Maximal Oxygen Uptake Is Achieved in Hypoxia but Not Normoxia during an Exhaustive Severe Intensity Run

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Highly aerobically trained individuals are unable to achieve maximal oxygen uptake (VO₂max) during exhaustive running lasting ~2 min, instead VO₂ plateaus below VO₂max after ~1 min. Hypoxia offers the opportunity to study the VO₂ response to an exhaustive run relative to a hypoxia induced reduction in VO₂max. The aim of this study was to explore whether there is a difference in the percentage of VO₂max achieved (during a 2 min exhaustive run) in normoxia and hypoxia. Fourteen competitive middle distance runners (normoxic VO₂max 67.0 ± 5.2 ml.kg⁻¹.min⁻¹) completed exhaustive treadmill ramp tests and constant work rate (CWR) tests in normoxia and hypoxia (FiO₂ 0.13). The VO₂ data from the CWR tests were modeled using a single exponential function. End exercise normoxic CWR VO₂ was less than normoxic VO₂max (86 ± 6% ramp, P < 0.001). During the hypoxic CWR test, hypoxic VO₂max was achieved (102 ± 8% ramp, P = 0.490). The phase II time constant was greater in hypoxia (12.7 ± 2.8 s) relative to normoxia (10.4 ± 2.6 s) (P = 0.029). The results demonstrate that highly aerobically trained individuals cannot achieve VO₂max during exhaustive severe intensity treadmill running in normoxia, but can achieve the lower VO₂max in hypoxia despite a slightly slower VO₂ response.

Keywords: VO₂, VO₂ kinetics, severe intensity, hypoxia, treadmill running

INTRODUCTION

Middle distance (800–3000 m) running performance is dependent on the speed that an athlete can sustain for the duration of the event. This speed is dependent on the ability of the locomotor muscles to produce power and resist fatigue (di Prampero et al., 1986; Lacour et al., 1990). The relatively high speed sustained throughout middle distance running events results in an energy demand in excess of the maximal aerobic energy yield (~110–120%), as assessed via pulmonary oxygen uptake (VO₂) and, thus necessitates the integrative contribution from both aerobic and anaerobic pathways (Lacour et al., 1990; Craig and Morgan, 1998; Spencer and Gastin, 2001; Duffield et al., 2005). The 800 m event, for example, requires an ~66 and 34% relative contribution from aerobic and anaerobic metabolism, respectively (Spencer and Gastin, 2001).

The overall energy demand of middle distance running events places these events within the severe, or possibly the extreme intensity domain (Jones and Burnley, 2009). It is assumed that during exercise within the severe or extreme intensity domain, VO₂ will project exponentially toward the maximal rate of pulmonary oxygen uptake (VO₂max) until VO₂max is achieved,
or exhaustion occurs (Whipp, 1994; Gaesser and Poole, 1996; Poole and Richardson, 1997; Hill and Ferguson, 1999; Jones and Burnley, 2009). However, research utilizing exhaustive constant work rate (CWR) treadmill running of ~2 min and highly aerobically trained middle distance runners (VO_{2max} ≥ 60 ml.kg^{-1}.min^{-1}) has found that VO_{2} does not achieve VO_{2max} despite sufficient time for the full response to develop (Draper and Wood, 2005a,b; Sandsals et al., 2006; James et al., 2007a,b, 2008). Instead, a submaximal steady state VO_{2} is achieved following ~1 min of exercise with no evidence of a further increase in VO_{2} (Draper and Wood, 2005b).

Previous studies using cross-sectional designs have shown that individuals with a greater VO_{2max} achieve a lower percentage of VO_{2max} (%VO_{2max}) during exhaustive CWR treadmill running of ~2 min (Draper and Wood, 2005a; James et al., 2007a). However, it should be recognized that individuals with a larger VO_{2max} typically have faster VO_{2} kinetics (Draper and Wood, 2005b; Kilding et al., 2006; Ingham et al., 2007; Marwood et al., 2010). It is therefore unclear why individuals whom possess a large VO_{2max} and faster VO_{2} kinetics achieve a lower %VO_{2max} than lesser aerobically trained individuals during exercise of this type.

