

REVIEW ARTICLE

Exposure to Microplastics and Nanoplastics through Animal-Derived Foods: A One Health Perspective on the Convergence of Environmental and Health Risks

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Abstract

Microplastics (MPs) and nanoplastics (NPs) pose a significant One Health challenge that transcends the boundaries of the environment, animal and human health, due to their widespread presence in global food systems. This narrative review draws on peer-reviewed literature published up to May 2026 to assess environmental entry points, animal exposure pathways, veterinary health outcomes and impacts on human health. Our analysis confirms that there is strong evidence regarding environmental contamination and entry into aquatic food chains; however, it reveals significant knowledge gaps at the animal-to-human interface, particularly concerning terrestrial farm animal products such as meat, milk and eggs. Toxicological studies indicate that multi-organ pathology can occur at high exposure concentrations; however, inferences regarding real-world dietary exposure are limited by methodological constraints, such as the use of pure polystyrene spheres at doses up to 100,000 times higher than environmental levels and the absence of standardized analytical protocols. Crucially, there is no longitudinal epidemiological evidence for a causal relationship between dietary microplastic ingestion from animal-derived foods and specific clinical outcomes in humans. We propose an integrated One Health surveillance architecture that unites environmental monitoring, animal sentinels, and human biomonitoring. Prioritizing standardized methods, terrestrial product contamination data, and prospective cohort studies is essential for an evidence-based risk assessment that does not undermine the proven nutritional benefits of ADFs.

Keywords: Microplastics, Nanoplastics, One Health, Animal-derived foods, Food safety, Environmental contamination, Human health, Risk assessment

INTRODUCTION

Animal-derived foods (ADFs), including meat, dairy products, fish and eggs, remain important components of human diets. They provide high-quality protein and essential micronutrients, particularly for growing children, pregnant women, and malnourished populations. Low intake of these foods has been linked to stunted growth, anemia and poor bone development. However, ADFs can also act as pathways through which environmental pollutants enter the human food supply. When farm animals, poultry or fish are reared under production conditions where they come into contact with contaminated

feed, water or soil, pollutants can enter animal production systems and ultimately reach the human food chain ^[1]. This dual role-nutritional value on the one hand and potential contaminant transfer on the other-makes ADFs a suitable framework for examining microplastic (MP) risks from a One Health perspective. MPs are generally defined as solid plastic particles ranging from 1 µm to 5 mm in size, whereas nanoplastics (NPs) comprise smaller plastic particles in the approximately 1-1000 nm size range ^[2]. Although size boundaries may vary among classification frameworks, these ranges are widely used in current environmental and food-safety literature.



The One Health perspective is important because MPs move across the boundaries of the environment, animal health and human health [3]. They can pass from soil and water to plants, animals and, ultimately, human consumers. Addressing this movement requires collaboration between environmental scientists, veterinarians, food safety experts and public health researchers. Within this framework, indirect exposure pathways are just as important as direct ones. Ecosystem degradation, produce contaminated via fertilizers or irrigation, and the long-term accumulation of plastics in the oceans and on agricultural land are among these pathways. These processes may increase future exposure risks for both animals and humans [4,5].

Modern agriculture is heavily reliant on plastic materials. Plastic mulch films, sewage sludge used as fertilizer, and irrigation with wastewater introduce MPs into agricultural soils, where they can persist and accumulate over time. In some regions, concentrations of up to 80,000 particles per kilogram of soil have been reported [6]. These concentrations are of particular significance when assessed in terms of animal exposure pathways. Earthworms can ingest MPs from the soil and are subsequently consumed by poultry; this creates a direct, biologically mediated transfer pathway that bypasses the plant route [7]. Grasses, which are an important component of ruminant diets, may also be affected by MP contamination. Experimental studies show that MPs reduce grass seed germination by 17-30% and shoot biomass by approximately 21% [8]. Furthermore, MPs can be taken up via the roots and transported within the plant through the xylem vessels, potentially becoming part of the forage [9]. Olmo and Holman [10] detected between 36 and 300 MP particles per kilogram in animal feed and up to 7,900 particles per kilogram in surrounding soil. MPs have also been detected in honey; here, contamination may occur via pollen, airborne dust or broader environmental exposure [11]. These findings demonstrate that soil, air, water and plants are not separate compartments; rather, they are interconnected reservoirs that can directly supply animal production systems.

