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From DNA to Multiorgan Effects: An Analysis of Everyday Chemical Toxicity and Hidden Toxic Threats to Human Health

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ABSTRACT

Exposure to hazardous chemicals has become increasingly embedded in everyday life through food, medicines, air pollution, cosmetics, and other consumer products. However, toxicity is still frequently examined separately according to the type of toxicant or target organ, leaving the relationships among genetic damage, exposure pathways, and multiorgan effects insufficiently integrated. This study aims to analyze patterns of everyday chemical toxicity from the genetic level to their manifestations in various organs. A qualitative document analysis was conducted using one core manuscript covering chemical carcinogenesis, genetic toxicology, hepatotoxicity, respiratory toxicity, neurotoxicity, and skin toxicity, supplemented by relevant scientific literature. The analysis involved in-depth reading, coding, categorization, thematic synthesis, and narrative interpretation. Three main findings were identified. First, chemical exposure may induce genetic damage that serves as an early pathway in the development of chronic disorders. Second, the liver and respiratory system emerged as the dominant target organs in the analyzed document because of their roles in xenobiotic metabolism and pollutant entry. Third, the nervous system and skin exhibited risks of toxic accumulation that may not be immediately recognized, particularly following repeated low-dose exposure. These findings indicate that everyday chemical toxicity should be understood across biological levels and organ systems. However, the results should be interpreted as a conceptual synthesis rather than as quantitative evidence derived from primary measurements. Future research should employ biomarkers, biomonitoring, and multiroute exposure analysis to examine these relationships more empirically and assess their causal implications.

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Introduction

Exposure to hazardous chemicals has become a public health concern that is increasingly embedded in everyday life. Such exposure may originate from food, medicines, air pollution, cosmetics, household products, industrial activities, and rapidly changing urban environments. The World Health Organization reported that the combined effects of ambient and household air pollution remain associated with approximately 6.7 million premature deaths annually, while most of the world's population lives in areas where air quality does not meet WHO guidelines (World Health Organization [WHO], 2024a). In addition, mercury-containing skin-lightening products remain a global health concern because they may cause kidney, nervous system, and skin disorders (WHO, 2024b). These facts indicate that toxic threats arise not only from chemical accidents or industrial exposure, but also from routine exposures that appear ordinary, familiar, and frequently remain unnoticed.

Within modern toxicology, chemical exposure can no longer be narrowly understood as an isolated toxic effect affecting only one organ. Chemicals may enter the body through oral, inhalation, and dermal routes and subsequently trigger biological changes at the molecular, cellular, tissue, and organ levels. DNA damage, oxidative stress, inflammation, mitochondrial dysfunction, and bioaccumulation are important mechanisms linking everyday exposure to long-term health disorders. The exposome framework strengthens this perspective by viewing human health as the result of total lifetime exposure, including interacting environmental, behavioral, social, and biological factors (Wan et al., 2025). Therefore, toxicity should be interpreted as a process occurring across biological levels and body systems rather than merely as localized damage caused by a single substance.

Several recent studies have explained the toxic effects of chemicals on particular organs or biological systems. Research on xenobiotics indicates that chemicals may induce liver injury through enzymatic bioactivation, oxidative stress, DNA damage, inflammation, and immune dysfunction (Wang et al., 2025). Studies of air pollution have shown that long-term exposure to ambient pollutants may reduce lung function and increase the risk of respiratory disorders (Gross et al., 2025). Heavy metals have also been associated with neurotoxicity through oxidative stress, mitochondrial dysfunction, DNA damage, and disruption of cellular regulation (Jomova et al., 2025). Meanwhile, mercury in skin-lightening products remains a concern because it is associated with dermal, neurological, and renal effects (Bastiansz et al., 2022). These studies demonstrate the broad scope of chemical toxicity, although most continue to examine exposure and target organs separately.

The research gap addressed in this article arises from the fragmented nature of toxicological research, which often examines individual organs, substances, or exposure routes separately, such as genotoxicity, hepatotoxicity, respiratory toxicity, neurotoxicity, and dermatotoxicity. In real-life settings, however, chemical exposure is often repeated, low-dose, multiroute, and cumulative. Consequently, early genetic damage, the prominence of particular target organs, and toxic accumulation in the nervous system and skin should not be interpreted as separate phenomena, but as interconnected components of a biological pathway. The main gap addressed in this article is the limited availability of integrative analyses connecting genotoxicity, target organs, exposure routes, and hidden toxic accumulation within a unified analytical framework.

