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Perspective

Advancing Health Risk Assessment: Integrating Exposure Routes and Bioavailability to Quantify Internal Dose

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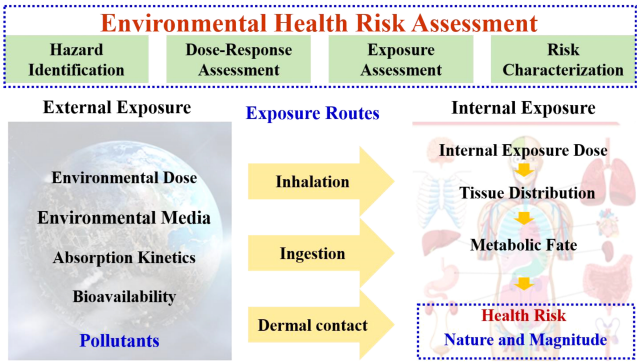
Highlights

- Exposure routes influence the internal dose, distribution, and metabolism of pollutants.
- Aggregate exposure assessment calls for weight-based integration of multiple routes.
- Probabilistic parameters and bioavailability data improve the accuracy of risk assessment

Abstract: As the linkage between environmental pollution and health outcomes, exposure routes characterize how pollutants enter the human body, constituting the foundation of health risk assessment. Pollutants primarily enter body through three major routes: ingestion, inhalation, and dermal contact. The exposure routes govern the internal dose, tissue distribution, and metabolic fate of various environmental pollutants, fundamentally shaping the nature and magnitude of associate health risks. From the perspective of exposure routes, current risk assessment models exist several limitations, including the lack of systematic integration across multiple exposure routes; reliance on fixed default exposure parameters that fail to reflect population heterogeneity; dependence on external exposure dose such as daily intake, without accounting for bioavailability; and omission of special exposure routes. Therefore, modern health risk assessment frameworks must evolve to incorporate: integrated multi-route exposure assessments, probabilistic parameter distributions, and bioavailability-corrected effective dose. Only through such comprehensive improvements can achieve accurate characterization of exposure risks and provide a robust scientific basis for precision prevention and control.



1. Introduction

The environment, serving as the external determinant of human health, exerts substantial influence on population health outcomes. However, rapid industrialization and urbanization have led to widespread releases of various pollutants into environmental media, resulting in severe public health challenges of global concern. Pollution is responsible for approximately 9

million premature deaths annually, accounting for nearly one-sixth of all deaths worldwide [1]. The 2021 Global Burden of Disease Study further confirms this burden, estimating about 19% of global mortality can be attributed to environmental risk factors [2]. However, adverse health effects from environmental pollution may occur only when exposure takes place. As emphasized in the U.S. Environmental Protection Agency (EPA) risk assessment guidelines, a comprehensive health risk evaluation must include exposure assessment to determine whether pollutants can reach human receptors via specific routes.

In essence, exposure routes, which determining whether and how pollutants reach the body, serve as the critical bridge that transforms environmental pollution into biologically effective internal doses. Understanding the mechanisms and contributions of exposure routes is fundamental to accurate health risk assessment. This perspective mainly examines how exposure routes influence the health risks of pollutants, reviews current limitations in existing risk assessment frameworks, and proposes strategies to refine and improve risk assessment models.

2. Basic Classification of Exposure Routes

Pollutants primarily enter the human body through three major exposure routes: inhalation, ingestion, and dermal contact (Figure 1). Inhalation occurs when pollutants are drawn into the lungs during breathing, making this route particularly relevant for volatile organic compounds (VOCs), fine particulate matter (PM), and hazardous gases [3]. Ingestion involves the intake of pollutants via contaminated drinking water or food. The ingestion route is especially significant for heavy metals (e.g., lead, cadmium) and persistent organic pollutants (POPs), which can easily accumulate in biological tissues and magnify through the food chain [4]. Dermal contact allows pollutants to penetrate the skin and enter systemic circulation, especially lipophilic substances such as polycyclic aromatic hydrocarbons (PAHs) and phthalates [5]. Beyond these primary routes, certain special exposure routes warrant attention in specific populations or scenarios. Maternal-offspring transmission, occurring via placental transfer during pregnancy or through breastfeeding, is a critical indirect exposure route that enables the intergenerational transfer of toxicants such as methylmercury and per- and polyfluoroalkyl substances (PFAS), and pathogens like hepatitis B virus (HBV) [6]. This route is of particular relevance in transgenerational risk assessment. Additionally, ocular exposure, though often overlooked, serves as a supplementary route for various pollutants in industrial, agricultural, or occupational scenarios. While its contribution to systemic dose accumulation is generally limited, localized ocular exposure can lead to severe acute injury, inflammation, or long-term damage, highlighting its importance in specific scenarios. Within the classic four-step risk assessment framework, a complete exposure pathway, encompassing the full chain from pollutant source to human contact, is the prerequisite for health risk occurrence. Accurate dominant exposure route identification is therefore essential for both hazard characterization and risk assessments. The route through which a pollutant enters the human body is jointly determined by its physicochemical properties and the environmental matrix in which it resides. Specifically, the environmental matrix acts as a carrier that defines how and where human exposure occurs, while the physicochemical properties govern its speciation, mobility, and bioaccessibility in that matrix, as well as its capacity to cross biological barriers.

