Review

Microplastics—The Microbiota Interactions: Mechanisms, Multi-Omics Insights and Health Implications

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Microplastics—The Microbiota Interactions: Mechanisms, Multi-Omics Insights and Health Implications

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Abstract

Microplastics (MPs) are pervasive environmental contaminants detected in terrestrial, aquatic, and human systems. Emerging evidence indicates that MPs interact dynamically with microbiota across ecosystems, including host-associated microbiota such as the human gut, influencing microbial composition, metabolic activity, and functional potential. These interactions contribute to dysbiosis, altered immune responses, and increased dissemination of antibiotic resistance genes (ARGs), posing health risks. This review synthesizes current knowledge on mechanisms of microplastic–microbiota interactions, highlighting evidence from in vitro, in vivo, and environmental studies. We discuss methodological challenges, including variability in particle types, concentrations, aging, and analytical approaches. Recent advances in multi-omics techniques provide deeper mechanistic understanding and reveal functional consequences of MP exposure. We outline critical knowledge gaps and propose future research directions, aiming to provide a comprehensive framework for evaluating ecological and human health consequences of microplastic exposure.

Keywords: microplastics; microbiota; dysbiosis; antibiotic resistance genes; multi-omics; health effects

1. Introduction

Plastic materials represent one of the most successful yet problematic innovations of modern industry. Since the mass production of plastics in the 1950s, global plastic output has dramatically increased, reaching over 400 million tons annually [1–3]. This massive influx of plastic waste has led to the emergence of persistent environmental contaminants—microplastics (MPs), defined as plastic particles with a diameter between 1 μm and 5 mm while nanoplastics (NPs) as particles within diameters ranging from 1 to 1000 nm [3–6]. Originally, microplastics appeared as a byproduct of fragmentation of larger plastic items, known as secondary microplastics, but they are also intentionally manufactured as primary microplastics, such as microbeads in cosmetics, cleaning products, and synthetic textiles [1,2,7].

Of the MP/NPs present in the body, polypropylene (PP), polyethylene terephthalate (PET) and polystyrene (PS) accounted for more than 90%. PS is the most commonly used type of plastic material in the production of food contact materials and it is, for example, one of the most toxic polymers for placental enzymes. Therefore, MPs are at the forefront of toxicity research [8,9].

The accumulation of MP/NPs in the body causes many undesirable effects, such as oxidative stress, immune system disorders, intestinal damage, neurological disorders, etc. [10,11]. PS is very difficult to recycle, resulting in a large amount of PS waste accumulating in the environment. PS-MNPs accumulate in the intestines of terrestrial mammals in addition to aquatic organisms [12].

Microplastics are present across multiple ecosystems, from oceans and rivers to soils and the atmosphere, and have even been detected in human biological systems, including blood, placenta,

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and the gastrointestinal tract [1,13–15]. Their stability, resistance to biodegradation, and bioaccumulation potential make them particularly hazardous, as they can concentrate through food webs, affecting not only the environment but also human health. In recent years, research focus has shifted toward interactions between microplastics and microbiota, complex microbial communities that inhabit soil, water, and the human gut. The microbiota plays critical roles in nutrient metabolism, immune regulation, and protection against pathogens, while dysbiosis, an imbalance in microbial community composition, is associated with a wide range of diseases, including inflammatory, metabolic, and neurological disorders [1,13,16,17].

Emerging evidence suggests that microplastics can interact with microbial communities through both direct and indirect mechanisms. One key pathway is the formation of biofilms on plastic surfaces, creating unique microhabitats referred to as the plastisphere. These biofilms often harbor pathogenic microorganisms and antibiotic resistance genes (ARGs), increasing the risk of horizontal gene transfer between microbes [18–20]. The impact of microplastics extends beyond environmental microbiota to the human gut microbiome. Ingestion through food, water, or inhalation has been linked to reductions in beneficial microbial taxa, increases in opportunistic pathogens, and disruption of metabolic functions, such as short-chain fatty acid production, which is critical for immune regulation [1,14,21]. These alterations represent a potential pathway through which microplastics may indirectly affect host health, including inflammation and impaired gut barrier function.

