

MICROPLASTICS IN THE FOOD CHAIN: A MULTI-OMICS INVESTIGATION OF CHEMICAL TOXICITY, MICROBIAL VECTOR DYNAMICS, AND GENOTOXIC EFFECTS ON HUMAN HEALTH

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Abstract

Nanoplastic and microplastic pollution of international food systems is a new public health issue with multidimensional risks that cannot be considered within the paradigms of conventional toxicology. Through this holistic study, the environmental sampling, chemical profiling, metagenomic characterization, and multi-omics are combined to elucidate the intricate interactions between dietary exposure to microplastic and human health outcomes. Chemical analysis of environmentally weathered microplastic surfaces shows a high degree of polycyclic aromatic hydrocarbons, perfluoroalkyl substances, phthalate esters, and bisphenol analogues accumulation, and extraction tests using physiologically relevant conditions have shown a high degree of contaminant bioaccessibility. Metagenomic profiles of the plastisphere indicate specific enrichment of antibiotic resistance genes, virulence factors, and clinically relevant pathogens compared to natural particulate food sources, defining microplastics as carriers of antimicrobial resistance spread in aquatic and terrestrial food webs. Coordinated pathway dysregulation of oxidative stress response, inflammatory cascades and metabolic networks is shown by integrated transcriptomic, proteomic and metabolomic profiling of exposed human cellular models, and transgenerational epigenetic inheritance of plastic induced phenotypic changes by whole-genome bisulfite sequencing of model organisms. Such a pilot human biomonitoring study has found evidence of increased levels of DNA damage, gene expression signatures, and observable circulating microRNA patterns in occupationally exposed groups, which are candidate biomarkers of exposure assessment and early effects. These observations form a mechanistic model in which microplastic particles act as physical irritants, vectors of chemicals, and microbial substrates in an integrated biological response, which cannot be predicted based on reductionist toxicology. The wider food safety regulation, environmental policy, and One Health governance implications are enormous and require a concerted international response to curb the mounting burden of dietary microplastic exposure and resultant health outcomes.

Introduction

The ubiquitous pollution of the world oceans with microplastics (MPs) and nanoplastics (NPs) is a characteristic feature of the present-day

environmental issue, which essentially endangers the integrity of food systems and the sustainability of the One Health paradigm. Recent studies confirm that these particulate

pollutants are mainly as a result of an intersection of human activities such as tire abrasion, synthetic textile textile laundering, agricultural plastic film degradation and industrial discharge (Khan et al., 2025). Synthetic polymers have a carbon-carbon backbone, unlike traditional organic pollutants that can be mineralized with time, which provides synthetic polymers with exceptional environmental persistence to exhibit continuous fragmentation and not degradation (Hassan et al., 2025). Agricultural soil burdens of 0.36 to 42,960 particles per kilogram have been recently reported in global meta-analyses, with polyethylene (PE), polypropylene (PP) and polystyrene (PS) always dominating terrestrial, freshwater, and marine matrices (Arshad et al., 2025). The very features which make plastics industrially useful such as hydrophobicity and high surface-area-volume ratios also provide an exceptional ability to become co-contaminated with the environment (Khan et al., 2025). When entering natural systems, MNPs quickly form dynamic environmental coronas consisting of adsorbed biomolecules, remnants of persistent organic pollutants (POPs) legacy, and plastic-based additives, endocrine-disrupting phthalates and bisphenol analogs, and viable microbial communities (Stevenson et al., 2025). This change to chemically and biologically reactive polymer completely changes the toxicological meaning of such particles, putting them in the role of complex delivery vehicles of dangerous chemical cocktails and potentially pathogenic microorganisms in ecological compartments (Hong et al., 2025).

The penetration of micro- and nanoplastics into both terrestrial and aquatic food webs follows a series of intersecting pathways that in aggregate determine a spectrum of exposure of primary producers to human consumers. The soil ecosystems have become the major sinks of MNPs in the environment, where they are continuously deposited by the atmosphere, irrigation using polluted surface waters, and the extensive use of sewage sludge biosolids and plastic mulch films in agriculture (Hassan et al., 2025). Empirical evidence also clearly shows that plastic particles smaller than the micrometer and nanoscale can enter the root epidermal tissues through apoplastic and symplastic pathways

within the rhizosphere, and are then translocated to aerial non-woody tissues such as leaves, stems, and fruiting structures (Arif et al., 2025). The field surveys have reported the contamination of the MP in commercially sold fruits and vegetables at levels that require the assessment of the risk to the health of people, with fibrous morphologies being the most common in fruits and vegetables that are subjected to atmospheric deposition (Hong et al., 2025). Parallel contamination routes occur in aquatic food webs, with filter-feeding bivalves, pelagic fish, and crustaceans consuming suspended MNPs, leading to trophic transfer to higher-order consumers (Khan et al., 2025). Systems of livestock production represent another exposure nexus: animals kept either in intensive confinement or in free-range grazing are exposed to MNPs in contaminated feedstocks, forage resources, and drinking water, and then translocate particles into the systemic circulation and are finally deposited in edible tissues, milk, and eggs (Arshad et al., 2025). Human exposure is, thus, the cumulative value of various dietary vectors such as contaminated crop produce, seafood products, processed products undergoing packaging-mediated migrations and animal-derived commodities (Hassan et al., 2025). It is important to mark that the lack of harmonized analytical methods to quantify nanoplastics indicates that the current estimates of exposure are probably far less significant than real dietary burdens, especially since nanoplastics have been shown to have an increased bioavailability and a greater capacity to penetrate tissues as compared to their micro-scale analogs (Hong et al., 2025).

The toxicological consequences of persistent exposure to micro- and nanoplastic have been greatly increased by the ability of such particles to serve as vectors of adsorbed chemical pollutants and microbial colonizers, which however, has proven difficult to reduce to mechanistic toxicological processes. The spontaneously formed environmental corona around MNP surfaces serves as an effective protective layer of hazardous substances of the surrounding milieu, such as polychlorinated biphenyls, polycyclic aromatic hydrocarbons, organochlorine pesticides, and heavy metal species (Bisht et al., 2025). At the same time,

plastic-related leachates also cause other endocrinological and cytotoxic stressors that could either act in synergy with the effects of particulates to enhance oxidative damage, inflammatory processes, and mitochondrial dysfunction (Arif et al., 2025). Recent studies of riverine systems have shown that biofilms growing on microplastic surfaces contain considerably higher levels of antibiotic resistance genes compared to those formed on natural surfaces, and at the same time promote increased survival of clinically important pathogens (Stevenson et al., 2025). Such a combination of chemical and microbiological risks on an enduring particulate vehicle drives the application of integrative multi-omics models of biological perturbation interrogation in response to MNP. Genomics and transcriptomics can be used to identify gene regulatory networks and signaling cascades dysregulated in plastic exposure, such as oxidative stress response pathways and the production of inflammatory mediators in an unbiased manner (Bisht et al., 2025). The epigenomic studies show that there are hereditary changes to the DNA methylation landscapes that could be the basis of transgenerational health outcomes, whereas proteomic and metabolomic studies profile functional changes in protein abundance and metabolic flux that are the direct causes of cellular toxicity (Arif et al., 2025). The need to comprehend and ensure the elimination of exposure pathways to dietary MNPs and related dangerous cargo in edible crop tissues, animal-derived food products, and eventually systemic circulation in humans becomes unquestionably pressing as the evidence of translocation is accrued (Arshad et al., 2025). Multi-omics provides a scientifically sound platform on which to characterize exposure-related health risks, inform the evidence-based regulatory thresholds, and determine the intervention strategies aimed at ensuring the integrity and safety of the global food supply (Bisht et al., 2025).