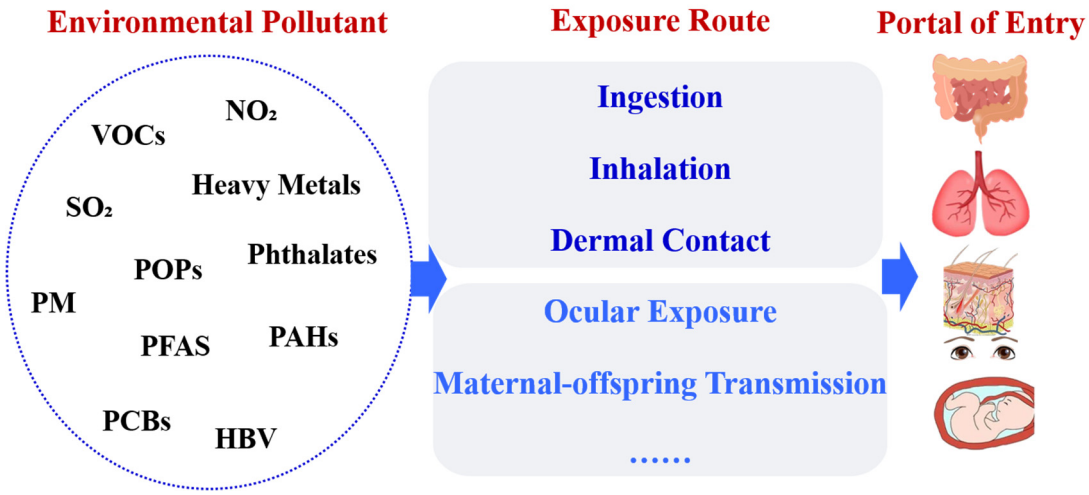


Figure 1. Overview of pollutant exposure routes, including major and special routes and their corresponding portals of entry.

3. Why Exposure Routes Critical

Different exposure routes directly influence the bioavailability and absorption kinetics of pollutants, thereby profoundly shaping the internal dose. In general, inhalation exhibits the highest absorption rate, due to the large alveolar surface area and extremely thin air-blood

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barrier, enabling rapid transfer of pollutants into systemic circulation [7]. Ingestion primarily relies on passive diffusion across the intestinal epithelium, although absorption can be modulated by factors such as gastrointestinal pH, transit time, and microbial metabolism. In contrast, dermal contact typically exhibits the lowest absorption rate, largely owing to the protective function of stratum corneum [8]. The environmental matrix in which pollutants reside determines their primary exposure route, which in turn significantly influences its bioavailability. Thus, the bioavailability of pollutants may span 1–2 orders of magnitude across different exposure routes, even at identical environmental concentrations. For example, a murine study demonstrated route-dependent bioavailability of PFAS: approximately 73–98% via ingestion, 34–67% via inhalation, and 5–8% through dermal exposure [9]. Moreover, in global daily intake of synthetic musks, dermal exposure accounts for the largest share (92.66% of total intake), while inhalation and oral ingestion contribute 3.77% and 4.03%, respectively [10]. These substantial differences underscore that route-specific variations in bioavailability and absorption kinetics fundamentally shape the internal dose of environmental pollutants.

