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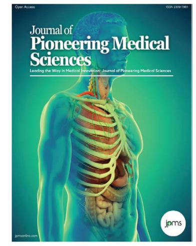

Microplastics in the Human Food Chain: Emerging Public Health Challenges, Biological Mechanisms, Health Consequences and Future Directions

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Abstract Microplastics have emerged as pervasive environmental contaminants due to the exponential growth in plastic production, inadequate waste management and their persistence in terrestrial, freshwater, marine and atmospheric ecosystems. Their widespread presence in seafood, drinking water, fruits, vegetables, meat, dairy products, processed foods and food packaging has made dietary ingestion a major route of human exposure, raising significant public health concerns. This review summarizes current evidence on microplastic contamination of the human food chain, exposure pathways, biological fate, mechanisms of toxicity, health effects and future research priorities. Experimental studies indicate that microplastics induce oxidative stress, chronic inflammation, immune dysregulation, mitochondrial dysfunction, endocrine disruption, gut microbiome dysbiosis and cellular injury, while also serving as carriers for heavy metals, persistent organic pollutants and pathogenic microorganisms. The detection of microplastics in human blood, placenta, lungs, breast milk, reproductive tissues and arterial plaques confirms systemic exposure and underscores the need for comprehensive health risk assessment. Emerging evidence suggests possible associations with gastrointestinal, cardiovascular, respiratory, metabolic, reproductive, neurological, hepatic and renal disorders; however, definitive causal relationships remain unconfirmed because of limited human epidemiological evidence and methodological heterogeneity. Given the ubiquitous nature of exposure, a precautionary public health approach is warranted. Future efforts should prioritize standardized analytical methods, long-term epidemiological studies, strengthened food safety surveillance, sustainable plastic management and multidisciplinary research within a One Health framework to better characterize health risks, inform evidence-based policies and reduce human exposure to this growing environmental threat.

Key Words Microplastics, Human Food Chain, Public Health, Food Safety, One Health

INTRODUCTION

Plastics have become indispensable in modern society because of their durability, versatility and low cost. However, the exponential increase in global plastic production, now exceeding 400 million tonnes annually, coupled with inadequate waste management and the extensive use of single-use plastics, has resulted in widespread environmental contamination. Instead of completely degrading, discarded plastics fragment into microplastics (<5mm) and nano-plastics, which persist in terrestrial, freshwater, marine and atmospheric ecosystems. Their small size, environmental persistence and mobility enable them to infiltrate ecological food webs and ultimately enter the human food chain [1,2].

Dietary ingestion is recognized as the primary route of human exposure to microplastics. These particles have been detected in seafood, drinking water, fruits, vegetables, meat, dairy products, table salt and processed foods, while food packaging, processing and storage further contribute to contamination. Inhalation of airborne particles and occupational exposure provide additional exposure pathways, making microplastics an almost unavoidable component of daily life [3,4].

Recent detection of microplastics in human blood, placenta, breast milk, lungs, liver, reproductive tissues and arterial plaques have raised growing concerns regarding their potential health effects. Experimental evidence suggests that microplastics can induce oxidative stress, chronic inflammation, immune dysregulation, mitochondrial dysfunction, endocrine disruption and gut microbiome dysbiosis. Moreover, they may act as vectors for heavy metals, persistent organic pollutants, pharmaceutical residues and pathogenic microorganisms, potentially amplifying their biological toxicity. However, despite increasing experimental evidence, definitive causal relationships with human diseases remain unestablished because of limited epidemiological studies, methodological heterogeneity and uncertainties regarding long-term exposure [5,6].

From a public health perspective, microplastics represent an emerging challenge at the interface of environmental health, food safety, toxicology and preventive medicine. This review critically synthesizes current evidence on microplastic contamination of the human food chain, exposure pathways, biological fate, mechanisms of toxicity, health implications and future research priorities. By adopting a One Health perspective, it aims to provide an updated understanding of this emerging environmental threat and identify strategies to reduce human exposure while strengthening food safety and protecting population health.

