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Changes in systolic arterial pressure variability are associated with the decreased aerobic performance of rats subjected to physical exercise in the heat

Flávia C. Müller-Ribeiro[a], Samuel P. Wanner[a], Weslley H.M. Santos[a], Milene R. Malheiros-Lima[a], Ivana A.T. Fonseca[a], Cândido C. Coimbra[b], Washington Pires[b,c,*]

[a] Exercise Physiology Laboratory, Department of Physical Education, School of Physical Education, Physiotherapy and Occupational Therapy, Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil
[b] Department of Physiology and Biophysics, Institute of Biological Sciences, Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil
[c] Department of Physical Education, Institute of Life Sciences, Universidade Federal de Juiz de Fora, Governador Valadares, MG, Brazil

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ABSTRACT

Enhanced cardiovascular strain is one of the factors that explains degraded aerobic capacity in hot environments. The cardiovascular system is regulated by the autonomic nervous system, whose activity can be indirectly evaluated by analyzing heart rate variability (HRV) and systolic arterial pressure (SAP) variability. However, no study has addressed whether HRV or SAP variability can predict aerobic performance during a single bout of exercise. Therefore, this study aimed to investigate whether there is an association between cardiovascular variability and performance in rats subjected to treadmill running at two ambient temperatures. In addition, this study investigated whether the heat-induced changes in cardiovascular variability and reductions in performance are associated with each other. Male Wistar rats were implanted with a catheter into their carotid artery for pulsatile blood pressure recordings. After recovery from surgery, the animals were subjected to incremental-speed exercise until they were fatigued under temperate (25 °C) and hot (35 °C) conditions. Impaired performance and exaggerated cardiovascular responses were observed in the hot relative to the temperate environment. Significant and negative correlations between most of the SAP variability components (standard deviation, variance, very low frequency [VLF], and low frequency [LF]) at the earlier stages of exercise and total exercise time were observed in both environmental conditions. Furthermore, the heat-induced changes in the sympathetic components of SAP variability (VLF and LF) were associated with heat-induced impairments in performance. Overall, the results indicate that SAP variability at the beginning of exercise predicts the acute performance of rats. Our findings also suggest that heat impairments in aerobic performance are associated with changes in cardiovascular autonomic control.

1. Introduction

Physical exercise can be one of the most stressful conditions with which the body has to cope. Depending on its intensity, exercise may require a metabolic rate up to 15–20 times greater than the resting value (Ainsworth et al., 2011). Therefore, coordinated physiological responses are required to supply adequate oxygen and nutrients to the working muscles. The physiological response to match the augmented metabolic demands is characterized by substrate mobilization, blood flow redistribution, and increases in mean arterial pressure (MAP), heart rate (HR), minute ventilation, and oxygen consumption (Laughlin, 1999; Pires et al., 2007, 2013; Whipp et al., 1984).

Physically active individuals and athletes often exercise in hot environments that markedly influence their exercise-induced physiological adjustments. For instance, prolonged physical exertion under environmental heat stress is associated with higher cardiovascular strain characterized by an increased demand for blood in the skin vessels and active muscles, which is counterbalanced by a reduction in the renal, splanchnic, and non-exercising muscle blood flow (Radigan and Robinson, 1949; Rowell et al., 1968). In addition, increased HR and decreased stroke volume are reported in response to exercise in the heat relative to exercise in temperate conditions (Crandall and

* Correspondence to: Laboratory of Endocrinology and Metabolism, Department of Physiology and Biophysics, Universidade Federal de Minas Gerais, Av. Antônio Carlos, 6627, Belo Horizonte, MG, Brazil.
E-mail address: washpires@gmail.com (W. Pires).

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Gonzalez-Alonso, 2010; Galloway and Maughan, 1997; Pires et al., 2013). Of note, these cardiovascular adjustments result from changes in the autonomic outflow to the heart and blood vessels that can be indirectly determined by assessing HR and blood pressure variability (Goldstein et al., 2011).

It is well established that hot environments effectively impair the physical (aerobic) performance of different animal species (Cheuvront et al., 2010; Febbraio et al., 1996; Nielsen et al., 1993; Rodrigues et al., 2003; Wanner et al., 2014). The attainment of critically high levels of core body temperature (T_{CORE}) as well as both metabolic and central nervous system (CNS)-mediated mechanisms have been proposed to be modulating factors for physical performance in heat (Cheung and McLellan, 1998; Cheung and Sleivert, 2004; Cheuvront et al., 2010; Nielsen et al., 1993; Wanner et al., 2014). Cardiovascular mechanisms also seem to play a role, as suggested by the observations that, at least in humans, high skin temperature and T_{CORE} impair aerobic performance in tandem, primarily through elevated cardiovascular strain rather than a deterioration in CNS function or skeletal muscle metabolism (Cheuvront et al., 2010; Cuddy et al., 2014).

Heart rate variability (HRV) has been considered one of the most promising and non-invasive assessment tools for monitoring individual adaptation to chronic exercise (Buchheit et al., 2007; Hautala et al., 2009; Plews et al., 2013). Decreases in vagal-derived indices of HRV are thought to be associated with negative adaptations in response to endurance training loads (Bosquet et al., 2008; Hynynen et al., 2008; Plews et al., 2013), whereas increases in vagal-derived indices of HRV have been related to improved physical performance consequent to endurance training (Atlaoui et al., 2007; Garet et al., 2004; Plews et al., 2013). Overtraining, resulting from heavy endurance training without adequate recovery, also induces changes in autonomic function, as evidenced by decreased HRV (Lehmann et al., 1993; Uusitalo et al., 1998).

