Investigating the hydration status, metabolic and hematologic responses and cognitive-motor performance in cyclists under a high heat stress environment


ABSTRACT: The aim of this study was to evaluate the hydration status, metabolic responses and cognitive-motor performance during a combined cycling race in the heat. The combined cycling race (such as a simulated cycling race), consisted of 67 km under heat stress conditions. Before and after cycling race, ten male cyclists (30.5 ± 4.9 years) were evaluated for hydration status, biochemical and hematological parameters and cognitive-motor performance. Blood samples were collected for biochemical and hematological analysis. The hydration status was assessed by evaluating a change in body mass percentage loss (Δ% BM), urinary color, specific gravity and blood parameters. To evaluate cognitive-motor performance, we measured immediate memory, motor coordination and reaction time. Environmental conditions during exercise were an average of 27.1 ± 0.7 °C for Wet-Bulb Globe Temperature (WBGT) Index. The hydration status (Δ% BM: -3.26 ± 0.57), demonstrated that the cyclists were dehydrated after race. We found an increase in the concentrations of ammonia (Δ%: 74.94 ± 18.64) and its metabolites (urea and urate) after the race. The concentrations of blood glucose and lactate were not significantly different after the race. Exercise promoted a significant increase in enzyme biomarkers for muscle damage. Red blood cells counts were unchanged. However, leukocyte count (Δ%: 178.55 ± 28.64) increased significantly. No changes were observed in the cognitive-motor tasks. Under these conditions, the results of the present study indicate that dehydration and/or hyperammonemia does not negatively affect cognitive-motor performance in acclimated cyclists.

Key Words: Dehydration; Exercise; Hot temperature; Ammonia; Psychomotor performance.

RESUMO: O objetivo desse estudo foi avaliar o estado de hidratação, respostas metabólicas e desempenho cognitivo-motor durante uma corrida de ciclismo simulada no calor. A corrida de ciclismo consistiu de 67 km sobre condições de estresse térmico. Antes e após a corrida, dez ciclistas (30,5±4,9 anos) foram avaliados para o estado de hidratação, parâmetros bioquímicos e hematológicos e desempenho cognitivo-motor. Amostras sanguíneas foram coletadas para a análise bioquímica e hematólogica. O estado de hidratação foi avaliado através de mudanças no peso corporal (Δ% BM), cor da urina, gravidade específica e parâmetros sanguíneos. Para avaliar o desempenho cognitivo-motor, nós mensuramos a memória imediata, coordenação motora e o tempo de reação. Condições ambientais durante o exercício estavam em uma média de 27,1 ± 0,7 °C segundo o índice de estresse térmico (WBGT index). O estado de hidratação (Δ% BM: -3,26 ± 0,57) demonstrou que os ciclistas estavam desidratados após a corrida. Nós encontramos um aumento nas concentrações de amônia (Δ%: 74,94 ± 18,64) e seus metabólitos (ureia e urato) após a corrida. As concentrações de glicose sanguínea e lactato não foram diferentes antes e após a corrida. O exercício promoveu um aumento significativo nos biomarcadores enzimáticos de lesão muscular. A contagem de células sanguíneas vermelhas não foi diferente. Entretanto, a contagem de leucócitos aumentou significativamente (Δ%: 178,55 ± 28,64). Não foi observado diferenças no desempenho cognitivo-motor. Os resultados do presente estudo indicam que desidratação e hiperammonemia não afetam negativamente o desempenho cognitivo-motor em ciclistas aclimatados.

Palavras-chave: Desidratação; Exercício; Temperatura alta; Amônia; Desempenho psicomotor.
Introduction

Exercise in the heat induces dehydration and increases metabolic and hematological systems response, which may decrease exercise performance. Understanding this problem can help us (especially physiologists, coaches and athletes) to improve exercise capability and athletic performance.

It is suggested that dehydration causes the deterioration of motor skill performance and disturbances in cognitive function (termed cognitive-motor performance) during exercise, such as motor coordination, reaction time and memory. It has been postulated that symptoms appear when dehydration exceeds 2% body mass loss.

Likewise, environmental heat stress causes severe metabolic disorders during exercise, including increases in blood ammonia concentrations (hyperammonemia). It has been shown that hyperammonemia is highly toxic and can lead to cerebral function impairment, altering glutamatergic neurotransmission.

