52, 53]. It is likely that improvements in mitochondria function and content, that are associated with exercise training [54], underpin the correlation between aerobic fitness and repeated-sprint ability. Additionally, muscle O_2 availability between sprint efforts likely affects mitochondrial oxidative phosphorylation, which would explain the connection between PCr resynthesis and O_2 availability [43, 46]. Therefore, the ability to deliver O_2 to the locomotor muscles during rest periods between sprints is critical to maintaining maximal sprint performance [35, 47].

3.2 Muscle oxygenation and repeated-sprint exercise

Muscle O_2 availability during repeated-sprint exercise is critical for supporting PCr resynthesis, which underpins the capacity to maintain power out over a sprint series [36, 46]. Changes in local O_2 balance (delivery vs. consumption) can

be measured in real-time with near-infrared spectroscopy (NIRS) [55]. The NIRS technology relies on the relative transparency of biological tissue to near-infrared light (650–950 nm), and light absorption of deoxyhaemoglobin and oxyhaemoglobin [56]. The concentration of deoxyhaemoglobin ([HHb]) and oxyhaemoglobin ([O₂Hb]) rises and falls, respectively, proportional to an increase in metabolic activity in the underlying tissue and display similar kinetics to pulmonary VO₂ [50, 57]. The analysis is typically focused on [HHb] since it is less sensitive to fluctuations in total haemoglobin, is assumed to reflect venous [HHb] and thus muscular oxygen extraction, and because [O₂Hb] is influenced by rapid blood volume and perfusion variations due to the skeletal muscle pump.

Because PCr resynthesis is achieved through oxidative processes [46, 58], the availability of muscle O₂ during rest periods is critically important for metabolic recovery. In maximal voluntary isometric handgrip exercise, reoxygenation rate measured as the rate change of [O₂Hb] during recovery was strongly correlated with the recovery of muscle PCr ($r^2 = 0.939$) [47]. Therefore, factors affecting muscle reoxygenation between sprint efforts will likely affect PCr resynthesis and repeated-sprint performance.

Vastus lateralis reoxygenation capacity can be attenuated by performing low-intensity activity (jogging/cycling) between sprint efforts [50, 59]. By reducing O₂ availability, the restoration of peak cycling power and peak running speed following periods of 'active' recovery is 3–7% lower compared to passive rest. The time to exhaustion is also lowered by performing 'active' recovery when performing 15-s sprints, repeated every 15 s (745 ± 171 s vs. 445 ± 79 s; –60%) [60]. Performing active recovery between sprints, muscle tissue reoxygenation is impaired through the constant O₂ uptake supporting the metabolic requirements of the active recovery. Therefore, PCr resynthesis is likely blunted because ATP from oxidative phosphorylation is devoted directly to maintain muscle contractions, rather than towards PCr resynthesis [41, 59].

The influence of limited reoxygenation on repeated-sprint ability has also been highlighted by manipulating the F_IO₂. When performing ten 10-s sprints with 30 s of passive rest and inspiring a hypoxic gas mixture (F_IO₂ = 0.13), reoxygenation was attenuated by 11% [35]. There was a $\approx 8\%$ reduction in total mechanical work in hypoxia compared to normoxia, and the reduction in work was strongly correlated with the attenuated muscle reoxygenation ($r = 0.78$; 90% confidence interval: 0.49, 0.91). Since PCr resynthesis has similar recovery kinetics to reoxygenation [47], it is likely that muscle PCr recovery was hindered by limited O₂ availability. Therefore, enhancing the capacity to reoxygenation the muscle between sprints is likely to have positive benefits for repeated-sprint ability.

There exists a positive relationship between aerobic fitness and repeated-sprint ability, which may in part be explained by superior reoxygenation capacity [31, 34, 52, 53]. After 8 weeks of endurance training, although the initial sprint performance is typically unaffected [61] (presumably because improvements in aerobic function do not support the anaerobic nature of an isolated sprint), muscle oxygenation was reported to be 152% higher prior to the commencement of the second sprint following training. Consequently, the decrement in performance within the subsequent sprint was attenuated by 26% [61]. It is likely that by improving O₂ delivery to the locomotor muscle, O₂ availability for oxidative phosphorylation was enhanced, and in turn, the phosphocreatine shuttle system [39, 40].