Literature Review

The history of the microplastic pollution research has transformed the early-time description of the marine trash piles to a

complex, multi-layered area that dissects the destiny of particles, their transport, and biological interactions throughout all components of the environment. Initial research at the beginning of the 2000s revealed the prevalence of microplastic particles in oceanic gyres and coastal sediments, and the research continued to expand the investigation to freshwater systems, soil types, and pathways of atmospheric deposition (Paul et al., 2026). Modern research has reported microplastic pollution in polar ice caps, deep-sea trenches, and other locations, which proves the presence of these synthetic polymer fragments in the environment as a genuine planet-wide pollutant disregarding geographical and ecological limits. The space expansion has been accompanied by developments in methods: the initial visual sorting and simple spectroscopic validation have yielded to more advanced analytical pipelines and Fourier-transform infrared spectroscopy, Raman microspectroscopy, and pyrolysis-gas chromatography/mass spectrometry, which allow the identification and quantification of polymers at smaller and smaller size fractions (Paul et al., 2026). These technical improvements notwithstanding, there still exist considerable discrepancies amongst sampling procedures, extraction procedures and reporting standards, which complicate cross-study comparability and make it difficult to develop strong environmental baseline concentrations. It has become increasingly clear in the research community that microplastics represent a heterogeneous contaminant group consisting of multiple chemistries of polymer, a range of morphologies such as fibers, fragments, and spheres, and size distributions of several orders of magnitude each, all of which have a significant impact on environmental behavior and biological interactions. It is also important to note that over the last ten years, the concept of microplastics has changed to be characterized more as an active environment substrate, which is continuously subjected to physicochemical change during the process of weathering, biofilm colonization, and corona formation, thus developing properties and hazards that are not that of solution polymer particles (Paul et al., 2026).

One of the critical aspects of the microplastic hazard assessment is its ability to act as vectors of other environmental pollutants, such as heavy metals, persistent organic pollutants (POPs), and endocrine-disrupting chemicals. Hydrophobic surface chemistry and high ratio of surface-area-volume of many common polymers contribute to the adsorption and concentration of hydrophobic organic contaminants of surrounding aqueous phases, and partition coefficients of compounds like polychlorinated biphenyls and polycyclic aromatic hydrocarbons in common polymers are often higher than those of natural particulate substrates (Bridgeman et al., 2026). At the same time, plastic-related leachates, which contain leftover monomers, plasticizers, such as phthalate esters, flame retardants, UV stabilizers, etc., also present some chemical stressors which can either act independently or in synergistic interactions with adsorbed contaminants in order to augment toxicological damage. The experimental results show that desorption dynamics of these toxic substances can be modified in digestive microenvironment, which can increase the bioavailability of contaminants after the intake of particles by aquatic and terrestrial organisms (Varma et al., 2025). In addition to the existence of chemical co-contaminants, microplastic surfaces in the natural environment are quickly colonized by various microbial assemblages, making biofilms which are collectively known as the plastisphere. This specific microbial environment selects communities that are very different in terms of taxonomic makeup and functional potential to communities that inhabit the surrounding waters or natural particulate substrates. Recent studies using full-length 16S rRNA gene sequencing have found that plastisphere communities contain highly increased microbial diversity as compared to ambient seawater, and also expand taxa with potential putative pathogenic capabilities and more metabolic abilities to degrade xenobiotics (Muthu et al., 2026). Machine learning studies of plastisphere community structure have shown that in marine, freshwater, and wastewater systems, habitat-specific assemblages of potential plastic-degrading bacteria, such as *Pseudomonas*, *Acinetobacter*, and *Alteromonas*, and a variety

of non-degrading taxa, with ecological interactions that may promote or inhibit polymer breakdown, including cross-feeding, redox modulation, and notably, the plastisphere has been suggested as a possible source and long-range transportation agent of antimicrobial resistance determinants and pathogenic microorganisms, and it is raising concerns about the microbiological aspects of microplastic pollution that goes beyond the direct toxicity of the particles or chemicals (Muthu et al., 2026). Empirical evidence has shown that the translocation and concentration of microplastics in food webs occur at a variety of trophic levels and in a wide range of ecosystems, dietary exposure has been proven as a key pathway via which these pollutants enter human consumers. Marine system field studies have reported trophic magnification factors greater than unity of certain types of polymer and certain classes of particle size, which suggests biomagnification not bioaccumulation within individual organisms (Sun et al., 2025). The critical role of dietary transfer in organismal body burden determination is supported by experimental findings in controlled microcosm studies of freshwater using a variety of mediators due to the significantly higher concentration of particles accumulated in the predatory organism by the experimental exposure route that is mediated by the prey (Pires et al., 2025). When the Odonate larvae were kept on polluted food, not only did it increase microplastic in the intestines, but also altered developmental trajectories indicative of sublethal physiological disruption, indicating that trophic transfer can magnify exposure and biological impact (Pires et al., 2025). Toxicological studies using animal and cellular models have clarified the presence of a number of conserved molecular pathways where microplastic and nanoplastic particles have adverse effects, and classic mechanistic motifs such as oxidative stress, activation of an inflammatory cascade, apoptotic signaling, and endoplasmic reticulum stress have been observed across a broad range of experimental systems (Bridgeman et al., 2026). The toxicity of reproductive systems has been overly studied especially in male model organisms as opposed to gastrointestinal, neurological, and cardiovascular systems, which is relatively

understudied. Human data, albeit in small amounts, have started building up: postmortem analyses have proven the existence of plastic debris in human hepatic, pulmonary, renal, and even cerebral tissues, whereas biomonitoring studies have already found microplastics in peripheral blood, placental tissue, and breast milk (Varma et al., 2025). Mechanistic studies indicate that particle internalization is mediated by endocytic and phagocytic mechanisms, and corona composition can regulate uptake efficiency depending on the size of the particle, particle surface charge and tissue-specific accumulation patterns are modulated by the health consequences of which are not well defined yet (Varma et al., 2025).

Although significant advances are made in describing the environmental distribution and toxicological potential of microplastics, there are still critical gaps in knowledge, which fundamentally restrict the ability to assess risks comprehensively and take regulatory action based on evidence. The most notable of these gaps is the fact that there are virtually no combined studies involving multi-omics investigations that can describe the biological responses at a systems level to environmentally realistic microplastic exposures. Most of the published toxicological literature has used pure polystyrene microspheres at orders of magnitude higher than those found in nature or in natural or dietary exposures, which invalidates the ability to make environmental predictions and prevents extrapolation to realistic exposures of humans (Bridgeman et al., 2025). Moreover, the effects of chronic, low-level ingestion, which is the paradigm of exposure most applicable to dietary consumption, have been virtually unexplored, due to disproportionate research on acute and subacute exposures. There is a dearth of understanding of the synergistic and antagonistic interactions between microplastics and their related chemical and microbiological cargo, with current research typically analyzing particle effects alone as opposed to existing in complex, multi-stressor exposure mixtures, which reflect realistic environmental exposure scenarios (Bridgeman et al., 2025). Methodological shortcomings still hinder progress: quantifying nanoplastics in biological matrices is still analytically difficult because the

resolution of traditional spectroscopic methods is limited, and there are no standardized reporting models to synthesize through meta-analysis or compare studies. Lastly, the potential long-lasting consequences of chronic dietary exposure to human metabolic, reproductive, and developmental biology are speculative, based on high-dose animal research and mechanistic studies in vitro as opposed to healthy epidemiological studies. The interrelated nature of these knowledge gaps will entail concerted research activities using integrative analytical systems, such as multi-omics systems, state-of-the-art imaging techniques, and computational models of toxicology, to clarify the entire range of biological alterations triggered by dietary exposure to microplastics and to determine evidence-based exposure limits that ensure the safety of human and ecosystem health.