It is well-known that acute hypoxic exposure results in significant reductions in VO_{2max} relative to values obtained in normoxic conditions (Dill et al., 1966; Dill and Adams, 1971; Engelen et al., 1996; Woorons et al., 2005; Calbet et al., 2015), and the decrement in VO_{2max} is linearly associated to the fraction of inspired oxygen (FiO_{2}) (Lawler et al., 1988). Acute hypoxic exposure, therefore, allows the VO_{2max} of highly aerobically trained individuals to be artificially and temporarily reduced. Whilst it is recognized that hypoxia may slow VO_{2} kinetics relative to normoxia (Engelen et al., 1996), the magnitude of slowing suggests that VO_{2} kinetics will remain sufficiently fast to permit the manifestation of its full response within <1 min, although evidence from exercise within the severe intensity domain is limited (Heubert et al., 2005). Therefore, hypoxia might provide the opportunity to explore whether highly aerobically trained individuals who are unable to achieve VO_{2max} during an exhaustive (~2 min) CWR treadmill run in normoxia can achieve a hypoxia reduced VO_{2max} during a time matched, thus relative intensity matched CWR treadmill run performed in hypoxia.

The purpose of this study, therefore, was to investigate the effect of artificially lowering VO_{2max} in trained individuals on their ability to attain VO_{2max} during an exhaustive treadmill run. We hypothesized that highly aerobically trained individuals would be unable to attain VO_{2max} during a CWR run lasting ~2 min performed in normoxia, but would be able to achieve a hypoxic reduced VO_{2max}.

**METHODS**

**Subjects**

Thirteen males and one female (mean ± SD: age 21 ± 3 y, height 1.76 ± 0.06 m, mass 66.0 ± 7.0 kg) volunteered for the study. All were trained middle distance runners with an 800 m seasonal best of <130 s. Written and informed consent was obtained prior to data collection. Subjects were instructed to report to all testing sessions in a similar state, following their usual pre-competition routine. The study was approved by the institutional ethics committee.

**General Procedures**

Subjects completed a laboratory familiarization session which was also used to determine appropriate speeds for the CWR tests. The speeds of the CWR tests were adjusted to ensure exhaustion between 105 and 135 s. All tests were performed in an environmental chamber (Sanyo Gallenkamp, PLC, Loughborough), on the same motorized treadmill (ELG 55, Woodway GmbH, Weil am Rhein, Germany). Air temperature and humidity were controlled at ~16°C and ~40%, respectively. FiO_{2} was manipulated to reflect normoxia (FiO_{2} 0.21) or hypoxia (FiO_{2} 0.13) by a hypoxic unit (Sporting Edge UK Ltd, Sherfield-on-Lodden).

Following familiarization, subjects visited the laboratory on four occasions to a complete ramp incremental tests and CWR tests, in normoxia and hypoxia. The speed of the treadmill was increased by 0.1 km.h^{-1} every 5 s (1.2 km.h^{-1}.min^{-1}) during the ramp incremental tests, the starting speeds were selected to elicit exhaustion in 8–12 min (Buchfuhrer et al., 1983) in both conditions. The speeds of the CWR tests were based on trial runs completed during the familiarization sessions.

If exhaustion was not achieved between 105 and 135 s, the treadmill speed was adjusted and subjects repeated the test on a different day. Trials were randomized to minimize any order effects.

Prior to each CWR run, subjects performed a warm-up on an identical treadmill outside of the environmental chamber. Subjects ran for 5 min at 12 km.h^{-1}, 2 min at 15 km.h^{-1}, and performed 3 × 10 s runs at the speed of the subsequent CWR test interspersed with 30 s of rest. Following the warm-up the subject entered the environmental chamber. Subjects were encouraged to perform light stretching for 2 min. Following the warm-up and stretching routine, subjects straddled the treadmill for 5 min, allowing the belt to move at the required speed for the test. Heart rate (HR) (recorded every 5 s) and breath-by-breath (VO_{2}) data were recorded during this period to determine baseline values.