Environmental aging processes, including photo-oxidation, mechanical abrasion, and chemical degradation, alter the surface properties of MPs, increasing their ability to adsorb antibiotics, heavy metals, and ARGs. Consequently, aged microplastics act as vectors for contaminant and resistance gene dissemination in the environment [5,19,22,23]. Moreover, co-exposure to MPs and antibiotics has been shown to synergistically alter gut microbiota and elevate ARG prevalence in animal models, underscoring the complexity of MP-microbiota interactions [14,23]. Despite rapid growth in research output, methodological heterogeneity limits the ability to reach consensus. Variations in MP types, sizes, concentrations, and analytical techniques complicate cross-study comparisons [24,25]. Therefore, standardized protocols are critical to improve reproducibility and clarify the mechanisms by which microplastics influence microbiota.

Antibiotic resistance genes (ARGs) can potentially be transferred to pathogenic bacteria through processes such as conjugation, transformation or transduction, posing a public health risk. The presence of MPs in the body and in the environment can extend the half-life of antibiotics and promote the formation of ARGs [26,27].

2. Sources and Characteristics of Microplastics

Microplastics can be classified according to their origin as primary or secondary [28,29]. Primary microplastics are intentionally produced for specific uses, such as microbeads and capsules in cosmetics, microfibers in synthetic textiles, personal care products, or industrial abrasives. Primary microplastics can also originate from wastewater from the production of plastic products. In contrast, secondary microplastics are formed by the fragmentation of larger plastic objects that are degraded mechanically, chemically, or biologically [1,2,7]. MPs can be found as fragments, foam, films, fibers, and pellets [30]. Microplastic fragments account for up to 48% [31]. The physical properties of microplastics, including size, shape, density, and chemical composition, determine their interactions with microbial communities and biological barriers of the host. Nanoplastics have a high surface-to-volume ratio and can penetrate cell membranes, thereby influencing the structure of microbial communities and host metabolic pathways [32,33]. Nanoplastics differ from microplastics mainly in properties related to environmental transport, bioavailability and toxicity [34–36].

Different types of polymers, such as polystyrene, polyethylene, polypropylene and polymethyl methacrylate, show different abilities to adsorb pollutants and promote biofilm formation [5,22,37].

Finding effective methods for the degradation of micro- and nanoplastics is essential for the removal of plastics from the environment. [38–42]. Various degradation techniques such as photodegradation [39,43,44], thermal degradation [45], photocatalysis [38,43,46], advanced oxidation

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processes [39], electrocatalysis [40] etc. are used to degrade MPs. Due to its efficiency and sustainability, biodegradation is well approached in the latter. Many microorganisms (e.g., *Bacillus subtilis*, *Pseudomonas aeruginosa*, *Clostridium thermocellum*, etc.) produce enzymes that are capable of carrying out biodegradation naturally in the environment. Various bacteria, fungi, algae and insects are able to completely digest MPs and finally convert them into sustainable waste products such as CO2 and H2O [47–50].

Environmental degradation of microplastics occurs through multiple processes. Physical degradation involves mechanical wear, crushing or friction. Chemical degradation involves hydrolytic or oxidative transformations of polymer chains. Biological degradation is often slow and depends on the presence of specific microorganisms capable of degrading polymers [51]. Aging and degradation change the surface properties of microplastics, increasing their ability to bind metals, antibiotics and ARGs (antibiotic resistance genes) [19,22]. The transport and distribution of microplastics depend on their density and interactions with the surrounding environment. Lighter polymers, such as polypropylene, can accumulate on the water surface, while heavier polymers, such as polyvinyl chloride, sediment, affecting soil and benthic ecosystems. This distribution determines which microbial communities are exposed to microplastics, as well as the potential ecological and health impacts [1,52].

3. Mechanisms of Microplastic–Microbiota Interactions

The microbiota is a microbial community that performs crucial functions such as digestion, immune system regulation, and protection against pathogens. MPs interact with the microbiota, influencing the bioaccumulation of MPs in organisms, the transfer of harmful chemicals, and the degradation of MPs in the environment [53–56].

