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**Review Article**

# Micro and Nanoplastics in Biological Systems: A One Health Review of Environmental Toxicity, Microbial Dysbiosis, and Human Health Risks

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## Abstract

Micro- and nanoplastics have emerged as biologically active contaminants that connect environmental pollution with ecosystem dysfunction, microbial imbalance, animal toxicity, and human health uncertainty. This One Health review critically synthesizes current evidence on the sources, physicochemical characteristics, environmental fate, biological toxicity, microbial interactions, and medical relevance of micro- and nanoplastics in biological systems. These particles originate from primary manufactured materials and secondary fragmentation of plastic waste, synthetic textiles, agricultural plastics, tyre wear, packaging, wastewater, and atmospheric deposition. Their toxicity is strongly influenced by particle size, shape, polymer type, surface chemistry, aging, biofilm formation, and contaminant adsorption. In plants and ecosystems, micro- and nanoplastics can impair seed germination, root architecture, photosynthesis, nutrient cycling, soil microbial activity, crop productivity, and ecological stability. Animal studies further demonstrate oxidative stress, inflammation, neurotoxicity, reproductive injury, developmental disruption, gut damage, and metabolic disturbance, although many experiments still rely on high doses and pristine laboratory particles. Microplastics also create plastisphere biofilms that alter soil, aquatic, and gut microbiomes and may facilitate pathogen survival and antimicrobial resistance gene dissemination. Human exposure occurs through food, drinking water, air, dust, and consumer products, with increasing detection in blood, lung, placenta, breast milk, vascular tissues, and other biological samples. However, causal links with chronic disease remain uncertain due to limitations in detection, dose standardization, contamination control, and long-term epidemiological evidence. Overall, micro- and nanoplastics represent an emerging cross-sectoral biological threat requiring standardized monitoring, realistic toxicology, biosensor development, biotechnology-based remediation, safer material design, and integrated One Health risk assessment.

**Keywords:** Microplastics; Nanoplastics; One Health; Environmental toxicity; Microbial dysbiosis; Antimicrobial resistance; Human health risk.



## INTRODUCTION

Plastic pollution has become one of the most serious environmental challenges of the twenty-first century because plastics are produced in large quantities, used in almost every sector of human life, and persist for long periods after disposal. In recent years, scientific concern has shifted from visible plastic waste to

microscopic plastic particles known as microplastics and nanoplastics. These particles are now recognized as emerging contaminants because they can move through air, water, soil, food chains, and biological systems (Jiang *et al*., 2020; Thompson *et al*., 2024).

Microplastics are generally defined as plastic particles smaller than 5 mm, while nanoplastics are usually described as plastic particles in the nanoscale range. However, the exact size boundary for nanoplastics may vary among studies, with some researchers defining them as particles below 100 nm and others extending the range up to 1000 nm (Jiang *et al*., 2020; Masseroni *et al*., 2022). Their small size, high surface-area-to-volume ratio, and ability to interact with chemicals, microbes, and biological membranes make them more concerning than larger plastic debris. The global emergence of micro- and nanoplastics is directly linked to the rapid increase in plastic production and poor waste management. A recent material-flow analysis reported that global plastic production reached approximately 400 million tonnes in 2022, with most of this production still dependent on virgin plastic rather than recycled material (Houssini *et al*., 2025). This continuous production, combined with limited recycling efficiency, has increased the accumulation of plastic residues in terrestrial, freshwater, marine, and atmospheric environments.

Micro- and nanoplastics originate from both primary and secondary sources. Primary microplastics are intentionally manufactured in small sizes for use in cosmetics, industrial abrasives, paints, coatings, textiles, and biomedical or consumer products. Secondary microplastics are formed when larger plastic materials break down due to sunlight, heat, mechanical abrasion, oxidation, and biological weathering (Jiang *et al*., 2020; Thompson *et al*., 2024). Over time, these processes generate smaller particles that can persist in the environment and become widely distributed. Rivers are one of the major transport pathways through which land-based plastic waste enters marine ecosystems. Meijer *et al.,* (2021) estimated that more than 1000 rivers are responsible for nearly 80% of global riverine plastic emissions into the oceans. This shows that plastic pollution is not only a marine problem but also a land-based and freshwater problem. Urban runoff, wastewater discharge, agricultural drainage, industrial waste, and mismanaged municipal waste all contribute to the movement of plastic particles across ecosystems. Recent studies also show that microplastics are not restricted to water bodies. Atmospheric transport has become an important pathway for the global movement of plastic particles. Airborne microplastics can travel over long distances and deposit in remote environments, including mountains, agricultural areas, polar regions, and inland ecosystems (Brahney *et al*., 2021; Thompson *et al*., 2024). This indicates that micro- and nanoplastics are mobile, persistent, and transboundary pollutants.

The biological concern of micro- and nanoplastic pollution is increasing because these particles can enter living organisms through ingestion, inhalation, dermal contact, and food-chain transfer. Microplastics have been reported in food, drinking water, air, aquatic organisms, terrestrial animals, and human biological samples (Li *et al*., 2023; Thompson *et al*., 2024). Experimental studies suggest that exposure to micro- and nanoplastics may be associated with oxidative stress, inflammation, immune disturbance, metabolic changes, reproductive toxicity, and gut microbiome imbalance. However, human evidence is still developing, and more standardized long-term studies are needed to confirm causal health effects (Li *et al*., 2023; Tran *et al*., 2026). From a One Health perspective, micro- and nanoplastics represent a shared environmental, biological, and medical concern. These particles connect polluted ecosystems with plants, animals, microbes, food chains, and human populations. Their ability to persist in the environment, interact with microbial communities, carry chemical pollutants, and enter biological tissues makes them important for environmental toxicology, microbiology, zoology, biotechnology, and medicine. Therefore, understanding the global emergence of micro- and nanoplastic pollution is essential for risk assessment, public health protection, and the development of sustainable monitoring and remediation strategies.

Micro- and nanoplastics are highly relevant to biological systems because their small size allows them to interact with living organisms at ecological, physiological, cellular, and molecular levels. Unlike larger plastic debris, microplastics and especially nanoplastics can become bioavailable to plants, microorganisms, animals, and humans through soil, water, air, food, and biological surfaces. Their biological relevance is not limited to physical presence; these particles may also act as carriers of chemical additives, heavy metals, persistent organic pollutants, antibiotics, and microorganisms, thereby increasing their potential to disturb normal biological functions (Alqahtani *et al*., 2023; Thompson *et al*., 2024). Recent reviews emphasize that microplastics are now widely distributed in natural environments and that evidence of biological effects is reported across different levels of biological organization, from cells and tissues to organisms and ecosystems (Thompson *et al*., 2024).

The relevance of micro- and nanoplastics to plant systems is particularly important because plants are the first biological entry point in many terrestrial food chains. In agricultural environments, micro- and nanoplastics may originate from plastic mulch films, sewage sludge, compost fertilizers, irrigation water, pesticide packaging, atmospheric deposition, and municipal waste. Once present in soil or on plant surfaces, these particles can interact with roots, leaves, rhizosphere microorganisms, and edible plant tissues. Lin *et al.,* (2025) reported that micro- and nanoplastics

may enter plants through endocytosis, apoplast pathways, cracks in root tissues, and leaf stomata. Their review further described possible effects on seed germination, plant growth, photosynthesis, oxidative stress, antioxidant defenses, cytotoxicity, genotoxicity, fruit yield, and nutritional quality (Lin *et al*., 2025). Micro- and nanoplastics are also relevant to microbial systems because they can modify microbial habitats and community composition. In soil, aquatic systems, and the gut, plastic particles provide artificial surfaces for microbial attachment and biofilm formation. These plastic-associated microbial communities are often referred to as the "plastisphere." This is biologically significant because microbial biofilms on plastic surfaces may alter nutrient cycling, pathogen survival, and the movement of antibiotic-resistance genes. In gut systems, microplastic exposure has been associated with changes in intestinal microbial balance, barrier function, and inflammatory responses, although the exact mechanisms remain under investigation (Li *et al*., 2023; Tran *et al*., 2026). The connection between microplastics, microbial dysbiosis, and antimicrobial resistance makes this issue important for microbiology, environmental biotechnology, and public health.

In animal biological systems, micro- and nanoplastics are relevant because they can be ingested, accumulated, and transferred through food webs. Aquatic organisms, soil invertebrates, insects, fish, birds, and mammals may encounter these particles through contaminated food, water, sediment, and air. Experimental studies and reviews suggest that exposure may cause oxidative stress, inflammation, metabolic disturbance, neurotoxicity, reproductive toxicity, developmental toxicity, and immune alteration in different animal models (Alqahtani *et al*., 2023; Li *et al*., 2023). Zebrafish, rodents, and invertebrate models are commonly used to investigate the biological behavior of micro- and nanoplastics because they allow researchers to examine tissue accumulation, gut injury, microbiome changes, developmental effects, and molecular toxicity pathways.

Nanoplastics are especially important in biological systems because they may behave differently from larger microplastics. Due to their nanoscale size, large surface-area-to-volume ratio, and high surface reactivity, nanoplastics may show greater cellular uptake, stronger interaction with proteins and membranes, and higher potential to cross biological barriers. Tran *et al.,* (2026) noted that nanoplastics may have enhanced cellular uptake and greater translocation across biological barriers compared with larger microplastic particles. However, current human studies often fail to clearly distinguish between microplastics and nanoplastics because analytical methods are still limited, particularly in complex biological tissues (Tran *et al*., 2026). This makes nanoplastics a major concern in nanotoxicology and biomedical research. Human biological relevance is increasingly recognized because microplastics have been detected in several human samples and tissues. Leslie *et al.,* (2022) provided early evidence that plastic particles are bioavailable for uptake into the human bloodstream, while Jenner *et al.,* (2022) detected microplastics in human lung tissue using micro-Fourier-transform infrared spectroscopy. A 2024 scoping review also reported growing evidence of microplastics in human tissues and organs, while emphasizing that entry routes, analytical standardization, and health implications remain important research gaps (Roslan *et al*., 2024). These findings suggest that human exposure is not only external but may involve internal biological contact with tissues, blood, and organs. However, the health consequences should be interpreted carefully because many studies are observational, small-scale, and methodologically variable.

The toxicological relevance of micro- and nanoplastics is mainly linked with oxidative stress, inflammation, DNA damage, metabolic disruption, immune responses, neurotoxicity, reproductive toxicity, and developmental effects. Li *et al.,* (2023) summarized evidence from cell, organoid, and animal models and reported that microplastic exposure may induce oxidative stress, DNA damage, organ dysfunction, metabolic disorder, immune response, neurotoxicity, and reproductive or developmental toxicity. Similarly, Alqahtani *et al.,* (2023) described possible mechanisms through which micro- and nanoplastics may cross biological barriers and contribute to inflammatory reactions, metabolism-related disorders, and gastrointestinal or pulmonary effects. These mechanisms are important because they provide a biological basis for understanding how environmental exposure may translate into organism-level and health-related outcomes.

Overall, micro- and nanoplastics are relevant to biological systems because they connect environmental contamination with plant health, microbial ecology, animal toxicology, and human medicine. Their ability to interact with cells, tissues, microbiomes, biological barriers, and food chains makes them a major One Health concern. Current evidence supports their importance as emerging biological stressors, but it also highlights major knowledge gaps, especially regarding environmentally realistic exposure levels, long-term accumulation, nanoplastic detection, dose–response relationships, and direct causal links with human disease (Masseroni *et al*., 2022; Tran *et al*., 2026). Therefore, studying micro- and nanoplastics in biological systems is essential for environmental safety, food-chain protection, biomedical risk assessment, and the development of biotechnology-based detection and remediation strategies. The One Health approach is highly relevant to micro- and nanoplastic research because plastic pollution does not remain confined to one environmental compartment or one biological group. One Health recognizes that the health of humans, animals, plants, microorganisms, and ecosystems is closely connected and interdependent

(One Health High-Level Expert Panel [OHHLEP] *et al*., 2022). In the context of micro- and nanoplastics, this framework is important because plastic particles move across soil, water, air, food chains, animal bodies, microbial communities, and human tissues. Therefore, micro- and nanoplastic pollution should not be studied only as an environmental issue, but as an integrated biological and public-health concern.

Micro- and nanoplastics first enter the One Health cycle through environmental systems such as soil, freshwater, marine ecosystems, wastewater, agricultural land, and air. Once released into these systems, plastic particles may persist for long periods and interact with organic matter, chemical pollutants, heavy metals, pesticides, antibiotics, and microorganisms. Thompson *et al.,* (2024) reported that microplastics are now widely distributed in the natural environment and that evidence of harm exists at multiple levels of biological organization. This environmental persistence is important because contaminated ecosystems become continuous exposure sources for plants, animals, microbes, and humans.

The environmental importance of micro- and nanoplastics is especially clear in agricultural and aquatic ecosystems. In soil, microplastics may alter soil structure, water movement, nutrient cycling, microbial activity, and plant performance. In aquatic ecosystems, plastic particles can be ingested by plankton, invertebrates, fish, and other organisms, allowing their movement through food webs. Chaudhary *et al.,* (2025) reviewed evidence showing that microplastics can enter plant systems through roots and leaves, affect photosynthesis and nutrient uptake, and induce oxidative stress, cytological abnormalities, and genotoxicity in plants. These effects are important from a One Health perspective because plant health is directly linked with food security, animal nutrition, ecosystem stability, and human exposure through edible crops.

Animals represent a major biological link between environmental contamination and human health. Aquatic animals, terrestrial wildlife, soil organisms, livestock, and laboratory models may be exposed to micro- and nanoplastics through contaminated food, water, sediments, soil, and air. Prata *et al.,* (2021) emphasized that microplastic pollution affects animal, human, and environmental health simultaneously and should be evaluated through a transdisciplinary One Health approach. In animals, micro- and nanoplastics may cause physical blockage, feeding disturbance, oxidative stress, inflammation, reproductive impairment, developmental effects, and tissue-level toxicity depending on particle size, polymer type, dose, exposure route, and biological model. These animal effects are not isolated because animals can act as ecological indicators, food-chain intermediates, and experimental models for understanding possible human health risks. Microorganisms are another central component of the One Health importance of micro- and nanoplastics. Plastic particles provide artificial surfaces for microbial colonization and biofilm formation, creating a plastic-associated microbial habitat known as the "plastisphere." Bocci *et al.,* (2024) reported that freshwater plastisphere communities may contain diverse microbial taxa, including potentially pathogenic bacteria and mobile genetic elements such as antibiotic-resistance genes. This is important because microplastics may not only transport particles through ecosystems but may also transport microbial communities and resistance-associated genes across environmental and biological boundaries.

The connection between microplastics and antimicrobial resistance is one of the most important One Health concerns. Antimicrobial resistance already links humans, animals, microbes, agriculture, water systems, and clinical settings. Microplastics may intensify this problem by providing surfaces where bacteria can attach, form biofilms, exchange genetic material, and survive environmental stress. Witsø *et al.,* (2025) found that plastisphere samples from freshwater, raw wastewater, and treated wastewater contained antimicrobial-resistance genes and potential bacterial hosts, with wastewater-associated plastispheres showing high resistome diversity. Similarly, Joo *et al.,* (2025) described the plastisphere as a potential reservoir for antibiotic-resistance genes and antibiotic-resistant bacteria. These findings support the need to study microplastics not only as toxic particles but also as microbial and genetic vectors within the One Health framework.

The human-health relevance of micro- and nanoplastics is also increasingly recognized. Human exposure may occur through ingestion of contaminated food and water, inhalation of airborne particles, and possibly contact with consumer products. Recent reviews have reported microplastics in human biological samples and tissues, but they also emphasize that analytical methods, contamination control, exposure assessment, and causal interpretation remain major limitations (Roslan *et al*., 2024; Thompson *et al*., 2024). This means that the presence of microplastics in human samples is concerning, but health outcomes should be interpreted carefully until stronger epidemiological and mechanistic evidence is available.

A One Health approach is necessary because micro- and nanoplastic toxicity may involve combined effects rather than single-particle exposure alone. Plastic particles can interact with microbial biofilms, environmental chemicals, metals, antibiotics, pesticides, and biological fluids. After entering organisms, they may interact with gut microbiota, epithelial barriers, immune cells, and metabolic pathways. Li *et al.,* (2023) summarized evidence that microplastic exposure may be associated with oxidative stress, DNA damage, immune responses, neurotoxicity, reproductive toxicity,

developmental toxicity, and metabolic disturbance in experimental systems. These mechanisms create biological links among environmental toxicology, microbiology, zoology, biotechnology, and medicine. The One Health importance of micro- and nanoplastics is also practical for surveillance, risk assessment, and policy. If plastic pollution is studied only in water, soil, animals, microbes, or humans separately, the full pathway of exposure and harm remains incomplete. A One Health framework allows researchers to connect environmental contamination with microbial dysbiosis, animal toxicity, food-chain transfer, antimicrobial resistance, and human-health uncertainty. It also encourages collaboration among environmental scientists, microbiologists, zoologists, clinicians, toxicologists, agricultural scientists, and biotechnology researchers. Therefore, micro- and nanoplastic pollution should be treated as a cross-sectoral biological threat requiring integrated monitoring, standardized detection methods, mechanistic toxicity studies, and coordinated environmental-health policies. Overall, the One Health perspective provides the most suitable framework for understanding micro- and nanoplastics because these particles connect ecosystems, animals, microbes, and humans through shared exposure routes and biological interactions. Current evidence shows that micro- and nanoplastics can affect environmental quality, plant health, animal biology, microbial communities, antimicrobial-resistance dynamics, and potential human health outcomes. Cross-taxa nanotoxicology frameworks further demonstrate that oxidative stress, immune modulation, genotoxic and epigenetic alterations, histopathological injury, behavioral responses, and molecular biomarkers can be integrated across environmental and biomedical systems to strengthen One Health risk assessment (Khan *et al*., 2). However, stronger long-term studies are still needed to determine environmentally realistic doses, causal disease relationships, nanoplastic behavior, and combined effects with chemical and microbial contaminants. Thus, One Health is not only a conceptual approach but also a necessary scientific strategy for evaluating the full biological significance of micro- and nanoplastic pollution.

## Sources, Classification, and Environmental Fate of Micro- and Nanoplastics

### Primary and Secondary Sources of Micro- and Nanoplastics

Micro- and nanoplastics originate from multiple human activities and are generally classified into primary and secondary particles according to their mode of formation. Primary microplastics are intentionally manufactured at microscopic sizes for direct use in commercial, industrial, cosmetic, biomedical, or technological applications, whereas secondary microplastics are formed unintentionally through the fragmentation, abrasion, and weathering of larger plastic materials after production, use, or disposal (Song, 2024). This classification is important because primary sources may be controlled through product regulation and manufacturing restrictions, while secondary sources require broader interventions in waste management, material design, consumer behavior, and environmental remediation (Song, 2024; Kamalasekaran & Sundramoorthy, 2026).

Primary micro- and nanoplastics include plastic particles that are deliberately produced in small sizes for specific functions. These may include microbeads in personal care products, resin pellets used in plastic manufacturing, industrial abrasives, plastic microspheres, and engineered polymer particles used in selected pharmaceutical, biomedical, and consumer applications (Song, 2024). Such particles can enter the environment directly during production, transportation, industrial processing, product use, or disposal. Because they are already microscopic at the time of release, primary microplastics may be more readily transported through wastewater, runoff, and atmospheric pathways than larger plastic debris (Song, 2024; Kamalasekaran & Sundramoorthy, 2026).

Cosmetics and personal care products represent an important primary source of microplastics. Microplastic particles have been used in exfoliating scrubs, facial cleansers, toothpastes, shower gels, sunscreen products, and other rinse-off or leave-on formulations to improve texture, viscosity, appearance, stability, or abrasive function (Song, 2024). During routine use, these particles may be washed into domestic wastewater systems and transported to wastewater treatment plants. Although treatment plants can retain many microplastic particles, complete removal is difficult, and retained particles may later enter terrestrial environments through sewage sludge application on agricultural land (Acharya *et al*., 2021; Kamalasekaran & Sundramoorthy, 2026).

Secondary micro- and nanoplastics are produced when larger plastic materials break down into smaller fragments. This process occurs through ultraviolet radiation, thermal oxidation, mechanical abrasion, wave action, soil friction, wind transport, microbial activity, and chemical weathering (Kamalasekaran & Sundramoorthy, 2026). Common parent materials include plastic bags, bottles, food packaging, fishing nets, agricultural mulch films, greenhouse covers, synthetic ropes, foams, disposable products, and other mismanaged plastic waste. Over time, these larger materials become brittle, crack, fragment, and generate microplastic particles; with continued degradation, some particles may reach the nanoscale range (Kamalasekaran & Sundramoorthy, 2026; Song, 2024).

Agriculture is a major source of secondary microplastics in terrestrial ecosystems. Plastic mulch films, greenhouse covers, irrigation pipes, polymer-coated fertilizers, seed coatings, compost, sewage sludge, and contaminated irrigation water can introduce

plastic residues into agricultural soils (Kamalasekaran & Sundramoorthy, 2026). Once these materials are present in soil, sunlight, temperature changes, tillage, soil abrasion, and biological activity can fragment them into smaller particles. This is important because soil microplastics may persist for long periods and can interact with soil structure, water movement, nutrient cycling, plant roots, and soil microorganisms (Kamalasekaran & Sundramoorthy, 2026).

