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Research Article

Human biomonitoring evidence of microplastics: What do we really know about internal exposure and health risk?

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ABSTRACT

The environmental prevalence of microplastics (MPs) and nanoplastics (NPs) is a growing concern due to their potential for human exposure. Human biomonitoring (HBM) studies have detected MPs in biological samples such as blood, feces, placenta, and respiratory tissues; however, significant uncertainty remains regarding their associated health risks. This systematic review combines bibliometric analysis and content synthesis of 197 Scopus-indexed articles (2015–2025) to critically assess the evidence on internal exposure and health risk. Although MPs are frequently measured in the surrounding environment, including air, water, food, and various tissues, the biologically relevant internal dose (i.e., the fraction that undergoes absorption, systemic distribution, and retention in target organs) remains largely unknown. Ingestion and inhalation are the primary routes of exposure; however, the mere presence of MPs in biological matrices does not indicate a quantifiable internal dose. Moreover, no study has demonstrated a direct causal relationship between MP detection and clinically adverse health effects in humans. Existing evidence is limited by methodological heterogeneity, the absence of standardized dose metrics, and a heavy reliance on animal studies, where exposure levels often exceed realistic human conditions by several orders of magnitude. Therefore, current evidence does not support a causal link between internally accumulated microplastics and adverse health outcomes in humans. Future research should prioritize harmonized approaches to internal dose assessment, biokinetic modeling, and well-designed epidemiological studies that directly link biomonitoring data to clinically relevant health outcomes. It is important to clarify that the detection of MPs in human matrices including blood, placenta, and feces is not in question; rather, the critical gap lies in translating these detection data into a quantifiable internal dose and establishing causal links with adverse health outcomes.

1. Introduction

The rapid increase in plastic production and consumption over the last few decades has caused extensive environmental pollution by microplastics (MPs) and nanoplastics (NPs). As shown in Fig. 1a, a significant source of secondary microplastics originates from fragmented, mismanaged plastic waste, which is found in terrestrial, freshwater, and marine environments. Once released, microplastics are transported to and retained in environmental sinks, such as soils and sediments (see Fig. 1b), where their high persistence makes them very difficult to remove [1–3]. The presence of microplastics in various environmental compartments has become a growing concern, as it serves as a medium for continuous human exposure through the food chain, drinking water, and air [4,5] (see Fig. 2).

The expanding contamination of ecosystems has led to increasing documentation of microplastics in biological systems, including those relevant to human exposure. The presence of microplastics in seafood, consumable organisms, and items intended to come into contact with food (see Fig. 1c) indicates ingestion as a primary route of exposure [6]. Recent human biomonitoring (HBM) studies have identified

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microplastics in several biological matrices and demonstrated that at least some particles can cross biological barriers (see Fig. 1d), including feces, blood, lung fluids, urine, and placenta [7–9]. In vivo and *in vitro* studies have reported that exposure to microplastics induces oxidative stress, inflammatory responses, and metabolic disturbances [10,11].

Research indicates a substantial gap between the environmental-to-human contamination continuum illustrated in Fig. 1 and the understanding of biologically significant internal exposure and associated health risks, despite advances in this field. A large number of HBM studies provide evidence of particle presence; however, most do not assess internal dose, residence time, or the systemic distribution of these particles. Furthermore, methodological heterogeneity across studies leads to inconsistent reporting and complicates risk assessment [12–14]. Consequently, relatively few studies demonstrate clinically meaningful correlations between microplastic exposure and human health outcomes. Due to this limited evidence, it remains unclear whether microplastics represent transient exposure that can be effectively cleared by the body or whether it accumulates and impose a significant biological burden.

The present study aims to systematically evaluate the existing literature on HBM of microplastics within the framework of the environment-to-human exposure model shown in Fig. 1, using a combined bibliometric and content analysis approach. This study seeks to characterize and evaluate research streams in HBM microplastic literature, including biological matrices, microplastic characteristics, and exposure pathways, while distinguishing between mere detection and biologically incorporated dose. Furthermore, this review aims to clarify what is currently understood about internal exposure and associated health risks, identify key methodological gaps, and define priorities for future research to support improved human health risk assessments of microplastics. Unlike traditional narrative reviews, this study integrates bibliometric mapping with thematic content analysis to provide a structured and reproducible synthesis of the literature, thereby offering a robust foundation for identifying knowledge gaps and guiding future research priorities.

2. Data acquisition and methodology

This study explores HBM of microplastics through a systematic review integrating bibliometric analysis and content-based synthesis to provide a comprehensive overview of current research on internal exposure pathways, detection methods (including exposure assessment methodologies), and potential health risks. It also aims to identify gaps in the literature, emerging research trends, and directions for strengthening the scientific basis for future policy development. The bibliometric procedure comprised four consecutive stages: formulation of search criteria, document screening, bibliometric processing, and content-based interpretation, with a specific focus on HBM evidence, internal exposure, and the health risk implications of microplastics. A structured literature search was conducted in the Scopus database using the query: **TITLE-ABS-KEY (microplastic)***, combined with health- and exposure-related terms including "human health," "HBM," "internal exposure," "bioaccumulation," "toxicological effects," "ingestion," and "inhalation." The search covered publications from database inception to 11 November 2025, yielding 10,307 documents. After restricting the dataset to the 2015–2025 period, final-stage publications, and research articles only, a total of 8350 records were retained. Subsequently, 1957 documents were excluded based on thematic irrelevance, including studies focused exclusively on environmental distribution, ecotoxicology in non-human species, materials science, water and wastewater treatment engineering, modeling approaches, and non-health-related reviews. This screening process resulted in 197 articles meeting the final inclusion criteria. The bibliometric analysis included publication characteristics, international collaboration and co-citation networks, keyword co-occurrence patterns, and the geographical distribution of research activity. These quantitative analyses were complemented by a content-based synthesis that critically evaluated: (i) HBM matrices and analytical approaches, (ii) evidence of internal exposure to microplastics, (iii) toxicological and mechanistic findings relevant to human health risk, (iv) limitations in the exposure–dose–effect relationship, and (v) major research gaps. The results were further interpreted in the context of public health relevance and alignment with Sustainable Development Goals (SDG) 3 (Good Health and Well-being) and SDG 6

Fig. 1. Environmental-to-human continuum of microplastics contamination.

(Clean Water and Sanitation).

3. Results and discussion

3.1. Annual trends in microplastics research relevant to HBM and health risk

Based on the reporting activity of leading countries, the duration of publication records, and the countries involved, the growth of scientific literature on microplastics and human health over time is illustrated through the five most active countries from 2015 to 2022: the United Kingdom, China, Germany, India, and the United States. A sustained scientific concern regarding toxicological assessment and exposure mechanisms can be observed across both developing and developed countries. The data also indicate that after 2018, South Asian countries, particularly China, India, and South Korea, experienced a substantial increase in research activity related to microplastics and human health, especially in port cities. While Europe continues to lead in the number of publications, the data also suggest a growing trend in international collaboration within the field, including strong contributions from European countries.

Research on microplastics and human health is increasing ($R^2 = 0.94$). Early studies were primarily published within the environmental sciences. However, fields such as toxicology, medicine, public health, water resources, and materials science have increasingly incorporated microplastic-related research. As interest in environmental toxicants and human exposure pathways expanded after 2020, research on microplastics and health similarly accelerated. The field is evolving beyond fundamental ecotoxicological principles toward a stronger focus on human health risk assessment and the need for integrated approaches to sustainability.

The complete list of the top 10 most frequently co-cited authors, along with their citation frequencies and centrality scores, is provided in Suppl. 1.

The bibliometric analysis demonstrated considerable variation in citation strength and structural influence among the top 10 most frequently co-cited authors in microplastic to human health research. Earlier publications from 2015 to 2018 appear to play a dominant role in accumulating co-citation frequency. This is consistent with the foundational nature of seminal works published during this period [15–17], which established methodological benchmarks and highlighted health-relevant implications of microplastic exposure. In particular, the

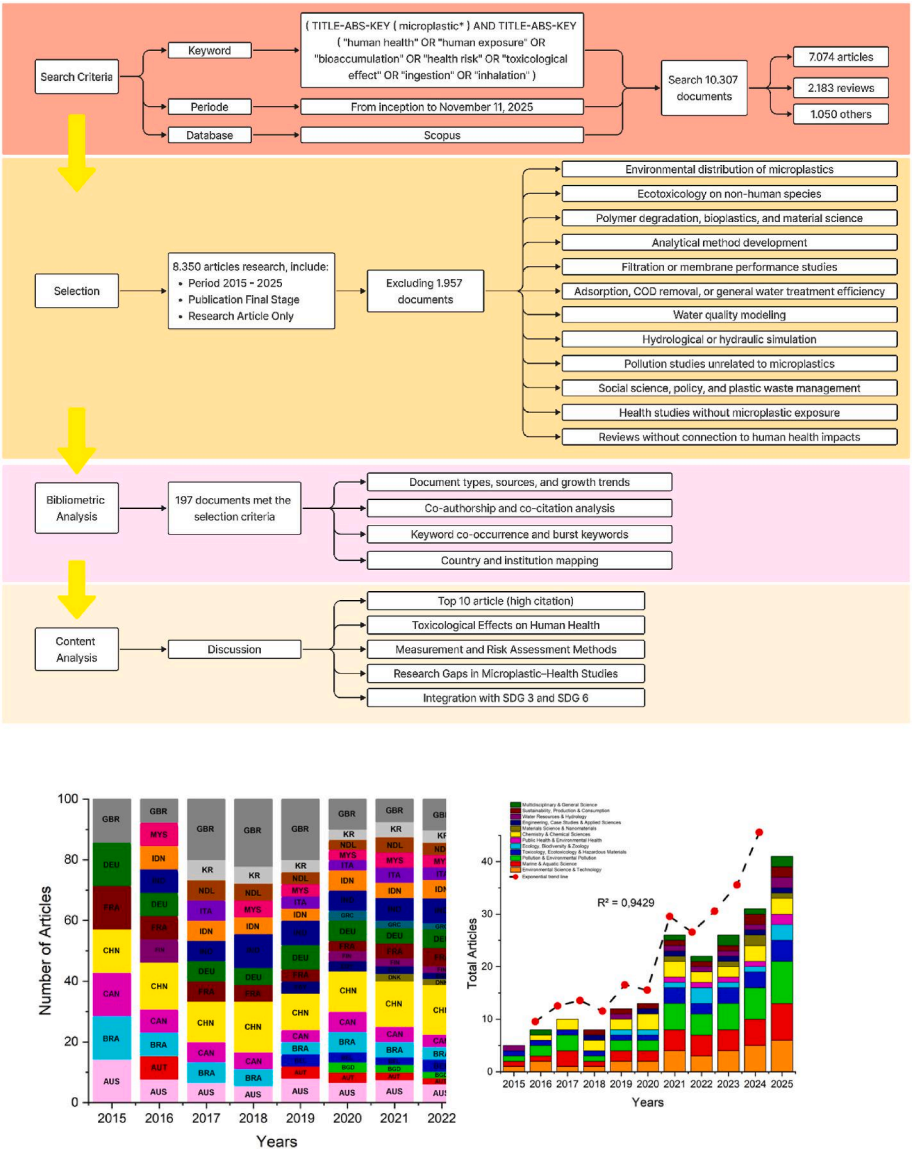

Fig. 2. (a) Bibliographic analysis procedure; (b) Country distributions; (c) Total document type based on subject related to microplastics–human health research.

study by Cole et al. (2015) in *Environmental Science and Technology* reached the highest frequency (97), followed by Weithmann et al. (2018) in *Science Advances* (85) and Nelms et al. (2018) in *Environmental Pollution* (91). These findings indicate that ecological investigations of trophic transfer and environmental pathways remain central to the intellectual foundation of the field. Meanwhile, more recent biomedical-oriented studies, such as Leslie et al. (2022), which documented plastic particles in human blood, and Jenner et al. (2022), which detected airway deposition, achieved high citation frequencies of 65 and 70, respectively, despite being published within the last three years. This trend suggests an emerging shift from environmental occurrence toward direct human exposure and clinical evidence. This shift may explain the increasing co-citation density in human health-related publications after 2020. Authors with relatively high centrality, such as Nelms et al. (0.05), Schirinzi et al. (0.04), and Mason et al. (0.03), demonstrate stronger bridging capacity across research clusters, indicating that their work functions as an interdisciplinary link between toxicology, environmental chemistry, and HBM. Conversely, authors with low or zero centrality, such as Amato-Lourenço et al. (2021), may exhibit high citation counts but limited influence within the broader knowledge network, possibly due to a narrower thematic scope. Overall, these patterns highlight the importance of highly cited early studies in shaping the current knowledge base, as well as the rapid emergence of clinical and biomedical evidence in steering the research agenda toward human health outcomes.