It has been suggested that hyperammonemia during exercise may cause central fatigue by altering cerebral function, which manifests as cognitive-motor disturbances (ataxia, lethargy and stupor), similar to the symptoms of dehydration and hepatic encephalopathy (situation in which cerebral function deteriorates due to failure in liver function). On the other hand, it is also possible that dehydration in athletes who are acclimated to exercise in the heat does not induce increased blood ammonia sufficient to impair cognitive-motor performance under a low heat stress environment.

Furthermore, this metabolic alteration can result in increases in oxidative stress, which leads to muscle damage as evidenced by the release of biomarkers (such as creatine kinase, lactate dehydrogenase, aspartate aminotransferase and alanine aminotransferase) and variations in different hematological variables, such as white blood cells, red blood cells and platelets counts. To the best of our knowledge, there is no information available about ammonia metabolism and cognitive-motor performance after exercise under heat stress conditions. In addition, studies observing athletes during exercise in the field in order to mimic both the real challenges and conditions that are faced during sports situations, such as Sportomics approach, are limited in number and scope.

Environmental conditions play a critical part during endurance exercise performance. Cycling races are among the most strenuous of endurance events, especially in the heat. Therefore, in the present study, we evaluated the hydration status and responses of metabolism and cognitive-motor performance during a combined (simulated) cycling race in the heat. We also sought to examine whether these conditions produce a rise in markers of muscle damage and hematological variables. Our hypothesis was that when exercising in high-heat stress conditions, dehydration and/or hyperammonemia impairs cognitive-motor performance and increases muscle damage and hematological changes.

Methods

Subjects

Ten endurance-trained male cyclists, with similar levels of maximal oxygen consumption (VO2max) (30.5 ± 4.9 years; 67.7 ± 1.5 kg; 1.74 ± 0.02 m; 59.41 ± 2.4 mL.kg⁻¹.min⁻¹) participated as volunteers. Cyclists have been training and competing for at least seven months and were acclimatized to training in the heat (repeated training–heat exposures over seven months). Either diseases or the use of ergogenic aids were exclusion criteria. The nature of the study and the procedures involved were described to all of the participants, and written informed consent was obtained from all of the cyclists. The Ethics Committee for Human Research at the Federal University of Alagoas approved all of the procedures involving human subjects (017640/2011-61).
The race and experimental design

One week before race, participants completed a dietary and anthropometric assessment and received an individualized diet plan corresponding to the following: 15% of the recommended energy intake was from protein, 25% was from lipids and 65% was from carbohydrates. The subjects were asked to start the planned diet at least two days before the race. The combined cycling race (such as a simulated cycling race) consisted of 67 km, between 09:00 AM and 01:00 PM. The cyclists cycled on a regular highway without transit interruption, with a safety car to protect them and were allowed to drink water *ad libitum* during race, but not carbohydrate. Also, the cyclists were instructed to maintain their typical cycling race intensity. Heart rate (HR) was recorded at the end of the race using a heart rate monitor (Polar® FT1, Kempele, Finland). Before (Pre) and after (Post) cycling race, cyclists were evaluated for hydration status, biochemical and hematological parameters and cognitive-motor performance.

Hydration status

The hydration status was assessed by evaluating percentage changes in body mass (Δ% BM), urine color and specific gravity (SG). The urine color was evaluated according to Armstrong *et al.*[^20] and SG was measured using a manual refractometer (Biobrix®, São Paulo, Brazil). To evaluate the consumption of a low-carbohydrate diet, urine samples were used for the ketonuria analysis by qualitative reagent strips for urinalysis (Biocolor/Bioeasy®, Minas Gerais, Brazil). We considered the absence of ketonuria to be a positive test for adequate carbohydrate ingestion before a cycling race that does not induce early hyperammonemia.

To further assess the hydration status, we recorded the exercise time in hours (h), as well as the total fluid intake (water) consumed by each cyclist (mL) to calculate the sweat rate (SR) according to Casa *et al.*[^5], as described below:

\[
\text{SR} = \frac{[(\text{BMpre} - \text{BMpost}) + \text{FIt} - \text{VUt}]}{\text{T}}
\]

Where BMpre is the body mass (kg) before cycling, BMpost is the body mass (kg) after cycling, FIt is the total fluid intake consumed in the exercise, VUt is the total urine volume (L) after exercise, and T is time (h).

