



# Propionyl-L-carnitine corrects metabolic and cardiovascular alterations in diet-induced obese mice and improves liver respiratory chain activity

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**AIMS:** Obesity is a primary contributor to acquired insulin resistance leading to the development of type 2 diabetes and cardiovascular alterations. The carnitine derivate, propionyl-L-carnitine (PLC), plays a key role in energy control. Our aim was to evaluate metabolic and cardiovascular effects of PLC in diet-induced obese mice.

**METHODS:** C57BL/6 mice were fed a high-fat diet for 9 weeks and then divided into two groups, receiving either free- (vehicle-HF) or PLC-supplemented water (200 mg/kg/day) during 4 additional weeks. Standard diet-fed animals were used as lean controls (vehicle-ST). Body weight and food intake were monitored. Glucose and insulin tolerance tests were assessed, as well as the HOMA(IR), the serum lipid profile, the hepatic and muscular mitochondrial activity and the tissue nitric oxide (NO) liberation. Systolic blood pressure, cardiac and endothelial functions were also evaluated.

**RESULTS:** Vehicle-HF displayed a greater increase of body weight compared to vehicle-ST that was completely reversed by PLC treatment without affecting food intake. PLC improved the insulin-resistant state and reversed the increased total cholesterol but not the increase in free fatty acid, triglyceride and HDL/LDL ratio induced by high-fat diet. Vehicle-HF exhibited a reduced cardiac output/body weight ratio, endothelial dysfunction and tissue decrease of NO production, all of them being improved by PLC treatment. Finally, the decrease of hepatic mitochondrial activity by high-fat diet was reversed by PLC.

**CONCLUSIONS:** Oral administration of PLC improves the insulin-resistant state developed by obese animals and decreases the cardiovascular risk associated to this metabolic alteration probably via correction of mitochondrial function.

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