Fatigue During High-Intensity Endurance Exercise: The Interaction Between Metabolic Factors and Thermal Stress

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Abstract

Mitchell JB, Rogers MM, Basset JT, and Hubing KA. Fatigue during high-intensity endurance exercise: The interaction between metabolic factors and thermal stress. J Strength Cond Res 28(7): 1906–1914, 2014—The purpose of this study was to examine the effects of hot (37°C) and cool (10°C) environments on cycling time to exhaustion (TTE), pH, lactate, and core temperature (Tc). Eleven endurance-trained subjects completed 4 TTE trials: Hot 80% VO2max (H80), Cool 80% (C80), Hot 100% (H100), and Cool 100% VO2max (C100). Esophageal temperature and blood was sampled before, every 5 minutes, at exhaustion, and 3 minutes after exercise and analyzed for lactate, pH, and HCO3-. Multifactorial analysis of variance with repeated measures was used to determine differences between mean values (±SD). Time to exhaustion was shorter in H100 and C100 vs. H80 and C80 (5.64 ± 1.49 minutes, 5.83 ± 1.03 minutes, 12.82 ± 2.0 minutes, and 24.85 ± 6.0 minutes, respectively) and shorter in H80 vs. C80 (p < 0.01). The pH at exhaustion was different among all conditions (7.17 ± 0.06, 7.15 ± 0.07, 7.21 ± 0.04, and 7.24 ± 0.06 units for H100, C100, H80, and C80, respectively, p = 0.02). The Tc at exhaustion was lower in H100 and C100 (37.93 ± 0.67 and 37.62 ± 0.58°C) vs. H80 and C80 (38.54 ± 0.51°C and 38.53 ± 0.38°C) (p < 0.01). In H80 and C80, the higher Tc likely played a greater role in the termination of exercise, whereas, in H100 and C100, pH and metabolic changes may have been more important. Despite these differences, neither an upper limit for Tc nor a lower limit for pH was identified; thus, fatigue based entirely on peripheral factors was not supported, and a combination of peripheral and central processes must be considered. The practical implications of these findings are that aerobic exercise at or near VO2max may be impacted more by metabolic factors, whereas lower intensities (~80% VO2max) may be affected more by heat stress; these differences should be considered when training for events of this type.

Keywords: acidosis, heat, exhaustion, performance

Introduction

Fatigue during exercise, defined as the inability to maintain the desired force output, has been attributed to a variety of factors (4,24). These include energy substrate depletion, pH changes, metabolite accumulation, fluid and ionic shifts, temperature effects, neuromuscular dysfunction, and the influence of the central nervous system (CNS) (4,5,9,23,24,34). The latter can be conceptualized as fatigue of the CNS that results in reduced central drive, or it has been proposed that the CNS regulates exercise output to avoid extreme alteration in the tissues that may lead to organ damage (22,24,30). Despite the difficulty in identifying the precise cause of fatigue, it is known that the intensity and duration of the exercise and the environment in which the exercise is carried out will interact to produce different physiological states and different performance outputs (9,17). Much of what is known about intensity or environmental effects has been derived from the examination of prolonged lower intensity exercise (e.g., 60–80% VO2max), where fatigue is usually associated with substrate depletion, fluid loss, cardiovascular dysfunction, and heat storage (6,12,23). Higher intensity aerobic exercise (e.g., >80% of VO2max) can, however, produce both rapid heat storage (19) and significant metabolic changes (8,12,17); thus, the cause of fatigue during this type of exercise becomes more complicated, especially when the exercise is conducted in a thermally stressful environment. Compared with lower intensity aerobic exercise, the cause of fatigue during higher intensity aerobic exercise in the heat has not been studied extensively.

High-intensity aerobic exercise is associated with peripheral tissue alterations such as elevations in hydrogen ions, lactate, extracellular potassium, and inorganic phosphate (1). Decreased pH has been associated with fatigue through a variety of proposed peripheral mechanisms; however, there is evidence to suggest that pH may not be the only or even the primary causal factor in fatigue during high-intensity exercise.
exercise (1,20). Although, it is well recognized that the lactate accumulation that occurs with high-intensity exercise does not play a direct role in fatigue (3,10,26), blood lactate levels, especially when expressed as a percentage of \( V_{\text{O2 max}} \), can be used to predict performance (21). If there are no definitive mechanistic links between peripheral physiological changes and reduced neuromuscular function, it may be that the typical metabolic markers of fatigue are factors monitored centrally to regulate the peripheral disturbances brought on by intense exercise (14,22).