The main contribution of this article lies in its interpretation of everyday chemical toxicity as a process progressing from the DNA level to multiorgan effects. The article does not only ask which substances are hazardous, but also examines how exposure operates, through which routes chemicals enter the body, which organs most frequently emerge as targets in the reviewed document, and why certain toxic effects may remain hidden for long periods before becoming clinically recognizable. Therefore, everyday chemical exposure is positioned as both a biological and social issue because its sources are often embedded in consumption practices, household environments, transportation, cosmetics, and urban air quality.

Based on this gap, the study was guided by three main questions. First, how can everyday chemical exposure be understood as a trigger of genetic damage that may develop into chronic disorders? Second, how do the liver and respiratory system emerge as dominant target organs in the reviewed document, and does this dominance reflect biological vulnerability or merely thematic prominence within the documentary data? Third, how can the nervous system and skin be understood as sites of hidden toxic accumulation, particularly following repeated low-dose exposure to heavy metals and cosmetic chemicals? These questions are important because the analysis should not merely identify affected organs, but should also distinguish thematic prominence in the document from biological vulnerability that requires further empirical examination.

Accordingly, this study aims to analyze patterns of everyday chemical toxicity integratively, from genetic damage to multiorgan effects. It employs document analysis of a core manuscript covering chemical carcinogenesis, genetic toxicology, hepatotoxicity, respiratory toxicity, neurotoxicity, and skin toxicity, supplemented by relevant scientific literature. This article is expected to make a conceptual contribution to the development of more integrative toxicological research, particularly by clarifying the relationships among daily exposure, biological mechanisms, target organs, and public health risks. However, because the study is based on document analysis, its findings should be interpreted as a conceptual synthesis rather than as causal evidence derived from primary data.

Method

This study employed a qualitative approach with a document analysis design. This design was selected because the primary data consisted of written materials containing conceptual and substantive discussions of chemical carcinogenesis, genetic toxicology, hepatotoxicity, respiratory toxicity, neurotoxicity, and skin toxicity. Operationally, the study was positioned as qualitative document analysis using a thematically oriented qualitative content analysis approach. This means that the documents were examined through content reading, coding, categorization, and theme development rather than through statistical measurement or empirical hypothesis testing. Document analysis was used to extract meaning and systematically develop an understanding of textual data, while qualitative content analysis was used to organize the document content into categories and themes relevant to the research objectives (Lyhne et al., 2025; Taylor et al., 2024).

The primary source of this study was one core academic manuscript discussing chemical toxicity from the genetic level to its effects on various target organs. The manuscript was purposively selected because it covered themes consistent with the focus of the article, namely the relationships among genotoxicity, exposure pathways, target organs, and hidden toxic accumulation. In addition to the core manuscript, the study used relevant scientific literature to strengthen the interpretation, compare the findings, and situate the analysis within current toxicological research. Therefore, the methodological position of this study was transparent. The main findings were developed from the analysis of the core document, while the supporting literature was used to enrich the discussion rather than serve as a new primary dataset.

The subjects of the study were not individuals or human participants, but the documents and units of meaning contained within them. The units of analysis included paragraphs, sentences, key terms, and substantive descriptions explaining types of toxicants, exposure routes, biological mechanisms, and affected target organs. Several content domains were identified from the core manuscript, including chemical carcinogenesis, genetic toxicology, hepatotoxicity, respiratory toxicity, neurotoxicity, and skin toxicity. These domains were then condensed into three main themes: genetic damage as an initial pathway to chronic disorders, the prominence of the liver and respiratory system as target organs in the document, and the nervous system and skin as sites of toxic accumulation that may not be immediately recognized.

The analytical procedure was conducted in several stages. The first stage involved a comprehensive reading of the core manuscript to obtain a general understanding of its content, structure, and direction of discussion. The second stage involved identifying units of meaning related to exposure sources, toxicity mechanisms, target organs, and biological effects. The third stage involved assigning initial codes to relevant sections of the text. The fourth stage involved grouping the codes into broader categories. The fifth stage involved formulating the main themes based on the relationships among categories. These stages followed the principles of qualitative content analysis, which emphasize systematic data reduction, categorization, abstraction, and interpretation of meaning (Lyhne et al., 2025; Nicmanis, 2024).

