

Protective effects of herbal extracts against drug-induced toxicity: a review of pharmacological evidence and mechanisms

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Received: 23 April 2026; Revised: 10 June 2026; Accepted: 25 June 2026; Published: 26 June 2026

Drug-induced toxicity is a major challenge in pharmacotherapy and is considered one of the leading causes of treatment interruption, hospitalization, and progressive organ dysfunction worldwide. Several commonly prescribed drugs (including acetaminophen, cisplatin, methotrexate, and doxorubicin) are well-known to exert toxic effects on organs such as the liver, the kidneys, and the heart, and although such drugs might differ in their pharmacological actions and therapeutic applications, they can frequently induce tissue injury through overlapping molecular mechanisms. In fact, oxidative stress, mitochondrial dysfunction, the activation of inflammatory signalling, and apoptosis are considered amongst the most important pathogenic pathways involved in drug-induced toxicity. Excessive generation of reactive oxygen species disrupts intracellular redox balance and promotes lipid peroxidation, protein oxidation, DNA damage, and impairment of normal cellular metabolism. Additionally, mitochondrial injury contributes to ATP depletion and the activation of apoptotic pathways, while the inflammatory mediators further amplify tissue injury and accelerate organ dysfunction. The acetaminophen-induced hepatotoxicity is primarily linked to the formation of a highly reactive metabolite that depletes intracellular glutathione stores and induces severe oxidative stress in the hepatocytes. Similarly, the cisplatin-induced nephrotoxicity involves an

excessive production of reactive oxygen species, inflammatory cytokine release, mitochondrial injury, and structural damage to the renal tubular cells. The doxorubicin-induced cardiotoxicity has been linked to mitochondrial dysfunction, iron-mediated oxidative reactions, calcium imbalance, and apoptosis of cardiomyocytes, ultimately leading to impaired cardiac performance and irreversible cardiac injury in severe cases. These overlapping pathogenic pathways emphasize the central role of oxidative and inflammatory mechanisms in the progression of drug-induced toxicity across multiple tissues and organ systems. In recent years, herbal extracts and phytochemicals have attracted considerable interest as potential protective agents against drug-induced toxicity. Numerous bioactive compounds isolated from medicinal plants have demonstrated antioxidant, anti-inflammatory, antiapoptotic, and cytoprotective properties in experimental studies. For example, silymarin has been reported to stabilize cellular membranes, enhance antioxidant enzyme activity, and reduce lipid peroxidation in hepatocytes, while curcumin has been shown to exert potent anti-inflammatory effects through the inhibition of the nuclear factor kappa-B signalling pathways and the suppression of inflammatory mediator production. Another example is epigallocatechin gallate (the major catechin found in green tea), which has been shown to act as a powerful free

Mohammad T. M., Fadheel Q. J., Kadhim A. S.: Protective effects of herbal extracts against drug-induced toxicity: a review of pharmacological evidence and mechanisms. *Colloq. Erud.* 1: 49–50 (2026).
<https://doi.org/10.5281/zenodo.20920114>

This proceedings abstract is published in *Colloquia Erudita – Volume 1: Proceedings of the 3rd International Babylon Conference on Clinical and Experimental Pharmacological Research* (Hillah; June 29–30, 2026); a volume of proceedings edited by Rafal J. Al-Saigh, Argyro Kyriakaki, and Apostolos Zarros.

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radical scavenger and to be able to protect macromolecules from oxidative injury. Finally, thymoquinone has been shown to exhibit both antioxidant as well as anti-inflammatory properties that can counteract the tissue damage caused by toxic metabolites, whereas ginsenosides have been shown to improve mitochondrial function, regulate apoptosis-related pathways, and enhance endogenous antioxidant defences. Experimental studies suggest that these phytochemicals may attenuate tissue injury, preserve mitochondrial integrity, improve biochemical markers of organ function, and enhance cellular resistance against toxic insults. Interestingly, several studies also indicate that herbal compounds may support cellular recovery processes and improve tissue regeneration following drug-induced toxicity. This review summarizes the principal molecular mechanisms underlying drug-induced toxicity and critically evaluates current evidence regarding the protective effects of selected herbal extracts and phytochemicals, while discussing their potential therapeutic significance and the existing limitations for their clinical application.

Keywords

drug toxicity; herbal extracts; mitochondrial dysfunction; oxidative stress; phytochemicals

Acknowledgements

The authors would like to express their appreciation to the staff and colleagues at the College of Pharmacy of the University of Babylon for their encouragement and assistance.

Conflicts of interest statement

None to declare.

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