Synthetic textiles are another important source of microplastic pollution, especially in the form of microfibers. Polyester, nylon, acrylic, polypropylene, and other synthetic fabrics can release microscopic fibers during washing, wearing, drying, and textile manufacturing (Acharya *et al*., 2021). Acharya *et al.,* (2021) described synthetic textile-based microfibers as a major source of environmental microplastics and reported that laundering is an important pathway through which fibers enter wastewater systems. These fibers may be released into aquatic systems through treated effluents or transferred to terrestrial systems through sewage sludge application (Acharya *et al*., 2021).

Tyre and road-wear particles are also major sources of secondary microplastics. These particles are generated when vehicle tyres undergo abrasion during contact with road surfaces. Tyre-wear particles are complex because they may contain rubber polymers, road dust, mineral particles, brake-wear residues, and traffic-related contaminants (Giechaskiel, 2024). Giechaskiel (2024) reported that tyre abrasion contributes substantially to unintentionally released microplastics and may enter the environment through road runoff, stormwater, roadside soil deposition, river transport, and airborne dust. This makes tyre wear an important urban and transportation-related source of microplastic contamination (Giechaskiel, 2024).

Wastewater treatment plants are important pathways for both primary and secondary microplastics. They receive microplastics from household laundry, cosmetics, industrial discharge, urban runoff, textile effluents, and consumer products (Acharya *et al*., 2021; Kamalasekaran & Sundramoorthy, 2026). Although wastewater treatment can remove a large fraction of microplastics from water, particles may still be released through treated effluent, while captured particles may accumulate in sewage sludge. When sludge is used as fertilizer, microplastics may be transferred from urban wastewater systems to agricultural soils, creating a link between domestic activities, wastewater management, crop production, animal exposure, and human food-chain risks (Acharya *et al*., 2021; Kamalasekaran & Sundramoorthy, 2026).

Rivers, stormwater, and surface runoff are major transport routes for microplastics derived from land-based sources. Plastic waste from urban areas, roads, wastewater discharge, agricultural land, and mismanaged solid waste can be transported through drainage systems and rivers into marine environments (Meijer *et al*., 2021). Meijer *et al.,* (2021) estimated that more than 1000 rivers account for approximately 80% of global riverine plastic emissions into the ocean, with annual emissions ranging from 0.8 to 2.7 million metric tonnes. This evidence shows that microplastic pollution is not only a marine issue but also a land-based, freshwater, urban, agricultural, and waste-management problem (Meijer *et al*., 2021).

Atmospheric deposition is also an emerging source and transport pathway for micro- and nanoplastics. Small plastic particles and fibers released from textiles, tyres, road dust, construction materials, degraded plastic waste, and urban surfaces can become airborne and later deposit onto soil, water bodies, crops, and indoor surfaces (Kamalasekaran & Sundramoorthy, 2026). This pathway is important because it allows plastic particles to move beyond their original release site and contaminate remote or non-point-source environments. Atmospheric movement also increases the possibility of inhalation exposure in humans and animals (Kamalasekaran & Sundramoorthy, 2026).

Overall, primary sources of micro- and nanoplastics mainly include intentionally manufactured microscopic plastic particles used in cosmetics, industrial materials, plastic production, and specialized applications. In contrast, secondary sources are broader and often more difficult to control because they result from the fragmentation and wear of larger plastic products during use, disposal, and environmental weathering (Song, 2024). Major secondary sources include packaging waste, agricultural plastics, synthetic textiles, tyre wear, road dust, fishing gear, construction materials, and discarded consumer plastics (Acharya *et al*., 2021; Giechaskiel, 2024; Kamalasekaran & Sundramoorthy, 2026). Identifying these sources is essential for understanding how micro- and nanoplastics enter ecosystems, interact with microbes, accumulate in animals, contaminate food chains, and contribute to human exposure.

**Size, Shape, Polymer Type, and Surface Chemistry**

The behavior and toxicity of micro- and nanoplastics are strongly influenced by their size, shape, polymer type, and surface chemistry. These physicochemical properties determine how plastic particles move in the environment, how easily they are ingested or inhaled, how they interact with microorganisms and pollutants, and how they cross biological barriers. Recent reviews emphasize that micro- and nanoplastics should not be treated as a single uniform contaminant because their effects vary according to particle size, morphology, polymer composition, weathering state, additives, surface charge, and biofilm or biocorona formation (Lamoree *et al*., 2025; Li *et al*., 2023). Lamoree *et al.,* (2025) specifically reported that polymer type, size, shape, and biocorona

are key factors controlling the behavior and potential health effects of micro- and nanoplastics.

Particle size is one of the most important characteristics of micro- and nanoplastics because it determines environmental mobility, bioavailability, cellular interaction, and analytical detectability. Microplastics are commonly described as plastic particles smaller than 5 mm, whereas nanoplastics are generally considered smaller than 1 µm, although there is still no fully standardized global definition for nanoplastic size boundaries (Lai *et al*., 2022; Lamoree *et al*., 2025). This lack of standardization makes comparison among studies difficult because some studies classify nanoplastics as particles below 100 nm, while others extend the upper limit to 1000 nm (Lai *et al*., 2022). Smaller particles are more concerning because they have a larger surface-area-to-volume ratio and may interact more strongly with cells, proteins, lipids, pollutants, and microbial biofilms. The FDA also notes that micro- and nanoplastics occur in a wide variety of sizes and that nanoplastic detection remains especially difficult because available methods are less reliable for very small particles.

Size also affects the environmental fate of plastic particles. Larger microplastics may remain in sediments, soils, wastewater sludge, or the gastrointestinal tract of organisms, while smaller microplastics and nanoplastics may remain suspended for longer periods, travel farther, and potentially cross biological barriers more efficiently (Lamoree *et al*., 2025; Tang, 2024). In toxicological studies, particle size is frequently linked with differences in uptake and biological response. Tang (2024) noted that weathering can alter the brittleness, density, size, shape, and surface charge of microplastics, while smaller particles may show different cellular uptake and transport behavior compared with larger particles. However, size-dependent toxicity is not always consistent across studies because dose, polymer type, cell model, exposure duration, and particle preparation methods differ widely (Tang, 2024).

Particle shape is another major factor controlling microplastic behavior. Microplastics may occur as fibers, fragments, films, foams, beads, pellets, granules, or irregular particles (Lusher *et al*., 2020). These shapes are not only descriptive categories; they also provide clues about possible sources and environmental behavior. For example, fibers are commonly associated with synthetic textiles and fishing materials, films may originate from plastic bags and agricultural mulch, fragments often result from the breakdown of packaging or rigid plastics, and beads or pellets may reflect primary plastic production or personal-care product sources (Lusher *et al*., 2020; Song, 2024). Lusher *et al.,* (2020) recommended that researchers classify microplastic morphology using size, shape, texture, optical properties, and physical behavior to improve comparability among studies.

Shape affects how plastic particles move, settle, aggregate, and interact with organisms. Fibers may remain suspended or be transported over long distances because their elongated morphology can reduce settling velocity compared with spherical particles. Fragments and irregular particles may have sharp edges and rough surfaces that increase contact with biological tissues and adsorption sites for pollutants. Films and foams may behave differently due to their low density, flexibility, and larger surface area (Lusher *et al*., 2020; Tang, 2024). In a freshwater case study from the Vaal River, Saad *et al.,* (2024) reported that detected microplastics ranged from 0.06 to 4.95 mm, more than 89% were smaller than 2 mm, and the dominant shapes were fibers and fragments, showing the importance of size–shape characterization in environmental monitoring.

The polymer type of micro- and nanoplastics is also essential because different polymers have different densities, chemical structures, hydrophobicity, crystallinity, degradation behavior, and additive profiles. Commonly reported environmental polymers include polyethylene (PE), polypropylene (PP), polyethylene terephthalate (PET), polystyrene (PS), polyvinyl chloride (PVC), polyurethane (PU), polyamide (PA), and synthetic rubbers from tyre-wear particles (Li *et al*., 2023; Easton *et al*., 2025). PE and PP are often associated with packaging, films, bags, containers, and consumer plastics; PET is widely used in bottles and textile fibers; PS is associated with foams and disposable items; PVC may originate from construction and industrial materials; and tyre-wear particles arise from transport-related abrasion (Easton *et al*., 2025; Kamalasekaran & Sundramoorthy, 2026).

Polymer type influences whether a particle floats, sinks, persists, or adsorbs pollutants. Low-density polymers such as PE and PP may initially float in aquatic systems, whereas higher-density polymers such as PET and PVC are more likely to settle into sediments, although biofouling and mineral attachment can change these behaviors over time (Chen *et al*., 2025). Polymer type also affects microbial colonization. Chen *et al.,* (2025) reported that plastic material type can shape biofilm structure and function; for example, PE and PP may support different biofilm communities compared with PET, while biofilm formation can alter density, hydrophilicity, roughness, and migration behavior of particles.

Surface chemistry is a key determinant of how micro- and nanoplastics interact with pollutants, microbes, and biological tissues. Freshly produced plastic particles may have relatively smooth and hydrophobic surfaces, but environmental weathering can introduce cracks, pores, oxygen-containing functional groups, altered crystallinity, roughness, and changes in surface charge (Tang, 2024; Hanun *et al*., 2023). These surface changes can increase adsorption sites and modify

interactions with metals, antibiotics, pesticides, organic pollutants, and microbial extracellular polymeric substances. Hanun *et al.,* (2023) reported that aged PET microplastics showed surface wrinkles, cracks, oxidation-related functional groups, increased surface area, and enhanced sorption of oxybenzone compared with virgin microplastics.

Surface chemistry also controls the ability of microplastics to carry chemical contaminants. Hydrophobic interactions, electrostatic attraction, hydrogen bonding, π–π interactions, and surface roughness can all contribute to pollutant adsorption (Easton *et al*., 2025; Hanun *et al*., 2023). Easton *et al.,* (2025) examined ceftazidime adsorption on several polymer types, including PET, PE, PS, PU, and tyre-wear particles, in different shapes and sizes. Their study showed that weathered particles can have higher sorption due to cracks, fragmentation, and increased adsorption sites, with weathered PET showing the highest adsorption among the tested materials (Easton *et al*., 2025). This demonstrates why aged environmental particles may behave differently from pristine laboratory particles.

Biofilm formation further modifies surface chemistry. Once microplastics enter aquatic, soil, or wastewater environments, bacteria, fungi, algae, and extracellular polymeric substances may colonize their surfaces, forming the "plastisphere." Biofilm attachment can increase surface roughness, hydrophilicity, density, functional-group diversity, and pollutant-retention capacity (Chen *et al*., 2025). Chen *et al.,* (2025) explained that biofilms can make microplastic surfaces looser and more porous, promote pollutant capture, introduce functional groups through extracellular polymeric substances, and alter the environmental behavior of particles. This makes biofilm-coated microplastics more realistic environmental models than pristine particles used in many laboratory studies.

In biological systems, surface chemistry may also determine the formation of a biocorona, which is a layer of proteins, lipids, metabolites, and other biomolecules adsorbed onto the particle surface after contact with biological fluids. This corona can change particle size, charge, aggregation, cellular recognition, uptake, and immune response (Lamoree *et al*., 2025; Tang, 2024). Tang (2024) noted that transformed microplastics may develop surface coronas that alter zeta potential, sorption behavior, aggregation, and cellular internalization. Therefore, the biological identity of micro- and nanoplastics may differ greatly from their original manufactured identity after environmental aging or biological exposure.

Overall, size, shape, polymer type, and surface chemistry are central to understanding the environmental fate and biological effects of micro- and nanoplastics. Size influences transport, uptake, and barrier crossing; shape affects suspension, ingestion, tissue contact, and source identification; polymer type determines density, persistence, degradation, and additive composition; and surface chemistry controls adsorption, biofilm formation, pollutant transport, and biological interactions (Lusher *et al*., 2020; Lamoree *et al*., 2025; Easton *et al*., 2025). Therefore, future studies should report these characteristics clearly and consistently instead of describing micro- and nanoplastics as a single general pollutant category.

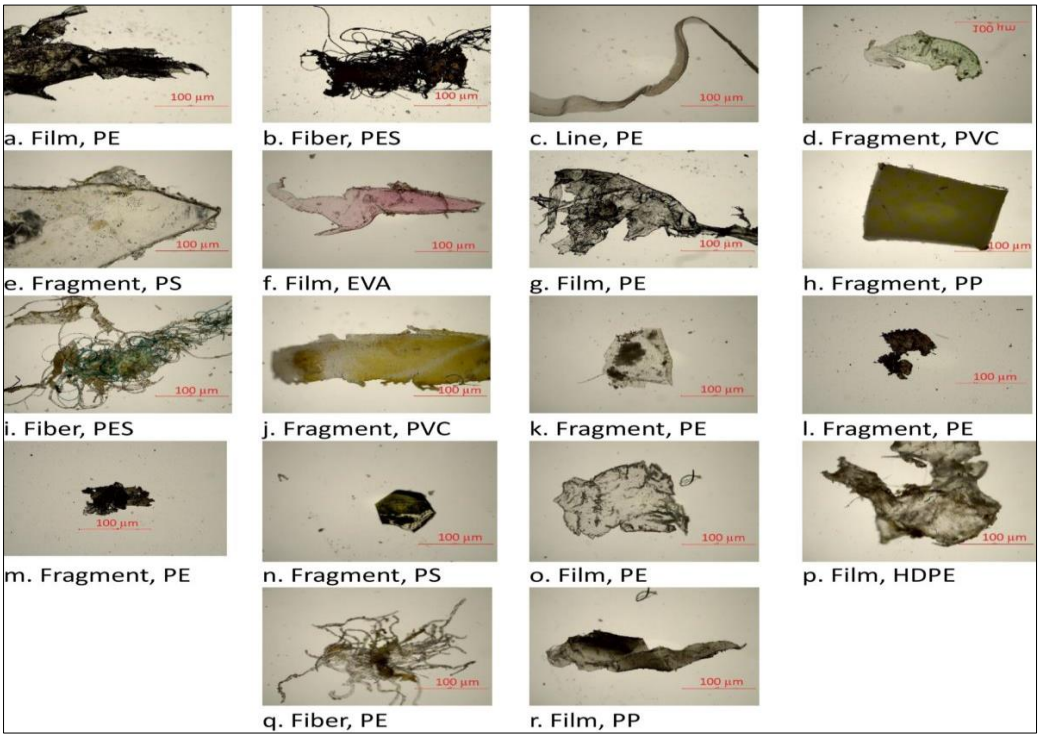

**Figure 1: Physicochemical Characterization of Microplastics Based on Size, Shape, and Polymer Type**

This figure shows representative microplastic particles with different morphologies, including films, fibers, fragments, and irregular particles, along with polymer identities such as PE, PP, PVC, PS, HDPE, and PES. It supports Section 2.2 by visually demonstrating that microplastics are not uniform contaminants; their environmental behavior and toxicity depend on particle size, morphology, polymer composition, density, and surface characteristics. 

**Persistence, Transport, and Bioavailability in Soil, Water, and Food Chains**

Micro- and nanoplastics show high environmental persistence because most conventional polymers are resistant to complete biological degradation and instead undergo slow fragmentation into smaller particles. Their long-term persistence is controlled by polymer type, particle size, crystallinity, density, additives, environmental temperature, ultraviolet radiation, oxygen availability, microbial activity, and mechanical abrasion (Kamalasekaran & Sundramoorthy, 2026). In environmental systems, this means that plastic particles may not disappear after weathering; rather, larger debris can progressively generate microplastics and nanoplastics that become more mobile and biologically available over time (Kamalasekaran & Sundramoorthy, 2026). Recent reviews therefore describe micro- and nanoplastics as persistent contaminants that can remain in soil, water, sediment, wastewater sludge, and biological systems for long periods (Rashid *et al*., 2025; Zhou *et al*., 2020).

In soil environments, micro- and nanoplastics can act both as long-term sinks and dynamic transportable particles. Agricultural soils are especially vulnerable because plastic mulching, biosolids, compost, organic fertilizers, wastewater irrigation, atmospheric deposition, and runoff can continuously introduce plastic particles into the soil matrix (Boctor *et al*., 2025). Boctor *et al.,* (2025) reported that soil can function as a major entry point for micro- and nanoplastics into terrestrial ecosystems and noted that field data usually show lower but cumulatively significant levels of contamination, often below 0.1% w/w, compared with many laboratory toxicity studies that use exaggerated concentrations. This difference is important because laboratory studies may overestimate acute toxicity, while field conditions may better reflect long-term accumulation and chronic exposure risk (Boctor *et al*., 2025).

The transport of micro- and nanoplastics in soil depends on particle properties and soil conditions. Smaller particles, low-density polymers, irregular fragments, fibers, and weathered particles may move differently through soil pores compared with larger or denser particles. Soil texture, porosity, organic matter, pH, mineral content, moisture, wet–dry cycles, root channels, and microbial biofilms can either enhance particle mobility or promote retention in specific soil layers (Azeem *et al*., 2021; Rashid *et al*., 2025). Azeem *et al.,* (2021) explained that tillage, irrigation, rainfall, wet–dry cycles, and soil organisms can promote vertical movement of plastic particles in soil. Earthworms, mites, collembolans, and other soil fauna may transport particles through burrowing, ingestion, egestion, casting, and adhesion to body surfaces, thereby increasing the movement of micro- and nanoplastics from surface soil into deeper layers (Azeem *et al*., 2021).

Micro- and nanoplastics can also influence the bioavailability of other contaminants in soil. Their surfaces may adsorb pesticides, heavy metals, antibiotics, endocrine-disrupting chemicals, persistent organic pollutants, and plastic additives. This sorption capacity depends on particle aging, surface roughness, oxygen-containing functional groups, polymer type, hydrophobicity, and soil chemistry (Azeem *et al*., 2021; Boctor *et al*., 2025). Aged particles may have cracks, pores, and oxidized surfaces that increase contaminant adsorption, while biofilms and organic matter can further alter their reactivity. As a result, micro- and nanoplastics may act as carriers that modify the persistence, mobility, and bioavailability of co-existing pollutants in soil–plant systems (Azeem *et al*., 2021; Boctor *et al*., 2025).

In aquatic environments, micro- and nanoplastics are transported by rivers, wastewater effluents, stormwater, runoff, tides, waves, currents, and sediment resuspension. Their fate depends strongly on density, size, shape, surface charge, biofouling, aggregation, and interactions with suspended particles (Pal *et al*., 2025). Low-density polymers may initially float, while high-density particles may settle into sediments. However, biofilm formation and mineral attachment can increase particle density, causing even buoyant plastics to sink or accumulate in sediments (Pal *et al*., 2025). Aquatic microplastics can also adsorb and transport contaminants such as heavy metals, persistent organic pollutants, PFAS, and other chemical residues, which may influence toxicity across aquatic food webs (Pal *et al*., 2025).

Sediments are important long-term reservoirs for micro- and nanoplastics in aquatic systems. Once particles settle, they may persist in benthic habitats and become available to sediment-feeding organisms. Resuspension during floods, storms, dredging, and hydrological disturbance can return these particles to the water column, allowing repeated exposure to plankton, invertebrates, fish, and other aquatic organisms (Pal *et al*., 2025). This cycling between water and sediment increases the environmental residence time of microplastics and supports their continuous interaction

with aquatic biota, microbes, and pollutants (Pal *et al*., 2025; Kamalasekaran & Sundramoorthy, 2026).

Bioavailability refers to the fraction of micro- and nanoplastics that can be taken up, ingested, inhaled, adsorbed, or internalized by organisms. In soil–plant systems, bioavailability depends on particle size, surface charge, polymer type, aggregation state, soil chemistry, plant species, exposure route, and root or leaf structure (Azeem *et al*., 2021; Chaudhary *et al*., 2025). Nanoplastics are generally considered more bioavailable than larger microplastics because their small size may allow closer interaction with cell walls, root pores, apoplastic spaces, xylem, and biological membranes (Azeem *et al*., 2021; Sun *et al*., 2020). Sun *et al.,* (2020) provided direct evidence that both positively and negatively charged nanoplastics can accumulate in *Arabidopsis thaliana*, with surface charge affecting accumulation pattern and biological response.

Plant uptake is especially important for food-chain exposure. Micro- and nanoplastics can interact with roots, root hairs, stomata, leaf surfaces, rhizosphere microorganisms, and vascular tissues. Chaudhary *et al.,* (2025) reported that microplastics can enter plants through roots and leaves and may translocate to stems, leaves, and edible tissues, raising concerns for food security and human exposure. Zytowski *et al.,* (2024) also demonstrated uptake and translocation of nanoplastics in edible parts of mono- and dicot vegetables, supporting the possibility that plant-based foods may become an exposure route under certain conditions. However, the extent of plant uptake remains variable and depends on particle size, surface chemistry, plant species, exposure concentration, and experimental design (Azeem *et al*., 2021; Zytowski *et al*., 2024).

In aquatic food chains, microplastics can become bioavailable through direct ingestion by plankton, molluscs, crustaceans, fish, and other organisms. Because some particles overlap in size with natural food items, they may be mistaken for prey or ingested accidentally during feeding (McHale & Sheehan, 2024). McHale and Sheehan (2024) observed trophic transmission of small plastic spheres from copepods to fish predators, showing that microplastics can move through aquatic food chains by both direct and indirect consumption. However, trophic transfer should not always be interpreted as permanent biomagnification, because retention, egestion, particle size, feeding ecology, and organism physiology strongly influence whether particles accumulate across trophic levels (Gao *et al*., 2024; McHale & Sheehan, 2024).