Sources of Microplastics and their Entry into the Human Food Chain

Microplastics have become ubiquitous environmental contaminants because of their persistence, mobility and resistance to degradation. They are widely distributed across terrestrial, freshwater, marine and atmospheric ecosystems, enabling continuous movement through air, water, soil, living organisms and ultimately the human food chain. Understanding their sources and pathways is essential for assessing human exposure and developing effective public health interventions.

Definition and Classification

Microplastics are synthetic polymer particles measuring less than 5mm in diameter, while particles smaller than 1μm are commonly referred to as nano-plastics. Based on their origin, microplastics are classified as primary or secondary. Primary microplastics are intentionally manufactured for industrial and commercial applications, including resin pellets, industrial abrasives, synthetic textile fibres, paints, coatings and personal care products. Secondary microplastics are formed through the fragmentation of larger plastic items under the influence of ultraviolet radiation, mechanical abrasion and environmental weathering. They occur as fibres, fragments, films, foams and beads, with their size, shape, polymer composition and surface characteristics influencing their environmental behaviour, bioavailability and toxicity. Weathered microplastics also exhibit an increased capacity to adsorb heavy metals, persistent organic pollutants and microorganisms, potentially enhancing their biological effects [1,7].

Major Environmental Sources

The widespread occurrence of microplastics reflects the extensive production, use and improper disposal of plastic materials. Major environmental sources include single-use plastic packaging, synthetic textiles, tire wear particles, industrial emissions, agricultural plastics, wastewater and sewage sludge. Plastic bottles, food containers, shopping bags and packaging materials gradually fragment into secondary microplastics after entering the environment. Synthetic fabrics release microscopic fibres during washing, many of which escape wastewater treatment and contaminate aquatic ecosystems. Similarly, tire abrasion and road dust contribute substantially to environmental microplastic pollution, while agricultural practices involving plastic mulch films, greenhouse covers, irrigation systems and sludge application introduce microplastics into cultivated soils. Industrial processes, including plastic manufacturing and transportation of resin pellets, represent additional sources. Furthermore, atmospheric transport enables long-distance dispersal of airborne microplastics, explaining their presence even in remote mountain and polar regions [8,9].

Entry into the Human Food Chain

Microplastics enter the human food chain through multiple interconnected pathways. Marine and freshwater organisms, particularly fish, shellfish, mussels, oysters and shrimp, ingest suspended microplastics directly or indirectly through contaminated prey, making seafood one of the most important dietary sources of exposure. Agricultural contamination occurs through sewage sludge application, contaminated irrigation water, degradation of agricultural plastics and atmospheric deposition, leading to the presence of microplastics in fruits, vegetables, cereals and other crops. Animal-derived foods such as meat, milk, eggs and honey may also become contaminated through polluted feed, water or processing environments [1,10].

Drinking water represents another important exposure source, with microplastics consistently detected in both bottled and tap water. Food processing, packaging, transportation and storage further contribute to contamination through the release of particles from plastic cutting boards, conveyor belts, disposable utensils, tea bags, food containers and packaging materials, particularly during heating or repeated use. Consequently, microplastics have become deeply integrated into modern food production and consumption systems, making chronic dietary exposure virtually unavoidable. This widespread contamination highlights the need for standardized monitoring, improved food safety surveillance, sustainable plastic management and preventive strategies to reduce long-term human exposure.

Human Exposure and Biological Fate of Microplastics

Microplastics have become an unavoidable component of human exposure because of their widespread occurrence in food, drinking water, air and consumer products. While dietary ingestion is considered the primary exposure route, inhalation of airborne particles and, to a lesser extent, dermal contact also contribute to lifetime exposure. Their biological fate depends on particle size, shape, polymer composition, surface chemistry and associated contaminants, which collectively influence their absorption, distribution, persistence and elimination.

Human Exposure Pathways

Dietary ingestion represents the major source of microplastic exposure. Microplastics have been detected in seafood, freshwater fish, shellfish, drinking water, table salt, fruits, vegetables, cereals, meat, dairy products, honey, tea and processed foods. Food packaging, storage, processing and heating in plastic containers further increase contamination. Although seafood has traditionally been regarded as the primary dietary source, increasing evidence indicates that terrestrial foods and drinking water also contribute substantially to overall exposure [11,12].

