

Article

Microplastics and Human Health: Exposure Pathways, Contributive Roles, and Mitigation Strategies

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ABSTRACT

The sophistication of human life with the inventions and infrastructure development that followed the industrial revolution has one unintended consequence—plastic pollution. Their exponential growth, accumulation in all the environmental systems, persistence and low rate of decay have made them unique polluters of this century, to which no wise management practices and mitigation strategies are known currently. The micro/nano sizes of plastic debris not only concerned in the natural environment; they also possess emergency risks to human health. Through an extensive literature search, we retrieved a total of 4583 records from the Scopus database appeared in last decade (2016–2025). By adopting PRISMA guidelines, we selected 932 literatures for extensive scoping review of the exposure pathways and mechanistic evidence linking microplastics to human health. The review has synthesized the evidences on microplastics contamination of external and internal human systems and traced the occurrence, types, abundance, distribution, and modes of transportation and bioaccumulation reported in the literature. The dominant mode of microplastics transport includes ingestion, followed by inhalation, dermal contact, and injection. Translocation of microplastics through circulatory systems within the human body endangers vital organs including brain and placental tissues, raising concern about potential systemic health risks. Review and examination of the microplastics exposure to humans with reference to the relative vulnerabilities of various organs revealed that, the gastrointestinal tract-digestive system, followed by liver-hepatic system, kidney-renal system, lungs-respiratory system, brain and nerves-the nervous system, reproductive system, cardiovascular and circulatory system, skin-integumentary system and lymphoid organs and tissues-immune system are all prone to microplastic-nanoplastic contaminants and are subjected to various levels of threats. Owing to their bioaccumulation capacity, slow rate of decay and persistence in almost all the media, effective mitigation/management strategies are yet to be devised and their effectiveness in tackling the menace is validated. With about 50 primary types of polymers, producing over 60,000 formulations, at an approximate rate of 8% increase every year, result in an alarming rate of plastic refuse in natural environments, often unmanaged is a looming threat to environmental systems and ecological niches. Addressing this situation requires urgent, policy initiatives, and microscale management practices, that include plans and strategies for source reduction, environmental intervention, legal and mandatory management/reuse/recycle practices at microscale.

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Research Highlights

- Systematic Scoping review of 932 studies out of 4583 records from the Scopus database (2016–2025) on microplastics and human health.
- Microplastics enter human circulatory system through various pathways and threatens multiple organ systems.
- Source reduction, environmental intervention, policy initiatives, and microscale management strategies are recommended.

1. Introduction

Plastics have become an integral part of daily life in this anthropocene era due to its convenience and comfort to use, and resistance to degradation property [1]. The commercial manufacturing of plastics had begun in the mid-20th century, and since then the production has increased substantially, to meet the exponential growth of their usage. The less expensive, water and corrosion resistance, chemically inert, thermal and electrical insulation properties, and its impressive cost to performance ratio make plastic an easy and efficient alternative for the conventional material for household and industrial usage [2]. Despite its benefits, the non-existent and or poor disposal practices, the plastic debris has become a major part of solid waste causing serious environmental problem. It is estimated that only 9% of the plastic waste generated globally are recycled, while the significant proportion end up in landfills or not properly managed [3]. It is estimated that, in 2010, 192 coastal countries have generated around 275 million metric tons (MT) of plastic debris, of which 4.8 to 12.7 million MT entered the ocean [4]. The widespread application of plastics in contemporary society, with an estimated 320 to 390 million tons manufactured annually, with a substantial portion accumulating in landfills and natural environments, eventually migrating to aquatic ecosystems, has led to significant environmental issues owing to their persistence/very low rate of decay [5–7]. Once plastic debris is exposed to the environment, due to mechanical, chemical and biological actions, they become disintegrated into smaller fragments and get accumulated [2, 8]. This ubiquity, coupled with low degradation rates, results in the fragmentation of larger plastic items into microplastics (MPs) (1 μm –5 mm) or nanoplastics (NPs) (<1000 nm), which are now pervasive across global ecosystems [9–11] ranging from icy peaks of mountains to the abyssal depths of oceans, in addition to the vast plastic floating islands supporting a plethora of micro-macro organisms in the open oceans, that earned them the dubious name *plastisphere* [12].

Microplastics are defined as plastic debris having a size of less than 5 mm diameter [12], often concerned as a severe contaminant to the environment and possesses several threats to living things [10]. Microplastics, depending on their origin, can be classified into primary microplastics and secondary microplastics. The primary

microplastics are generated from the raw materials for manufacturing consumer plastic goods, while secondary microplastics are derived from the progressive degradation of macroplastics once it is exposed to the environment. Majority of the microplastics in the environment which possesses public health risk are of secondary origin, mainly derived from the larger plastic litter [13, 14]. The minute secondary plastic particles pose an escalating risk to human and environmental health due to their continuous accumulation and the potential for widespread exposure through various pathways [15, 16]. Due to their microscopic size and chemical composition, they could bypass the biological barriers and potentially accumulate in the vital organs and even infiltrate the circulatory system, causing immune disorders, inflammation, and systemic toxicity [17, 18]. Microplastics, while being transferred through the food chain, they potentially could carry harmful chemicals such as persistent organic pollutants (POPs), further intensifies its impact on human health [7]. The pervasive presence and the potential health impact of the microplastics necessitate a comprehensive understanding of their formation, toxicological profiles, and the subsequent detrimental health effects [19].

Several recent reviews have studied microplastics and human health, but each has focused on a specific aspect of the topic. Existing systematic reviews have generally focused on a limited number of organs or health outcomes. For example, [20] examined digestive, reproductive, and respiratory effects, while [21] focused specifically on liver toxicity. Ref. [22] explored the presence and distribution of microplastics in human tissues but did not comprehensively assess their related health effects. Recent studies [23, 24] have examined the effects of microplastics across different organs and their interactions. However, these reviews are not PRISMA-based systematic reviews. Further, they also do not include bibliometric analyses of the research domain or provide a structured assessment of microplastics mitigation and management strategies. Concurrently, [25] has examined the scientometric and bibliometric analysis on global research trends on microplastics and public health, and [26] reviewed the environmental fate and removal technologies to mitigate microplastics pollution. While these studies provide useful insights into publication trends, research collaborations, and major keywords, they do not systematically synthesize the

available evidence on human health or assess mitigation and management approaches. This review aims to present a PRISMA-based systematic synthesis of microplastics-associated health implications spanning the major human organ systems, bibliometric mapping of the research landscape and its thematic development, and a structured evaluation of mitigation and management strategies organized across upstream, midstream, and downstream interventions.

Given cognizance to all these, there is an urgent requirement in examining the current understanding of microplastics as environmental contaminants, focusing on their ubiquitous presence and the multifaceted mechanisms through which they interact with biological systems, ultimately impacting human health [27]. This review aims to synthesize current understanding on the health impacts of microplastics to draw inferences on the challenges in their mitigation and management strategies. The review is carried out in a comprehensive manner, following the standard procedure of systematic literature review and subjective interpretation to explore the mechanisms of microplastics toxicity, including inflammatory responses, oxidative stress, and cellular dysfunction, alongside the limitations of current analytical methods.