All tests started with the subjects lowering themselves onto the moving treadmill belt. The treadmill was fitted with two handrails, which subjects used to tilt themselves onto or clear of the belt. The subject remained in contact with these rails at the start of the test for as long as necessary to reach the required leg speed (typically 2–3 s). The test was stopped when subjects were unable to continue and lifted themselves clear of the treadmill belt.

**Data Acquisition**

Throughout testing, subjects wore a chest strap and HR was measured using short-range telemetry (810i; Polar Electro Oy, Kempele, Finland), and breathed through a low-dead space (90 ml), low resistance (5.5 cm H_{2}O at 510 L.min^{-1}) mouthpiece and turbine assembly. Gases were collected continuously from the mouthpiece through a 2 m sampling line (0.5 mm internal diameter) to a quadrupole mass spectrometer (MSX 671: Ferraris
Respiratory Europe Ltd, Hertford, UK) where they were analyzed for O2, CO2, Ar and N2. Expired volumes were determined using a turbine volume transducer (Interface Associates, Alifioieja, US). The mass spectrometer and turbine were calibrated before each test using mixtures of known composition (Linde Gas, London, UK), and a 3 L calibration syringe (Hans Rundolf, KS), respectively. Two identical quadrupole mass spectrometers were used; one was placed outside the environmental chamber to accurately determine the internal environmental conditions, this system was calibrated against outside atmospheric air (20.94% O2, 0.04% CO2, 0.93% Argon, and 78.08% N2) and a normoxic gas bottle (14.99% O2, 5.01% CO2, 5.02% Argon, and 74.98% N2). The second system was placed inside the environmental chamber and was calibrated against the environmental conditions provided by the other mass spectrometer and a gas bottle of known composition; the normoxic gas bottle was used during normoxic testing, and a gas bottle composed of 5% O2, 5.01% CO2, 5.02% Argon, and 84.97% N2 was used in hypoxia. The volume and concentration signals were time aligned, accounting for transit delay in capillary gas and analyser rise time relative to the volume signal. VO2, VCO2, VE were calculated for each breath.

**Data Analysis**

Moving 15 s averages were used to calculate VO2, VCO2 and VE for every complete 15 s period throughout all tests. VO2 max was defined as the highest 15 s VO2 value attained during the ramp incremental tests, and VO2peak was the highest 15 s VO2 value achieved during the CWR tests. HR was recorded every 5 s and the highest value achieved during the ramp incremental test was taken as maximum HR (HR max) and the highest value recorded during the CWR exercise was the peak HR (HR peak).

The breath-by-breath VO2 data from the CWR tests were initially examined to exclude errant breaths caused by coughing, swallowing, etc., and values lying more than 4 SD from the local mean were removed. Subsequently, the breath-by-breath data were converted to second-by-second data using linear interpolation and time aligned to the start of the test. The first 15 s of data were removed to account for the cardio-dynamic phase (Murias et al., 2011). A single exponential model was used to characterize VO2 kinetics as described in the following equation:

\[ \text{VO2(t)} = \text{VO2 baseline} + A(1 - (e^{-(t/b)})) \]  

(1)

where VO2 (t) represents the absolute VO2 at a given time (t), VO2 baseline is the average of the VO2 measured over the final 120 s of quiet standing, A is the asymptotic amplitude, \( \tau \) is the time constant of the exponential response and \( \delta \) is a delay. No parameters were constrained.

**Statistical Analysis**

Data were tested for normality (Duffy and Jacobsen, 2001) and was found to be normally distributed. Two-way (test \times condition) repeated measures ANOVA was employed to determine the effect of hypoxia on VO2, minute ventilation (VE), ventilatory equivalents (i.e., VE/VO2, VE/ VCO2) and HR. Post hoc t-tests with Bonferroni correction were used to explore the origin of any significant interaction effect. Paired t-tests were used to explore differences in estimates of the modeled VO2 data in normoxia and hypoxia. Pearson's Product Moment Correlation was used to investigate the relationship between VO2 max, CWR running speed, and the % VO2 max achieved during the CWR tests. The relationship between the difference in running speed and the difference in % VO2 max achieved during the normoxic and hypoxic CWR tests was also investigated. Statistical significance was set at \( P < 0.05 \). Data are presented as mean ± SD unless otherwise stated.