3.3. Heightened inspiratory muscle work

As described in Section 2, respiratory muscle work has been implicated as a limiting factor of limb O₂ perfusion during continuous exercise [9]. However,

competition between locomotor and respiratory muscle for available cardiac output does not appear to be a significant limiting factor of performance during repeated-sprint exercise. In our recent work, we examined the influence of inspiratory muscle loading on oxygenation trends in repeated-sprint exercise [62]. Participants were asked to perform ten 10-s cycle ergometer sprints, each separated by 30 s of passive rest. Inspiratory loading was achieved by placing a plastic disk with a 10-mm opening over the inspiratory side of a two-way non-rebreathing valve (Figure 1). Inspiratory muscle force development (calculated as the integral of inspiratory mouth pressure, multiplied by respiratory frequency) was similar to others who have shown vastus grater lateralis muscle deoxygenation with inspiratory loading during exercise [63]. In response, whole-body VO_2 measured at the mouth was elevated by 4–5% during both the sprint and recovery phases (Figure 2) [62]. This occurred even though total sprint work was similar between the conditions. The elevation in VO_2 was likely driven by a heightened oxygen O_2 uptake by the respiratory muscle to accommodate for the additional inspiratory muscle work [23, 63]. Importantly, Tissue Saturation Index ($\text{TSI} = [\text{O}_2\text{Hb}] \div ([\text{O}_2\text{Hb}] + [\text{HHb}])$, expressed in %) measured at the sixth intercostal space, was comparable between the conditions, which suggests that O_2 supply to the respiratory muscles remained proportional to the metabolic activity. The change in inspiratory muscle work did not translate into compromised vastus lateralis oxygenation.

The intermittent nature of repeated sprints is likely a key mediating factor for which O_2 delivery can be maintained to both locomotor and respiratory



Figure 1.
 Representation of how inspiratory loading was achieved [62]. A plastic disk with a 10-mm opening was placed over the inspiratory side of a two-way non-rebreathing valve (Hans Rudolph Inc., Kansas, United States of America) attached to the distal end of a bidirectional turbine and held in place by the internal ridge of a rubber tubing adaptor. A pressure transducer was attached to the saliva port of the non-rebreathing valve via Tygon tubing to assess inspiratory mouth pressure.

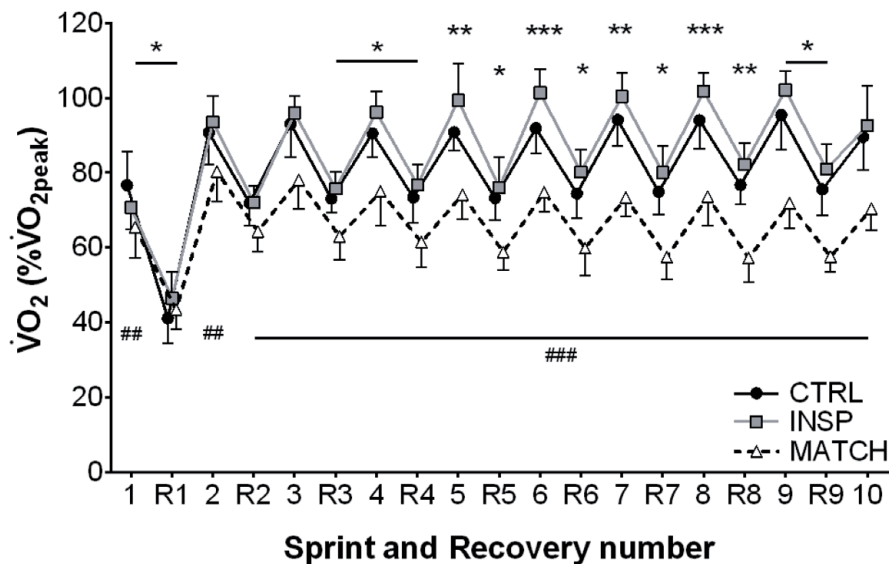


Figure 2.