2. Methodology

The study firstly adopted a systematic review and bibliometric analysis approach to investigate the global trend in research on microplastics and their implications for human health, with a special focus on mitigation and management strategies. An extensive literature search was conducted using the SCOPUS database to identify peer-reviewed academic publications related to microplastics and human health. The search aimed to retrieve relevant articles published within the last decade to ensure the inclusion of recent scientific evidence. The search was performed using the Boolean expression as follows:

TITLE-ABS-KEY (microplastic*) AND (TITLE-ABS-KEY (Human Health) OR TITLE-ABS-KEY (Mitigation AND Management)) AND (PUBYEAR > 2015 AND PUBYEAR < 2026) AND (LIMIT-TO (DOCTYPE, "ar") OR LIMIT-TO (DOCTYPE, "ch")) AND (LIMIT-TO (LANGUAGE, "English"))

2.1. Eligibility Criteria

Studies were eligible for inclusion if they met the following PECO-based criteria: (i) Population—studies involving human populations or human-relevant biological systems (e.g., *in vitro* human cell models, human biomonitoring studies); (ii) Exposure—studies reporting exposure to microplastics or associated additives/contaminants through any route such as ingestion, inhalation, and dermal contact; (iii) Comparator—studies with or without an explicit comparison group (e.g., varying exposure levels, exposed vs. non-exposed populations), considering the heterogeneity of the literature; and (iv) Outcome—studies reporting human health outcomes, toxicological impli-

cations, exposure pathways, or mitigation/management strategies addressing microplastics-related health risks.

To address this, the literature search strategy was designed to capture studies that explicitly discuss microplastics, their occurrence, exposure pathways, toxicological impacts, health implications in humans, mitigation and management policies and strategies to reduce such impacts. Filters were applied to limit the results to articles and book chapters published in English, excluding preprints and conference proceedings, with a publication timeframe restricted to last decade (between 2016 and 2025). There were no restrictions on study location in the search process to ensure the inclusion of recent contributions to microplastics pollution impact on human health across the globe. The literature selection process followed the four-stage procedure, including stages of identification, screening, eligibility, and inclusion, recommended by Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) [28]. Since this review aimed to map the literature on microplastics and human health and identify major research trends through bibliometric analysis, we followed the PRISMA extension for Scoping Reviews (PRISMA-ScR) [29], along with the standard PRISMA 2020 guidelines for the identification and screening stages.

2.2. Screening Process

We initially retrieved a total of 4583 records were based on the predefined search keywords from the Scopus database. The literature search was conducted in September 2026, and all retrieved records were exported for further screening and analysis. In the duplicate removal stage, ten duplicate records were identified and removed. Later, these 4573 records were screened based on titles and abstracts to determine their relevance to the research objectives.

Titles and abstracts of all records retrieved from Scopus were screened by the lead reviewer against the eligibility criteria above, using the Rayyan screening tool. Records considered irrelevant to microplastics, human health, or mitigation/management strategies at this stage were excluded. At this stage, a total of 3563 unrelated records were excluded, and only 1010 studies that met the inclusion criteria were retained for further analysis. Full texts of the remaining records were then retrieved and assessed for eligibility by the same reviewer. During full-text review stage, 78 records were removed due to the unavailability of full text and no reports were excluded on eligibility grounds ($n = 0$). To minimize the risk of selection error inherent to single-reviewer screening, a random sample of around 10% of excluded records at both the title/abstract and full-text stages was independently re-checked by the rest of the reviewers, and any discrepancies were resolved before finalizing the selection. After removing the duplicates and applying the inclusion and exclusion criteria, a total of 932 records were used for the review and bibliometric analysis. The full study selection process is summarized in the PRISMA 2020 flow diagram (Figure 1), which

reports the number of records identified, screened, excluded, sought for retrieval, not retrieved, and ultimately included at each stage. An array of subject fields, medium of plastic pollution and modes of transport and bioaccumulation could be discerned from perusal of these 932 publications.

2.3. Bibliometric Analysis

Bibliometric analysis was conducted to identify keyword connection, keyword trend, citation trends, and thematic development within the field. The yearly trend of publication record and citation record for the study period (2016–2025) are given as Figure 2 and Figure 3 respectively.

We used VOSviewer Version 1.6.20 to construct network visualizations including Keyword co-occurrence networks to identify major research themes such as plastic types, pollutions and human exposure (Figure 4). While

preparing the Network Visualization diagram using the VOSviewer, we used the binary counting method; the minimum number of occurrences of a term was set as 10. Out of 21,385 terms, 678 terms met the threshold. We chose 60% of the most relevant terms (407 terms) based on the number of occurrences and relevance score for the preparation of network visualization map.

Further, we extracted the top 20 keywords and made the heat map indicating their occurrence in the last decade (Figure 5). The year-wise trend in the occurrence of these keywords for the last decade is given as Supplementary Figure S1. From the network and heat map, it is inferred that polyethylene-polypropylene microplastic samples are widely studied as they affect ecosystems and threatens humans by exposure to its toxicity. The detailed examination of pathways to human exposure, vulnerability to various human organ systems and strategies for mitigation and management are presented hereunder.

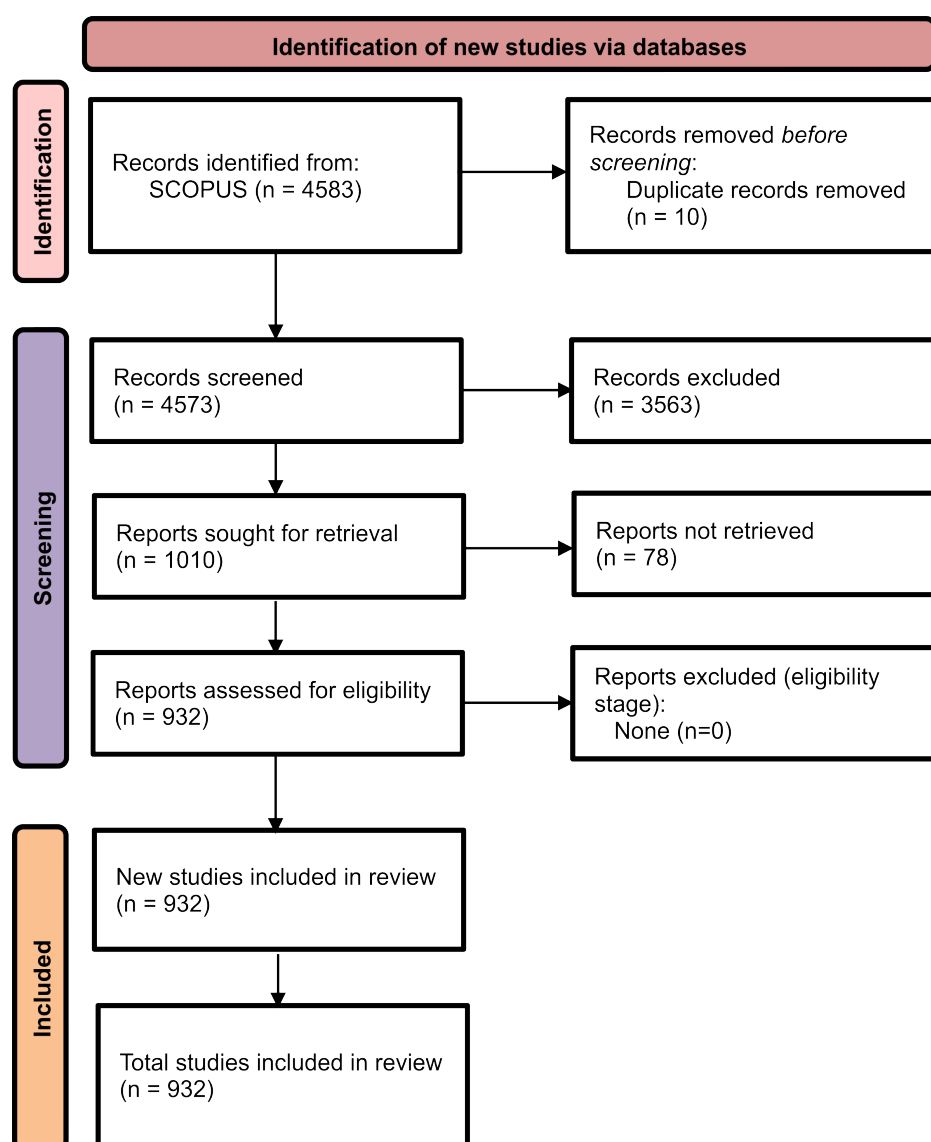


Figure 1. The PRISMA Framework adopted for the systematic literature review on human health impacts, and mitigation and management strategies as retrieved from SCOPUS database (2016–2025).

