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Relatório Final

*(Anexo 8)*

Lacerda ACR, Gripp F, Rodrigues LOC, Silami-Garcia E, Coimbra DD, Prado LS. 
Acute heat exposure increases high-intensity performance during sprint cycle exercise

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Abstract The purpose of this study was to investigate the effects of acute heat exposure at thermal balance on high-intensity performance during sprint cycle exercise. Nine healthy male subjects were tested in three different, well-controlled environments in an environmental chamber: T (22°C, 65% RH), H1 (30°C, 55% RH) and H2 (35°C, 62% RH), each test being carried out on a different day following a randomized sequence. After 30 min of exposure to the set environment, subjects performed the 30-s sprint cycle exercise. Heart rate, rectal and skin temperatures were measured prior to exercise, at rest, before and after environmental exposure, and after exercise. There were no differences in subjects’ core temperature or heart rate prior to exercise. However, skin temperature was significantly higher in hot trials compared with the control throughout the experimental session (P < 0.05). Peak power was significantly higher in the hot environments compared with the control. Mean power was higher only in H2 compared with T (P < 0.05). This difference in power output was the consequence of a faster pedaling cadence in the hot trials (P < 0.05). Plasma ammonia was higher in the hot trials versus control at 4 min post-sprint. No differences in blood lactate levels at 3 min post-sprint were observed between tests. The results of this study suggest that the exposure to hot environment caused an improvement in power output for a single 30-s sprint. This increase in power output was associated with an elevation in plasma ammonia suggestive of an increase in adenine nucleotide loss.

Keywords High-intensity exercise · Temperature · Power

Introduction

There is little information available concerning the influence of exposure to hot environments on anaerobic performance (Ball et al. 1999; Falk et al. 1998; Hoffman et al. 1997; Rotstein et al. 1998). Furthermore, the results of these studies are conflicting. In some studies, high temperatures have been reported not to affect performance during high-intensity, short duration exercise (Backx et al. 2000; Bar-Or 1987; Davies and Young 1983; Dotan and Bar-Or 1980; Gray and Nimmo 2001; Hue et al. 2003; Rowell 1974), while other studies have shown heat exposure to decrease (Drust et al. 2005) or improve anaerobic performance (Ball et al. 1999; Falk et al. 1998; Linnane et al. 2004; Sargeant 1987). Nevertheless, Ball et al. (1999) and Falk et al. (1998) demonstrated an increase in peak and mean power during high-intensity, intermittent exercise performed following heat exposure. However, no difference in blood lactate was observed between the different environmental conditions, indicating that the
increased performance was not related to blood lactate production. In these studies, blood ammonia was not measured and this information may be helpful in understanding the effect of heat exposure on anaerobic metabolism.

Ball et al. (1999) also showed a higher pedaling cadence during higher performance in the heat, thus suggesting that exposure to a warm environment may have led to a favorable shift in the force/velocity relationship of muscle fibers. An improvement in performance during an initial bout of high-intensity cycle exercise following hyperthermia compared to controls was also observed by Linnane et al. (2004).

Furthermore, they observed higher plasma ammonia concentrations after sprints in the hot trial versus the control trial. However, blood lactate levels were higher in the hot trial compared to the control trial only after the second sprint (Linnane et al. 2004).

Therefore, we investigated the effect of heat exposure on high-intensity performance during a sprint cycle exercise. We hypothesized that when exercising in conditions of high ambient heat, but in which rectal temperature is within normal range, the ability to generate maximum short-term power is enhanced.

Methods

Subjects

Nine healthy males participated in the study. Their mean (+SEM) height, weight, age, body fat, estimated peak oxygen consumption, and body surface area were 177.9 ± 2.1 cm, 68.5 ± 2.4 kg, 23.8 ± 1.7 yr, 9.3 ± 0.1%, 55.7 ± 3.5 ml kg⁻¹ min⁻¹, and 1.85 ± 0.03 m², respectively. Testing took place during the months of September–October, which is springtime in Brazil. The mean temperature and air relative humidity (RH) at this time of the year are −23.4°C and −57.6%, respectively (5th district of Meteorology/INMET).

