FENOFIBRATE UPREGULATES TARGET ENZYMES OF FATTY ACID OXIDATION WITHIN WHITE ADIPOSE TISSUE AND LIVER.

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It is well established that fenofibrate (stimulant of peroxisome proliferator-activated receptor a – PPARa) administration decreases plasma lipids and adiposity. The experiments reported here were designed to study the effect of fenofibrate on the target enzymes of liver and white adipose tissue (WAT). Wistar male rats fed a balanced diet (C) or a balanced diet plus 100 $mg.Kg^{-1}bw.day^{-1}$ fenofibrate (C+F), during 9 days. After decapitation, the tissues were removed, total mRNA extracted and gene expression of PPARa, acyl-CoA oxidase (ACO) and carnitine palmitoyl transferase (CPT1) was evaluated by RT-PCR. The liver mRNA expression of PPARa, ACO and CPT1 were 200%, 180%, and 50% increased in the treated group when compared to control group, respectively. In relation to WAT, the fenofibrate increased the expression of PPARa, ACO and CPT1 by 80%, 300% and 250%, respectively. Although both, the peroxisomal and the mitochondrial compartments contribute to increase oxidation of fatty acids, our data provide evidence that the hipolipidemic effect of fenofibrate is mediated, mainly, by hepatic peroxisomal oxidation of fatty acids. Besides its effects on liver, fenofibrate seems to play a relevant role on the metabolism of adipose tissue, which may contribute to decrease adiposity, probably as a result of the local increased fatty acid oxidation.

Key words: fatty acid oxidation, fenofibrate, PPARa, ACO, CPT1.

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