

available at www.sciencedirect.comwww.elsevier.com/locate/brainres**BRAIN
RESEARCH****Research Report****Sinoaortic denervation prevents enhanced heat loss induced by central cholinergic stimulation during physical exercise**

Washington Pires^a, Samuel P. Wanner^b, Milene R.M. Lima^a, Bernardo M.S. Oliveira^a,
 Juliana B. Guimarães^b, Daniel C. de Lima^b, Andréa S. Haibara^b, Luiz O.C. Rodrigues^a,
 Cândido C. Coimbra^b, Nilo R.V. Lima^{a,*}

^aExercise Physiology Laboratory, Department of Physical Education, School of Physical Education, Physiotherapy and Occupational Therapy, Federal University of Minas Gerais, Belo Horizonte, Minas Gerais, Brazil

^bDepartment of Physiology and Biophysics, Institute of Biological Sciences, Federal University of Minas Gerais, Belo Horizonte, Minas Gerais, Brazil

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ABSTRACT

The present study investigated whether the effects of central cholinergic stimulation on thermoregulation during exercise are modulated by arterial baroreceptors. Wistar rats were submitted to sinoaortic denervation (SAD) or sham denervation (SHAM) and then fitted with a chronic guide cannula into the lateral cerebral ventricle. After 2 weeks, a catheter was implanted into the ascending aorta, and a temperature sensor was implanted into the peritoneal cavity. Two days later, the rats were submitted to exercise on a treadmill at 18 m/min until fatigued. Thermoregulatory and cardiovascular responses were measured after injection of 2 μ L of 10 mM physostigmine (Phy) or 0.15 M NaCl solution (Sal) into the cerebral ventricle. In SHAM rats, Phy injection induced a greater exercise-induced increase in blood pressure and lower increase in heart rate than Sal treatment. In the SAD group, the attenuation of heart rate in response to Phy was blocked despite an exaggerated increase in blood pressure. SHAM rats treated with Phy had a higher increase in tail skin temperature compared to Sal injection (31.9 ± 0.4 °C Phy-SHAM vs. 30.1 ± 0.6 °C Sal-SHAM, 5 min after injection; $p < 0.05$), resulting in a lower exercise-induced increase in core temperature. In contrast, SAD blocked the Phy injection effects in thermoregulatory responses during exercise (tail temperature: 30.1 ± 1.2 °C Phy-SAD vs. 29.5 ± 1.2 °C Sal-SAD, 5 min, $p = 0.65$). Therefore, we conclude that the enhancement of cutaneous heat loss induced by central cholinergic stimulation during exercise is mediated primarily by arterial baroreceptors.

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1. Introduction

Prolonged physical exercise performed in cool and warm environments induces an increase in core body temperature (T_{core}) because activation of heat loss has a longer time-constant than heat production (Webb, 1995). High T_{core} increases the risk

of heat stroke that is ultimately associated to endotoxemia and may lead to death (Gisolfi, 2000). Therefore, it is important to understand how heat is dissipated during exercise to protect the brain and other vital organs from thermal damage.

Animal models are employed in an attempt to better understand the central pathways and neurotransmitters

* Corresponding author. Fax: +55 31 3409 2325.
 E-mail address: nilo@ufmg.br (N.R.V. Lima).

controlling heat loss mechanisms during exercise (Hasegawa et al., 2005; Rodrigues et al., 2009; Wanner et al., 2007). In rodents, the increased blood flow in cutaneous tail vessels is the major mechanism by which these species dissipate heat produced during exercise (Gisolfi and Christman, 1980), and heat exchange between the body and environment can be estimated through the measurement of tail skin temperature (Romanovsky et al., 2002).

Central cholinergic stimulation enhances tail heat loss mechanisms in both resting and running rats (Pires et al., 2007; Primola-Gomes et al., 2007). In rats allowed to freely move, intracerebroventricular (icv) injection of the anti-cholinesterase agent physostigmine (Phy) may or may not decrease T_{core} depending upon the dose injected and ambient temperature (Fehlner and Gordon, 1985; Lin et al., 1980). However, during physical exercise, icv Phy clearly attenuates the exercise-induced increase in T_{core} , even when injected in doses that do not change T_{core} in resting rats (Pires et al., 2007; Rodrigues et al., 2004). Despite the many studies showing that cutaneous heat loss is enhanced in response to central cholinergic stimulation, the mechanism underlying this effect is not yet fully elucidated.