HRV analysis has been extensively used as a tool to predict chronic fatigue and recovery in athletic training (Atlaoui et al., 2007; Plews et al., 2013); however, the use of this tool for predicting acute performance (e.g., total exercise time during a bout of exercise) is still unclear. Because impaired performance in the heat is associated with greater cardiovascular strain resulting from changes in the autonomic outflow to the heart and vessels, we hypothesized that the heat-induced changes in cardiovascular variability parameters would be associated with heat-induced impairment in aerobic performance. Therefore, the present study first investigated whether the cardiovascular variability parameters are associated with physical performance in temperate and hot environments. We then investigated whether the heat-induced changes in cardiovascular variability parameters are associated with reduced performance in such environmental conditions. Importantly, we evaluated the association between physical performance and the data regarding not only HRV but also systolic arterial pressure (SAP) variability. Indeed, arterial pressure is the regulated parameter in cardiovascular control (Dampney, 1994; Dampney et al., 2002) and, thus, SAP variability may be more associated with performance than HRV.

2. Materials and methods

2.1. Animals

Experiments were performed on six adult male Wistar rats weighing 280–350 g that were supplied by the Animal Resources CEBIO at the Universidade Federal de Minas Gerais (Belo Horizonte, Brazil). Animals were housed collectively and maintained in a room with a 14-h light-10-h dark cycle and ambient temperature controlled at 24 ± 1 °C. After the implantation of an arterial catheter, the rats were housed individually. The rats had free access to water and food. All experimental procedures were approved by the local Ethics Committee for Care and Use of Laboratory Animals (protocol 178/10) and carried out in accordance with the Guidelines of the Committee's Principles Manual.

2.2. Experimental design

Most of the data presented in this paper consist of a deeper analysis of cardiovascular and performance data that have been taken from a previously published manuscript (Pires et al., 2013); therefore, the present paper proposes a new analysis aimed at understanding whether there is an association between cardiovascular variability and aerobic performance during treadmill running in rats.

The rats were initially familiarized with exercising on the treadmill and then subjected to an incremental-speed exercise to evaluate their intrinsic aerobic capacity. On the following day, the animals were anesthetized with ketamine-xylazine (90 and 10.5 mg/kg body mass, respectively, i.p.). A small incision was made in the neck, and a polyethylene catheter (PE-10 connected to a PE-50; Becton Dickinson, Franklin Lakes, NJ, USA), filled with heparin diluted in isotonic saline, was inserted into the left common carotid artery. The free end of the PE-50 tubing was tunneled subcutaneously and exteriorized at the cervical dorsal area (Pires et al., 2007), and the incision in the neck was closed with small sutures. The adequacy of anesthesia was verified by the absence of a withdrawal response to nociceptive stimulation of a hind paw. Immediately after surgery, rats received an intramuscular prophylactic dose of antibiotics (pentabiotic, 48,000 IU/kg) and a subcutaneous injection of analgesic medication (flunixin meglumine, 1.1 mg/kg).

The rats were then allowed to recover in their home cages for 48 h before experiments began. The blood pressure and HR were recorded from the indwelling carotid arterial catheter connected to a blood pressure transducer (model MP 100 A-CE, Biopac Systems). All animals for which data were reported remained in good health, as assessed by appearance, behavior, and maintenance of body weight, throughout recovery from the surgical procedures and experimental trials. At the completion of the experiments, rats were euthanized with an overdose of ketamine-xylazine (240 and 30 mg/kg body mass, respectively, i.p.).

2.3. Experimental trials

Each rat was subjected to two experimental trials that consisted of incremental-speed running on a treadmill under temperate (25 °C) or hot (35 °C) conditions. A two-day interval was allowed between the trials. All experiments were performed between 0800 and 1600 h, and care was taken to test the same animal at the same time of day. The experiments in the temperate environment were always performed prior to those in the heat. A non-randomized experimental design was selected because of a concern regarding the occurrence of heat-related disorders after running at 35 °C, which would have prevented us from subjecting the rats to subsequent exercise at 25 °C. In this context, there is evidence that rats cannot restore their normal T_{CORE} circadian rhythm during the 10 days that follow severe heat exposure (Leon and Helwig, 2010). In addition, it has been demonstrated that, following a 5-d familiarization protocol, repeated exposure to physical exercise does not influence performance during the second exercise bout compared to that during the first bout (Kunstetter et al., 2014; Pires et al., 2013).

The temperature inside the treadmill chamber was set at 25 °C or 35 °C. The temperate environment was selected to be at 25 °C because previous data suggest that temperatures ranging between 24° and 26 °C correspond to the lower extremity of the thermoneutral zone of resting rats inside the chamber that contains the treadmill belt (Wanner et al., 2015a, 2015b). The hot environment was selected to be at a temperature of 35 °C because even non-exercising rats exposed to this temperature level display marked hyperthermia and activation of cutaneous heat loss (Lima et al., 2013). To heat the environment