**RESULTS**

The VO2 max measured in the normoxic ramp incremental test was 4.40 ± 0.42 L.min⁻¹ (67.0 ± 5.2 ml.kg⁻¹.min⁻¹) and HR max was 185 ± 7 bpm. Hypoxia reduced VO2 max to 2.97 ± 0.27 L.min⁻¹ (45.1 ± 3.0 ml.kg⁻¹.min⁻¹; \( P < 0.001 \)) and HR max to 181 ± 6 bpm; \( P < 0.05 \).

The average speed utilized for the normoxic CWR trials was 22.0 ± 1.0 km.h⁻¹ which resulted in a trial duration of 114 ± 11 s (range: 100 s to 130 s). The speed of the hypoxic CWR trial was performed at a significantly slower speed (20.5 ± 1.0 km.h⁻¹; \( P < 0.001 \)) to ensure a similar duration of trial between conditions. The duration of the hypoxic CWR trial (114 ± 11 s, range: 105 s to 135 s) was not significantly different to the duration of the normoxic CWR trial (114 ± 5 s, range: 105 s to 125 s; \( P > 0.05 \)). Normoxic VO2 max was not achieved during the normoxic CWR trials (3.79 ± 0.47 L.min⁻¹; 86 ± 6% VO2 max; \( P < 0.05 \); Figure 1). However, subjects attained hypoxic VO2 max during the hypoxic CWR trial (3.02 ± 0.30 L.min⁻¹; 102 ± 8%; \( P > 0.05 \); Figure 1). VO2 max was inversely associated with %VO2 max achieved during the hypoxic \( (r = -0.64, P < 0.05) \) and hypoxic \( (r = -0.68, P < 0.01) \) CWR trials, and when the normoxic and hypoxic trials were combined \( (r = -0.85, P < 0.001; \) Figure 2). Condition-specific HR max was attained during normoxic (189 ± 7 bpm) and hypoxic (181 ± 7 bpm) CWR trials \( (P < 0.05) \). The parameters of the modeled VO2 data are presented in Table 1. No relationships were observed between speed and % VO2 achieved during the CWR trials performed in normoxia \( (r = 0.34, P > 0.05) \), hypoxia \( (r = -0.16, P > 0.05) \), or the difference in speed and VO2 between the normoxic and hypoxic CWR trials \( (r = -0.05, P > 0.05) \).

No significant interaction effect was observed for VE \( (P > 0.05) \) with no significant main effect for condition (normoxia, 137.3 ± 17.5 L.min⁻¹; hypoxia 130 ± 16.2 L.min⁻¹; \( P > 0.05 \)), but a significant main effect for test (ramp, 128.0 ± 16.6 L.min⁻¹; CWR, 139.2 ± 16.0 L.min⁻¹; \( P < 0.001 \)). There was a significant interaction effect for VE/VO2 \( (P < 0.05) \) with significant main effects for condition (normoxia, 35.3 ± 6.7 L.min⁻¹; hypoxia 43.7 ± 4.7 L.min⁻¹; \( P < 0.001 \)) and test (ramp, 128.0 ± 16.6 L.min⁻¹; CWR, 139.2 ± 16.0 L.min⁻¹; \( P < 0.001 \)). There was a significant interaction effect for VE/VCO2 \( (P < 0.01) \) with significant main effects for condition (normoxia, 28.2 ± 3.7 L.min⁻¹; hypoxia 31.6 ± 6.0 L.min⁻¹; \( P < 0.05 \)), but no significant difference for test (ramp, 29.8 ± 5.0 L.min⁻¹; CWR, 26.2 ± 3.4 L.min⁻¹; \( P > 0.05 \)).
DISCUSSION