3.2. Thematic keyword clusters in microplastics research addressing HBM and health effects

The presence of frequently used keywords such as “human,” “health risk,” “bioaccumulation,” “ingestion,” “exposure route,” “urine,” “feces,” “plastic particle,” “PS particle,” and “nanoplastic” indicates that a substantial proportion of publications explicitly focuses on the direct pathways of microplastic entry into the human body and their subsequent accumulation in biological systems (see Fig. 3). Several laboratory-oriented terms, including “controlled study,” “concentration,” “particle dose,” and “exposure experiment,” reflect that most investigations of human microplastic exposure are dominated by laboratory-scale analyses rather than population-level monitoring. The co-occurrence of polymer-related expressions such as “PS,” “PE,” and “PP” in Cluster 1 confirms that these polymer types remain central to experimental human health evaluations, possibly due to their environmental prevalence and suitability for laboratory exposure models (see Fig. 4).

When examining the cluster structures, results from Cluster 2 reveal biological consequence-oriented keywords related to “metabolism,” “oxidative stress,” “tissue accumulation,” and “biomarker response,” suggesting that a major research focus is directed toward mechanistic toxicology, biochemical alterations, and inflammatory responses in human cells and organs as a result of microplastic exposure. Moreover, the grouping of terms such as “urine,” “feces,” “blood,” and “pulmonary tissue” within Cluster 3 further demonstrates that HBM studies have recently gained increasing scientific attention, indicating a growing shift from environmental occurrence studies toward biomedical detection, biotransport, and internal organ deposition of microplastics in humans. Collectively, the hierarchical dendrogram (Fig. 3) provides a clear thematic overview, showing that research on microplastics and human interaction primarily evolves around three major axes: (i) exposure pathways and polymer types, (ii) biological and toxicological mechanisms, and (iii) human tissue detection and health risk implications.

3.3. HBM evidence: what has been detected?

3.3.1. Biological matrices used in HBM

The most direct evidence of internal exposure to MPs comes from HBM studies. However, findings from these studies are difficult to interpret due to complex anatomical and physiological barriers, as well as biological defense mechanisms that regulate the absorption and distribution of particles within the human body. To date, a variety of human biological materials have been documented to contain MPs, including blood, lung tissue, and other respiratory samples (sputum or bronchoalveolar lavage fluid), urine, thrombi, and placental tissues. This diversity of tissues reflects different exposure routes (primarily ingestion and inhalation) and varying degrees of biological barrier translocation, and the biological interpretation is highly matrix-dependent. A comprehensive overview of the interpretation and methodological limitations for each biological matrix is presented in Suppl. 2.

Due to their non-invasive nature and their ability to indicate oral intake and intestinal passage of microplastics, feces are the most extensively studied biomonitoring matrix. Almost every study has documented the presence of MPs in human feces, with the overwhelming majority being fibrous and fragmented. Such MPs are indicative of exposure through food and the environment and do not reflect the accumulation of MPs in body tissues [18]. Given the physiological functions of the human body, MPs in feces are more representative of excreted particles than of those that have been absorbed and accumulated. Barriers in the gastrointestinal (GI) system, copious mucus secretion, and intestinal peristalsis all serve to eliminate larger particles; therefore, the presence of MPs in feces cannot be equated with systemic internal doses [19–21]. Some studies provide evidence of oral exposure

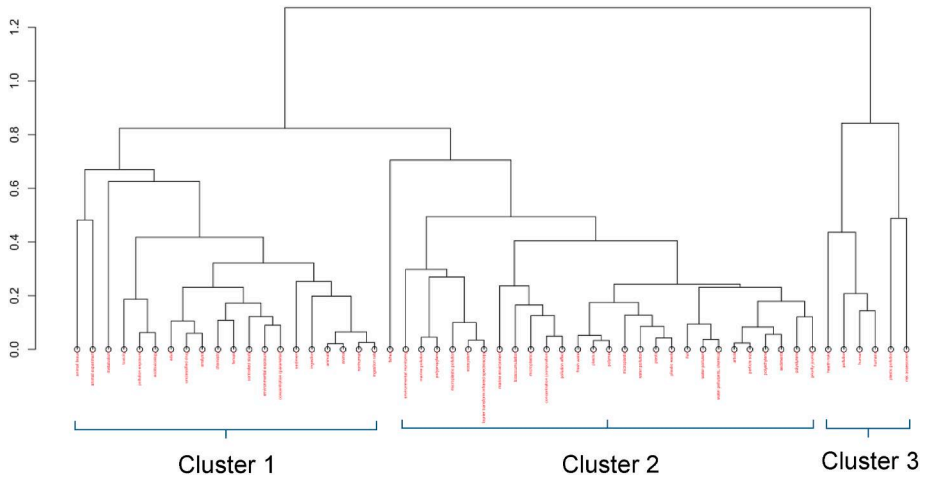

Fig. 3. Tree dendrogram of hierarchical cluster analysis of keywords used in research publications on microplastics and human health (bibliometric data as of November 11, 2025).

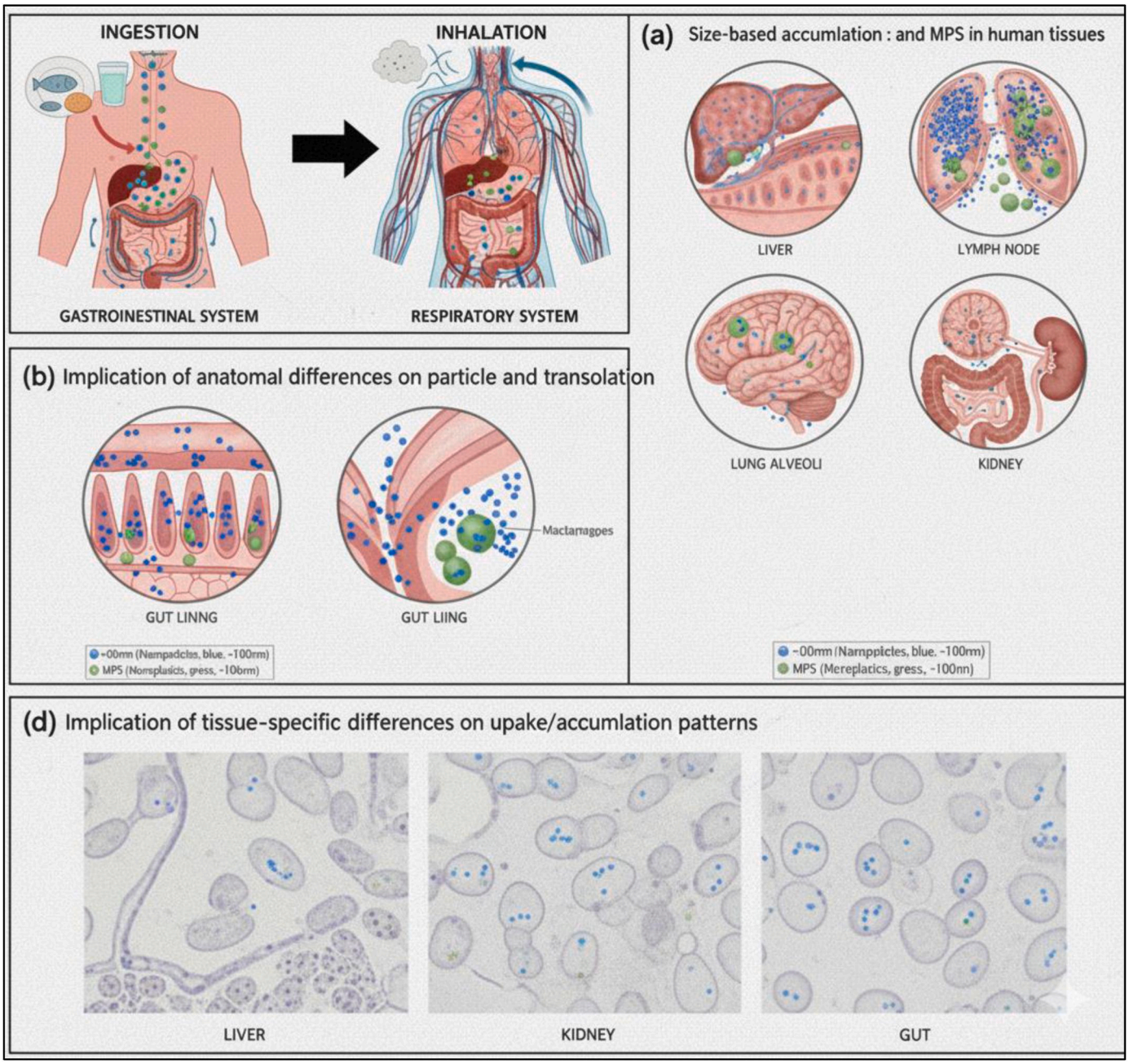

Fig. 4. Conceptual illustration of anatomical and physiological determinants of NPs and MPs uptake, translocation, and accumulation in the human body: (a) comparison of human entry pathways, gastrointestinal versus respiratory systems; (b) organ-level distribution and deposition of microplastics (5–20 μm) in the liver, kidney, and gut, indicating systemic translocation beyond the primary entry site; (c) size-based accumulation of NPs and MPs in human tissues; (d) implications of tissue-specific differences on uptake and accumulation patterns.

and unabsorbed intestinal passage of microplastics, as summarized in Suppl. 2.

In comparison, blood represents a more informative internal compartment, as it indicates that: (i) MPs have crossed the epithelial barrier. The pioneering research by Leslie et al. [9] was the first to identify and quantify polymer mass concentrations in total human blood, demonstrating that plastic particles ≥700 nm are bioavailable in the human bloodstream. These findings were corroborated by subsequent studies that focused on particle-count metrics and described associations between lifestyle-related MPs exposure and varying levels of coagulation disorders [22]. Despite this, blood-based data, as discussed in the matrix-specific constraints in Suppl. 2, are further complicated by low MPs concentrations, heterogeneous particle distribution, and a high potential for analytical contamination.

Respiratory matrices, such as lung tissue and sputum, demonstrate that inhalation must be considered an important pathway of exposure. MP fibers of different polymers and concentrations, affected by smoking and occupational exposure, have been detected in the sputum of patients with pulmonary diseases [23]. However, the respiratory system possesses robust biological defenses. The mucociliary escalator removes most inhaled particles and prevents them from reaching the alveoli. Therefore, translocation across the alveolar barrier is believed to occur only for very small particles and primarily under conditions of chronic and intense exposure [24–26]. As noted in Suppl. 2, respiratory matrices primarily reflect local exposure rather than total systemic burden.