Inhalation is another important pathway, particularly in indoor environments where synthetic fibres released from textiles, carpets, furniture and household dust accumulate. Outdoor exposure occurs through tire wear particles, industrial emissions, road dust and degraded plastic waste. Fine airborne particles can penetrate deep into the respiratory tract, while occupational exposure may be considerably higher among workers in textile, plastic manufacturing, recycling and waste management industries [13,14].

Compared with ingestion and inhalation, dermal exposure appears to contribute minimally. Nevertheless, contact with cosmetics, synthetic textiles, medical devices and contaminated water may result in limited skin exposure, particularly when the skin barrier is compromised.

Absorption, Distribution and Bioaccumulation

Following ingestion, most microplastics are excreted through the gastrointestinal tract. However, smaller microplastics and nano-plastics may cross the intestinal barrier through endocytosis or paracellular transport, entering the bloodstream or lymphatic system. Particle uptake may be enhanced when intestinal permeability is increased due to inflammation or gastrointestinal disease.

Recent studies have identified microplastics in human blood, placenta, breast milk, lungs, liver, kidneys, reproductive tissues and atherosclerotic plaques, suggesting systemic distribution after absorption. Their accumulation appears to depend on particle size, polymer type, surface properties and interactions with circulating biomolecules. Experimental studies indicate that prolonged low-dose exposure may result in gradual bioaccumulation in organs such as the liver, lungs, kidneys, brain and reproductive tissues. However, the extent and clinical significance of tissue accumulation in humans remain uncertain [15,16].

Elimination and Current Knowledge Gaps

Faecal excretion is considered the primary route of microplastic elimination, indicating that most ingested particles are not systemically absorbed. Smaller particles entering the circulation may be cleared through hepatobiliary or renal pathways or by immune-mediated phagocytosis, although these mechanisms remain poorly understood. The lack of standardized biomarkers and harmonized analytical methods currently limits accurate assessment of internal microplastic burden [17,18].

Despite growing evidence of systemic exposure, important uncertainties remain regarding absorption efficiency, long-term tissue persistence, dose-response relationships and health effects under environmentally relevant conditions. Therefore, standardized biomonitoring, improved analytical techniques and large prospective human studies are essential for accurately characterizing the toxicokinetics and public health implications of chronic microplastic exposure.

Biological Mechanisms of Microplastic Toxicity

Although microplastics have been detected in environmental matrices, food products and human tissues, their health effects remain incompletely understood. Current evidence, derived primarily from *in vitro*, animal and limited human studies, suggests that microplastics exert toxicity through multiple interconnected biological pathways rather than a single mechanism. Their toxic potential depends on particle size, shape, polymer composition, surface chemistry, degree of weathering and associated contaminants (Table 1).

Table 1: Major biological mechanisms underlying microplastic toxicity and their potential health implications

Biological mechanism	Principal molecular events	Major target organs	Potential health outcomes
Oxidative stress	Excess ROS generation, antioxidant depletion	Multiple organs	Cellular injury, aging, chronic diseases
Chronic inflammation	Cytokine release, NF-κB activation	Gut, lungs, cardiovascular system	Chronic inflammatory diseases
Immune dysregulation	Altered macrophage and lymphocyte function	Immune system	Infection susceptibility, immune imbalance
Mitochondrial dysfunction	Reduced ATP production, apoptosis	Liver, kidney, brain	Organ dysfunction
Endocrine disruption	Hormone receptor interference, chemical leaching	Endocrine and reproductive organs	Infertility, metabolic disorders
Gut microbiome dysbiosis	Altered microbial diversity, impaired gut barrier	Gastrointestinal tract	Dysbiosis, inflammatory bowel disease
Genotoxicity and epigenetic changes	DNA damage, altered gene expression	Multiple tissues	Carcinogenesis, developmental effects
Carrier effect	Transport of heavy metals, POPs, pathogens	Multiple organs	Enhanced toxic exposure

