Results TAA increased, within 6 h, hepatic tumour necrosis factor- α production, interleukin 1 β , nitrite levels, inducible nitric oxide synthase expression and myeloperoxidase activity. CYP 2E1 inhibitors showed significant inhibition of tumour necrosis factor α , interleukin 1 β , nitrite, and myeloperoxidase activity after TAA treatment. In addition, acetamide, but not TAA-S-oxide, increased myeloperoxidase activity and all tested proinflammatory mediators generation.

Conclusion The authors conclude that acetamide-associated neutrophil activation is involved, at least partially, in TAA-induced hepatic inflammation. Further, exposure to acetamide-based compounds may be a risk factor of acute hepatic inflammation.

Competing interests None.

Keywords acetamide, hepatic inflammation, inflammatory cytokine, neutrophil infiltration, nitric oxide, thioacetamide.

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EXPOSURE TO ACETAMIDE-BASED COMPOUNDS IS A RISK FACTOR OF ACUTE HEPATIC INFLAMMATION

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Introduction Acetamide-based compounds widely used in industries and their toxic effect have not been studied. Thioacetamide (TAA) is widely used in industry and is known to be a hepatotoxicants in experimental animals. However, the mechanism underlying TAA-induced acute inflammation is still unclear. The authors investigated the mechanisms and the involvement of main TAA metabolites in acute hepatic inflammation induced by TAA-in rats.

Methods Acute hepatic inflammation was induced by TAA (0, 10, 30 and 100 mg/kg, intraperitoneally), while the inflammatory indicators including cytokines and nitric oxide were determined 0, 1, 3, 6 and 12 h after TAA administration. Hepatic pro-inflammatory cytokines were measured quantitatively using ELISA. SKF525A (cytochrome P450 2E1 (CYP 2E1) inhibitor) were used to examine the role of cytochrome in TAA-induced acute hepatic inflammation. In addition, TAA-S-oxide and acetamide were also used to examine the involvement of TAA metabolites in the early stage of TAA-induced hepatic inflammation.