The exposure route is also a key determinant of pollutant biodistribution and target organ specificity. Although most pollutants eventually reach systemic circulation, their initial distribution varies significantly depending on the entry route. Inhaled pollutants first enter the pulmonary circulation; ingested pollutants are transported via the digestive system to the liver; and dermally absorbed pollutants initially accumulate in local skin tissues [11]. These differences in distribution contribute to distinct patterns of primary target organs: respiratory system for inhalation, liver for oral ingestion, and skin for dermal contact. Unlike orally ingested pollutants, which undergo hepatic first-pass metabolism, inhaled and dermally absorbed pollutants can bypass the liver and its detoxification processes. This allows them to enter systemic circulation directly, where they may bind to serum proteins or rapidly distribute to sensitive organs, including the brain and heart, potentially leading to neurotoxic or cardiotoxic effects [12]. Moreover, the toxic effects vary significantly across different target organs, reflecting route-specific mechanisms of damage. For example, PAHs via inhalation primarily induce oxidative stress and inflammatory responses in respiratory epithelial cells, and lead to persistent cough, headaches, dyspnea, and even lung cancer under chronic exposure. Following ingestion, PAHs are associated with an increased risk of gastric, colorectal, and liver cancers. Dermal exposure allows PAHs to accumulate in the skin, forming a repository, where cutaneous enzymes can bioactivate them into carcinogenic metabolites [11]. Moreover, PAHs can reach the cardiovascular system either directly through inhalation or via systemic circulation, contributing to hypertension, heart rate variability, and ischemic heart disease.

Metabolism is a pivotal process that determines whether a pollutant is detoxified or bioactivated. The liver is the primary site of pollutant metabolism, but significant metabolic activity also occurs in extrahepatic target tissues, such as the respiratory tract, gastrointestinal tract, and skin, highlighting marked exposure route-specific differences. Phase II enzymes, including glutathione S-transferases (GSTs), UDP-glucuronosyltransferases (UGTs), sulfotransferases (SULTs), and epoxide hydrolases (EHs), are widely expressed across multiple tissues and primarily mediate basic detoxification function [13]. In contrast, certain isoforms of Phase I metabolic enzymes, such as cytochrome P450 enzymes (CYPs), exhibit pronounced tissue-specific expression patterns [14]. Although the liver exhibits the most abundant and diverse of CYP enzymes, peripheral tissues also express multiple CYP subtypes. For instance, CYP2A13 and CYP2F1 show particularly high expression specificity in the respiratory system, while CYP3A4 and CYP3A5 are abundantly present in the digestive system [15]. CYPs are capable of metabolically activating inhaled procarcinogens such as PAHs, tobacco-derived N-nitrosamines, and 1,1-dichloroethylene into reactive electrophiles that form DNA adducts, thereby increasing the risk of pulmonary carcinogenesis [16]. The skin, though less metabolically active, contains enzymes such as esterases and aryl hydrocarbon hydroxylases that can perform preliminary transformations of certain pollutants. Notably, due to differences in enzyme distribution and target organ, route-specific metabolic systems can convert the same pollutant into distinct metabolites, leading to distinct toxicological profiles. For instance, the synthetic tonalide undergoes photochemical reactions with amino acids on the skin surface, forming imine byproducts that act as potential sensitizers, mediating skin-specific immunotoxicity [10].

Exposure routes serve as a critical bridge between external exposure and internal dose, fundamentally shaping the nature and magnitude of health risks by modulating the internal dose, tissue distribution, and metabolic fate of pollutants. The same pollutant can elicit markedly different health effects depending on different exposure routes [17]. Therefore, risk assessment must adopt a route-specific perspective, to accurately identify the dominant exposure routes and their relative contributions, thereby advancing the scientific accuracy of risk assessment and the effectiveness of risk management strategies.

4. Limitations of Current Risk Assessment: A Perspective from Exposure Routes

Current risk assessment frameworks, while well-established in structure, exhibit significant limitations when reviewed through the lens of exposure routes. First,

although pollutants typically exert adverse effects through multiple concurrent exposure routes, current risk assessment models often lack integrated analysis of multi-route exposure. Conventional approaches rely on single-route assessments or simple linear summation across multiple routes [18]. However, such additive models overlook synergistic interactions among exposure routes, the cumulative burden on target organs, and the barrier effects of first-pass metabolism. Failure to integrate multi-route exposures can result in underestimation of the internal dose and health risks, particularly in cases of chronic, low-level, and multi-route co-exposure.