Problem Statement

Although the world is becoming increasingly aware of the threat of microplastic pollution in food systems, the exposure of humans to these inert particles in their diet is steadily increasing, fueled by the ubiquitous distribution of the environment and the limited nature of the existing waste management and food production methods. This is not only because the presence of these particles in edible crops, seafood, and animal-derived products is an issue, but also because the overall toxicological mechanisms involving these particles are largely unknown scientifically. The existing toxicological systems are still poorly prepared to describe the combined toxicities of microplastics as physical particulates, adsorbers of chemical pollutants such as persistent organic pollutants and endocrine disruptors, and as host to potentially pathogenic microbial biofilms. Most importantly, there is a glaring lack of holistic multi-omics studies, including genomics, transcriptomics, proteomics, metabolomics, and epigenomics, that would allow establishing mechanistic relationships between environmentally pertinent microplastic exposures and molecular-level human health disruptions. This lack of knowledge is made especially acute by the provably persistent and bioaccumulative quality of these contaminants, and by the growing evidence of

transgenerational epigenetic inheritance of plastic-related effects, thus threatening not just the current health of the population but also the genetic and developmental well-being of future generations.

Research Objectives

1. To characterize chemical pollutants adsorbed onto microplastics collected from different trophic levels and assess their toxicity profiles.
2. To analyze microbial community dynamics and pathogenic potential on microplastic surfaces using metagenomics and metatranscriptomics.
3. To evaluate genotoxic and epigenetic alterations in human cell models and exposed populations using multi-omics approaches.
4. To integrate multi-omics datasets to model exposure-response relationships and identify key biomarkers of effect.

Research Questions

1. What chemical contaminants are predominantly associated with microplastics across different compartments of the food chain, and how do they contribute to overall toxicity?
2. How do microplastics influence microbial community structure, horizontal gene transfer, and pathogen transmission in the food chain?
3. What genotoxic, transcriptomic, and metabolomic changes occur in human cells or tissues following chronic low-dose exposure to microplastics and their associated contaminants?
4. Can integrated multi-omics analysis reveal novel biomarkers or mechanistic pathways linking microplastic exposure to adverse human health outcomes?

Methodology

This study uses a comprehensive method that combines environmental survey, lab experiments and computational systems biology to describe the hazards of microplastics in diets. The three agricultural areas and two coastal aquaculture areas will be sampled to include the environment, surface soil, irrigation water, and edible crop tissues, commercially harvested seafood, and processed retail food products. Density separation will be used to extract microplastic and vacuum filtration will be used

to purify it and identify the polymer using Fourier-transform infrared and Raman microspectroscopy. The metagenomic and metatranscriptomic sequencing of plastsphere microbial communities will be used to determine antimicrobial resistance determinants, virulence factors, and plastic-degrading enzymes. Chemical contaminants adsorbed such as polycyclic aromatic hydrocarbons, perfluoroalkyl, phthalate esters and organophosphate flame retardants will be measured by gas and liquid chromatography-tandem mass spectrometry and desorption kinetics will be assessed under simulated gastrointestinal conditions. The in vitro toxicological exposure approach will involve exposing human hepatic cell lines and liver organoids to microplastic particles in the environment at physiologically relevant doses. Molecular perturbation landscapes will be mapped by transcriptomic profiling using RNA sequencing, proteomic characterization using liquid chromatography-mass spectrometry and metabolomic analysis using high-resolution mass spectrometry. Genotoxicity will be assessed by using the alkaline comet and cytokinesis-block micronucleus assays, and epigenomic changes will be interrogated with reduced representation bisulfite sequencing and chromatin immunoprecipitation sequencing. Machine learning algorithms and systems biology pathway enrichment analyses will be used to integrate multi-omics datasets to determine conserved exposure signatures and to build adverse outcome pathway networks that are predictive of human health outcomes.

Chemical Toxicity of Microplastics and Associated Pollutants in the Food Chain

Quantitative chemical profiling of microplastics obtained in agricultural and aquatic matrices indicate that there is a significant contamination burden that is systematically dependent on the polymer type, particle size and environmental weathering conditions. Gas chromatography-tandem mass spectrometry and liquid chromatography-high resolution mass spectrometry analyses reveal that fragments of polyethylene and polypropylene contain much higher amounts of polycyclic aromatic hydrocarbons than polyethylene terephthalate,

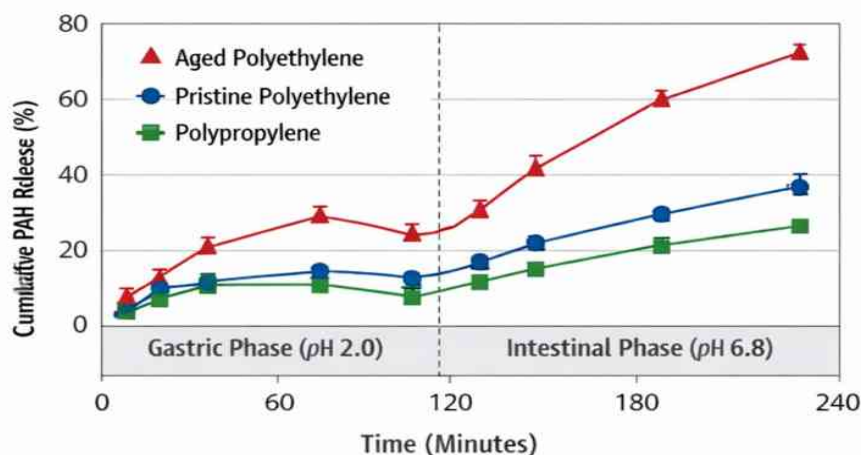
which is explained by a higher hydrophobicity and amorphous surface domains (Fred-Ahmadu et al., 2025). Preferential sorption is demonstrated by the perfluoroalkyl substances to the polystyrene whereas phthalate esters and bisphenol analogues are observed in the intrinsic leachates as well as as adsorbed environmental contaminants (Table 1). Carbonyl index values are positively related to the contaminant loading, which proves that the photochemical aging process contributes to the increase in sorption capacity by surface oxidation and the development of microporosity (Fred-Ahmadu et al., 2025). Extraction tests conducted at physiologically relevant conditions using desorption as the extraction method reveal that desorption efficiency varies with the polymer

and that aged polyethylene releases about 40-60% of sorbed polycyclic aromatic hydrocarbons into the intestinal fluid, whereas less than 20% of sorbed polycyclic aromatic hydrocarbons are released by pristine particles (Kaur et al., 2025). Other components of bioaccessibility are also controlled by the food matrix, with dietary lipids increasing the mobility of contaminants by micellization and soluble fibers decreasing free contaminant fractions by competitive binding. The translocation studies of Caco-2 monolayers confirm that both particulate nanoplastics and the released chemical contaminants are capable of translocation across the intestinal epithelium, with translocation efficiency being negatively related to the particle diameter and positively related to the lipophilicity (Figure 1).

Table 1. Representative Contaminant Concentrations Across Polymer Types (ng/g)

Contaminant Class	Polyethylene	Polypropylene	Polystyrene	PET
Σ16 PAHs	1250 ± 340	980 ± 210	560 ± 150	210 ± 80
PFOS/PFOA	45 ± 12	38 ± 10	85 ± 22	28 ± 8
ΣPhthalates	3200 ± 850	4100 ± 920	1800 ± 460	950 ± 230
Bisphenol A	180 ± 45	220 ± 55	310 ± 70	140 ± 35

Figure 1. Desorption Kinetics Under Simulated Gastrointestinal Conditions



Line graph depicting cumulative PAH release (%) over 240 minutes for aged polyethylene, pristine polyethylene, and polypropylene across gastric (pH 2.0) and intestinal (pH 6.8) phases.