Table 2: Current evidence linking microplastic exposure with organ-specific health effects

Organ system	Principal biological mechanisms	Reported experimental findings	Current human evidence	Overall certainty
Gastrointestinal	Dysbiosis, inflammation, increased permeability	Gut barrier disruption, IBD-like changes	Limited	Moderate
Cardiovascular	Oxidative stress, endothelial dysfunction	Atherosclerosis, vascular inflammation	Emerging	Low-Moderate
Respiratory	Inhalation, chronic inflammation	Fibrosis, airway injury	Limited	Low
Endocrine & Metabolic	Hormonal disruption, oxidative stress	Insulin resistance, obesity	Limited	Low
Reproductive	Endocrine disruption, oxidative stress	Reduced fertility, placental transfer	Emerging	Low-Moderate
Neurological	Neuroinflammation, mitochondrial dysfunction	Cognitive impairment, behavioural changes	Very limited	Low
Liver & Kidney	Oxidative injury, apoptosis	Hepatic steatosis, renal injury	Limited	Low
Cancer	DNA damage, chronic inflammation, contaminant transport	Carcinogenic mechanisms demonstrated experimentally	No causal human evidence	Very Low

Oxidative Stress and Inflammation

Oxidative stress is considered the primary mechanism underlying microplastic toxicity. Following cellular uptake, microplastics stimulate excessive production of reactive oxygen species (ROS), overwhelming antioxidant defence systems and resulting in lipid peroxidation, protein oxidation, DNA damage and mitochondrial injury. Persistent oxidative stress subsequently activates inflammatory signalling pathways, particularly NF-κB and MAPK, leading to increased production of pro-inflammatory cytokines, including IL-1β, IL-6, IL-8 and TNF-α. Chronic low-grade inflammation and immune dysregulation may contribute to the development of cardiovascular, metabolic, respiratory and gastrointestinal disorders, although evidence in humans remains limited [19-21].

Cellular Dysfunction, Genotoxicity and Endocrine Disruption

Microplastics can disrupt normal cellular function by impairing mitochondrial activity, reducing ATP production, altering intracellular calcium homeostasis and inducing apoptosis. Smaller particles, particularly nano-plastics, exhibit greater cellular uptake and may therefore possess higher toxic potential. Experimental studies have also demonstrated oxidative DNA damage, chromosomal abnormalities and epigenetic alterations, including changes in DNA methylation and microRNA expression, suggesting possible long-term effects on gene regulation and disease susceptibility [22,23].

In addition to their physical effects, microplastics serve as carriers of endocrine-disrupting chemicals such as bisphenols and phthalates, which interfere with estrogenic, androgenic and thyroid hormone signalling. These interactions have been associated with impaired reproductive function, metabolic disorders and developmental abnormalities in experimental models.

Gut Microbiome Alterations and Carrier Effect

As the gastrointestinal tract is the primary site of exposure, chronic ingestion of microplastics may alter gut microbial composition, reduce beneficial bacteria, impair intestinal barrier integrity and promote chronic intestinal inflammation. Such gut microbiome dysbiosis has been linked to metabolic disorders, inflammatory bowel disease and immune dysfunction, although evidence in humans remains limited.

Microplastics also act as environmental vectors because of their large surface area and hydrophobic properties. They readily adsorb heavy metals, persistent organic pollutants (POPs), polycyclic aromatic hydrocarbons (PAHs), pharmaceutical residues and pathogenic microorganisms, facilitating their transport into biological systems. Furthermore, microbial biofilms ("plastisphere") formed on microplastic surfaces may harbour pathogenic and antibiotic-resistant microorganisms, potentially amplifying toxic effects.

Overall, current evidence supports the biological plausibility that microplastics contribute to adverse health effects through oxidative stress, chronic inflammation, mitochondrial dysfunction, endocrine disruption, gut microbiome alterations and contaminant transport. However, most mechanistic studies rely on high-dose experimental models that may not accurately represent environmental exposure. Therefore, standardized toxicological methods, environmentally relevant exposure models and well-designed human studies are needed to establish dose-response relationships and clarify the clinical significance of chronic microplastic exposure [24,25].