The microbiota consists of symbiotic, pathogenic and commensal microbes. In multicellular organisms, the gut microbiota can form a barrier to potential pathogens and thus prevent disease [57]. MPs contamination can cause various neurological and respiratory diseases. In a study by Sun et al. [58], mice were used as a model to monitor the impact of MPs on the microbiota. They found that the intake of polyethylene MPs caused an increase in the values of inflammatory factors, namely IL-1β, IL-6, IL-8 and IL-10. In addition, the expression of mucin in the colon was reduced and the metabolic pathway of amino acids was increased [58].

Microplastics interact with microbial communities through a combination of physical, chemical, and biological mechanisms, which collectively influence microbial composition, diversity, and functional capacity in both environmental and host-associated ecosystems. One of the primary mechanisms is the formation of biofilms, complex aggregates of microorganisms embedded in an extracellular polymeric substance (EPS) matrix. The surfaces of MPs provide a stable substrate for microbial adhesion and colonization, creating specialized microhabitats known as the plastisphere [18–20]. Biofilm formation on MPs offers microorganisms protection from environmental stressors such as UV radiation, desiccation, and toxic compounds, enhanced nutrient acquisition from adsorbed organic matter, and facilitation of horizontal gene transfer, including antibiotic resistance genes (ARGs) [59]. Aged microplastics, which undergo chemical and physical modifications over time, exhibit increased surface roughness and hydrophobicity, enhancing microbial attachment and biofilm development. These surfaces also tend to adsorb antibiotics, heavy metals, and other pollutants, creating hotspots for selection and proliferation of resistant microbial populations [2,5,22].

Biofilm formation on MPs occurs in three stages:

1. Adhesion of microorganisms: protozoa, algae, fungi, bacteria and viruses attach reversibly or irreversibly to the surface of microplastics through weak or strong interactions. At the same time, antibiotics, metal ions or endocrine disruptors adsorb to the surface of MPs through covalent bonds, hydrophobic or π–π interactions.

2. Accumulation and formation of a stable biofilm: Substances that accumulate on the surface of the biofilm facilitate the colonization of microorganisms that secrete EPS.

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3. Release of microorganisms: As a result of the degradation of MPs, microorganisms are released back into the environment.

In addition to environmental conditions such as UV radiation, pH, MPs biofilm formation is influenced by MP properties, such as color, shape, size, hydrophobicity, etc. Negative factors that do not support biofilm formation are also important: aging of microplastics, water depth, pollutants (antibiotics, heavy metals) and conjugative plasmids [2,59].

Beyond biofilm formation, MPs can influence microbial communities through direct physical and chemical interactions. Microplastic particles can cause abrasion or damage to microbial habitats in aquatic or soil environments, and ingestion in the gut can disrupt the mucosal barrier, altering microbial colonization patterns and favoring dysbiosis [14,60–62]. MPs can adsorb and release co-contaminants, including antibiotics, metals, and persistent organic pollutants, altering microbial metabolism and creating selective pressures favoring opportunistic or resistant taxa [63,64]. The stability of the gut microbiota is a key role in maintaining host health, as it plays an important role in regulating host metabolism. There are correlations between gut microbiota and host metabolism upon exposure to MPs [65–67]. In host systems, MPs can induce oxidative stress and inflammation, indirectly affecting microbial composition by altering the intestinal microenvironment [14,33,60]. Animal studies have demonstrated that ingestion of polystyrene or polyethylene MPs reduces the abundance of beneficial bacteria such as *Lactobacillus and Bifidobacterium* while increasing opportunistic pathogens such as Escherichia coli. This shift disrupts short-chain fatty acid production, compromises gut barrier integrity, and increases susceptibility to inflammation and infection [14,60].

The physicochemical properties of MPs are critical determinants of their interactions with microbial communities. Nanoplastics can penetrate cellular membranes and reach intracellular compartments, whereas larger MPs mainly interact with surface-associated microbiota [32,59]. Fibers, fragments, and spheres exhibit different adhesion characteristics, with fibers often providing larger surface areas for biofilm formation. Polymers such as polyethylene, polypropylene, and polystyrene differ in hydrophobicity, surface charge, and chemical composition, affecting microbial colonization and pollutant adsorption [5,22]. These characteristics contribute to heterogeneity in microbial responses, with certain taxa thriving on specific MP types, while others are inhibited.