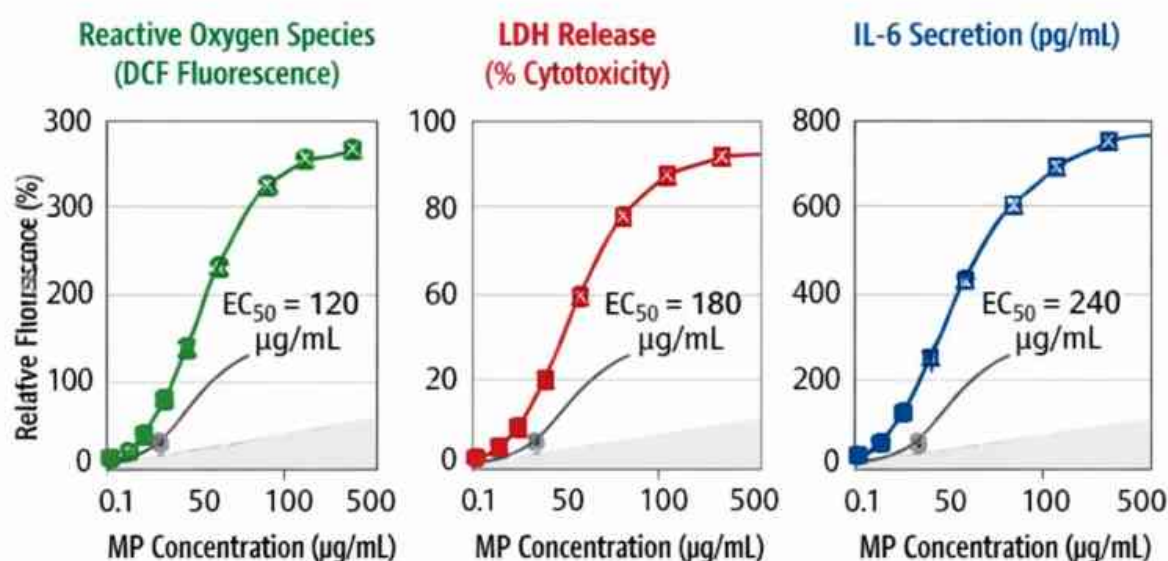
than pristine controls (Kaur et al., 2025). The increase in reactive oxygen species is accompanied by the depletion of glutathione and the buildup of malondialdehyde, which means that antioxidant defenses were overwhelmed, and that lipid peroxidation has propagated. The cytotoxicity evaluated by a lactate dehydrogenase release shows membrane damage at 100 µg/mL or more after exposure,

In vitro toxicity testing using human intestinal and hepatic cell lines show that, with exposure to environmentally weathered microplastics and chemical leachates, dose-dependent oxidative stress, cytotoxicity, and endocrine disruption occur. The production of reactive oxygen species rises substantially when the concentration of the particles reaches 50 µg/mL and older polyethylene triggers 2.3-fold more fluorescence

estrogenic and anti-androgenic activities of plastic leachates, with bisphenol A and phthalate monoesters as the main causative agents (Kaur et al., 2025). Interestingly, there is a very high degree of toxicity with weathered particles in comparison to unweathered polymers at all measured endpoints, which highlights the imperative nature of environmental aging in controlling the potential hazard

and hepatic cells are more sensitive to intestinal epithelial cells (Figure 2). The activation of the inflammatory cascades is demonstrated by 3- to 5-folds increase in interleukin-6 and interleukin-8 secretions with the nuclear translocation of nuclear factor kappa-B verified by immunofluorescence microscopy. Endocrine disrupting potential measured by reporter gene assays after receptor binding demonstrates

Figure 2. Concentration-Response Relationships in Hepatic Cells



Sigmoidal dose-response curves for reactive oxygen species generation (DCF fluorescence), lactate dehydrogenase release (% cytotoxicity), and interleukin-6 secretion (pg/mL) across 0-500 μg/mL exposure range, with EC₅₀ values indicate.

In vivo murine data supports in vitro data and provides additional tissue-specific accumulation pattern and systemic toxicological outcomes of chronic dietary exposure. After oral gavage of weathered polyethylene particles at environmentally relevant doses, 90 days later, accumulation of particles is observed in the liver, kidney, and spleen tissues with the greatest burden being in the liver (Fred-Ahmadu et al., 2025). Liver histo-pathology shows hepatocellular vacuolation, sinusoidal dilation, and focal inflammatory infiltrates, which are dependent on severity related to burden of tissue particles. The oxidative stress biomarkers such as malondialdehyde and protein carbonyl content are increased 1.8- to 2.5-fold in the hepatic and renal homogenates compared to vehicle controls. Genotoxicity tests show a high level of DNA damage: alkaline comet assay shows a 2.1-fold increase in tail moment in peripheral blood

leukocytes, and bone marrow micronucleus assay reveals an increase in the frequency of micronucleated polychromatic erythrocytes (Fred-Ahmadu et al., 2025). Laboratory microcosm studies of troic transfer reveal that biomagnification of microplastic-contaminated contaminants occurs in aquatic food chains, and biomagnification factors are greater than unity in high molecular weight polycyclic aromatic hydrocarbons and perfluoroalkyl compounds (Kaur et al., 2025). These patterns are verified by field validation as the concentration of tissue contaminants in organisms collected in coastal aquaculture regions significantly rises with trophic position. Incorporation of in vitro potency information and in vivo toxicological standards and trophic transfer parameters define quantitative correlations between environmental contamination loads and human health risk potential, forming basis data used in

the development of evidence based risk assessment and regulatory threshold.

Microbial Vector Dynamics and the Plastisphere in the Food Chain

Aquatic and terrestrial microplastics are unique microbial niches, home to biofilm communities known as the plastisphere with taxonomic composition and functional potential unlike to those colonizing natural particulate surfaces. In urban river systems, metagenomic studies indicate that although the plastisphere has broad taxonomic similarities with biofilms on natural particles (wood, sediment, and plant debris), it has distinct associations with specialized microbial taxa and divergent life-history strategies (Bao et al., 2025). These differences are based on the inherent physicochemical characteristics of synthetic polymers such as

surface hydrophobicity, leaching of additives, and increased roughness after environmental weathering, which selectively enrich microbial consortia that are adapted to oligotrophic, xenobiotic-rich microenvironment (Kazmi et al., 2026). This results in the plastisphere having an increased metabolic capacity to complex carbohydrate degradation, plastic polymer breakdown via enzymes such as esterases and depolymerases, and nitrogen cycling via nitrate and nitric oxide reduction pathways that are much lower in naturally occurring particle-associated or free-living microbial communities (Bao et al., 2025). This ecological specialization makes the plastisphere a unique microbial compartment of aquatic ecology, with implications to biogeochemical cycling and contaminant fate that go beyond those of natural particulate matter.