Sprint and recovery pulmonary oxygen uptake (VO_2) expressed as a percentage of VO_{2peak} for control (CTRL), inspiratory muscle loading (INSP), and worked matched (MATCH) exercise. The symbols represent comparisons between INSP and CTRL (), INSP and MATCH (#). The number of symbols; one, two, and three denote likely, very likely and almost certainly respectively, that the chance of the true effect exceeds a small (-0.2 to 0.2) effect size. Values are presented as mean \pm SD. Reproduced from Rodriguez et al. [62] under a Creative Commons Attribution 4.0 licence.*

muscles. Others have demonstrated that the addition of an inspiratory load while exercising $>95\%$ VO_{2max} , results in a decrease in limb perfusion and O_2 delivery, mediated by sympathetically-activated vasoconstriction in the locomotor muscles [2, 3]. Whereas during moderate intensities ($50\text{--}75\%$ VO_{2max}), there is no change in vascular resistance or blood flow [23]. Even though repeated-sprint exercise can elicit $>90\%$ of VO_2 peak, it is not sustained throughout the entire protocol and can fluctuate between 70 and 90% of VO_{2max} between sprint and recover phases [50, 62]. The fluctuation in metabolic demands between the phases likely minimises the potential for a competition for available cardiac output. Moreover, since VO_2 was able to increase, these data highlight the capacity of the cardiorespiratory system to rapidly adjust and meet the additional metabolic demands imposed by inspiratory loading even during severe exercise [64]. In instances where blood flow is impacted by additional respiratory muscle work, there is no compensatory increase in VO_2 [2, 3]. Therefore, having the capacity to increase VO_2 may be a crucial factor in maintaining O_2 supply to all active muscles during high-intensity exercise and, thereby, sustain prolonged periods of physical activity.

3.4 Acute environmental hypoxia

To further explore the role of O_2 availability in balancing the metabolic demands of the locomotor and respiratory muscles, we asked participants to exercise in an environment where the O_2 concentration had been reduced to 14.55% [65]. Participants completed the same protocol as previously described (ten 10-s sprints, 30 s of passive rest) while vastus lateralis and intercostal muscle oxygenation was assessed with NIRS. Surprisingly, there was no clear difference in repeated-sprint ability in hypoxia compared to normoxia. However, there was a clear reduction in vastus lateralis muscle oxygenation similar to previous research (Figure 3) [35, 66]. Ventilation patterns (respiratory frequency and inspiratory volume) and inspiratory pressure generation were similar between conditions. Therefore, the O_2 cost of

