
Chloroacetonitrile induces oxidative stress and apoptosis in mouse fetal liver.

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Chloroacetonitrile (CAN) is a disinfection by-product of chlorination of drinking water. The present study was designed to investigate the potential adverse effects of maternal exposure to CAN on fetal liver in mice. Based on an initial dose-response experiment, CAN (25mg/(kg/day)) was given orally to pregnant mice at gestation day (GD) 6, till GD 18. Fetuses were collected and fetal livers were used for assessing oxidative status, apoptosis and histopathological changes. Maternal exposure to CAN resulted in observed oxidative stress and redox imbalance in fetal liver tissues as marked by significant decrease in reduced glutathione (GSH) and elevation of oxidized glutathione (GSSG), malondialdehyde (MDA) and 8-hydroxy-2'-deoxyguanosine (8-OHdG) in genomic DNA. Further, CAN induced apoptosis as indicated by a significant increase in binding of Hoechst reagent to damaged DNA fragments of fetal liver and enhancement of the activity of caspase-3 in cytosolic fractions of fetal livers. Histopathological examination of fetal livers of CAN-treated mice showed hepatocytes with vacuolated cytoplasm, karyolysis and karyorrhexis as well as depletion of their glycogen content. In conclusion, maternal exposure to CAN adversely affects mouse fetal livers as evidenced by the induction of oxidative stress, apoptosis and histopathological changes.

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