Exercise in a hot environment (\( \geq 35^\circ \text{C} \)) reduces maximal aerobic capacity (12,28) and is associated with an accelerated onset of fatigue accompanied by greater heat storage and systemic and metabolic changes compared with exercise in a neutral environment (\( \sim 22^\circ \text{C} \)) (17,25). At the submaximal exercise intensities typically studied during the examination of fatiguing exercise responses in hot environments (60–80% of \( V_{\text{O2 max}} \)), it has been suggested that fatigue may be associated with a critical level of core temperature (\( T_c \)), possibly because of the reduced cortical drive and muscle recruitment (5,12,23). During exercise at higher intensities, such as those above 80% of \( V_{\text{O2 max}} \), other fatigue-inducing factors may be active; thus, a critical \( T_c \) may not be present. An integrated model of fatigue suggests that alterations in multiple peripheral factors occurring during high-intensity exercise in the heat may work with central responses to produce fatigue (14,22). Furthermore, it has been suggested that the rate of heat gain may be a more important factor than an absolute temperature in mediating the interaction between peripheral and central factors associated with fatigue (30,31). The concept that exercise is terminated to avoid extreme disturbances may be applied to physiological responses other than heat, such as changes in pH or lactate (2,7,22). A complete understanding of the cause of fatigue is important to practitioners so that they have the information needed to improve training methods and other preparations for a variety of competitive events taking place in a range of environments.

The purpose of this study was to examine the interaction between thermal and metabolic changes associated with fatigue during exercise in the heat and to relate these changes to possible causes of fatigue. Specifically, we compared time to exhaustion (TTE) cycle ergometry in hot (37\(^\circ\)C) and cool (10\(^\circ\)C) environments during 2 exercise intensities (80 and 100% of \( V_{\text{O2 max}} \)). Because of the documented negative effects of a hot environment on maximal aerobic capacity and exercise performance assessed at submaximal intensity, we hypothesized that TTE would be significantly reduced at both the exercise intensities performed in a hot compared with a cool environment. The basis for this hypothesis was that thermal stress would be the predominant causal factor in fatigue, overriding metabolic factors associated with high-intensity exercise.

**Methods**

**Experimental Approach to the Problem**

This experiment was conducted using a repeated measures, condition (80 and 100% \( V_{\text{O2 max}} \)) by environment and (hot and cool) by time (sampling time points), randomized (order of treatment) counterbalanced design. Subjects completed 4 experimental trials requiring that each subject complete a TTE ride at: (a) 80% \( V_{\text{O2 max}} \) in a cool environment (C80); (b) 80% \( V_{\text{O2 max}} \) in a hot environment (H80); (c) 100% \( V_{\text{O2 max}} \) in a cool environment (C100); and (d) 100% \( V_{\text{O2 max}} \) in a hot environment (H100). The temperatures for the cool and hot environments were 10.17 ± 2.55\(^\circ\)C and 36.61 ± 0.88\(^\circ\)C with relative humidities of 25.85 ± 5.86% and 57.05 ± 7.34%, respectively. These environmental conditions were selected to create distinctly different levels of thermoregulatory strain, and the exercise intensities were selected to emphasize high-intensity exercise with a substantial anaerobic energy contribution, thus creating large changes in such metabolic factors as pH and other metabolites. The 4 conditions used in this design allowed us to compare fatigue responses occurring in the presence of both large temperature increases and large metabolic disturbances.

**Subjects**

Eleven endurance-trained male athletes aged 21–40 years were recruited from local triathlon and cycling clubs using the following inclusion criteria: (a) endurance-trained cyclists or triathletes (\( > 100 \) miles per week cycling) currently competing in local/national road races or triathlons for at least the previous 12 months; and (b) a maximal aerobic capacity (\( V_{\text{O2 max}} \)) >3.75 L⋅min\(^{-1}\) (Table 1). These criteria were used to avoid training effects over the course of the study and to ensure that they were capable of completing the protocol at a high level and without undue risk. The study was conducted during the months of January through March. The study was approved by the institutional review board for research with human subjects. Informed consent was obtained from each subject, and they also completed a medical history questionnaire.

**Table 1. Subject characteristics (mean ± SD).**

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Body fat (%)</th>
<th>( V_{\text{O2 max}} ) (L⋅min(^{-1}))</th>
<th>Peak power (W)</th>
</tr>
</thead>
<tbody>
<tr>
<td>32.6 ± 4.9</td>
<td>185.4 ± 7.7</td>
<td>82.5 ± 8.8</td>
<td>13.8 ± 3.6</td>
<td>4.23 ± 0.49</td>
<td>333 ± 29.6</td>
</tr>
</tbody>
</table>

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Preliminary Testing

On the preliminary test day, subjects completed a medical history questionnaire and an informed consent form, and a 7-site (13) skinfold assessment was completed by a trained technician. The same day, they completed an incremental exercise cycle ergometer test to assess maximal oxygen consumption (\(V_{\text{O2,max}}\)); the results of this test were used to determine the target workloads for the experimental trials.

