



## ADIPOSE TISSUE MORPHOLOGY AND METABOLIC VARIATIONS ON PPAR $\gamma$ LYS CRE OBESE MICE SUBMITTED TO MODERATE TRAINING

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Obesity is a public health problem presenting high prevalence worldwide. Exercise is considered a non-pharmacological therapy not only for maintenance of body weight excess but chronic inflammatory diseases such as diabetes type 2. Peroxisome proliferator-activated receptor gamma (PPAR- $\gamma$ ) agonists are widely used as anti diabetic drugs, additionally, this transcription factor is highly expressed in macrophages, immunological cells whose functions are related to metabolic variations, especially in adipose tissue. Starting from the interface from exercise on metabolic homeostasis and macrophage interaction on metabolism, we aimed to assess the effects of PPAR- $\gamma$  deletion in macrophages in the metabolic response of the exercise in a diet-induced state of obesity. Two animal strains were used: CreLox for PPAR- $\gamma$  (KO) in myeloid cells and litter control animals (WT). Each genotype was divided into 3 subgroups 1) chow diet sedentary; 2) high fat diet sedentary (HF) 3) high fat diet and moderate intensity training (HFT). The experimental protocol lasted 12 weeks (4w diet + 8w diet and training). Were evaluated body weight and adiposity index (sum of fat pads), fasting glucose and insulin, glucose (GTT) and insulin (ITT) tolerance tests, HOMA-IR, area under curve (AUC) in GTT and decay constant in ITT (kITT). Histological sections stained with eosin and hematoxylin from subcutaneous adipose tissue (SAT) were obtained. Body weight was higher in HF groups but restored by exercise. Adiposity index significantly increase only in KO HF despite of the adipocyte area in SAT had increased in both HF groups. Glucose tolerance indicated by AUC was observed in both HF genotypes independently of exercise although insulin resistance detected by ITT test was observed only in WT HF thus reversed by exercise. Fasting insulin remained elevated in KO HFT even though HOMA-IR of this group was reduced possibly due to its low fasting glucose. Exercise reduced adipocyte area only in KO mice. Exercise seems to be an important mechanism of insulin sensibility independent of PPARg in macrophages, furthermore mechanisms of insulin pathways on adipose tissue of PPARg Lys Cre mice need to be considered. On the other hand adipose tissue morphology was affected by the lack of PPARg.



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