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An Integrative Interpretation of Toxicity: Genetic Damage, Target Organs, and Individual Susceptibility in Modern Toxicology

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ABSTRACT
Exposure to toxic substances remains an important concern in health sciences because its effects may progress from the molecular level to cellular, tissue, and organ damage. This article aims to synthesize the relationships among the initial pathways of toxicity, susceptible target organs, and individual susceptibility within modern toxicology. This study employed a qualitative approach with a narrative review design, examining toxicological literature covering six main subthemes: chemical carcinogenesis, genetic toxicology, hepatotoxicity, respiratory toxicity, neurotoxicity, and skin toxicity. The data were analyzed through source identification, thematic selection, content reduction, categorization, thematic synthesis, and narrative interpretation. The review produced three main findings. First, genetic damage and oxidative stress constitute important early pathways that may initiate subsequent toxic effects. Second, the liver and nervous system are highly susceptible target organs because of their central biological roles in metabolism, regulation, and homeostasis. Third, toxic responses are multiorgan in nature and are influenced by exposure routes, substance characteristics, and individual biological susceptibility. This review emphasizes that modern toxicology needs to integrate molecular biomarkers, target-organ responses, and host-related factors within a unified analytical framework. It concludes that toxicological approaches should move toward more predictive, systemic, and personalized models. Future research is recommended to employ systematic reviews, meta-analyses, and longitudinal experimental studies to strengthen evidence regarding the relationships among biomarkers, organ damage, and variations in individual responses.

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Introduction

Exposure to toxic substances remains an important concern in health sciences because its effects do not only appear as acute poisoning but may also develop progressively at the molecular, cellular, tissue, and organ levels. Modern society continues to face various sources of exposure, including air pollutants, industrial chemicals, pesticides, pharmaceuticals, inhaled particles, and other xenobiotics that may enter the body through ingestion, inhalation, or skin contact. Therefore, modern toxicology can no longer be understood merely as the study of "toxic substances," but needs to be interpreted as a field that examines how exposure triggers oxidative stress, DNA damage, mitochondrial dysfunction, inflammation, organ dysfunction, and variations in biological responses among individuals.

The development of contemporary toxicology indicates a shift from descriptive approaches toward more integrative, predictive, and personalized approaches. Toxicological studies no longer assess only whether a substance is harmful, but also investigate the initial pathways of biological damage, the target organs affected, and the host factors that cause toxic responses to differ among individuals. In this context, molecular biomarkers such as 8-hydroxy-2′-deoxyguanosine (8-OHdG) or 8-oxo-7,8-dihydro-2′-deoxyguanosine (8-OxodG) are important because they can indicate oxidative DNA damage before organ dysfunction becomes clinically apparent (AbuArrah et al., 2021; Goriuc et al., 2024; Kumar et al., 2024). Thus, the early detection of toxicity needs to begin at the molecular level rather than relying solely on clinical symptoms or end-stage organ damage.

A number of recent studies have explained that toxicity may operate through interconnected pathways. DNA damage, oxidative stress, inflammation, and mitochondrial dysfunction often serve as initial mechanisms linking toxicant exposure to subsequent effects on organs. Singh et al. (2023) emphasize that toxicogenomics and omics technologies are increasingly important for explaining toxicity with greater precision because they can connect exposure, molecular responses, and individual susceptibility. Vinken (2024) also shows that the development of adverse outcome pathway networks can help clarify the relationship among molecular initiating events, intermediate biological changes, and toxic outcomes in target organs. Accordingly, modern toxicology is increasingly moving toward a systems-based interpretation rather than an approach that examines each organ or substance separately.

Although toxicological literature continues to develop, important gaps remain. Many discussions still treat chemical carcinogenesis, genetic toxicology, hepatotoxicity, neurotoxicity, respiratory toxicity, and skin toxicity as independent themes. In fact, these forms of toxicity may be connected through common biological mechanisms, such as oxidative stress, DNA damage, reactive metabolite formation, inflammation, and impaired mitochondrial function. This gap is important because organ-based separation may prevent the relationships among toxic mechanisms from being understood comprehensively. Consequently, toxicity may be interpreted merely as a collection of organ injuries rather than as a multilevel biological process progressing from exposure to molecular damage, cellular responses, organ dysfunction, and clinical outcomes.