Second, the most common approach in research is to adopt the standard exposure assessment models developed by the U.S. Environmental Protection Agency (EPA), with modifications based on the physicochemical properties of specific pollutants and site-specific exposure parameters [19]. However, these widely used models typically rely on daily intake (DI) as a proxy for exposure dose, rather than biologically effective dose, the concentration of pollutant actually absorbed and elicited toxic effects [20]. Relying on DI without relative bioavailability (RBA) correction effectively assumes all environmental pollutant mass as equally bioavailable, which is a fundamental oversimplification that can lead to orders-of-magnitude errors in risk assessment. The environmental matrix in which a pollutant resides significantly influences its bioavailability; for instance, soil can markedly reduce the bioaccessibility of arsenic by binding it to organic matter [21]. Moreover, key physicochemical properties also strongly influence the uptake of pollutants. For example, dietary lipids can significantly enhance the bioavailability of highly lipophilic organic pollutants (e.g., polychlorinated biphenyls), whereas hydrophilic metals (e.g., arsenic, cadmium) depend on specific ion transporters for intestinal absorption and exhibit minimal dermal penetration [22]. In inhalation, insoluble particles may be removed via mucociliary clearance, while water-soluble gases (e.g., SO_2) are rapidly absorbed in the upper airways [23]. Ignoring these chemical- and route-specific dynamics introduces systematic biases in internal dose estimation, potentially leading to either over- or underestimation of health risks. Given these limitations, relying solely on DI without accounting for matrix- and route-specific RBA [24] can lead to inaccurate risk characterization. Although bioavailability remains poorly characterized for many pollutants, several studies have begun incorporating bioavailability data derived from in vitro gastrointestinal simulation assays to refine dose estimates for ingested pollutants, in recent years [25]. Nevertheless, the bioavailability of pollutants via inhalation and dermal contact is also significantly modulated by physiological barriers and cumulative exposure effects. To date, RBA correction factors for these routes remain underdeveloped and lack broad empirical validation across diverse populations and exposure scenarios.

Third, risk assessment practices often oversimplify or standardize exposure routes, relying on default parameters and generic models. These approaches typically adopt the "average adult" model and use uniform physiological parameters (respiratory rate, water intake, soil ingestion rate, skin area to weight ratio), failing to account for the inter-individual variability of exposed populations. Especially for sensitive populations, including children, pregnant women, and older adult, "average adult" model tends to underestimate their susceptibility to pollutants. For instance, children are more likely to ingest pollutants adhered to dust due to their frequent hand-to-mouth activity. Older individuals experience increased dermal permeability of pollutants due to age-related degradation of the skin barrier. Moreover, children generally experience higher internal doses than adults under comparable environmental conditions, owing to their higher inhalation rate, water intake, and food consumption per unit body weight compared to adults [26]. The 2025 blood lead incident at Shihe Peixin Kindergarten in Tianshui City clearly demonstrates this difference. The abnormal blood lead ratio was 82.4% among staff members, and as high as 98.4% among children, highlighting the physiological susceptibility of children [27].

Finally, conventional risk assessments primarily focus on the three classic exposure routes, while often excluding special exposure routes such as maternal-offspring transmission and ocular exposure. However, these special routes exhibit higher absorption and heightened sensitivity to pollutants. Prenatal or early-life exposure may lead to amplified toxic effects, and the eyes are particularly vulnerable owing to their delicate structure and rich vascularization. Unfortunately, current assessment frameworks lack standardized methods and key parameters to evaluate these routes, including placental transfer rates, milk-to-plasma concentration ratios, and ocular absorption fractions. Although these special exposure routes are not commonly encountered in general assessments, their potential health impacts should not be overlooked, especially in vulnerable subgroups.

Exposure routes play a critical role in influencing the health risks of environmental pollutants. However, current risk assessment models exhibit several limitations when reviewed from an exposure route perspective, as shown in Figure 2. These include: (1) the lack of systematic integration of multiple exposure routes; (2) the use of fixed, default exposure parameters that fail to capture population heterogeneity; (3) the reliance on external exposure metrics such as daily intake (DI), without accounting for bioavailability; and (4) the frequent omission of special exposure routes. Failure to fully incorporate these limitations undermines the scientific rigor and accuracy of risk characterization.