More recently, MPs have been identified in placentas, urine, and

thrombi [27,28], suggesting that some particles may be capable of breaching biological barriers and disseminating throughout the body. Nevertheless, most of the available evidence remains limited due to small sample sizes and is often confined to specific clinical settings. Furthermore, biological defense mechanisms, such as macrophage-mediated phagocytosis, immune activation via NF-κB signaling, and clearance by the reticuloendothelial system, indicate that the translocation and retention of MPs are highly selective and strongly dependent on particle size and surface characteristics [29–31]. The interpretative limitations associated with these matrices are likewise summarized in Suppl. 2.

The predominant focus on fecal studies, combined with the relative scarcity of studies on blood and tissue, as well as the lack of standardized methodologies for biomonitoring microplastics in humans, highlights major gaps in current knowledge regarding human exposure assessment. The cross-matrix synthesis presented in Suppl. 2 emphasizes that existing biomonitoring evidence should primarily be interpreted as evidence of presence rather than as evidence of a biologically significant systemic dose or associated health risk. Therefore, for a more meaningful assessment of HBM findings related to human health risks, it is essential to integrate evidence of microplastic presence in specific biological matrices with an understanding of anatomical and biological protective barriers.

3.3.2. *Particle characteristics in human samples*

Studies on HBM indicate that MPs found in distinct human biological matrices exhibit considerable differences in particle size, polymer composition, polymer form, and quantification techniques. Current studies report that most detected particles overwhelmingly fall within the micro-size category, which reflects both biological limitations in particle translocation and constraints of current analytical methods. The differences presented in Table 3 reflect the diversity of matrices and methodologies used in laboratory analyses, highlighting the wide range of human biological samples in which MPs have been detected.

Mass-based assessments using pyrolysis-GC/MS in blood target particles greater than or equal to 700 nm and report polymer concentrations in the micrograms per milliliter range. These studies most commonly detect MPs composed of polyethylene (PE), polyethylene terephthalate (PET), and styrene-based polymers, while polypropylene (PP) often remains below the quantification limit [9]. Particle sizes inferred from mass concentrations suggest the presence of submicron to micro-sized particles, consistent with the greater bioavailability of smaller particles compared to larger fragments. Particle-count approaches provide additional insights into characteristics associated with specific methodologies. For example [22], used micro-Fourier transform infrared (μ-FTIR) spectroscopy and reported an average concentration of 4.2 particles mL^{-1} of MPs in human blood, dominated by polystyrene (PS) and PP, with most particles in the 20–50 μm (μm) size range. Fragment-shaped particles were more prevalent than fibers, suggesting a more direct consumer plastic fragmentation source rather than textile-derived origins. These findings, presented in Table 1, indicate that methodological selection significantly influences the reported size range and polymer composition of MPs in blood samples.

Fecal analysis continues to provide evidence of the wide range of polymers and morphological characteristics present in the gastrointestinal matrix. Table 1 demonstrates that the majority of MPs identified in fecal matter are fibers and fragments composed of polymers such as ethylene vinyl alcohol (EVOH), PP, PET, low-density polyethylene (LDPE), and PS. These polymers are commonly encountered across diverse diets and environments, suggesting an absence of selective biological retention [18]. Fecal analysis should therefore be interpreted as primarily reflecting excretion rather than retention processes.

Another example illustrating matrix-specific variability is found in biomonitoring of the respiratory system. Sputum samples show a predominance of particles smaller than 500 μm, with inhalation identified as the primary exposure route. This is consistent with the dominant presence of coarse polyurethanes (PU), polyesters, and fiber-like morphologies [23]. These data, as summarized in Table 1, align with the expected behavior of fibrous airborne particles, including their tendency for downward sedimentation within the respiratory tract.

Recent studies examining urine and thrombotic material suggest systemic redistribution of MPs with distinct characteristics. Urinary MPs are typically composed of small fragments (4–15 μm) made of PE, PP, polyvinyl chloride (PVC), and polyvinyl alcohol (PVA), indicating potential renal elimination pathways for smaller particles [28]. In contrast, thrombi contain MPs with higher mass concentrations and mean diameters of approximately 35 μm. These MPs are predominantly composed of PE, PA66, and PVC, suggesting possible local retention in diseased tissues [27]. Table 1 synthesizes the profiles of MPs in urine and thrombi, capturing variability in particle characteristics across matrices associated with elimination and pathological retention.

The information presented in Table 1 illustrates the reported characteristics of MPs in human samples, highlighting both biological processes and the influence of research design. The predominance of microplastic constituents, coupled with the limited detection of nanoscale particles, suggests a likely underestimation of smaller, potentially more bioreactive constituents. These bioreactive particles are likely underrepresented, which limits cross-study comparability and hinders comprehensive integration of available data on internal microplastic exposure in humans.

3.3.3. *Methodological heterogeneity and uncertainty*

Key for interpreting the heterogeneity observed across HBM studies is the systematic comparison of analytical techniques. The field is dominated by two main families of methods, namely mass spectroscopy-based methods, mainly pyrolysis-gas chromatography/mass spectrometry (Py-GC/MS), and spectroscopy-based methods such as Fourier Transform Infrared Spectroscopy (FTIR) and Raman. Four basic differences among these platforms affect reported MP characteristics and inferences about internal exposure. In terms of size detection limits, Py-GC/MS detects particles of ≥700 nm, while μ-FTIR is typically limited to ≥10–20 μm and μ-Raman to ≥1–10 μm. Therefore, spectroscopic methods are systematically biased against the submicron and nanoplastic fractions, which are the very fractions capable of traversing biological barriers with high physiological absorptive capacity into systemic circulation.

When it comes to quantification metrics, Py-GC/MS currently provides mass concentrations (μg/g or μg/mL), which relate to classical

Table 1
Reported size ranges, polymer types, and methodological approaches for microplastics in human biological samples.

Matrix	Size range reported	Dominant polymers	Predominant morphology	Quantification approach	References
Blood	≥700 nm (mass-based); 10–100 μm (count-based)	PET, PE, PS, PP	Fragments > fibers	Mass (μg mL^{-1}); particle counts	[9,22]
Feces	Broad micro-size range	EVOH, PP, PET, LDPE, PS	Fibers, fragments	Particle counts	[18]
Sputum/ respiratory	20–500 μm (median ≈75 μm)	PU, PES, CPE	Fiber-like, elongated	Particle counts	[23]
Urine	4–15 μm	PE, PP, PVC, PVA	Small fragments	Particle counts	[28]
Thrombi	Mean ~ 35 μm	PE, PA66, PVC	Heterogeneous	Mass (μg g^{-1}) + imaging	[27]

toxicological dose metrics, while FTIR and Raman determine particle counts per volume or mass. These two sets of metrics cannot be converted into one another without making assumptions about particle size and density. Spectroscopic FTIR allows for the characterization of the size, shape, and color of particles, unlike Py-GC/MS, which destroys the sample and provides no morphological information. Regarding polymer identification, all methods can identify polymers, but each will have a different spectral library and potential for interference, such as fluorescence observed in Raman and atmospheric contamination in FTIR. These differences become evident in blood analyses. For example, in Ref. [9], Py-GC/MS polymer mass concentrations (1.6 μg/mL) were mostly PET and PE, whereas [22] reported particle numbers dominated by PS and PP in μ-FTIR data, with concentrations ranging from 4.2 particles/mL for 20–50 μm. Importantly, these variable results do not necessarily represent conflicting conclusions, as different methods recover only specific fractions of the total particle burden. Consequently, this study highlights that any conclusion regarding "internal exposure" or "internal dose" should clarify the analytical approach applied and demonstrate why cross-study comparisons can only be regarded as semi-quantitative until harmonized reference materials and cross-validation studies become available. A comparative overview of these technical characteristics is provided in Table 2.

The differences reported in certain characteristics of MPs, such as quantification, morphology, polymer type, and size, reflect variations in methodology and the specific approaches employed in HBM studies. These differences can be attributed to the biological context, variations in detection principles, differences in size cut-offs, and inconsistencies in quantification and reporting metrics across studies. Table 3 provides a comparative synthesis of reported characteristics of microplastics within human biological matrices, as well as the inferred biological consequences of microplastics.

Studies employing mass-based analysis using Py-GC/MS for blood samples typically focus on microplastic particles ≥700 nm. These studies report polymer concentrations in micrograms per milliliter and most frequently detect PE, PET, and styrene-based polymers, while PP is often below the limit of quantification [9]. Most mass-based concentrations indicate a substantial presence of submicron to small micro-sized particles, which, in many biological contexts, are capable of crossing epithelial barriers and becoming internalized. This observation is consistent with experimental findings showing that particles smaller than 10–20 μm are readily internalized via endocytosis and translocated across epithelial barriers [32,33]. The blood-based studies summarized in Table 3 are largely characterized by methodological bias and limited variability in approaches used to quantify microplastics.

Alternatively, certain particle count–based methods yield different but potentially complementary information. Using μ-FTIR spectroscopy [22], reported an average concentration of 4.2 MP particles mL^{-1} in human blood, with the majority identified as PS and PP, and most particles ranging from 20 to 50 μm in size. The study also noted that fragment-shaped particles were significantly more abundant than fibers, suggesting that fragmented consumer plastic products may represent a major source of exposure. The substantial variation between mass-based and particle count–based results underscore the extent to which methodology influences observed MP characteristics, as illustrated in Table 3 through the wide range of reported particle sizes and inferred biological impacts.

Table 2
Comparative analytical characteristics of major MP detection techniques in HBM studies.

Feature	Py-GC/MS	μ-FTIR	μ-Raman
Size detection limit	≥700 nm	≥10–20 μm	≥1–10 μm
Quantification metric	Mass (μg/g or μg/mL)	Particle count	Particle count
Morphological information	None	Yes (size, shape, color)	Yes (higher resolution)
Particle size info	No	Yes	Yes
Sample destruction	Yes	No	No
Nanoplastic detection	Possible (indirect)	No	Limited (fluorescence)
Primary bias	Underestimates large particles	Misses submicron particles	Misses submicron; fluorescence

While analyzing various fecal samples, studies conducted in gastrointestinal matrices highlight the consistent presence of heterogeneous particle sizes, morphological attributes, and varying polymer compositions. Reports on fecal samples reveal a predominance of fragments and polymers, specifically EVOH, PP, PET, LDPE, and PS [18]. Some observations point to diverse dietary and environmental exposure sources, in addition to the relative efficacy of the digestive system in relation to particle size, retention, and subsequent excretion. Smaller microplastic particles, as available experimental evidence suggests, are retained for longer periods or are more likely to interact with gastrointestinal tissues, whereas larger particles are more readily excreted via feces or eliminated through sedimentation [34,35]. Therefore, MPs in feces, as shown in Table 3, primarily indicate the filtering out of systemically unexposed microplastics and the biological selection of particles based on size.

Monitoring of the respiratory system demonstrates a distinct particle profile. In the case of sputum, MPs largely consist of particles <500 μm. Dominant polymer types include PU and polyester, accompanied by frequent observations of fibrous and elongated morphologies [23]. These features align with inhalation exposure pathways and the selective retention of fibrous particles in the respiratory tract. In terms of respiratory interaction, smaller particles are more likely to reach the deeper peripheral regions of the lungs and, therefore, more likely to interact with respiratory tissues, as evidenced in experimental and inhalation toxicology studies [36,37]. Thus, as summarized in Table 3, evidence from sputum primarily reflects localized inhalation exposure.

MPs exhibiting specific characteristics have also been detected systemically, with recent findings reported in urine and thrombotic material. Urine samples have been shown to contain polymeric fragments (4–15 μm) composed of PE, PP, PVC, and PVA, which may support the hypothesis of renal clearance of smaller particles [28]. Conversely, thrombi have been reported to contain PE, PA66, and PVC, with particles averaging approximately 35 μm, suggesting potential pathological retention within tissues [27]. The contrasting profiles presented in Table 3 illustrate the biological and pathological factors influencing the distribution and retention of microplastic particles.