Subjects were informed of the nature of the study and all signed informed consent forms in accordance with the requirements of our Institution’s Ethical Committee on Human Experimentation (protocol ETIC 112/00). Subjects were requested not to change their dietary habits nor to perform any physical activity or drink any alcoholic beverage in the 24 h prior to the test. In addition, they were requested to avoid eating any food or drinking any beverage containing caffeine in the 48 h prior to the test, to inform staff of the use of any medication, to sleep well the night before the test, and to drink plenty of fluids (at least 500 ml) 2 h before the test (ACSM 1996). Subjects’ compliance with these instructions was verbally confirmed upon arrival at the laboratory.

Experimental design

The study design included four preliminary sessions, each performed on a different day followed by three balanced-order experimental tests, also performed on different days. The first preliminary session included a physical examination, anthropometric measurements (height, and weight), skin-fold thickness measurements at three sites (chest, abdomen, and thigh) for the assessment of percentage of body fat (Brozek et al. 1963; Jackson and Pollock 1978), and determination of peak oxygen consumption (VO₂peak), using a progressive test on a Monark standart ergometer cycle. The test consisted of cycling initially at 25 W and thereafter increasing by 25 W every 2 min until fatigue. The test was terminated when subjects could no longer maintain the required power despite verbal encouragement from the investigator. The greatest power value obtained by each one of the subjects was registered and VO₂peak was determined by using the equation: VO₂peak (ml O₂ kg⁻¹ min⁻¹) = 300 + (12 × power (W))/body mass (kg) (ACSM 1995). These procedures were carried out in a normal temperature environment: 22°C, 65% (RH), in an environmental chamber (Clark and Edholm 1985; Haynes and Wells 1986; Pandolf et al. 1986).

Three subsequent sessions were performed to familiarize the subjects with the proceedings. During these sessions, the subjects performed 3 min of low intensity cycling interspersed with 2–3 all-out sprints of 4–7 s duration against the highest resistance that would still allow a sprinting pace (Dotan and Bar-Or 1983).

On the other three occasions, subjects were asked to come to the laboratory to perform the Wingate Anaerobic Test (Bar-Or 1987; Dotan and Bar-Or 1983; Vandewalle et al. 1987). During this test, the volunteers were asked to perform one sprint of 30 s of intense cycling on the ergometer against a resistance determined according to the subject’s body mass (75 g kg⁻¹ body mass). The sessions were performed in T (22°C, 65% RH), H1 (30°C, 55% RH) or H2 (35°C, 62% RH) environments. The hot environments were chosen to simulate competition conditions.

Climatic chamber protocol

During the test, the subjects were dressed in shorts, socks and running shoes. Prior to entering the climatic chamber, they had a 15-min period of rest in a room near the climatic chamber. Upon entry to the climatic
chamber, subjects stayed seated for 30 min. Subsequently, they were asked to perform a 5-min warm-up on a cycle ergometer, similar to the exercise previously performed during the familiarization procedure. One minute following the warm-up, subjects performed a sprint consisting of 30 s intense cycling on the cycle ergometer. Thereafter, subjects remained in each of the respective environments in the climatic chamber during the 20 min of recovery.

Power output was recorded online by a computer connected to the Monark standard cycle ergometer during the 30-s sprint test. For this purpose, a MCE model software (Multi Cycle Ergometer, 2.3 version, Warsaw Sports Institute, Poland) was used. An electronic sensor was placed on the pedal in order to measure the occurrence of a complete pedal cycle. Sensor status was sampled at 1.000 Hz and fed online the MCE software. Since pedal is coupled to the wheel, consisting a pedal-wheel system, one complete pedal revolution corresponds to 3.71 wheel rotations, which, in turn, corresponds to $2\times25\times3.71 = 6\ m$ (Monark standard cycle ergometer). The power output was calculated as function of the time taken for each pedal revolution cycle according to the formula 1. Thus, a time table of power output throughout the 30 s recording period allowed the software to calculate specific parameters for analysis, such as peak and mean power, time to peak power, and pedal cadence.