Food-chain bioavailability also includes human dietary exposure. Microplastics have been reported in several food and drink categories, including seafood, salt, water, milk, tea, honey, and other food matrices, although analytical methods and contamination controls vary among studies (Udovicki *et al*., 2022). Udovicki *et al.,* (2022) noted that potential toxic consequences of ingested microplastics may arise not only from the particles themselves but also from monomers, additives, sorbed contaminants, and microorganisms colonizing particle surfaces. The U.S. FDA similarly states that micro- and nanoplastics may be present in foods primarily because of environmental contamination where foods are grown or raised, but it also emphasizes that current evidence does not demonstrate that detected levels in foods pose a confirmed risk to human health (U.S. Food and Drug Administration, 2024).

Overall, the persistence, transport, and bioavailability of micro- and nanoplastics are interconnected processes. Persistence allows plastic particles to remain in soil, water, and sediments; transport moves them across environmental compartments; and bioavailability enables their interaction with plants, microbes, animals, and humans. In soil, particles can accumulate, migrate downward, bind pollutants, and enter plant systems. In water, they can float, sink, aggregate, resuspend, and move through aquatic food chains. In food systems, they may become available through contaminated crops, seafood, drinking water, and other dietary sources (Boctor *et al*., 2025; Pal *et al*., 2025; Udovicki *et al*., 2022). Therefore, understanding these processes is essential for One Health risk assessment, food-chain protection, and the development of standardized monitoring and remediation strategies.

This figure illustrates how micro- and nanoplastics enter agricultural soils through plastic mulch, biosolids, wastewater, atmospheric deposition, tyre wear, and runoff. After entering soil and water systems, these particles may persist, migrate vertically or horizontally, interact with microbes and contaminants, enter plants and livestock, and ultimately reach humans through contaminated food chains.

**Environmental Toxicity in Plants and Ecosystems**

**Effects on Plant Growth, Physiology, and Crop Productivity**

Micro- and nanoplastics are increasingly recognized as emerging stressors in plant systems because they can interact with soil structure, root surfaces, plant tissues, nutrient cycles, and physiological processes. In agricultural ecosystems, these particles may originate from plastic mulch films, sewage sludge, compost, wastewater irrigation, synthetic textiles, packaging residues, and atmospheric deposition. Once present in soil or water, they may influence seed germination, root elongation, shoot development, chlorophyll content, photosynthesis, oxidative balance, nutrient uptake, and final crop yield (Chaudhary *et al*., 2025; Jia *et al*., 2023; Lin *et al*., 2025). Recent reviews emphasize that plant responses are not uniform because phytotoxicity depends on particle size, shape, polymer type, concentration, surface charge, exposure duration,

plant species, growth stage, and soil conditions (Chaudhary *et al*., 2025; Lin *et al*., 2025).

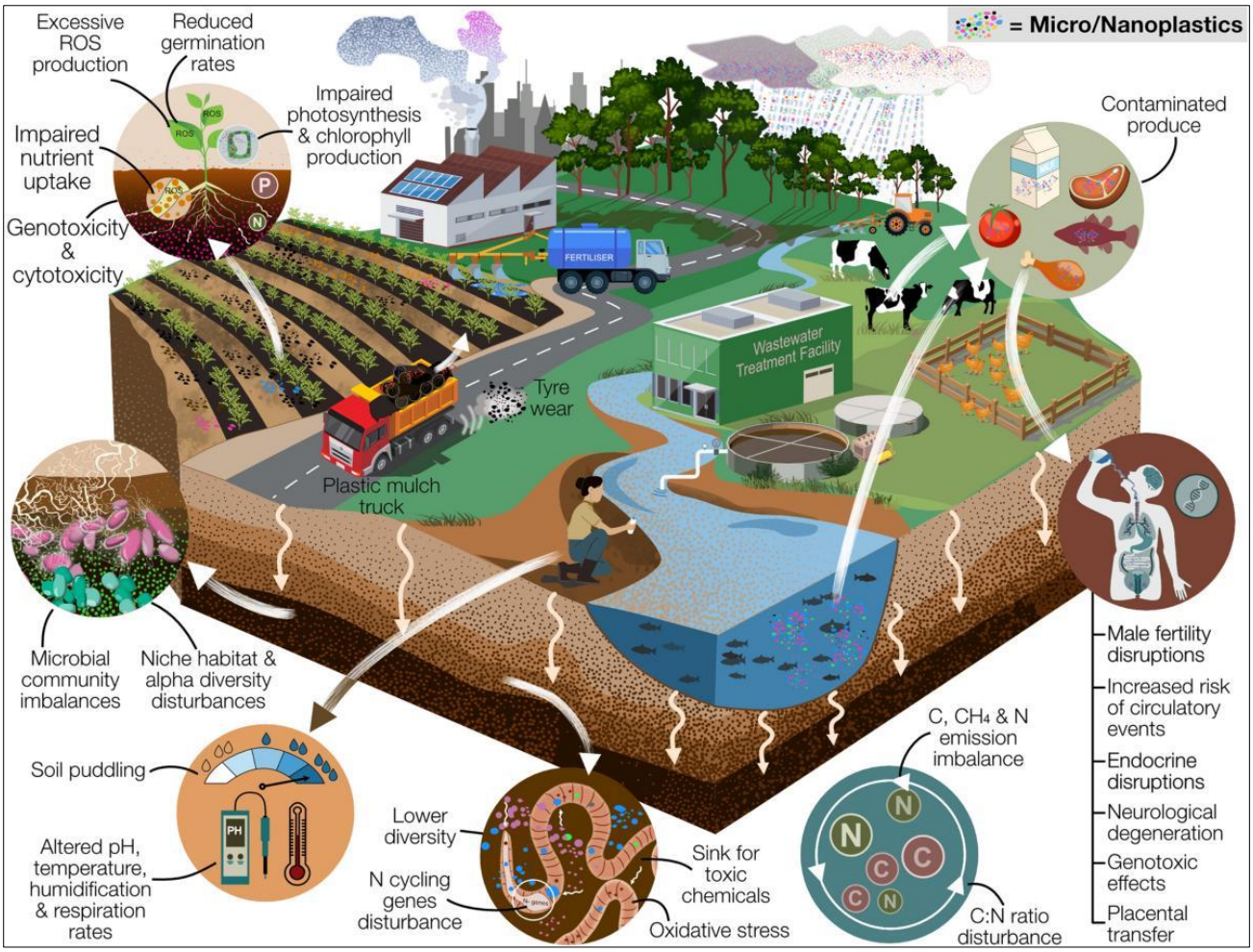


**Figure 2: Persistence, Transport, and Bioavailability of Micro- and Nanoplastics from Agricultural Soil to Food Webs and Humans**

One of the earliest visible effects of micro- and nanoplastic exposure occurs during seed germination and early seedling establishment. Plastic particles may adhere to the seed coat, block pores, reduce water uptake, interfere with oxygen diffusion, or release toxic leachates that disturb early metabolic activity (Jia *et al*., 2023). Reduced germination percentage, delayed germination, and weaker seedling vigor have been reported in several plant species exposed to polyethylene, polystyrene, polycarbonate, high-density polyethylene, and other plastic particles (Jia *et al*., 2023). However, the response is not always linear; some low-dose exposures may stimulate germination or root growth, possibly due to changes in soil aeration or mild stress-induced priming, while higher doses often produce inhibitory effects (Jia *et al*., 2023; Wong & Taylor, 2025).

Root systems are especially vulnerable because they are the first plant organs exposed to soil micro- and nanoplastics. Plastic particles may accumulate on root surfaces, enter through cracks or apoplastic spaces, and interfere with root elongation, root hair formation, lateral root development, and water or nutrient uptake (Jia *et al*., 2023; Lin *et al*., 2025). Mechanical blockage of root pores and disruption of root architecture can reduce the absorptive surface area of plants, thereby limiting water, nitrogen, phosphorus, potassium, calcium, magnesium, and micronutrient acquisition (Chaudhary *et al*., 2025; Jia *et al*., 2023). In soil-grown crops, these root-level disturbances may reduce biomass accumulation and weaken plant establishment, particularly under high microplastic concentrations or combined stress conditions such as drought, salinity, heavy metals, or pesticide contamination (Lin *et al*., 2025).

Micro- and nanoplastics also affect shoot growth and leaf development. Inhibition of root function may indirectly reduce shoot height, leaf area, petiole number, fresh weight, dry weight, and overall biomass because shoots depend on roots for water and mineral supply (Jia *et al*., 2023). A recent soil-based experiment using environmentally relevant concentrations of polystyrene microplastics reported species-specific effects in edible crops: in cherry radish, root length and

fruit diameter decreased by 35.0% and 20.4%, respectively, while in Chinese cabbage, leaf area and petiole number decreased by 35.9% and 41.7%, respectively (Chen *et al*., 2025). These findings show that even when the same polymer is used, crop responses may differ depending on plant morphology, organ development, and species-specific physiology (Chen *et al*., 2025).

Photosynthesis is one of the most important physiological processes affected by microplastic stress. Microplastics can reduce chlorophyll content, disturb chloroplast structure, alter stomatal conductance, impair gas exchange, reduce Rubisco activity, and disturb photosystem performance (Jia *et al*., 2023; Wong & Taylor, 2025). Wong and Taylor (2025) reviewed terrestrial plant studies and reported that many studies found reductions in chlorophyll content and photosynthesis under microplastic exposure. In maize, high microplastic exposure was associated with reduced net carbon fixation, stomatal conductance, and transpiration, while in wheat, low concentrations produced stimulatory effects but higher concentrations reduced physiological performance (Wong & Taylor, 2025). This indicates that microplastic effects on photosynthesis can be dose-dependent and may vary between crop species (Wong & Taylor, 2025).

Recent global-scale evidence further highlights the potential agricultural importance of photosynthetic inhibition. Zhu *et al.,* (2025) conducted a multiecosystem meta-analysis using 3,286 data points and estimated that microplastic exposure reduced photosynthesis by 7.05–12.12% across terrestrial plants, marine algae, and freshwater algae. Their model also estimated that microplastic-induced reductions in photosynthesis could correspond to annual losses of 4.11–13.52% in major crop production, equivalent to 109.73–360.87 million metric tons per year for major crops (Zhu *et al*., 2025). These estimates are important for food-security discussion, but they should be interpreted carefully because global projections depend on model assumptions, available datasets, and the difference between controlled experiments and real field conditions (Zhu *et al*., 2025).

Micro- and nanoplastic exposure may also trigger oxidative stress in plants. Oxidative stress occurs when the production of reactive oxygen species exceeds the plant's antioxidant defense capacity. Under microplastic exposure, plants may show increased levels of reactive oxygen species, hydrogen peroxide, malondialdehyde, lipid peroxidation, and altered activities of antioxidant enzymes such as superoxide dismutase, catalase, peroxidase, and ascorbate peroxidase (Jia *et al*., 2023; Lin *et al*., 2025). At moderate stress levels, antioxidant enzymes may increase as a protective response; however, under high or prolonged exposure, antioxidant capacity may decline, leading to membrane damage, metabolic imbalance, cytotoxicity, and reduced plant growth (Jia *et al*., 2023).

Nutrient uptake and metabolic regulation are also affected by microplastic contamination. Microplastics may physically block root surfaces, alter rhizosphere chemistry, adsorb essential nutrients, change soil pH and water-holding capacity, and disturb microbial processes involved in nutrient cycling (Chaudhary *et al*., 2025; Chen *et al*., 2025). In rice, lettuce, wheat, soybean, and other crops, studies reviewed by Jia *et al.,* (2023) reported changes in carbohydrate metabolism, nitrogen metabolism, amino acid pathways, antioxidant metabolism, and gene expression under microplastic exposure. These metabolic changes are important because they may influence not only plant growth but also crop nutritional quality and stress tolerance (Jia *et al*., 2023; Lin *et al*., 2025).

The effect of microplastics on crop productivity is complex and sometimes contradictory. Several studies report reduced biomass, impaired grain development, decreased yield components, and altered crop quality, while other studies report neutral or even stimulatory effects under specific conditions (Jia *et al*., 2023; Wong & Taylor, 2025). Wong and Taylor (2025) noted that long-term exposure to microplastics or plastic mulch residues has been linked with decreased yield in rice, cotton, and maize, but they also highlighted that some experiments reported increased root or shoot biomass depending on polymer type, dose, and crop species. Therefore, microplastic exposure should not be described as uniformly toxic in all situations; instead, its effects should be interpreted through a dose–response and context-dependent framework (Wong & Taylor, 2025).

Overall, micro- and nanoplastics can affect plant growth, physiology, and crop productivity through multiple direct and indirect pathways. Direct effects include seed-coat blockage, root adsorption, tissue entry, oxidative stress, chlorophyll reduction, photosynthetic inhibition, and altered metabolism. Indirect effects include changes in soil structure, nutrient availability, water retention, microbial communities, and co-contaminant bioavailability (Chaudhary *et al*., 2025; Jia *et al*., 2023; Lin *et al*., 2025). Current evidence suggests that these particles may threaten crop performance and food security, especially under long-term field exposure and combined environmental stress. However, more standardized field studies are needed to confirm environmentally realistic dose–response relationships, crop-specific sensitivity, and long-term impacts on yield and nutritional quality.

**Table 1: Effects of Micro- and Nanoplastics on Plant Growth, Physiology, and Crop Productivity**

| Plant/Crop | Plastic Type | Exposure Condition | Growth Effect | Physiological/Biochemical Effect |
|---|---|---|---|---|
| Rice | Microplastics | Soil exposure | Reduced growth and possible yield decline | Altered nutrient uptake, oxidative stress, and disturbed metabolic activity |
| Wheat | Microplastics | Soil/laboratory exposure | Reduced root and shoot growth | Reduced chlorophyll content and photosynthetic performance |
| Maize | Microplastics | Soil exposure | Reduced biomass accumulation | Decreased gas exchange, stomatal conductance, and carbon fixation |
| Chinese cabbage | Polystyrene microplastics | Soil exposure | Reduced leaf area and petiole number | Oxidative stress and redox imbalance |
| Cherry radish | Polystyrene microplastics | Soil exposure | Reduced root length and fruit diameter | Stress-pathway disturbance and impaired root development |
| Lettuce | Micro-/nanoplastics | Soil or hydroponic exposure | Reduced seedling growth and biomass | Altered antioxidant enzyme activity and nutrient imbalance |
| Arabidopsis | Nanoplastics | Controlled experimental exposure | Altered root growth and plant development | Particle accumulation, surface-charge-dependent response, and oxidative stress |
| Algae | Microplastics | Aquatic exposure | Reduced growth rate | Photosynthetic inhibition and cellular stress |

**Soil Health, Nutrient Cycling, and Ecological Imbalance**

Soil health is a key indicator of ecosystem stability because it integrates physical structure, chemical fertility, microbial activity, organic-matter turnover, and biological diversity. Micro- and nanoplastics can disturb this balance by altering soil aggregation, porosity, water-holding capacity, pH, nutrient availability, microbial community composition, enzyme activity, and soil-fauna performance. These changes are important because soil is not only a passive sink for plastic particles, but also an active biological system where microorganisms, roots, organic matter, minerals, and soil animals regulate nutrient cycling and ecosystem functioning (Aralappanavar *et al*., 2024; Cheng *et al*., 2024). Cheng *et al.,* (2024) reported that polyethylene microplastics altered soil nutrient-cycling indicators and reduced overall soil ecosystem function in a size- and dose-dependent manner.

Microplastics can affect soil physical health by changing soil structure and water movement. After entering soil, plastic particles may become incorporated into soil aggregates or occupy pore spaces, which can modify bulk density, permeability, aeration, and water retention. These physical changes may indirectly influence root growth, microbial respiration, nutrient diffusion, and organic-matter decomposition. Cheng *et al.,* (2024) noted that microplastics can alter aggregate structure, bulk density, porosity, permeability, and water-holding capacity after being incorporated into the soil matrix. Such structural disturbance may create uneven microhabitats, causing some soil zones to become more aerated and biologically active while others become compacted or less suitable for microbial and root activity (Cheng *et al*., 2024).

Soil biochemical properties are also highly sensitive to microplastic contamination. In an experimental study, Cheng *et al.,* (2024) tested polyethylene microplastics of two sizes, 13 μm and 130 μm, at concentrations of 0%, 1%, 3%, 6%, and 10% w/w. The smaller 13 μm particles significantly reduced soil respiration, β-glucosidase activity, catalase activity, pH, available phosphorus, dissolved reactive phosphorus, dissolved organic carbon, and available potassium in most cases, while urease activity increased. High concentrations of 130 μm particles also reduced pH, total dissolved nitrogen, available phosphorus, and available potassium, but increased soil respiration. These findings show that microplastic effects on soil health are strongly dependent on particle size and exposure concentration (Cheng *et al*., 2024).

Microplastics may disrupt nutrient cycling by modifying the activity and diversity of soil microorganisms. Soil bacteria and fungi regulate decomposition, mineralization, nitrogen transformation, phosphorus solubilization, carbon turnover, and enzyme production. Aralappanavar *et al.,* (2024) reviewed evidence showing that microplastics can affect soil microbial diversity and microbial functions related to nutrient and carbon cycling. Their review reported that plastisphere communities and microplastic-contaminated soils may show microbial communities that differ from uncontaminated soils, often with reduced diversity and altered functional capacity (Aralappanavar *et al*., 2024). This is important because microbial diversity supports ecosystem resilience, and any

disruption in microbial composition may affect nutrient availability and soil fertility.

Carbon cycling is one of the most important soil processes influenced by microplastics. Plastic particles are carbon-rich polymers, and their presence in soil may alter soil organic carbon stability, dissolved organic carbon, microbial biomass, soil respiration, and greenhouse-gas emissions. Chang *et al.,* (2024) reviewed evidence showing that microplastics can affect carbon stability, carbon dioxide emission, methane emission, microbial community structure, enzyme activity, and functional genes associated with carbon metabolism. The same review emphasized that biodegradable microplastics often show stronger effects than conventional non-biodegradable microplastics, but the direction and magnitude of the effect depend on polymer type, particle size, concentration, biodegradability, and soil type (Chang *et al*., 2024).

Microplastics can influence soil organic carbon dynamics through both physical and biological pathways. Physically, they may disturb soil aggregation and reduce the protection of organic matter within stable aggregates. Biologically, they may change microbial biomass, microbial respiration, carbon-degrading enzymes, and the availability of dissolved organic carbon. Chang *et al.,* (2024) explained that microplastics may modify soil carbon cycling by changing soil physicochemical and microbiological properties, while the exact mechanisms remain incompletely understood. Because soil carbon storage is closely linked with soil fertility and climate regulation, microplastic-induced changes in carbon cycling may have broader ecological implications beyond crop production alone (Chang *et al*., 2024).

Nitrogen cycling is another critical process that may be affected by microplastics. Nitrogen availability in soil depends on microbial processes such as mineralization, nitrification, denitrification, nitrogen fixation, and ammonia volatilization. Sarfraz *et al.,* (2025) emphasized that microplastics may alter microbial activity and enzyme function, thereby affecting nitrogen fluxes in agricultural soils. These disturbances may influence nitrate and ammonium availability, nitrogen retention, gaseous nitrogen losses, and plant nitrogen uptake. Because nitrogen is essential for plant growth and crop productivity, microplastic interference with nitrogen cycling may reduce soil fertility and contribute to long-term agricultural instability (Sarfraz *et al*., 2025).

Soil enzymes are useful indicators of nutrient cycling because they reflect microbial activity and organic-matter transformation. Enzymes such as β-glucosidase, urease, catalase, phosphatase, protease, and dehydrogenase participate in carbon, nitrogen, phosphorus, and oxidative-stress processes. Cheng *et al.,* (2024) found that polyethylene microplastic exposure changed enzyme activities involved in soil nutrient cycling, especially β-glucosidase, catalase, and urease. These enzyme-level changes suggest that microplastics can alter the biochemical pathways responsible for nutrient release, nutrient retention, and microbial metabolism (Cheng *et al*., 2024).

Microplastics may also create ecological imbalance by affecting soil fauna. Earthworms, collembolans, mites, nematodes, and other soil organisms contribute to litter decomposition, soil mixing, aggregate formation, microbial regulation, and nutrient redistribution. Lin *et al.,* (2020) reported from a field-based microplastic addition experiment that microplastics negatively affected soil fauna but stimulated microbial activity, indicating that microplastics can disturb the balance between soil animal communities and microbial processes. Such imbalance is ecologically important because soil fauna and microorganisms normally work together to maintain decomposition, nutrient cycling, and soil structure (Lin *et al*., 2020).

Earthworms are especially important because they are considered soil ecosystem engineers. They improve soil aeration, organic-matter turnover, nutrient mixing, and aggregate formation. Guo *et al.,* (2023) reviewed microplastic toxicity to earthworms and reported that polyethylene and polystyrene are among the most commonly studied polymers in earthworm-toxicity research. Their analysis showed that microplastic toxicity mechanisms mainly involve histopathological damage, oxidative stress, and carrier effects for heavy metals or organic pollutants. They also reported that when microplastic concentration in soil exceeds 0.1% w/w, earthworm growth may be affected and oxidative stress may be induced, with possible neural and DNA damage (Guo *et al*., 2023).