During the coding process, the researchers did not calculate theme frequencies as empirical quantitative data. Categorization was conducted to assist in developing thematic patterns and clarifying the structure of the findings. Therefore, terms such as dominant, strong, or prominent were interpreted as thematic tendencies within the document rather than as measures of empirical prevalence or proportion. Any reference to intensity or thematic mapping functioned only as an analytical aid to indicate the main focus of the document content and was not treated as a statistical measurement. This clarification was important to prevent the findings from being misinterpreted as quantitative results derived from primary data.

Data analysis was conducted inductively while maintaining a clear connection between the original text and the researchers' interpretations. After the initial codes had been collected, the data were reduced by combining similar codes and organizing them into more abstract categories. These categories were subsequently interpreted as three main themes corresponding to the research objectives. To maintain analytical traceability, each theme was reviewed against the context of the core document through repeated reading. This process ensured that the interpretations remained grounded in the original source and that the relationships among the data, categories, and themes could be academically justified (Nicmanis, 2024; Taylor et al., 2024).

The data were presented narratively and analytically. Each theme was reported as a main finding and then discussed by comparing it with relevant toxicological literature. The data were not presented using inferential statistics, but through thematic descriptions explaining the relationships among chemical exposure, biological mechanisms, toxicant entry routes, and target organs. Through this form of presentation, the study sought to develop a conceptual understanding of everyday chemical toxicity as a phenomenon operating across biological levels and organ systems.

This study has methodological limitations. Because it was based on document analysis of a single core manuscript, the findings were not intended to provide empirical generalizations, prevalence estimates, or evidence of causal relationships. The study also did not use primary laboratory data, biomarkers, exposure measurements, or population-based epidemiological data. Therefore, the findings are more appropriately understood as a conceptual synthesis useful for mapping the relationships among genotoxicity, target organs, exposure pathways, and the cumulative risks of everyday chemical exposure. Future research should employ biomonitoring designs, epidemiological studies, biomarker analyses, or exposome approaches to examine these relationships more empirically.

Results

Chemical Toxicity Begins with Genetic Damage and Progresses toward Chronic Disorders

The first finding indicates that chemical toxicity in the analyzed manuscript begins at the molecular level, particularly through damage to genetic material, before progressing to more severe clinical disorders. The sections on chemical carcinogenesis and genetic toxicology show that chemical exposure may alter DNA sequences, induce mutations, and promote abnormal cell formation through the stages of initiation, promotion, and progression. This indicates that toxic

effects are not always immediately visible, but may remain latent and only emerge after repeated or cumulative exposure over a long period.

In the analyzed document, genetic damage emerged as one of the central themes because it provides the basis for explaining how chemicals may contribute to chronic disease. Exposure to substances such as benzo[a]pyrene, aflatoxins, and other environmental chemicals was described as having the potential to disrupt genomic stability and trigger cellular changes that are not visible during the early stages. Therefore, the novelty of this finding lies in emphasizing that everyday chemical toxicity should be examined from the stage of cellular and genetic damage, rather than only after clinical symptoms or organ damage have become apparent.

The Liver and Respiratory System as Dominant Target Organs in the Reviewed Document

The second finding shows that the liver and respiratory system were the two most prominent target organs in the analyzed document. The liver was positioned as the principal organ involved in xenobiotic metabolism and detoxification, making it vulnerable to chemical compounds, toxic doses of medicines, and reactive metabolites. In the manuscript, liver toxicity was described as potentially progressing to steatosis, hepatitis, cirrhosis, and liver cancer. Meanwhile, the respiratory system was presented as a major entry route for various air pollutants, including carbon monoxide, nitrogen oxides, sulfur dioxide, hydrocarbons, and particulate matter generated by transportation and industrial activities.

However, the prominence of the liver and respiratory system in these findings should be interpreted cautiously. This prominence mainly indicates that these two organs appeared most frequently as thematic focuses in the reviewed document. It does not mean that they are always the most vulnerable organs for every type of toxicant. The biological vulnerability of an organ depends on the type of substance, dose, exposure duration, route of entry, metabolism, and the body's defense capacity. Therefore, this finding is more appropriately interpreted as a thematic tendency in which the liver and lungs occupy central positions in the manuscript, while also recognizing that both organs have a biologically high likelihood of exposure because of their roles in metabolism and gas exchange.

