ABSTRACT

ROLE OF THE RENIN-ANGIOTENSIN SYSTEM IN RESISTANCE EXERCISE-INDUCED CARDIAC HYPERTROPHY

Author: VALÉRIO GARRONE BARAUNA

Adviser: PROFa. DRa. EDILAMAR MENEZES DE OLIVEIRA

Besides the well-known effects of Ang II in stimulating pathological pressure-overload cardiac hypertrophy, little information is available regarding the role of Renin-Angiotensin-System (RAS) in the exercise training-induced cardiac hypertrophy. 64 male Wistar rats were divided into 6 groups: Sedentary, Trained, Sedentary or Trained + Losartan (20mg/Kg/d, n=7) and Sedentary or Trained + Salt (NaCl 1%). The exercise protocol was: 4 x 12 bouts, 5x/week during eight weeks, with 65-75% of 1 Repetition Maximum (1RM). Using LV weight/body weight ratio and echocardiography (ECHO) we have observed cardiac hypertrophy in the Trained group without any impairment in ventricular function. Concerning RAS, neither ACE, analyzed by fluorometric assay (systemic and local in the heart), nor Renin, by RIA, activities were altered after resistance training. In addition, using Western blotting analysis, no change was observed in cardiac Ang II and AT2 receptor levels while the AT1 receptor expression was upregulated in Trained groups by 31,4%. Administration of the AT1 receptor antagonist (losartan) prevented left ventricle hypertrophy in response to the resistance training. The administration of salt, to inhibit the renin activity, did not prevent the cardiac hypertrophy. These results suggest that the AT1 receptor participates in resistance-training-induced cardiac hypertrophy without an increase in Ang II concentration in the heart. A possible mechanism is the direct activation of the AT1 receptor by mechanical stretching of cardiomyocytes.

Keywords: Resistance-training; Renin-Angiotensin System; Cardiac Hypertrophy.