

Biomarkers of Toxicity for Monitoring Adverse Drug Reactions

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DESCRIPTION

Biomarkers of toxicity have become an essential component of modern pharmacology and toxicology, particularly in the monitoring and prediction of adverse drug reactions. In my opinion, their growing importance reflects a necessary shift from traditional approaches that relied heavily on observable clinical symptoms to more precise, mechanism-based methods of detecting harm at an earlier stage. Adverse drug reactions remain a major challenge in healthcare systems worldwide, often leading to hospital admissions, treatment failure, and in severe cases, life-threatening complications. The use of reliable biomarkers offers an opportunity to identify toxicity before irreversible damage occurs, thereby improving patient safety and therapeutic outcomes.

A biomarker of toxicity can be described as a measurable biological indicator that reflects exposure to a drug, a biological response to that exposure, or the degree of organ dysfunction caused by it. These biomarkers may include enzymes, proteins, genetic markers, metabolites, or physiological changes that signal disruption in normal biological processes. In the context of adverse drug reactions, they serve as early warning signals that allow clinicians to adjust treatment before significant harm develops. I believe this represents a major advancement in personalized medicine, as it enables healthcare providers to tailor drug therapy based on individual risk profiles rather than relying on generalized dosing strategies. One of the most significant applications of toxicity biomarkers is in the detection of drug-induced organ injury. For example, liver toxicity is a common reason for drug withdrawal from the market, and biomarkers such as alanine aminotransferase and aspartate aminotransferase have long been used to assess hepatic damage. However, these traditional markers often indicate injury only after substantial damage has already occurred. Emerging biomarkers, including micro ribonucleic acids and keratin-18 fragments, show promise in detecting liver injury at much earlier stages. In my view, this shift toward earlier detection is crucial, as

it provides a window of opportunity to prevent irreversible organ failure and improve clinical decision making. Similarly, biomarkers are increasingly being explored for the detection of kidney, cardiac, and neurological toxicity. Kidney injury molecule one and neutrophil gelatinase associated lipocalin are examples of renal biomarkers that can detect kidney injury more sensitively than traditional measures such as serum creatinine. Cardiac troponins are widely used in detecting heart muscle damage, while emerging neurological biomarkers such as glial fibrillary acidic protein are being investigated for brain injury. The expanding range of these indicators highlights the growing sophistication of toxicological monitoring systems. I believe that integrating these biomarkers into routine clinical practice will significantly enhance the ability to detect adverse drug reactions across multiple organ systems.

Despite their potential, the implementation of toxicity biomarkers in clinical practice faces several challenges. Variability in biomarker expression among individuals, lack of standardization in measurement techniques, and limited validation across diverse populations remain significant barriers. Additionally, some biomarkers may be influenced by factors unrelated to drug exposure, such as underlying diseases or environmental stressors, which can complicate interpretation. In my opinion, addressing these challenges requires coordinated efforts between researchers, clinicians, and regulatory authorities to establish standardized protocols and validate biomarkers across large-scale clinical studies.

Another important consideration is the integration of biomarkers with advanced technologies such as pharmacogenomics and computational modeling. Combining genetic information with toxicity biomarkers can provide a more comprehensive understanding of why certain individuals are more susceptible to adverse drug reactions. This integrated approach supports the development of precision medicine, where drug therapy is customized based on both biological response and genetic predisposition.

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