In addition, other hydration markers were obtained using blood parameters including the blood urea nitrogen (BUN)/creatinine ratio and the plasma volume loss estimate (Δ% PV), with hemoglobin (Hb) and hematocrit (Hct) counts according to Dill and Costill[^21] and calculated as:

\[
\Delta \% \text{ PV} = \left( \frac{\text{Hbpre}}{\text{Hbpost}} \right) \times \left( \frac{1 - \text{Hctpost}}{1 - \text{Hctpre}} \right) - 1 \right) \times 100
\]

Biochemical and hematological analyses

Pre and post race, blood samples were obtained from the median antecubital vein, of which 3 mL were used for biochemical analysis, and 2 mL was collected in a vacuum tube with the anticoagulant EDTA for hematological analysis. The blood samples used for biochemical analysis were immediately centrifuged (3000 g) to avoid the loss of volatile compounds. The serum was aliquoted and stored at 4 °C. To prevent loss, seric ammonia was measured immediately, and the other biochemical analyses were performed within a 24 h period.

The biochemical determination of urea, ammonia, BUN, urate, glucose, lactate, creatinine, enzyme creatine kinase (CK), lactate dehydrogenase (LDH), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were measured using commercial spectrophotometric assays (Biospectro®, Paraná, Brazil).

The hematological analysis was performed by a hematology analyzer (Human®, Hessen, Germany) to obtain
the following counts and/or indexes: erytrocite, Hb, Hct, mean cell volume (MCV), mean cell hemoglobin (MCH), mean cell hemoglobin concentration (MCHC), red cell distribution width (RDW), platelet count (Plt), mean platelet volume (MPV), leukocytes and its subpopulations (neutrophils, lymphocytes, monocytes, eosinophils and basophils).

**Cognitive-motor performance**

To evaluate cognitive-motor performance, we used measures of immediate memory, motor coordination and simple reaction time (SRT)\(^{11,22-27}\). Immediate memory was evaluated as described by McCrory et al.\(^{28}\) and McCrory et al.\(^{29}\). A list of five words (one word per second - s) was given, and the cyclist was asked to repeat as many words as possible in any order. The same list was repeated three times in 10 s intervals (Figure 1). The maximum number of correct words remembered was taken as the score.

**Figure 1.** Immediate memory test.

Motor coordination was evaluated using the finger-to-nose test adapted from McCrory et al.\(^{28}\) and McCrory et al.\(^{29}\). Briefly, while seated and facing the examiner (having their dominant arm laterally extended at a 90° angle in relation to the body and their eyes open – Figure 2A), the cyclists were asked to touch the tip of their nose with their extended index finger (Figure 2B) and return to the initial position. Cyclists were asked to repeat this procedure as quickly and as accurately as possible, five times. The cognitive tests were filmed for analysis by five independent evaluators at a later time. All measures were timed using a stopwatch accurate to 0.01 s.

**Figure 2.** (A) Initial position; (B) Finger to nose repetitions using index finger to touch the tip of the nose.
The SRT, as described by Eckner et al. and Eckner et al., was obtained immediately after the finger-to-nose test. While seated, the cyclists had their dominant arm flexed and abducted to 90°, rested on a flat surface with their outstretched hand in a neutral position, also forming the same angle perpendicular to the thumb. The zero mark of the apparatus was positioned inside the open hand of the cyclist. The examiner (standing) vertically suspends the apparatus, allowing the spacer portion of the device to rest inside the open hand of the test participant (Figure 3A). When the examiner drops the apparatus, the test participant catches it as quickly as possible (Figure 3B). The cyclists were tested in 8 trials, and the test average was determined. The fall distance was measured from the superior surface of the weighted disk to the most superior aspect of the cyclist’s hand. The fall distance was converted into a reaction time using the formula for a body falling under the influence of gravity: \( d = \frac{1}{2} gt^2 \), where \( d \) (cm) is the distance, \( g \) is the acceleration due to gravity and \( t \) is the time (in milliseconds - ms).