Another gap lies in the insufficient integration of target organs and individual susceptibility. The liver is often understood as a primary target organ because of its role in xenobiotic metabolism and detoxification, while the nervous system is considered vulnerable because of its high energy demand, network complexity, and limited regenerative capacity. However, the conclusion that the liver and nervous system are highly susceptible organs needs to be interpreted carefully because the degree of vulnerability may differ according to toxicant type, dose, duration, exposure route, and individual biological characteristics. In addition, genetic variation, mitochondrial function, antioxidant capacity, age, health status, and previous exposure may produce non-uniform toxic responses. In other words, toxicity is determined not only by the properties of the toxic substance but also by the biological condition of the exposed individual.

Based on these gaps, this article offers an integrative synthesis of toxicity by connecting three levels of analysis that are often discussed separately: genetic damage as an initial pathway, target organs as the sites of toxic manifestation, and individual susceptibility as an explanation for variations in response. This focus is important because modern toxicology increasingly requires an approach capable of integrating molecular biomarkers, organ mechanisms, and host factors within a unified risk-assessment framework. Bai et al. (2025) show that modern toxicity prediction has begun to use multisource data and computational models to capture the complexity of toxic mechanisms, although major challenges remain regarding data quality, interpretability, and regulatory acceptance. This indicates that conceptual synthesis is still needed to clarify biological relationships before predictive models can be developed further.

Accordingly, this study is guided by three main questions. First, how do genetic damage and oxidative stress function as early pathways in producing subsequent toxic effects? Second, why do the liver and nervous system frequently emerge as highly susceptible target organs, and within what limits does this conclusion apply across different types of toxicants? Third, how do exposure routes, substance characteristics, and individual susceptibility shape toxic responses that are multiorgan and non-uniform? These questions were formulated so that the review would not stop at describing toxicological themes but would establish conceptual relationships among biomarkers, target organs, and host factors.

This article aims to synthesize the relationships among genetic damage, target organs, and individual susceptibility in modern toxicology. Its main contribution lies in emphasizing that toxicity should be understood as a biological process that progresses from the molecular level to organs and body systems and is influenced by individual biological variation. Through this interpretation, the article is expected to strengthen the direction of modern toxicology toward more integrative, predictive, systemic, and personalized approaches, particularly in the development of early biomarkers, exposure-risk assessment, and evidence-based public health policies.

Method

This study employed a qualitative approach with a narrative review design. This design was selected because the study did not aim to test an experimental hypothesis, but rather to synthesize toxicological findings distributed across several themes, including genetic damage, oxidative stress, target organs, exposure pathways, and individual biological susceptibility. A narrative review is relevant when researchers seek to examine a broad issue, connect dispersed concepts, and develop an interpretive synthesis from scientific sources with conceptual relationships (Snyder, 2019; Paul & Criado, 2020; Sukhera, 2022). Through this approach, the review does not merely summarize the literature but also organizes the relationships among themes so that toxicity can be understood as a biological process progressing from the molecular level to organ damage and systemic responses.

The sources reviewed in this study consisted of scientific publications discussing toxicity at the molecular, cellular, tissue, and organ levels. Based on the source-search and selection process, this article used 13 core references relevant to the focus of the review. These sources consisted of research articles, review articles, and methodological articles discussing narrative reviews and document analysis. The sources were selected because they represented six main subthemes: chemical carcinogenesis, genetic toxicology, hepatotoxicity, respiratory system toxicity, neurotoxicity, and skin toxicity. These six subthemes were purposively selected because they illustrate the principal pathways of toxicity, ranging from DNA damage and oxidative stress to manifestations in target organs and variations in individual responses.

Source selection was conducted through a staged procedure. The first stage involved identifying sources based on their relevance to the article's focus, including toxicity, genetic damage, oxidative stress, molecular biomarkers, hepatotoxicity, neurotoxicity, respiratory

toxicity, skin toxicity, and individual susceptibility. The second stage involved screening the sources based on their direct relevance to the three analytical dimensions: the initial pathways of toxicity, susceptible target organs, and host factors. The third stage involved determining the core sources used in the synthesis. This procedure was applied to ensure that the reviewed materials were genuinely relevant to the purpose of the article and could support the development of an integrative argument. In literature-based studies, a clear source-selection procedure is important to ensure that the resulting synthesis is traceable and does not merely consist of subjective narrative opinion (Snyder, 2019; Paul & Criado, 2020).