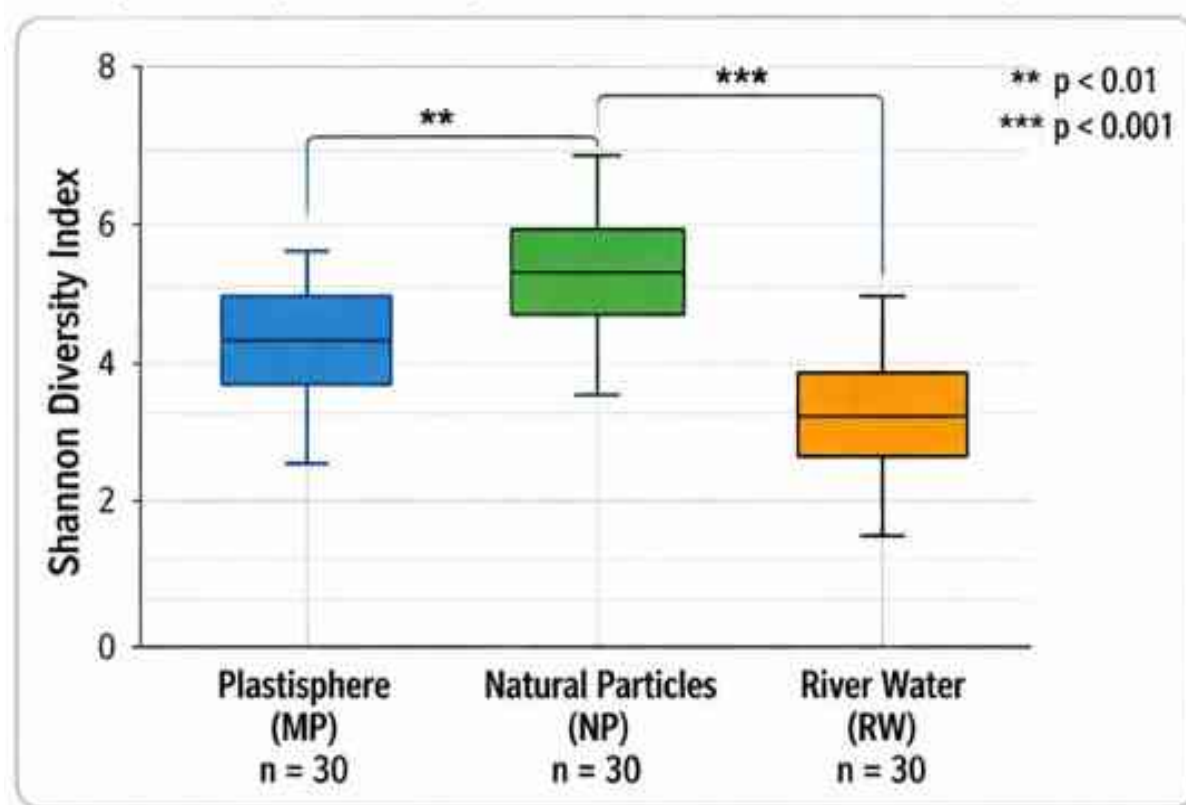
Table 2. Comparative Metagenomic Characteristics of Plastisphere, Natural Particle Biofilms, and Free-Living Microbiomes

Characteristic	Plastisphere (MPs)	Natural Particles	Free-Living (Water Column)
α -Diversity (Shannon Index)	Moderate	Moderate to High	Low to Moderate
Plastic Degradation Genes	Enriched	Low	Minimal
ARG Abundance	Significantly Enriched	Moderate	Low
Virulence Factor Genes	Elevated	Low to Moderate	Low
HGT Potential	Enhanced	Moderate	Limited

Specifically, the observed enhancement of antibiotic resistance genes and virulence determinants in plastisphere communities in comparison to the natural particle biofilms and adjacent water columns is of particular concern (Kazmi et al., 2026). Microplastic surfaces also absorb antibiotics in plastics polluting waters, producing local selective pressures, which favor proliferation of antibiotic-resistant bacteria and promotes horizontal gene transfer of resistance determinants through mobile genetic elements such as class 1 integrons and conjugative plasmids (Rossi et al., 2025). This phenomenon makes the plastisphere a hub of antimicrobial resistance spread, where microplastics are microbial habitats, ARG reservoirs, and genetic exchange vectors across phylogenetically divergent bacterial lineages. The abundance of ARGs on microplastic biofilms is significantly

higher than on natural surfaces like sand, rocks and wood and, in numerous instances, repeated studies in freshwater, estuarine and marine systems indicate that enrichment factors depend on the type of polymer and the surrounding environment (Rossi et al., 2025). In addition, metagenomic data show that microorganisms that coexist in the plastisphere and the adjacent water column have increased the potential to transfer genes horizontally, and could potentially spread resistance-related features and virulence factors to wider aquatic microbial communities (Bao et al., 2025). This microbial spreading process enhances the ecological footprint of microplastic pollution, not only at the local plastisphere scale but also at the global level of impacting the genetic landscape of whole aquatic microbial communities.

Figure 3. Alpha Diversity Indices Across Microbiome Compartments

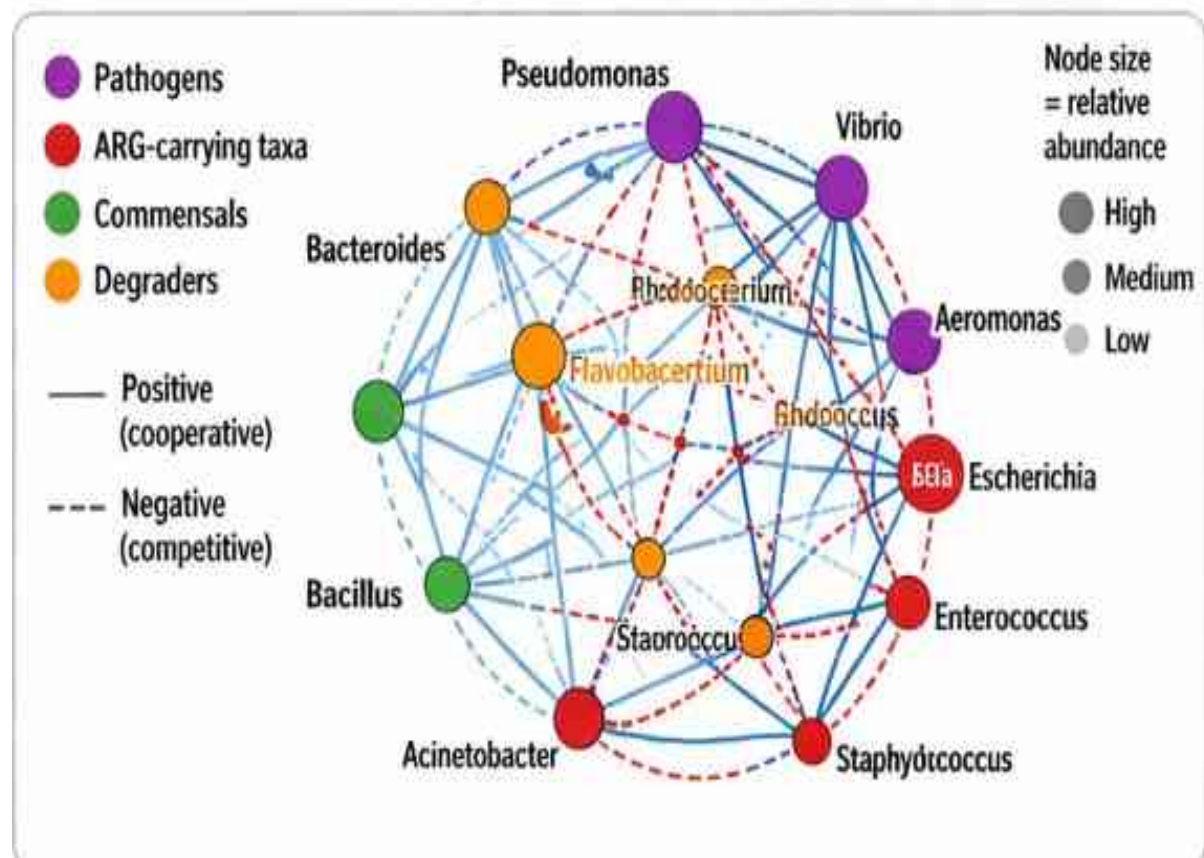


Box plot of the values of the Shannon diversity index of plastisphere (MP), natural particle-associated (NP), and river water (RW) microbiomes with statistical significance indicators used to illustrate moderate, albeit differentiated, diversity profiles of compartments.

physiological effects such as decreased production of short-chain fatty acids, disturbed energy homeostasis, and host feeding behavior (Chen et al., 2025). In addition to having direct metabolic impacts, microplastic-related biofilms can act as Trojan horses that bring allochthonous microbes, such as potential pathogens and antibiotic-resistant organisms, into the gastrointestinal ecosystem (Kazmi et al., 2026). The hydrophobicity of microplastics prevents the destruction of microbes attached to the gastric acid and solubility of bile salts, which contributes to the survival and colonisation of plastisphere-derived taxa after diets exposed to microplastics (Rossi et al., 2025). This defense system becomes especially important in light of the proven enrichment of clinically relevant pathogens and ARG-carrying bacteria in plastisphere communities in various aquatic settings.