\(V_{\text{O2,max}}\) Test. All exercise tests were performed on a Monark cycle ergometer (Ergometer 894E; Monark Exercise AB, Vansbro, Sweden) that was equipped with a digital display for monitoring cycle cadence. Subjects wore a heart rate (HR) monitor (Polar Electro E600; Polar Electro Inc., Lake Success, NY, USA) and were fitted with a mouthpiece to collect respiratory gas exchange. The incremental test began at a power output of 80 W and increased by 40 W every 3 minutes for the first 12 minutes, after which the output was increased by 40 W every 2 minutes until termination. Subjects were allowed to self-select a cadence between 85 and 95 revolutions per minute (90.7 ± 2.4), which was then used for all subsequent tests. Termination was based on the inability to maintain a constant cycle cadence, and test validity was based on achieving 2 of the following 3 criteria: a respiratory exchange ratio of 1.1 or greater, reaching an age-predicted maximal HR, and a leveling off of \(V_{\text{O2}}\) with an increased load. Oxygen consumption was measured continuously through a gas analysis system (TrueOne 2400 Metabolic; Parvo Medics, Inc., Sandy, UT, USA) with a reporting interval of 30 seconds and calibrated according to the manufacturer’s specifications.

Learning Trial. A minimum of 2 days after the preliminary test day, subjects completed a TTE learning trial. This trial was conducted at 80% of \(V_{\text{O2,max}}\) in the environmental chamber under hot conditions (36°C) and did not involve blood sampling or measurement of esophageal temperature. Heart rate, oxygen consumption, and TTE were recorded. The primary purpose of this trial was to acclimate the subjects to the experimental test conditions and the TTE protocol.

Experimental Testing

At the same time of the day for each trial and after a day of rest, subjects reported to the laboratory after a minimum of a 4-hour fast, at least 3 days after the learning day. Hydration was standardized through instructions to consume a liter of water the evening before testing for those tested in the morning and 500 ml during 2–3 hours before each trial for those tested later in the day. We did not, however, quantify hydration status with body mass, plasma volume, or other hydration measures. After resting in a supine position for 15 minutes, a catheter was inserted in an antecubital vein, and a baseline blood sample was collected. To determine \(T_e\), an esophageal thermometer (4400 9FR probe; Cincinnati Sub-Zero, Cincinnati, OH, USA) was inserted through the nasal passage to a depth corresponding to 25% of the subject’s height and was monitored by a digital telethermometer (Model 8502-12; Cole-Parmer Instrument Company, Vernon Hills, IL, USA). After baseline temperatures had been recorded, the subjects completed a 5-minute warm-up at room temperature on a Monark cycle ergometer at an intensity corresponding to 60% of their \(V_{\text{O2,max}}\). After the warm-up, subjects entered the environmental chamber and were positioned on the Monark ergometer (previously described). Subjects wore the HR monitor and were fitted with a mouthpiece for respiratory gas analysis. There was a 4-minute time delay from the end of the warm-up to the beginning of each trial. Subjects were allowed to get the cadence to their predetermined level without any resistance in a 10-second period before beginning the TTE ride and were required to maintain the given workload until exhaustion, which was defined as a drop in the required cycling cadence by 10 revolutions per minute for >20 seconds. The predetermined cadence from the initial \(V_{\text{O2,max}}\) test for each subject was used in all 4 time trials. The power outputs for the 80 and 100% intensities were 267.6 ± 25.3 W and 334.6 ± 31.7 W, respectively. During each trial, respiratory gas data and HR were monitored continuously using the equipment described previously. No fluid consumption was allowed during the trial. Esophageal temperature was monitored continuously and recorded at 1-minute intervals. The subjects remained in the chamber, seated on the ergometer for 3 minutes until the final blood sample was taken.

Blood Collection and Analysis

Through an indwelling venous catheter, blood samples were obtained before exercise, every 5 minutes during exercise, at exhaustion, and at 3 minutes of recovery for analysis of pH, bicarbonate, and lactate. The 3-minute recovery sample was obtained to allow for efflux of these substances into the blood. After each blood sample, the catheter was rinsed with sterile physiological saline to prevent clotting. Lactate was assayed in triplicate (coefficient of variation [CV] < 3%) using an enzymatic spectrophotometric assay (16). Blood pH, and bicarbonate, levels were determined using a blood gas/electrolyte analyzer (ABL 77 Series; Radiometer, Copenhagen, Denmark). This device has been evaluated by Lindemans et al. (15) and has been shown to have within-day and between-day CV of 0.7 and 0.9%, respectively. These blood measures were selected to determine the level of a variety of metabolic responses that are known to be elevated when there is a substantial contribution of anaerobic metabolism.