The principle novel finding of the current study was that despite being unable to attain $\dot{V}O_2^{\text{max}}$ during normoxic CWR running lasting $\sim 2$ min, highly aerobically trained individuals could achieve a hypoxia reduced $\dot{V}O_2^{\text{max}}$ during CWR running of a matched duration, thus of a similar relative intensity. This is the first study to demonstrate that subjects whose $\dot{V}O_2$ plateaued below $\dot{V}O_2^{\text{max}}$ during an exhaustive CWR run, were subsequently able to attain $\dot{V}O_2^{\text{max}}$ when the exercise bout was replicated in hypoxic conditions despite a slowed $\dot{V}O_2$ response.

Previous research has demonstrated that during normoxic CWR running lasting $\sim 2$ min, more highly aerobically trained individuals achieved a lower % $\dot{V}O_2^{\text{max}}$ (Draper and Wood, 2005a; James et al., 2007a). In agreement with these findings, the current study reported an inverse association between $\dot{V}O_2^{\text{max}}$ and the % $\dot{V}O_2^{\text{max}}$ achieved during the normoxic CWR trial (Figure 2). To gain further insight into the relationship between $\dot{V}O_2^{\text{max}}$ and % $\dot{V}O_2^{\text{max}}$ achieved during the present study investigated whether a hypoxia induced reduction in $\dot{V}O_2^{\text{max}}$ may permit highly aerobically trained individuals to attain $\dot{V}O_2^{\text{max}}$ during exhaustive CWR running at a matched relative intensity. The acute hypoxic exposure reduced $\dot{V}O_2^{\text{max}}$ by $\sim 32\%$, consistent with previous reports (Engelen et al., 1996; Martin and O’Kroy, 1993; Woorons et al., 2005), and subject to this reduction $\dot{V}O_2^{\text{max}}$ was achieved (Figure 1B). No relationship was observed between % $\dot{V}O_2^{\text{max}}$ achieved and the running speed during the CWR tests in normoxia or hypoxia, nor the difference in speed between conditions (i.e., normoxia and hypoxia) and the difference in % $\dot{V}O_2^{\text{max}}$ achieved (all $P > 0.05$), suggesting that $\dot{V}O_2^{\text{max}}$ may be an important parameter in determining whether an individual may be able to achieve their $\dot{V}O_2^{\text{max}}$ during this type of exercise. Furthermore, these findings highlight that further improvements in $\dot{V}O_2^{\text{max}}$ are of less benefit to high-intensity exercise performance compared to similar gains in anaerobic capability. These findings perhaps seem incongruous with the high $\dot{V}O_2^{\text{max}}$ values typically reported in elite 800 m runners (Svedenhag and Sjödin, 1984; Ingham et al., 2008) that they are apparently unable to fully utilize. However, such a high $\dot{V}O_2^{\text{max}}$ value may be due to the high volume of interval training performed by these athletes (Helgerud et al.,...
2007). There have certainly been instances where performance at altitude would indicate that the decrement in VO$_{2\text{max}}$ may not substantially impair performance. For example, Ralph Doubell equalled the World Record at the 1968 Mexico Olympics which was performed at an altitude of 2,240 m above sea level; a feat that would seem implausible if one's VO$_{2\text{max}}$ was a necessity for optimum performance.