The Nervous System and Skin Show Risks of Toxic Accumulation That May Remain Unrecognized

The third finding indicates that the nervous system and skin are two biological areas associated with hidden toxic accumulation. In the nervous system, exposure to heavy metals such as lead and cadmium was described as potentially causing memory impairment, hyperactivity, sensory and motor disturbances, insomnia, and encephalopathy, particularly among vulnerable groups such as children. In the skin, chemicals such as mercury in cosmetics were described as cumulative toxicants that may be absorbed gradually and cause dermatitis, irritation, edema, and long-term systemic disorders.

This finding confirms that toxicity does not occur only through internal organs, but may also develop through external exposure routes that are often underestimated, such as cosmetic use or repeated dermal contact. In a social context, dermal exposure is important because it is related to everyday consumption practices, beauty standards, and access to products that may not be safe. Therefore, the nervous system and skin are important indicators of toxic risks that develop gradually, repeatedly, and are often recognized only at a later stage.

Overall, the three findings show that chemical toxicity in the analyzed manuscript forms an interconnected pattern consisting of chemical exposure, progressive biological disturbance, and disease manifestations in multiple organs. Exposure may occur through food, air, medicines, and cosmetics and may enter the body through oral, inhalation, and dermal routes. Toxic substances may then trigger genetic, metabolic, and physiological changes that may eventually develop into disorders of the liver, lungs, nervous system, and skin. These findings emphasize that everyday

toxic threats should be understood as progressive, cumulative, multisystem phenomena that are relevant to public health.

Discussion

Chemical Toxicity as a Trigger of Genetic Damage and the Early Development of Chronic Disease

The findings indicate that chemical toxicity does not stop at surface-level exposure, but may begin at the molecular level through DNA damage, mutations, disruption of genomic stability, and abnormal cell formation. This finding supports recent studies that position genotoxicity as an important pathway in the development of chronic disease, particularly when chemicals or reactive metabolites induce oxidative stress and interfere with DNA repair mechanisms. Casella and Ballaz (2024) emphasized that extremely small particles may disrupt proteostasis and mitochondrial function and induce oxidative stress associated with cellular damage. Zhang et al. (2022) also showed that environmental chemicals may interfere with DNA repair and potentially produce carcinogenic effects.

This finding strengthens the understanding that cancer and degenerative disorders do not always begin with visible clinical symptoms, but may originate from molecular disturbances that persist unnoticed for long periods. In the context of everyday exposure, this is important because people often perceive chemicals as hazardous only when they produce immediate symptoms. In reality, some exposures may act slowly, repeatedly, and cumulatively. Picinini-Zambelli et al. (2025) showed that various emerging pollutants in aquatic environments have genotoxic potential and should be understood as long-term biological threats. Therefore, the contribution of this discussion lies in positioning genotoxicity as the beginning of a broader toxic pathway rather than as an isolated biological event.

However, claims regarding genetic damage must be interpreted proportionally. This article did not use primary biomarker data, laboratory measurements, or epidemiological analysis. Therefore, the relationship between chemical exposure and DNA damage presented in this manuscript represents a conceptual synthesis based on documentary evidence and supporting literature. Accordingly, the finding is more appropriately positioned as an initial mapping of possible biological pathways rather than as direct causal evidence.

The Liver and Respiratory System: Prominent in the Reviewed Document and Biologically Relevant

The findings show that the liver and respiratory system were the most prominent target organs in the analyzed document. However, it is important to clearly distinguish between prominence in the reviewed data and biological vulnerability. Prominence in the reviewed data means that the liver and respiratory system appeared most frequently as the focus of discussion in the manuscript. Biological vulnerability, in contrast, means that these organs are physiologically more likely to be affected because of their roles in metabolism, detoxification, and direct contact with airborne pollutants.

The thematic prominence of the liver has a strong biological basis. The liver is the primary organ responsible for xenobiotic metabolism, meaning that many chemical compounds are processed through it. Wang et al. (2025) showed that xenobiotics may cause liver injury through enzymatic bioactivation, oxidative stress, DNA damage, inflammation, and immune dysfunction. Yilmaz et al. (2025) also emphasized that emerging environmental pollutants, including microplastics and nanoplastics, may contribute to liver injury and metabolic dysfunction. Therefore, the liver occupies a paradoxical position. It protects the body from foreign substances, yet this protective role also makes it particularly vulnerable to toxic exposure.