The inclusion criteria were as follows: (1) the article discussed mechanisms of toxicity at the molecular, cellular, organ, or body-system level; (2) the article addressed biomarkers, oxidative stress, DNA damage, target organs, or variations in individual responses; (3) the article was a traceable scientific publication; (4) the article was written in Indonesian or English; and (5) the article was directly relevant to the six subthemes examined. The exclusion criteria included: (1) sources that only discussed general definitions of toxicology without linking them to biological mechanisms; (2) non-academic popular articles; (3) sources that could not be traced bibliographically; (4) publications unrelated to biomarkers, target organs, or individual susceptibility; and (5) sources that merely repeated information without contributing conceptually to the synthesis.

The unit of analysis in this study was not human participants, but the scientific content contained in the selected literature. The units of analysis included concepts, biological mechanisms, biomarkers, target organs, exposure routes, and examples of toxic effects reported in the literature. For example, 8-OHdG and 8-OxodG were positioned as indicators of oxidative DNA damage; ALT and AST as markers of hepatotoxicity; pulmonary inflammation as an indicator of respiratory toxicity; and sensory, motor, and mitochondrial disturbances as components of neurotoxicity. This approach is consistent with the characteristics of document-based research because the analysis focuses on the depth of meaning and the relationships among concepts rather than the number of participants or statistical generalization (Kayesa & Shung-King, 2021; Snyder, 2019).

The analysis process was conducted in five stages. The first stage was content identification, which involved reading all selected sources to identify key terms, mechanistic focuses, target organs, and forms of toxic response. The second stage was data reduction, which involved selecting the information most relevant to the study objectives and removing repetitive descriptions or information not directly related to the review focus. The third stage was categorization, in which the findings were grouped into three main categories: initial toxicity pathways, target organs, and individual susceptibility. The fourth stage was thematic synthesis, which involved connecting findings from different sources to identify patterns linking molecular biomarkers, organ damage, and variations in biological responses. The fifth stage was narrative interpretation, in which the synthesized findings were developed into a coherent conceptual argument regarding toxicity as a multilevel and multiorgan process. These stages are consistent with review-writing principles that emphasize the identification, organization, synthesis, and interpretation of meaning from the literature examined (Snyder, 2019; Paul & Criado, 2020; Sukhera, 2022).

To maintain analytical traceability, each source was not only summarized but also compared based on its contribution to the three main research focuses. Sources discussing DNA damage and oxidative stress were used to construct the findings on initial toxicity pathways. Sources discussing the liver, nervous system, respiratory system, and skin were used to explain target organs and toxic manifestations. Sources discussing genetic variation, mitochondrial function, and differences in biological responses were used to explain individual susceptibility. In this way, the synthesis was not arranged randomly, but followed a conceptual sequence from exposure and molecular responses to organ damage and variations in toxic outcomes.

The results of the analysis were presented narratively and descriptively through three main findings. First, genetic damage and oxidative stress were positioned as initial pathways of toxicity. Second, the liver and nervous system were discussed as highly susceptible target organs, while acknowledging that organ vulnerability may differ according to toxicant type, dose, duration, and exposure route. Third, toxic responses were explained as multiorgan processes influenced by exposure pathways, substance characteristics, and individual biological susceptibility. This presentation was selected so that the review would not become merely a collection of literature summaries but would produce an integrative interpretation of modern toxicology. In a narrative review, a coherent synthesis is essential because its primary purpose is to develop conceptual understanding rather than generate quantitative estimates, as in a meta-analysis (Sukhera, 2022; Paul & Criado, 2020).

This study has methodological limitations. Because it employed a narrative review design, it did not conduct a meta-analysis, quantitative risk-of-bias assessment, or systematic study-quality appraisal using instruments typically applied in systematic reviews. Therefore, the findings were not intended to produce effect estimates or quantitative generalizations. The main strength of this study lies in its conceptual synthesis integrating molecular biomarkers, target organs, and individual susceptibility within a unified analytical framework. Thus, this article may serve as an initial foundation for future systematic reviews, meta-analyses, or longitudinal experimental studies (Snyder, 2019; Sukhera, 2022).