The VO$_{2}$ kinetics were similar to values previously reported during investigations utilizing similarly highly aerobically trained runners during CWR running lasting ~2 min (Draper and Wood, 2005a,b; Draper et al., 2008). The phase I time delays are also similar to those reported by Wilkerson et al. (2004). Consistent with Engelen et al. (1996), we found a slower phase II $\tau$ in the hypoxic condition (Table 1). However, it should be noted that despite a slower phase II $\tau$, resulting in ~10 s difference in the attainment of the VO$_{2}$ amplitude, hypoxic VO$_{2\text{max}}$ was achieved. Conversely, despite faster VO$_{2}$ kinetics normoxic VO$_{2\text{max}}$ was not attained. Instead, there was an evident VO$_{2}$ plateau in normoxia at ~86% normoxic VO$_{2\text{max}}$. The occurrence of a VO$_{2}$ plateau, rather than a continued trajectory toward VO$_{2\text{max}}$ and indeed the energy demands of the exercise, questions contemporary models of VO$_{2}$ kinetics during CWR exercise lasting ~2 min in this highly aerobically trained group. Interestingly, this same response is not evident during exhaustive cycle ergometry of a similar duration whereby VO$_{2}$ continues to increase throughout, although maximum values are not attained (Draper et al., 2003). At present the reasons for the differences between exercise modes in VO$_{2}$ response to severe intensity exercise are unclear. Increasing oxygen uptake has been associated with reduced efficiency arising from factors, such as metabolite accumulation, limitations in substrate availability, pH disturbance, increased muscle temperature, and altered motor unit recruitment (Grassi et al., 2015). Indeed, it is well established that the patterns of muscle action, including the relative proportion of eccentric and concentric contraction and the contribution of the stretch-shortening cycle differ between running and cycling (van Ingen-Schenau et al., 1997; Bijker et al., 2002). These effects might, at least in part, contribute to the between mode differences in VO$_{2}$ kinetics, particularly for higher work rates where the use of elastic energy is optimized (Dalleau et al., 1998); whether or not increased stored energy during the stretch shortening cycle can help maintain efficiency despite increased metabolic fatigue warrants further investigation.

Consistent with previous investigations, we found that HR$_{\text{max}}$ was greater in normoxia than hypoxia (Benoit et al., 1995; Mollard et al., 2007). However, similar to our VO$_{2}$ findings normoxic HR$_{\text{max}}$ was not achieved during the normoxic CWR test (Draper and Wood, 2005a,b), but hypoxic HR$_{\text{max}}$ could be achieved during the hypoxic CWR trial. Assuming HR$_{\text{max}}$ is needed to achieve maximal cardiac output (Q$_{\text{max}}$), these findings suggest that Q$_{\text{max}}$ was not achieved during the normoxic CWR test. Despite a lower HR$_{\text{max}}$ in hypoxia relative to normoxia, previous findings have shown that hypoxia has no effect on Q$_{\text{max}}$ (Mollard et al., 2007), implying a compensatory increase in maximal stroke volume in hypoxia. Therefore, the inability to achieve VO$_{2\text{max}}$ in the normoxic CWR trial may be associated with submaximal cardiac output. However, further investigation that assesses cardiac output and blood flow is necessary to gain insight into Q$_{\text{max}}$ as a potential limiting factor in the attainment of VO$_{2\text{max}}$ during this type of exercise.

Although end exercise VO$_{2}$ was greater during the CWR trials relative to the ramp incremental tests, this was not different between normoxia and hypoxia. Furthermore, we observed no differences in ventilatory equivalents between conditions (i.e., normoxia and hypoxia). These similar ventilatory responses might serve to attenuate or prevent the exercise induced arterial hypoxemia that has been described in highly aerobically trained individuals (Dempsey et al., 1984; Powers et al., 1988, 1992; Caillaud et al., 1993; review Prefaut et al., 2000). In normoxia, the increased VE during CWR exercise would likely increase the work of breathing thereby compromising limb muscle blood flow (Wetter et al., 1999). In hypoxia, the PO$_{2}$ is in the steep portion of the oxygen-hemoglobin dissociation curve and increased VE could have pronounced effects on arterial oxygen concentration and may help to preserve muscle VO$_{2}$ despite reduced limb blood flow. However, in normoxia the PO$_{2}$ is in the flatter region of the oxygen-hemoglobin dissociation curve and the same increases in VE would be less effective in altering arterial oxygen concentration relative to hypoxia. As a consequence the increased work associated with breathing would result in little/small increases in arterial oxygen concentration and reduce muscle blood flow and thus muscle VO$_{2}$. However, it should be noted that exercise induced arterial hypoxemia has also been reported during different exercise modalities, such as cycling (Powers et al., 1988), whereas the phenomenon whereby VO$_{2}$ attains a plateau below VO$_{2\text{max}}$ has only been reported in highly aerobically trained individuals during CWR running exercise lasting ~2 min. The mechanistic origin(s) for this phenomenon is currently unknown and requires further research.