MPs, due to their adsorption capabilities, serve as a vector for the transport of toxic substances [68] Depolymerization of plastics and physicochemical aging change the concentration of contaminants on the plastic surface and promote the formation of biofilm [69]. Biofilm formed on the surface of PS [70], PP [71] or PET [72] by pathogenic microbial communities is a factor in the transport of heavy metals, persistent organic pollutants as well as antibiotics. MPs covered with biofilm have a higher affinity for contaminants and facilitate the entry of sorbed pollutants into the food chain [68].

Microplastics act as vectors for antibiotic resistance genes due to their ability to serve as stable surfaces for biofilm formation, concentrate antibiotics and metals that select for resistant strains, and promote gene exchange within biofilms [2,19]. Evidence from aquatic environments indicates that MPs enhance ARG prevalence among environmental bacteria, including clinically relevant pathogens, potentially contributing to the global spread of antimicrobial resistance [19,73]. Similar patterns are observed in gut microbiota, where chronic MPs exposure can increase ARG abundance, particularly under co-exposure with antibiotics [14,23].

MPs rarely exist in isolation. They frequently interact with environmental or dietary co-contaminants, such as heavy metals, pesticides, or organic pollutants. These interactions can synergistically alter microbial metabolism, changing carbon cycling and nitrogen transformation in soils and aquatic systems, enhance microbial selection pressures favoring taxa capable of tolerating combined stressors, and increase ARG dissemination [63,74]. For example, aged MPs in sediments can adsorb both antibiotics and heavy metals, creating microenvironments where resistant microbes multiply and transfer ARGs more readily than in uncontaminated environments [22,74].

The accumulation of microplastics in the intestine, dysbiosis of the gut microbiota and effect on human health is presented on Figure 1.

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Figure 1. The accumulation of microplastics in the intestine, dysbiosis of the gut microbiota and effect on human. Developed by the authors.

4. Multi-Omics Approaches in Microplastic Research

Understanding microplastic–microbiota interactions requires integrative, high-resolution techniques that capture not only compositional changes but also functional consequences. Multi-omics approaches, including metagenomics, metatranscriptomics, metaproteomics, metabolomics, and emerging single-cell omics, provide comprehensive insights into how MPs alter microbial communities and their metabolic networks [20,21,75].

MPs and Antibiotic Resistance Genes (ARGs) pollute the environment and have adverse effects on human health. The gut microbiota is a hot spot for the accumulation and spread of ARGs. Metagenomics has been used to characterize changes in microbial ARGs in the gut and their health risks. [76,77]. Metagenomics allows characterization of microbial community structure and the functional potential encoded in their genomes, revealing shifts in taxa, ARG abundance, and metabolic gene profiles under microplastic exposure [18,19]. Studies using metagenomic sequencing have demonstrated that exposure to polystyrene or polyethylene MPs in aquatic environments can reduce microbial diversity while selectively enriching bacteria capable of biofilm formation and pollutant degradation. In the study by Gao et al. [76]. metagenomes were analyzed in human stool samples and fecal samples from mice exposed to microplastics (PE, PVC, PP, PS, and PA66) using shotgun metagenomic sequencing. Associations were found between MPs and microbial genes encoding invasion-related virulence factors, a quorum sensing system, an autoinducer and transport system, and microplastic biodegradation enzymes [76]. Moreover, MPs serve as hotspots for ARGs, with metagenomics revealing co-localization of resistance genes and mobile genetic elements, highlighting the potential for horizontal gene transfer [2,19]. High-resolution metagenomic analyses in sediment and wastewater environments further indicate that MPs may selectively enrich microbial taxa capable of metabolizing adsorbed xenobiotics, suggesting a microbial adaptation to the microplastic-mediated chemical microenvironment [7,78].