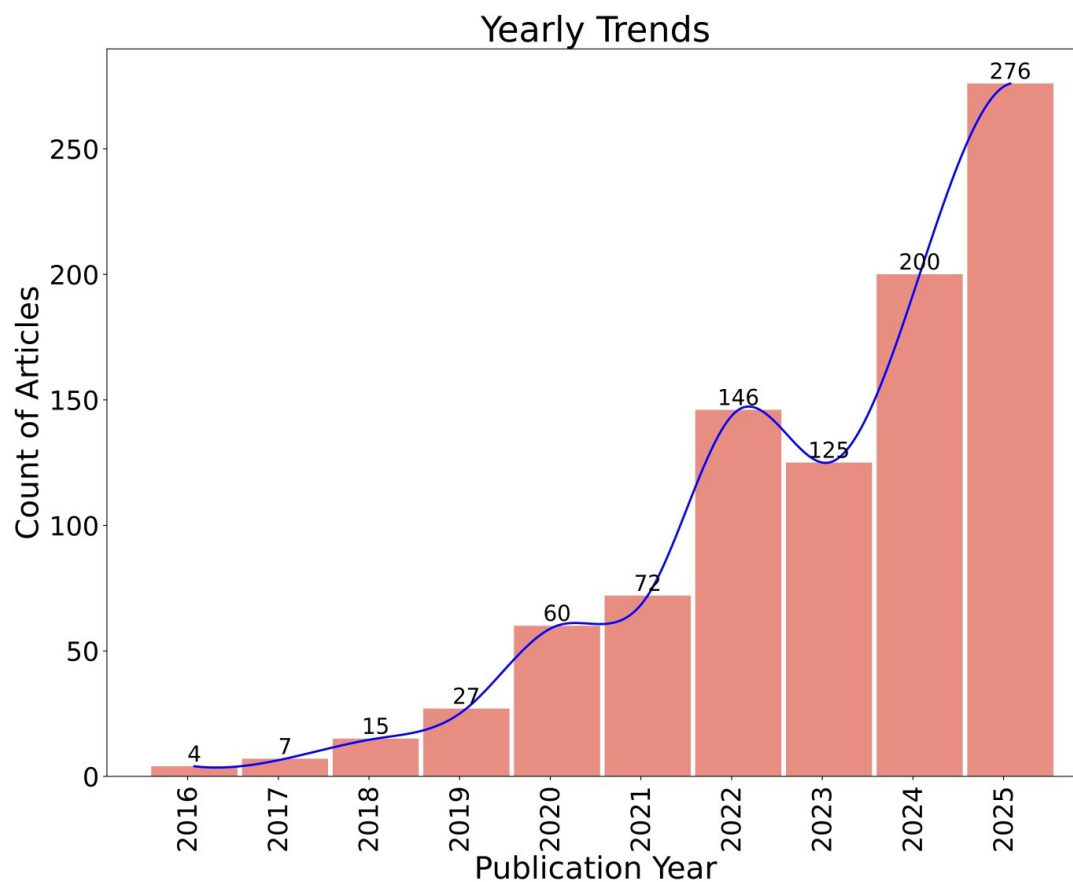


Figure 2. The yearly trend of articles published related to microplastics pollution and human health impacts, and its management and mitigation strategies as retrieved from SCOPUS database (2016–2025). An exponential increase in number of publications could be discerned from this graph.

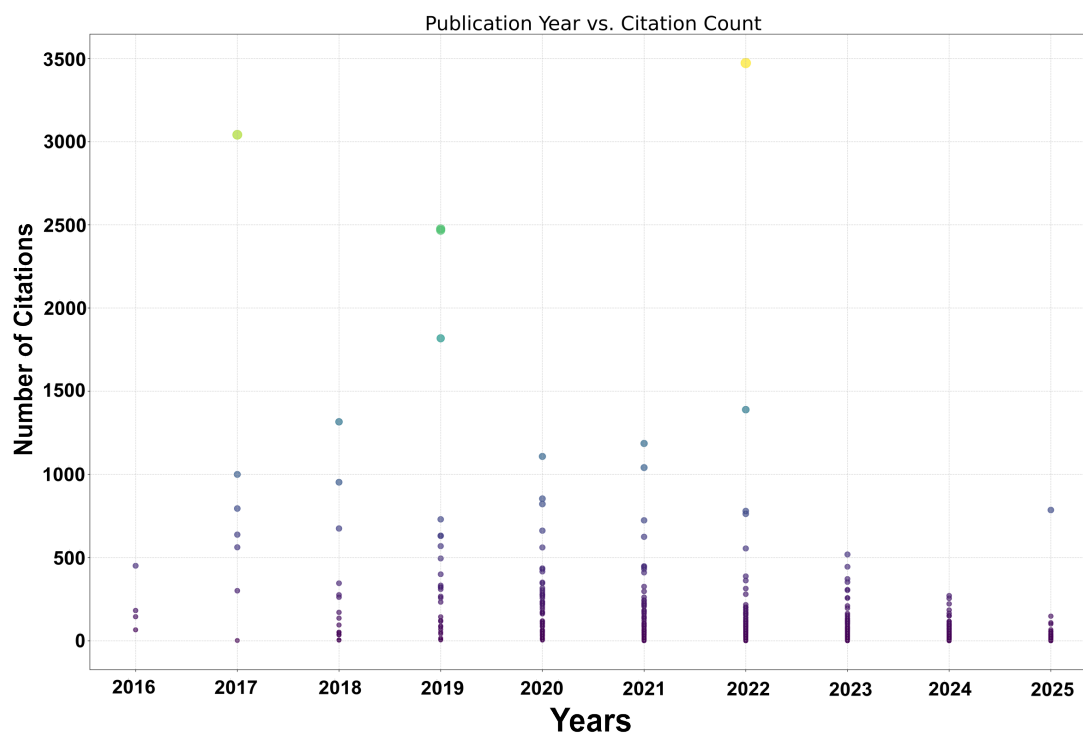
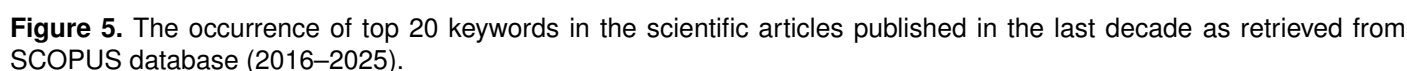
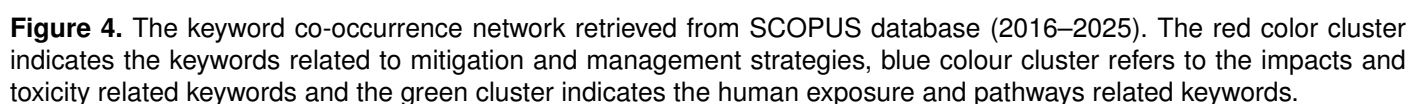


Figure 3. The yearly trend of citation count of articles related to microplastics pollution and human health impacts, and its management and mitigation strategies as retrieved from SCOPUS database (2016–2025). The increasing citation count in the last decade indicates the relevance of the research domain.