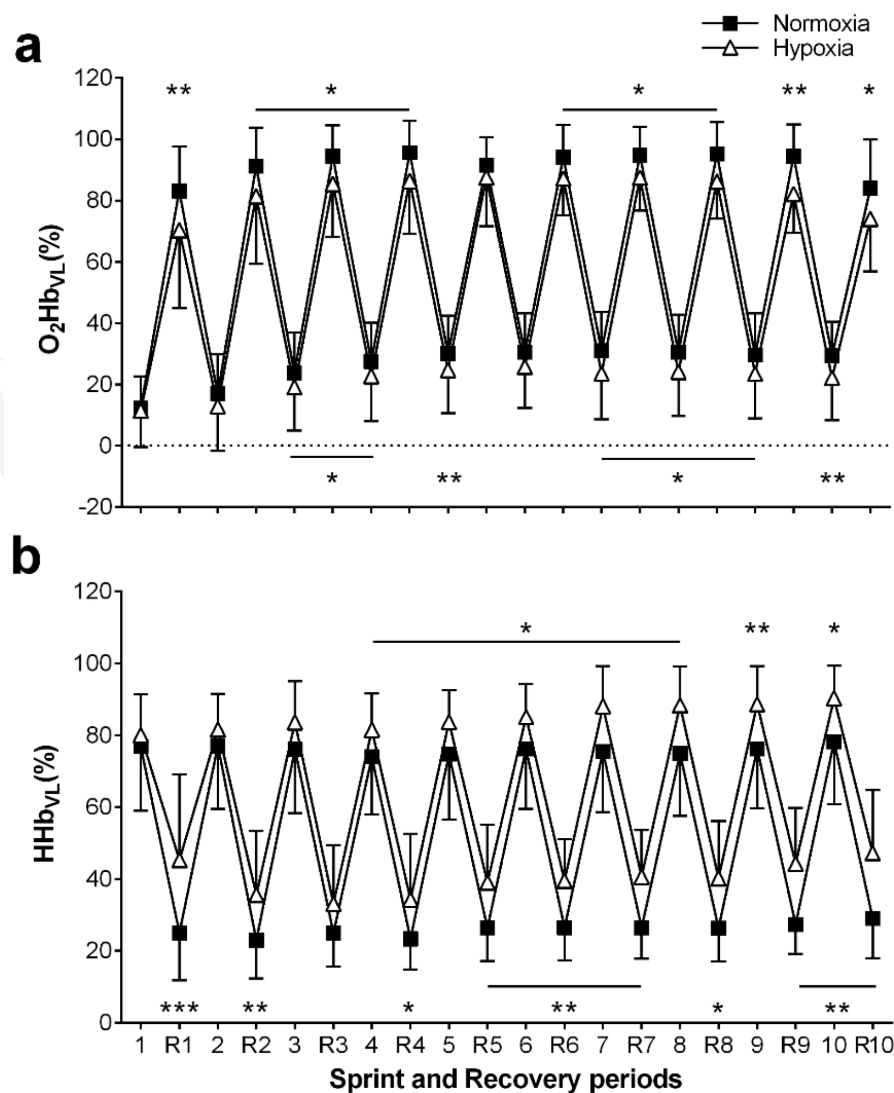


Figure 3. *Vastus lateralis* muscle oxygenation trends during repeated-sprint exercise in normoxia and hypoxia expressed as a percentage of arterial occlusion. (a) Change of vastus lateralis oxyhaemoglobin (O_2Hb_{VL}); and (b) vastus lateralis deoxyhaemoglobin (HHb_{VL}). The number of symbols (*); one, two and three denote likely, very likely and most likely respectively, that the chance of the true effect exceeds a small (-0.2 to 0.2) effect size. Results are presented as mean \pm SD. Reprinted from Rodriguez et al. [65], with permission from Elsevier.

exercise hyperpnoea was likely similar between conditions [67]. However, there was no clear difference in respiratory muscle oxygenation ($[O_2Hb]$ and $[HHb]$) during exercise in hypoxia when compared to normoxia (**Figure 4**) [65]. Based on this new evidence, it appears that O_2 delivery is preferentially distributed to the respiratory muscles to maintain the metabolic function of the respiratory muscles.

Locomotor muscle O_2 availability during rest phases between sprints is a strong determining factor of metabolic recovery [36, 37, 44], and thus performance over multiple sprints [35]. Based on our early research, it seems that locomotor muscle O_2 availability is compromised in hypoxia in favour of the respiratory muscles. Others have reported exaggerated deoxygenation of the respiratory muscles in hypoxia during voluntary isocapnic hyperpnoea [68]. However, their hypoxia gas mixture (10% O_2) resulted in a lower arterial O_2 saturation of 82% (estimated via pulse oximetry) compared to the average 87% in subjects of the study discussed here [65]. A hypoxic threshold may exist where respiratory muscle O_2 delivery can be maintained close to the rate of that during exercise in normoxia. If arterial hypoxemia was greater, further desaturation of the respiratory muscles may have been detected. Amann et al. [28] have reported a link between inspiratory muscle