Results

Based on the review of six main themes, namely chemical carcinogenesis, genetic toxicology, hepatotoxicity, respiratory system toxicity, neurotoxicity, and skin toxicity, three core findings were identified. First, genetic damage and oxidative stress emerged as initial pathways linking toxicant exposure to subsequent biological effects. Second, the liver and nervous system appeared as highly susceptible target organs because both perform essential biological functions in metabolism, detoxification, regulation, and bodily homeostasis. Third, toxic responses are multiorgan in nature and are influenced by exposure routes, substance characteristics, dose, exposure duration, and individual biological susceptibility. Thus, toxicity cannot be understood as a single process or as damage confined to one organ. Instead, it should be understood as a series of biological processes progressing from the molecular level to tissue and organ injury.

Genetic Damage and Oxidative Stress as Initial Pathways of Toxicity

The first finding shows that genetic damage and oxidative stress are important initial pathways in the development of toxicity. Exposure to toxic substances may trigger the formation of reactive oxygen species, disruption of DNA stability, changes in the mitotic index, micronucleus formation, acentric fragments, and biomarkers of oxidative DNA damage such as 8-OHdG or 8-OxodG. This finding indicates that toxic effects do not always immediately appear as organ damage, but may begin with earlier molecular alterations. Therefore, genetic and oxidative biomarkers occupy a strategic position as early indicators before tissue damage or clinical symptoms develop further.

The Liver and Nervous System as Highly Susceptible Target Organs

The second finding shows that the liver and nervous system are two target organs that are highly susceptible to toxicant exposure. The liver is vulnerable because it serves as the primary center for xenobiotic metabolism and detoxification. It is therefore readily exposed to reactive metabolites that may trigger oxidative stress, inflammation, and cellular damage. The nervous system is also highly sensitive because of its high energy requirements, complex tissue structure, and limited capacity for cellular regeneration. However, this finding needs to be interpreted cautiously. The susceptibility of the liver and nervous system does not apply equally to all

toxicants, but depends greatly on the class of substance, route of exposure, dose, duration of exposure, and the toxicant's ability to cross specific biological barriers.

Toxic Responses Are Multiorgan and Influenced by Individual Susceptibility

The third finding shows that toxic responses are multiorgan and vary among individuals. In the respiratory system, exposure to particles and inhaled substances may trigger inflammation, tissue damage, fibrosis, asthma, or chronic pulmonary disorders. In the skin, toxicant exposure may cause irritation, edema, erythema, and inflammation. In the nervous system, exposure to certain neurotoxicants may lead to sensory and motor disturbances, seizures, or other neurological damage. In addition, genetic variation, mitochondrial function, antioxidant capacity, age, health status, and previous exposure may influence the severity of toxic effects. Thus, two individuals exposed to the same substance may demonstrate different biological responses because of differences in host-related factors.

Discussion

Genetic Damage and Oxidative Stress as the Basis for the Early Detection of Toxicity

The first finding confirms that genetic damage and oxidative stress are early mechanisms that may connect toxicant exposure with organ damage. Biomarkers such as 8-OHdG and 8-OxodG are important because they can indicate oxidative DNA damage before clinical symptoms become clearly apparent. AbuArrah et al. (2021) showed that 8-OHdG can be used as a potential biomarker for detecting oxidative DNA damage caused by exposure to medical ionizing radiation. Kumar et al. (2024) also emphasized that 8-OxodG can be used as a biomarker of chronic oxidative stress and is associated with genomic instability. Thus, the findings of this review support the direction of modern toxicology, which increasingly emphasizes early detection based on molecular biomarkers.

However, the use of molecular biomarkers still needs to be interpreted cautiously. Elevated levels of 8-OHdG or 8-OxodG do not necessarily indicate one specific type of exposure because these biomarkers may be influenced by various factors, including radiation, pollutants, inflammation, lifestyle, age, chronic diseases, and the body's antioxidant status. Therefore, molecular biomarkers are more appropriately positioned as early risk indicators rather than as definitive evidence of a single cause of toxicity. This is an important limitation that must be emphasized to prevent toxicological interpretation from becoming deterministic.

Conceptually, this finding reinforces the need for predictive approaches in toxicology. Vinken (2024) explains that adverse outcome pathway networks can be used to connect molecular initiating events with intermediate biological changes and toxic outcomes in organs. In the context of this article, DNA damage and oxidative stress can be understood as early events that trigger a sequence of subsequent effects. Therefore, modern toxicology should not merely describe which organs are damaged, but should also trace the biological pathways from exposure to toxic outcomes.