Statistical Analyses

Data from the experimental trials were analyzed using either a 2-factor or a 3-factor repeated measures analysis of variance (ANOVA). The first factor was “intensity” and had 2 levels: 80 and 100% of \(V_{\text{O2,max}}\); the second factor was “environment” and also had 2 levels: hot and cool; and for those variables that were sampled multiple times within a condition, the third factor was “time” with a variable number of levels depending on the sampling frequency. A 2-factor ANOVA was used for performance (TTE) based on
only intensity and environment as independent variables. A Huynh–Feldt correction was applied to the repeated measures analyses to correct for multiple sampling in the time factor. A Newman–Keuls post hoc analysis was used to isolate the location of significant main effects and interactions detected by the ANOVA. Using a correlation matrix, Pearson’s correlations were conducted between all dependent variables with an emphasis on examining the relationship between the changes in $T_c$, pH, and lactate on TTE. Power calculations for the dependent measures analyzed produced values of 0.80 or higher. All data are reported as the mean ± SD, and significance was accepted at an alpha level of $p \leq 0.05$.

**Results**

**Performance Data**

The analysis of the TTE results produced a 2-way interaction such that the H80 and C80 responses were significantly different ($p < 0.001$) from each other, with C80 being approximately twice as long as H80 (Figure 1). In addition, both the lower intensity responses were greater than those observed in the H100 and C100 conditions; however, the 2 high-intensity conditions did not differ from each other.

**Cardiorespiratory Data**

A significant ($p < 0.001$) intensity by time interaction was found for oxygen consumption with greater levels in H100 and C100 compared with H80 and C80 at all time points (Table 2). A significant ($p < 0.001$) intensity by time interaction was found for ventilation with greater levels in H100 and C100 compared with H80 and C80 at all time points (Table 2). In addition, a significant ($p = 0.001$) environment by time interaction was also present for ventilation with greater levels at 3 minutes and exhaustion in H80 and H100 compared with C80 and C100. Finally, a significant ($p < 0.001$) intensity by time interaction was present for HR with greater values during H100 and C100 compared with H80 and C80 at 2 and 3 minutes but not at exhaustion (Table 2).

**Temperature and Metabolic Data**

At exhaustion, a significant ($p = 0.01$) environment by time interaction showed that the $T_c$ responses in H80 and C80 were significantly greater than those in H100 and C100 (Figure 2). Although greater by $-0.30^\circ$ C, the $T_c$ in H100 was not significantly different ($p = 0.29$) from that observed in C100. As shown in Figure 2, at 3 minutes of recovery, similar trends were observed. The rates of heat gain were $0.13 \pm 0.03^\circ$ C·min$^{-1}$, $0.06 \pm 0.03^\circ$ C·min$^{-1}$, $0.22 \pm 0.05^\circ$ C·min$^{-1}$, and $0.13 \pm 0.03^\circ$ C·min$^{-1}$ for H80, C80, H100, and C100, respectively. For these data, a main effect for environment (hot greater than cool, $p < 0.001$) and a main effect for intensity (100 greater than 80, $p < 0.001$) was found; however, there was not a significant interaction ($p = 0.52$).