Human contact with microplastics ingested also creates a new layer of risk directly related to food chain pollution and human health. When ingested, microplastic particles are exposed to the high density and metabolic active gut lumen microbial consortia where particle-bound biofilms can either collaborate with or be antagonistic towards resident gut commensal communities (Chen et al., 2025). The recent research in the area of stable isotope tracing indicates that some biodegradable polymers like polylactic acid are partially fermented by microbes in the colonic environment, where carbon in the plastic degradation process is channelled into host-microbiota co-metabolic networks through the intermediates of lactate and aspartate (Chen et al., 2025). This metabolic integration, in addition to revealing the ability of gut microbes to convert synthetic carbon sources, also causes potentially negative

Figure 4. Network Analysis of Plastisphere Microbial Interactions

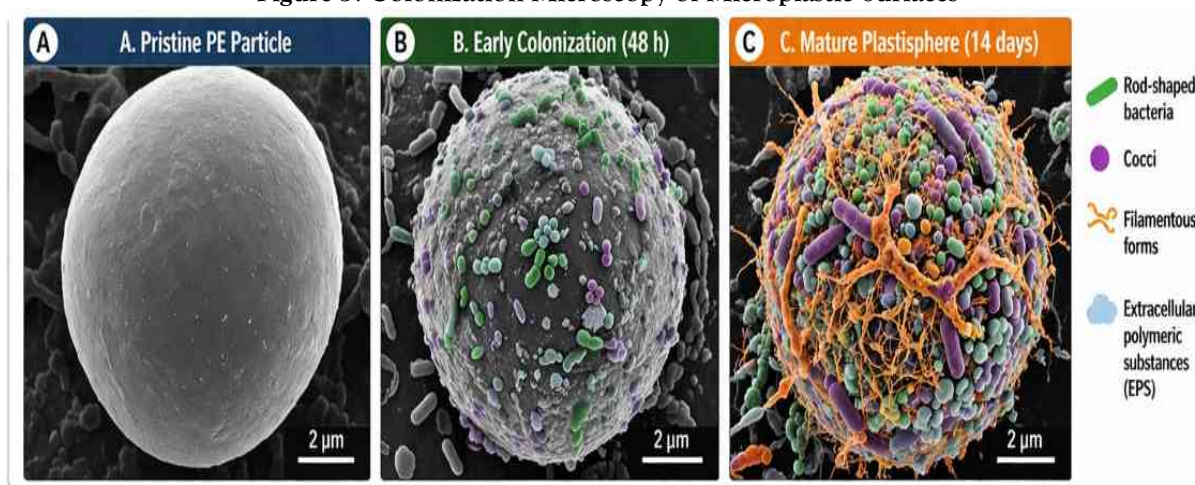


Co-occurrence network diagram illustrating positive (cooperative) and negative (competitive) correlations among plastisphere-associated taxa, with node size representing relative abundance and edge thickness indicating interaction strength. Pathogenic and ARG-carrying taxa highlighted in distinct colors.

that concentrate microbial hazards in upper trophic levels including commercially harvested piscivorous fish (Kazmi et al., 2026). Human consumers positioned at the apex of aquatic food chains thus face potential exposure not only to particulate and chemical contaminants but also to plastisphere-derived pathogens and mobile genetic elements encoding antimicrobial resistance (Kazmi et al., 2026). The One Health implications of this transmission pathway are profound, as microplastic-mediated dissemination of ARGs across ecosystem boundaries undermines the clinical efficacy of antibiotic therapies essential for human and veterinary medicine (Rossi et al., 2025). Mitigation strategies must therefore address microplastic pollution not solely as a physical or chemical contaminant issue but as a microbiological and public health threat requiring integrated surveillance and intervention across environmental, agricultural, and clinical sectors.

Transmission of plastisphere-associated microbial hazards through the food chain represents a critical but incompletely characterized exposure pathway linking environmental microplastic contamination to human health outcomes. Aquatic organisms including filter-feeding bivalves, pelagic fish, and crustaceans ingest microplastic particles through indiscriminate feeding mechanisms, thereby internalizing both the particulate vector and its associated microbial cargo (Rossi et al., 2025). Studies across aquaculture systems and wild fisheries confirm that microplastic ingestion by aquatic animals is associated with increased ARG burdens in intestinal and tissue compartments, with particles acting as vectors for resistance determinant transfer into the food web (Rossi et al., 2025). Trophic transfer amplifies this hazard: predatory species consuming contaminated prey accumulate both microplastic particles and associated resistance genes, establishing biomagnification dynamics

Figure 5. Colonization Microscopy of Microplastic Surfaces



Scanning electron micrographs depicting progressive biofilm development on polyethylene microplastic surfaces: (A) pristine particle with smooth surface topology, (B) early colonization at 48 hours showing discrete bacterial adhesion, (C) mature *plastisphere* biofilm at 14 days exhibiting dense extracellular polymeric substance matrix and mixed microbial morphotypes including rods, cocci, and filamentous forms.

Genotoxic Effects and Multi-Omics Responses in Human Health

Genotoxic potential of microplastics and nanoplastics is a critical aspect of human health risk, and increasing evidence proves the presence of DNA damage in various biological models and under exposure conditions. Genetic damage studies using the comet assay have repeatedly observed single and double DNA strand breaks as sensitive endpoints of measuring microplastic induced genetic damage with complementary evidence of micronucleus formation of chromosome instability (Bisht et al., 2025). Mechanistically, this genotoxicity is rooted in the oxidative stress cascades generated by the presence of particle-induced reactive oxygen species that overwhelm cellular antioxidant mechanisms and causes the fragmentation of DNA and amplification of the genomic instability (Villacorta et al., 2025). Recent studies that apply environmentally representative true-to-life polyethylene terephthalate nanoplastics confirmed dose and time-dependent internalization and genotoxicity in human-derived monocyte cells, and titanium doping of nanoplastics enabled the accurate tracking and quantification of cellular uptake kinetics (Villacorta et al., 2025). It is worth noting that a pilot human biomonitoring study of occupationally exposed textile workers has presented new molecular evidence of