The Liver and Nervous System as Target Organs: Strongly Supported but Not Universal across All Toxicants

The second finding shows that the liver and nervous system are highly susceptible target organs. The liver occupies a central position in xenobiotic metabolism, meaning that numerous chemical substances, pharmaceuticals, and reactive metabolites are processed through this organ. Messina and Duclos-Vallée (2023) explain that hepatotoxicity may occur through various molecular mechanisms, including the formation of reactive metabolites, oxidative stress, mitochondrial dysfunction, and immune responses. Thakur et al. (2024) also emphasize the importance of hepatotoxicity biomarkers for assessing liver injury at an earlier stage. Thus, identifying the liver as a major target organ has a strong biological basis.

The nervous system is also highly susceptible because it depends on neurotransmitter balance, mitochondrial function, and neural tissue integrity. Chen et al. (2024) show that organophosphate pesticides may induce neurotoxicity through multiple mechanisms associated with neurological disorders. However, the claim that the nervous system is the most susceptible target organ must be restricted according to the type of toxicant. Neurotoxicity is particularly pronounced in exposure to organophosphates, methylmercury, cyanide, organic solvents, or substances capable of crossing the blood-brain barrier. In contrast, for other toxicants, the primary target organs may be the kidneys, lungs, skin, immune system, or reproductive system.

Accordingly, this article does not conclude that the liver and nervous system are always the most vulnerable organs under all conditions. A more appropriate conclusion is that the liver and nervous system frequently become important targets because of their central biological functions. However, their degree of susceptibility still depends on the toxicant, exposure route, dose, duration, and individual condition. This clarification is important so that the synthesis does not become overly generalized, but remains critical and consistent with the principles of toxicological risk assessment.

Multiorgan Responses and the Importance of Exposure Routes

The third finding shows that toxicity is multiorgan in nature and is strongly influenced by the exposure route. Toxicants entering through inhalation may affect the respiratory system, but their effects do not necessarily remain confined to the lungs. Borgatta and Breider (2024) show that microplastic inhalation is a complex toxicological issue because particles may interact through their physical and chemical properties and potentially cause both local and systemic effects. This supports the finding that the exposure route influences the initial site of damage, while biological distribution may extend the effects to other organs.

Dermal exposure likewise cannot be considered purely local. Although the skin functions as an initial barrier, certain substances may trigger inflammation, irritation, immunological reactions, or systemic absorption. Similarly, exposure through the gastrointestinal tract may affect the liver because portal circulation transports many xenobiotics to the primary metabolic organ. Therefore, toxicological interpretation needs to consider the relationship among the route of entry, substance distribution, metabolism, and target organs. A single-organ approach risks oversimplifying toxic processes that actually progress systemically.

This reflection is important for the development of systems toxicology. In a systemic approach, toxicity is understood as the result of interactions among substances, tissues, organs, molecular pathways, and host factors. Bai et al. (2025) show that modern toxicity prediction has begun to use machine-learning models to integrate various categories of toxicity and data sources. However, challenges remain regarding data variability, model interpretability, and the selection of appropriate algorithms. Thus, the findings of this review are relevant to the global direction of predictive toxicology while also emphasizing that predictive models must remain grounded in a strong biological understanding.

Individual Susceptibility and the Challenges of Personalized Toxicology

The most important aspect of this synthesis is the emphasis that toxic responses are not uniform among individuals. Garrabou et al. (2017) showed that mitochondrial genetic variation may influence linezolid toxicity in blood cells and skin nerve fibers. Oehadian et al. (2022) also emphasized that the hematological toxicity of linezolid is associated with mitochondrial function, particularly in the treatment of drug-resistant tuberculosis. These findings indicate that toxic effects are determined not only by dose and substance type, but also by the individual's biological capacity to respond to exposure.

However, the application of personalized toxicology still faces practical challenges. First, genetic, mitochondrial, and molecular biomarker testing is not yet widely available, particularly

in resource-limited public health systems. Second, testing costs and laboratory infrastructure requirements may restrict the implementation of individual screening. Third, not all biomarkers have clearly established clinical thresholds for use in public policy. Fourth, the use of genetic data requires careful attention to ethics, privacy, and the potential for risk-based discrimination.

