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Multifaceted potential of Thioacetamide as a Hepatotoxicant model for Screening of Hepatoprotective agents

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ABSTRACT

The paper, textile, lab, and leather industries all once employed the organosulfur chemical thioacetamide (C_2H_5NS) because of its excellent fungicide property but soon its use was banned as it was reported to cause hepatotoxicity. Thioacetamide has been listed as a model hepatotoxicant to cause acute and chronic liver damage because of its effects on DNA, RNA, and protein synthesis. Thioacetamide is bioactivated twice, first to the reactive metabolite sulfine and then to sulfene. Sulfine causes the nuclear volume and Ca^{++} concentration to rise, the nucleoli to grow, the permeability of cells to change, and the inhibition of mitochondrial activity. Nitric oxide synthase and NF-κB are released by sulfene at the same time, which causes centrilobular necrosis, protein denaturation, and lipid peroxidation. When this substance is used orally over an extended period of time, it can induce liver nodules, liver cell adenomas, cholangiomas, and hepatocarcinomas that are histologically identical to those brought on by viral hepatitis, hepatic fibrosis, hepatic cirrhosis, and ultimately hepatocellular carcinomas. Thus, this chapter presents an overview on multifaceted potential of thioacetamide as a hepatotoxicant model for screening of hepatoprotective agents.

INTRODUCTION

Thioacetamide was first developed by Childs and Siegler in 1945 to stop deterioration of oranges before developing it into a fungicide. Thioacetamide with chemical formula C_2H_5NS and molecular weight