The prominence of the respiratory system may also be explained by the fact that the lungs are a major entry point for numerous airborne pollutants. Hamanaka and Mutlu (2025) explained

that particulate matter is associated with various respiratory diseases through inflammation, oxidative stress, and lung tissue injury. Gross et al. (2025), through a systematic review and meta-analysis, showed that long-term exposure to ambient air pollution has adverse effects on adult lung function. Therefore, the lungs are not only an entry route for pollutants, but also an initial site of interaction between environmental particles and biological tissues.

Nevertheless, the liver and lungs should not be concluded to be the most vulnerable organs for all toxicants. Under certain types of exposure, the kidneys, reproductive system, endocrine system, immune system, or nervous system may become the primary targets. Therefore, the more accurate conclusion is that the liver and respiratory system are prominent in the reviewed document and have a strong biological basis for vulnerability, but their susceptibility still depends on the type of toxicant, exposure route, dose, duration, and individual characteristics.

The Nervous System and Skin as Sites of Hidden Toxic Accumulation

The findings indicate that the nervous system and skin are important areas for understanding hidden toxic risks. In the nervous system, exposure to heavy metals such as lead, cadmium, and mercury may induce neurotoxicity through oxidative stress, mitochondrial dysfunction, enzyme disruption, DNA damage, and altered gene regulation. Jomova et al. (2025) explained that heavy metals have extensive effects on human health, including neurological disorders. Yu et al. (2024) also emphasized that heavy metals may induce neurotoxicity through epigenetic mechanisms and disturbances in gene expression.

This discussion is important because neurotoxicity does not always develop rapidly or become easily recognizable. Repeated exposure to low doses of heavy metals may progress gradually, particularly among vulnerable populations such as children, pregnant women, informal workers, and communities living in polluted environments. Therefore, risks to the nervous system should be considered not only in the context of intensive industrial exposure, but also in relation to environmental exposure, consumption, and everyday products containing contaminants.

The findings regarding skin toxicity indicate that dermal exposure may serve as an important route for toxic substances to enter the body. This can be observed in the use of mercury-containing skin-lightening cosmetics. Bastiansz et al. (2022) showed that mercury continues to be detected in skin-lightening products across several countries and is associated with dermal, neurological, and renal effects. Cadungog et al. (2025) also emphasized that skin-lightening cosmetics sold online continue to pose serious health risks despite the existence of international regulatory limits.

Therefore, dermatotoxicity should not be reduced to skin irritation alone. Repeated dermal exposure may provide an entry route for systemic disorders, particularly when hazardous substances are used over long periods. In a social context, this issue is related to the normalization of cosmetic use, pressure arising from beauty standards, and limited product safety literacy. Therefore, skin toxicity should be understood within a multiorgan exposure network rather than as an isolated cosmetic issue.

Everyday Toxicity within the Exposome and Cumulative Exposure Framework

Conceptually, the findings support the need to understand toxicity through the exposome framework, which refers to the totality of exposures experienced by an individual throughout life. Everyday chemical exposure does not occur through a single route, but through multiple sources such as food, air, medicines, cosmetics, workplaces, household environments, and consumer products. The exposome approach helps explain that health risks are not determined by one substance alone, but by combinations of repeated and cumulative exposures that interact with biological and social factors.

This framework is important because low-dose exposures are often considered harmless when evaluated separately. However, repeated exposure through different routes may create a

cumulative toxic burden. Wan et al. (2025) emphasized that the exposome approach is useful for understanding relationships between environmental exposure and biological responses more comprehensively. Therefore, this article does not only discuss toxicity according to target organs, but also directs attention to the total exposure burden experienced in everyday life.

The implication is that future toxicological research should move from single-substance or single-organ studies toward multiroute, multiorgan, and multibiomarker approaches. Further studies should develop biomonitoring, exposure biomarkers, early-effect biomarkers, and analyses of vulnerable populations. Such approaches are important for examining the relationship between low-dose daily exposure and multiorgan damage more empirically and for generating evidence that is more relevant to public health policy.