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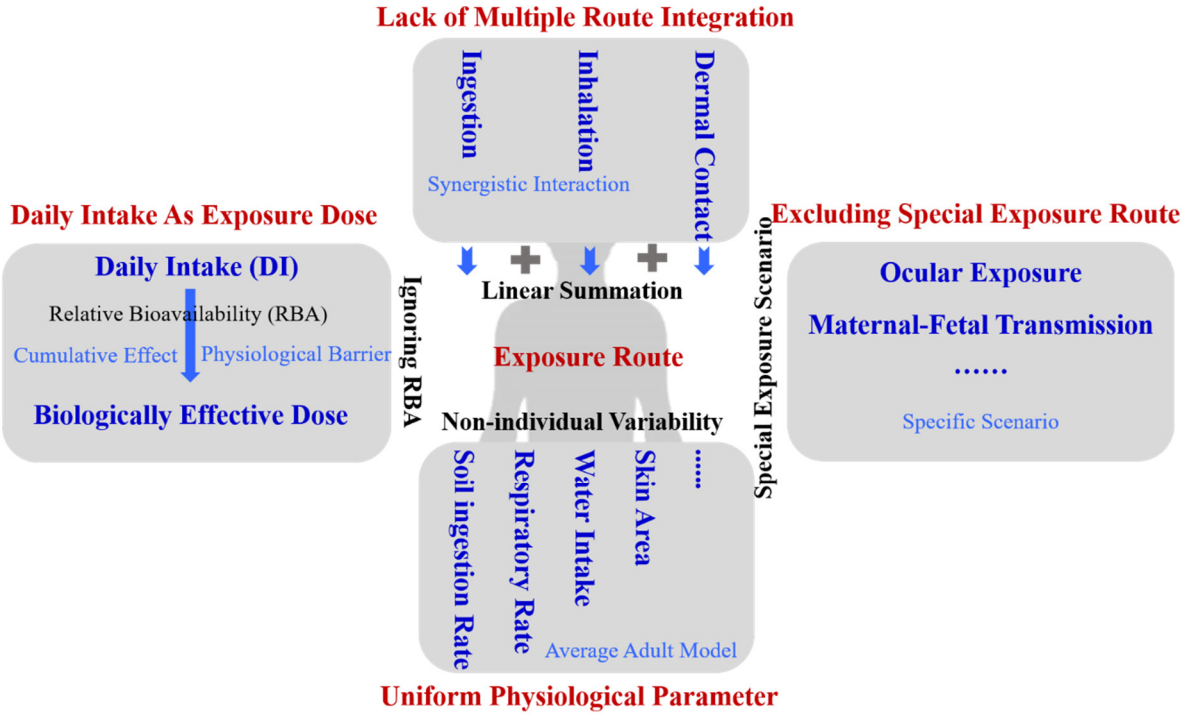

Figure 2. Limitations of current risk assessment models from exposure route perspective.

5. Recommendations for the Development of Risk Assessment

Although the current environmental health risk assessment framework has become standardized, exemplified by the widely adopted four-step approach, it is increasingly revealing limitations in addressing the complex exposure scenarios. Future advancements must drive the model toward more refined and individualized approaches. Building on this foundation, Figure 3 summarizes key recommendations for advancing future risk assessment research.

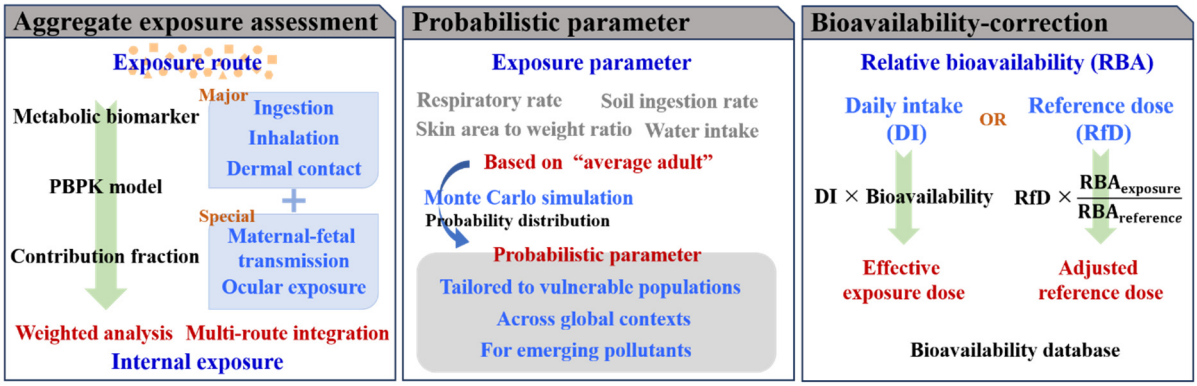

Figure 3. Recommendations for advancing risk assessment.

