Potentiation of ischemia-reperfusion liver injury by hyperthyroidism in the rat

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Parameters related to hepatic oxidative stress, cell injury, and liver histology were determined in control rats and in animals treated with 3,3',5- triiodothyronine (T3), after in vitro perfusion under normoxic or ischemia- reperfusion conditions. Thyroid calorigenesis was found concomitantly with higher rates of hepatic O2 consumption and thiobarbituric acid reactive substances (TBARS) formation, glutathione (GSH) depletion, enhanced TBARS/GSH ratio as indicator of oxidative stress, and higher sinusoidal lactate dehydrogenase (LDH) efflux compared to control values, assessed under normoxic conditions. Perfused livers from control animals subjected to ischemia-reperfusion exhibited significant increases in the TBARS/GSH ratio and in the sinusoidal LDH efflux over values obtained under normoxic conditions, concomitantly with the appearance of small foci of necrotic cells in centrilobular and midzonal areas of the liver lobule. These parameters were further modified in the liver of hype