Microplastic-induced ecological imbalance is not limited to individual organisms. Changes in soil physical structure, microbial community composition, enzyme activity, nutrient cycling, and soil-fauna health may collectively reduce ecosystem multifunctionality. Ecosystem multifunctionality refers to the ability of soil to maintain several functions at the same time, including nutrient retention, microbial activity, organic-matter decomposition, carbon storage, water regulation, and plant support. Cheng *et al.,* (2024) found that overall soil ecosystem function decreased after microplastic addition, with soil respiration, available phosphorus, dissolved reactive phosphorus, and β-glucosidase activity acting as important predictors of ecosystem function. This suggests that microplastics may disturb multiple soil functions simultaneously rather than affecting only one isolated pathway (Cheng *et al*., 2024).

Microplastics can also intensify the ecological effects of other pollutants. Their surfaces may adsorb heavy metals, pesticides, antibiotics, persistent organic

pollutants, and plastic additives. Once present in soil, these particles may change the mobility, persistence, and bioavailability of co-contaminants. Guo *et al.,* (2023) highlighted that microplastics can act as carriers of heavy metals and organic pollutants, contributing to combined toxicity in earthworms. This carrier role is important because agricultural soils are often exposed to multiple stressors at the same time, including fertilizers, pesticides, wastewater residues, heavy metals, and plastic mulch fragments (Guo *et al*., 2023; Aralappanavar *et al*., 2024).

Overall, micro- and nanoplastics can threaten soil health by disturbing physical structure, biochemical properties, microbial functions, nutrient cycling, soil fauna, and ecosystem multifunctionality. Their effects are complex and may be negative, neutral, or occasionally stimulatory depending on polymer type, particle size, dose, exposure duration, soil properties, and organism group. However, current evidence shows that microplastics can alter carbon cycling, nitrogen cycling, enzyme activity, microbial diversity, earthworm health, nutrient availability, and soil ecosystem function (Aralappanavar *et al*., 2024; Chang *et al*., 2024; Cheng *et al*., 2024; Guo *et al*., 2023). Therefore, microplastic contamination should be considered a soil-health issue, not only a plastic-waste issue.

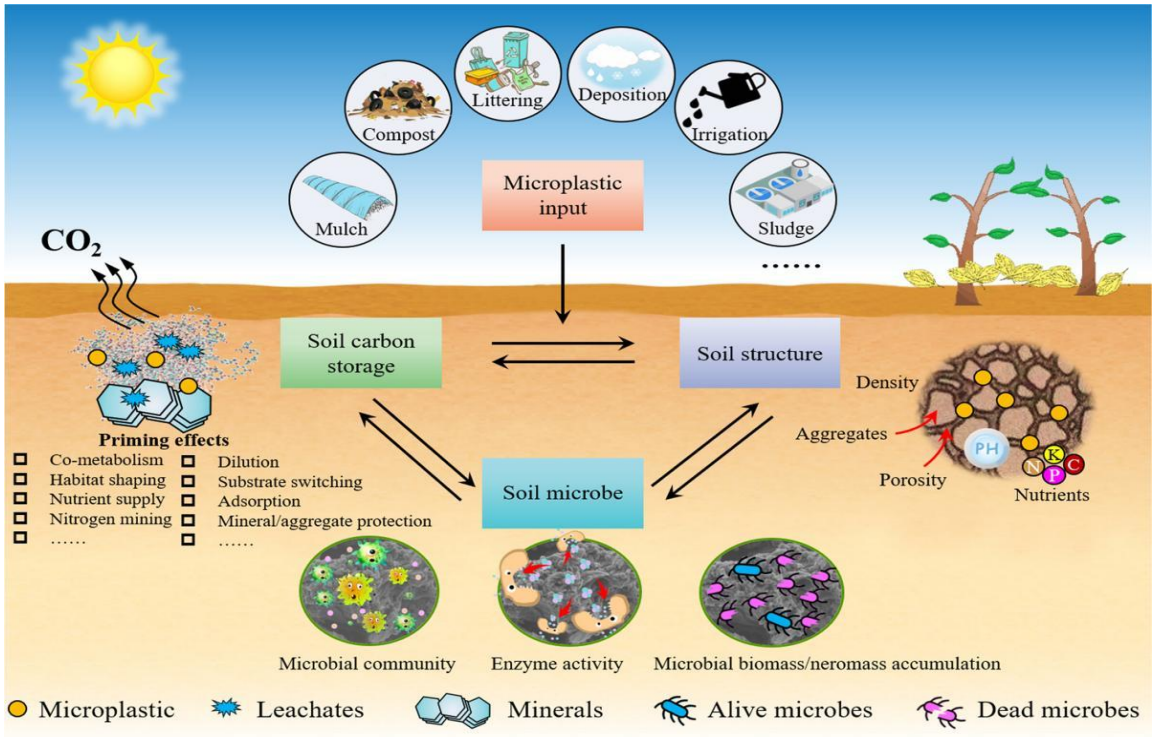


**Figure 3: Microplastic-Induced Disruption of Soil Health, Microbial Activity, and Nutrient-Cycling Functions**

This figure summarizes how microplastics enter soil through agricultural and environmental pathways and disturb soil structure, microbial communities, enzyme activity, and soil carbon processes. It supports the concept that microplastics can create ecological imbalance by modifying soil physicochemical properties, microbial habitats, nutrient availability, and ecosystem functioning.

**Food-Chain Transfer and Ecological Risk Assessment**

Food-chain transfer is one of the most important ecological concerns associated with micro- and nanoplastics because these particles can move from environmental compartments into organisms and then from prey to predators. In aquatic ecosystems, microplastics may be ingested directly from water, sediment, suspended particles, or biofilms, while indirect ingestion can occur when predators consume contaminated prey. This trophic movement is important because it links environmental contamination with ecological exposure across plankton, invertebrates, fish, birds, mammals, and humans. However, trophic transfer should be interpreted carefully because the movement of particles through food chains does not automatically mean biomagnification at higher trophic levels (Miller *et al*., 2020; Pitt *et al*., 2024).

A major distinction is needed among bioaccumulation**,** trophic transfer, and biomagnification. Bioaccumulation refers to the net uptake of particles by an organism from all exposure routes, including water, sediment, food, respiration, or direct contact. Trophic transfer refers to movement from one trophic level to another, such as from prey to predator. Biomagnification refers to an increase in contaminant concentration from lower to higher trophic levels. Miller *et al.,* (2020) emphasized that these terms are often used in

microplastic literature, but field evidence does not clearly support microplastic biomagnification across a general marine food web, even though bioaccumulation within organisms and trophic transfer in laboratory settings have been reported.

In aquatic food chains, lower trophic organisms such as algae, zooplankton, copepods, bivalves, and small crustaceans are particularly important because their particle-size feeding range overlaps with many microplastics. Microplastics may be mistaken for food, trapped in feeding appendages, or ingested along with natural particles. Experimental work by Costa *et al.,* (2020) demonstrated trophic transfer of fluorescent polyethylene microplastics from contaminated copepod nauplii to *Aurelia* jellyfish ephyrae. After 24 hours, the jellyfish ingested microplastic-contaminated prey, showing that microplastics can move between trophic levels through predator–prey interactions, although the study did not observe clear immobility or pulsation-frequency effects in jellyfish under the tested conditions (Costa *et al*., 2020).

Recent experimental evidence also supports the movement of microplastics through aquatic food chains involving algae, copepods, and fish. McHale and Sheehan (2024) reported that microplastics negatively affected algal growth and copepod survival and observed trophic transmission of small plastic spheres from copepods to fish predators. Their findings are important because they show that microplastics may affect food chains not only by direct ingestion, but also by altering lower trophic resources and prey quality. This means that ecological risk may arise through both particle transfer and disruption of food-web function (McHale & Sheehan, 2024).

Marine food-web evidence remains complex and sometimes contradictory. In a systematic review and meta-analysis of 116 publications, Miller *et al.,* (2020) reported that microplastic contamination occurs across marine species and trophic levels, and that bioaccumulation occurs within many marine organisms. However, their analysis did not support clear biomagnification of microplastics from lower to higher trophic levels in a general marine food web. They also noted that feeding strategy may be more important than trophic level in explaining body burden, because filter feeders and organisms with high particle-processing rates may accumulate more microplastics than some higher predators (Miller *et al*., 2020).

Pitt *et al.,* (2024) reached a similar conclusion in a critical review of microplastics in marine food webs. They reported convincing evidence for trophic transfer, mixed evidence for bioaccumulation, and no clear evidence for biomagnification of microplastics. They also emphasized that nanoplastics remain a major uncertainty because smaller particles may behave differently from larger microplastics due to greater potential for tissue translocation, cellular interaction, and longer retention. Therefore, ecological interpretation should distinguish between larger microplastics and nanoplastics rather than treating all plastic particles as one uniform contaminant class (Pitt *et al*., 2024).

Food-chain transfer is not limited to aquatic systems. In terrestrial and agricultural ecosystems, micro- and nanoplastics can enter soils through plastic mulch, biosolids, compost, wastewater irrigation, atmospheric deposition, and organic fertilizers. From soil, they may interact with roots, soil fauna, rhizosphere microorganisms, and edible plant tissues. Boctor *et al.,* (2025) described agricultural soils as important entry points for micro- and nanoplastics into food webs and highlighted concerns about plant uptake, soil-organism exposure, and human food-chain infiltration. Their review also noted that micro- and nanoplastic uptake by plants depends on plant type, agricultural practice, and soil properties (Boctor *et al*., 2025).

Plant uptake is a critical pathway for food-chain transfer because crops connect contaminated soils with herbivores, livestock, and humans. Li *et al.,* (2020) provided experimental evidence that submicrometre- and micrometre-sized plastic particles can enter wheat and lettuce roots through crack-entry sites at lateral-root emergence and then translocate from roots to shoots. This finding is important because it suggests that plastic particles may move beyond external root surfaces and enter aboveground plant tissues under certain exposure conditions. However, plant uptake depends strongly on particle size, polymer type, surface charge, exposure medium, plant species, and transpiration rate, so field-level transfer still needs more realistic assessment (Li *et al*., 2020; Azeem *et al*., 2021).

Human dietary exposure represents the upper end of the food-chain-transfer pathway. Microplastics have been reported in food and drink categories such as seafood, salt, water, processed foods, honey, and plant-based foods, although reported levels vary widely because analytical methods, particle-size limits, contamination control, and polymer identification are not standardized across studies. Udovicki *et al.,* (2022) reported that potential toxic consequences of ingested microplastics may arise from the particles themselves, diffused monomers and additives, sorbed contaminants, and microorganisms colonizing plastic surfaces. The same review emphasized that true human exposure remains uncertain because many studies differ in sampling and detection methods (Udovicki *et al*., 2022).

Ecological risk assessment of micro- and nanoplastics requires more than measuring particle abundance. Risk depends on particle concentration, size, shape, polymer type, surface chemistry, weathering state, biofilm formation, contaminant load, exposure duration, organism sensitivity, and ecosystem context. Hooge *et al.,* (2024) proposed an ecological risk-assessment

framework for microplastics in agricultural soils amended with biosolids and emphasized that risk evaluation should consider not only microplastic abundance but also particle properties such as size, shape, and polymer type. Their framework identified earthworms, springtails, and soil microbiomes as important ecological receptors because these organisms regulate soil health, organic-matter turnover, nutrient cycling, and ecosystem function (Hooge *et al*., 2024).

Several index-based models have been proposed to evaluate microplastic contamination and ecological risk. Christian *et al.,* (2022) described models such as plastic contamination factor, pollution load index, plastic bioconcentration or accumulation factor, plastic-biota-sediment accumulation factor, biota accumulation load index, polymer risk index, polymer ecological risk index, plastic estimated daily intake, and plastic carcinogenic risk. These models are useful because they convert complex environmental data into interpretable indicators for comparing sites, polymers, exposure routes, and intervention priorities. However, index-based assessments should be used carefully because they depend on data quality, polymer-hazard assumptions, sampling design, and the availability of toxicological thresholds for different organisms (Christian *et al*., 2022).

Ecological risk assessment is still limited by major knowledge gaps. Many laboratory studies use concentrations, particle types, or exposure durations that do not fully represent field conditions. Environmental particles are often weathered, irregular, chemically aged, biofilm-coated, and mixed with other pollutants, whereas laboratory particles are frequently pristine, spherical, and used at higher concentrations. In addition, many studies measure ingestion or accumulation but do not always measure depuration, tissue translocation, chronic reproductive effects, population-level consequences, or ecosystem-level outcomes. Miller *et al.,* (2020) and Hooge *et al.,* (2024) both emphasized the need for environmentally realistic exposure conditions, standardized methods, and receptor-specific risk assessment to improve confidence in ecological interpretation.

Overall, micro- and nanoplastics can enter food chains through soil, water, sediment, plants, invertebrates, fish, livestock, and human diets. Current evidence supports trophic transfer and organism-level bioaccumulation, but clear biomagnification of microplastics across whole food webs is not consistently supported. Nanoplastics may present greater uncertainty because their small size may increase cellular uptake, tissue movement, and retention. Therefore, ecological risk assessment should integrate particle properties, exposure pathways, trophic transfer, organism sensitivity, co-contaminants, and One Health relevance. A strong risk-assessment approach should move beyond simple particle counts and include biological effects, food-web interactions, and realistic environmental exposure scenarios.

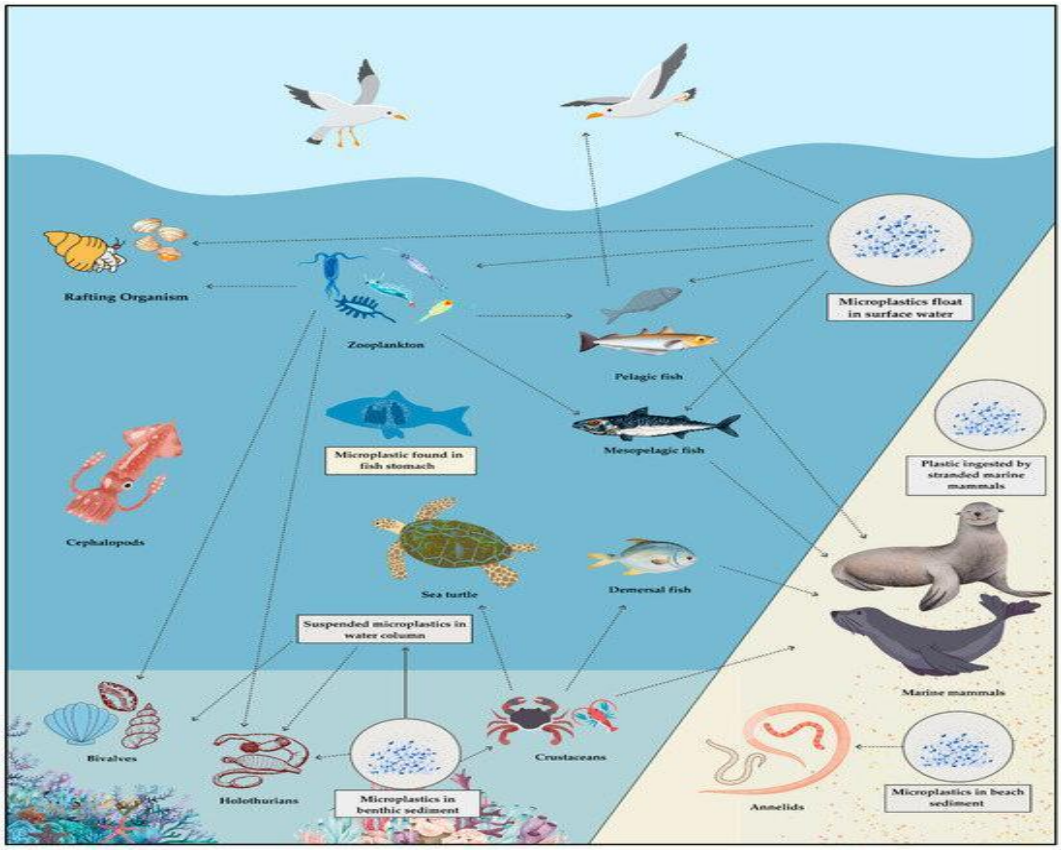

**Figure 4: Food-Chain Transfer and Ecological Risk Pathways of Microplastics in Aquatic Ecosystems**

This figure illustrates how microplastics accumulate in aquatic compartments, including surface water, sediments, beaches, and the water column, before entering food webs through ingestion by zooplankton, bivalves, crustaceans, annelids, and fish. It further shows trophic transfer to higher consumers such as cephalopods, turtles, birds, and marine mammals, highlighting ecological-risk pathways across aquatic food chains.

**Toxicological Impacts in Animal Biological Models Evidence from Aquatic Organisms, Insects, and Wildlife**

Micro- and nanoplastics have been widely investigated in animal biological models because animals provide essential evidence for understanding environmental toxicity, food-chain transfer, tissue accumulation, physiological stress, and potential One Health risks. Aquatic organisms, insects, and wildlife are exposed through contaminated water, sediment, soil, air, prey, and food particles. These exposures may occur through ingestion, inhalation, dermal contact, gill uptake, trophic transfer, and interaction with biofilm-coated particles. Recent reviews emphasize that micro- and nanoplastics can affect organisms at multiple biological levels, including molecular signaling, oxidative balance, gut function, immune response, reproduction, development, behavior, and population fitness (Naz *et al*., 2024; Subaramaniyam *et al*., 2023). In aquatic systems, particles smaller than 10 µm are often reported as more toxic than larger particles because they may be more bioavailable and capable of stronger tissue interactions (Naz *et al*., 2024).

Aquatic organisms are among the most studied animal groups because rivers, lakes, estuaries, and oceans are major sinks and transport pathways for plastic particles. Fish, mollusks, crustaceans, zooplankton, jellyfish, amphibians, and benthic invertebrates may ingest microplastics directly from water or sediment or indirectly through contaminated prey. In fish, microplastic exposure has been associated with reactive oxygen species production, oxidative stress, inflammation, immunotoxicity, genotoxicity, DNA damage, altered gut microbiota, altered swimming and feeding behavior, and reduced growth or quality of fish tissues (Subaramaniyam *et al*., 2023). These outcomes are biologically important because fish are not only ecological consumers but also food resources for humans and wildlife.

Fish models provide strong evidence that microplastics can disturb physiological and biochemical homeostasis. Microplastic exposure may alter antioxidant enzymes such as superoxide dismutase, catalase, glutathione-related enzymes, and lipid peroxidation markers, indicating oxidative injury and stress-response activation (Subaramaniyam *et al*., 2023). Several studies summarized in recent reviews also report tissue-level effects in gills, gut, liver, kidney, and brain, where particles or particle-associated chemicals may contribute to inflammation, neurotoxicity, metabolic disturbance, and cellular damage (Ghosh, 2025; Subaramaniyam *et al*., 2023). However, the severity of these effects depends on polymer type, particle size, dose, exposure duration, fish species, developmental stage, and the presence of co-contaminants such as pesticides or metals.

Zooplankton and small crustaceans are important aquatic models because their feeding range overlaps with many microplastic sizes and because they connect primary producers with higher trophic levels. In *Daphnia magna*, Huang *et al.,* (2022) reported that microplastics were internalized by or attached to individuals and that high exposure delayed sexual maturity, reduced egg production, and affected offspring growth. Their study found that the number of eggs after 7 days decreased under higher microplastic exposure, while delayed maturation suggested impaired reproductive timing (Huang *et al*., 2022). Similar long-term evidence reported that microplastics caused parental and juvenile mortality and reduced somatic growth, reproduction, and population growth rate in *Daphnia magna* under 21-day bioassays (Guilhermino, 2021). These findings show that microplastic toxicity may affect not only individual survival but also reproductive output and population dynamics.

Nanoplastics may produce stronger biological concern than larger microplastics because their small size increases surface reactivity, cellular contact, and potential translocation across biological barriers. Thakur *et al.,* (2025) reviewed nanoplastic toxicity in aquatic organisms and reported that nanoplastics may enter aquatic species through ingestion, inhalation-like routes in aquatic respiration, and dermal or epithelial exposure. Their review described oxidative stress as one of the main toxicological mechanisms in aquatic organisms exposed to nanoplastics, with possible downstream effects on tissues, organs, and ecosystem-level health (Thakur *et al*., 2025). This is important for your article because nanoplastics may represent a more difficult risk-assessment problem than larger microplastics due to analytical limitations and limited chronic, environmentally realistic exposure data.

Insects are increasingly recognized as important biological models for microplastic toxicity, especially in terrestrial and agricultural ecosystems. Terrestrial insects can encounter microplastics through contaminated soil, air, food, nectar, pollen, water, nesting material, and plant surfaces. A recent review on terrestrial insects explained that plastic ingestion can lead to accumulation and may support transfer to higher trophic levels through predator–prey interactions (Sucharitakul *et al*., 2024). This is important because insects contribute to pollination, decomposition, biological pest control,

nutrient cycling, and food-web stability; therefore, microplastic effects on insects may have ecological consequences beyond individual toxicity.

The fruit fly *Drosophila melanogaster* is a useful insect model because it allows controlled evaluation of survival, feeding behavior, gut damage, cellular toxicity, and sex-specific responses. El Kholy *et al.,* (2023) exposed adult *Drosophila* to polystyrene microplastic beads and found that flies could distinguish and avoid microplastic-treated food. The study reported sex-specific toxicity, with males showing greater sensitivity; all male flies exposed to 0.5 µg/mL polystyrene microplastics died after 14 days, while some females survived longer. The authors also observed reduced food intake, decreased survival, reduced starvation resistance in males, and concentration-dependent necrosis and apoptosis in midgut cells (El Kholy *et al*., 2023). These findings suggest that microplastic exposure can affect insect fitness through feeding disturbance, gut injury, survival reduction, and cellular damage.