Ultimately, the synthesis across matrices shown in Table 3 indicates that the reported characteristics of microplastics in human samples (i.e., their presence and the bias introduced by analytical methods) are shaped by the interplay between biological selectivity and methodological variability. The predominance of micro-sized particles and the absence of nanoplastics appear to be systematic, suggesting that smaller, highly mobile, and potentially more bioactive particles may be underrepresented [38–40]. Furthermore, the heterogeneity of methodologies across studies contributes to limited comparability and an incomplete understanding of microplastic exposure in the human body.

3.4. Internal exposure pathways and biological interactions

3.4.1. Dominant human exposure pathways

The primary ways in which humans are exposed to MPs and NPs include ingestion and respiratory inhalation. This is because plastic particles are ubiquitous, being present throughout environmental systems as well as in man-made systems that come into contact with humans. Plastic particles occur in all aspects of daily life, and ingestion and inhalation represent the routes of exposure that pose the greatest potential risk. This is due to the widespread presence of particles at low concentrations in food, environmental ecosystems, and the atmosphere. Environmental exposure to micro- and nanoplastics, combined with

Table 3
Reported characteristics of microplastics in human biological matrices with interpretative biological notes.

Matrix	Dominant size range	Dominant polymers	Dominant morphology	Quantification method	Key biological implications	References
Blood	≥700 nm (mass); 20–50 μm (count)	PE, PET, PS, PP	Fibers > fragments	Py-GC/MS; μ-FTIR	Evidence of translocation of small particles; Method bias	[9,22]
Feces	Wide micro range	EVOH, PP, PET, LDPE, PS	Fibers, fragments	Particle counting	Excretion-dominated; biological size selection	[18]
Sputum	<500 μm (median ≈75 μm)	PU, PES	Elongated fibers	Particle counting	Local inhalation exposure	[23]
Urine	4–15 μm	PE, PP, PVC, PVA	Small fragments	Particle counting	Elimination of small particles	[28]
Thrombi	Average ~ 35 μm	PE, PA66, PVC	Heterogeneous	Mass + imaging	Potential pathological retention	[27]

other microchemical and biological pollutants, is illustrated in Fig. 5, which presents the main pathways of environmental co-exposure. The diagram highlights the primary routes by which micro- and nanoplastics enter the human body, namely ingestion through food and drinking water and inhalation from the air.

One of the most prominent routes of human exposure to microplastics is ingestion, which occurs through the consumption of food and drinking water. Originating from terrestrial sources and fragmenting into microplastics, contaminants can enter marine and freshwater systems via industrial discharge, plastic degradation, land-based runoff, wastewater effluents, and atmospheric deposition. The vertical distribution of microplastics in the water column is strongly influenced by particle density, size, and shape, as well as the presence of biofilms. Microplastics remain detectable in drinking water regardless of the treatment processes applied. Although water treatment removes the majority of suspended particles, residual microplastics persist, resulting in continuous low-level human exposure through drinking water [2,41,42]. Consumption of food, particularly seafood, further amplifies exposure, as food can act as a transfer vector for microplastics through trophic transfer. Numerous studies have reported the presence of microplastics across different levels of the aquatic food web, where it can bioaccumulate in organisms and subsequently be consumed by humans. Evidence of microplastics has been reported in commercially important fish species from field studies in Australia and Fiji. Even after removal of the gastrointestinal tract, an average of 1.58 particles per fish was detected, indicating that internal fish tissues remain a relevant exposure pathway [43]. Experimental studies have also demonstrated trophic transfer of MPs across multiple levels of the food web [44].

Mytilus spp. and other filter feeders are of particular concern because these organisms are commonly consumed whole. Microplastics have been detected in nearly all coastal areas of Norway, with abundances reaching up to 2.3 particles per individual. Consequently, the potential for human exposure through mollusk consumption is considerable [45].

At a deeper level than the detection of MPs in seafood commodities, knowledge about bioaccumulation and biomagnification is fundamental to understanding the potential human health impacts of environmental occurrence. Bioaccumulation refers to the overall increase in MP concentrations in an organism through uptake from surrounding water, sediment, or food, whereas biomagnification refers to the gradual increase in concentration across higher trophic levels in a food chain. A systematic synthesis by Ref. [46] critically collated available data on MP distributions in natural aquatic systems and found that sediments, rather than water, mussels, or fish, contain the largest proportion of the total MP load. Notably, the study reported a positive correlation between MP abundance in mussels and sediments, suggesting a potential link between environmental MP reservoirs and accumulation in filter-feeding organisms. This is mechanistically plausible, as mussels continuously filter large volumes of water and retain particles within the 1–100 μm size range. Bioaccumulation in mussels, which are often eaten whole and may involve imperfect gastrointestinal removal, therefore represents a direct and measurable exposure route relevant to human health. Nonetheless, the study also suggests a surprising lack of consistent

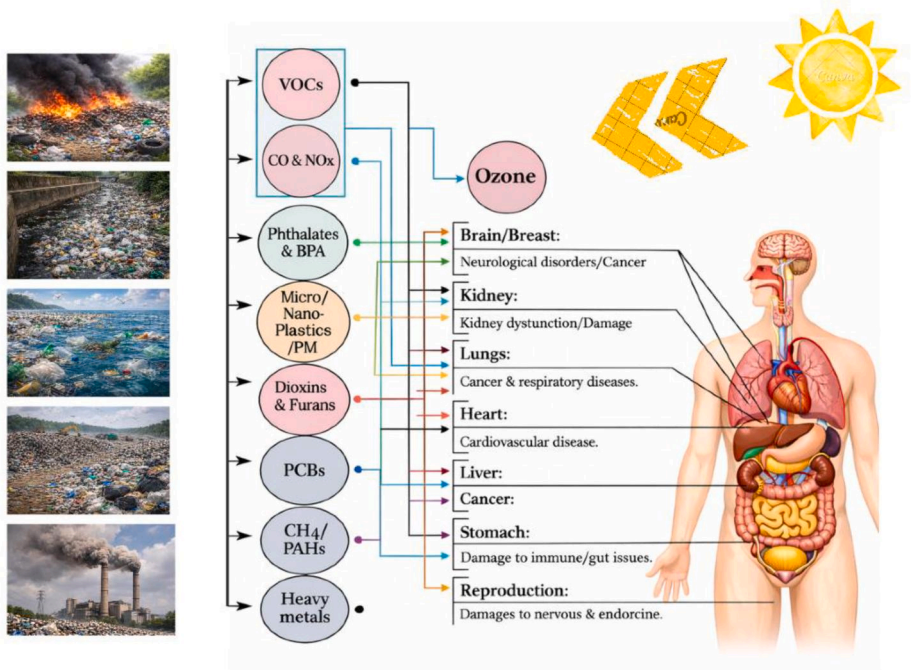

Fig. 5. Schematic illustration of environmental co-exposures involving micro/nanoplastics and associated pollutants and their dominant pathways of human exposure.

evidence for biomagnification, defined as the stepwise enrichment of MP concentrations at higher trophic levels. This is not merely an academic distinction; it has important implications for risk assessment. If biomagnification were occurring, top predators, including humans, would be expected to show higher MP body burdens along the continuum of trophic position, independent of specific exposure pathways. The absence of such evidence suggests that dietary exposure contributing to human internal dose may be dominated by direct bioaccumulation in consumed organisms, such as bivalves and small fish, rather than by trophic amplification through complex food webs. This finding align68s with the broader argument of this review: detection does not necessarily imply a biologically relevant internal dose.

However, two major caveats to this conclusion require further discussion. First, many field studies quantif68y total MP particles without clearly distinguishing whether particles are loosely adhered to gastrointestinal surfaces or internalized within edible tissues, such as muscle or liver. The detection of MPs in fish liver [47] and the observation of trophic transfer under controlled laboratory conditions [44] suggest some degree of tissue-specific accumulation, although with relatively low transfer efficiency. Second, nanoplastics and smaller microplastics may behave differently from larger particles because their reduced size increases the likelihood of epithelial uptake, tissue translocation, and biological persistence. Therefore, future risk assessment should not rely solely on total particle counts, particularly when current evidence does not strongly support food-web biomagnification for larger particles. Size-selective analytical approaches capable of quantifying the nanoplastic fraction in human tissues should be prioritised in future HBM studies, because this fraction, rather than total particle abundance, may ultimately determine chronic health risk.

Additionally, coastal and estuarine ecosystems impacted by the inflow of treated domestic wastewater exhibit increased microplastic uptake in juvenile fish, indicating that anthropogenically influenced ecosystems play a significant role in human exposure risk [48,49]. Fig. 6 integrates these ecosystems and their relevance to human exposure, illustrating the biological pathways, trophic transfer mechanisms, and potential health impact pathways of MPs and NPs across terrestrial and aquatic environments.

Food-related exposure is not limited to seafood. Plastic food-contact materials, particularly single-use packaging and heated containers, have been reported to contribute to the release of MPs into food. Environmental redistribution processes, such as deep-sea currents that generate benthic hotspots, can resuspend deposited microplastics and reintroduce them into coastal ecosystems that support fisheries [50]. The detection of microplastics in large marine predators, including whale sharks, further underscores the global mobility of MPs and the extent of cross-ecosystem exposure potential [51].

Beyond ingestion, inhalation is increasingly recognized as a major exposure pathway. Airborne microplastics originate from atmospheric deposition, indoor dust, and emissions associated with urban and industrial activities. Direct HBM evidence confirms the presence of various MP polymers in sputum, demonstrating that airborne particles can be inhaled and deposited in the human respiratory tract [23]. Behavioral factors such as smoking habits, occupational exposure, and medical procedures involving the respiratory system have been shown to modulate inhalation exposure levels. Unlike ingestion, inhalation allows MPs to interact directly with pulmonary tissues without first encountering the gastrointestinal barrier.

In contrast, dermal exposure is currently considered a secondary pathway. Although contact with microplastic-containing products can occur, evidence supporting significant transdermal penetration and systemic absorption remains limited compared with ingestion and inhalation. Consequently, the contribution of dermal exposure to overall internal human exposure is considered relatively minor.

The dominance of ingestion and inhalation pathways is reflected in HBM data across multiple biological matrices. Table 4 summarizes evidence of microplastics detected in human biological samples, including respiratory tissues, blood, urine, feces, placenta, breast milk, and male reproductive tissues, together with analytical approaches, reported concentrations, and key findings relevant to internal exposure. The environmental and dietary exposure pathways detailed above are reflected in contamination patterns observed in environmental media and food webs relevant to humans. Table 5 summarizes instances of microplastics in water systems and edible organisms, underscoring the pathways through which microplastics enter human food supplies.