**Figure 3.** (A) Initial position; (B) Final position.

### Environmental conditions

During the combined cycling race, we used the ambient temperature, the relative humidity, and the global temperature to calculate the Wet-Bulb Globe Temperature (WBGT) Index (Instrutemp®, São Paulo, Brazil). We considered a WBGT of up to 22.2 °C to be a low heat stress environment.

### Statistical analysis

All values are expressed as mean ± SD. After testing for normality (Kolmogorov–Smirnov), the changes in the variables (Pre and Post cycling race) were analyzed using a paired t-test and Wilcoxon test for unpaired variables outside the normal distribution. The level of significance was set at \( P < 0.05 \).

### Results

We used a combined cycling race conducted in the heat to evaluate the hydration status and responses of metabolism and cognitive-motor performance in cyclists. The environmental conditions indicated a high thermal stress on race day (Table 1). The cyclists finished the race in ~ 2:30 h and the HR reached 146.6 ± 12.8 beats/min at the end of the race. Furthermore, the ketonuria analysis revealed that cyclists were not in ketosis before the race.

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We observed a significant increase in the concentrations of urea, urate, ammonia and creatinine after the race. On the other hand, the concentrations of blood glucose and lactate were not significantly different after the race. We also evaluated enzyme biomarkers for muscle damage in response to race in the heat. Exercise promoted a significant increase in CK and AST (Table 2).

<table>
<thead>
<tr>
<th>Table 1. Environmental conditions during race.</th>
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<tr>
<td>WBGT (°C)</td>
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<td></td>
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<tr>
<td>Ambient temperature (°C)</td>
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<tr>
<td>Global temperature (°C)</td>
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<tr>
<td>Relative humidity (%)</td>
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</table>

Wet-Bulb Globe Temperature (WBGT).

Red blood cells were unchanged, however platelet count increased significantly (Table 3).

<table>
<thead>
<tr>
<th>Table 2. Biochemical parameters before and after cycling in heat.</th>
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<tr>
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<tr>
<td>Ammonia (µmol/L)</td>
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<tr>
<td>Urea (mmol/L)</td>
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<tr>
<td>Urate (µmol/L)</td>
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<tr>
<td>Glucose (mmol/L)</td>
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<tr>
<td>Lactate (mmol/L)</td>
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<tr>
<td>Creatinine (µmol/L)</td>
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<tr>
<td>CK (µkat/L)</td>
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<tr>
<td>LDH (µkat/L)</td>
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<td>AST (µkat/L)</td>
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<td>ALT (µkat/L)</td>
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</tbody>
</table>

Values are expressed as mean ± SD. Percentage changes (Δ%). Enzyme creatine kinase (CK); lactate dehydrogenase (LDH); aspartate aminotransferase (AST) and alanine aminotransferase (ALT). *Significant changes between Pre and Post race P < 0.05.

<table>
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<tr>
<th>Table 3. The hematological parameters of red blood cells and platelets counts before and after cycling in heat.</th>
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<tr>
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<tr>
<td>Erythrocytes (x 10^{12}/L)</td>
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<tr>
<td>Hemoglobin (mmol/L)</td>
</tr>
<tr>
<td>Hematocrit (% x 0.01)</td>
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<tr>
<td>MCV (fl)</td>
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<tr>
<td>MCH (pg)</td>
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<tr>
<td>MCHC (g/L)</td>
</tr>
<tr>
<td>RDW (% x 0.01)</td>
</tr>
<tr>
<td>Plt (x 10^{7}/L)</td>
</tr>
<tr>
<td>MPV (fl)</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD. Percentage changes (Δ%). Mean cell volume (MCV), mean cell hemoglobin (MCH), mean cell hemoglobin concentration (MCHC), red cell distribution width (RDW), platelet count (Plt), mean olatelet volume (MPV). *Significant changes between Pre and Post race P < 0.05.
Leukocytes, neutrophils, monocytes and basophils counts were all significantly increased. On the other hand, lymphocytes and eosinophils were unchanged (Table 4).