3. Pathways to Human Exposure

Microplastics can enter the human body through various exposure pathways (Figure 6). Major routes of primary exposure to MPs/NPs are: ingestion, inhalation, dermal contact, with an extra route of injection that is less studied [30, 31]. Particle size is a critical modulator of toxicity, as it has been observed that microplastics can penetrate the biological barriers including the blood-brain and placental barriers, and even microplastics can accumulate in the kidney, liver, and brain, depending upon the size, shape, surface, polymer type, etc. [31–35]. Smaller plastic particles, including NPs, have increased probability of internalization and biodistribution across cells, tissues, and organs [33]. Many studies have already identified and characterized various types of microplastics from different human body parts (Figure 7).

3.1. Ingestion

Ingestion is the principal and most critical pathway of human MP exposure. The common polymers ingested include polypropylene (PP), polyethylene (PE), polyethylene terephthalate (PET), polystyrene (PS), polytetrafluoroethylene (PTFE), and polyamide (PA) [36, 37]. Estimated annual

intake is in the range of 39,000 to 52,000 particles, which increases to an alarming level to the tune of 74,000–121,000 when inhalation exposure [37] is accounted. Contaminants are ingested through many sources, such as drinking water (bottled water commonly showing higher concentrations than tap water), seafood (fish and bivalves), table salt, sugar, honey, and beer [18, 36, 37]. MPs also enter through the degradation of food packaging, tea bags, and infant feeding bottles [38, 39].

The gastrointestinal tract (GIT) is the principal accumulation site and is the first organ system affected upon ingestion [40]. Particles up to approximately 150 μm may cross the intestinal barrier [35, 41]. Internalization takes place primarily through the gastrointestinal epithelium via specialized M-cells in Peyer's patches or by paracellular transfer, known as persorption [30, 35]. *In vitro* and animal studies suggest this exposure may contribute to inflammation, oxidative stress, gut barrier dysfunction, and dysbiosis of the gut microbiome [42]. Fecal MP concentrations in inflammatory bowel disease (IBD) patients are significantly higher compared to those in healthy individuals [43]. Following absorption, MPs translocate via the portal circulation to secondary organs, most notably the liver, kidneys, and spleen [35].

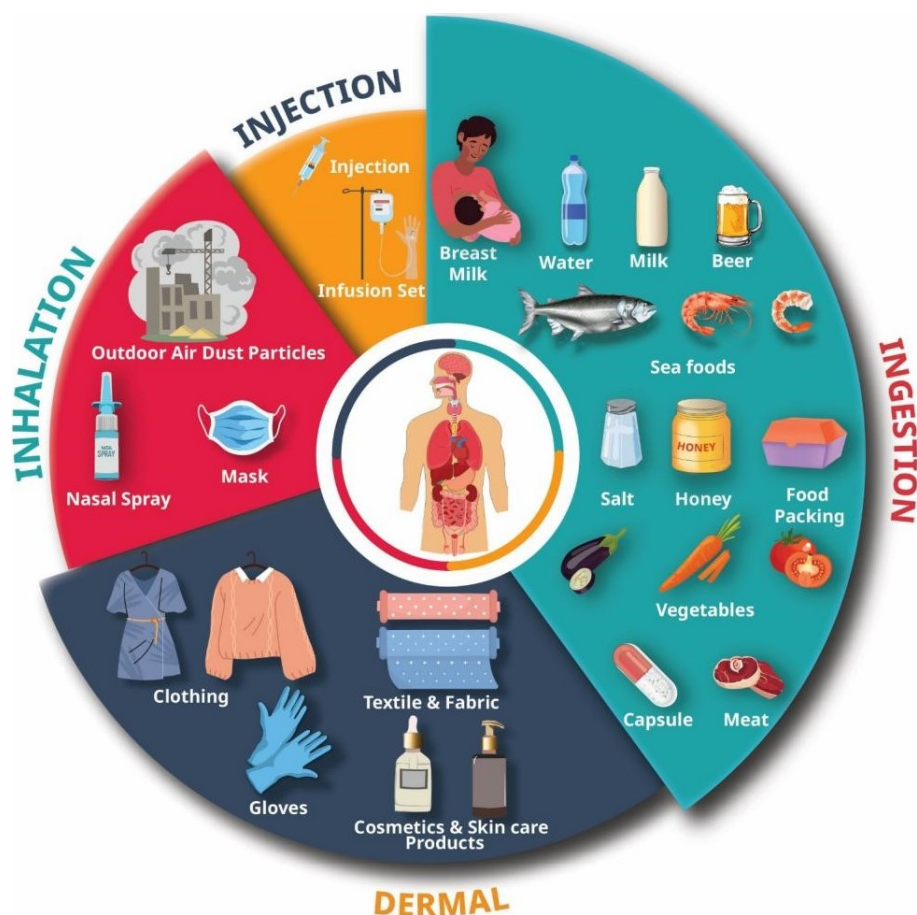


Figure 6. Human exposure pathways to microplastics and their associated sources. It is perceivable from this figure that, ubiquitous presence of MPs/NPs, make even the normal unassuming life-actions of humans invite active consumption of pollutions and lead to exponential bioaccumulation.