A significant ($p = 0.015$) intensity by environment by

<p>| Table 2. Cardiorespiratory responses (mean ± SD).* |</p>
<table>
<thead>
<tr>
<th>Condition</th>
<th>2 min</th>
<th>3 min</th>
<th>Exhaustion</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_o_2$ (L·min$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H80</td>
<td>3.07 ± 0.40</td>
<td>3.26 ± 0.40</td>
<td>3.93 ± 0.38</td>
</tr>
<tr>
<td>C80</td>
<td>2.97 ± 0.38</td>
<td>2.96 ± 0.42</td>
<td>3.64 ± 0.41</td>
</tr>
<tr>
<td>H100</td>
<td>3.70 ± 0.39†</td>
<td>3.98 ± 0.43†</td>
<td>4.18 ± 0.46†</td>
</tr>
<tr>
<td>C100</td>
<td>3.43 ± 0.35†</td>
<td>3.66 ± 0.37†</td>
<td>3.94 ± 0.51†</td>
</tr>
<tr>
<td>HR (b·min$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H80</td>
<td>153 ± 11</td>
<td>159 ± 19</td>
<td>184 ± 11</td>
</tr>
<tr>
<td>C80</td>
<td>148 ± 7</td>
<td>153 ± 7</td>
<td>180 ± 8</td>
</tr>
<tr>
<td>H100</td>
<td>164 ± 9†</td>
<td>171 ± 8†</td>
<td>181 ± 11</td>
</tr>
<tr>
<td>C100</td>
<td>161 ± 10†</td>
<td>169 ± 9†</td>
<td>178 ± 10</td>
</tr>
<tr>
<td>VE (L·min$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H80</td>
<td>75.97 ± 12.19</td>
<td>86.98 ± 12.16‡</td>
<td>148.74 ± 20.88‡</td>
</tr>
<tr>
<td>C80</td>
<td>70.98 ± 9.87</td>
<td>79.33 ± 11.11</td>
<td>127.81 ± 15.75</td>
</tr>
<tr>
<td>H100</td>
<td>106.81 ± 16.84‡</td>
<td>129.41 ± 18.78‡</td>
<td>164.29 ± 12.92‡</td>
</tr>
<tr>
<td>C100</td>
<td>101.07 ± 16.07‡</td>
<td>123.57 ± 15.55‡</td>
<td>151.59 ± 17.39‡</td>
</tr>
</tbody>
</table>

*HR = heart rate; VE = ventilation.†H80 and H100 are different from H80 and C80 based on an intensity by time interaction ($p < 0.001$).‡H80 and H100 are different from C80 and C100 based on an environment by time interaction ($p < 0.001$).
time interaction revealed that the pH responses were different among all conditions at exhaustion (Figure 3), with progressively lower values in the C80, H80, H100, and C100 conditions, respectively. At the recovery time point, the pH values in all conditions differed from each other with the exception of those observed in H100 and C100. Blood lactate levels differed only at the recovery time point, where a significant \( p = 0.036 \) intensity by time interaction showed that the levels in H80 and C80 were significantly lower than those in H100 and C100 (Figure 4). A significant \( p = 0.038 \) 3-way interaction revealed that the blood bicarbonate responses at exhaustion were significantly lower in H100 compared with C100 (18.02 ± 1.15 mM vs. 21.36 ± 0.68 mM), and the levels in H80 and C80 (15.58 ± 0.68 mM and 15.85 ± 0.77 mM) were significantly lower than in both the H100 and C100 trials. The bicarbonate levels at exhaustion in H80 and C80 did not, however, differ from each other.

Simple correlations were conducted within conditions to determine relationships between TTE and variables that might be related to performance (\( T_c \), lactate, pH). When expressed in absolute units or as the change in the various responses, the correlation coefficients for these comparisons ranged from a low of \( r = 0.04 \) (TTE vs. pH in H80) to a high of \( r = 0.54 \) (TTE vs. pH in C80); however, even the moderate correlation coefficients observed at the higher end of this range were not significant \( p = 0.086 \).

**DISCUSSION**

A primary finding of this study was that performance was impacted by an interaction between intensity and environment with varying thermal, metabolic, and cardiorespiratory responses observed at exhaustion among the trials. The greatest elevations in \( T_c \) were measured in the lower intensity conditions, suggesting that \( T_c \) is not always the primary determinant of exercise termination, and that similar absolute levels of elevated temperature may not be found consistently at the time of fatigue when the intensity and environment are systematically varied in relatively short bouts of exercise such as those used in this study. The pH levels in the higher intensity conditions underwent a greater decline compared with the lower intensity conditions, and recovery lactates were higher in the high-intensity conditions; thus, the metabolic state present at fatigue differed depending on the condition. The lack of relationships...
between TTE and $T_c$, pH, and lactate expressed either in absolute units or as a change in these variables suggests that these variables, by themselves, do not consistently explain fatigue under the conditions studied. The environmental impact on performance, resulting from the hotter and more humid conditions in the hot trials, was observed only in the lower intensity conditions, suggesting that in the high-intensity conditions, fatigue was because of other factors that occurred before the hot environment had a negative effect on performance.