Pollinating insects are particularly important because microplastic toxicity may affect ecosystem services and agricultural productivity. Sheng *et al.,* (2024) reviewed plastic pollution in agricultural landscapes and argued that nano- and microplastics may threaten pollination and biological pest control by affecting beneficial insects at organismal, farm, and landscape scales. Their review highlighted possible impacts on gene expression, organ damage, behavior, pathogen susceptibility, and interactions with other stressors such as antibiotics, pesticides, and climate stress (Sheng *et al*., 2024). Similarly, a systematic review summarized by the University of Freiburg reported that nano- and microplastics may damage bee digestive systems, weaken immunity, alter behavior, reduce pollination performance, and increase risks to biodiversity and food security (Sheng *et al*., 2024).

Wildlife evidence shows that microplastic exposure is not limited to laboratory models or aquatic organisms. Birds are increasingly used as bioindicators because they occupy diverse habitats, feed at different trophic levels, and may be exposed through ingestion, inhalation, prey consumption, contaminated sediments, and anthropogenic waste. Wang *et al.,* (2021) reviewed recent evidence on birds and plastic pollution and reported that macroplastics and microplastics can accumulate in different tissues of aquatic and terrestrial birds, raising concerns about digestive, physiological, and ecological impacts. More recent studies have also reported microplastic accumulation in various bird species, supporting their value as environmental sentinels for plastic exposure (Tatlı *et al*., 2025; Wang *et al*., 2021).

Inhalation is an emerging wildlife exposure pathway. Dziobak *et al.,* (2024) provided the first evidence of microplastic inhalation among free-ranging small cetaceans by analyzing exhaled breath samples from bottlenose dolphins in Sarasota Bay, Florida, and Barataria Bay, Louisiana. The study found suspected microplastics in all sampled dolphins, and Raman spectroscopy confirmed plastic composition in a subset of particles. The authors concluded that inhalation is a relevant exposure route for bottlenose dolphins, although further research is needed to determine health outcomes such as pulmonary inflammation, lung function impairment, or systemic translocation (Dziobak *et al*., 2024).

Overall, evidence from aquatic organisms, insects, and wildlife shows that micro- and nanoplastics can affect animal biological systems through multiple toxicological pathways. In aquatic organisms, major effects include ingestion, bioaccumulation, oxidative stress, inflammation, neurotoxicity, reproductive impairment, gut dysbiosis, and behavioral changes. In insects, microplastics may impair feeding, gut integrity, survival, immunity, and ecosystem services such as pollination and pest control. In wildlife, birds and marine mammals provide evidence that exposure can occur through ingestion and inhalation in real environments. However, current evidence also shows major research limitations, including inconsistent particle characterization, unrealistic laboratory doses, limited chronic field studies, insufficient nanoplastic detection, and weak understanding of population-level effects. Therefore, animal biological models remain essential for linking environmental exposure with mechanistic toxicity, ecological risk, and One Health relevance.

This figure illustrates how microplastics are ingested by aquatic organisms such as zooplankton, jellyfish, and fish, allowing particle transfer through food webs. It also highlights the role of microplastics as carriers of chemical contaminants, supporting toxicological outcomes such as bioaccumulation, oxidative stress, inflammation, feeding disruption, and reproductive impairment in animal models.

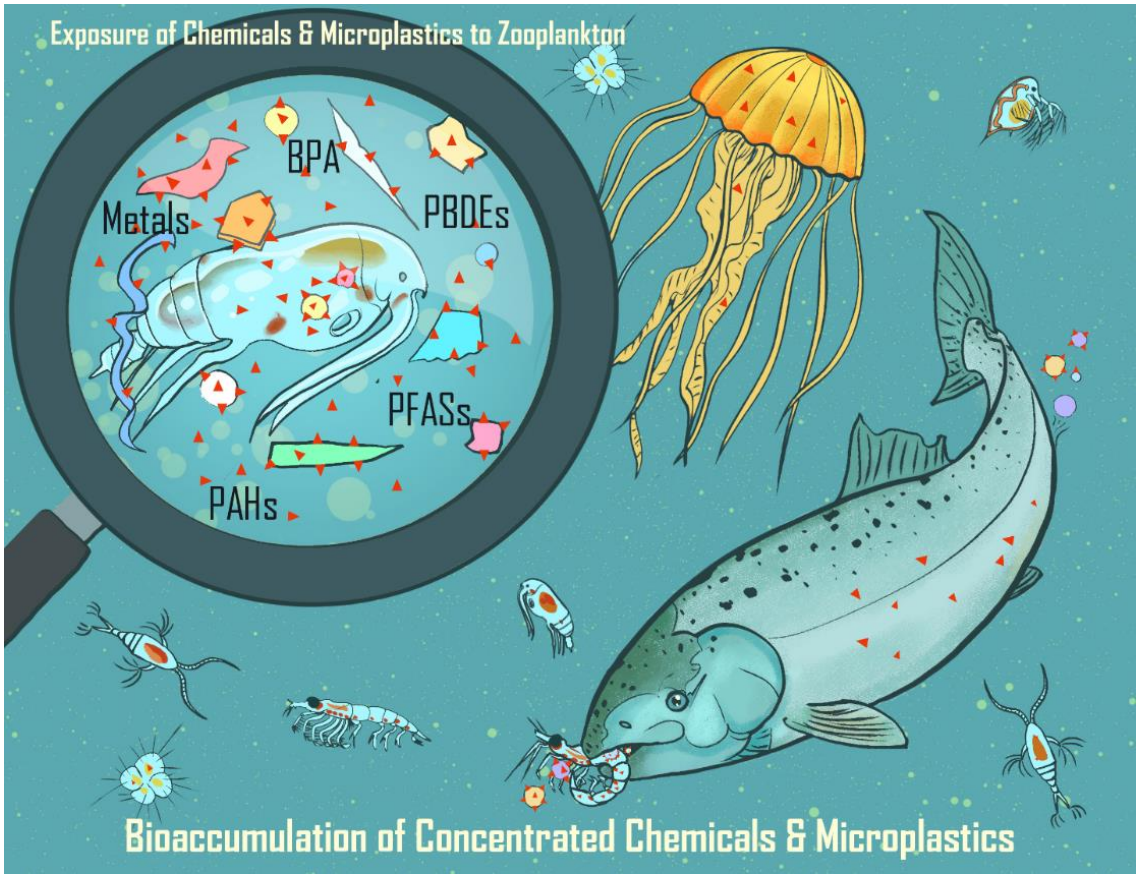


**Figure 5: Toxicological Pathways of Microplastics in Aquatic Organisms and Wildlife Models**

**Rodent and Zebrafish Models for Nanoplastic Toxicity**

Rodent and zebrafish models are widely used in nanoplastic toxicology because they provide complementary biological information. Rodents are valuable for evaluating mammalian biodistribution, intestinal absorption, systemic inflammation, organ-specific toxicity, reproductive outcomes, gut microbiota disruption, metabolic effects, and possible human-health relevance. Zebrafish are highly useful for developmental and ecotoxicological assessment because they have transparent embryos, rapid development, high fecundity, low maintenance cost, and measurable endpoints such as hatching rate, heart rate, body length, locomotor activity, oxidative stress, apoptosis, and gene-expression changes (Feng *et al*., 2022; Huang *et al*., 2024; Torres-Ruiz *et al*., 2021). Zebrafish therefore help bridge the gap between simple cell-culture studies and complex mammalian studies.

Nanoplastics are generally considered more biologically reactive than larger microplastics because their small size increases surface-area-to-volume ratio, tissue penetration potential, interaction with cell membranes, and ability to cross biological barriers. In rodent studies, this concern is especially important because ingested or inhaled particles may interact with the gastrointestinal barrier, gut microbiota, blood circulation, liver, kidney, reproductive tissues, placenta, and nervous system. In zebrafish, nanoplastics can affect embryonic development, larval behavior, oxidative balance, DNA repair pathways, and organ-specific accumulation (Feng *et al*., 2022; Du *et al*., 2024). Du *et al.,* (2024) reported that 50 nm, 100 nm, and 500 nm polystyrene particles were distributed mainly in the liver, spleen, and intestine of mice, with smaller particles showing stronger accumulation and easier tissue penetration.

Rodent models provide strong evidence that nanoplastic toxicity is influenced by particle size, dose, exposure route, and exposure duration. In BALB/c mice exposed by gavage for 28 days, Du *et al.,* (2024) found that polystyrene micro/nanoplastics of 50 nm, 100 nm, and 500 nm were distributed in major organs, including liver, spleen, kidney, brain, intestine, and testis. The study reported that smaller particles accumulated more widely and produced more obvious changes in liver, kidney, and heart-related biochemical indicators compared with larger particles. Although severe histopathological abnormalities were not observed at low dose, high-dose exposure produced size- and dose-dependent changes in hematological and biochemical parameters (Du *et al*., 2024).

The gastrointestinal tract is one of the most important targets of orally exposed nanoplastics. Recent mouse studies show that polystyrene nanoplastics can

disturb the intestinal microenvironment, increase intestinal permeability, alter tight-junction and mucin-related pathways, and induce gut microbiota dysbiosis. Hsu *et al.,* (2025) reported that exposure to 100 nm polystyrene nanoplastics in mice altered intestinal miRNAs, compromised tight junction protein ZO-1 and mucin-13 expression, increased intestinal permeability, and induced gut microbiota dysbiosis through bacteria–host interactions. These findings are important because intestinal barrier injury and gut microbial imbalance may provide mechanistic links between environmental nanoplastic exposure and systemic inflammation or metabolic disturbance (Hsu *et al*., 2025).

Rodent pregnancy models are also important for evaluating developmental and reproductive risk. Aghaei *et al.,* (2022) exposed pregnant mice to either 5 µm or 50 nm polystyrene particles in filtered drinking water and reported fetal growth restriction in late gestation, with a 12% decrease in fetal weight at the highest exposure concentration. This study is significant because it directly connects maternal exposure to micro- and nanoplastics with fetal and placental growth outcomes in a mammalian model (Aghaei *et al*., 2022). More recently, He *et al.,* (2025) reported that oral administration of approximately 100 nm polystyrene nanoplastics to pregnant mice caused placental injury, metabolic abnormalities, and adverse pregnancy outcomes, with gut microbiota contributing to fetal growth restriction through disruption of placental nicotinamide metabolism (He *et al*., 2025).

Nanoplastic neurotoxicity is another emerging concern in rodent models. Song *et al.,* (2025) investigated polystyrene nanoplastic toxicity across *Aurelia coerulea* polyps, microglial cells, and mice and reported oxidative-stress-related effects mediated through the MAPK pathway. In mice, polystyrene nanoplastics altered oxidative stress biomarkers and were associated with anxiety-like behavior and cognitive dysfunction (Song *et al*., 2025). These findings suggest that mammalian nervous-system endpoints should be included in future long-term nanoplastic toxicity studies, especially because oxidative stress, neuroinflammation, and mitochondrial dysfunction are repeatedly proposed as mechanisms of nanoplastic-induced neurotoxicity.

Zebrafish models provide strong evidence for developmental toxicity because embryos and larvae allow direct observation of early-life endpoints. Feng *et al.,* (2022) exposed zebrafish embryos to 100 nm polystyrene nanoplastics at different concentrations and reported decreased hatching and survival rates, reduced heart rate, shortened body length, suppressed behavioral activity, increased reactive oxygen species, altered superoxide dismutase and catalase activities, and changes in base excision repair pathway-related genes. Their study concluded that nanoplastics could induce developmental toxicity through oxidative stress activation and DNA repair pathway disturbance (Feng *et al*., 2022).

Zebrafish are also useful for neurobehavioral and reproductive toxicity assessment. Sarasamma *et al.,* (2020) exposed adult zebrafish to approximately 70 nm polystyrene nanoplastics and reported neurobehavioral impairment, reproductive toxicity, oxidative damage, and changes in multiple biomarkers. The study included behavioral assays such as novel tank, mirror biting, predator avoidance, social interaction, shoaling, and circadian rhythm assessment, showing that nanoplastic exposure may affect both physiological and behavioral endpoints in adult zebrafish (Sarasamma *et al*., 2020). This makes zebrafish particularly valuable for linking nanoplastic exposure with nervous-system function, reproductive disruption, and oxidative stress.

Recent reviews further support the importance of zebrafish embryos for micro- and nanoplastic toxicity assessment. Huang *et al.,* (2024) reviewed evidence showing that micro- and nanoplastic exposure can induce neurodevelopmental toxicity, immunotoxicity, gastrointestinal effects, microbiota dysbiosis, cardiac dysfunction, vascular toxicity, metabolic imbalance, oxidative damage, and apoptosis in zebrafish embryos. Torres-Ruiz *et al.,* (2021) also emphasized that zebrafish embryos are useful for studying polystyrene nanoplastic effects, but highlighted the need for better standardization of particle characterization, exposure design, and endpoint reporting.

Overall, rodent and zebrafish models show that nanoplastic toxicity is multi-systemic and mechanism-dependent. Rodents provide mammalian evidence for biodistribution, intestinal barrier injury, gut dysbiosis, organ toxicity, reproductive effects, fetal growth restriction, and neurobehavioral changes. Zebrafish provide high-resolution developmental, behavioral, oxidative-stress, apoptosis, and gene-expression endpoints. Across both models, recurring mechanisms include oxidative stress, inflammation, apoptosis, mitochondrial dysfunction, DNA damage or repair activation, gut microbiota disruption, and tissue-specific accumulation. However, many studies still use high experimental concentrations, pristine spherical polystyrene particles, and short exposure periods, which may not fully represent real environmental exposure. Future studies should use environmentally realistic doses, aged and irregular particles, mixed polymer types, chronic exposure designs, and standardized reporting of particle size, surface charge, polymer type, aggregation, and biological endpoints.

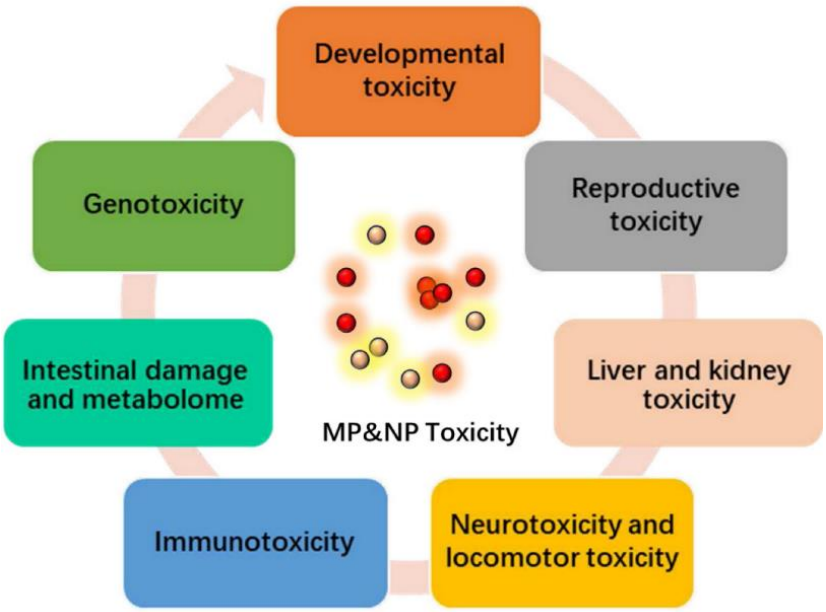


**Figure 6: In Vivo Toxicological Outcomes of Micro- and Nanoplastics in Zebrafish and Rodent Models**

This figure summarizes major in vivo toxicological endpoints of micro- and nanoplastic exposure, including developmental toxicity, reproductive toxicity, intestinal damage, immunotoxicity, genotoxicity, liver/kidney toxicity, neurotoxicity, and locomotor impairment. It fits Section 4.2 because zebrafish and rodent models are commonly used to evaluate biodistribution, oxidative stress, inflammation, developmental disruption, and organ-specific toxicity.

**Oxidative Stress, Inflammation, Genotoxicity, and Reproductive Toxicity**

Oxidative stress is one of the most frequently reported mechanisms of micro- and nanoplastic toxicity in animal and cellular models. After exposure, micro- and nanoplastics may interact with cell membranes, mitochondria, lysosomes, and intracellular proteins, leading to excessive production of reactive oxygen species (ROS). When ROS production exceeds antioxidant defense capacity, lipid peroxidation, protein oxidation, mitochondrial dysfunction, DNA damage, inflammation, apoptosis, and tissue injury may occur (Kadac-Czapska *et al*., 2024; Mahmud *et al*., 2024). Recent reviews show that oxidative-stress biomarkers commonly used in microplastic toxicology include superoxide dismutase, catalase, glutathione, glutathione peroxidase, glutathione S-transferase, malondialdehyde, hydrogen peroxide, and ROS levels (Panizzolo *et al*., 2023). Broader evidence from human cell-line nanotoxicology similarly identifies ROS accumulation, mitochondrial dysfunction, lipid peroxidation, disruption of redox homeostasis, altered antioxidant enzymes, and stress-related gene-expression changes as central mechanisms of particle-induced cellular injury (Shafiq *et al*., 2025).

Inflammation is closely linked with oxidative stress because ROS can activate inflammatory signaling pathways and cytokine responses. In fish models, microplastics have been reported to induce oxidative stress, inflammation, immunotoxicity, genotoxicity, DNA damage, gut-microbiota alteration, and behavioral changes. Subaramaniyam *et al.,* (2023) reported that microplastic exposure in fish can influence Nrf2, JNK, ERK, NF-κB, and MAPK signaling pathways, while modulating antioxidant enzymes such as superoxide dismutase, catalase, and glutathione-related systems (Subaramaniyam *et al*., 2023). These pathways are important because they connect oxidative injury with immune activation, inflammatory cytokine expression, apoptosis, and tissue-level damage.

At the cellular level, micro- and nanoplastics may trigger inflammation through several mechanisms. First, particles may physically interact with epithelial barriers and disturb membrane integrity. Second, smaller particles may be internalized by cells and accumulate in endosomes or lysosomes. Third, particle-induced mitochondrial stress may increase ROS generation, which can activate pro-inflammatory pathways. Fourth, aged or biofilm-coated particles may carry additives, metals, persistent organic pollutants, or microbial components that intensify inflammatory responses (Mahmud *et al*., 2024; Panizzolo *et al*., 2023). Therefore, inflammation caused by micro- and nanoplastics should be understood as both a direct particle effect and an indirect response to oxidative stress and co-contaminant exposure.

Genotoxicity is another important toxicological endpoint because DNA damage may affect cell survival, tissue function, reproduction, development, and long-term disease risk. Micro- and nanoplastics may

contribute to genotoxicity directly through particle interaction with cellular structures or indirectly through oxidative stress-mediated DNA damage. Tang (2025) reported that microplastics may induce genotoxic effects through direct interaction with chromosomes, DNA, and associated proteins, as well as indirectly through ROS production and inflammatory signaling. Similarly, Panizzolo *et al.,* (2023) reviewed biomarkers of oxidative stress, inflammation, and genotoxicity and concluded that both in vitro and in vivo evidence supports oxidative stress and inflammation as key mechanisms that may lead to immunotoxicity and genotoxicity (Panizzolo *et al*., 2023; Tang, 2025).

Zebrafish studies provide direct evidence linking nanoplastic exposure with oxidative DNA damage. Sökmen *et al.,* (2020) reported that 20 nm polystyrene nanoplastics could bioaccumulate in zebrafish embryo brain tissue and cause oxidative DNA damage. This finding is important because it shows that nanoscale plastic particles may reach sensitive tissues during early development and produce molecular-level damage (Sökmen *et al*., 2020). Zebrafish are particularly useful for genotoxicity and developmental toxicity assessment because their embryos allow evaluation of early-life endpoints, including hatching, growth, behavior, oxidative stress, apoptosis, and gene-expression changes.

Reproductive toxicity is one of the most biologically significant outcomes because it can affect fertility, fecundity, offspring quality, and population stability. Micro- and nanoplastics may impair reproduction through oxidative stress, endocrine disruption, inflammatory damage, apoptosis, altered steroidogenesis, testicular injury, ovarian dysfunction, sperm-quality reduction, follicular damage, and embryonic developmental disturbance (Ferrante *et al*., 2022; Xie *et al*., 2020). Ferrante *et al.,* (2022) reviewed evidence that micro- and nanoplastic contamination can disturb male and female reproductive systems through ROS generation, inflammation, apoptosis, hormonal imbalance, and altered reproductive signaling pathways.

Rodent models provide strong evidence for male reproductive toxicity. Xie *et al.,* (2020) reported that exposure to polystyrene microplastics induced reproductive toxicity in mice through oxidative stress and activation of the p38 MAPK signaling pathway. Their findings suggest that microplastic exposure may impair spermatogenesis and male reproductive function through redox imbalance and stress-signaling activation (Xie *et al*., 2020). Ilechukwu *et al.,* (2022) also reported that chronic ingestion of polystyrene microplastics caused reproductive dysfunction in male rats, supporting the concern that persistent exposure may affect reproductive tissues and fertility-related endpoints.

