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The relationship of angiotensin II in energy metabolism of adipocytes: production of lactate and glycerol.

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Abstract

Angiotensin II (AII) produced by Renin-Angiotensin-Aldosterone System (RAAS) through the classical pathway have been associated with the regulation of adipocyte glycolytic and lipolytic metabolism. This pathway can be potentiated by the increase of catecholamines. The Wistar-Kyoto (WKY) and Spontaneously Hypertensive Rats (SHR) strains exhibet high catecholamines pattern and have different adipocyte profiles. This work aims to evaluate the influence of AII in the production of glycerol and lactate from these different strains.

Key words: *Angiotensin II, glycerol, lactate.*

Introduction

Lipolytic and glycolytic activity are regulated by catecholamines, which act on the availability of stored energy, which are also involved with the Renin-Angiotensin-Aldosterone System (RAAS) activation, responsible for angiotensin II (AII) production in the classic pathway (1,2). Therefore, the AII may be related to lipolysis and glycolysis modulation in adipose tissue, where the AT1 and AT2 receptors blockade affect the adipocyte metabolism (3). The Spontaneously Hypertensive Rats (SHR), as its control Wistar-Kyoto (WKY), exhibit high catecholamines pattern and different weights and adipocytes profiles, which WKY is heavier, with higher area and diameter of their adipocytes (4). This work aims to evaluate the influence of AII in the production of glycerol and lactate from these different strains.

Results and Discussion

Ethical Committee approved the protocol under number 4073-1. Male 15-week-old Wistar (WIS), WKY and SHR rats (n=5-12) were used for the experiments. The normality was confirmed by Shapiro-Wilk test and then we performed Student's t-test for parametric and Mann-Whitney for nonparametric data. Our results were standardized by area due to the fact that the size influences the adipocyte metabolism. There is no difference between strains in the basal glycerol production (Figure 1A). AII did not increase significantly the glycerol production above basal levels (Figure 1B vs Figure 1A). However, SHR exhibited higher glycerol levels in presence of AII (Figure 1B) probably due to a decrease in glycerol production in WKY isolated adipocytes under the same stimulus (10^{-7}M) (Figure 1B).

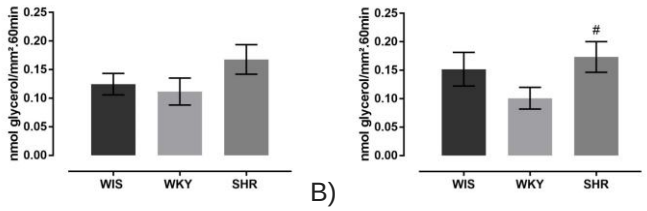

Figure 1. A) Basal production of glycerol from WIS, WKY and SHR; B) Basal production of glycerol incubated with AII (10^{-7}M) from WIS, WKY and SHR. p<0.05. n= 11-12

SHR exhibited higher basal lactate than the other strains (Figure 2A) and AII (10^{-7}M) does not affect the glycolytic activity above the basal levels (Figure 2B)

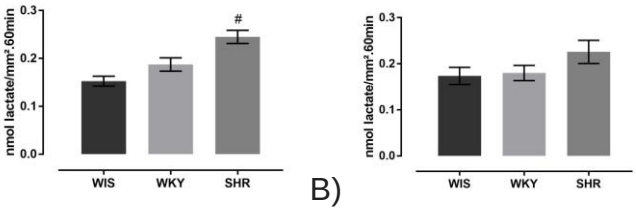

Figure 2. A) Basal production of lactate from WIS, WKY and SHR; B) Basal production of lactate incubated with AII (10^{-7}M) from WIS, WKY and SHR. p<0.05. n= 5-6

Conclusions

The lipolytic activity does not exhibit any difference in the basal production; however, AII seems to have anti-lipolytic in WKY isolated adipocytes. The glycolytic metabolism of the adipocyte is not affected by AII. However, SHR presents high basal lactate in relation to the other strains, which may be due to increased LDH activity and higher α_2 renal receptors density, which is associated with increased lactate efflux (5-7).

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