# Increases in tumor necrosis factor-? in response to thyroid hormone-induced liver oxidative stress in the rat

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Thyroid hormone-induced calorigenesis contributes to liver oxidative stress and promotes an increased respiratory burst activity in Kupffer cells, which could conceivably increase the expression of redox-sensitive genes, including those coding for cytokines. Our aim was to test the hypothesis that L-3,3?,5-triiodothyronine (T3)-induced liver oxidative stress would markedly increase the production of TNF-? by Kupffer cells and its release into the circulation. Sprague-Dawley rats received a single dose of 0.1 mg T3/kg or vehicle (controls) and determinations of liver O2 consumption, serum TNF-?, rectal temperature, and serum T3 levels, were carried out at different times after treatment. Hepatic content of total reduced glutathione (GSH) and biliary glutathione disulfide (GSSG) efflux were measured as indices of oxidative stress. In some studies, prior to T3 injection animals were administered either (i) the Kupffer cell inactivator gadolinium chloride (GdCl3), (ii) the antioxidants ?-t