Zebrafish also provide important evidence for reproductive and oxidative damage. Sarasamma *et al.,* (2020) reported that nanoplastic exposure caused neurobehavioral impairment, reproductive damage, oxidative damage, and biomarker responses in zebrafish. The study suggested that nanoplastics may induce oxidative stress in developing follicles and interfere with key regulatory genes involved in reproductive function. This is important because reproductive toxicity in fish can affect not only individual fertility but also population recruitment and aquatic ecosystem stability (Sarasamma *et al*., 2020).

The relationship among oxidative stress, inflammation, genotoxicity, and reproductive toxicity is highly interconnected. Oxidative stress can initiate lipid peroxidation and mitochondrial damage, which then activates inflammatory pathways such as NF-κB and MAPK. These inflammatory and oxidative pathways can damage DNA, disturb cell-cycle regulation, induce apoptosis, and impair gonadal tissues. In reproductive organs, such damage may reduce sperm quality, disrupt ovarian follicle development, alter sex-steroid hormone levels, damage the blood–testis barrier, and reduce fertility or offspring viability (Ferrante *et al*., 2022; Xie *et al*., 2020). Thus, reproductive toxicity may be considered a downstream outcome of multiple interacting molecular mechanisms rather than a single isolated effect.

Overall, current evidence indicates that micro- and nanoplastics can induce oxidative stress, inflammation, genotoxicity, and reproductive toxicity in animal biological models. The strongest recurring mechanism is ROS-mediated damage, which may activate inflammatory signaling, disturb antioxidant defenses, damage DNA, and impair reproductive tissues. However, interpretation must remain cautious because many studies use pristine polystyrene particles, high exposure doses, short experimental periods, and laboratory conditions that may not fully reflect real environmental exposure. Future studies should prioritize environmentally realistic concentrations, aged and irregular particles, mixed polymer types, chronic exposure models, multigenerational outcomes, and standardized biomarkers for oxidative stress, inflammation, DNA damage, and reproductive health.

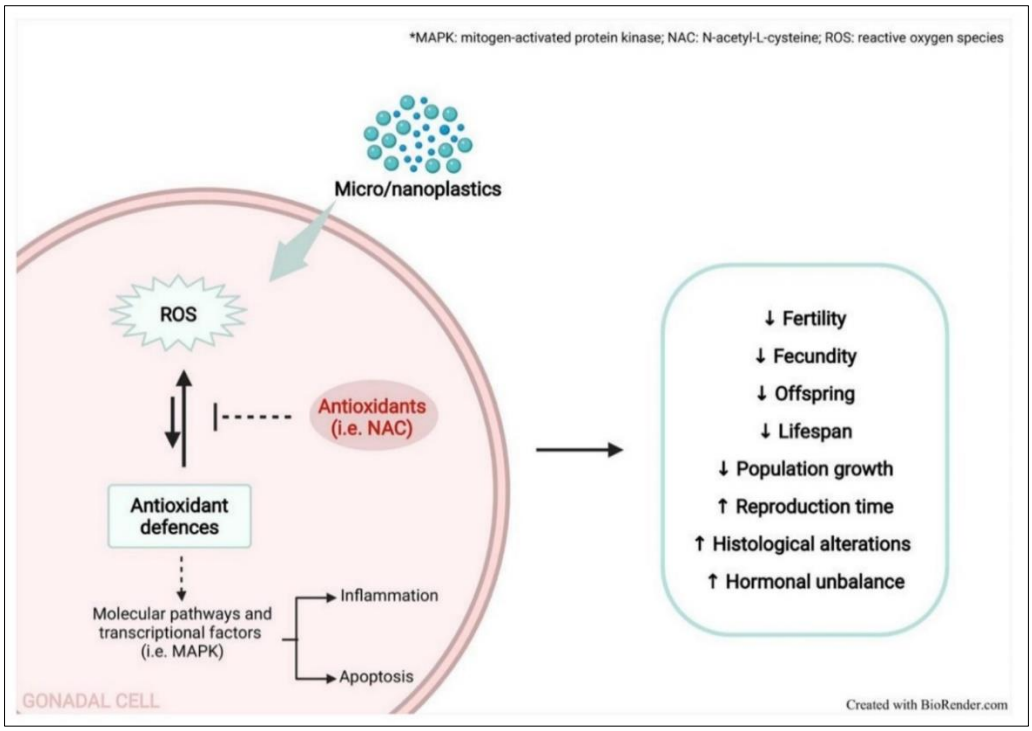


**Figure 7: ROS-Mediated Oxidative Stress, Inflammation, and Reproductive Toxicity Induced by Micro- and Nanoplastics**

This figure shows how micro- and nanoplastics can trigger ROS production, oxidative stress, inflammation, apoptosis, hormonal imbalance, and reproductive dysfunction in male and female systems. It supports Section 4.3 by visually linking molecular toxicity pathways with reproductive outcomes such as reduced fertility, impaired fecundity, histological alterations, and offspring-related effects.

**Microbial Dysbiosis and Antimicrobial Resistance**
**Microplastics as Carriers of Microbial Biofilms and Pathogens**

Microplastics are increasingly recognized as artificial surfaces that can support microbial attachment and biofilm formation in aquatic, soil, wastewater, and biological environments. Once released into the environment, microplastic particles do not remain biologically inert; instead, their surfaces may be rapidly colonized by bacteria, fungi, algae, protozoa, archaea, and viruses, forming a plastic-associated microbial habitat known as the plastisphere (Zhai *et al*., 2023; Zhang *et al*., 2026). This plastisphere can differ substantially from surrounding water, sediment, soil, or natural biofilms because plastic surfaces provide a persistent, hydrophobic, and mobile substrate for microbial colonization. Zhai *et al.,* (2023) reported that marine microplastics are colonized by diverse microorganisms, including bacteria, fungi, viruses, archaea, algae, and protozoans, and that plastisphere communities differ significantly from those in surrounding environments.

Biofilm formation on microplastics generally begins with the adsorption of organic molecules and conditioning films onto the plastic surface, followed by microbial attachment, extracellular polymeric substance production, biofilm maturation, and eventual detachment or dispersal of microbial cells. The initial microbial attachment is influenced by plastic hydrophobicity, surface roughness, polymer composition, particle aging, environmental nutrients, temperature, salinity, pH, light exposure, and the presence of pollutants (Bartkova *et al*., 2021; Zhang *et al*., 2026). Zhang *et al.,* (2026) explained that microorganisms adhere to microplastic surfaces and secrete extracellular polymeric substances, producing mature biofilms that can promote microbial interactions, horizontal gene transfer, pathogen enrichment, and later release of associated microorganisms back into the environment.

The ability of microplastics to carry microbial biofilms is important because biofilms can protect attached microorganisms from environmental stress. The extracellular polymeric substance matrix may shield microbial communities from ultraviolet radiation, desiccation, predation, disinfectants, chemical stress, and shear forces. This protective microenvironment may increase microbial survival and allow bacteria to remain attached to plastic particles during environmental transport (Witsø *et al*., 2024; Zhang *et al*., 2026). In wastewater-associated plastispheres, biofilms are particularly important because wastewater contains high microbial loads, nutrients, organic matter, antibiotic residues, and other contaminants that can support

microbial growth and resistance selection (Witsø *et al*., 2024).

Microplastics may also function as carriers of potential pathogens. In aquatic and wastewater environments, plastic particles can be colonized by microorganisms of public-health concern, including *Vibrio* spp., *Escherichia coli*, *Salmonella*, *Listeria*, *Klebsiella*, *Aeromonas*, *Acinetobacter*, *Pseudomonas*, and other opportunistic or foodborne taxa (Witsø *et al*., 2024; Cholewińska *et al*., 2025). Witsø *et al.,* (2024) detected genera containing foodborne pathogenic taxa, including *Salmonella*, *E. coli/Shigella*, *Listeria*, and *Campylobacter*, as well as opportunistic pathogens such as *Klebsiella pneumoniae*, *Aeromonas hydrophila*, *Serratia marcescens*, *Enterobacter* spp., and *Acinetobacter* spp. in wastewater plastispheres. They also confirmed enteropathogenic *E. coli* and *Listeria monocytogenes* using RT-qPCR, while viable *K. pneumoniae* and *Acinetobacter* isolates were recovered from plastispheres in raw and treated wastewater.

The role of wastewater treatment plants is particularly important because these facilities receive plastic particles, organic matter, antibiotics, resistant bacteria, viruses, and household, hospital, industrial, and agricultural inputs. Although wastewater treatment reduces many pollutants and pathogens, microplastics are not completely removed and can persist in effluents or sludge. Witsø *et al.,* (2024) reported that plastispheres formed on polypropylene, polyvinyl chloride, and high-density polyethylene surfaces in raw and treated wastewater carried potential foodborne bacteria and viruses, including *L. monocytogenes*, *E. coli*, norovirus, and adenovirus. Their findings suggest that wastewater-associated plastispheres may act as reservoirs for bacteria and viruses and may contribute to the movement of plastic-associated pathogens into aquatic environments or food systems if wastewater and plastic waste are not properly managed.

Microplastics may increase microbial-risk potential not only by providing attachment surfaces but also by concentrating pollutants that influence microbial selection. Plastic particles can adsorb antibiotics, heavy metals, organic pollutants, nutrients, and additives. When these pollutants accumulate within or near biofilms, they may create selective pressures that favor antibiotic-resistant bacteria and resistance genes (Bartkova *et al*., 2021; Ahmad *et al*., 2024). Bartkova *et al.,* (2021) described the plastisphere as a potential hotspot for antimicrobial-resistance evolution because microplastic surfaces can retain microbes, antibiotics, heavy metals, and other resistance-promoting elements in close proximity.

The close spatial arrangement of microbial cells within biofilms can promote horizontal gene transfer. Biofilms contain dense bacterial communities, extracellular DNA, plasmids, bacteriophages, and mobile genetic elements, creating favorable conditions for the exchange of antibiotic-resistance genes. Ahmad *et al.,* (2024) emphasized that microplastics in aquatic environments provide substrates for biofilm formation, where colonizing bacteria may exchange antibiotic-resistance genes through horizontal gene transfer. Similarly, Zhang *et al.,* (2026) identified microplastic biofilms as reservoirs for antibiotic-resistance genes and potential pathogens, highlighting their ecological and public-health relevance.

Recent metagenomic studies provide stronger evidence that plastispheres can carry complex resistomes. Witsø *et al.,* (2025) analyzed 36 metagenomes from plastispheres in freshwater, raw wastewater, and treated wastewater environments and constructed 537 high-quality metagenome-assembled genomes. Their results showed that the surrounding environment strongly influenced ARG abundance and diversity, with wastewater plastispheres carrying the highest ARG diversity. They also reported that beta-lactam, aminoglycoside, and tetracycline resistance mechanisms were among the most abundant resistance classes detected in plastisphere resistomes (Witsø *et al*., 2025).

River systems are also important because they can connect urban, hospital, industrial, agricultural, and wastewater sources with wider aquatic ecosystems. Alfonsi *et al.,* (2025) studied plastic fragments introduced into a river system and recovered cefotaxime-resistant Enterobacteriaceae from plastic and water samples. Among selected isolates, most plastic-derived and water-derived isolates were multidrug resistant, and some carbapenem-resistant strains carried clinically relevant resistance determinants such as KPC and NDM enzymes. Their study supports the concern that riverine plastic debris may act as a reservoir and vehicle for clinically relevant resistant bacteria and resistance genes (Alfonsi *et al*., 2025).

Experimental evidence from river water and sediment further supports the ability of microplastics to alter microbial communities and enrich pathogens or resistance genes. Cholewińska *et al.,* (2025) exposed Oder River water and sediment communities to microplastic particles and reported higher abundance of pathogenic bacteria such as *Aeromonas salmonicida*, *Vibrio* spp., *E. coli*, and *Salmonella* after seven days of incubation. The same study reported a higher number of antibiotic-resistance genes after seven days compared with the control group, suggesting that microplastic presence can influence both pathogen abundance and ARG patterns in river-associated microbial communities (Cholewińska *et al*., 2025).

From a One Health perspective, microplastics as biofilm and pathogen carriers create a link among environmental pollution, microbial ecology, animal exposure, food safety, and human health. Plastic-

associated pathogens may enter aquatic food chains through ingestion by zooplankton, bivalves, fish, and other organisms, while wastewater-associated plastispheres may contaminate irrigation water, agricultural soils, and fresh produce. However, the actual public-health risk depends on whether the microbes are viable, virulent, abundant enough to cause infection, and capable of transfer to hosts under real exposure conditions (Witsø *et al*., 2024; Witsø *et al*., 2025). Therefore, sequencing-based detection should be combined with cultivation, qPCR, metagenomics, virulence profiling, and exposure assessment to better determine risk.

Overall, microplastics can act as mobile microbial habitats that support biofilm formation, protect microorganisms, concentrate pollutants, and create favorable conditions for pathogen survival and antibiotic-resistance dissemination. Current evidence indicates that plastispheres may carry foodborne bacteria, opportunistic pathogens, viruses, antibiotic-resistant bacteria, antibiotic-resistance genes, and mobile genetic elements. However, risk assessment remains limited by methodological differences, incomplete viability data, limited field-scale studies, and insufficient knowledge of pathogen transfer from plastispheres to animals, crops, and humans. Future research should focus on realistic environmental exposure, viable pathogen detection, standardized microbial-risk methods, and One Health surveillance of plastisphere-associated pathogens.

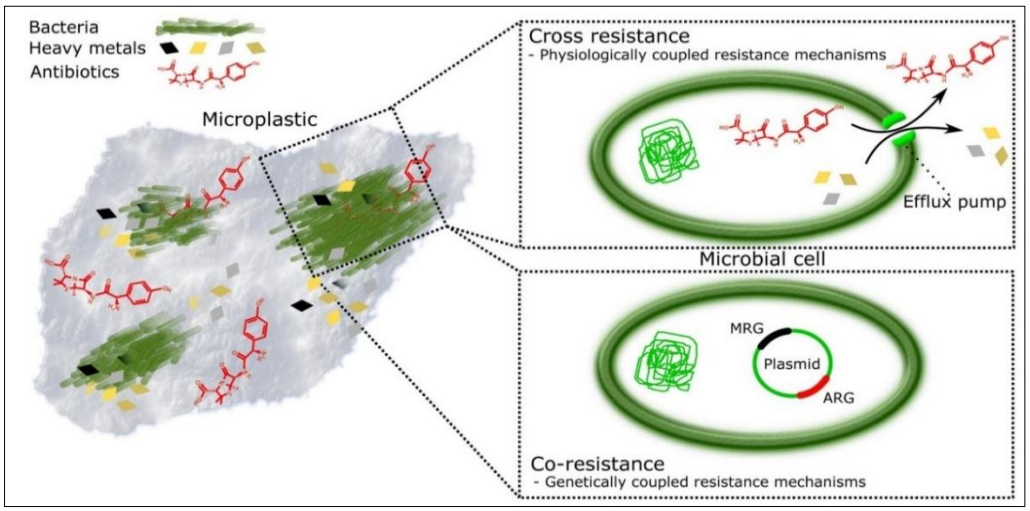


**Figure 8: Microplastics as Plastisphere Hotspots for Microbial Biofilm Formation, Pathogen Survival, and Antimicrobial-Resistance Dissemination**

This figure shows how microplastic surfaces act as miniature microbial habitats where bacteria attach, form biofilms, interact with antibiotics/heavy metals, and exchange antibiotic-resistance genes through mobile genetic elements.

**Alteration of Soil, Aquatic, and Gut Microbiomes**

Microplastics and nanoplastics are no longer considered only passive pollutants; they can act as active ecological surfaces that reshape microbial communities in soil, water, and the gastrointestinal tract. Their surfaces support microbial attachment and biofilm formation, producing a distinct "plastisphere" microbiome that may differ from surrounding soil, water, or gut microbial communities. This is important because plastisphere biofilms can concentrate nutrients, pollutants, antibiotics, metals, pathogens, and antibiotic resistance genes, creating favorable conditions for microbial dysbiosis and horizontal gene transfer (Yang *et al*., 2020; Witsø *et al*., 2025). Witsø *et al.,* analyzed 36 aquatic plastisphere metagenomes from freshwater, raw wastewater, and treated wastewater and showed that plastic-associated biofilms can carry antimicrobial resistance genes and potential pathogenic bacteria.

In soil ecosystems, microplastics can alter microbial diversity, soil enzyme activity, nutrient cycling, carbon cycling, and microbial network stability. These effects occur because microplastics change soil structure, water retention, aeration, organic matter distribution, and microbial colonization surfaces. A 2024 review in *Science of the Total Environment* reported that both microplastic-contaminated soils and plastisphere communities may show distinct and often lower microbial diversity compared with non-contaminated soils, with consequences for nutrient and carbon cycling (Aralappanavar *et al*., 2024).

Recent metagenomic evidence strongly supports the role of soil microplastics in antimicrobial resistance enrichment. Wang *et al.,* (2024) showed that increasing microplastic diversity in soil increased the abundance of antibiotic resistance genes, virulence factor genes, and mobile genetic elements. This means that the risk is not only linked to the amount of microplastic

pollution, but also to the diversity of polymer type, shape, and color present in soil. Their study further suggested that microplastic diversity can influence microbial adaptive strategies, including altered genetic diversity, enrichment of specific microbes, and functional metabolic changes associated with ARG dissemination.

In aquatic ecosystems, microplastics provide floating or sediment-associated surfaces for microbial colonization. These plastic surfaces may persist longer than natural organic particles and can transport microbial biofilms through rivers, wastewater streams, estuaries, aquaculture systems, and marine environments. Witsø *et al.,* (2025) identified 191 ARGs from 21 drug classes in aquatic plastispheres, with beta-lactam, aminoglycoside, tetracycline, macrolide, and lincosamide resistance genes being prominent in wastewater plastispheres. Raw wastewater plastispheres contained more ARGs than treated wastewater and river-water plastispheres, showing that wastewater-associated microplastics may act as important AMR reservoirs.

Microplastics can also disturb gut microbiomes after ingestion. In mammals, ingested plastic particles interact with mucus layers, epithelial barriers, bile acids, microbial metabolites, and immune cells. Su *et al.,* (2024) showed that polystyrene microplastics disrupted the microscopic structure of the mouse intestine, downregulated tight-junction genes such as Claudin-1, Claudin-2, Claudin-15, and Occludin, and altered gut microbiota diversity. Their findings also suggested sex-dependent effects, with stronger microbiome disturbance in female mice.

Gut microbiome dysbiosis caused by microplastics is closely linked with intestinal inflammation and metabolic disturbance. Hasegawa *et al.,* (2024) exposed mice to polystyrene microplastics under normal dietary conditions and found altered gut microbiota, increased intestinal inflammation, changes in nutrient-transport gene expression, and metabolic outcomes including dyslipidemia and fatty liver-related effects. This supports the idea that microplastics can disturb host metabolism partly through gut microbiome and immune pathways.

Nanoplastics may produce stronger biological effects than larger microplastics because of their smaller size, larger surface area, and greater interaction with epithelial and microbial interfaces. Hsu *et al.,* (2025) reported that polystyrene nanoplastics disrupted the intestinal microenvironment and barrier function by altering bacteria–host interactions through extracellular vesicle-delivered microRNAs. Similarly, Wang *et al.,* (2026) showed that chronic polystyrene nanoplastic exposure in young mice reduced oral and gut microbial diversity, altered microbial community structure, increased inflammatory cytokines, decreased antioxidant gene expression, and promoted aging-related markers through the oral–gut microbiome axis.

Overall, recent evidence from 2020–2026 shows that microplastics and nanoplastics can disturb microbial homeostasis across interconnected environmental and biological compartments. In soil, they alter microbial diversity, carbon/nutrient cycling, and resistance-gene profiles. In aquatic systems, they create plastisphere biofilms that may transport ARGs and pathogenic bacteria. In the gut, they promote dysbiosis, barrier dysfunction, inflammation, and metabolic imbalance. Therefore, microplastic-induced microbiome alteration should be discussed under a One-Health framework because soil, water, animals, humans, and antimicrobial resistance are directly connected through environmental exposure routes.

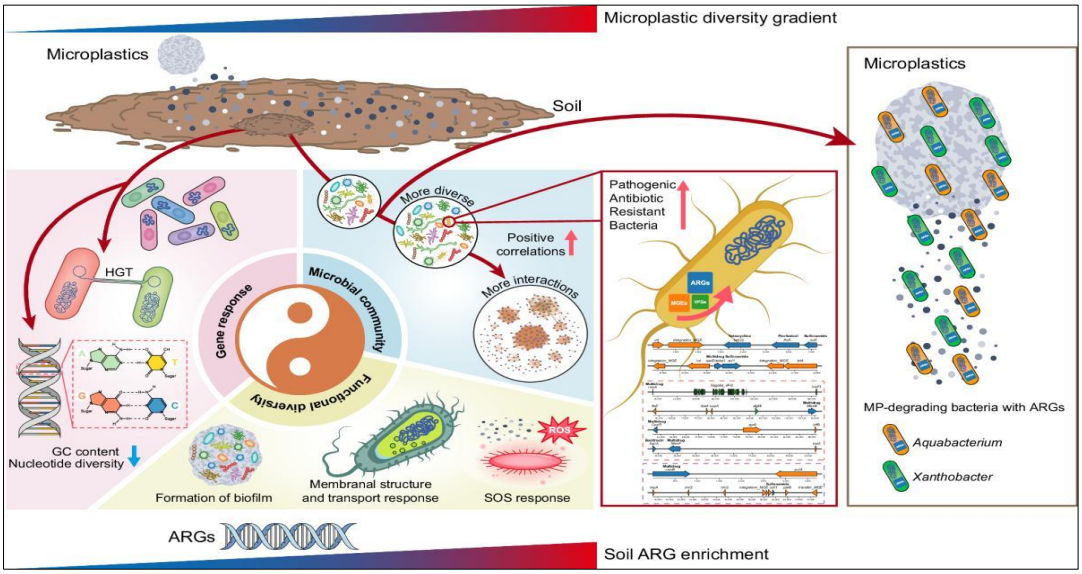


**Figure 9: Microplastic-induced microbiome dysbiosis and antimicrobial resistance dissemination across soil, aquatic, and gut ecosystems**

Figure 9. Microplastic-associated plastispheres as reservoirs of antimicrobial resistance genes in aquatic environments. Metagenomic analysis of plastisphere biofilms revealed distinct ARG profiles across freshwater, raw wastewater, and treated wastewater, supporting the role of microplastics as microbial and resistome hotspots. Adapted/reproduced from Witsø *et al.,* (2025) under the Creative Commons Attribution License.