Despite only one transition to the CWR trial in each condition, due to the large amplitude of the VO$_{2}$ response during this intensity of exercise there is a much greater signal/noise ratio when compared to exercise of a lower intensity (Lamarra et al., 1987). In lesser trained individuals with smaller VO$_{2}$ amplitude, thus smaller signal to noise ratio, Draper et al. (2008) demonstrated that two transitions would at worst (i.e., smallest signal to noise ratio) provide 95% confidence intervals of 1 s. Given that the current study recruited more highly aerobically trained individuals than Draper et al. (2008), thus a greater signal to noise ratio, it would be reasonable to

<table>
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<th>TABLE 1</th>
<th>The parameters of the modeled VO$_{2}$ response to CWR exercise in normoxia and hypoxia.</th>
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<tr>
<td></td>
<td>Normoxia</td>
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<tr>
<td>Ramp VO$_{2\text{max}}$ (L.min$^{-1}$)</td>
<td>4.40 ± 0.42</td>
</tr>
<tr>
<td>CWR VO$_{2\text{peak}}$ (L.min$^{-1}$)</td>
<td>3.79 ± 0.47</td>
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<tr>
<td>Baseline O$_{2}$ (L.min$^{-1}$)</td>
<td>0.60 ± 0.11</td>
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<tr>
<td>A (L.min$^{-1}$)</td>
<td>2.45 ± 0.50</td>
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<tr>
<td>Baseline + A (L.min$^{-1}$)</td>
<td>3.05 ± 0.51</td>
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<tr>
<td>$\tau$ (s)</td>
<td>10.4 ± 2.6</td>
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<td>$\delta$ (s)</td>
<td>7.6 ± 2.6</td>
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expect 95% confidence intervals of better than 2 s for \( \tau \). Furthermore, the current study design was sufficiently sensitive and had adequate power to detect differences in \( \tau \) between conditions.

In conclusion, the results of the present study demonstrate that highly aerobicly trained individuals whom are unable to achieve \( \text{VO}_2\text{max} \) during an exhaustive CWR run lasting \(~2\) min, are able to achieve a hypoxia reduced \( \text{VO}_2\text{max} \) despite exhibiting slower \( \text{VO}_2 \) kinetics. These data further support the notion that \( \text{VO}_2\text{max} \) is an important determinant of the %\( \text{VO}_2\text{max} \) that can be achieved during a short duration exhaustive CWR run. The present data demonstrate that ventilatory differences are unable to explain the inability to attain \( \text{VO}_2\text{max} \) during normoxic CWR trials. Future research should explore the possibility of an \( \text{O}_2 \) delivery or blood perfusion limitation during this type of exercise in highly aerobically trained runners. Future research should also consider utilizing an experimental condition in normoxia which uses gradient on the treadmill (or weighted vest) instead of hypoxia to slow the running speed down and induce task failure in \(~2\) min. This would aid in deciphering the novel finding of this study.

**ETHICS STATEMENT**

The study was approved by University of Gloucestershire Ethics Committee. All participants were provided with verbal and written information that detailed the rationale of the study, the test procedures, and any risks and benefits of participation. Participants were informed of their right to withdraw from the study at any time without penalty. All participants provided written informed consent detailing that they were willing to take part.

**AUTHOR CONTRIBUTIONS**

MB, CP, SD, JC, and CC were involved in conceptual design, data collection, interpretation, and manuscript preparation. All authors approve the submission of this work and agree to be accountable for all aspects of the work.

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

The reviewer NT and handling Editor declared their shared affiliation, and the handling Editor states that the process nevertheless met the standards of a fair and objective review.