Human Health Consequences of Microplastic Exposure

The widespread detection of microplastics in human tissues has raised growing concerns regarding their potential health effects (Table 2). Experimental studies indicate that chronic exposure may affect multiple organ systems through oxidative stress, inflammation, endocrine disruption, mitochondrial dysfunction and immune dysregulation. However, it is important to distinguish biological plausibility from clinical evidence. Most available data originate from laboratory and animal studies, while human epidemiological evidence remains limited. Consequently, although microplastics have been associated with several adverse biological effects, definitive causal relationships with human diseases have not yet been established [26-28].

Gastrointestinal Health

As the gastrointestinal tract is the primary site of exposure, it is considered one of the most vulnerable organs. Experimental studies suggest that ingested microplastics may disrupt intestinal barrier integrity, alter gut microbiota composition and promote chronic intestinal inflammation. These changes have been linked to increased intestinal permeability, inflammatory bowel disease, metabolic dysfunction and impaired nutrient absorption. Although microplastics have been detected in human faeces and intestinal tissues, direct evidence linking them to gastrointestinal diseases remains limited.

Cardiovascular and Respiratory Health

Recent detection of microplastics in human atherosclerotic plaques has raised concerns regarding cardiovascular toxicity. Experimental evidence indicates that microplastics may induce endothelial dysfunction, vascular inflammation, oxidative stress and platelet activation, processes associated with atherosclerosis and thrombosis. However, whether microplastics directly contribute to cardiovascular disease or simply accumulate in diseased tissues remains unclear.

Inhalation represents another important exposure route. Airborne microplastics can deposit throughout the respiratory tract, particularly in occupational settings. Experimental and occupational studies suggest that chronic exposure may contribute to airway inflammation, pulmonary fibrosis, reduced lung function and respiratory irritation. Nevertheless, epidemiological evidence linking environmental microplastic exposure to asthma, chronic obstructive pulmonary disease (COPD) or lung cancer remains insufficient.

Endocrine, Metabolic and Reproductive Health

Microplastics and their associated additives may interfere with endocrine signalling, particularly through chemicals such as bisphenols and phthalates. Experimental studies have linked exposure to insulin resistance, obesity, altered thyroid function, dyslipidaemia and metabolic syndrome, although evidence in humans is limited.

The reproductive system appears particularly susceptible because of endocrine disruption and oxidative stress. Animal studies have demonstrated impaired spermatogenesis, reduced sperm quality, ovarian dysfunction, altered hormone production and decreased fertility. The detection of microplastics in the placenta, breast milk, amniotic fluid and reproductive tissues has raised concerns regarding maternal foetal transfer and developmental toxicity. However, the clinical significance of these findings requires confirmation through long-term human studies.

Neurological, Hepatic and Renal Effects

Experimental evidence suggests that nano-plastics may cross the blood-brain barrier, inducing neuroinflammation, mitochondrial dysfunction and cognitive impairment. Alterations in the gut-brain axis have also been proposed as an indirect mechanism of neurotoxicity. However, convincing evidence in humans remains unavailable.

The liver and kidneys, as major organs involved in metabolism and excretion, are also potential targets. Animal studies have reported hepatic oxidative stress, lipid accumulation, inflammation and renal tubular injury following chronic exposure. Nevertheless, there is currently no conclusive evidence linking environmental microplastic exposure to clinically significant liver or kidney disease in humans.

Carcinogenic Potential

Whether microplastics contribute to carcinogenesis remains uncertain. Chronic inflammation, oxidative DNA damage, epigenetic alterations and endocrine disruption provide biologically plausible mechanisms for cancer development. In addition, microplastics can transport carcinogenic compounds such as heavy metals, polycyclic aromatic hydrocarbons and persistent organic pollutants. Despite these concerns, no robust epidemiological evidence currently demonstrates that environmental microplastic exposure directly causes cancer in humans.