Research Limitations

This study has several limitations. First, the findings were developed through document analysis and thematic synthesis and were not supported by primary laboratory data, biomarkers, or epidemiological analyses that could establish strong causal relationships. Second, because the primary source consisted of one core manuscript, the findings depended heavily on the scope, depth, and thematic tendencies of that document. Third, the thematic categories used in the study functioned as analytical aids rather than as empirical quantitative measures. Therefore, the findings cannot be interpreted as estimates of prevalence, risk proportions, or biological effect sizes.

Fourth, the discussion of target-organ prominence should be interpreted as prominence within the reviewed document rather than as evidence that these organs are always the most biologically vulnerable under every exposure condition. Fifth, the study did not systematically compare the strength of evidence across studies, as would be conducted in a systematic review or meta-analysis. Nevertheless, the principal strength of the study lies in its conceptual mapping of genotoxicity, exposure pathways, target organs, and hidden toxic accumulation within an integrative framework.

Conclusion

This study shows that everyday chemical toxicity is a progressive biological phenomenon that begins with genetic damage and may eventually manifest across multiple organs. Three main findings were identified: the role of genotoxicity as an early pathway to chronic disorders, the prominence of the liver and respiratory system as target organs in the reviewed document, and the importance of the nervous system and skin as sites of hidden toxic accumulation.

These findings emphasize that everyday chemical exposure should no longer be viewed as a simple risk because its effects may progress from molecular disturbances to systemic consequences. However, conclusions regarding target-organ prominence should be interpreted cautiously. The liver and respiratory system were prominent in the analyzed document and have a biological basis as organs actively involved in metabolism and direct contact with pollutants. Nevertheless, organ vulnerability still depends on the toxicant type, dose, duration, exposure route, and individual characteristics.

The principal strength of this study lies in its ability to provide a conceptual synthesis of the relationships among chemical exposure, biological mechanisms, exposure routes, and multiorgan effects within a unified analytical framework. However, the study is limited because it was based on document analysis of one core manuscript and was not supported by primary data, biomarkers, or epidemiological measurements. Therefore, the findings should be interpreted as conceptual mapping rather than causal evidence or quantitative estimates.

Future research should focus on empirical studies using biomonitoring, biomarker analysis, epidemiological research, and exposome approaches to examine the relationships among chemical exposure, duration, dose, route of entry, and biological damage more precisely.

Vulnerable groups such as children, women using cosmetic products, workers exposed to chemicals, and communities living in highly polluted areas should receive particular attention. Through this direction, toxicological research may progress from conceptual synthesis toward a stronger scientific basis for preventing health risks associated with everyday chemical exposure.

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Research Ethics Statement
This study was conducted in accordance with the ethical principles of scientific research, including academic honesty, transparency, objectivity, scientific responsibility, and the responsible use of published sources. Since the study employed qualitative document analysis and did not directly involve human participants, primary data collection, clinical intervention, biological specimen collection, or personally identifiable information, informed consent and formal ethical approval were not required. Nevertheless, the authors maintained academic integrity, accurately represented the analyzed documents and supporting literature, properly acknowledged all cited sources, and clearly distinguished conceptual interpretations from primary empirical evidence throughout the research and reporting process.

Author Contributions
Tiara Putri Maharani: conceptualization, identification and collection of documentary sources, data curation, qualitative content analysis, thematic coding, conceptual synthesis, interpretation of findings, writing of the original draft, manuscript revision, and preparation of the final version. Ahmad Irawan: methodology, validation of the analytical framework, academic supervision, substantive review, critical interpretation of the findings, manuscript review and editing, and approval of the final version.
All authors have read, reviewed, and approved the final version of the manuscript.

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

Artificial Intelligence Use Statement
The authors declare that artificial intelligence, if used, was employed only as a limited technical support tool for language editing, sentence refinement, grammar checking, translation assistance, and improving manuscript readability. All processes involving document selection, qualitative coding, thematic analysis, conceptual synthesis, scientific interpretation, academic argumentation, and conclusion development remain the full responsibility of the authors.

Data Availability Statement

The materials supporting the findings of this study consist of the core academic document, published scientific articles, official health information, analytical notes, coding records, thematic classifications, and conceptual synthesis materials related to genotoxicity, exposure pathways, target organs, and the multiorgan effects of everyday chemical exposure. Since this study was based on document analysis, no new primary dataset involving human participants or laboratory measurements was generated. Additional information regarding the reviewed materials and analytical procedures may be obtained from the corresponding author upon reasonable request.

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