

Influence of hyperthyroidism on lindane-induced hepatotoxicity in the rat

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Parameters related to hepatic oxidative stress, cell injury, phagocytic activity, and liver histology were studied in control rats and in animals subjected to l-3,3',5-triiodothyronine (T3) and/or lindane administration. Hyperthyroidism elicited a calorogenic response and increased rates of hepatic O₂ uptake, which were not modified by lindane treatment. T3 diminished serum lindane levels as well as those in the liver and adipose tissue, whereas lindane enhanced serum T3 levels in animals given T3. Compared with control rats, lindane significantly increased the rate of formation of thiobarbituric acid reactants (TBARS) by the liver, with no changes in either the reduced glutathione (GSH) content, the TBARS/GSH ratio as indicator of oxidative stress, or in the fractional rates of lactate dehydrogenase (LDH) and GSH efflux from perfused livers as integrity parameters. Hyperthyroidism induced GSH depletion in the liver, with a significant enhancement in the TBARS formation, the TBARS/GSH