**Formula 1:**

$$P = k/t_0$$

where $P = $ power output (W); $t_0 =$ measurement time related to one complete revolution; $k =$ variable expressed as:

$$k = 9.81 \times m_Q \times S_{erg}$$

where gravitational acceleration = $9.81\ \text{m/s}^2$; $m_Q =$ resistance determined according to the subject's body mass (75 $\text{g kg}^{-1}$ body mass); $S_{erg} =$ wheel surface traveled distance (m).

Blood lactate concentrations were measured before and after exposure to the different environments, and 3 min after the 30-s sprint test. A 20 $\mu l$ blood sample was taken from a hyperemized ear lobe and analyzed using the Accusport BM Lactate Kit (Boehringer, Germany).

Blood ammonia was measured 4 min after the 30-s sprint test in a 20 $\mu l$ blood sample collected from the hyperemized ear lobe. The sample was analyzed enzymatically using the Ammonia Checker II (Arkray, Japan).

Heart rate (HR), rectal and skin temperatures ($T_{re}$ and $T_{sk}$, respectively) were monitored every 10 min and were recorded immediately upon entry to the climatic chamber, continuing until 20 min into the recovery period. HR was measured using a heart rate monitor (Polar, Vantage NG model). $T_{re}$, considered an index of internal temperature, was measured using a rectal thermistor probe (YSI, Ohio) inserted 10 cm beyond the anal sphincter. $T_{sk}$ was determined using thermistors (YSI, Ohio) at four sites: chest ($T_{ch}$), upper arm ($T_{ar}$), thigh ($T_{th}$), and medial thigh calf ($T_{ca}$).

Mean skin temperature was calculated according to the Ramanathan equation: $T_{sk}$ ($C$) = 0.3 ($T_{ch} + T_{ar}$) + 0.2 ($T_{th} + T_{ca}$) (Ramanathan 1964). Calculation of the heat loss index (HLI) was based on the $T_{re}$ and $T_{sk}$ measurements according to the Romanovsky equation: ($T_{sk}$–environmental temperature)/( $T_{re}$–environmental temperature) (Romanovsky et al. 2002).

**Statistical analysis**

Mean and peak power, time to peak power, pedal cadence, and blood ammonia concentrations were analyzed using analysis of variance (ANOVA) for repeated measures. The variables blood lactate, rectal and skin temperature, heat loss index, and heart rate were analyzed using an analysis of variance (ANOVA) for repeated measures and subdivided parts. Student's $t$-test was performed to assess differences between the means, differences at $P < 0.05$ being considered significant.

**Results**

The peak and mean power, time to peak power, and pedal cadence developed during the 30-s sprint test are presented in Table 1. The peak power and pedal cadence were higher in H2 when compared to H1 and T. Moreover the peak power was higher in H1 when compared to T. Mean power was found to be higher in H2 compared to T. However, there was no difference in mean power between T and H1 or between H1 and H2. Time to peak power were higher in H1 and H2 when compared to T.

There was no difference in rectal temperature between the three environmental conditions during 30 min of exposure, indicating that subjects performed the experimental procedures at the same core temperature (Fig. 1a). However, after 20 min of the recovery period, rectal temperature was higher in H2 when compared to H1 and T. It was also higher in H1 compared with T. As
Table 1  Effect of the three environmental conditions—T (22°C/65% RH), H1 (30°C/55% RH) and H2 (35°C/62% RH)—on performance variables during sprint cycle exercise

<table>
<thead>
<tr>
<th>Performance variables</th>
<th>Environments</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T (22°C, 65% RH)</td>
<td>H1 (30°C, 55% RH)</td>
<td>H2 (35°C, 62% RH)</td>
</tr>
<tr>
<td>Peak power (W)</td>
<td>851 ± 38</td>
<td>880 ± 38*</td>
<td>903 ± 44*</td>
</tr>
<tr>
<td>Time to peak power (s)</td>
<td>2 ± 0</td>
<td>3 ± 0*</td>
<td>3 ± 0*</td>
</tr>
<tr>
<td>Mean power (W)</td>
<td>673 ± 32</td>
<td>688 ± 30</td>
<td>700 ± 31*</td>
</tr>
<tr>
<td>Pedal cadence (RPM)</td>
<td>133 ± 3</td>
<td>136 ± 3</td>
<td>139 ± 3**</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM

