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The effects of 4-nonylphenol administration induced oxidative damages in liver and contributes to hepatic steatosis in male rats

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An emerging literature suggests that early life exposure to 4-nonylphenol (4-NP), a widespread endocrine disrupting chemical, may increase the risk of metabolic syndrome. In this study, we investigated the hypothesis that intraperitoneal administration of 4-NP induce hepatic steatosis in rat. 24 males Sprague-Dawley rats were administered with 4-NP (0, 2, 10 and 50 mg/kg b.wt) in corn oil during 30 days. Liver histology, biochemical analysis and gene expression profiling were examined. After treatment, abnormal liver morphology and function were observed in the 4-NP-treated rat, and significant changes in gene expression as indicator of hepatic steatosis and apoptosis were observed compared with controls. Up-regulated genes involved in apoptosis, Hepatotoxity and OS, increased ROS and decrease of antioxidant enzyme were observed in the 4-NP exposed rat. Extensive fatty accumulation in liver section and elevated serum SGOT, SGPT, LDH and γ-GT were also observed. Incidence and severity of liver steatosis was scored and taken in to consideration (steatosis, ballooning and lobular inflammation). Hepatocyte apoptosis could promote NAFLD progression; Fas/FasL, TNF-α and Caspase-9 mRNA activation were important contributing factors to hepatic steatosis. These findings provide the first evidence that 4-NP affects the gene expression related to liver hepatotoxicity, which is correlated with hepatic steatosis.

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CCL18/PITPNM3 enhances migration, invasion, and EMT through the NF-κB signaling pathway in hepatocellular carcinoma

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Chemokine ligand 18 (CCL18) has been associated with hepatocellular carcinoma (HCC) metastasis. Here, we demonstrated a novel mechanism through which CCL18 enhances cell migration, invasion, and epithelial–mesenchymal transition (EMT) in HCC. (1) Using immunohistochemistry, we analyzed the expression of PITPNM3, a molecule that correlated with CCL18 signaling, in 149 HCC tissue specimens. The results showed that PITPNM3 expression is highly associated with tumor metastasis and differentiation; (2)in vitro experiments showed that CCL18 enhances cell migration, invasion, and EMT in PITPNM3 (+) HCC cells but not in PITPNM3(-) cells. Silencing of PITPNM3 by short interfering RNA (siRNA) inhibited the induction of cell migration, invasion, and EMT by CCL18; (3) Cell migration, invasion, and EMT induced by CCL18 accompanied with the phosphorylation of IKK and IKBα as well as p65 nuclear translocation in PITPNM3(+) HCC cells, but not in the cells that PITPNM3 is silenced with siRNA, implying that the activation of NF-κB signaling is involved in the action of CCL18/PITPNM3. These results suggest that CCL18 enhances HCC cell migration, invasion, and EMT through the expression of PITPNM3 and the activation of the NF-κB signaling pathway.

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