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PROCEEDINGS ABSTRACT

Molecular basis of drug-induced organ toxicity and advances in protective pharmacological interventions: a review

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Drug-induced toxicity is a major clinical and pharmacological challenge that continues to contribute significantly to patient morbidity, prolonged hospitalisation, and drug withdrawal worldwide. Although drugs are developed with the aim to provide therapeutic benefits, their biochemical and molecular interactions within the body are complex and may lead to unintended harmful effects involving multiple organ systems. Such adverse effects can arise through several mechanisms of toxicity, involving (but not limited to) dose-dependent toxicity, immune-mediated toxicity, mitochondrial dysfunction and oxidative stress, disruption of cellular homeostasis, and the activation of inflammatory or apoptotic signalling pathways; these processes may be occurring individually or may be synergistically leading to tissue injury and organ dysfunction. In addition, interindividual variability (particularly in terms of genetic polymorphisms affecting drug metabolism and transport, as well as detoxification pathways) can play a significant role in determining drug disposition and susceptibility to toxicity, making some patients more vulnerable than others even at standard therapeutic doses. In addition, environmental factors, drug interactions, and underlying comorbidities may further increase the risk and severity of toxic responses and drug adverse reactions. Among the organs / systems most frequently affected by drug-induced toxicity are the liver, the kidneys, the heart, and the auditory system. Hepatotoxicity is commonly associated with the cytochrome P450-mediated metabolism of drugs into reactive intermediates, which promote oxidative stress, the depletion of intracellular antioxidants such as glutathione, and the subsequent hepatocellular injury / necrosis. Nephrotoxicity often results from mitochondrial impairment, inflammatory responses, oxidative damage, as well as direct tubular cell damage, particularly with drugs such as aminoglycosides and cisplatin. Cardiotoxicity, especially the one induced by anthracyclines such as doxorubicin, is linked to multiple mechanisms including topoisomerase inhibition, iron dysregulation, calcium imbalance, excessive reactive oxygen species generation, and mitochondrial dysfunction, ultimately leading to myocardial injury and impaired cardiac function. Finally, ototoxicity, which is frequently associated with aminoglycosides and platinum-based anticancer drugs, involves a disruption of ion homeostasis, the excessive production of reactive oxygen species, and an irreversible damage to cochlear hair cells resulting in hearing impairment or permanent hearing loss. The better understanding of the molecular mechanisms involved in such cases of drug-induced organ injury has contributed to the development of several protective pharmacological strategies aimed at minimizing toxicity and improving patient safety. These strategies

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involve the use of antioxidant agents, enzyme modulators, mitochondrial protectants, transporter inhibitors, and immunomodulators. A common example of such strategies is the use of *N*-acetylcysteine in counteracting acetaminophen-induced hepatotoxicity by restoring intracellular glutathione levels. Dexrazoxane is also commonly employed for the reduction of the anthracycline-induced cardiotoxicity. Other approaches aim at the modulation of signalling pathways, the enhancement of endogenous antioxidant defences, the reduction of inflammatory responses, and the stabilization of mitochondrial integrity and function. The further development of pharmacogenomics, the establishment of nanotechnology-based drug delivery systems, and the launch of reliable artificial intelligence-driven toxicity prediction platforms are all gaining increasing attention for their potential to enhance drug safety and support personalised therapeutic strategies and precision medicine. These innovations may allow for the early identification of high-risk individuals, the better monitoring of drug responses, and a more precise optimization of dosing regimens in clinical practice. This review provides an overview of key molecular mechanisms underlying drug-induced organ toxicity and highlights established and emerging protective pharmacological strategies. A deeper understanding of these interconnected processes may support safer drug development, improve the early detection of adverse effects, and contribute to the development of more individualised treatment approaches, thereby enhancing clinical outcomes and minimizing drug-induced harm in diverse patient populations.

Keywords

cardiotoxicity; drug toxicity; hepatotoxicity; nephrotoxicity; protective strategies

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Conflicts of interest statement

None to declare.

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