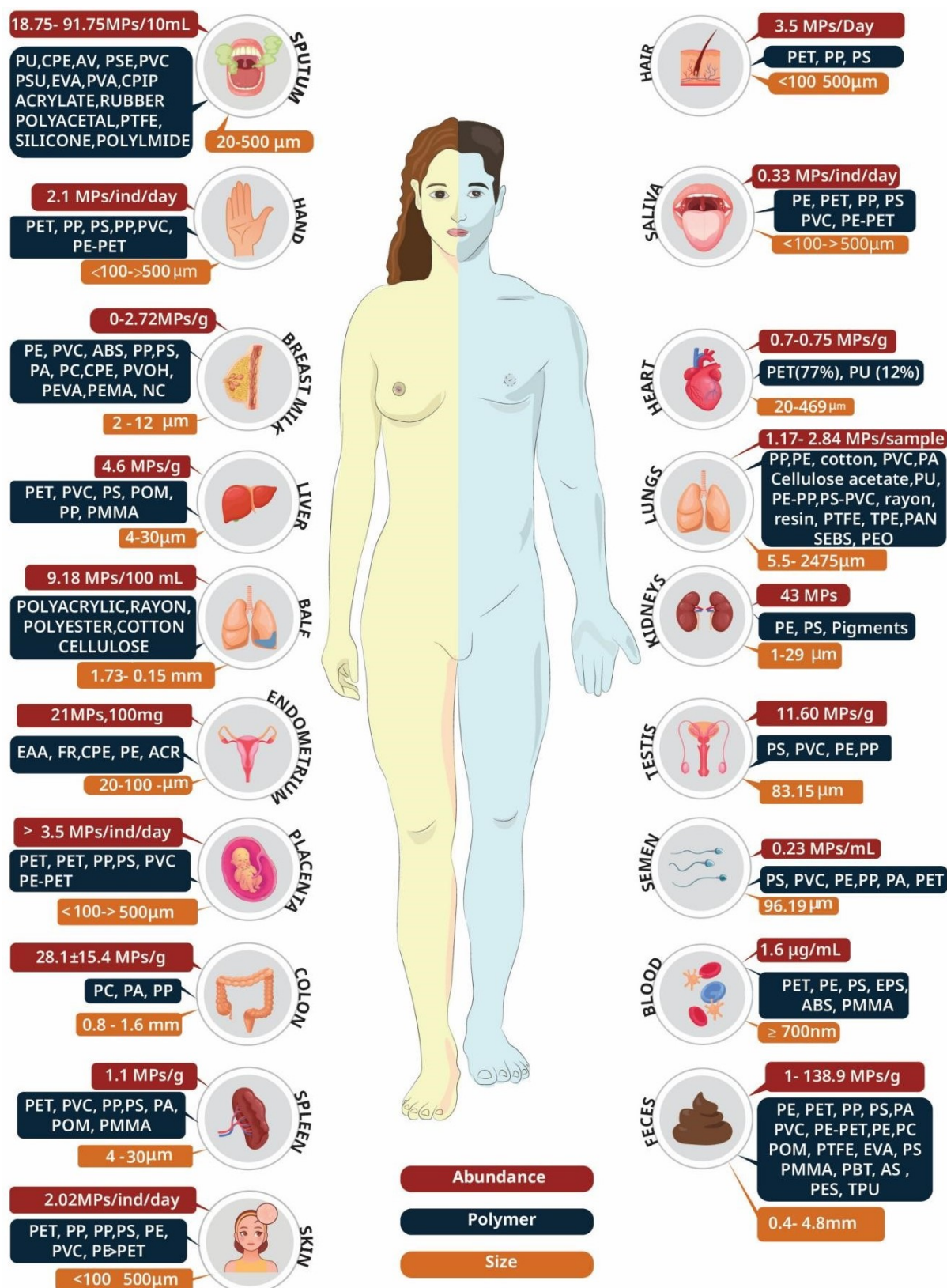


Figure 7. Schematic representation depicting the distribution, abundance, particle size ranges, and polymer types of microplastics detected in various human tissues, organs, and body fluids. Active accumulation of plastic pollutants within the human body, even while performing normal life-functions and organ specific types of pollutants can be inferred from the figure. Also recognizable is the size dependency of plastic accumulation within human body, which makes the MPs/NPs more lethal than other forms of pollution.

3.2. Inhalation

Inhalation which brings air particles into the respiratory tract and from thereon gets circulated all through the body is reported as a significant MP exposure pathway and is also considered as a subject matter of research than any other exposure pathway to human body. Commonly detected polymers in the air/atmosphere are: polyethylene (PE), polystyrene (PS), and polyethylene terephthalate [44–46]. Yet, precise data on their occurrence in varied environmental niche including, but not limited to open, meadowed, indoor, type of indoor are scarce [47]. Available data suggest that concretely, indoor air concentrations commonly ranged between 1 and 60 fibers/m³ and outdoor concentrations between 0.3 and 1.5 fibers/m³ [48]. Mainly, MPs come from synthetic textiles [37, 40], abrasion of materials such as tires and buildings [49, 50], and waste incineration [49], besides wear from road transport [47].

Particles $\leq 5 \mu\text{m}$ represent the most respirable fraction, enabling their penetration to the deep alveolar region and, thus, crossing the pulmonary barrier [35]. Particles of MPs can easily be inhaled and reach the lung tissue [35]. The respiratory system, lungs, and airways are considered the immediate target of MPs upon inhalation, where the exposure can provoke toxicity, oxidative stress, cytotoxicity, and inflammatory processes [30, 42, 46]. The presence of microfibrils and plastic particles in human lung tissue corroborates this pathway of exposure [51, 52]. Lung tissue has been suggested as a major site of retention; the highest accumulation was found to happen for particles in the range of 20–223 μm [52–54]. Chronic occupational exposure in plastic or synthetic textile industries is related to respiratory and interstitial lung diseases [49, 55].

3.3. Dermal

Dermal contact is considered a minor route of particle absorption owing to the protective efficacy of the stratum corneum [30, 56–58], but the affirmation of this presumption warrants further study and supporting data. The principal risk involves the release and absorption of chemical additives and monomers from MPs via the skin, rather than direct transdermal penetration [30, 59, 60]. Exposure sources include cosmetics and facial cleansers containing microbeads [61, 62], synthetic textile fibers, and atmospheric dust [49]. Dermal/epithelial tissue effects include oxidative stress, cytotoxicity, inflammation, and skin aging [42, 63, 64].

3.4. Injection

Exposure by injection is linked to the use of intravenous medical devices, such as syringes and infusion sets, and the solutions they carry, bypassing the natural barriers [65, 66]. The injection pathway is the least explored exposure route for microplastics. Nevertheless, it remains relevant in terms of toxicology, particularly among patients who require frequent parenteral drug administration or intravenous fluid therapy. These individuals are more vulnerable owing to their impaired physiological

states, combined with the direct entry into the systemic circulation of microplastic contaminants, thus allowing this route of distribution to reach many organ systems [67].

MPs have been detected in human blood, including the following polymers: polypropylene, polyethylene, polyethylene terephthalate, polystyrene, polytetrafluoroethylene, poly methyl methacrylate, confirming systemic transport via the circulatory system [68]. Particles $\leq 10 \mu\text{m}$ can cross the blood–brain barrier and the placental barrier. Particles introduced via injection vary from nanometers to 285.9 μm [65]. MPs in the 5–10 μm size range have been identified in the human placenta, hence indicating fetal exposure [69, 70]. After entering the bloodstream, MPs have been detected in human thrombi, implying potential risks for vascular occlusion, thrombosis, and disorders in coagulation [30, 71].

4. Microplastics Vulnerability to Human Organ Systems

Microplastics/nanoplastics infiltrate into nearly all human organ systems, causing raising risks of chronic diseases. Although decisive human studies are limited, increasing evidence from research studies underscores the importance of studying vulnerable human organ systems including gastrointestinal, liver-hepatic, renal, lungs, nervous, reproductive cardiovascular, integumentary and immune systems. Microplastics are now detected in nearly every major human organ system such as the lungs, brain, placenta, and digestive tract highlights a growing vulnerability across multiple systems.

4.1. Gastrointestinal Tract—Digestive System

The primary function of the gastrointestinal system is to digest food and help absorb nutrients for metabolism in a complex microbial environment [72, 73]. Microplastics enter the system when contaminated foods such as seafood, salt, sugar, milk and bottled water are ingested, and via swallowing inhaled particulates which have been cleared from the respiratory tract through the larynx [18, 42, 74]. Most of the larger particles, on entering, are excreted through faeces, while the smaller fragments may persist within the gut lumen, interact with biomolecules like proteins and lipids, or adhere to the apical surface of intestinal epithelial cells [70, 73] and may also form the loci for adsorption of gut materials including the semi-digested food and microbiota. Toxicokinetics involve a long residence time during which, the particles can go through biotransformation or biofilm formation mediated by colonic microbiota [18, 42]. Translocation to host tissues may occur by endocytosis via M cells of Peyer's patches, paracellular diffusion through tight junctions, or persorption, which includes the passage of particles up to 130 μm through gaps in villi, eventually resulting in accumulation in colonic tissue and systemic distribution [73, 75]. Intestinal dysbiosis, or, more specifically, the enrichment of pathobionts, may be the toxic effect of these. They also involve barrier dysfunction (leaky gut), reduced mucus secretion and local inflam-

matory responses signalled by cytokines namely, IL-6 and TNF- α [18, 76]. Mechanistic studies propose that oxidative stress and ROS-mediated epithelial cell apoptosis underlie these effects; based on largely preclinical evidence, these pathways have been hypothesized to correlate with IBD severity and colorectal/gastric cancer risk, though direct causal evidence in humans is limited [40, 77]. Experimental studies in animal models and cell cultures have demonstrated that MP exposure can induce inflammation, oxidative stress, gut barrier dysfunction, and dysbiosis of the gut microbiome [42]. However, direct evidence for these effects in humans remains limited, and the clinical significance of these findings is yet to be established.

