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Hepatotoxicity of formaldehyde in rats: Cellular and molecular mechanisms

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The present study was addressed to gain better understanding of the intimate mechanisms of FA hepatotoxicity. Adult female *Wistar rats* were distributed into: (1) control, (2) FA; 10 ppm for 15 days, (3) FA; 10 ppm for 30 days, (4): FA; 20 ppm for 15 days and (5): FA; 20 ppm for 30 days. Histopathological description, immunohistochemical examination and molecular genetics analyses were carried out. Liver histopathological findings of liver tissue sections revealed sinusoidal congestion, liver necrosis and fibrosis due to FA exposure. Liver tissue sections immunostained with the antibody for PCNA or cytochrome c showed strong positive immunoreactivity within numerous nuclei and the cytoplasm of numerous cells respectively. Exposure of rat to FA for 15 days evoked downregulation in liver Cyp2c6 and BHMT genes expression level. Exposure of rat to FA (20 ppm) for 15 or 30 days experienced downregulation in liver Mapk12 gene expression level. Liver HLA-A and GSTP1 genes expression of rats exposed to FA for 15 and 30 days showed upregulation except for 10 ppm of FA at 15 days. This study provides cellular and molecular evidences for FA-induced hepatotoxicity. Formaldehyde is reasonable to assume that apoptosis, oxidative stress and inflammation be involved in formaldehyde-induced hepatotoxicity.