In response to high-intensity exercise, elevations in $T_c$ up to 41.0°C have been reported after exercise in the heat and have been associated with an accelerated onset of fatigue (8,17). Of importance to our primary question, $T_c$ was significantly higher after exercise in H80 and C80 compared with H100 and C100; thus, a lower intensity combined with a longer duration produced the greatest heat storage but did not produce a more rapid termination of exercise. The failure to observe higher $T_c$ during the H100 and C100 trials does not parallel the expectation that greater elevations in $T_c$ would occur at higher relative workloads because of rapid rates of heat production (19); however, in most previous studies, lower intensities (<80% $\dot{V}_O_{2}$max) have been examined over longer periods of time (9,12,25). Therefore, a prolonged bout of exercise at, for example, 75% of $\dot{V}_O_{2}$max would likely lead to greater heat storage than one at 55% of $\dot{V}_O_{2}$max, resulting in an influence of exercise intensity on $T_c$ that is quite different from that observed in this study. Because this was a TTE model, comparing higher intensities where factors other than thermal strain may have contributed to fatigue, especially in the 100% conditions, there was inadequate time for significantly greater heat storage to occur. Although they used supramaximal repeated sprint exercise tests, Drust et al. (8) and Maxwell et al. (17) both reported that performance was impaired in a hot environment when $T_c$ was elevated. Although the differences in performance between C80 and H80 are in agreement with these earlier studies, the similarity in the performance results for the H100 and C100 conditions in the current investigation do not align with these findings. The variations in environmental and exercise protocols used in the previous studies make direct comparisons with the high-intensity conditions in the current study difficult. Specifically, the use of supramaximal exercise intensity, different combinations of environmental temperatures (20°C vs. 40°C (8) and 21°C vs. 33°C (17)), and intermittent sprint performance tasks over a different time periods (40+ minutes (8) and 150 seconds (17)) in the previous studies may explain the discrepancies.

The lack of similar $T_c$ responses at exhaustion among all the 4 conditions and the relatively modest increases observed even in the H80 and C80 conditions are not in agreement with the concept that exhaustion occurs at the same critical $T_c$ (12). It has been suggested that in trained individuals, a maximal $T_c$ of ~40°C may be tolerated, whereas in untrained individuals, lower values between 38 and 39°C may initiate fatigue (12). Our subjects were endurance-trained, yet the average peak $T_c$ of 38.5°C in the H80 and C80 conditions, although identical in both conditions, is well below the value proposed for trained subjects. Because the different $T_c$ responses observed between the 80 and 100% conditions do not support a critical $T_c$ at the higher exercise intensities used in this study, the question arises as to the role of muscle temperature elevations in causing fatigue, a response not measured in this study. Although some elevation in tissue temperature is beneficial to the muscle function, (23), there is no debate that high environmental temperatures accelerate heat storage or impair endurance performance when excessive $T_c$ and local tissue temperatures are present. The question arises as to whether a bout as short as 5–6 minutes in the 100% conditions could be limited by elevated $T_c$. Given the previous findings of reduced $\dot{V}_O_{2}$max in the heat (11,28) and the high rates of heat gain (~0.20°C·min⁻¹) with high-intensity exercise (19), it is conceivable that meaningful heat-induced alterations in tissue and systemic function can occur in a relatively short period of time. Because the elevations in the 100% conditions did not approach the previously postulated critical $T_c$ cut-offs, any effect of temperature would have to be either indirect through influences on other systems, that is CNS or cardiorespiratory, or based on other aspects of $T_c$ such as the rate of change.

The largest differences in performance were between the H80 and C80 conditions, yet the final $T_c$ was similar and the pH differences, although statistically significant, were not large; thus, the mechanism responsible for the nearly 2-fold increase in TTE in C80 is difficult to identify based on the final $T_c$ and metabolic responses. Clearly, the rate of onset of all these responses was slower in the C80 condition; thus, responses that may have limited performance in the C80 condition did not develop until much later. These results fit with the general expectation that exercise performance in the heat is impaired because of heat-induced factors, including cardiovascular strain, respiratory responses (VE was 20 L·min⁻¹ greater in H80 vs. C80), and possible fluid-related differences. Relative to the fluid status, we did not provide hydration during the exercise, and we did not measure responses indicative of fluid balance either as a pretrial measure or during the trials; thus, differences in this factor cannot be accounted for in this study. Given the relatively short duration of exercise in H80 (~12 minutes) where the greatest sweat loss would occur, even a relatively heavy sweat rate would be unlikely to produce a level of dehydration that would impair performance. In addition, although we did not measure skin temperature ($T_{sk}$), Cheuvront et al. (6) point out that higher $T_c$ can be tolerated when $T_{sk}$ is kept low. In the C80 trial, therefore, the subjects may have benefited from a lower $T_{sk}$, allowing them to continue with less cardiovascular strain.

It is not clear whether fatigue in this study was because of the peripheral or central mechanisms or a combination of...
the two (5,23). Further, if central effects are to be considered, it is necessary to explore the different hypotheses regarding CNS regulation of performance. Nybo (23) has suggested that an elevation to a critical level of $T_c$ might be related to a reduced CNS activation resulting from metabolic disturbances within brain tissue, or there may be peripheral metaboreceptors that generate afferent signals that are then processed to create inhibitory signals that reduce motor activation. Nybo and Nielsen (24) observed that hyperthermia was not associated with impaired muscular function; rather, a reduced central activation accounted for the reduction in maximal voluntary force development during prolonged isometric contractions. However, Noakes et al. (22) and others (14,30,31) have elaborated on the role of the CNS, proposing that exercise is terminated (30–32) as a preemptory measure to prevent damaging disruptions of homeostasis.

