

EARLY PPAR α ACTIVATION PROTECT AGAINST HEPATIC MICROSTEATOSIS INDUCED BY POST-NATAL OVERFEEDING

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Post-natal overfeeding leads to development of obesity and metabolic dysfunction. PPAR α activation improves steatosis and metabolic dysfunction associated with obesity. Thus, we have hypothesized that PPAR α would prevent the metabolic disorders and obesity caused by litter reduction. Wistar male offspring rats were divided into normal litter (NL - 9 pups) and small litter (SL - 3 pups) and were daily treated with an PPAR α agonist, fenofibrate, from post-natal day (PN) 1 until weaning (PN21), with either, fenofibrate (F - 12.5mg/kg) or vehicle (V). Forming the following groups: NL-V, NL-F, SL-V, SL-F. At PN120 animals were euthanized and histological evaluation of samples from liver, visceral (VAT), subcutaneous (SAT) and brown (BAT) adipose tissue were performed. Fenofibrate was able to prevent the development of microsteatosis in the liver caused by postnatal overfeeding. In regard to VAT, there was no difference in the mean adipocyte area, however in distribution analyzes it was observed higher frequency of larger adipocytes in SL groups, and no difference was observed in SAT. SL-V animals had increased lipid area in BAT, which was reduced in SL-F. Increased PPAR α activation induced by an agonist during lactation improved some histopathological abnormalities induced by postnatal overfeeding. This was proven by the decrease in microsteatosis in the liver and improvement in VAT and BAT morphology, thus implying a better metabolic health.

Keywords: PPAR α , Small Litter, Fenofibrate.

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