

The formation of oxidative stress condition in the experimental chemically induced hepatotoxicity.

[Rocz Akad Med Bialymst.](#) 2002;47:86-94.

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In spite of extensive studies concerning the chemically induced hepatotoxicity, no clear-cut practical conclusions were obtained. Wide chemical structure of compounds as well as the different way of administration are the most important factors. We used an acute administration of carbon tetrachloride and paracetamol and a chronic intoxication with acetylphenylhydrazine, cadmium chloride and ethanol. For the acute administration of chemicals in high doses, in the rat liver microsomes a decrease of cytochrome P450, aniline hydroxylase and glutathione (GSH) was noticed with an increase of lipid peroxides even at 2 hours after administration. 24 hours after the chemicals administration, in the blood of rats characteristic changes of hepatotoxicity such as increases glutamate-pyruvate transaminase, triglycerides and lipid peroxides, while GSH was decreased. In chronic administration, the cumulated concentration of chemical was true main factor. For both ways of chemicals administration, as a critical concentration was reached, an oxidative stress was produced, demonstrated by the increase of lipid peroxides and subsequently decrease of GSH. In both ways of administration, the oxidative stress was the precocious biochemical event occurring even at 2 hours and it triggers other metabolic changes which favor fatty infiltration and liver damage.