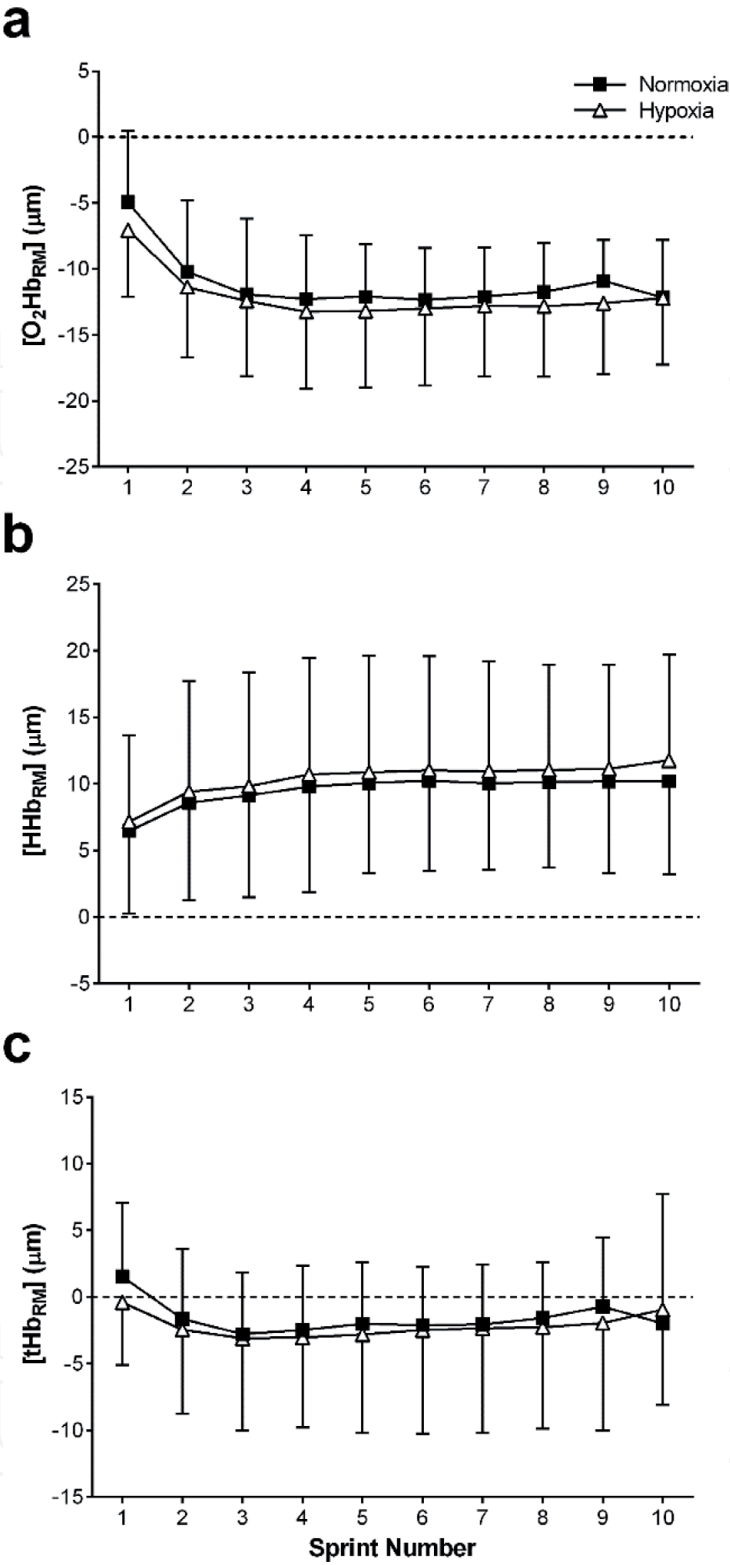


Figure 4. Respiratory muscle oxygenation trends during repeated-sprint exercise in normoxia and hypoxia expressed as an absolute change from baseline (horizontal line). (a) Concentration change from baseline of respiratory muscle oxyhaemoglobin ($[\text{O}_2\text{Hb}_{\text{RM}}]$); (b) respiratory muscle deoxyhaemoglobin ($[\text{HHb}_{\text{RM}}]$); and (c) respiratory muscle total haemoglobin ($[\text{tHb}_{\text{RM}}]$). There was no clear effect of hypoxia on respiratory muscle oxygenation compared to normoxia. Results are represented as mean \pm SD. Reprinted from Rodriguez et al. [65], with permission from Elsevier.

work in hypoxia and the development of quadriceps fatigue during high-intensity exercise. By reducing the work of breathing with PAV, the rate of fatigue developments can be attenuated [28]. Therefore, alleviating the O_2 cost of exercise hyperpnoea appears to be a pathway for enhancing limb O_2 delivery and exercise capacity in humans.