Table 4. The hematological parameters of white blood cells and its subpopulations counts before and after cycling in heat.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>Δ %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukocytes (x 10^9/L)</td>
<td>6.58 ± 1.58</td>
<td>18.45 ± 7.06*</td>
<td>178.55 ± 85.93</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>3.63 ± 1.10</td>
<td>14.58 ± 3.19*</td>
<td>344.68 ± 190.97</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>2.05 ± 0.81</td>
<td>2.35 ± 0.98</td>
<td>81.91 ± 60.88</td>
</tr>
<tr>
<td>Monocytes</td>
<td>0.57 ± 0.15</td>
<td>0.97 ± 0.27*</td>
<td>486.59 ± 803.85</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0.26 ± 0.09</td>
<td>0.25 ± 0.05</td>
<td>-1.70 ± 33.72</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.05 ± 0.02</td>
<td>0.21 ± 0.17*</td>
<td>-</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD. Percentage changes (Δ%). *Significant changes between Pre and Post race P < 0.05.

The BM decreased significantly between the Pre (67.7 ± 4.8 kg) and the Post time points (65.6 ± 5.5 kg), and the percentage change was Δ% BM = - 3.26 ± 1.83 (Figure 4A); SR = 1.86 ± 0.44 L/h, and Δ% PV = - 2.84 ± 9.14. There was no significant difference between the BUN/creatinine ratio Pre (15.57 ± 3.97) and Post (15.08 ± 4.61) race (Figure 4B). Likewise, there was no significant difference in SG (Pre: 1023.5 ± 7.9, Post: 1026.9 ± 5.1; P = 0.058) and urine color (Pre 5.2 ± 1.8, and Post, 6.5 ± 1.4) before and after the race (Figure 4C and 4D, respectively). These results suggest that cyclists were dehydrated both before and after the race.

In addition, we were not able to detect any significant difference in the cognitive-motor performances. There was no significant difference between the Pre (4.78 ± 0.73) and Post (4.53 ± 1.10) (zero point) time points in motor coordination (Figure 5A). Also, the reaction time and immediate memory tests showed no changes between the Pre (29.6 ± 6.4 cm; 13.1 ± 1.1) and Post (27.4 ± 5.3 cm; 12.4 ± 2.2) time points, respectively (Figure 5B and 5C, respectively).

Figure 4. (A) Body Mass (BM) and Δ% BM (inset); (B) Blood Urea Nitrogen (BUN)/Creatinine ratio; (C) Specific Gravity (SG); and (D) Urine Color Index, before and after cycling. * Significant changes (P < 0.05).
Figure 5. (A) Finger-to-nose test (in seconds – s); (B) Simple Reaction Time (SRT), measured in cm and SRT measured in ms (inset); and (C) Immediate Memory (amount of correct words remembered), before and after cycling.

Discussion

The aim of this study was to evaluate the hydration status and responses of metabolism and cognitive-motor performance after a combined cycling race conducted in the heat. Here we showed that cyclists were dehydrated. Furthermore, we report changes in metabolism, such as hyperammonemia, the presence of biomarkers of muscle injury in the bloodstream and leukocyte counts, but we were not able to detect any significant difference in the cognitive-motor performance.

It is known that exercise-induced dehydration may impair cognitive-motor performance\textsuperscript{10,30}. Furthermore, it has been suggested that cognitive-motor performance is impaired when there is a loss of more than 2 % of body mass\textsuperscript{5,8,31}. Subjects reported that their ability to concentrate and their alertness were reduced following fluid restriction (~ 2.7% body mass loss)\textsuperscript{32}. Cian \textit{et al.} \textsuperscript{33} demonstrated that dehydration affects the short term memory but not the long-term memory. On the other hand, dehydration did not impair the processes involved in reaction time. In this study, the subjects were dehydrated (loss > 3 % of body mass and increase in blood creatinine concentrations). Under these
conditions, the cyclists showed no any difference in the cognitive-motor performance.

Although cognitive-motor performance can be affected by dehydration, the mechanisms responsible for these effects have not been clearly elucidated. It is possible to postulate that the main mechanisms involved are: hypercortisolemia, elevated cerebral arginine vasopressin, enhanced nitric oxide synthase release, mitochondrial dysfunction, glutamate hypertransmission and cytokines elaboration.

On the other hand, several studies have shown that dehydration, *per se*, does not affect the cognitive-motor performance, but may be attributed to other factors involved, such as hyperammonemia. In addition, it is important to mention that an athlete’s acclimation to heat can influence these results due to a greater tolerance to heat stress. Therefore, it is possible that better acclimation to the heat may have led to a reduction in cognitive-motor impairment.

