OVERNOURISHMENT DURING LACTATION INDUCES METABOLIC AND HAEMODYNAMIC HEART IMPAIRMENT DURING ADULTHOOD


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Introduction and Objectives: Experimental studies demonstrated that adult mice overfed during lactation presents obesity associated with development of cardiovascular disease and energy metabolism impairment. Analyze hemodynamic, energy metabolism and cardiac remodeling in the heart of obese adult Swiss mice at baseline (CGBL, OGBL) and after ischemia/reperfusion (CGIR, OGIR).

Materials and Methods: Obesity was induced by litter size reduction method. Hearts from Control group (CG) and overfed group (OG) hemodynamics was studied by Langendorff technique analyzing EDP, Tau, Max dp/dt, Min dp/dt and heart rate; Western blotting analysis of IRβ protein, AKT, AMPK, PTP1B, IRS1, FABP, CD36; Real Time PCR for gene expression of ANP, BNP, CPT1 and UCP3; collagen quantification by histology. Insulin and leptin plasma levels were measured by ELISA.

Results and Conclusions: OG showed increased body weight, visceral fat, leptin and insulin when compared to CG. In hemodynamic studies we observed changes in OGBL with reduced Min dp/dT and in OGIR increased EDP and Max dp/dT compared to the respective OGIR. Increased OGIR IRS1, reduced pAKT1, AMPK, FABP and CD36 compared to CGIR. OGBL showed increased IRβ and reduced pAKT1 compared to CGBL, and increased pAMPK/AMPK, reduced PTP1B, IRS1 compared to OGIR. ANP gene expression was increased in OGIR compared to CGIR, BNP increased in OGBL compared to CGBL, increased CPT1 in OGBL compared to CGBL and OGIR and increased UCP3 in CGIR compared to CGBL and OGIR. Heart collagen was increased in OGIR compared to CGIR and OGBL, and reduced in CGBL compared to CGIR and OGBL. Overnutrition during lactation induce obesity in adulthood, increases leptin and insulin and impairs heart insulin signaling and fatty acids metabolism, which favors the development of hypertrophy and cardiac remodeling, impairment in heart hemodynamic and energy metabolism.

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Key Words: Heart; Postnatal Overfeeding; Ischemia/Reperfusion.