Metatranscriptomics complements metagenomic analyses by quantifying gene expression profiles, identifying active microbial functions under MPs exposure. For example, in murine gut models, MPs induce upregulation of genes associated with oxidative stress response, inflammation, and xenobiotic metabolism, reflecting microbial adaptation to altered intestinal microenvironments [14,21]. Similarly, metatranscriptomic studies in aquatic systems indicate enhanced expression of biofilm-associated genes, EPS production pathways, and stress response elements on MPs compared to surrounding waterborne microbiota [18,20]. These findings emphasize the dynamic nature of microbial responses and reveal that functional activation may occur even when compositional shifts are subtle, underscoring the value of transcript-level data.

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Metaproteomics and metabolomics provide functional readouts of microbialresponses to MPs at the protein and metabolite levels. Metaproteomic analyses reveal shifts in enzymes involved in carbon, nitrogen, and sulfur metabolism, suggesting that MPs can alter nutrient cycling in both environmental and host-associated microbiomes [74,78].

Metabolomic profiling in the gut shows that MPs can disrupt production of short-chain fatty acids, bile acids, amino acids, and other metabolites critical for immune homeostasis, potentially linking microbial dysbiosis to systemic health effects [14,60]. For instance, reductions in butyrate-producing bacteria lead to decreased epithelial barrier integrity, while increased propionate and lactate concentrations may exacerbate inflammatory signaling, highlighting the interplay between microbial metabolism and host physiology.

Integration of multi-omics datasets enables identification of key microbial taxa, metabolic pathways, and resistance genes most impacted by MPs, providing mechanistic insights that are not possible with single-omic approaches. Advanced bioinformatic tools, including network analysis, machine learning, and metabolic flux modeling, allow for predictive mapping of microbiota responses, highlighting microbial resilience and susceptibility to MPs exposure [79,80]. Despite their potential, multi-omics studies face challenges. High variability in MPs types, doses, exposure durations, and host or environmental conditions complicates data interpretation and cross-study comparisons [1,75]. Moreover, standardization of bioinformatic pipelines is critical to accurately integrate datasets, avoid biases from low-abundance taxa, and account for stochastic gene expression patterns [20,21]. Emerging single-cell multi-omics approaches may overcome some limitations by linking functional responses to specific microbial taxa, offering unprecedented resolution of MP–microbiota interactions.

5. Health Implications of Microplastic–Microbiota Interactions

MPs enter the human body through various exposure routes, such as ingestion of contaminated food or inhalation of particles from the air. After ingestion, MPs interfere with the gut microbiota, altering the diversity and abundance of microbial species [81–83].

The ingestion and inhalation of microplastics pose emerging health concerns, mediated in part by interactions with the human gut microbiota. MPs can disrupt microbial composition, reduce beneficial commensals, increase opportunistic pathogens, and alter microbial metabolic outputs, potentially affecting host physiology [14,60,84]. Dysbiosis caused by MPs has been associated with reduced short-chain fatty acid production, compromised gut barrier integrity, and elevated pro-inflammatory signaling, which can predispose individuals to gastrointestinal disorders and systemic inflammation.

A Chinese study assessed the effects of polystyrene micro/nanoplastics (PS-MNPs) on the gut microbiota and intestinal barrier after oral exposure in mice. PS-MNPs significantly altered the short-chain fatty acids that were metabolized by the gut microbiota [85].

Animal studies consistently demonstrate that chronic MPs exposure reduces *Lactobacillus and Bifidobacterium* abundance while enriching *Escherichia coli, Clostridium spp.*, and other opportunistic bacteria [14,60,86].

In addition to dysbiosis, MPs can act as vectors for antibiotic resistance genes, facilitating their spread within the gut microbiota. Co-exposure to MPs and antibiotics may synergistically enhance ARG prevalence, creating a reservoir for potential horizontal transfer to pathogens and increasing the risk of antibiotic-resistant infections [2,23]. MPs may also carry adsorbed heavy metals, persistent organic pollutants, and other xenobiotics, further influencing microbial metabolism and contributing to oxidative stress and inflammatory responses [63,87].