3.5 Respiratory muscle training

Aside from the structural characteristics of the pulmonary system, the relative strength of the respiratory muscles is likely to have a key role in the O₂ cost of exercise hyperpnoea. After 6-weeks of inspiratory muscle strength training, it has been demonstrated that ventilation O₂ efficiency can be enhanced [19]. Specific training targeting the inspiratory muscles (inspiratory muscle training, IMT) typically consists of inspiring against a closed valve set to open at $\approx 50\%$ of an individual's maximal inspiratory mouth pressure, repeated 30 times twice per day. Strengthening the inspiratory muscles has translated to reduced O₂ cost of voluntary hyperpnoea, attenuated exercise-induced respiratory muscle fatigue, attenuated vastus lateralis and respiratory muscle deoxygenation, and improved exercise capacity [19, 67–70]. However, respiratory muscle fatigue has not been clearly demonstrated for multiple-sprint work, and therefore ergogenic benefits of respiratory muscle training for improving repeated-sprint ability may be limited [71]. Nevertheless, evidence that IMT provides some benefit towards maintaining repeated-sprint performance exists, though the mechanisms are unclear [72, 73].

After a 6-week period of IMT, repeated-sprint ability was assessed in a group of recreational sprint sports players (soccer, rugby, field hockey and basketball) [72]. Performance was assessed during fifteen 20-m sprints, which participants were allowed a maximum of 30 s rest. Following the intervention, there were no clear changes in sprint times. However, self-selected recovery time was lessened by 6.9% (range: -0.9 to 14.5%). Strengthening the inspiratory muscles presumably reduced the O₂ cost of exercise hyperpnoea and blunted the respiratory muscle metaboreflex, which would, in turn, reduce O₂ competition between locomotor and respiratory muscles [9, 19, 74]. Through minimising O₂ competition, it is likely that the quality of metabolic recovery was enhanced with IMT, so that subjects could maintain performance with less rest between sprints [72]. But since there were no measurements of muscle oxygenation, it is difficult to separate potential changes in O₂ delivery from reduced feelings of dyspnoea that is associated with respiratory muscle training [69, 72].

The effectiveness of IMT on repeat-sprint ability and run time to exhaustion at 100% of the speed obtained during a maximal incremental exercise test has also been assessed in a group of professional female soccer players [73]. Repeated-sprint ability was assessed with six 40-m sprints (20 m + 180° turn + 20 m) with 20 s of passive rest between sprints. Vastus lateralis and intercostal muscle oxygenation was only examined during the time-to-exhaustion trials. There was no significant difference between the groups in repeated-sprint ability ($P > 0.05$). However the effect size for performance decrement was slightly larger in the IMT group post intervention (Cohen's $d = 0.84$ vs. 0.16). Similar, both placebo and experimental groups improved time to exhaustion with no significant difference between groups, but the effect size in the IMT group was larger (Cohen's $d = 0.74$ vs. 0.46). Specific training of the respiratory muscles, therefore, may only provide negligible/small performance benefits beyond professional soccer periodised training. Performance benefits were partly attributed to a blunted increase in respiratory muscle [HHb], with a concurrent increase in vastus lateralis [O₂Hb] [73]. In terms of the athlete's ability to preserve repeat-sprint performance, the IMT group also showed the greatest improvement in the capacity to maintain sprint time over multiple sprints. The blunted respiratory muscle metaboreflex in the exhaustion test may have also occurred during the repeated-sprint test. However, without muscle oxygenation measurements during the sprint trials, it is unclear if there were any changes to O₂ availability after training.

The few studies demonstrating enhanced repeated-sprint performance following IMT [72, 73] support the notion that respiratory muscle work plays a negative effect on high-intensity intermittent exercise. Training the respiratory muscles can reduce the O_2 cost of exercise hyperpnoea [19], and attenuate blood flow competition between the locomotor and respiratory muscles [70, 74]. However, there remains a very limited understanding of the role exercise hyperpnoea plays during repeated-sprint exercise. Research still needs to answer if the enhanced repeated-sprint ability following respiratory muscle training is derived from improved skeletal muscle oxygenation kinetics.

