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Published in:
Frontiers in Physiology
Publication date:
2017
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10.3389/fphys.2017.00096

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Citation for published version (APA):

Download date: 01. Jan. 2024
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 (VO₂max ≥ 60 ml.kg⁻¹.min⁻¹) has found that VO₂ does not achieve VO₂max 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 VO₂ is achieved following ~1 min of exercise with no evidence of a further increase in VO₂ (Draper and Wood, 2005b).

Previous studies using cross-sectional designs have shown that individuals with a greater VO₂max achieve a lower percentage of VO₂max (%VO₂max) 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 VO₂max typically have faster VO₂ 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 VO₂max and faster VO₂ kinetics achieve a lower %VO₂max than lesser aerobically trained individuals during exercise of this type.

It is well-known that acute hypoxic exposure results in significant reductions in VO₂max 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 VO₂max is linearly associated to the fraction of inspired oxygen (FiO₂) (Lawler et al., 1988). Acute hypoxic exposure, therefore, allows the VO₂max of highly aerobically trained individuals to be artificially and temporarily reduced. Whilst it is recognized that hypoxia may slow VO₂ kinetics relative to normoxia (Engelen et al., 1996), the magnitude of slowing suggests that VO₂ 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 VO₂max during an exhaustive (~2 min) CWR treadmill run in normoxia can achieve a hypoxia reduced VO₂max 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 VO₂max in trained individuals on their ability to attain VO₂max during an exhaustive treadmill run. We hypothesized that highly aerobically trained individuals would be unable to attain VO₂max during a CWR run lasting ~2 min performed in normoxia, but would be able to achieve a hypoxic reduced VO₂max.

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. FiO₂ was manipulated to reflect normoxia (FiO₂ 0.21) or hypoxia (FiO₂ 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 (VO₂) 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 lift 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 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 H₂O 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, Alifovieja, 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, 0.02% 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, V̇E were calculated for each breath.

### Data Analysis

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

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:

\[
\text{VO}_2(t) = \text{VO}_2 \text{baseline} + A(1 - (e^{-\nu(t-\delta)}))
\]

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, \( \nu \) 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 × condition) repeated measures ANOVA was employed to determine the effect of hypoxia on VO₂, minute ventilation (V̇E), ventilatory equivalents (i.e., V̇E/VO₂, V̇E/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 VO₂max, CWR running speed, and the % VO₂max achieved during the CWR tests. The relationship between the difference in running speed and the difference in % VO₂max 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 VO₂max 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 HRmax was 185 ± 7 bpm. Hypoxia reduced VO₂max to 2.97 ± 0.27 L.min⁻¹ (45.1 ± 3.0 ml.kg⁻¹.min⁻¹; \( P < 0.001 \)) and HRmax 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 135 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 VO₂max was not achieved during the normoxic CWR trial (3.79 ± 0.47 L.min⁻¹; 86 ± 6% VO₂max; \( P < 0.05 \); Figure 1). However, subjects attained hypoxic VO₂max during the hypoxic CWR trial (3.02 ± 0.30 L.min⁻¹; 102 ± 8%; \( P > 0.05 \); Figure 1). VO₂max was inversely associated with %VO₂max achieved during the normoxic (\( r = -0.64; P < 0.05 \)) 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 HRmax 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 V̇E (\( P > 0.05 \)) 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 V̇E/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 V̇E/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 current study, we 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.,...
TABLE 1 | The parameters of the modeled VO2 response to CWR exercise in normoxia and hypoxia.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normoxia</th>
<th>Hypoxia</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ramp VO2 (L.min(^{-1}))</td>
<td>4.40 ± 0.42</td>
<td>2.97 ± 0.27</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CWR VO2peak (L.min(^{-1}))</td>
<td>3.79 ± 0.47</td>
<td>3.02 ± 0.30</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Baseline O2 (L.min(^{-1}))</td>
<td>0.60 ± 0.11</td>
<td>0.67 ± 0.15</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>A (L.min(^{-1}))</td>
<td>2.45 ± 0.50</td>
<td>1.61 ± 0.27</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Baseline + A (L.min(^{-1}))</td>
<td>3.05 ± 0.51</td>
<td>2.28 ± 0.21</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>τ (s)</td>
<td>10.4 ± 2.6</td>
<td>12.7 ± 2.8</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>δ (s)</td>
<td>7.6 ± 2.6</td>
<td>7.4 ± 3.3</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

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 VO2 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 VO2\(_{\text{max}}\) 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 VO2\(_{\text{max}}\) during this type of exercise.

Although end exercise \(V_{E}\) 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 \(V_{E}\) 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 \(V_{E}\) 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 \(V_{E}\) 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 VO2\(_{\text{max}}\) 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 VO2 response during this intensity of exercise there is a much greater signal/noise ratio when compared to exercise of a lower intensity (Lamarr et al., 1987). In lesser trained individuals with smaller VO2 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
expect 95% confidence intervals of better than 2s for τ. Furthermore, the current study design was sufficiently sensitive and had adequate power to detect differences in τ between conditions.

In conclusion, the results of the present study demonstrate that highly aerobicly trained individuals whom are unable to achieve VO\(_{2max}\) during an exhaustive CWR run lasting ~2 min, are able to achieve a hypoxia reduced VO\(_{2max}\) despite exhibiting slower VO\(_2\) kinetics. These data further support the notion that VO\(_{2max}\) is an important determinant of the %VO\(_{2max}\) 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 VO\(_{2max}\) during normoxic CWR trials. Future research should explore the possibility of an 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.

**ETICS 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.

**REFERENCES**


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.

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