Besides aquatic ecosystems, terrestrial environments and sediments also serve as additional exposure routes related to food security. Microplastics found in biota and organisms associated with sediments reveal indirect human exposure through food produced in soils and soil-

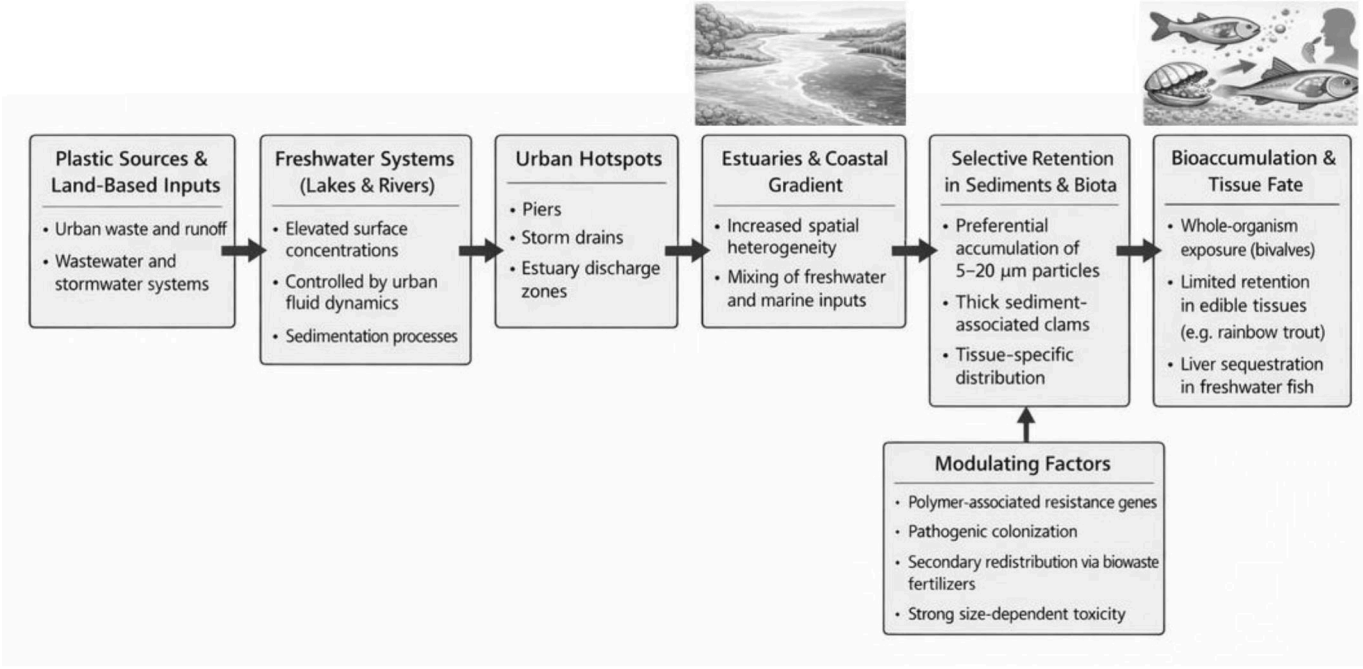

Fig. 6. Cross-ecosystem biological interactions and health-relevant effects of microplastics and nanoplastics.

Table 4
HBM evidence of microplastics in different biological matrices: Analytical approaches, concentrations, and key findings.

Biological Matrix	Study Location/ Population	Number of Samples	Analysis Method	MP Concentration	Polymer Types	Key Findings/Important Notes	References
Respiratory Tract (Lung & BALF)	Cancerous and non-cancerous lung tissue; GGN patients; Spanish population	13 lung tissue samples; BALF from 44 individuals	μ-FTIR, Raman, BALF-μFTIR	0.69 ± 0.84 MP/g (lung tissue); 9.18 ± 2.45 items/100 mL BALF	PP, PET, rayon, polyester, cellulose	Microfibers predominate (>90%); no significant difference between urban and rural populations; MPs detected in both tumor and normal tissues	[7,52–54]
Blood	Healthy volunteers, Netherlands	22	Double-shot Pyr-GC/MS	1.6 μg/mL	PET, PE, PS, PMMA	First study confirming MPs in human blood; PP < LOQ	[9]
Urine	Healthy volunteers (Italy)	6 (3 men, 3 women)	μ-Raman	Size: 4–15 μm (no mass quantification)	PP, PE, PVC, PVA	Possible gender-based differences in exposure or excretion	[28]
Feces (Stool)	Europe, Asia, China, Hong Kong, Indonesia, USA	>140 subjects (adults and infants)	FTIR, μ-Raman, Raman, HPLC-MS/MS, LDIR	1–138.9 MP/g; infants up to 82 μg/g	PP, PET, PE, PS, PVC, PA, PU, PLA	Higher concentrations in infants and IBS patients; PP often dominant and readily excreted	[55–59]
Placenta	Italy, Iran, China, Germany	>80 placentas	Raman, μ-Raman, LDIR	0.28–41.8 MP/g	PE, PS, PP, PVC, PA, PU	MPs can cross the placental barrier; higher concentrations associated with IUGR; potential links to personal care product use	[8,60] [61–63]
Breast Milk	Lactating mothers, Italy	34	Raman	0–2.72 MP/g (mean 0.51 ± 0.61)	PE, PVC, PP	Fragments and pigment particles predominate; potential risk of early-life exposure in infants	[8]
Testis	Donors, China	6 testis samples; 30 semen samples	LDIR, Pyr-GC/MS	Testis: 1.60 ± 15.5 MP/g; Semen: 0–2.06 MP/mL	PS (testis); PE, PVC (semen)	MPs detected in reproductive organs; potential implications for fertility	[31]

Table 5
Microplastic occurrence in environmental media and human-relevant exposure via seafood, aquatic systems, and food webs.

No.	Polymer	Experimental Media/ Model	Size	Exposure/Concentration	General/Accumulation Effect	References
1	PE + PP mixture	*Mytilus* spp., seawater	<5 mm	0.008–100 μg/L	Induced tissue-specific antioxidant responses (↑SOD, CAT), lysosomal enzyme changes, and the presence of MPs in the digestive gland, indicating sub-lethal stress in edible bivalves consumed by humans.	[64]
2	Mixed polymers (incl. PVC, PC)	Surface water, Manas River Basin	<5 mm	10–22 items/L	Seasonal variation with hazardous polymers highlights fluctuating exposure in inland drinking-water sources.	[41]
3	Mixed MPs	River macroinvertebrates	Environmental	Up to 0.14 MP mg^{-1}	Widespread ingestion by macroinvertebrates demonstrates the entry of MPs into freshwater food webs supporting human water and food resources.	[65]
4	PET, PE, PP	Mariculture cage biofilm (seawater)	Environmental	*In situ*	Plastic surfaces are colonized by potentially pathogenic bacteria (*Vibrio*, *Tenacibaculum*, *Escherichia*), acting as vectors into seafood chains.	[66]
5	Mixed MPs	Grey/harbour seals (GIT & feces)	≥100 μm	0–55 particles	Frequent presence in marine predators documents biomagnification potential and sentinel relevance to humans.	[67]
6	Mixed plastics	Plastisphere microbial communities, North Pacific Gyre	-	ARGs: 7.07×10^{-4}–1.21×10^{-2}	Floating plastics harbour antibiotic and metal resistance genes, raising concern about resistance vectors that may affect human pathogens.	[68]
7	Polyamide (PA), PET, acrylic	Demersal & pelagic fish (NE Atlantic)	Micro–macro	47.7% coastal fish	Identifies dominant polymers and documents higher coastal burdens, emphasizing increased risk for coastal fisheries and consumers.	[47]
8	Multiple polymers	*Mytilus* spp., Norway	<1 mm	1.5 ± 2.3 ind^{-1}	Shows mussels are suitable sentinels and indicates surprisingly high levels in the remote Barents Sea, suggesting a seafood exposure pathway.	[45]
9	Anthropogenic particles	Freshwater fish *Squalius cephalus*	Mean 2.41 mm	25% stomach, 5% liver	First report of MPs in the liver of wild freshwater fish, raising concerns about offal consumption and food safety.	[69]

based food chains, as summarized in Table 6.

Lastly, experimental studies on diet and co-exposure demonstrate the mechanisms of translocation, accumulation, and interactions between microplastics and co-occurring contaminants across various trophic levels, potentially extending to humans. These studies are synthesized in Table 7, which outlines dietary exposure pathways within the food web.

3.4.2. *Translocation, retention, and barrier interactions*

Human beings encounter MPs/NPs as a consequence of ingestion or inhalation. After entering the body, only a limited number of particles can permeate biological barriers. The body employs several strategies to limit the uptake and widespread distribution of foreign particles, including anatomical and physiological defenses such as the liver, the intestinal and respiratory tracts, as well as mucus layers. The efficiency of these barriers, however, is only partial and is highly influenced by particle size, stiffness, polymer type, and individual host characteristics, making these barriers inherently semipermeable [33,77,78].

Early studies have reported that certain MPs/NPs translocate through the intestinal epithelium, particularly in regions of the gastro-intestinal tract associated with Peyer's patches, via a combination of enterocyte transcytosis, endocytosis by M cells, and persorption under conditions such as epithelial cell turnover or compromised barrier integrity [14,78]. These processes allow some particles to penetrate the intestinal barrier and epithelial lining. However, it has been suggested

Table 6
Terrestrial and sediment biota exposed to microplastics and the implications for food security and human health.

No.	Polymer	Experimental Media/ Model	Size	Exposure/ Concentration	Accumulation Effect	References
1	Various MPs (PS over-represented)	Terrestrial soil fauna (review: earthworms, nematodes, beetles, etc.)	Mostly <1 mm; many PS microspheres	Broad range of experimental concentrations	Across earthworms, nematodes, beetles, etc., MPs caused shifts in gut microbiota and impaired growth, metabolism, reproduction, and survival, especially for small particles at high doses, mechanisms that may analogously threaten the human gut and soil-based food security.	[70]
2	LDPE MPs	Aquatic midge *Chironomus riparius* larvae	<32 and 32–45 μm, irregular	0.025 and 2.5 g kg^{-1} dry sediment for 48 h	Environmental LDPE levels altered energy reserves, increased antioxidant and detoxification enzyme activity, and changed the expression of endocrine-, immune-, and DNA repair–related genes.	[71]

Table 7
Dietary and Co-exposure studies in fish, invertebrates, and trophic chains.

No.	Model	Polymer	Size	Concentration	Duration	References
1	Rainbow trout (*Oncorhynchus mykiss*)	PE microspheres (dietary exposure)	10–300 μm	Up to ~9800 spheres g^{-1} food for 2 weeks	2 weeks + depuration	[72]
2	Copepod (*Calanus helgolandicus*)	PS	20 μm	75 MP/mL	Laboratory exposure	[15]
3	Algae → snail trophic chain	MPs + methamphetamine (MP mixture)	N/A	Methamphetamine EC50/LC50 shifted when combined with MPs	N/A	[73]
3	Fish (*Fundulus*, *Pimephales*)	Tire crumb rubber	38–355 μm	≤6 g/L	Laboratory exposure	[74]
4	Sea urchins (*Psammechinus miliaris*, *Paracentrotus lividus*)	PVC MPs	53–59 μm	25,000 MP L^{-1} (water), 0.5% mass in diet for 2 months	2 months	[75]
5	Freshwater fish (laboratory)	Mixed MPs (fibres & fragments, <5 μm)	<5 μm	Realistic exposure	N/A	[76]

that most particles do not cross the gut barrier, remain trapped within the intestinal lumen, and are ultimately excreted in feces [78]. Nevertheless, *in vivo* studies across several species indicate that smaller particles can translocate to secondary organs such as the liver and kidneys, most likely via the lymphatic and portal systems, where it may exert effects, particularly on immune function and oxidative stress pathways [19,67,79–81].

A critical factor influencing these processes is particle size. Mucus trapping, peristaltic propulsion, and depurative removal systems, as demonstrated in fish and invertebrates, effectively eliminate larger particles greater than 10 μm [19]. Particles within the 5–20 μm range exhibit longer retention times in tissues and greater persistence within food webs, increasing the potential for chronic exposure in the digestive systems of higher trophic organisms, including marine mammals and, by analogy, humans [67,79,80]. Furthermore, environmental aging alters the surface characteristics of microplastics, thereby modifying their interactions with biological tissues. These changes may influence feeding behavior, metabolism, and the capacity for translocation [15,82,83].

In the respiratory system, the first line of defense against MPs/NPs is mucociliary clearance. Translocation, which may involve alveolar epithelial transcytosis or migration of alveolar macrophages to the lymphatic system, occurs primarily with smaller particles smaller than 5 μm that can reach the alveolar region [5,14]. Some studies report that chronic exposure or specific occupational environments may promote the deposition of nanoscale particles in peripheral lung regions and potentially enable passage across the alveolar barrier [24,25,84]. Furthermore, the presence of larger MPs in human lung tissue may be explained by particles circulating in the bloodstream becoming mechanically trapped within the lung's fine capillary networks [5].