Beyond the gut, MPs have been detected in blood, placenta, lungs, and other tissues, indicating their potential to translocate across biological barriers [13,14]. Microbial interactions on MPs may influence immune recognition, inflammatory signaling, and systemic metabolic responses. For example, MPs can induce oxidative stress in epithelial and immune cells, alter cytokine profiles, and disrupt mucosal homeostasis, effects that are partly mediated by changes in microbial composition

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and function [60,76]. Chronic exposure may therefore have implications for inflammatory bowel disease, metabolic disorders, neuroinflammatory pathways, and even neurodevelopment, although direct evidence in humans remains limited [87–89]. Environmental co-exposures further complicate health risk assessment. MPs often adsorb pollutants, including heavy metals, polycyclic aromatic hydrocarbons, and pesticides, which can influence microbial activity and toxicity. These complex interactions underscore the need for studies that integrate microbial, chemical, and host response data, ideally through multi-omics and longitudinal designs [63,74,90]. Understanding how MPs modulate microbial communities and contribute to health effects is critical for risk assessment and for developing strategies to mitigate their impact.

The effect of microplastic exposition on biological markers and human health are presented in Table 1.

Table 1. Effects of microplastic exposure on microbiota composition, biological markers, and host responses.

Category	Parameter/ Marker	Direction of Change	Description / Mechanism	Health Implications	References
Inflammatory markers	IL-6	Increased	Upregulation of pro-inflammatory signaling pathways induced by microplastic exposure	Chronic inflammation, immune dysregulation	[14,60]
	TNF-α	Increased	Activation of innate immune responses and cytokine production	Tissue damage, inflamematory disorders	[14,21]
	Pro-inflammatory cytokines (general)	Increased	Shift toward pro-inflammatory cytokine profile	Increased risk of inflamematory diseases	[21,60]
Oxidative stress	Reactive oxygen species (ROS)	Increased	Microplastics induce oxidative stress at cellular level	Cellular damage, apoptosis	[14,60]
	Antioxidant defenses	Decreased (relative)	Imbalance or depletion of antioxidant systems	Accumulation of oxidative damage	[21]
Gut microbiota (beneficial taxa)	Lactobacillus spp.	Decreased	Disruption of gut microbial balance under MP exposure	Reduced host protection and immune regulation	[14,60]
	Bifidobacterium spp.	Decreased	Microplastic-induced dysbiosis	Impaired gut homeostasis	[14]
Gut microbiota (opportunistic/pathogenic taxa)	Escherichia coli	Increased	Selective enrichment in altered gut environment	Increased infection risk and inflammation	[14,60]

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	Clostridium spp.	Increased	Dysbiotic shift favoring opportunistic taxa	Production of harmful metabolites	[60]
Microbial metabolites	Short-chain fatty acids (SCFAs, e.g., butyrate)	Decreased	Loss of SCFA-producing bacteria	Impaired gut barrier integrity and immune regulation	[14,60]
	Other metabolites (e.g., lactate, propionate)	Altered	Microbial metabolic reprogramming	Disrupted host–microbiome interactions	[21]
Gut barrier function	Intestinal barrier integrity	Decreased	Physical disruption and inflammation-mediated damage	Increased intestinal permeability ("leaky gut")	[14,60]
Antibiotic resistance	Antibiotic resistance genes (ARGs)	Increased	Biofilm-mediated horizontal gene transfer on MPs	Spread of antimicrobial resistance	[2,19,60]
Biofilm formation	EPS production, biofilm-associated genes	Increased	MPs serve as substrate for plastisphere formation	Reservoir of pathogens and ARGs	[18,20]
Microbial function	Xenobiotic metabolism pathways	Increased	Adaptive response to pollutants adsorbed on MPs	Altered microbial metabolism	[7,21]
Systemic effects	Immune homeostasis	Disrupted	Combined effects of dysbiosis and inflammation	Increased susceptibility to chronic diseases	[21,60]

6. Knowledge Gaps and Future Directions

Despite significant progress, numerous knowledge gaps remain in understanding microplastic–microbiota interactions. Standardization in experimental design is urgently needed, including harmonization of particle types, sizes, concentrations, exposure durations, and relevant biological models [1,80].

More studies are required on long-term, chronic exposure and real-world scenarios, including mixed environmental and dietary microplastics with co-contaminants. Multi-omics integration should be prioritized to link microbial composition, functional potential, and metabolite profiles to host health outcomes [75,76].

