Liver and biliary levels of glutathione and thiobarbituric acid reactants after acute lindane intoxication

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The i.p. administration of 60 mg kg?1 body weight of lindane, the ??isomer of hexachlorocyclohexane, to fed rats led to an enhancement of hepatic lipid peroxidation after 24 h of treatment. This was evidenced by significant increases in the hepatic production and biliary release of thiobarbituric acid reactive substances, and in the biliary release of glutathione disulphide. Under these conditions, the content of cytochrome P450 was enhanced concomitantly with increases in the total microsomal oxygen uptake, superoxide radical generation and (+)?catechin (cyanid?3?ol) sensitive respiration. The glutathione status of hepatocytes was altered by lindane as the content and biliary release of glutathione disulphide was drastically augmented, leading to a decrease in the cellular and biliary GSH/GSSG ratios. It is suggested that lindane treatment leads to an induced oxidative capacity, which, in turn, alters the glutathione status of the liver tissue.