Once in the bloodstream, MPs exhibit rapid circulation. This is primarily due to the activity of macrophages within the reticuloendothelial system, particularly in the liver and spleen, which play a central role in particle capture and clearance [5,85]. Only a small fraction of particles may evade macrophage uptake, and this ability is largely dependent on particle size, surface properties, charge, and potential biofilm interactions.

Nanoplastics are hypothesized to interact with various biological barriers, including the blood–brain barrier (BBB) and the placental barrier. However, direct evidence in humans remains limited, with most findings derived from animal models, *in vitro* studies, and theoretical estimations based on particle size and physicochemical properties [14,86]. Consequently, the potential health effects associated with the crossing of these barriers remain uncertain. Overall, translocation of MPs/NPs across biological barriers in humans appears to be highly selective. While available evidence indicates that translocation can occur, it is constrained by the multilayered system of physiological defenses. Current findings suggest that, although microplastics may act as biological stressors, there is insufficient evidence to support long-term systemic retention or widespread accumulation in the human body [5,14]. These findings are summarized in Table 8.

3.5. From detection to health risk: where does current evidence fall short?

3.5.1. Detection versus internal dose

The last decade has seen explosive growth in studies detailing the presence of MPs and NPs in human biological matrices. These studies have begun to better inform the phenomenon of human exposure to plastic particles [12,90]. However, while there is a gap between establishing the presence of plastic particles and determining a biologically relevant internal dose, biomonitoring studies primarily focus on documenting the presence of plastic particles in human biological matrices, including tissue, blood, sputum, and feces. These studies document and characterize particle quantities, their distribution across various size classes, and the polymer types that comprise them. However, it fails to characterize, or even attempt to estimate, the fraction of particles that may be absorbed, systemically distributed, and/or capable of eliciting a toxicological response [5,14,91].

Fig. 7 illustrates microplastic contamination in water bodies and food systems from inland areas to coastal regions and provides a spatial and environmental framework to understand this gap. These gradients demonstrate diffuse environmental contamination and the various exposure pathways present across drinking water sources, freshwater

Table 8
Size-dependent translocation and clearance mechanisms of micro- and nanoplastics in human biological systems.

Biological Stage/Location	Major Biological Barriers	Translocation/Retention Mechanism	Dominant Size Range	References
Digestive tract (small intestine)	Intestinal epithelium, mucus, tight junctions	Enterocyte transcytosis; M-cell endocytosis (Peyer's patches); limited persorption	MPs <5–20 μm; NPs <100 nm	[5,14]
Lymphatic and portal system	Lymphatic and portal venous pathways	Transport of small particles to the liver and spleen	Small MPs (<5–10 μm)	[19] [87]
Liver and spleen (RES)	Reticuloendothelial system	Phagocytosis by macrophages; rapid clearance	Internalized MPs and NPs	[5,85]
Respiratory tract (alveolus)	Mucociliary clearance; alveolar epithelium	Alveolar transcytosis; macrophage migration to lymphatics	MPs <5 μm; NPs	[5,14]
Capillary network	Narrow capillary diameter	Mechanical trapping of circulating particles	MPs >5–10 μm	[5]
Blood–brain barrier (BBB)	Brain capillary endothelium	Suspected NP transcytosis; limited evidence	NPs <100 nm	[14,86]
Placental barrier	Trophoblast layer	Selective translocation of very small particles	NPs; very small MPs	[14]
Gastrointestinal elimination	Peristalsis, mucus	Fecal excretion	MPs >10 μm dominant	[5]
Respiratory defense	Mucociliary clearance	Clearance and re-ingestion	Large MPs and fibers	[25,84]
Skin (secondary pathway)	Stratum corneum	Limited penetration; increased in damaged skin	NPs	[88,89]

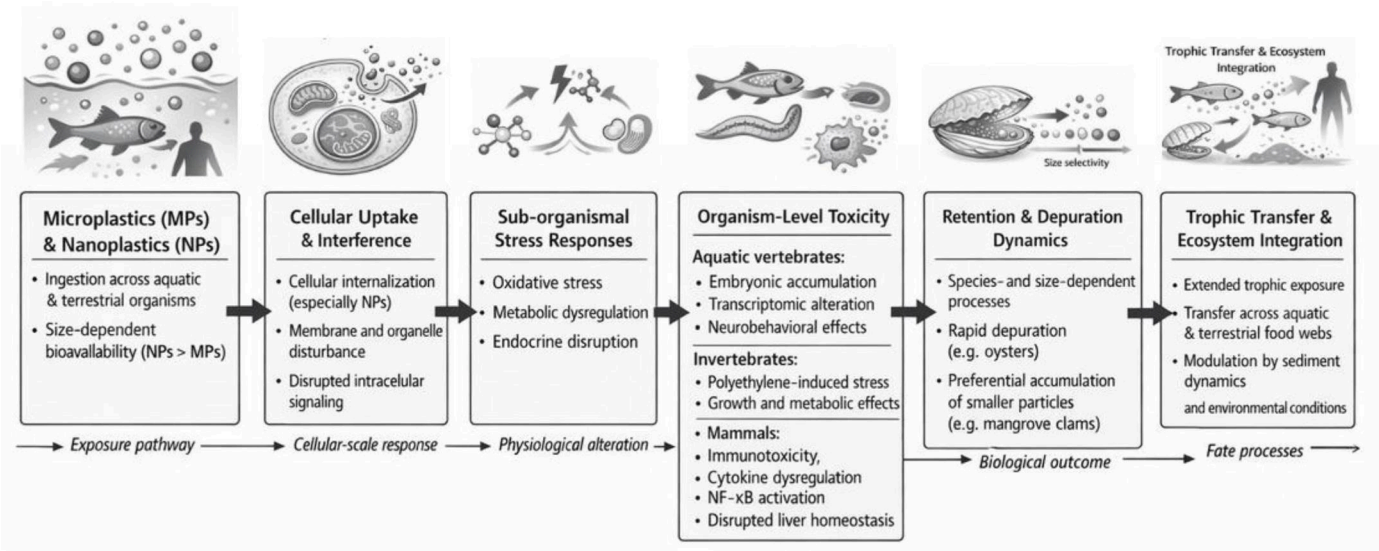

Fig. 7. Inland-to-coastal gradients of microplastic contamination in water and food systems.

systems, microplastic-contaminated wetlands, and coastal food webs. While these pathways shape external exposure scenarios, it provides little information regarding the internal dose that may be present.

Detection-focused evidence primarily reflects external contact and exposure rather than internal dose. It may not mean that MPs are absent from the human body — HBM studies have certainly found MPs in many biological matrices. But the presence of a particle in a biological sample is not equivalent to an internal dose that is relevant and able to cause systemic effects (i.e., distributed, retained in target organs, and causing toxicity). Microplastics found in feces mainly indicate unabsorbed material undergoing excretion and reflect oral exposure rather than internal burden [92]. Similarly, particles detected in sputum and ther respiratory samples primarily indicate deposition following inhalation and do not demonstrate systemic internalization or distribution to target organs [4,93].

The interpretation of microplastics detected in blood and tissues in terms of internal dose remains very limited. A lack of information on particle residence time, subcellular localization, and tissue-specific accumulation represents a critical gap in defining internal dose and biological relevance [25,85]. Therefore, the mere detection of microplastics in internal matrices does not necessarily indicate systemic exposure.

Fig. 8 further illustrates this conceptual distinction by schematically linking sources of environmental pollution, micro- and nanoplastic co-exposure, associated contaminants, and potential organ-specific health outcomes. Fig. 8 highlights that multiple exposure pathways and co-contaminants may converge at target organs, while metrics based solely on detection remain insufficient to address dose–effect relationships.

Internal dose is not determined solely by the number of particles present, but by the fraction capable of traversing biological barriers, evading physiological clearance, and interacting with target systems. While this is well established, the majority of the literature on microplastic bioavailability and inflammation lacks consideration of the substantial proportion of particles that remain undetected in both environmental and biological contexts. Most studies indicate that the majority of microplastics are not retained within biological systems following ingestion or inhalation, even after repeated exposure [13,25,63]. Less than 1% are absorbed. Most particles are eliminated via mechanisms such as the mucociliary escalator, macrophage-mediated phagocytosis, or the reticuloendothelial system [63,78,94]. Furthermore, many studies overlook biological clearance mechanisms, despite their potential to induce irritation and tissue damage [95]. As a result, systemic exposure is frequently overestimated when conclusions are based solely on detection frequency or particle counts.

Another major limitation is the lack of consistent and standardized metrics for measuring internal dose. Studies report results using markedly different measurement units, resulting in highly heterogeneous approaches. Metrics vary widely, including particle counts, mass-based measurements, aggregate mass descriptions, and distributions of

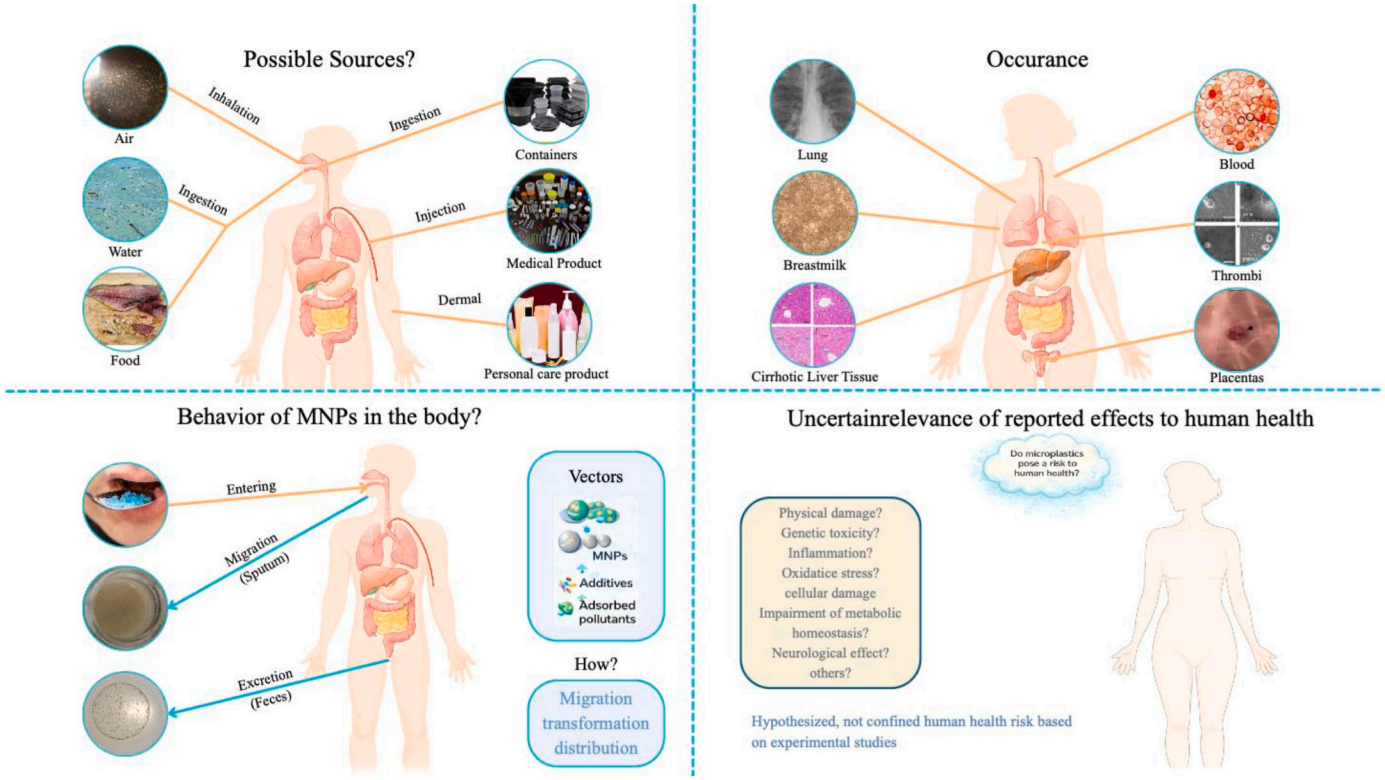

Fig. 8. Schematic overview of environmental pollution sources and co-exposure pathways linking micro/nanoplastics and associated contaminants to potential organ-specific health outcomes.

microplastic sizes. However, these approaches are not coherently integrated to define internal dose or its spatial and temporal dynamics [14,91]. Such heterogeneity not only prevents meaningful comparison across studies but also represents a major barrier to developing reliable dose–response frameworks and estimating the potential health risks of human exposure [12,78].