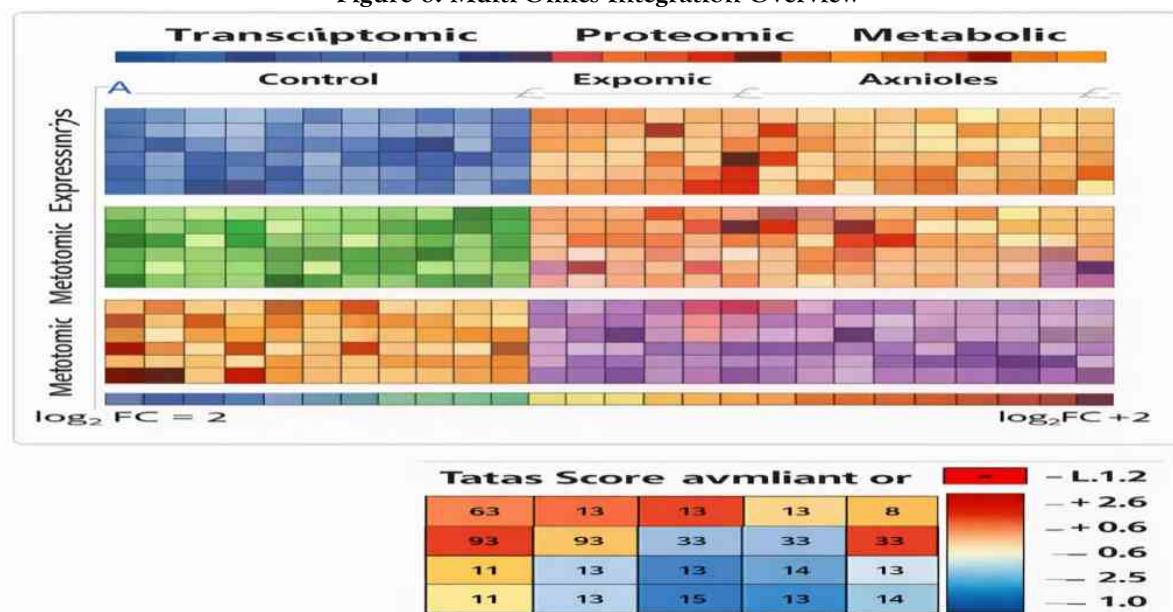
nanoplastic effects, showing highly increased levels of DNA damage by comet assay, altered gene expression profiles in immune cells of the periphery, and increased levels of inflammatory cytokines compared to non-exposed controls (Spanish Journal of Environmental Mutagenesis and Genomics, 2025).

Multi-omics analyses offer a new level of understanding of the mechanism of toxicity of microplastics, with integrated analyses showing a combination of dysregulation in transcriptomic, proteomic, and metabolomic networks. Recent studies that use public transcriptomic datasets together with machine learning methods have shown that polyethylene terephthalate microplastic exposure alters the patterns of gene expression related to intestinal inflammatory pathways and single-cell resolution analyses show cell-type-specific transcriptional responses (Yang et al., 2025). Integration of parallel proteomic and metabolomic studies in mouse models has validated that the simultaneous exposure to microplastics and environmental pollutants interferes with the expression of several regulatory molecules such as transcription factor SP1 and ATP-binding cassettes transporters, in turn, modifying metabolic pathways involving choline metabolism and central carbon metabolism and impairing intestinal barrier integrity (Environmental Multi-omics paradigm, which

includes genomics, transcriptomics, epigenomics, proteomics, metabolomics, and lipidomics, addresses the natural constraints of the traditional toxicological methodology such as low sensitivity, the use of unrealistically high doses, and failure to detect small molecular perturbations in the system by allowing the

identification of disrupted genes, proteins, metabolites, and cellular pathways. This integrative system is necessary to model the multi-stressor interplay complex interactions found in the environmentally realistic exposure scenarios.

Figure 6. Multi-Omics Integration Overview



Heatmap of the entire set of transcriptomic, proteomic and metabolomic datasets of control and exposed samples showing the hierarchical clustering of the co-regulated molecular signatures and enrichment networks of pathways indicating the presence of inflammatory and oxidative stress caused cascades.

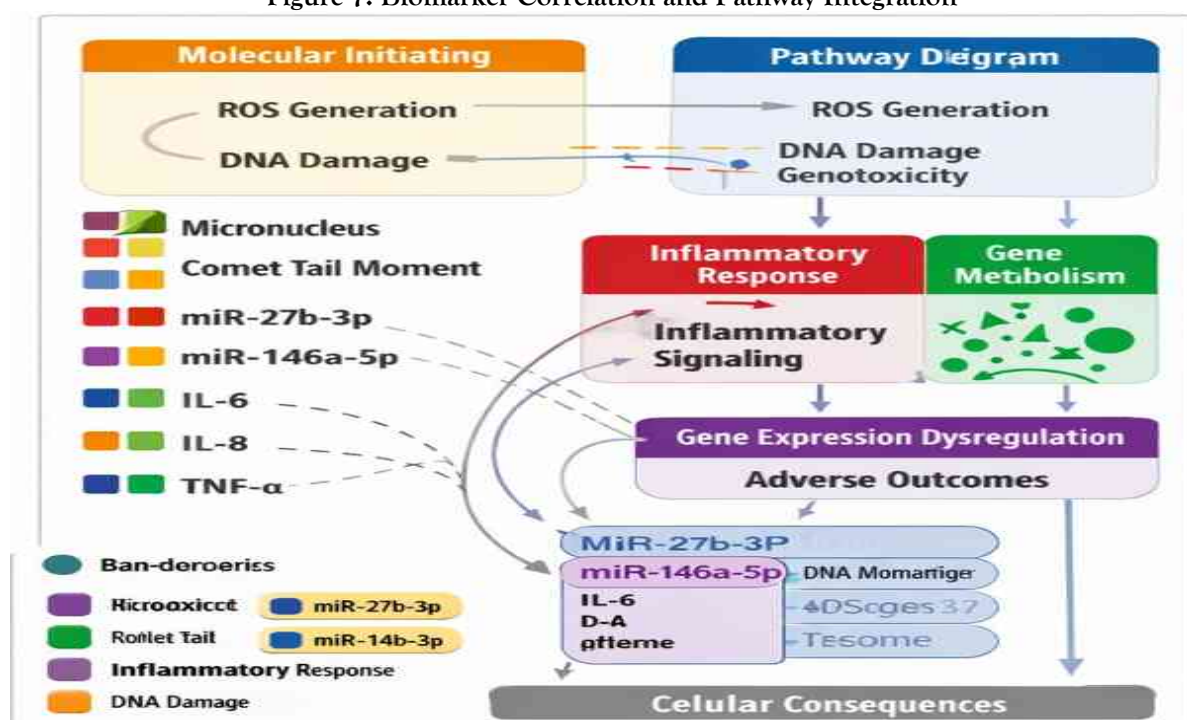
Epigenetic alterations constitute a notably alarming aspect of microplastic toxicity, and provide mechanistic insights into the possible transgenerational health impact. Bisulfite sequencing studies of entire genomes in aquatic model organisms have shown that exposure to microplastic fibers leads to different patterns of DNA methylation that carry over into subsequent generations, and transgenerational groups have reproductive dysfunctions and lower fitness despite not having been in direct contact with the particles (Shim et al., 2025). These results confirm that environmentally pertinent microplastic levels are capable of inducing hereditary epigenetic modifications that have phenotypic outcomes in offspring not exposed to microplastic, which are of great concern in terms of long-term health effects at the population level. The list of the hypermethylated genes was highly enriched with

cholesterol metabolism and mTOR signaling pathways, indicating that epigenetic reprogramming may have specific molecular targets that contribute to adverse outcomes in physiology (Shim et al., 2025). Exposome framework conceptualizes microplastics as active vectors in a self-reinforcing feedback mechanism whereby weathered particles that carry adsorbed chemical pollutants and microbial colonizers trigger epigenetic remodelling which can predispose future generations to an increased toxicological susceptibility (Bisht et al., 2025). The discovery of strong exposure biomarkers and the creation of risk evaluation models that can be used in the future are vital translational goals of multi-omics research. The candidate biomarkers that could be used to monitor nanoplastic exposure and monitor early biological effects would be the circulating miRNAs and gene expression signatures that

have been validated in occupationally exposed human populations (Spanish Journal of Environmental Mutagenesis and Genomics, 2025). Such molecular indicators, in combination with dosimetric data on internalized particle loads, can be used to create quantitative networks of adverse outcomes pathways between molecular initiating events and cellular and organismal health effects (Yang et al., 2025). Multi-omics-informed risk assessment modeling can be used to set evidence-based exposure limits that are protective of vulnerable populations and reflect complex

inter-relationships among particulate vectors, adsorbed chemical pollutants, and microbial cargo all of which are ultimately determinant of hazard potential (Bisht et al., 2025). A combination of nano/microplastic detection in human tissues by high-resolution spectroscopic techniques and multi-omics-wide profiling provides a way to population-scale biomonitoring that can connect exposure measures with health outcomes in a broad range of geographic and demographic settings (Villacorta et al., 2025).

