

P-7 Effects of Nitric Oxide on Hepatotoxicity Induced by Acetaminophen

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Nitric oxide(NO) is a readily diffusible, short-lived free radical with a multitude of organ-specific regulatory functions. However, while NO can regulate a number of hepatocyte functions, it is unknown whether nitric oxide production in liver is related to hepatotoxicity. Using isolated SD rat hepatocytes in primary culture, we investigated the effect of endogenously (endotoxin and cytokines) and exogenously(NO donor, S-nitroso-N-acetyl-DL-penicillamine) synthesized nitric oxide on hepatotoxicants-induced hepatocellular injury. To observe whether nitric oxide production in liver is related to hepatotoxicity *in vivo*, we also observed the indicative constituents(ALT, AST), the level of NO in serum of ICR mice administered acetaminophen(340mg/kg, P.O), lipopolysaccharide (NOS inducer ; 120 μ g/mouse, I.P) and N^G-monomethyl-L-arginine(NOS inhibitor ; 5mg/mouse, I.V). We observed the serum level of indicative constituents(ALT, AST etc) *in vivo* experiment, and LDH leakage and MTT assay *in vitro* experiment to estimate the degree of hepatotoxicity. In our *in vitro* and *in vivo* experiment we suggested that inhibition of endogenously and exogenously synthesized nitric oxide potentiated hepatotoxicity induced by hepatotoxicants in low level of NO. We suggested that NO has hepatoprotective effect in hepatotoxicity induced by acetaminophen.

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