*H2 significantly different from T (P < 0.05)
* H2 significantly different from H1 (P < 0.05)
* H1 significantly different from T (P < 0.05)

shown in Fig. 1b skin temperature changed around a new equilibrium point according to the environmental chamber. In the environmental chamber, mean skin temperature at rest following 30 min of environmental exposure was (T = 28.9 ± 0.4°C, H1 = 33.4 ± 0.5°C, H2 = 35.5 ± 0.3°C; P < 0.05). The heat loss index was lower in H2 throughout the entire experiment compared to the other environmental conditions. However, there was a significant reduction in heat loss index in this hotter environment during the first 10 min of exposure, thereafter a steady state was achieved and maintained until the end of the experiment (Fig. 1c).

There was no difference in heart rate between the three environmental conditions during 30 min of exposure. However, following exercise, heart rate increased significantly only in H2 compared with T (Fig. 2).

There was no difference in blood lactate concentration between environmental conditions during any of the experimental procedures. However, blood lactate concentration was higher after exercise compared to resting conditions (Fig. 3).

Ammonia blood concentration after exercise was higher in H1 and H2 compared with T (T = 147 + 19 μmol/l, H1 = 184 + 16 μmol/l, H2 = 189 + 16 μmol/l; P < 0.05). However, there was no difference between H1 and H2 (Fig. 4).

Discussion

The main finding of this study is that heat exposure increased high-intensity power output during sprint cycle exercise compared to a normal temperature environment (Clark and Edholm 1985; Haymes and Wells 1986; Pandolf et al. 1986). This improvement was more evident in peak power than in mean power. An improvement in high-intensity power output has also been observed by Linnane et al. (2004). These investigators suggested that the increase in core body temperature was responsible for the improvement in performance during an initial bout of high-intensity cycle exercise.

In the present study, there was no difference in resting rectal temperature and heart rate following 30 min of exposure to the different environmental conditions. However, following high intensity exercise, rectal temperature was higher in the hotter conditions compared to the normal temperature environment at 20 min of recovery. These results suggest that the higher power output in combination with the environmental factor, resulted in the higher core body temperature during the recovery period. This supposition can be supported by the lower heat loss index in the hotter condition, suggesting either heat gain or negligible heat loss.

The improvement in high-intensity power output in the present study was circa 6% in maximal peak power and circa 4% in mean power in the hotter environment compared to the control condition. This increase in power was associated with higher plasma ammonia concentrations as a consequence of AMP deamination to IMP, which contributes to the maintenance of the energy charge within the working muscle cell, thus enabling a higher rate of cross-bridge turnover (Kara 2001).

The results from this study support similar investigations into the effects of acute temperature changes on power output (Amsussen and Boje 1945; Sargeant 1987). Sargeant (1987) suggested that power production increases by an average of 4% for every 1°C rise in muscle temperature, which is a consequence of a higher rate of cross-bridge cycling.

It is important to mention that power output was not corrected for flywheel acceleration in the present study, which consists a limitation for data interpretation. According to Lakomy (1986), the highest power output value over 5 s during the Wingate test was...
found to be underestimated by 29.4% when unadjusted values were used. However, the data in the present study clearly showed a higher peak power output in the hotter conditions.