**Role of Micro- and Nanoplastics in Spreading Antimicrobial Resistance Genes**

Micro- and nanoplastics are increasingly recognized as active vectors in the environmental spread of antimicrobial resistance genes (ARGs). Their ecological importance is linked to their small size, large surface-area-to-volume ratio, persistence, hydrophobic surface, and ability to support dense microbial biofilms known as the plastisphere. These biofilms can recruit antibiotic-resistant bacteria, enrich ARGs, concentrate mobile genetic elements such as plasmids, integrons, and transposons, and promote close cell-to-cell contact required for horizontal gene transfer (HGT) (Zhang *et al*., 2026). In this context, plastic particles do not merely transport microorganisms passively; they create microhabitats where resistant bacteria can survive, exchange genes, and disperse across soil, wastewater, freshwater, estuarine, marine, and host-associated environments. Zhang *et al.,* (2026) described microplastic biofilms as important reservoirs and preferred interfaces for ARG dissemination because ARGs and mobile genetic elements are often enriched on microplastic surfaces compared with surrounding water or sediment.

One major pathway by which micro- and nanoplastics promote ARG spread is the enrichment of resistant microbial communities on plastic surfaces. In landfill leachate, Shi *et al.,* (2020) showed that exposure to nano/microplastics increased ARG abundance, particularly under long-term exposure, and this enrichment was associated with increased bacterial abundance and potential ARG-carrying genera such as *Pseudomonas*, *Syntrophomonas*, and *Desulfotomaculum*. Their study further reported that reactive oxygen species production and increased bacterial membrane permeability may allow more bacteria to become ARG recipients through mobile genetic element-mediated transfer. This evidence indicates that polluted waste environments such as landfill leachate can become important reservoirs where microplastics, ARGs, and resistant bacteria co-propagate (Shi *et al*., 2020).

Nanoplastics may pose an even greater resistance-transfer risk than larger microplastics because they interact more strongly with bacterial membranes and extracellular DNA. Wang *et al.,* (2022) compared polystyrene microplastics and nanoplastics using a plasmid-borne ampicillin resistance transformation model in *Escherichia coli*. They found that polystyrene nanoplastics significantly increased transformation efficiency by 2.8–5.4-fold and transformation frequency by 3.2–8.4-fold, whereas larger microplastics showed no comparable effect. Mechanistically, nanoplastics promoted ARG uptake by inducing reactive oxygen species, activating the SOS response, increasing membrane permeability, and altering bacterial secretion systems. These findings support the view that particle size is a critical determinant of ARG transfer risk, with nanoscale plastics capable of directly enhancing bacterial acquisition of resistance genes (Wang *et al*., 2022).

Evidence from agricultural environments also demonstrates that microplastics can intensify ARG co-occurrence and horizontal transfer. Yu *et al.,* (2023) investigated chicken farms and surrounding farmlands and found a significant correlation between microplastics and ARGs. Chicken feces showed particularly high levels of both pollutants, with microplastics reaching 14.9 items/g and ARG abundance reaching $6.24 \times 10^8$ copies/g. Their conjugative transfer experiments further showed that microplastic exposure increased bacterial ARG transfer frequency by 1.4–1.7-fold. The authors linked this enhancement to altered expression of genes involved in stress response, membrane transport, conjugation, plasmid replication, and transfer regulation. This suggests that livestock-associated environments may function as hotspots for the co-spread of microplastics and ARGs, especially where manure, antibiotics, resistant bacteria, and plastic debris are simultaneously present (Yu *et al*., 2023).

Aquatic environments provide another important route for microplastic-mediated ARG dissemination. Plastics floating in rivers, wastewater systems, estuaries, and marine habitats can develop biofilms that differ from free-living microbial communities and may contain clinically relevant resistance genes. Witsø *et al.,* (2025) analyzed 36 plastisphere metagenomes from freshwater, raw wastewater, and treated wastewater environments and reconstructed 537 high-quality metagenome-assembled genomes. They found that wastewater plastispheres carried the highest ARG diversity, with resistance to beta-lactams, aminoglycosides, and tetracyclines among the most abundant mechanisms. Importantly, ARGs were linked to both environmental bacteria and potential pathogens, supporting the role of aquatic plastispheres as environmental reservoirs and vectors of antimicrobial resistance (Witsø *et al*., 2025).

Biofilm formation on plastic surfaces can directly increase conjugative ARG transfer. Zhou *et al.,* (2024) reported that microplastic biofilms in estuarine environments enhanced ARG conjugative transfer by 7.2–19.6 times compared with suspended bacteria, with polyethylene biofilms showing the strongest promotion. The proposed mechanism involved increased bacterial density inside biofilms, higher donor–recipient collision

frequency, upregulation of outer membrane proteins, enhanced conjugative pili synthesis, and activation of DNA replication and transfer systems. This evidence clearly shows that plastisphere biofilms can function as HGT hotspots where ARG transfer becomes more efficient than in freely suspended bacterial communities (Zhou *et al*., 2024).

Recent work further shows that polymer type, particle size, and chemical composition influence ARG dissemination. In mariculture sediments, Liu *et al.,* (2025) reported that polystyrene and polyvinyl chloride microplastics increased the relative abundance of ARGs by 1.41–2.50-fold and 2.01–2.84-fold, respectively, compared with controls. Their findings suggested that polymer type may exert a stronger influence than particle size, with PVC promoting deterministic microbial community assembly and enriching ARG-hosting pathogens, whereas PS promoted horizontal gene transfer within the plastisphere. This is highly relevant to aquaculture and seafood safety because mariculture sediments can connect plastic pollution, resistant bacteria, aquatic animals, and human food-chain exposure (Liu *et al*., 2025).

Soil ecosystems are also vulnerable to microplastic-driven resistome expansion. Wang *et al.,* (2024) demonstrated that increasing microplastic diversity in soil increased the abundance of ARGs, virulence factor genes, and mobile genetic elements. This finding is important because real-world soils rarely contain a single polymer type; instead, they often contain mixed plastics differing in shape, color, polymer chemistry, additives, and weathering status. Such diversity may increase ecological niches for different bacterial hosts and promote ARG dissemination through combined effects on microbial community structure, metabolic pathways, genetic diversity, and mobile element abundance (Wang *et al*., 2024).

Tire wear particles represent another important microplastic-related AMR vector because they contain polymeric particles, metals, and chemical additives. Jaffer *et al.,* (2024) showed that tire wear particles significantly enhanced horizontal transfer of ARGs from an *E. coli* donor to *Pseudomonas* and natural lake microbial communities. The study also found that metals associated with tire wear particles further promoted HGT, suggesting that plastic-associated co-contaminants may intensify resistance spread. This is important because tire wear particles are continuously released into road dust, stormwater runoff, rivers, lakes, and coastal environments, creating a realistic pathway for ARG movement across urban and aquatic ecosystems (Jaffer *et al*., 2024).

Mechanistically, micro- and nanoplastics promote ARG spread through several interconnected processes. First, they provide surfaces for biofilm growth, increasing bacterial density and donor–recipient contact. Second, they enrich ARG hosts, pathogens, and mobile genetic elements. Third, they adsorb antibiotics, metals, disinfectants, and plastic additives, creating selective pressure for resistant bacteria. Fourth, they induce oxidative stress, increase membrane permeability, alter ATP levels, and activate SOS and conjugation-associated genes, thereby enhancing transformation and conjugation. Kang *et al.,* (2026) experimentally showed that polystyrene micro/nanoplastics of 20 nm, 120 nm, and 1 μm can affect ARG transfer in both Gram-negative and Gram-positive bacterial systems. Notably, environmentally relevant concentrations as low as 0.1 mg/L facilitated AMR transfer, and 20 nm polystyrene nanoparticles increased transfer frequency by up to 8.9-fold in tested systems (Kang *et al*., 2026).

Overall, evidence from 2020–2026 demonstrates that micro- and nanoplastics are important environmental amplifiers of antimicrobial resistance. They spread ARGs by serving as physical carriers, selective biofilm habitats, co-contaminant concentrators, and active promoters of horizontal gene transfer. Their impact is observed across landfill leachate, agricultural systems, soil, wastewater, mariculture sediments, estuaries, lakes, and other aquatic environments. From a One-Health perspective, this is highly concerning because microplastic-mediated ARG dissemination connects environmental pollution with animal production, food safety, ecosystem health, and human exposure. Therefore, future AMR risk assessment should include not only antibiotic use and resistant bacteria but also microplastic abundance, polymer type, particle size, weathering status, additive chemistry, and plastisphere-associated mobile genetic elements.

## Human Health Risks and Medical Relevance

### Human Exposure Through Food, Water, Air, and Consumer Products

Human exposure to microplastics and nanoplastics occurs through multiple daily routes, mainly ingestion, inhalation, and limited dermal contact. The most important exposure sources include contaminated food, drinking water, bottled beverages, indoor and outdoor air, household dust, food-contact materials, cosmetics, plastic packaging, and other consumer products. The World Health Organization emphasized that human risk assessment should consider not only plastic particles themselves but also their polymer type, particle size, additives, residual monomers, adsorbed pollutants, and microbial biofilms attached to plastic surfaces (World Health Organization [WHO], 2022). WHO also identified food, water, and air as major environmental exposure routes for nano- and microplastic particles.

Food is one of the major pathways of human exposure because microplastics can enter the food chain through contaminated soil, irrigation water, aquatic environments, food processing, and food packaging.

Microplastics have been reported in seafood, table salt, drinking water, bottled water, honey, fruits, vegetables, and processed foods. During food production and storage, plastic packaging and food-contact materials may also release particles directly into edible products. Lee *et al.,* (2023) reported that microplastics have been detected in both food consumed by humans and airborne environments, indicating that ingestion and inhalation are key exposure routes with potential health relevance.

Drinking water and bottled water are especially important because they are consumed daily. Qian *et al.,* (2024) developed stimulated Raman scattering microscopy for single-particle chemical imaging of nanoplastics and applied it to bottled water analysis. Their study showed that bottled water can contain large numbers of plastic particles, particularly nanoscale particles that may have been underestimated in earlier studies due to analytical limitations (Qian *et al*., 2024). This finding is medically relevant because nanoplastics may have greater biological interaction potential than larger microplastics due to their smaller size and higher surface-area-to-volume ratio.

Food-contact plastic products can also release substantial microplastic loads. Li *et al.,* (2020) investigated polypropylene infant feeding bottles and reported that these bottles released microplastics at levels as high as 16,200,000 particles per liter during infant formula preparation. This is highly relevant for early-life exposure because infants have developing gastrointestinal, immune, and metabolic systems, and bottle-fed infants may experience repeated daily exposure through formula preparation practices (Li *et al*., 2020).

Household food-preparation materials are another overlooked source of microplastic ingestion. Yadav *et al.,* (2023) showed that plastic chopping boards can release microplastics into food during cutting activities. Their study estimated that a person may be exposed annually to 14.5–71.9 million polyethylene microplastics and up to 79.4 million polypropylene microplastics from chopping-board use. Although their short-term toxicity assessment did not show adverse effects on mouse fibroblast cell viability after 72 hours, the study confirms that routine kitchen practices can directly introduce microplastics into human food (Yadav *et al*., 2023).

Single-use consumer plastic products may release even smaller particles, especially under hot-water or hot-beverage conditions. Zangmeister *et al.,* (2022) reported that common single-use plastic products can release trillions of sub-100 nm nanoparticles per liter into water during normal use. This is important because nanoscale particles may interact more easily with biological barriers, cells, and proteins than larger particles. Therefore, hot beverages, plastic-lined cups, plastic food bags, and other heat-exposed consumer products may represent important exposure sources (Zangmeister *et al*., 2022).

Airborne exposure is another medically relevant route, especially in indoor environments where synthetic textiles, carpets, furniture, plastic materials, household dust, and poor ventilation may increase inhalable microplastic levels. Inhaled microplastics may deposit in different regions of the respiratory tract depending on their size, shape, density, and aerodynamic behavior. Jenner *et al.,* (2022) detected microplastics in human lung tissue using μFTIR spectroscopy, analyzing 13 lung tissue samples and showing that microplastics can be present in human respiratory tissue. This supports inhalation as a biologically relevant pathway for internal exposure (Jenner *et al*., 2022).

Evidence of internal exposure has also been reported in human blood. Leslie *et al.,* (2022) detected and quantified plastic particles in human blood samples, showing that plastic particles are bioavailable for uptake into the human bloodstream. This finding suggests that at least some plastic particles, especially smaller particles, may cross biological barriers and enter systemic circulation. However, the detection of particles in blood does not prove direct disease causation; rather, it provides important evidence of internal exposure and highlights the need for further toxicokinetic and clinical studies (Leslie *et al*., 2022).

Dermal exposure may occur through cosmetics, personal care products, synthetic clothing, and skin-contact consumer products, but current evidence suggests that intact skin is generally a less important route than ingestion and inhalation. Lane *et al.,* (2025) noted that dermal penetration is mainly relevant for very small particles, particularly nanoscale particles, while oral and inhalation exposure remain the dominant routes for human health-risk assessment. Therefore, consumer products are most concerning when they contact food, beverages, hot liquids, indoor air, or infant feeding materials (Lane *et al*., 2025).

Overall, human exposure to micro- and nanoplastics is continuous, multi-source, and medically relevant. Food and water mainly contribute through ingestion, air and dust contribute through inhalation, and consumer products contribute through food contact, packaging, heat-driven release, and household contamination. Current evidence strongly supports the presence of microplastics and nanoplastics in exposure media and selected human biological samples, but health-risk assessment remains limited by methodological differences in sampling, contamination control, size detection limits, polymer identification, and dose estimation. Therefore, future medical research should prioritize standardized exposure assessment, vulnerable populations such as infants and children, and mechanistic links between particle exposure, oxidative

stress, inflammation, immune dysregulation, gut microbiome disruption, and systemic distribution.

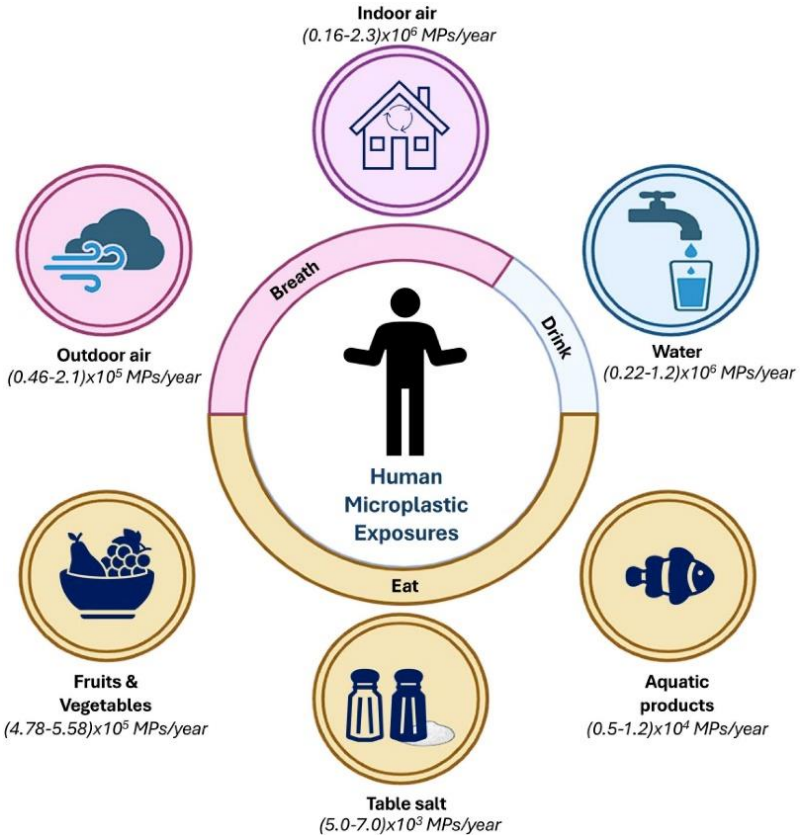


**Figure 10: Human exposure pathways of micro- and nanoplastics through food, water, air, and consumer products**

Human exposure pathways of micro- and nanoplastics through food, drinking water, air, and consumer products. The figure illustrates multiple daily exposure routes, including ingestion through water, salt, seafood, fruits and vegetables, and inhalation through indoor and outdoor air. Adapted from Bora *et al.,* (2024) under the Creative Commons Attribution Licens.

**Potential Effects on Gut Health, Immunity, Metabolism, and Inflammation**

Micro- and nanoplastics are increasingly discussed as potential disruptors of gastrointestinal health because the gut is one of the first biological interfaces exposed after ingestion. After entering the digestive tract through contaminated food, drinking water, beverages, and food-contact materials, plastic particles may interact with mucus, epithelial cells, gut microbiota, immune cells, and microbial metabolites. Current evidence suggests that micro- and nanoplastics can disturb gut homeostasis through four interconnected pathways: gut microbiome dysbiosis, intestinal barrier dysfunction, immune modulation, and metabolic inflammation (Bora *et al*., 2024; Thin *et al*., 2025). However, it is important to write carefully that most mechanistic evidence still comes from animal models, in vitro gut systems, and human-associated observational studies rather than large-scale clinical trials.

A major gut-related effect of microplastic exposure is intestinal dysbiosis, which means disruption of the normal balance between beneficial and harmful gut microorganisms. A systematic review of preclinical evidence reported that microplastics may trigger dysbiosis characterized by enrichment of bacterial groups such as Firmicutes, Proteobacteria, and Chlamydia, along with reduction of Bacteroidetes in several experimental models (Souza-Silva *et al*., 2022). More recently, a 2025 systematic review focusing on human-relevant studies concluded that microplastics are consistently associated with altered microbial composition, reduced microbial diversity, impaired short-chain fatty acid production, and changes in immune and metabolic pathways (Thin *et al*., 2025). This is medically important because the gut microbiome regulates nutrient metabolism, epithelial barrier integrity, immune tolerance, inflammation control, and protection against pathogens.

Human evidence is still limited, but some studies suggest biologically relevant associations. Yan *et al.,* (2022) detected 15 types of microplastics in human feces and reported that fecal microplastic concentration was positively correlated with inflammatory bowel disease status and disease severity. Polyethylene terephthalate and polyamide were among the dominant

polymers, and the primary particle shapes were sheets and fibers (Yan *et al*., 2022). This finding does not prove that microplastics cause IBD, but it supports the need to investigate whether chronic microplastic exposure may worsen gut inflammation in susceptible individuals.

Microplastics may also damage the intestinal barrier, which normally prevents pathogens, toxins, and inflammatory molecules from entering systemic circulation. Experimental studies show that plastic particles can reduce mucus secretion, alter goblet-cell function, downregulate tight-junction proteins, and increase intestinal permeability. Su *et al.,* (2024) reported that polystyrene microplastics affected mouse intestinal microstructure, reduced the expression of tight-junction-related genes, and altered gut microbiota, with stronger microbiome effects observed in female mice. Similarly, continuous oral exposure to micro- and nanoplastics in adult mice induced gut microbiota dysbiosis, intestinal barrier impairment, and immune dysfunction (Zhang *et al*., 2023). These findings suggest that epithelial barrier damage may be a central mechanism linking plastic exposure to gut inflammation.

Nanoplastics may be particularly concerning because their smaller particle size allows closer interaction with epithelial surfaces, mucus, extracellular vesicles, and bacterial cells. Hsu *et al.,* (2025) showed that 100 nm polystyrene nanoplastics accumulated in mouse intestinal tissues and disrupted the intestinal microenvironment by altering bacteria–host interactions. Their study found that nanoplastic exposure reduced tight-junction protein ZO-1 and mucin-13 expression, increased intestinal permeability, induced gut microbiota dysbiosis, and changed extracellular-vesicle-mediated communication between goblet-like cells and bacteria (Hsu *et al*., 2025). This provides strong mechanistic evidence that nanoplastics can affect gut health not only by physical irritation but also by interfering with molecular signaling between host cells and microbes.

Microplastic exposure may also activate immune and inflammatory pathways. The gut contains a large proportion of the body's immune cells, and any disturbance in epithelial integrity or microbial balance can activate local and systemic inflammation. Hasegawa *et al.,* (2024) exposed C57BL/6J mice to polystyrene microplastics through drinking water for six weeks and reported altered gut microbiota, increased intestinal natural killer cells, intestinal inflammation, altered nutrient-transporter gene expression, increased serum lipid levels, and aggravated fatty liver-related outcomes. The study concluded that high concentrations of polystyrene microplastics disturbed the intestinal environment and modulated nutrient metabolism even under normal dietary conditions (Hasegawa *et al*., 2024).