4.2. Liver—Hepatic System

Following intestinal uptake, MPs are thought to translocate through the portal circulation or lymphatic system, although the extent of systemic transport in humans remains uncertain [42, 78]. Once in the liver, microplastics can be internalized by hepatocytes or specialized immune cells known as Kupffer cells [78]. The liver acts as a critical site for biotransformation, xenobiotic metabolism, and energy homeostasis [62, 73]. Human Toxicokinetic data suggests that chronic liver disease, such as cirrhosis, significantly favors the accumulation of MPs, as “leaky gut” conditions associated with portal hypertension facilitates higher translocation rates [78]. Bioaccumulation of different polymers namely, the PS, PVC, PET, PMMA has been confirmed in human cirrhotic liver tissue at concentrations significantly higher than in healthy controls [78]. Animal studies have shown that MP exposure can induce hepatic fibrosis, steatosis, and glucose/lipid metabolism disorders [73, 76, 79]. Whether these effects occur in humans remains unknown, though the detection of MPs in cirrhotic liver tissue [78] raises concerns that warrant further investigation. The mechanisms underlying these associations have been characterized primarily in experimental models [73, 76]. The mechanism of toxicity involves mitochondrial dysfunction and DNA damage triggered by oxidative stress, which activates signalling pathways namely, cGAS/STING and Wnt/ β -catenin, promoting the expression of pro-inflammatory cytokines and fibronectin [42, 62, 80].

4.3. Kidneys and Urinary Tract—Renal System

The renal system regulates the internal environment of the body by filtering the blood and retaining the nutrients [73]. Renal Systems functions also as the primary pathway for eliminating metabolic waste. Microplastics enter the kidneys through systemic circulation after entering through the lungs or intestines [42, 62]. In the renal system, microplastics have been reported to accumulate in the kidneys, while some particles may be excreted in bile and urine. In a preliminary study, irregularly shaped microplastic particles measuring 4–15 μm were detected in human urine samples, suggesting that microplastics can reach and be eliminated through the urinary system; how-

ever, the mechanisms responsible for their presence in urine remain unclear [73]. Experimental studies have reported elevations in nephrotoxicity biomarkers following MP exposure [62]. Additional signs of toxicity reported in animal models include histopathological changes, weight loss, and impaired renal function [62, 76]. Mechanically, microplastics induce toxicity through oxidative stress and mitochondrial reactive oxidative species (ROS) production, as well as through the stimulation of MAPK and AKT/mTOR pathways, pathways that have been proposed, based on preclinical data, as potential contributors to chemically induced carcinogenesis [62, 73].

4.4. Lungs—Respiratory System

Gas exchange is the primary function of the lungs, facilitating the uptake of oxygen and removal of carbon dioxide from the body [42, 73]. Microplastics enter the lungs via inhalation of synthetic textile, building material, and tire wear airborne particulates [42, 74]. The mucociliary escalator is responsible for clearing most microplastic particles toward the mouth so they can be swallowed; however, a subset of these smaller and lighter particles (<2.5 μm) reaches the deepest regions of the lungs (i.e., alveoli and acini) [42, 75] wherein their chances of bioaccumulation is more. Certain fibre types are highly biopersistent and are able to avoid clearance [75] creating the potential for tissue embedding. MPs can translocate across the air-blood barrier (ABB) through either passive diffusion or active cellular uptake, permitting MPs to reach the bloodstream [42, 73]. Bioaccumulation in human lung tissue, sputum, and bronchoalveolar lavage fluid are evidently documented [52, 81]. Experimental studies report pulmonary fibrosis, chronic interstitial pneumonia, and restrictive ventilatory dysfunction as toxicity effects associated with MP exposure [62, 76]. Mechanistically, the MPs induce the generation of reactive oxidative species (ROS) and resultant oxidative stress, lysosomal damage, and the release of pro-inflammatory cytokines (TNF- α , IL-6, IL-8), and disruption of the structural integrity of surfactant proteins in the lung. All these mechanisms that have been proposed as contributing factors in asthma pathogenesis and lung cancer risk, based largely on experimental and mechanistic evidence rather than direct human causal data [18, 73].

4.5. Brain and Nerves—Nervous System

The brain and the nervous system regulates learning, memory, and physiological activities [42, 73]. The brain is mainly accessed by MPs via the blood stream and by crossing the blood-brain barrier (BBB). Microplastics can also bypass the BBB using the olfactory route through the nasal cavity [42, 73]. Animal studies indicate preferential accumulation in regions including the cortex, hippocampus and cerebellum where the distribution of smaller nanoplastics (NPs; $\leq 3 \mu\text{m}$) are more likely to cross through these biological barriers than larger MPs [42, 73]. Bioaccumulation of MPs in the human brain (olfactory bulb) and cerebrospinal fluid has been confirmed [82, 83]. Toxicologi-

cal studies in animal models have reported neurotoxicity, microglial activation, and blood-brain barrier damage following MP exposure [62, 76]. These experimental findings have raised concerns about potential cognitive effects and neurodegenerative disease risk, but it must be emphasized that these associations remain speculative in humans and have not been demonstrated in epidemiological or clinical studies [62, 76]. Oxidative stress-induced cellular damage has been proposed as a major mechanism, with animal studies reporting reduced acetylcholine levels, decreased dendritic spine density, and associated cognitive deficits. Based on this preclinical evidence, some authors have hypothesized a link to increased susceptibility to neurodegenerative diseases such as Alzheimer's and Parkinson's disease; however, this remains speculative in the absence of human clinical or longitudinal epidemiological data [42, 62]. The evidence in humans is currently limited to detection studies.

4.6. Reproductive Organs—Reproductive System

Reproductive systems play essential roles in regulating hormonal balance, gamete production, and foetal development [42]. Microplastics can travel through the bloodstream and enter reproductive organs, breaking through the blood-testis barrier and through the placental barrier [18, 76]. They can accumulate in the reproductive organs, including testes, and ovaries, as well as within syncytiotrophoblasts that line the placenta [42]. Nanoparticles, which are up to 240 nm in size, appear to be particularly effective in transferring transplacentally, also reaching foetal organs such as the brain and liver [18]. Accumulations of microplastics have been detected in the human placenta, meconium, and breast milk [18, 42]. Animal studies have reported decreased sperm motility and concentration, ovarian inflammation, thinning of the uterine endometrium, and reduced pregnancy rates following MP exposure [18, 42]. Human evidence for these reproductive effects is currently lacking, though the detection of MPs in placenta and breast milk [69] underscores the need for further investigation [18, 42, 76]. The toxicity is mediated by the generation of ROS, creating oxidative stress, which activates apoptotic pathways, including p38 MAPK, disrupting maternal-fetal immune balance in experimental models; authors have speculated this could contribute to metabolic or neurodevelopmental issues in offspring, though this extrapolation from preclinical data to human transgenerational effects has not been directly demonstrated [18, 72].

4.7. Heart and Blood Vessels—Cardiovascular and Circulatory System

The cardiovascular and circulatory system is responsible for the delivery of oxygen and maintaining the body's ability to prevent excessive bleeding [73]. MPs can enter the circulatory system by being absorbed through the gastrointestinal or respiratory system [34]. MP's can attach to the surface of red blood cells (RBCs) or aggregate in

the cytoplasm of the endothelial cell or bind to plasma proteins [18, 73]. The average concentration of MPs in human blood has been determined to be 1.6 µg/mL using toxicokinetic analyses [64]. Bioaccumulation of MPs is evidenced by the discovery of MPs in human blood and human thrombi (blood clots) [18, 76]. Experimental studies report cardiovascular toxicity effects including hemolysis (the destruction of red blood cells), aortic inflammation, and impaired cardiac contractility following MP exposure [18, 73]. Whether these effects translate to human cardiovascular disease remains to be determined, although the detection of MPs in human blood and thrombi [68, 71] provides a plausible biological basis for concern. Possible mechanisms responsible for these effects include the activation of platelets, disruption of the vascular barrier, and induction of myocardial pyroptosis and cardiac fibrosis through the activation of the Wnt/ β -catenin and NLRP3/caspase-1 pathways [18, 62].