3.6 Evidence for hyperventilation

Hyperventilation is demarcated when alveolar ventilation disproportionately rises relative to CO_2 production causing a decrease in the pressure of alveolar CO_2 , and an increase in the pressure of alveolar O_2 [75]. Hyperventilation readily occurs during high-intensity exercise and can constrain a fall in arterial O_2 and pH [75, 76]. Though this was not directly examined in our research [62, 65], some evidence of hyperventilation occurring during repeated-sprint exercise was present. As depicted by the data of a representative subject (**Figure 5**), the partial pressure of end-tidal oxygen ($P_{ET}O_2$) and carbon dioxide ($P_{ET}CO_2$) rose and fell respectively from baseline over the course of the repeated-sprint protocol [62]. The wave-like pattern in $P_{ET}O_2$ and $P_{ET}CO_2$ appears to be linked to the phase of the protocol (sprint vs. rest) and occurs at exercise onset. This pattern is suggestive of a locomotor respiratory coupling, in which breathing frequency matches the cadence of locomotor exercise [77].

Further new evidence of hyperventilation comes from our recent hypoxia research [65]. Arterial hypoxemia is a potent stimulus of ventilation [26–28]. However, as we reported, there was no clear difference in either inspiratory volume, respiratory frequency or inspiratory mouth pressure during the repeated-sprint protocol. One may argue that participants were already operating at their

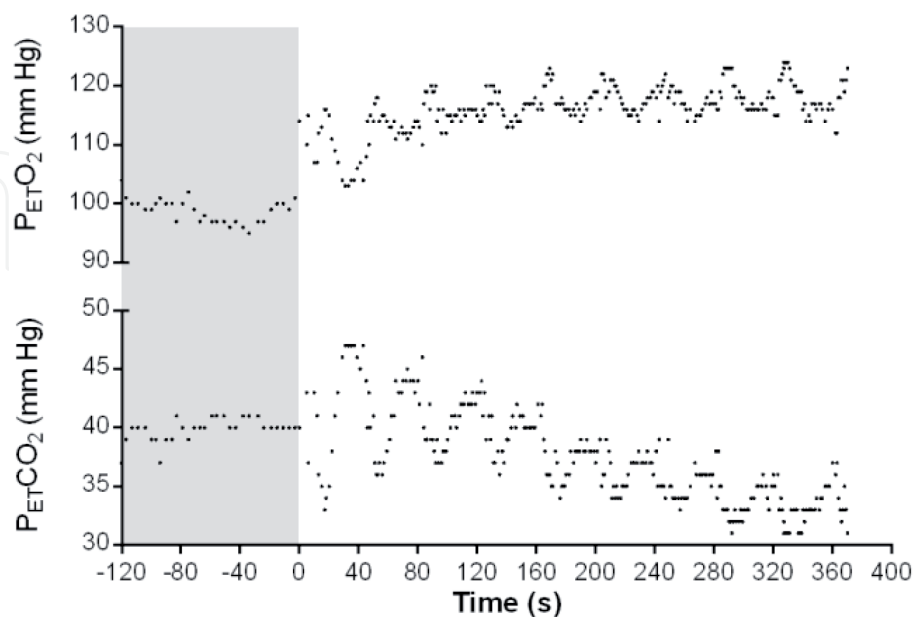


Figure 5.

Partial pressure of end-tidal oxygen ($P_{ET}O_2$) and carbon dioxide ($P_{ET}CO_2$) recorded on a breath-by-breath basis during repeated-sprint exercise. Data are from a single subject collected as part of the study by Rodriguez et al. [62] during the control exercise condition. The exercise protocol consisted of ten 10-s sprints, separated by 30 s of passive rest so that a sprint commenced every 40 s. The grey shaded area represents the 2-min baseline period observed prior to the commencement of warm-up.

upper limits of ventilation, and thus arterial hypoxemia could not have had an additive effect. Although appealing, such hypothesis requires additional work to determine the influencing factors of exercise hyperpnoea over a variety of sprint durations.

4. Conclusion

The findings of our research do not support heightened inspiratory muscle work as being a limiting factor in vastus lateralis muscle oxygenation in normoxia. The intermittent nature of repeated-sprint activity is likely a key mediating factor for which O₂ delivery can be maintained to both the locomotor and respiratory muscles. Moreover, reducing the relative intensity of exercise hyperpnoea through inspiratory muscle training shows limited benefits for enhancing repeated sprint ability. Inspiratory muscle work appears to play a more influential role under conditions of arterial hypoxemia. Our research showed that locomotor muscle oxygenation can be compromised through preferential O₂ delivery to the respiratory muscles. It is yet to be seen if inspiratory muscle training could be of benefit to exercise under these conditions.

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