These and other conceptual and methodological limitations are summarized in Table 9. Each limitation reflects a gap in linking particle detection to a biologically relevant internal dose.

Although detection-based data are essential for demonstrating human exposure to microplastics, it remains insufficient to address the critical question of dosimetry, namely, the biologically relevant internal dose. Integrated approaches combining HBM, pharmacokinetic modeling, predictions of barrier crossing, and particle distribution and elimination dynamics are required prior to establishing dose–response relationships and conducting meaningful human health risk assessments [14,25,96].

Table 9
Methodological and conceptual limitations in linking microplastic detection to internal dose.

Aspect	Detection-based evidence	Internal dose relevance	Limitation
Biological matrices	Feces, blood, sputum, tissues	Systemic availability unclear	Presence does not equal absorption
Reported metrics	Particle counts, polymer types, sizes	Dose at target organs	Lack of residence time data
Barrier crossing	Rarely quantified	Determines bioavailability	Uptake fraction largely unknown
Clearance processes	Often ignored	Controls internal burden	Rapid elimination not accounted for
Standardization	Highly heterogeneous	Needed for dose–response relationships	No unified internal dose metric

3.5.2. *Internal dose versus adverse health outcomes*

There is an increasing number of experimental and observational studies documenting the diverse biological impacts of MPs/NPs; however, a definitive link between internal dose and adverse health outcomes in humans remains unestablished. The main reason is that studies, both *in vitro* and *in vivo*, do not align the internal dose levels used in exposure toxicity experiments with the internal doses that might reasonably occur in humans under chronic environmental exposure scenarios [91,97].

Importantly, the abundance of microplastics in mussels has been shown to be positively correlated with sediment concentrations, indicating bioaccumulation via water-borne pathways. A systematic review by Refs. [46,98] summarized data on microplastic abundance across natural aquatic environments and found that microplastics are more abundant in sediments than in water, mussels, or fish. Mussel microplastic abundance was highly correlated with that in sediments, further supporting bioaccumulation through water-borne pathways. However, pathways of biomagnification remain unclear, and additional evidence is needed to demonstrate whether microplastics can be transferred trophically through aquatic food webs across diverse environmental conditions and ecosystems worldwide. These findings have direct implications for human health risk assessment, as seafood consumption, particularly of filter-feeding organisms such as mussels, represents a primary route of human exposure. Meanwhile, smaller microplastics and nanoplastics are more capable of crossing biological barriers and accumulating within specific organs, raising concerns about chronic health effects under low-dose, long-term exposure scenarios. Such bioaccumulation may help explain microplastic concentrations over time in different organs and provides a theoretical basis for risk assessment models. This perspective is particularly relevant given the historical absence of standardized internal dose measurements, which should

consider not only the presence of microplastics in biological matrices but also their potential for trophic transfer and tissue retention with gradual accumulation. Despite these considerations, the referenced study concluded that no clearly established biomagnification pathway exists and that current evidence does not support trophic transfer of microplastics across aquatic food webs on a global scale.

Most studies identify particle size as a key determinant of the biological behavior and toxicity of micro- and nanoplastics. Smaller particles have been shown to permeate biological barriers more efficiently than larger ones, which explains the focus on smaller microplastics and nanoplastics in many aquatic studies [36,99]. Particles smaller than 10 μm and 200 nm (microplastics and nanoplastics, respectively) have the capacity to induce oxidative stress, inflammatory responses, and metabolic disruption, and have also been shown to permeate cellular membranes via endocytosis [25,32,38,40,100]. However, many toxicological studies assessing the effects of micro- and nanoplastic particles employ exposure concentrations that far exceed realistic environmental levels.

Additionally, numerous toxicological studies utilize specific polymer types, such as PS, with homogeneous particle sizes and high exposure doses. This further limit their external relevance to human exposure scenarios, which are typically more heterogeneous, chronic, and lower in dose [91,97]. Supporting evidence indicates that the uptake of MPs/NPs is minimal, often less than 1%, with most particles being eliminated through bodily excretion. Therefore, internal human doses are expected to be low [78]. This contributes to the difficulty of accurately assessing health risks and increases the likelihood of overestimating risk when extrapolating toxicological findings directly to humans. The discrepancy between experimental doses and realistic human internal doses is particularly evident in rodent models used to simulate ingestion exposure. These models are commonly employed to study the systemic distribution and biological effects of MPs/NPs. Table 10 summarizes particle size, exposure models, doses, exposure duration, and major biological effects reported in numerous *in vivo* studies. Notably, the majority of adverse effects observed in these studies occur at doses significantly higher than those typically encountered in the environment, rendering them less representative of real-world human exposure conditions.

Apart from possible dose discrepancies, interpretative constraints are further reinforced by the fact that, although microplastics have been identified in numerous human tissues, including blood, placenta, and lungs, most HBM studies have not been accompanied by evidence of tissue dysfunction or clinically measurable changes [14,85]. This underscores that the presence of internal particles does not necessarily indicate functional toxicity, and that the detection of particles in the body cannot be unconditionally regarded as a health risk or concern without quantitative assessment of the relevant biologically active internal dose.

The absence of a universally accepted biologically relevant internal dose further amplifies existing knowledge gaps. There is no consensus on the specific particle size, number, and surface properties that are likely to induce damage in humans. In addition, important phenomena, such as the formation of the biological corona, interactions with plasma proteins, modulation of immune responses, chemical leachates, and adsorbed pollutants, have not been systematically incorporated into dose–response frameworks [10,119].

Further research is required to accurately establish the relationship between the internal dose of microplastic particles and adverse health outcomes in humans. The toxicological literature highlights the potential biological threats associated with exposure to MPs/NPs; however, current evidence remains insufficient to determine the mechanisms linking internal dose to adverse health effects in humans. These uncertainties emphasize the need for comprehensive investigations into the biological impacts of MPs/NPs. Establishing such relationships requires the quantification of internal dose under realistic exposure scenarios, supported by clinically relevant biomarkers of effect. Moreover, evidence should encompass the distribution, metabolism, and elimination of microplastics within the human body [14,78,85].

3.5.3. *Reliance on experimental and animal-based evidence*

The potential human health risks of microplastics are still largely based on empirical research from animal studies and *in vitro* experiments. These studies serve an important function in establishing early mechanisms of toxicity, such as oxidative stress, inflammation, mitochondrial disruption, metabolic dysregulation, disturbance of the epithelial barrier, and redox imbalance [91,97,120]. However, the predominance of experimental studies also presents critical challenges for applying their findings to human exposure scenarios.

There is a clear misalignment between microplastic exposures in experimental settings and those experienced in real-world environments. Many *in vitro* and *in vivo* studies utilize microplastic concentrations that do not reflect the chronic, low-level exposures humans experience over their lifetimes; as a result, associated health risks may be overestimated [5,78,121]. Furthermore, many studies rely on a single model polymer, often PS, with consistent size, shape, and surface charge, which does not reflect the substantial variability of real-world microplastic exposure.

Biological differences between animal models and humans further strengthen translational limitations. Differences in anatomy, physiology, particle kinetics, and biological barriers limit the direct applicability of animal-based toxicological studies to humans [78]. For instance, microplastic absorption, distribution, and elimination processes in rodents are not fully representative of adult human pharmacokinetics. Consequently, animal studies provide evidence that is more inferential than empirical when establishing causal links between microplastic exposure and human health effects [85].

When considered alongside HBM, these limitations become particularly pertinent. HBM assesses human exposure to environmental pollutants by analyzing biological samples (blood, urine, feces, or tissue) for the presence of pollutants or their metabolites and breakdown products [122]. It offers a direct approach to confirming the presence of microplastics in the human body by integrating multiple exposure pathways, including air, food, water, and personal care products, as well as routes such as inhalation, ingestion, and dermal absorption [123]. However, the presence of particles in biological matrices does not necessarily indicate biologically significant internal exposure or associated health risks.

Microplastics have been detected in a wide range of human tissues, including the respiratory tract, blood, urine, feces, placenta, and even breast milk. However, many HBM studies lack evidence of tissue dysfunction or clinically measurable changes [12,14]. The human body possesses multiple biological barriers and clearance mechanisms, such as the mucociliary escalator, macrophage phagocytosis, and lymphatic transport, which help limit the penetration and accumulation of particles [124–126]. The biokinetics of microplastics in humans remain poorly understood, in contrast to engineered nanoparticles, which have been studied more extensively [62,127].

To date, the most detailed insights into microplastic–cell interactions have come from *in vitro* studies, which provide controlled conditions for systematically examining particle uptake, immune activation, and inflammatory responses. Key findings from *in vitro* studies are summarized in Table 11. Biological responses appear to depend on particle size, polymer type, concentration, and cell type.

Alongside these findings, studies investigating the potential impacts of microplastics have reported cellular inflammation, granuloma formation, and fibrosis [129], oxidative stress [9], and dysfunction of respiratory and gastrointestinal epithelial cells [4,130,131]. Biotransformation of microplastics during digestive transit and alterations in gut microbiota composition have also been documented in static and dynamic gastrointestinal models [100,132,133]. Most observed biological effects occur at microplastic concentrations far exceeding those realistically encountered in the environment, and under acute rather than

Table 10
Oral exposure in rodent models (Gavage, drinking water, and dietary routes).

