

# Thyroid hormone preconditioning: Protection against ischemia-reperfusion liver injury in the rat

Fernández, Virginia

Castillo, Iván

Tapia, Gladys

Romanque, Pamela

Uribe-Echevarría, Sebastián

Uribe, Mario

Cartier-Ugarte, Denise

Santander, Gonzalo

Vial, María T.

Videla, Luis A.

Recently, we reported that oxidative stress due to 3,3',5- triiodothyronine (T3)-induced calorogenesis up-regulates the hepatic expression of mediators promoting cell protection. In this study, T3 administration in rats (single dose of 0.1 mg/kg intraperitoneally) induced significant depletion of reduced liver glutathione (GSH), with higher protein oxidation, O<sub>2</sub> consumption, and Kupffer cell function (carbon phagocytosis and carbon-induced O<sub>2</sub> up-take). These changes occurred within a period of 36 hours of T3 treatment in animals showing normal liver histology and lack of alteration in serum AST and ALT levels. Partial hepatic ischemia-reperfusion (IR) (1 h of ischemia via vascular clamping and 20 h reperfusion) led to 11-fold and 42-fold increases in serum AST and ALT levels, respectively, and significant changes in liver histology, with a 36% decrease in liver GSH content and a 133% increase in that of protein carbonyls. T3 administration in a time window of 48 hours was substantial