The exposure of subjects to hotter conditions should have increased muscle temperature. However, as this variable was not measured in the present study, the
Fig. 4 Effect of three environmental conditions—T (22°C/65% RH), H1 (30°C/55% RH) and H2 (35°C/62% RH)—on blood ammonia concentrations after sprint cycle exercise. Data are expressed as mean ± SEM. *H2 significantly different from T (P < 0.05); † H1 significantly different from T (P < 0.05)

precise magnitude of this increase is unknown. This supposition was based on the study of Webb (1992) that showed that skeletal muscle temperature is part of the shell temperature (i.e. skin and subcutaneous tissue temperature), at least at rest in hot conditions. In the current study, skin temperature was related to the environmental thermal situation. So, the improvement in sprint performance in hot conditions resulted from an increased rate of cross-bridge cycling and high-energy phosphate turnover in the heated muscle. This suggestion is supported by the observation that there was a higher pedal cadence in hot conditions that may have been a function of a rightward shift in the power/velocity characteristics of the muscle as a consequence of an increased cross-bridge cycling rate. Similarly, Ball et al. (1999) reported that high-intensity exercise performed under conditions of thermal stress resulted in a higher pedal cadence.

Falk et al. (1998) suggested that better anaerobic performance in the heat could involve a warm-up effect. According to these authors, higher muscle temperature may improve performance by possibly increasing the speed of contraction or by increasing the rate of metabolic processes. In the current study, greater ammonia formation during sprint exercise in hot conditions was observed. These findings are in agreement with Linnane et al. (2004). In maximum sprint exercise of a short duration, all motor units should be fully recruited. Thus, the temperature related increase in cross-bridge/energy turnover should occur in all fiber types. However, in the fastest fibers where the rate is already high, any increase leads to an even more rapid depletion of high-energy phosphate and hence inosine monophosphate and ammonia production (Karatzafis et al. 2001). Furthermore, phosphocreatine is depleted and adenosine triphosphate concentration decreases within a few seconds in the fastest fibers under normal conditions in this type of exercise. Having a hot muscle, the depletion occurs earlier, thus increasing blood ammonia concentrations. However, as ammonia concentrations were not measured when volunteers were at rest and as they had been exposed to the different environments, we cannot categorically affirm that the higher ammonia values observed following exercise in the hot environments compared to normal temperature condition are related to the greater effort expended in these environments. If we take into account that the basal levels of healthy men are around 20.0±8.0 μg/dl (Luck et al. 1925) it seems plausible that the exercise was related to the higher blood ammonia levels.

Ball et al. (1999) reported that heat exposure may have induced catecholamine release. To reinforce this supposition, these authors cited Powers et al. (1982) and Brenner et al. (1997), who reported a significant rise in noradrenaline and a small but significant rise in adrenaline concentration at rest after exposure to heat compared to thermoneutral environments. It is known that increased catecholamine levels stimulate a higher rate of glycolysis and, consequently, blood lactate accumulation (Vandermalle et al. 1987). In the present study, the blood lactate concentration after exercise was higher than that found at rest. However, the increase in peak and mean power was achieved in hot environments compared to thermoneutral condition without any apparent difference in lactate concentrations. Furthermore, as previously mentioned, there were no differences in resting heart rate between the environments studied, indicating that catecholamine levels could not have been altered by the environments. Moreover, Linnane et al. (2004) observed that plasma noradrenaline and adrenaline were unaltered at rest and before exercise under hyperthermia and thermoneutral conditions.

Similar increases in blood lactate concentration in the hot condition compared to the control condition after sprint were observed in the present study. This finding is in agreement with Ball et al. (1999), who found no differences in blood lactate following repeated sprints in a hot environment compared to a control trial, a surprising finding given that subjects...
completed more work in both sprints in the heat. Several other studies have failed to show differences in blood lactate concentrations following brief maximal exercise between thermoneutral and hot environments (Backx et al. 2000; Falk et al. 1998; Linnane et Linnane et al. 2004).

Sargeant (1987) stated that the effects of temperature change on muscle power output were velocity specific. The average increase in power output was ~4% per degree Celsius rise in muscle temperature. He also reported an improvement in maximal peak power of approximately 10% per degree Celsius at the pedal cadence of 140 rev. min⁻¹. In the current study, the pedal cadence in the hotter condition was ~139 rev. min⁻¹ and the increase in maximal peak power and mean power output was 6 and 4%, respectively, in the hot trial compared to control trial.

In conclusion, the exposure to hot environment caused an improvement in power output for a single 30-s sprint. This increase in power output was associated with an elevation in plasma ammonia suggestive of an increase in adenine nucleotide loss.

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