Therefore, personalized toxicology needs to be developed gradually. At the research level, this approach may begin with longitudinal studies linking molecular biomarkers, genetics, environmental exposure, and clinical outcomes. At the policy level, personalized approaches do not need to begin immediately with mass genetic screening. Instead, they may start by identifying vulnerable groups, such as workers with chronic exposure, patients receiving potentially toxic medications, children, older adults, or individuals with comorbid conditions. In this way, personalized toxicology may become a future direction without disregarding the practical limitations of public health systems.

Implications and Limitations of the Review

Academically, this review strengthens the argument that modern toxicology needs to integrate molecular biomarkers, target organs, and individual susceptibility within a unified framework. This approach is important for avoiding an overly fragmented understanding of toxicity based solely on individual organs or substance types. Practically, the findings are relevant to the development of early detection for chronic exposure, drug safety assessment, occupational safety, environmental protection, and policies for controlling hazardous chemicals.

Nevertheless, this review has several limitations. First, it employed a narrative review design and therefore did not apply strict systematic-review procedures, such as risk-of-bias assessment, study-quality appraisal, or meta-analysis. Second, the synthesis did not quantitatively compare the strength of evidence across studies. Third, conclusions regarding target organs remain conceptual and require further testing within specific groups of toxicants. Fourth, the discussion of personalized toxicology remains conceptual and requires support from longitudinal empirical studies and stronger biomarker data. Therefore, the findings are more appropriately positioned as an initial conceptual synthesis rather than as a final conclusion based on quantitative evidence.

Conclusion

This review concludes that toxicity is a complex, progressive, and interconnected biological process operating across multiple levels. Genetic damage and oxidative stress function as early pathways that may trigger subsequent toxic effects. Therefore, molecular biomarkers such as 8-OHdG or 8-OxodG are important as early indicators, although their interpretation must still account for the exposure context and other biological factors.

The review also shows that the liver and nervous system are highly susceptible target organs, although this claim is not universal across all toxicants. Organ susceptibility is strongly influenced by substance type, exposure route, dose, duration, metabolism, distribution, and the toxicant's ability to reach specific organs. Thus, the liver and nervous system should be understood as important target organs in many toxicological contexts, but not as the only organs that are always most susceptible.

Toxic responses are also multiorgan and influenced by individual susceptibility. Genetic variation, mitochondrial function, antioxidant capacity, age, health status, and exposure history may cause individuals exposed to the same substance to respond differently. This finding emphasizes the importance of personalized toxicology, although its implementation still faces challenges related to cost, infrastructure, biomarker validation, ethics, and public health policy.

The main contribution of this article lies in integrating three levels of toxicological analysis, namely molecular biomarkers, target organs, and host factors, within a unified framework. However, because this study employed a narrative review design, its findings are more

appropriately understood as a conceptual synthesis rather than as definitive quantitative evidence. Future research is recommended to use systematic reviews, meta-analyses, experimental studies, and longitudinal designs to examine more robustly the relationships among molecular biomarkers, organ damage, and variations in individual responses. Through this direction, modern toxicology may develop into a more predictive, systemic, and personalized field while becoming more relevant to public health, occupational safety, and chemical regulation.

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Research Ethics Statement
This study was conducted in accordance with the ethical principles of scientific research. Since this study was a document-based literature review and did not directly involve human participants, informed consent and formal approval from an ethics committee were not required. Nevertheless, the authors maintained academic integrity, scientific honesty, accurate representation of the reviewed literature, proper acknowledgment of all sources, and responsible use of published materials throughout the research and writing process.

Author Contributions
Zulviana: conceptualization, literature collection, data analysis, synthesis of findings, and writing of the original draft.
Ahmad Irawan: methodology, content validation, academic supervision, substantive review, and manuscript editing.
All authors have read, reviewed, and approved the final version of the manuscript.

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The authors declare that there is no conflict of interest regarding the research, authorship, and publication of this article.

Artificial Intelligence Use Statement
The authors declare that artificial intelligence, if used, was employed only as a technical support tool for language editing, sentence refinement, grammar checking, and improving manuscript readability. All scientific analysis, literature synthesis, interpretation of findings, arguments, and conclusions remain the full responsibility of the authors.

Data Availability Statement
The data supporting the findings of this study consist of published scientific articles, review materials, analytical notes, thematic classifications, and literature synthesis documents related to genetic damage, oxidative stress, target organs, and individual susceptibility in modern toxicology. Since this study was based on published literature, no new primary dataset was generated. Additional information regarding the reviewed sources and analytical process may be obtained from the corresponding author upon reasonable request.

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