4.8. Skin—Integumentary System

As the body's first layer of protection, the skin forms a physical, chemical, and biological barrier against injury and infection [42, 72]. Physical injuries on the skin surface may allow MPs namely, the personal care products (scrubs, soaps, cosmetic items etc.), textiles and atmospheric fallout [42, 74] to penetrate. The penetration of intact skin is limited to nanoplastics that are lower than <4 nm in size (<4 nm), whereas damaged skin may allow larger (>45 nm) nanoplastic particles to penetrate through the skin. The information that larger sizes of nanoparticles have been detected inside hair follicles and sweat glands [72, 84] raises alarm bells. There are additional reports of MPs detected in the stratum corneum (layer of skin) and human hand skin [42, 74]. Types of adverse health effects from microplastic toxicity include inflammation, phototoxicity, and irritation to the skin [42, 72]. One of the proposed mechanisms of nano-plastic toxicity involves the activation of the NLRP3 inflammasome and the generation of mitochondrial reactive oxygen species (ROS) in the keratinocytes that make up the uppermost layer of the skin. This mechanism has been proposed, based on *in vitro* keratinocyte studies, as a potential contributor to proliferative skin pathology; a direct causal role in human skin cancer has not been established [18, 77].

4.9. Lymphoid Organs and Tissues—Immune System

The immune system is responsible for distinguishing between the body's own cells (self) and those that have invaded the body (non-self) and also those that are bodily-created, but are detriment to the healthy body functions, and it removes anything that is not part of the body (foreign) from the body [42, 73]. MPs enter the immune system when they are phagocytosed by macrophages and dendritic cells at the location where they enter the body [42, 73]. Inside the body, the microplastic particles will often be trapped inside a lysosome (an organelle that contains the enzymes needed to digest foreign material) of

an immune cell and afterwards will migrate to the lymphatic system (lymph nodes and spleen) [42, 74]. The toxicokinetics indicate that immune cells die approximately three times more quickly upon contact with MPs compared to other foreign bodies [77]. Studies of bioaccumulation show that MPs are found in the lymph nodes and spleen of humans, resulting in various types of granulomatous inflammation [42, 74]. Experimental toxicology data indicated that there is an overall negative effect on the immune system, with decreased production of T-cells (immunosuppression), increased levels of hypersensitivity (immunostimulation), and the increased risk for developing autoimmune diseases [42, 74]. It is important to note that clinical evidence linking MP exposure to immunosuppression or autoimmune disease in humans does not currently exist, and these findings represent mechanistic hypotheses that require further investigation [42, 74]. The mechanism includes oxidative bursts and the release of cytokines (tryptophan-6, IL-8, and TNF-alpha) and disruption of immune homeostasis [42, 73].

4.10. Interpretation of the Evidence

Evidence regarding the health effects of microplastics varies substantially across study types. Detection of MPs in human tissues confirms exposure but does not by itself demonstrate toxicity or disease causation. Most mechanistic evidence derives from *in vitro* experiments and animal models, often using particle types, sizes, concentrations, and exposure conditions that may not fully reflect human exposure. Human epidemiological studies remain limited and are largely observational, precluding causal inference. Throughout this review, experimental findings are distinguished from evidence obtained in human studies whenever possible.

5. Mitigation and Management Strategies

MPs have become ubiquitous in all the ecosystems and their extensive dispersion raises global environmental concern impacting terrestrial and aquatic ecosystems, biodiversity, and most importantly, human health [85, 86]. The pathways of microplastics into the food chain of marine, terrestrial, and other lesser known ecosystems and their detrimental effect on these organisms are documented. There is an increasing concern over the potential infiltration of these harmful elements into the food chain of human through the consumption of contaminated seafood, agricultural products, and even through atmospheric deposition [87, 88]. Every year, an estimated 1.7 million tons of plastic debris end up in the ocean, and drinking water can contain up to 4.34 microplastics particles per litre [7]. Globally, around 50 primary types of polymers and approximately 60,000 plastic formulations are available in the market [12]. The global production of such plastic materials is expected to increase by 8% every year. This surge in production has turned plastic pollution a global pressing issue and there is an urgent requirement for the formulation of strategies for effective management and reduction

[47]. Addressing this requires a multi-faceted approach encompassing source reduction, environmental interception, remediation, and robust policy interventions [89, 90]. Figure 8 represents a schematic diagram on relevance of mitigation and management strategies to reduce the human exposure to microplastics pollution. By extensive review of the literatures, it is understood that microplastics pollution mitigation and management strategies require a hierarchical approach which includes (a) upstream (source reduction), (b) midstream (interception), and (c) downstream (remediation) interventions, supported by policy and technological innovation. However, the strength of evidence and technological readiness supporting these interventions varies significantly across stages, ranging from empirically validated regulatory outcomes to early-stage laboratory demonstrations.

5.1. Source Reduction (Upstream Interventions)

The upstream intervention or the reduction of the source material is considered as the most effective strategy, as this focuses on preventing microplastics from entering the environment in the first place. This includes measures such as:

- **Reducing Plastic Production and Consumption:** Global efforts to curb the exponential growth of plastic production are crucial. This includes promoting sustainable alternatives and reducing reliance on single-use plastics [91]. The consumer behavior based contributions is an important part in regulating the market and excluding the single-use plastics from the circular economy [92]. However, evidence for this measure remains largely policy-level and reported reductions in aggregate plastic production or consumption following such initiatives are not often quantified in the literature. Further, the behavioural adoption of alternatives to single-use plastics varies widely by region and socioeconomic context.
- **Design for Degradability and Circularity:** Developing plastics that are inherently biodegradable or easily recyclable can reduce their environmental persistence. Further, implementing circular economy models, where materials are kept in use for as long as possible, and Extended Producer Responsibility (EPR) schemes shall encourage manufacturers to take greater responsibility for their products' end-of-life [86]. Though conceptually attractive, EPR schemes and circular-design mandates remain predominantly at the policy-formulation stage, with limited empirical data available so far on their measurable impact on microplastics pollution [93].
- **Eliminating Primary Microplastics:** Specific regulatory measures are vital for addressing the potential sources of primary microplastics. Legislative bans on micro-beads in personal care products, as implemented in several countries, are a direct example [94]. Some studies reported measurable declines

in microbeads concentrations in surface waters following legislative action [95]. Furthermore, stringent regulations are needed to prevent the release of pre-production plastic pellets (nurdles) from industrial settings and synthetic fibres from textiles. This might involve mandating advanced filtration systems in washing machines or improvements in textile manufacturing processes such as enclosure of operations, improved ventilation system, and use of wet scrubbers to trap particles at the point of origin itself [96]. Notably, primary microplastics constitute only a small fraction of total environmental microplas-

tics load, while the majority is coming from secondary fragmentation of larger plastic debris, which the source upstream bans do not address.

Altogether, upstream interventions offer the most preventive form of control and is the only level concerning the reduction of future microplastics generation at its origin stage. Further, none of these measures address the extensive volume of microplastics already present in soils, water bodies, and the atmosphere, highlighting the necessity of the midstream and downstream interventions discussed below.



Figure 8. Schematic diagram on the relevance of mitigation and management strategies to reduce the human exposure to microplastics pollution.

