PROTEIN RESTRICTION DURING PERI-PUBERTAL PHASE PROGRAMS TO THE METABOLIC AND RENIN-ANGIOTENSIN DYSFUNCTION OF MALE ADULT RATS

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Introduction: Protein restriction (LP) in perinatal life induces to hypertension related to renin-angiotensin system and metabolic dysfunction in adulthood, however, the implication of renin angiotensin system and metabolism in the hypertension induced by peri-pubertal protein restriction is unknown. **Objective:** This study aims to evaluate whether protein restriction in peri-pubertal phase induces hypertension related to renin-angiotensin system and metabolic dysfunction. **Method:** After approval by the ethic committee (nº3353060421), Wistar rats at post-natal days (PN) 30 were fed with a low-protein diet (4%) for 30 days and then fed a 20.5% protein diet for 60 days (LP), for dietary recovery. Control animals (NP) consumed a diet with 20.5% protein throughout the protocol. At PN 120 cardiovascular parameters were evaluated. The T-Student test was used for statistical analysis. **Results:** At PN 120 LP animals showed had a lower body weight and nasal-anal length (P=0.0001; P=0.0103) with an increase in retroperitoneal fat (P=0.0289). The LP animals showed higher levels of glucose (P=0.0457) and triglycerides (P=0.0444) with an increase in triglyceride glucose index (TYG) (P=0.0045), however, with no change in total cholesterol and HDL. LP rats showed long-term increased mean arterial pressure (p=0.047) but heart rate remained unchanged. LP rats showed attenuated depressor response to the ACE inhibitor (enalapril) compared with control animals (p = 0.015). In the angiotensin 2 dose response curve, The LP animals showed an increased pressor response to angiotensin 2 at the low dose (50 ng/Kg) (p=0.007), and a reduction in the pressor response at the intermediary dose (200 ng/Kg) (P=0.0219) and high dose (400 ng/Kg) (p=0.015). **Conclusion:** Protein restriction in peri-pubertal phase leads to

hypertension in adulthood, supported by both metabolic and renin-angiotensin system impairment.

Keywords: Metabolism; Angiotensin 2; Hypertension.

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