Although we do not have data that can be used to directly assess either of the aforementioned centrally mediated mechanisms of fatigue, some interpretation in the context of CNS regulation is possible. If $T_c$ is a monitored variable, the dissimilar responses when comparing the 100 and 80% conditions suggest that different variables may provide meaningful afferent information depending on the nature of the exercise. The physiological state found in the 80% conditions may have been such that $T_c$ responses represented more useful afferent information than in the 100% conditions because the levels in the latter conditions were not high enough to either alter CNS function or warrant curtailment of exercise. It has been proposed that the rate of heat gain is an important variable used by the CNS to regulate performance in the heat (12,30). Rates of heat gain of 0.15–0.20°C min$^{-1}$ have been reported during high-intensity exercise in the heat (19). In this study, the analysis of the rate of heat gain indicated that exercising at a higher intensity, especially in the heat, produced a greater rate of heat gain, although H100 and C100 resulted in significantly lower absolute $T_c$ at exhaustion compared with H80 and C80. Based on this analysis, our findings do not support the conclusion reported by others (12,30) that fatigue is related to the rate of heat gain. This conclusion is based on the fact that similar rates of gain in the H80 and C100 conditions corresponded to different performance durations, and different rates in the H100 and C100 conditions corresponded to similar durations. A significant relationship between TTE and either the absolute level or the change in temperature may have provided stronger evidence of $T_c$ as a regulator of exercise output; however, we observed no significant correlations based on any of these methods of expressing $T_c$. It should be kept in mind, however, that our fixed intensity TTE model does not allow the same level of interpretation of the anticipatory regulation as that provided by Tucker et al. (30) because they used a fixed RPE with variable pacing; thus, different regulatory dynamics are involved relative to performance.

Given that these exercise bouts required a substantial aerobic energy contribution, even in the higher intensity conditions, the role of cardiorespiratory function in fatigue should also be considered. Gonzalez-Alonso and Calbet (11) found that a reduction in cardiac output and mean arterial pressure led to a reduced $V_{O2,max}$ (>0.40 L min$^{-1}$ reduction) in heat stressed individuals exercising at a near-maximal intensity. These reductions were associated with declines in muscle blood flow and $O_2$ delivery. Our data do not allow a similar analysis of causes of fatigue based on cardiovascular responses; however, the comparison of $V_{O2}$ at exhaustion in H100 (4.18 L min$^{-1}$) indicates that it was nearly maximal ($V_{O2,max} = 4.23$ L min$^{-1}$) and was actually slightly higher in H100 compared with that observed in C100 (3.94 L min$^{-1}$). Although HR responses differed because of intensity early in the ride, at exhaustion, the similar responses did not reflect the effects of either environment or intensity, potentially identifying a maximal HR response as a marker of fatigue independent of the duration of exercise. The same cannot be said for ventilation responses because they were affected by both the intensity and environment. The elevated ventilation may have been partially because of the respiratory compensation of acidosis as indicated by the greater ventilatory responses in the 100% conditions where pH was lower. The elevated ventilation in the H80 compared with the C80 condition does not appear to be in response to pH; thus, it is likely that it is because of the effects of heat alone.

The pH levels observed at exhaustion and recovery across all the 4 conditions suggest that the contribution of pH to fatigue differed depending on the intensity and the environment. Although there is controversy regarding the role of pH in fatigue (4,33), an accumulation of hydrogen ions has been identified as a possible causal factor in fatigue; thus, the lower pH levels in both the 100% conditions compared with those in the 80% conditions suggest that pH may have played a greater role in fatigue during the 100% trials compared with the 80% trials. A peripheral fatigue model would suggest that hydrogen ion levels in the tissue brought about changes in cellular function that affected force output, and therefore, TTE; however, because of the differences in the absolute level of pH across all the 4 conditions, it does not appear that a single level of hydrogen ion concentration is responsible for a peripherally based fatigue mechanism. In fact, we found no meaningful relationship between absolute or change in pH and TTE; however, because of the differences in the absolute level of pH across all the 4 conditions, it does not appear that a single level of hydrogen ion concentration is responsible for a peripherally based fatigue mechanism. In fact, we found no meaningful relationship between absolute or change in pH and TTE; however, because of the differences in the absolute level of pH across all the 4 conditions, it does not appear that a single level of hydrogen ion concentration is responsible for a peripherally based fatigue mechanism. In fact, we found no meaningful relationship between absolute or change in pH and TTE; however, because of the differences in the absolute level of pH across all the 4 conditions, it does not appear that a single level of hydrogen ion concentration is responsible for a peripherally based fatigue mechanism.
If pH is an important factor in fatigue, and if peripheral mechanisms do not adequately explain fatigue, as in the case of $T_e$, it may be that pH is a monitored variable, and a change in exercise output may be dictated by the CNS to avoid large disturbances in hydrogen ion concentration (14, 22, 31). In this study, the largest changes in pH occurred with the 100% conditions where exercise termination occurred the earliest. Other authors have reported significant correlations between the rate of change in various variables and performance, for example, RPE (7); however, with a TTE model, it is not statistically viable to conduct this analysis because the 2 variables are not independent of each other because of the fact that performance time is a component of both the $X$ and $Y$ variables.