5.2. Environmental Interception (Midstream Interventions)

The midstream intervention strategies are designed to capture or reduce microplastics after they are generated but before they reach natural ecosystems such as rivers, oceans, soils, atmosphere, and biosphere. The environmental interception strategies bridge the gap between upstream intervention (source reduction) and downstream remediation (environmental clean-up). Midstream interception strategies primarily focus on designing engineering systems, altering infrastructure, and establishing environmental barriers to prevent microplastics from entering or dispersing into environmental compartments. This stage includes measures such as:

- *Optimization of Wastewater Treatment Technologies:* Wastewater treatment plants are the significant point sources for microplastics pollution and their release into the environments. Due to the varied physical and chemical properties, microplastics interact differently with the treatment technologies [97]. Adaptive treatment and processing technologies such as membrane filtering [98], tertiary treatment processes [99], ozonation followed by a granular activated carbon [100], and advanced oxidation processes (AOPs) [26] have been adopted for the microplastics pollution treatment. However, removal efficacies across these technologies vary considerably and are influenced largely by influent characteristics and treatment configuration.
- *Stormwater Management:* Urban runoff carries microplastics from tire wear, abrasions from the road markings containing synthetic resins, and degraded plastic litter. Retrofitting stormwater systems with hydrodynamic separators, biofiltration, and other green infrastructure solutions would help intercept these particles and effectively mitigate the microplastics from reaching the aquatic environment [96]. Green infrastructure demands regular maintenance to remain effective as the Captured microplastics may also be released again during heavy storms [101].
- *Fishing gear management:* Fishing gears and nets are a crucial part of the fishing industry and are manufactured from synthetic materials such as nylon, polypropylene, and polyester. Abandoned, lost, or discarded fishing gear is a major source of macroplastics that degrades into microplastics [102, 103]. Implementing “ghost gear” recovery programs and promoting more sustainable fishing practices are important. The scale and efficacy of such ghost-gear recovery programs are constrained by the funding availability, enforcement capacity, and jurisdictional limits in international waters, making their measurable contribution to reducing microplastics generation difficult to assess at a global scale.

Midstream interception is effective only when the captured material is properly managed downstream, and

its performance is regarded highly site- and technology-specific. Captured microplastics can re-enter the environment if the collected material is not properly managed, resulting that interception alone is not a comprehensive solution. This highlights the need for the remediation measures as downstream interventions discussed below.

5.3. Remediation Measures (Downstream Interventions)

Downstream interventions refer to remediation strategies applied after microplastics have already entered natural environments, such as marine ecosystems, freshwater systems, soils, or the atmosphere. Unlike upstream prevention and midstream interception, downstream remediation involves the removal, degradation, or neutralization of microplastics pollutants already present in natural environments. These strategies are essential for reducing ecological risks, protecting biodiversity, and limiting human exposure. Methods for removal of microplastics encompass physical adsorption and filtration, biological processes such as removal and ingestion, as well as chemical treatments [104].

- *Physical removal:* The ability of the microalgae to sorb tiny plastic particles has been studied [105]. The presence of an anionic polysaccharide in the algal chemical structure could efficiently sorb positively charged microplastics [104]. Engineered adsorbents, such as functionalized nanomaterials, activated carbon, biochar, and chitosan-based materials, show promise in capturing microplastics due to their high surface area and tailored functionalities [96, 106, 107]. Membrane technologies have been successful in removal of microplastics from the polluted aquatic environment. However, their efficacy depends primarily on their durability, influent flux, size, and the concentration of microplastics. It is evident that the combination of advanced filtration technologies with the biological and sedimentation processes could effectively treat the microplastics out of the wastewater [104]. Most evidence for these physical removal methods comes from laboratory-based or small-scale experiments. Their effectiveness also varies with polymer type, particle size, and environmental conditions [108]. However, studies testing these methods under real-world field conditions are still limited.
- *Chemical degradation:* Chemical methods play an increasingly crucial role in the mitigation and management of microplastics pollution, offering distinct advantages in degrading these persistent contaminants by altering the chemical structure of microplastics and ultimately breaking them down into less harmful compounds [109, 110]. The use of coagulants, for instance, iron or aluminium salts, and flocculants help to aggregate microplastics particles, making them heavier and easier to remove through sedimentation or filtration [111]. Advanced Oxidation Processes

(AOPs) such as Photo catalysis [112, 113], Fenton, Photo-Fenton, and Fenton-like processes [114], Electrochemical treatment [115, 116], and Ozonation [117] have been experimented and found to be effective for the chemical degradation of microplastics. However, methods discussed here are often requiring high amounts of energy and reagents, making them cost-intensive and limit their use at large scales. Some methods, especially advanced oxidation processes, may also produce secondary by-products whose environmental toxicity is not yet well understood [118].

- **Bioremediation:** Biological degradation, commonly referred to as bioremediation, involves the use of microorganisms such as bacteria, fungi, algae, and their enzymes to break down plastic polymers into simpler and less harmful compounds. This is an emerging and environmentally sustainable approach for mitigating microplastics pollution and involves. Bioremediation methods involve enzymatic degradation using microorganism [119], particularly involving diverse communities of microbes with complementary metabolic capabilities can lead to more effective plastic degradation [120]. However, it is evident that physical properties of plastics, such as crystallinity, can potentially limit the effectiveness of enzymatic attack [121]. The integration of crustacean nanochitosan in the water treatment systems has been found to be effective in mitigating nanoplastics in the aquatic environment [107]. Enzymatic and microbial degradation is generally slower than chemical methods, and its efficiency depends on polymer structure and environmental conditions [122]. Therefore, contemporary bioremediation methods may not be fast enough to keep up with the increasing accumulation of microplastics without substantial optimization for field-scale application.

Collectively, the downstream remediation literature is largely based on laboratory-scale proof-of-concept studies. Standardized measures for comparing the removal efficiency, cost effectiveness, and environmental safety across these methods are still lacking, and evidence of successful scaling of these technologies for field-scale deployment is still limited.

6. Conclusion

The collective evidence from published scientific studies leaves makes it known unequivocally that microplastics pose a persistent and escalating threat to ecosystems, organisms, and human health. Microplastics researches on human organ system are already established links between microplastic exposure and inflammation, immune disruption, cardiovascular disease, reproductive harm, and other potential health risks. Their persistence in tissues such as the lungs, brain, placenta, and digestive tract underscores a growing vulnerability across multiple biological systems.

Owing to their myriad formulations that account for tens of thousands of polymer varieties and their varied interactions with natural environment and degradation, devising a universal method of mitigation and management is neither feasible nor effective. Also fact is that the microplastic pollutant is here to stay and therefore targeted approaches are required—combining upstream source reduction, legal curtailments on production of single use plastics and ecosystem-specific downstream management strategies. The challenge that lies ahead is designing and implementing ecosystem-specific eradication strategies to protect biodiversity and human health against this durable pollutant.

This review has some limitations related to study design and appraisal. First, given the scale of the literature identified ($n = 932$) and the review's dual scoping and bibliometric objectives, formal quality appraisal was not applied. Second, screening, extraction, and appraisal were each conducted by a single reviewer, with only a random subset re-checked for consistency, which may introduce selection or assessment bias despite these consistency checks. Third, this review relied solely on a single database, Scopus, since it offers a broad coverage of peer-reviewed literature. However, such single-database searches may omit some studies indexed in PubMed, Web of Science, or Embase, particularly in clinical/toxicological human health studies. As a result, the synthesis presented here should be interpreted as a broad, evidence-mapping account of the microplastics–human health literature rather than a fully graded or risk-of-bias-adjusted body of evidence. Future work incorporating independent dual-reviewer screening, multi-database searching, and a full-corpus quality appraisal would strengthen confidence in the specific health-effect associations discussed.

Supplementary Materials

The supporting information can be downloaded at: <https://media.sciltp.com/articles/others/2610131457482071/ESRS-26040196-SM.pdf>.

Author Contributions

V.A.: Investigation, Methodology, Writing—original draft; K.A.P.: Investigation, Methodology, Software, Writing—original draft; K.B.: Supervision, Validation, Writing—Review & Editing; M.R.: Conceptualization, Methodology, Writing—Review & Editing; All authors have read and agreed to the current version of the manuscript.

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Conflicts of Interest

The authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies

The authors utilized the Rayyan AI assisted tool for the systematic literature review. The output was reviewed and edited as necessary by all the authors. AI-assisted tools were used for language editing and improving the clarity, grammar, and readability of the text.

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