Overall, current evidence indicates that microplastics may adversely affect multiple organ systems; however, most findings are based on experimental models employing exposure levels that may not accurately reflect real-world conditions. Human studies remain limited by methodological heterogeneity, inadequate exposure assessment and the absence of long-term follow-up. Therefore, well-designed prospective cohort studies, standardized biomonitoring and environmentally relevant toxicological investigations are essential to clarify the true health burden of chronic microplastic exposure and support evidence-based public health policies.

Public Health Implications

The pervasive presence of microplastics in food systems, drinking water, air and human tissues has transformed plastic pollution into an emerging public health concern. Unlike many environmental contaminants, microplastic exposure is continuous, lifelong and nearly universal. Although current evidence is insufficient to establish definitive causal relationships with specific diseases, the widespread nature of exposure warrants a precautionary public health approach. Even modest health effects could translate into a substantial population-level burden because of the large number of exposed individuals [29,30].

Food Safety and Vulnerable Populations

The detection of microplastics in seafood, drinking water, fruits, vegetables, cereals, dairy products, meat and processed foods highlights food contamination as a major public health issue. Besides the particles themselves, microplastics may carry plastic additives, heavy metals, persistent organic pollutants and pathogenic microorganisms, complicating food safety risk assessment.

Table 3: Current knowledge gaps and future research priorities in microplastic research

Research domain	Current limitations	Future priorities	Public health relevance
Exposure assessment	Lack of standardized sampling and analytical methods	International harmonization of detection protocols	Reliable exposure estimation
Human epidemiology	Few prospective human studies	Large multicentre longitudinal cohorts	Establish causality
Toxicology	High-dose laboratory models	Chronic low-dose environmentally relevant exposure studies	Improved risk assessment
Nano-plastics	Limited detection capability	Development of sensitive analytical technologies	Better understanding of systemic toxicity
Biomonitoring	Absence of validated biomarkers	Identification of biomarkers of exposure and early biological effect	Population surveillance
Food safety	Limited routine monitoring	National surveillance of food and drinking water	Consumer protection
Mechanistic studies	Incomplete understanding of molecular pathways	Multi-omics and systems biology approaches	Identification of therapeutic targets
Policy and regulation	Lack of global exposure guidelines	International regulatory frameworks and coordinated surveillance	Evidence-based policymaking

Consequently, strengthening food safety surveillance, improving food packaging practices and reducing plastic contamination throughout the food supply chain have become important public health priorities.

Certain populations may be particularly vulnerable to microplastic exposure. Pregnant women, infants and children are of special concern because microplastics have been detected in the placenta and breast milk, suggesting potential early-life exposure during critical developmental stages. Older adults and individuals with chronic diseases may also be more susceptible because of reduced physiological resilience. In addition, workers employed in plastic manufacturing, textile production, recycling and waste management may experience higher occupational exposure, emphasizing the need for workplace monitoring and preventive measures.

Environmental Justice, One Health and Policy

Microplastic pollution also raises important concerns regarding environmental justice. Communities living near industrial areas, landfills or regions with inadequate waste management infrastructure often experience disproportionately higher exposure. Low- and middle-income countries are particularly affected because of rapid urbanization, increasing plastic consumption and limited environmental monitoring and waste management systems.

Addressing microplastic pollution requires a One Health approach that recognizes the interconnectedness of environmental, animal and human health. Reducing plastic waste, improving recycling systems, promoting sustainable packaging and adopting circular economy strategies can simultaneously protect ecosystems, strengthen food security and reduce human exposure.

Governments and regulatory agencies should prioritize standardized monitoring methods, harmonized analytical protocols, routine surveillance of food and drinking water and evidence-based exposure guidelines. At the same time, effective public communication should provide balanced, science-based information that encourages practical exposure-reduction measures without generating unnecessary public concern.

Overall, microplastics represent an emerging public health challenge requiring coordinated action across environmental management, food safety, occupational health, research and policy. Future efforts should focus on standardized biomonitoring, longitudinal epidemiological studies and multidisciplinary collaboration to generate robust evidence that supports effective public health interventions and evidence-based regulatory policies.