The pollutants typically exert adverse effects through multiple concurrent exposure routes, thus there is an urgent need of "aggregate exposure assessment" that integrates doses across all relevant routes. When entering the body via different exposure routes, pollutants undergo divergent first-pass effects, resulting in route-specific metabolic profiles. Through advanced correlation analysis, route-specific metabolic biomarkers can be identified to distinguish the biological signatures of different exposure routes [28]. These biomarkers combined with physiologically based pharmacokinetic (PBPK) models, which simulate the absorption, distribution, metabolism, and excretion of pollutants in the human body, allow for the quantitation of contribution fraction of each exposure route and facilitate a weight-based integration of multiple exposure routes. Furthermore, in specific exposure scenarios, special exposure routes may become dominant routes. Neglecting these routes may create blind spots in risk assessment. It is therefore essential to incorporate these

routes into the aggregate exposure assessment. This aggregate exposure assessment not only identify the dominant routes of pollutant exposure but also simulate the dynamics of internal dose, thereby advancing the development of precision risk assessment.

Additionally, to address the limitation of relying on fixed default parameters based on the "average adult", it is recommended to adopt probabilistic methods such as Monte Carlo simulation. By assigning probability distributions to all input parameters, these methods generate a probabilistic distribution of risk estimates, thereby quantifying uncertainty and variability in exposure assessments [29]. The probabilistic distribution parameters are drawn from databases, such as the Exposure Factors Handbook (EFH). However, databases limitations, such as insufficient population representativeness and missing key parameters, directly introduce uncertainties into the final risk assessment, compromising its accuracy [30]. Thus, there is a need to develop exposure parameter handbooks tailored to vulnerable populations and global contexts, and to routinely incorporate their distinct exposure patterns and heightened susceptibility into standard risk assessment practices. This is especially critical for emerging pollutants, which despite typically occurring at trace levels in environmental matrices, pose potential health risks due to their persistence, bioaccumulation, or endocrine-disrupting properties, thereby necessitating more refined and route-specific exposure parameters [31].

To address the common practice of using intake metrics as proxies for actual exposure dose, it is essential to establish an exposure route-specific bioavailability database that systematically compiles the bioaccessibility and bioavailability data of various pollutants across different environmental media. During the exposure assessment phase, RBA data can be used to adjust intake doses to effective doses, the fraction of pollutants that is actually absorbed and biologically active. In the dose-response assessment phase, incorporating RBA into the derivation of toxicity reference dose (RfD) aligns risk characterization more closely with actual physiological responses. The bioavailability of pollutants is governed by multiple factors, including chemical speciation, environmental matrix characteristics, and individual physiological conditions. Thus, establishing a comprehensive bioavailability database would significantly improve the accuracy of risk assessments.

The current risk assessment paradigm centered on pollutants, reliant on default parameters, and based on linear summation across exposure routes, is inadequate for accurately reflecting complex exposure scenarios. Future risk assessment models must evolve toward greater precision, individualization, and integration. Key advancements should include: integrated multi-route exposure assessment, probabilistic parameter distributions that account for population heterogeneity, and bioavailability-corrected effective dose estimation. Such comprehensive developments will enable accurate characterization of exposure risks and provide a scientific foundation for precision prevention and control.

Author Contributions

Y.Z.: Writing—original draft preparation, investigation, conceptualization; G.L.: Writing—review and editing, conceptualization; J.G.: Writing—review and editing; T.A.: Writing—review and editing, conceptualization, supervision, funding acquisition. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

This study did not involve human participants or experimental animals.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable. No original data were generated in this study.

Conflicts of Interest

The authors declare that they have no conflicts of interest in this work. Given the role as Editor-in-Chief, Taicheng An had no involvement in the peer review of this paper and had no access to information regarding its peer-review process. Full responsibility for the editorial process of this paper was delegated to another editor of the journal.

Use of AI and AI-Assisted Technologies

No AI tools were utilized for this paper.

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