No	Model	Polymer	Size	Exposure	Concentration	Duration	Effects	References
1	C57BL/6 mice	PS (pristine or fluorescent)	50, 500, 5000 nm, alone or mixture	Oral gavage	2.5, 25, 50, 125, 250, 500 mg/kg bw	Single dose (24 h); daily for 28 days	Size-dependent accumulation with broad organ distribution; induced intestinal oxidative stress, barrier dysfunction, and inflammation, enhanced by co-exposure and mitigated by antioxidants.	[101]
2	Wistar rats	PS (mixture of 25 and 50 nm)	25 and 50 nm mixture	Oral gavage	1, 3, 6, 10 mg/kg bw/day	Daily for 35 days	Testicular accumulation associated with reduced sex hormones, impaired sperm quality, and testicular atrophy.	[60]
3	CD-1 mice	PE (coated with phthalate esters)	0.4–3.2 μm	Oral gavage	100 mg/kg bw	Daily for 30 days	Gut–liver–testis accumulation of phthalate-associated MPs induced oxidative stress and impaired spermatogenesis.	[29]
4	C57BL/6 HOTT reporter mice	PS (fluorescent, carboxyl- or sulphate-coated)	1, 4, 10 μm mixture	Oral gavage	1.25–34 mg/kg bw, adjusted for surface area	Three times per week for 28 days	No significant accumulation or treatment-related adverse effects observed.	[100]
5	ICR mice	PS (pristine or fluorescent)	5 and 20 μm	Oral gavage	0.01–0.5 mg/day	Up to 28 days	Size-dependent accumulation in the liver, kidney, and gut caused oxidative stress and metabolic dysregulation.	[33]
6	C57BL/6 mice	PE	10–150 μm	Diet (mixed in basal feed)	6, 60, 600 μg/day (assuming 3 g feed)	35 days	Intestinal inflammation, immune pathway activation, and gut microbiota alterations, dose-dependent.	[102]
7	SPF C57BL/6 mice	PS (fluorescent)	60 nm	Oral gavage	50 μg/mL (500 μL)	Single dose; 3 days	Accumulation detected only in the stomach, intestines, and liver.	[103]
8	ICR mice	PS (pristine)	5 μm	Drinking water	100 and 1000 μg/L	42 days	Gut accumulation induced microbiota dysbiosis, mucus depletion, barrier impairment, and metabolic alterations.	[104]
9	ICR mice (dams, F1, F2)	PS	5 μm	Drinking water (during pregnancy and lactation)	100 and 1000 μg/L	≈6 weeks	Transgenerational metabolic disruption with gut dysbiosis and hepatic lipid alterations	[105]
10	ICR mice (offspring)	PS	0.5 and 5 μm	Drinking water (during pregnancy)	100 and 1000 μg/L	≈6 weeks	Prenatal exposure disrupted offspring lipid metabolism in a sex-dependent manner.	[105]
11	Fischer 344 rats	PS (fluorescent, non-coated, aminated, carboxyl-modified)	50 nm	Oral gavage	125 mg/kg bw	Single dose, 6 h	Low systemic bioavailability with no acute histopathological effects.	[106]
12	C57BL/6 mice	PS (fluorescent)	5 μm	Drinking water	20 mg/kg/day bw	30 days	Hepatic accumulation induced inflammation, oxidative stress, and hepatocellular injury.	[33]
13	CD-1 mice	PE and PS suspended in organic flame retardants (OFR)	0.5–1.0 μm	Drinking water	2 mg/L (PE or PS) in 10 or 100 μg/L OFR	90 days	Liver and gut accumulation caused inflammation, oxidative stress, and metabolic and neurotoxicity markers, amplified by OFR co-exposure.	[103]
14	BALB/C mice	PS (fluorescent)	0.5, 4, 10 μm	Oral gavage	10 mg/mL	Daily for 28 days	Testicular accumulation induced inflammation, reduced sperm quality, and blood–testis barrier disruption.	[107]
15	ICR mice	PS	0.5 and 50 μm	Drinking water	100 and 1000 μg/L	35 days	Reduced adiposity and intestinal mucus, with altered gut microbiota and hepatic lipid metabolism.	[108]
16	Swiss mice	PUR	250 nm	Oral gavage and intraperitoneal	2, 5, 10 mg/kg bw	10 days	Systemic inflammation with renal, hepatic, and pulmonary injury, and increased visceral adiposity.	[109]
17	C57BL/6 mice	PS	5 μm	Drinking water	500 μg/L	28 days	Exacerbation of colitis with increased intestinal permeability and hepatic lipid peroxidation.	[110]
18	BALB/c mice	PS	5.0–5.9 μm	Oral gavage	0.01, 0.1, 1 mg/day (± NAC or p38 MAPK inhibitor)	Daily for 42 days	Oxidative stress-mediated male reproductive toxicity, reversible by antioxidant treatment and p38 inhibition.	[111]
19	ICR mice	PS	5 μm	Drinking water	100, 1000, 10,000 μg/L	35 days	Testicular injury linked to inflammatory activation and impaired antioxidant defense	[112]
20	Wistar rats	PS	0.5 μm	Drinking water	0.015, 0.15, 1.5 mg/kg/day	90 days	Ovarian oxidative stress with reduced follicular reserve and inflammasome activation.	[113]
21	Wistar rats	PS	0.5 μm	Drinking water	0.015, 0.15, 1.5 mg/day	90 days	Ovarian fibrosis and reduced reserve associated with oxidative stress and apoptotic signaling.	[114]

(*continued on next page*)

Table 10 (*continued*)

No	Model	Polymer	Size	Exposure	Concentration	Duration	Effects	References
22	ICR mice	PE (surface-modified)	40–48 μm	Oral gavage	3.75, 15, 60 mg/kg bw	Daily for 90 days (some >21 days lactation)	Immune and reproductive alterations in adults, with developmental effects in offspring.	[20]
23	Wistar rats	PS	25 and 50 nm	Drinking water	1, 3, 6, 10 mg/kg bw	Daily for 5 weeks	No significant effects on body weight or neurobehavior.	[115]
24	Wistar rats	PS	0.5 μm	Drinking water	0.5, 5, 50 mg/L	Daily for 90 days	Cardiac accumulation induced oxidative stress-mediated myocardial apoptosis and fibrosis.	[116]
25	Wistar rats	PS (25 and 50 nm mixture)	25 and 50 nm mixture	Oral gavage	1, 3, 6, 10 mg/kg bw/day	Daily for 5 weeks	Mild thyroid endocrine disruption with elevated liver injury markers.	[117]
26	C57BL/6J mice	PS	0.5 μm	Oral (gavage/diet)	0.5 mg/day	4 weeks	Chronic exposure induced NK cell-mediated hepatic inflammation.	[118]

Table 11
In Vitro mechanistic evidence of cellular uptake, immune activation, and inflammatory responses induced by microplastics.

No.	Polymer	Experimental Media/Model	Size	Exposure/Concentration	Accumulation Effect	References
1	PS	Murine macrophage and epithelial cell lines	0.2–6.0 μm	Up to ≥250 μg mL^{-1}	Preferential PS uptake by macrophages with stress responses; minimal epithelial uptake.	[128]
2	PS MPs	Human-relevant cell systems (*in vitro*)	Nano–micro scale PS particles	Size- and concentration-dependent exposures	Size- and dose-dependent pro-inflammatory immune activation	[10]

chronic exposure scenarios [78]. Furthermore, microplastics may act as vectors for organic pollutants and heavy metals, potentially enhancing the toxicity of co-occurring substances. This has been demonstrated in co-exposure studies involving human liver cells and polychlorinated biphenyls (PCBs) [134]. Such complexity further complicates efforts to directly link microplastics to specific health outcomes.

Microplastics may pose potential risks to human health. While animal studies and quasi-experimental investigations provide valuable mechanistic insights, there remains a lack of data on internal human doses of microplastics and an absence of clinically relevant evidence linking exposure to adverse health outcomes. This is the primary reason why a direct causal relationship between microplastic exposure and adverse health effects has not yet been established.

3.5.4. *Absence of epidemiological and longitudinal human data*

There are numerous reports documenting the presence of microplastics in different human biological matrices; however, evidence linking microplastic exposure to relevant population-level health consequences from an epidemiological and longitudinal perspective remains a significant gap in the literature. Many human studies to date are limited to the detection and characterization of microplastics in blood, lungs, placenta, feces, and other tissues, without establishing causal relationships with specific health outcomes [13,14]. The absence of longitudinal studies, particularly those employing cohort and case-control designs, limits the potential to establish explanatory relationships between microplastic exposure and subsequent health effects. Most estimates of health risks continue to be based on empirical studies and animal models, which serve as the primary basis for these estimates and the main source of exposure ranges, yet may underestimate actual human exposure risks [85,91]. Furthermore, the chronic and lifelong nature of microplastic exposure, coupled with the absence of systematic evaluations of latent, cumulative, or transgenerational effects, as well as unexplained outcomes, further contributes to uncertainty [97].

The breadth of particulate exposure suggests that the lack of early evidence does not imply the absence of risk. Studies have demonstrated the toxicity of certain particulate matter, such as PM_{10} and $PM_{2.5}$, due to their chemical composition and size distribution [135], and the history of asbestos provides an example of materials once considered safe that were later found to be carcinogenic based on long-term epidemiological evidence [136]. Given the current and increasing global reliance on plastics, these parallels are highly relevant. The most robust epidemiological evidence currently available comes from occupational studies involving high levels of inhalation exposure. Evidence on the adverse health effects of nylon flock fibers and PVC dust exposure has documented pulmonary inflammation, interstitial fibrosis, restrictive lung disease, and an increased risk of lung cancer [135,137]. Although direct causal relationships have not yet been fully established, these associations cannot be dismissed, particularly in light of observed health improvements following cessation of exposure. Recent research has also shown that inhalation of microplastics occurs in various occupational groups, such as couriers and workers in the footwear industry, with microplastics identified in sputum, nasal lavage, and bronchoalveolar lavage fluids [138]. Nonetheless, these studies rarely assess specific health outcomes.

Although existing studies have illustrated multiple pathways through which humans may be exposed to microplastics, the assessment of associated health risks remains limited due to the absence of comprehensive epidemiological and longitudinal research. Until there is clearer delineation of exposure–outcome relationships, improved estimation of internal doses, and better characterization of population-level burdens, it remains difficult to establish direct causal links between microplastic exposure and adverse health effects.

4. Conclusion

The presence of MPs in human biological matrices is well-documented and not disputed to avoid any misinterpretation. This analysis confirms detection in feces, blood, lung tissue, placenta, urine, and breast milk. However, detection alone does not constitute evidence of a biologically significant internal dose or health risk. This study synthesized evidence from 197 Scopus-indexed articles (2015–2025) and critically evaluated HBM data on MPs exposure, internal dose, and health risk. The results confirm the presence of MPs in a variety of human matrices, including feces (1–138.9 MP g^{-1}), blood (1.6 μg mL^{-1} or 4.2 particles mL^{-1}), lung tissue samples (~0.69 ± 0.84 MP g^{-1}), placenta (0.28–41.8 MP g^{-1}), urine (particle size range: 4–15 μm), and breast milk (0–2.72 MP g^{-1}). Despite widespread detection, such measurements primarily reflect external exposure and biological presence and do not provide a quantifiable and biologically relevant internal dose. Ingestion and inhalation remain the primary exposure routes; however, current evidence suggests that less than 1% of ingested or inhaled particles are absorbed into tissues, as most MPs are cleared from organ systems via biological clearance pathways, including mucociliary

transport (inhaled MPs), gastrointestinal excretion (ingested MPs), and phagocytic uptake by macrophages for intracellular degradation. Particle behavior is size-dependent, imposing further constraints on systemic exposure. Translocation beyond primary entry barriers (e.g., epithelia) is predominantly observed for particles with diameters <5–20 μm (or nanoscale fractions). High methodological heterogeneity represents a primary limitation in the reviewed studies, including variations in detection methods (e.g., Py-GC/MS vs. FTIR/Raman), size detection thresholds (≥700 nm or ≥10–20 μm), and reported metrics (mass versus particle counts). These disparities result in heterogeneous findings that compromise comparability across studies, thereby precluding the establishment of standardized internal dose parameters. Furthermore, toxicological evidence has largely relied on experimental approaches using exposure levels many orders of magnitude higher than realistic environmental conditions, thus providing limited direct relevance to human health risk assessments. Importantly, no study has established a causal relationship between the detection of MPs in human tissues and clinically observable adverse health effects. The lack of longitudinal and epidemiological data, along with the absence of validated dose–response frameworks, remains a major challenge for risk characterization. Current HBM evidence indicates human exposure rather than a confirmed health risk. Future studies should focus on (i) standardization of internal dose metrics, (ii) biokinetic and particle fate modeling, and (iii) large-scale epidemiological investigations that explicitly connect quantified internal exposure with clinically relevant endpoints. Two key gaps must be addressed to transition from detection-based science to robust, evidence-based human health risk assessment of microplastics.

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CRediT authorship contribution statement

Adil Syah Johari: Data curation, Formal analysis. **Mochamad Arief Budihardjo:** Conceptualization, Data curation, Formal analysis. **Syafrudin:** Resources, Software. **Annisa Sila Puspita:** Writing – original draft, Writing – review & editing. **Amin Chegenizadeh:** Software, Validation, Visualization. **Akbar Bayu Saputro:** Methodology, Project administration, Visualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

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