It is recognized that hyperammonemia can induce central nervous system dysfunction, which is associated with impairments in cognitive-motor performance, by causing an imbalance in the neurotransmitters. Exercise-induced hyperammonemia is independent of environmental temperature. Previous studies have suggested that a hot environment contributes to exacerbated exercise-induced hyperammonemia. Mohr et al. demonstrated exacerbated exercise-induced hyperammonemia in individuals undergoing prolonged exercise in the heat (40°C) compared to those undergoing prolonged exercise at 20°C. There was a significant increase of ammonia (~70%) in long-distance runners after 2 h of exercise in ambient temperatures ranging from ~25°C to 28°C. Similarly, in our study, cycling in the heat increased the concentrations of ammonia by ~75% after ~2:30 h of racing in an ambient temperature of ~31°C.

The condition of hyperammonemia is linked to increased urea and urate blood levels, which are ammonia metabolites. Urea blood levels reflects amino acid deamination, which can be exacerbated by both low-carbohydrate diet (such as ketogenic diet to induce ketosis and ketonuria) and heat. In the present study, we were not able to measure any ketonuria, but there was an increase in urea concentrations, as well as the maintenance of the glycemia and blood lactate concentrations. The data presented here may indicate an additive effect of the heat and the dehydration. However, Prado et al. also showed that significant increases in blood urea levels were associated with no changes in glucose and lactate levels in cyclists during prolonged exercise, but under thermoneutral conditions (temperature, ~23°C and relative humidity, ~60%).

Furthermore, urate also increased significantly. Urate is a metabolite derivative from deamination of adenosine monophosphate that is pronounced during exercise. In this way, the results of the present experiment are consistent with Bessa et al. who showed a significant increase in the concentration of urate after a long-distance cycling race.

There is evidence that increased levels of urate from exercise promotes reactive species and free radicals production, which leads to cellular muscle damage and allows biomarkers to be released into the bloodstream for hours or days after the exercise session. Although Nybo et al. did not detect changes in CK concentrations immediately after a soccer match held in a hot environment, other reports have demonstrated that heat contributes to the exacerbation of CK concentrations. In the current study, we noted an increase in CK and AST levels immediately after a cycling race, most likely as a result of the heat. Our data are in agreement with a previous report of changes in injury biomarkers after a high-intensity ultraendurance cycling.

We also demonstrated an increase in platelet and white blood cells counts after a cycling race, but not in red blood cells. It is suggested that platelet count is increased by exercise, and this response depends on several factors including exercise intensity, exercise duration and the physical fitness status of the individual, but future studies are needed to elucidate the effect of exercise in hot environments.

It is also important to highlight that one of the most studied aspects of exercise and the immune system is the
changes in leukocyte numbers (mainly neutrophils) in circulating blood. It is believed that during exercise, the main source of circulatory neutrophils are primary (bone marrow) and secondary (spleen, lymph nodes, gut) lymphoid tissues, as well as marginated neutrophils from the endothelial wall of peripheral veins. Leukocytosis was observed in athletes who participated in prolonged cycling sessions. Mestre-Alfaro et al. have reported increased leukocytosis by neutrophilia after prolonged exercise in the heat (TA ~ 32 ºC e UR ~ 85 %). In our study, we measured an increase in leukocytes (~ 180 %) in blood after race due to the increase of neutrophils (~ 345 %), monocytes (~ 80 %) and basophils (~ 485 %). On the other hand, although hematocrit and hemoglobin have been proposed as biomarkers of hydration status, no changes were observed in red blood cell distributions or other associated indexes.

This field study did not demonstrate a hydrated group (control group) and we did not verify body temperature, a potential limitation of the current study, but this parameter should be evaluated in future studies. On the other hand, the present study presents data obtained during a combined cycling race as an approach to mimic the challenges and conditions that are faced during sports training and competition. Such design allows for description of the real responses during a cycling race, generating cognitive-motor responses closer to real conditions, such as Sportomics approach.

Conclusions

Collectively, the results of the present study indicate that, under these conditions, dehydration and/or hyperammonemia does not negatively affect cognitive-motor performance in acclimated cyclists. Furthermore, we report changes in enzyme biomarkers of muscle damage levels and leukocyte counts. This combined information can provide important information during exercise in the heat and to assist coaches with training management and performance improvement.

Acknowledgments

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