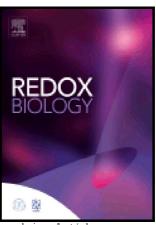
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Mechanisms of acetaminophen-induced liver injury and its implications for therapeutic interventions

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Abstract

Acetaminophen (APAP) overdose is the leading cause of drug-induced acute liver failure in many developed countries.

Mitochondrial oxidative stress is considered to be the predominant cellular event in APAP-induced liver injury.

Accordingly, N-acetyl cysteine, a known scavenger of reactive oxygen species (ROS), is recommended as an effective

clinical antidote against APAP-induced acute liver injury (AILI) when it is given at an early phase; however, the narrow

therapeutic window limits its use. Hence, the development of novel therapeutic approaches that can offer broadly

protective effects against AILI is clearly needed. To this end, it is necessary to better understand the mechanisms of APAP

hepatotoxicity. Up to now, in addition to mitochondrial oxidative stress, many other cellular processes, including phase

1

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