Diet may modify microplastic toxicity. Okamura *et al.,* (2023) showed that polystyrene microplastics produced stronger metabolic disturbance in mice fed a high-fat diet than in those fed a normal diet. In high-fat-diet mice, microplastic exposure was associated with higher blood glucose, higher serum lipid concentrations, increased nonalcoholic fatty liver disease activity scores, more inflammatory cells, fewer anti-inflammatory cells, increased expression of inflammation- and nutrient-transport-related genes, and higher abundance of *Desulfovibrio* in the intestine (Okamura *et al*., 2023). This suggests that microplastics may have stronger effects in metabolically vulnerable conditions such as obesity, high-fat diet exposure, leaky gut syndrome, insulin resistance, or fatty liver disease.

Microplastic-induced gut disruption may also influence metabolism through altered short-chain fatty acids, bile acid signaling, lipid absorption, glucose metabolism, and gut–liver communication. The gut microbiota normally produces short-chain fatty acids such as acetate, propionate, and butyrate, which help maintain epithelial barrier function, regulate inflammation, and support metabolic homeostasis. Thin *et al.,* (2025) reported that microplastic exposure was associated with impaired short-chain fatty acid production and altered metabolic pathways in human-relevant gut microbiome studies. In animal models, Hasegawa *et al.,* (2024) and Okamura *et al.,* (2023) further showed links between microplastic exposure, altered intestinal microbiota, dyslipidemia, glucose intolerance, and fatty liver-related outcomes. These findings support the concept that microplastics may influence systemic metabolism through the gut–liver axis.

Microplastics can also act as carriers of chemical additives and pollutants, which may intensify gut toxicity. Deng *et al.,* (2020) demonstrated that microplastics can adsorb and release phthalate esters in the mouse gut, resulting in intestinal accumulation of phthalates and aggravated intestinal inflammation and permeability compared with exposure to microplastics or phthalates alone. This is important because real-world microplastics are rarely "clean" particles; they may contain plasticizers, stabilizers, pigments, metals, persistent organic pollutants, endotoxins, and microbial biofilms. Therefore, gut toxicity may result from combined effects of the plastic particle, its chemical additives, and its adsorbed environmental contaminants.

Overall, evidence from 2020–2026 suggests that micro- and nanoplastics may affect gut health by disturbing microbial composition, weakening mucus and tight-junction barriers, activating immune responses, increasing intestinal permeability, altering microbial metabolites, and contributing to metabolic inflammation. The strongest mechanistic evidence comes from animal and cellular models, while human evidence is emerging but still limited. Therefore, in a medical relevance section, micro- and nanoplastics should be described as potential risk modifiers for gut inflammation, immune dysregulation, metabolic disturbance, and chronic

disease susceptibility, rather than as fully proven direct causes of human disease.

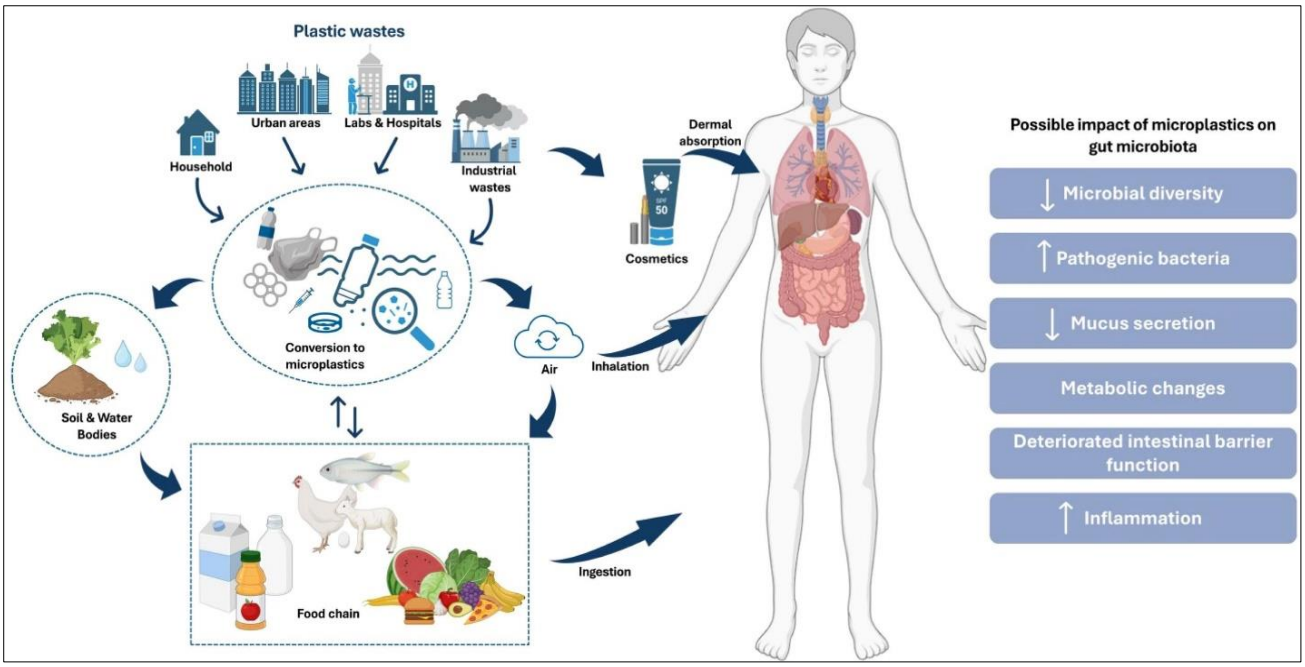


**Figure 11: Micro- and nanoplastic-induced gut dysbiosis, barrier disruption, immune activation, and metabolic inflammation**

This figure illustrates how micro- and nanoplastics can disturb gut homeostasis by altering gut microbiota, weakening the intestinal mucus and tight-junction barrier, and increasing intestinal permeability. These changes may activate immune and inflammatory pathways, disturb short-chain fatty acid production, and contribute to metabolic dysfunction such as lipid imbalance, glucose disturbance, and gut–liver inflammation.

**Translational Concerns: Tissue Accumulation, Chronic Disease Risk, and Clinical Uncertainty**

The translational relevance of micro- and nanoplastics is based on a central concern: these particles are no longer detected only in the external environment but also in human biological samples and tissues. Recent human biomonitoring studies have reported microplastics or nanoplastics in blood, lung tissue, placenta, breast milk, stool, semen, thrombi, vascular plaques, and post-mortem organs, suggesting that at least some particles can cross biological barriers and reach internal compartments (Leslie *et al*., 2022; Jenner *et al*., 2022; Ragusa *et al*., 2022; Roslan *et al*., 2024). A 2024 scoping review screened 3,616 records and included 26 in vivo human studies, reporting microplastics in 8 of 12 human organ systems, including cardiovascular, digestive, endocrine, respiratory, reproductive, urinary, lymphatic, and integumentary systems (Roslan *et al*., 2024).

Tissue accumulation is medically important because particle retention may expose organs to chronic low-grade physical, chemical, and biological stress. Microplastics may act not only as inert foreign particles but also as carriers of plastic additives, adsorbed environmental pollutants, metals, endocrine-disrupting compounds, and microbial biofilms. Once present in tissues, they may theoretically contribute to oxidative stress, epithelial or endothelial injury, immune-cell activation, mitochondrial stress, and inflammatory signaling. However, this does not mean that every detected particle automatically causes disease; rather, detection in tissue provides evidence of internal exposure and raises translational questions about dose, persistence, clearance, and long-term biological effects.

One of the strongest human translational signals comes from cardiovascular research. Marfella *et al.,* (2024) analyzed carotid atherosclerotic plaques from patients undergoing carotid endarterectomy and detected polyethylene in 150 patients and polyvinyl chloride in 31 patients. During an average follow-up of 34 months, patients with micro- and nanoplastics in their plaques had a higher risk of myocardial infarction, stroke, or death from any cause than those without detected particles (Marfella *et al*., 2024). This study is important because it links plastic particles in human vascular tissue with clinical outcomes, but it remains observational and cannot prove direct causation.

Additional vascular evidence comes from thrombus studies. Wang *et al.,* (2024) used multimodal detection methods to identify and quantify microplastics in thrombi surgically retrieved from cerebral arteries,

coronary arteries, and deep veins. Their study investigated whether microplastic levels were associated with thrombotic disease severity, supporting the hypothesis that plastic particles may be relevant to vascular pathology and clot-related disorders (Wang *et al*., 2024). These findings are translationally important because vascular inflammation, endothelial dysfunction, and thrombosis are central mechanisms in cardiovascular and cerebrovascular disease, but larger longitudinal cohorts are needed before microplastics can be considered established clinical risk factors.

Placental accumulation is another major concern because the placenta is a temporary but highly active maternal–fetal interface. Ragusa *et al.,* (2021) first reported microplastic fragments in human placenta, and Garcia *et al.,* (2024) later quantified nano- and microplastics in 62 placental samples using pyrolysis gas chromatography–mass spectrometry. Garcia *et al.,* (2024) reported that microplastics were detected in all 62 placental samples, with concentrations ranging from 6.5 to 685 µg/g of placental tissue and an average of 126.8 ± 147.5 µg/g. Polyethylene was the most prevalent polymer, accounting for 54% of total nano- and microplastic burden, while PVC and nylon each represented approximately 10% (Garcia *et al*., 2024).

From a translational perspective, placental findings are important because fetal development is highly sensitive to inflammation, oxidative stress, endocrine disruption, and impaired nutrient exchange. However, current placental studies mainly demonstrate presence and concentration, not direct clinical harm. Therefore, microplastic detection in placenta should be interpreted as a warning signal for developmental and reproductive toxicology rather than definitive proof of adverse pregnancy outcomes. Future studies should connect placental particle burden with birth weight, preterm birth, placental inflammation, maternal exposure patterns, and long-term child health.

Respiratory accumulation is also clinically relevant because inhaled microplastics may deposit in the upper or lower respiratory tract depending on size, shape, and aerodynamic behavior. Jenner *et al.,* (2022) analyzed 13 human lung tissue samples using µFTIR spectroscopy and identified 39 microplastics in 11 of 13 samples, with an average of 1.42 ± 1.50 microplastics per gram of tissue before background correction. The detection of particles in deeper lung regions supports inhalation as a biologically plausible route for internal exposure (Jenner *et al*., 2022).

The detection of plastic particles in blood provides another important translational link because systemic circulation may distribute particles or particle-associated chemicals to distant tissues. Leslie *et al.,* (2022) reported plastic particles in human blood, with a mean quantifiable concentration of approximately 1.6 µg/mL in a small donor group. This finding suggests that plastic particles are bioavailable for uptake into the bloodstream, although the study did not determine long-term retention, organ distribution, clearance rates, or disease causality (Leslie *et al*., 2022).

Brain accumulation has recently become one of the most debated areas. Nihart *et al.,* (2025) reported micro- and nanoplastics in post-mortem human brain, liver, and kidney samples using complementary detection approaches. Their data suggested that normal decedent brain samples had micro- and nanoplastic concentrations 7–30 times higher than liver or kidney samples, and dementia cases showed even greater presence. Importantly, the authors clearly stated that these data are associative and do not establish a causal role in neurological disease (Nihart *et al*., 2025).

Chronic disease risk remains biologically plausible but clinically uncertain. Experimental studies suggest that micro- and nanoplastics may contribute to inflammation, oxidative stress, gut microbiome disruption, epithelial barrier dysfunction, lipid metabolism disturbance, endothelial dysfunction, and immune activation. These mechanisms overlap with pathways involved in inflammatory bowel disease, metabolic syndrome, cardiovascular disease, fatty liver disease, respiratory disorders, reproductive dysfunction, and neurodegenerative disease. However, human evidence is still mostly cross-sectional, observational, or based on small tissue studies. Therefore, the strongest wording for a review article is that micro- and nanoplastics may act as potential chronic disease risk modifiers, not yet as proven independent disease causes.

Clinical uncertainty is a major limitation in this field. First, analytical methods differ widely across studies, including Raman microscopy, µFTIR, laser direct infrared spectroscopy, pyrolysis-GC/MS, and electron microscopy. Each method has different detection limits, contamination risks, polymer identification strengths, and limitations for nanoplastic detection. Roslan *et al.,* (2024) emphasized that extraction methods and analytical approaches vary substantially across human tissue studies, limiting direct comparison between studies (Roslan *et al*., 2024).

Second, there is no standardized clinical biomarker for microplastic exposure. Current studies report results using different units, including particles per gram, particles per milliliter, µg/g tissue, µg/mL blood, or polymer mass concentration. This makes it difficult to establish dose–response relationships, safe exposure thresholds, or clinically meaningful reference ranges. Lane *et al.,* (2025) noted that acceptable human health risk assessment methods for nano- and microplastic particles are still lacking because exposure and hazard assessments remain limited, and they recommended improved exposure scenarios, demographic-specific models, quality control, and probabilistic exposure assessment (Lane *et al*., 2025).

Third, detection does not equal disease causation. Many human studies confirm presence of particles but do not prove whether particles-initiated disease, accumulated because of pre-existing disease, or were influenced by sampling and analytical artifacts. For example, vascular plaque studies suggest a relationship with cardiovascular outcomes, but patients already had carotid artery disease. Brain studies show association with dementia status, but they cannot prove that plastic particles caused dementia. Therefore, future translational research must include larger cohorts, longitudinal follow-up, exposure history, standardized detection methods, contamination-controlled protocols, mechanistic biomarkers, and clinical endpoints.

Overall, the translational concern is not simply that micro- and nanoplastics are present in humans, but that they may persist in biologically sensitive tissues, interact with immune and metabolic pathways, and contribute to chronic disease susceptibility under long-term exposure. Current evidence supports internal exposure and possible tissue accumulation, while disease causality remains uncertain. Therefore, medical relevance should be framed carefully: micro- and nanoplastics represent an emerging environmental health concern with plausible links to inflammation, vascular disease, metabolic dysfunction, reproductive health, and neurotoxicity, but definitive clinical risk thresholds and causal pathways remain unresolved.

## Research Gaps, Future Directions, and Conclusion

### Major Research Gaps in Detection, Dose Standardization, and Long-Term Toxicity

Although micro- and nanoplastic research has expanded rapidly, major gaps still limit reliable health-risk assessment. The first gap is detection and characterization. Current methods such as Raman spectroscopy, FTIR, pyrolysis-GC/MS, and microscopy differ in sensitivity, particle-size limits, and polymer-identification accuracy, making results difficult to compare across studies. Nanoplastics are especially challenging because they are smaller, more reactive, and harder to isolate from biological samples (Nene *et al*., 2025; Roslan *et al*., 2024).

A second major gap is dose standardization. Studies report exposure as particles/L, particles/g, mg/L, µg/g tissue, particle number, mass, or surface area. This prevents accurate comparison between studies and weakens dose–response interpretation. Future studies should report multiple dose metrics together, including particle number, mass, size distribution, shape, polymer type, and surface chemistry (Lane *et al*., 2025; Vogel *et al*., 2024). Another limitation is the use of unrealistic experimental models. Many toxicology studies use pristine spherical polystyrene particles at high doses, while real-world exposure involves aged, irregular, mixed-polymer particles carrying additives, pollutants, metals, and microbial biofilms. Therefore, future work should use environmentally realistic particles and exposure concentrations (Prata *et al*., 2024).

Long-term toxicity remains poorly understood. Most available studies are acute or short-term, whereas human exposure occurs continuously through food, water, air, dust, and consumer products. More chronic low-dose studies are needed to assess tissue accumulation, inflammation, immune disruption, metabolic effects, reproductive toxicity, neurotoxicity, and carcinogenic potential (WHO, 2022; Kühnel *et al*., 2026). Overall, future research must focus on standardized detection protocols, validated reference materials, harmonized dose reporting, realistic exposure models, and long-term human cohort studies. Without these improvements, it will remain difficult to determine whether micro- and nanoplastics are only markers of exposure or true contributors to chronic disease risk.

### Future Directions in Biotechnology, Biosensors, Remediation, and Safer Materials

Future research should move from simple detection of micro- and nanoplastics toward prevention, real-time monitoring, biological degradation, and safer material design. A key biotechnology direction is the development of engineered enzymes and microbial systems capable of degrading plastic polymers under controlled conditions. PETase, MHETase, cutinases, and engineered PET hydrolases are promising for PET depolymerization, especially in industrial recycling systems. For example, FAST-PETase was designed using machine-learning-guided enzyme engineering and degraded untreated post-consumer PET from 51 thermoformed products within one week under laboratory conditions (Lu *et al*., 2022). However, enzymatic and microbial approaches still require improvement in stability, substrate range, degradation speed, and safety before large-scale environmental application (Choi *et al*., 2024).

Biosensor development is another important future direction. Current microplastic detection methods such as FTIR, Raman spectroscopy, and pyrolysis-GC/MS are accurate but often expensive, time-consuming, and laboratory-dependent. Biosensors may provide faster, cheaper, and field-deployable detection. Recent reviews highlight optical, electrochemical, microbial, aptamer-based, and enzyme-based biosensors as promising tools for micro- and nanoplastic monitoring (Daoutakou & Kintzios, 2025). A 2025 ACS Sensors study developed a green fluorescent protein-based microbial biosensor coupled with Raman spectroscopy, showing the potential of engineered microbes for rapid environmental microplastic detection (Choi *et al*., 2025).

Remediation strategies should combine physical, chemical, and biological approaches. Current and emerging technologies include membrane filtration, adsorption, coagulation–flocculation–sedimentation, electrocoagulation, advanced oxidation, biochar-based

adsorption, microalgal biopolymers, and bioremediation. A review of water and wastewater technologies emphasized that future remediation must be efficient, scalable, cost-effective, and environmentally safe (Yang *et al*., 2023). Microalgal extracellular polymeric substances and biopolymers are especially promising because they can aggregate nano- and microplastics and may replace hazardous synthetic flocculants in wastewater treatment (Cunha *et al*., 2020).

Safer material design should become a core prevention strategy. Future plastics should be designed for reduced toxicity, lower additive hazard, improved recyclability, controlled degradation, and minimal microplastic release across their life cycle. The OECD recommends integrating sustainable chemistry into plastic material selection to protect human and environmental health throughout the product life cycle (OECD, 2021). However, biodegradable plastics should not be treated as automatically safe because some biodegradable materials can still fragment into biodegradable microplastics before full mineralization. Therefore, safer materials must be evaluated under real soil, freshwater, marine, composting, and human-exposure conditions (Costa & Lackner, 2025).

Overall, future work should prioritize a source-control and circular-economy approach: reduce unnecessary plastic production, design safer polymers, improve recycling, monitor particles using portable biosensors, remove particles from wastewater before environmental release, and use biotechnology mainly in controlled systems. The strongest future direction is not one single solution but an integrated framework combining biotechnology, biosensing, remediation engineering, green chemistry, and policy-based prevention.

## CONCLUSION

Micro- and nanoplastics have emerged as persistent biological contaminants that connect environmental pollution with ecosystem instability, microbial dysbiosis, animal toxicity, food-chain exposure, antimicrobial resistance, and human-health uncertainty. This review highlights that these particles are not uniform pollutants; their behavior and toxicity depend on size, shape, polymer type, surface chemistry, weathering status, biofilm formation, and contaminant load. Their widespread sources, including plastic waste, textiles, tyre wear, agricultural plastics, wastewater, packaging, and atmospheric deposition, allow continuous movement across soil, water, air, food webs, and biological systems.

From a One Health perspective, micro- and nanoplastics represent a shared threat to ecosystems, animals, microbes, and humans. In plants and soils, they can disturb seed germination, root growth, photosynthesis, nutrient cycling, microbial activity, and soil health. In animal models, exposure has been associated with oxidative stress, inflammation, gut injury, reproductive toxicity, developmental effects, neurotoxicity, and metabolic disruption. In microbial systems, plastic particles form plastisphere biofilms that may alter microbial communities and facilitate the spread of antimicrobial-resistance genes. Your article strongly emphasizes that the plastisphere is not only an ecological surface but also a potential microbial and genetic vector within the One Health framework.

Human exposure occurs through contaminated food, water, air, dust, and consumer products. Recent detection of microplastics in human biological samples and tissues raises important medical concerns, but current evidence is still limited by methodological differences, contamination risks, weak dose standardization, and insufficient long-term epidemiological studies. Therefore, micro- and nanoplastics should be viewed as emerging risk modifiers rather than fully proven direct causes of chronic disease. Stronger mechanistic studies, realistic exposure models, and human cohort data are needed to clarify their role in inflammation, metabolic disorders, immune dysfunction, reproductive health, cardiovascular risk, and neurological outcomes.

Overall, this review supports the conclusion that micro- and nanoplastic pollution is not only an environmental issue but a cross-sectoral biological and public-health challenge. Future progress requires standardized detection methods, harmonized dose metrics, long-term toxicity studies, portable biosensors, biotechnology-based remediation, safer polymer design, improved waste management, and circular-economy strategies. A One Health framework provides the most suitable scientific approach because it connects environmental contamination, microbial ecology, animal biology, food-chain safety, and human medicine into one integrated risk-assessment model. Micro- and nanoplastics should therefore be prioritized in future environmental monitoring, biomedical research, public-health planning, and sustainable material innovation.

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