There was no difference in lactate at exhaustion, despite the fact that pH was different between trials at this time point, thus emphasizing the dissociation between these metabolites in contributing to fatigue. It has been established that hydrogen ions are released by sources other than lactic acid, explaining the disassociation between lactate and acidosis (27). At 3 minutes of recovery, however, there was a higher lactate level in the 100% intensity conditions that was not apparent, until the efflux of lactate into the blood had occurred (3). The absence of an environmental effect on lactate is consistent with the findings of Starkie et al. (29), who reported no differences in muscle lactate after 20 minutes of exercise in heated and chilled legs. High lactate levels have been associated with fatigue; however, any causal link between lactate and fatigue is questionable because lactate itself does not negatively affect cell function (4, 10). Because high lactate levels are associated with fatigue and because there is no viable peripherally based mechanism to explain the association between lactate and fatigue, it may be that its influence on performance is mediated through central processes (2, 4, 22, 26).

The bicarbonate results paralleled earlier research, in which bicarbonate decreased as a result of exhaustive cycling exercise regardless of the environment or intensity (18). There was, however, a significantly higher bicarbonate concentration at exhaustion in the C100 condition compared with all other conditions with a concomitantly lower pH. This finding suggests that the bicarbonate buffering reaction did not occur to the same extent in this condition compared with the others. Alterations in blood flow (increased subcutaneous flow paired with decreased venous return, cardiac output, and muscle blood flow) typically explain many of the differences in the metabolic responses observed in hot compared with neutral or cool environments (11, 12); however, the low pH in combination with a higher bicarbonate at exhaustion in the C100 condition are not responses that would be altered by the thermoregulatory redistribution of blood flow. Regardless, the reduced activity of the bicarbonate buffering system may have contributed to fatigue either through peripheral or centrally mediated mechanisms.

The results of this study support the concept that the exercise intensity and environment interact to produce different TTE results in trained individuals, extending previous findings to include exercise at high percentages of $V_{\text{O}_2}\text{max}$. The fact that there were no differences between the 2 trials completed at 100% of $V_{\text{O}_2}\text{max}$ was counter to our hypotheses and suggests that high intensity minimized the impact of environment. Across all the 4 conditions, no critical level of any single variable was associated with fatigue. Given that the analysis of single variables indicating changes in tissue conditions did not provide definitive conclusions regarding the cause of fatigue, it is reasonable to consider that the causes of fatigue are multiple, including peripheral and CNS factors, and that the causes of fatigue are task and environment specific (4, 22, 23). The fact that the absolute levels of $T_e$, pH, and lactate did not produce strong relationships with TTE suggests that either these variables individually are not causal, that absolute levels may not be the aspect of these variables that is most important. If these, and possibly other variables, do in fact interact with the CNS, the question remains as to which and how many variables act in a regulatory fashion under various exercise or environmental conditions? Future studies should be designed to examine these questions and to explore more thoroughly the impact of absolute levels, the change, or the rate of change of key variables as the best predictors of fatigue.

**Practical Applications**

To optimize training and prepare for competition, it is important that practitioners have a sound understanding of the cause of fatigue in individuals engaged in various types of physical activity under different environmental conditions. The findings from this study suggest that for higher intensity exercise, metabolic responses appear to override the negative impact of the hot environment (similar performance in H100 and C100). Based on this result, when preparing for high-intensity competitions in thermally stressful environments, the practitioner should place greater emphasis on training and other strategies to optimize metabolic responses as opposed to thermal responses. Conversely, at lower intensities, thermal factors should be accounted for to a greater extent; thus, interventions such as preexercise cooling, adequate acclimation, or other strategies to mitigate thermal effects may be appropriate.

**References**


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