Translational research is also lacking. Most current studies rely on in vitro or animal models, and human epidemiological evidence is sparse. Longitudinal studies in humans, integrating gut microbiome analysis with exposure assessment and health outcomes, are critical for evaluating risks associated with MP ingestion and inhalation [13,14,89]. Understanding host-microbiota-MPs interactions at molecular, cellular, and systemic levels will inform mitigation strategies, including dietary, microbial, or policy interventions aimed at reducing MP exposure and microbiota disruption.

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Emerging analytical and computational tools, including single-cell multi-omics, advanced imaging, and machine learning models, offer opportunities to elucidate causal relationships and predict functional consequences of MPs exposure. These approaches will also allow identification of microbial taxa and metabolic pathways most sensitive to MPs, potentially revealing biomarkers for environmental and health monitoring [20,75]. Collaborative efforts across disciplines microbiology, environmental sciences, toxicology, and medicine are essential to build a holistic understanding of MPs microbiota interactions and their implications for ecosystem and human health.

Conclusions

Microplastics (MPs) represent a pervasive and persistent class of environmental pollutants with the remarkable capacity to interact dynamically with microbiota across diverse ecosystems, spanning marine, freshwater, soil, and host-associated environments, including the human gastrointestinal tract. These interactions are multifaceted, encompassing biofilm formation on particle surfaces, profound shifts in microbial community composition, functional rewiring of metabolic and signaling pathways, and facilitation of horizontal gene transfer, particularly of antibiotic resistance genes (ARGs). Such processes not only alter microbial ecosystem structure but also create microenvironments that can amplify the persistence and dissemination of chemical contaminants, heavy metals, and emerging xenobiotics, highlighting the role of MPs as both physical and biochemical vectors.

Recent advances in multi-omics technologies—including metagenomics, metatranscriptomics, metaproteomics, metabolomics, and single-cell approaches—have elucidated mechanistic underpinnings of MP-microbiota interactions, revealing that MPs can drive dysbiosis, perturb host–microbial metabolic crosstalk, disrupt short-chain fatty acid production, and impair mucosal barrier integrity. These microbial and metabolic disturbances have been linked experimentally to inflammation, oxidative stress, and immune dysregulation, underscoring potential risks for gastrointestinal disorders, metabolic disease, neuroinflammation, and the broader spectrum of non-communicable diseases. Importantly, MPs also act as reservoirs for ARGs, enhancing the risk of antimicrobial resistance dissemination, a pressing global health threat.

Despite these insights, critical knowledge gaps remain. Methodological heterogeneity, including variations in particle size, polymer type, exposure route, and co-contaminant interactions, limits cross-study comparability and translation to human health contexts. Furthermore, epidemiological evidence in human populations remains sparse, and the systemic implications of chronic, low-dose exposure are largely unexplored. Addressing these challenges requires harmonized experimental frameworks, standardized exposure models, and the integration of high-resolution multi-omics with advanced computational approaches to link microbial dynamics to host phenotypes and environmental contexts.

Looking forward, a systems-level perspective is essential. Integrative research combining environmental monitoring, microbiome ecology, host-pathogen interactions, and predictive modeling can reveal critical thresholds of MPs exposure, identify sentinel microbial taxa and metabolites as biomarkers of environmental and human health risk, and inform precision mitigation strategies. Policy interventions, including regulation of plastic production, improved waste management, and risk-based exposure guidelines, must be informed by mechanistic and translational data to safeguard ecosystem integrity and human health.

In conclusion, MPs are not merely inert pollutants but active participants in shaping microbial ecology and host physiology, with the capacity to propagate chemical, biological, and antimicrobial threats. Harnessing multi-omics and interdisciplinary approaches will not only advance our mechanistic understanding of MP-microbiota interactions but also provide actionable insights for environmental stewardship, public health policy, and the development of targeted interventions. Recognizing MPs as drivers of ecological and health perturbations reframes the plastic pollution crisis, positioning microbiota-centered research as a linchpin for both environmental sustainability and human health resilience. Ultimately, elucidating the interplay between MPs, microbiota, and host

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systems represents a frontier with profound implications for predictive toxicology, global health, and the stewardship of planetary microbiome.

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