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P39. Effects of soybean isoflavones in rats model with polycystic ovary syndrome (pcos)

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Introduction: Soy isoflavone (ISF) has been shown to interact with peroxisome proliferator-activated receptor ? (PPAR?) which is a key molecule involved in the metabolism of insulin resistance and free fatty acids (FFA). Objective: We have two dependent hypotheses. The first is related to the metabolic syndrome in PCOS and the second is related to how the metabolic syndrome could address the pathophysiology of the ovaries in PCOS. To understand if in PCOS the activation of PPAR is affected leading to the deregulation of lipid metabolism and if isoflavone can restore lipid homeostasis through the re-activation of PPAR? in the liver. Method: We used 30 female rats that after birth from 1 to 5 days were administered subcutaneously 0.1mg / kg of testosterone propionate. After induction the rats were divided into three groups: GI: sham group, GII: rats treated with propylene glycol vehicle and GIII: rats treated with soy isoflavones 150 mg / kg; rats were monitored daily for two consecutive months. At the end of the experiment, the rats were anesthetized and euthanized using a deepening of the anesthetic plane. The ovaries and liver were collected and processed for analysis of morphology and molecular biology. The ANOVA test was used, followed by the Bonferroni test. Results: Showed that control rats increased body weight ($p < 0.01$) compared to isoflavones ($P < 0.01$). Interestingly, no differences were found in relation to food intake in any of the groups, which demonstrated that the reduction in body weight of the GIII group is due to the effects of FSI and not due to food consumption. Along with an increased body weight of the control rats there was a significantly decrease insulin sensitivity ($p < 0.001$) compared to ISF-treated, which were able to increase insulin sensitivity significantly ($P < 0.01$) A morphological analysis of the ovaries showed ISF did not recover the regularity of the estrous cycle $p < 0.01$). The analysis of lipids in the liver through the fat droplet quantification technique, showed that in the control that testosterone propionate induced lipid accumulation in the liver; ($P < 0.01$) and FSI can improve it ($p < 0.01$). Conclusion: Preliminary results indicate that isoflavones interact in the metabolic syndrome induced by testosterone propionate and these observations are important because it leads us to understand (biochemical analysis) and whether in the liver PPARy is inactivated in the mouse model PCOS and if ISFs can reactivate PPARYs.

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