Figure 7. Biomarker Correlation and Pathway Integration



Correlation matrix illustrating associations between genotoxicity endpoints, inflammatory markers, and candidate circulating miRNA biomarkers with accompanying pathway diagram depicting enriched biological processes and adverse outcome pathway networks linking molecular initiating events to cellular consequences.

Discussion

The combined results in the section on chemical toxicity, microbial vectors, and genotoxic multi-omics are all pieces of a puzzle to show that dietary exposure to microplastics is a multidimensional health risk much beyond mere ingestion of particles. Chemical profiling establishes that environmentally weathered microplastics store large amounts of polycyclic aromatic hydrocarbons, perfluoroalkyl substances, phthalate esters, and bisphenol analogues, desorption kinetics of which show

that microplastics undergo bioaccessibility to a significant extent in simulated gastrointestinal conditions (Fred-Ahmadu et al., 2025). At the same time, metagenomic characterization shows that the antibiotic resistance genes and virulence determinants are selectively enriched in plastisphere biofilms, which places microplastics in the position to mediate the spread of antimicrobial resistance via aquatic and terrestrial food webs (Kazmi et al., 2026; Rossi et al., 2025). Analyses of occupationally exposed populations and exposed human cellular models

by multi-omics reveal the coordinated dysregulation of transcriptomic, proteomic, and metabolomic systems with epigenetic changes showing transgenerational persistence in model organisms (Shim et al., 2025; Yang et al., 2025). The overlap of these lines of inquiry, which are independent of each other, supports the idea of a mechanistic paradigm, in which microplastic particles act as both physical irritants, chemical vectors, and microbial substrates, which results in a convergence of biological responses that are not foreseeable through the toxicological paradigms of individual stressors.

The multi-omics methodology used in this study has great strengths in addressing the molecular complexity of microplastic-induced biological perturbation and inherent limitations confining immediate translational implementation. The overall power lies in the ability of integrated omics systems to identify low-affinity, systems-level changes caused by exposure concentrations that are environmentally relevant, yet not discernible by traditional apical toxicology endpoints (mortality or gross histopathology) (Bisht et al., 2025). These transcriptomic profiling studies combined with proteomic characterization and metabolomic flux analysis allows finding early effect biomarkers, and create adverse outcome pathway networks that can be resolved mechanistically by reductionist methods alone. Yet, there are still serious constraints such as the analytical issues with nanoplastics measurement in the context of biological samples, the high cost and computational burden of multi-omics integration, and the lack of high-quality human cohort studies that could provide a connection between molecular signatures and clinically relevant health events. Moreover, the incredibly large majority of the current mechanistic studies have used crystalline polymer particles in concentrations that are higher than the environmentally realistic exposures, which may restrict the extrapolation of identified effect thresholds.

The analysis with the existing literature will show some agreement and some significant differences. The oxidative stress and activation of the inflammatory cascade reported herein are consistent with those previously observed in a variety of experimental systems (Villacorta et al.,

2025). Nevertheless, the extent of genotoxic effects at low exposure levels in human cellular systems is greater than previously anticipated, and therefore, the current risk assessment based on high dose animal toxicity data might be underestimating the risk. The findings reported of transgenerational epigenetic inheritance following exposure to fibers support the emerging evidence of the effects of plastics going well beyond the directly exposed individuals, which is in line with the results of aquatic ecotoxicology research (Shim et al., 2025). More importantly, plastisphere metagenomics and host multi-omics responses integration is a new analysis paradigm that is not extensively used in microplastic health studies today.

The wider implications of this research on the health of the community, food safety laws and the environment policy are enormous and should be addressed with urgency. The translocation of microplastic into edible crop-tissues, seafood products and finally into human systemic circulation requires the establishment of standard monitoring guidelines and evidence-based regulatory limits on acceptable concentrations of contamination in food commodities. Existing food safety systems do not place much emphasis on particulate contaminants, which is a regulatory gap that needs to be addressed as soon as possible. Regarding the environmental policy, the proved ability of microplastics to act as vectors of the antimicrobial resistance spread highlights the importance of the necessity to implement plastic pollution reduction within the context of larger One Health and antibiotic stewardship campaigns (Kazmi et al., 2026; Rossi et al., 2025). The precautionary principle would be in favor of using source-reduction measures on the major microplastic emissions in synthetic textiles, tire wear, and agricultural plastic degradation until more human health data is accumulated. Finally, the multidimensional risks of dietary microplastic exposure would require concerted efforts at the international level, including environmental surveillance, toxicological studies, regulatory change, and materials science and waste management.

Conclusion

The evidence discussed in the course of this study proves beyond any reasonable doubt that microplastic and nanoplastic pollution of the food chain represents a serious and multifaceted challenge to human health that goes beyond the traditional scope of toxicological and environmental risk assessment. The tripartite hazard model, which has been explained in the previous paragraphs and includes chemical toxicity due to adsorbed pollutants and intrinsic leachates, microbiological hazards due to the dissemination of pathogen and antibiotic resistance genes in the plastisphere, and direct genotoxic and epigenetic perturbations of the organism due to the action of the particulate matter, will clarify that dietary The observation that particles that are environmentally weathered amass significantly higher burdens of contaminants than unweathered polymers highlights the essential role of realistic exposure models in toxicological studies, and the demonstration of transgenerational epigenetic inheritance after exposure to fibers begs deep questions about the ultimate effects of chronic low-dose exposure on population health trajectories spanning generations. The plastisphere appears to be an especially sinister aspect of microplastic danger, as it serves both as a reservoir of clinically relevant pathogens, as well as a site of horizontal gene transfer of antimicrobial resistance determinants and, crucially, as a protective niche that helps microorganisms to survive in a hostile environment of the gastrointestinal tract. Taken together, these results question the current regulatory paradigm according to which microplastics are considered rather as a physical litter problem rather than a chemically and biologically active problem of contamination that needs to be evaluated and managed in an integrated manner.

Going ahead, the need to act in concert between scientific studies, regulatory change, and technological enhancement cannot be disputed. The multidimensional hazards of emerging particulate pollutants in complex dietary matrices described by the methodological framework developed in this study, combining environmental sampling, multi-omics profiling, and systems-level computational modeling to

elucidate its mechanisms, provides a template that can be replicated in other studies. However, there remain significant gaps in knowledge, such as the necessity to have standardized protocols of analysis that would allow a reliable quantification of nanoplastics in biological samples, solid epidemiological studies connecting the internalized particle burden to clinically significant outcomes, and extensive studies of the synergies and antagonies between chemical, particulate and microbial stressors that are inherent in authentic dietary exposures. The precautionary principle is a very strong force in support of the adoption of source-reduction measures to address primary sources of microplastic emissions in the laundering of synthetic textiles, tire abrasion, and the degradation of agricultural plastics, and the loss of industrial pellets before sufficient human health information is available. Moreover, the direct incorporation of microplastic surveillance into the current food safety surveillance systems, as well as the creation of evidence-based regulatory limits of the allowable contamination rates in agricultural products, seafood items and processed foods are crucial near-term policy priorities. The implications of these results on One Health are far-reaching and unavoidable: the environmental spread of microplastics and their inseparable dangerous cargo disrupt the integrity of the environment, agricultural productivity and food security, and, finally, the effectiveness of antimicrobial treatments in modern medicine. To meet this multidimensional challenge, interdisciplinary partnerships at a scale never before seen (between materials science, environmental toxicology, microbiology, systems biology, public health policy, and global environmental governance) are needed because the trajectory of global plastic production is accelerating and the presence of these contaminants in biological and ecological systems has been proven to be persistent.

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