Future Directions and Research Priorities

Despite significant advances in microplastic research, major knowledge gaps remain regarding their long-term effects on human health. Current evidence is largely derived from experimental studies, while robust epidemiological data remain limited. Inconsistencies in sampling methods, analytical techniques, particle characterization and exposure assessment further complicate comparison among studies and hinder evidence-based risk assessment. Addressing these challenges requires coordinated multidisciplinary research integrating toxicology, epidemiology, environmental science, food safety, analytical chemistry and public health (Table 3).

Standardized Exposure Assessment and Human Studies

A major priority is the development of internationally standardized protocols for the detection, characterization and quantification of microplastics in environmental samples, food products and human tissues. Harmonized analytical methods will improve data comparability and strengthen risk assessment. Equally important is the establishment of large prospective cohort studies incorporating standardized biomarkers, repeated biological sampling, dietary assessment, occupational history and long-term clinical follow-up to clarify dose-response relationships and establish causal links between exposure and disease.

Environmentally Relevant Toxicological Research

Most experimental studies employ particle concentrations that exceed typical environmental exposure levels and often use uniform polymer types that do not reflect real-world conditions. Future research should utilize environmentally relevant concentrations, diverse polymer compositions, weathered particles and chronic low-dose exposure models. Greater attention should also be directed toward nanoplastics because of their increased potential for systemic absorption and biological interactions. Advances in multi-omics approaches, including genomics, proteomics, metabolomics and epigenomics, may further improve understanding of molecular mechanisms and facilitate the identification of reliable biomarkers of exposure and early biological effects.

Public Health, Policy and Sustainable Interventions

Strengthening surveillance of microplastics in food, drinking water and environmental media should become a public health priority. Standardized monitoring programs, safer food-contact materials and improved food processing practices are essential to minimize human exposure. Future policies should support harmonized exposure guidelines, international regulatory frameworks and integration of microplastic monitoring into existing environmental and food safety programs. At the same time, reducing plastic pollution through sustainable product design, circular economy strategies, improved recycling and reduced reliance on single-use plastics will provide long-term benefits for both environmental and human health.

Overall, future research should move beyond documenting the presence of microplastics toward determining who is exposed, at what levels, through which pathways and with what health consequences. Achieving this goal will require standardized methodologies, multidisciplinary collaboration and a One Health approach that recognizes the interconnectedness of environmental sustainability, food safety and human health. Such evidence will be essential for guiding regulatory policies and developing effective strategies to reduce human exposure to this emerging environmental contaminant.

CONCLUSIONS

Microplastics have become ubiquitous environmental contaminants, with widespread occurrence in ecosystems, food systems, drinking water and human tissues, making human exposure virtually unavoidable. Their presence throughout the food chain has elevated plastic pollution from an environmental issue to an emerging public health concern. Experimental evidence suggests that microplastics can induce oxidative stress, inflammation, immune dysregulation, endocrine disruption, mitochondrial dysfunction and gut microbiome alterations, providing biologically plausible mechanisms for adverse health effects. However, despite growing evidence of systemic exposure, definitive causal relationships between environmental microplastic exposure and specific human diseases remain unestablished because of limited epidemiological data and methodological inconsistencies. From a public health perspective, the pervasive nature of exposure, contamination of food systems and potential risks to vulnerable populations highlight the need for a precautionary approach. Reducing plastic pollution at its source, strengthening food safety surveillance, improving waste management, promoting sustainable packaging and increasing public awareness are essential strategies for minimizing human exposure. Equally important is the adoption of **a** One Health approach that recognizes the interconnectedness of environmental, animal and human health. Future research should prioritize standardized analytical methods, robust human biomonitoring, environmentally relevant toxicological studies and large prospective epidemiological cohorts to clarify exposure-response relationships and support evidence-based risk assessment. Collectively, these efforts will be critical for informing regulatory policies, advancing sustainable plastic management and protecting human health from the growing challenge of microplastic pollution.

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