The thermoregulatory responses induced by central cholinergic stimulation occur in parallel to changes in the cardiovascular system (Pires et al., 2007). Icv Phy increases mean arterial pressure (or in the case of running rats, enhances the exercise-induced increase), and it is followed by a decrease in heart rate (or attenuation of the increase), indicating activation of the baroreflex. Considering that the large increase in tail skin temperature caused by icv Phy was closely correlated with the high systolic arterial pressure response (Pires et al., 2007), it is worth testing if this enhanced heat loss is also a consequence of arterial baroreceptor activation. There is evidence showing that the cutaneous sympathetic vasoconstrictor tonus is barosensitive (Johnson and Gilbey, 1998; Owens et al., 2002; Rathner et al., 2008) and that stimulation of baroreceptors modulates heat loss through the tail (O'Leary and Johnson, 1989; Zhang et al., 2003).

Taking these data into account, we hypothesized that the Phy-induced heat loss could result from activation of a non-thermoregulatory reflex. Therefore, the present study was aimed at investigating whether the arterial baroreceptors mediated the enhanced tail heat loss caused by central cholinergic stimulation.

2. Results

2.1. Effect of different doses of icv physostigmine on cardiovascular and thermoregulatory responses during exercise in intact rats

As shown in Table 1, central cholinergic stimulation with the two low doses of physostigmine 5 mM (Phy 5) and 10 mM (Phy 10) did not change exercise performance when compared to saline (Sal) treatment. However, physostigmine 40 mM (Phy 40) significantly decreased exercise performance by approximately 80% as shown by both time to fatigue and workload. When rats were injected with the high dose of Phy, they ran for only 11 min, and at the point of fatigue, presented a higher mean arterial pressure (MAP) as well as lower heart rate (HR) and tail skin temperature (T_{tail}) when compared to the other three experimental conditions (Table 1). This result may be explained by the fact that the effects of Phy last for approximately 20 min after the icv injection (Fig. 1A and B), and therefore, the drug was still acting at the point of fatigue.

Running on the treadmill increased MAP, HR, T_{tail} and T_{core} in all experimental trials and these parameters remained elevated until the point of fatigue (Fig. 1A–D). However, icv Phy 5 induced a higher increase in MAP and T_{tail} and attenuated the exercise-induced tachycardia. In addition, the increase in MAP and T_{tail} was even more pronounced during the Phy 10 when compared to both the Sal and Phy 5 trials. Phy 10 also attenuated the increases in HR and T_{core} , indicating the existence of a dose-dependent effect. Icv injection of Phy 40 did not induce any additional effect in the parameters recorded when compared to the Phy 10 trial.

2.2. Effect of sinoaortic denervation (SAD) on cardiovascular and thermoregulatory responses induced by Phy in resting or running rats

As expected, icv injection of Phy 10 into resting, sham-denervated (SHAM) animals induced an increase in MAP relative to those injected with Sal (133 ± 6 mm Hg Phy-SHAM vs. 107 ± 4 mm Hg Sal-SHAM, 4 min after injection; $p < 0.05$;

Table 1 – Effects of icv physostigmine on exercise performance as wells as on cardiovascular and thermoregulatory parameters in running rats.

Parameters	Sal (n=23)	Phy [5 mM] (n=8)	Phy [10 mM] (n=10)	Phy [40 mM] (n=5)
Time to fatigue (min)	51 ± 4	51 ± 9	47 ± 8	11 ± 2 *
Workload (kgm)	270 ± 20	258 ± 40	236 ± 34	70 ± 9 *
Mean arterial pressure (mm Hg)				
Pre-exercise	112 ± 2	106 ± 2	111 ± 3	109 ± 6
Fatigue	125 ± 3 #	120 ± 2 #	126 ± 2 #	143 ± 4 # *
Heart rate (beats·min ⁻¹)				
Pre-exercise	369 ± 7	363 ± 13	374 ± 13	386 ± 14
Fatigue	487 ± 8 #	472 ± 26 #	464 ± 21 #	437 ± 21 # *
Tail temperature (°C)				
Pre-exercise	26.8 ± 0.1	26.8 ± 0.3	26.9 ± 0.2	26.5 ± 0.6
Fatigue	32.3 ± 0.1 #	32.9 ± 0.2 #	32.3 ± 0.3 #	30.8 ± 0.1 # *
Intraperitoneal temperature (°C)				
Pre-exercise	38.0 ± 0.1	38.2 ± 0.2	37.7 ± 0.2	38.1 ± 0.2
Fatigue	38.8 ± 0.1 #	38.7 ± 0.2 #	38.2 ± 0.3 #	38.4 ± 0.1 #

* Significantly different compared to Sal [NaCl 0.15 M], Phy [5 mM], and Phy [10 mM] conditions.

Significantly different compared to corresponding pre-exercise values.

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