Entry into the environment is only the first stage. The critical question is whether environmental MPs are transferred from farms to animal products such as meat, milk and eggs, and through which mechanisms this transfer occurs. The transfer can take place via various direct and indirect routes. Animals may ingest MPs via contaminated drinking water and feed; free-range poultry, meanwhile, may be indirectly exposed by consuming earthworms that have accumulated soil-borne plastic particles [12]. Secondary contamination may arise from residues carried by the wind from feed packaging bags, irrigation hoses, silage covers, bale netting and nearby landfill sites. The agricultural equivalents of polyethylene

packaging -which can break down into MPs and leach into food [13] are found in the plastic materials used in feed storage and processing. Once inside the animal's body, small particles-particularly those under 150 µm can pass through the intestinal wall, enter the bloodstream and accumulate in organs such as the liver, kidneys and muscles [14]. MPs have been detected in meat, liver, kidneys, raw milk and eggs [15,16]. Consequently, at a mechanistic level, the pathway from agricultural plastic waste to potential human dietary exposure appears plausible. However, this should not be taken as evidence of a public health crisis on a real-world scale. Interpreting the current evidence is complex. The detection of MPs in ADFs is not sufficient to classify the issue as a confirmed One Health risk. Many experimental studies use exposure concentrations that are hundreds or thousands of times higher than typical real-world exposure levels. These studies often rely on pure plastic spheres rather than the worn and irregular particles found in food systems. Analytical inconsistencies -such as the lack of standardized methods, inconsistent reporting units and the risk of procedural contamination-also limit interpretation. There is still no validated and rapid test procedure for MPs in food. Although evidence increasingly suggests that MPs are widespread, can move through food chains and can cause harm in laboratory animals at very high doses, a clear causal link between the consumption of contaminated meat or milk and specific human diseases has not yet been established.

This review addresses this gap by tracing the chain of evidence from environmental contamination through to animal exposure and human dietary intake. It identifies which links are well-established, which remain weak, and which systemic barriers limit the ability to draw stronger conclusions. Given their position at the intersection of environmental, veterinary and human health, the review focuses specifically on food of animal origin as a route of exposure. In doing so, it demonstrates how the widespread use of plastics challenges the One Health perspective, whilst also highlighting where stronger scientific evidence is required before regulatory decisions or risk communication strategies can be developed.

DATA SOURCES AND SEARCH STRATEGY

This narrative review is based on a comprehensive literature search conducted in the Web of Science, Scopus and PubMed databases. The search focused on studies addressing MP contamination in food of animal origin and the associated food chain pathways. No restriction was applied to the starting publication year, and all relevant publications available up to 1 May 2026 were considered for inclusion in the review. Prior to the main

search, a preliminary review was conducted to identify the most commonly used terminology in the field. Search terms combined concepts related to MPs, NPs and polymer identification with descriptors pertaining to ADFs and animal production systems. These included “microplastic”, “nanoplastic”, “polymer”, “polymer type”, “polymer identification”, “polymer composition”, “animal-derived food”, “livestock”, “poultry”, “cattle”, “sheep”, “goat”, “pig”, “meat”, “milk”, “eggs”, “dairy”, “seafood”, “aquaculture”, “fish”, “shellfish”, “food safety”, “animal health”, “One Health” and “trophic transfer”. Boolean operators were used to combine these terms and, where necessary, to broaden or narrow the search.

This review focuses on studies reporting the detection, quantification or characterization of MP in ADFs, including meat, milk, eggs, seafood, dairy products and related matrices. Studies on animal feed were considered where they provided information on the transfer of MP to food products intended for human consumption. Research on aquatic organisms was included where these organisms form part of the human food chain. Studies on environmental matrices, plant systems or toxicological mechanisms were utilized where they contributed to the interpretation of food chain transfer or the broader One Health context. Studies on honey were also considered where they related to ADFs and environmental exposure pathways. Original research articles formed the main basis of the review. Review articles, conference proceedings, books and book chapters were utilized where they provided background information, a conceptual framework or methodological context. Publications in languages other than English were excluded. After duplicate records were removed, the publications were assessed for their relevance to the objectives of the review by examining the title, abstract and, where necessary, the full text. The selected literature was categorized thematically in terms of food type, contamination levels, polymer composition, analytical methods, potential sources of contamination, food safety implications and its relevance to One Health.

ONE HEALTH EVIDENCE-CHAIN FRAMEWORK

A structured evidence-chain framework is needed to connect environmental contamination, animal exposure and human dietary intake in the assessment of MP and NP risks through ADFs. Environmental contamination leads to animal exposure through a wide array of pathways, including direct ingestion, inhalation, trophic transfer, and the movement of biological and environmental vectors. Food-producing animals may be exposed to MPs and NPs through ingestion or inhalation of contaminated environmental matrices, including soil, water, air and

commercial feed ^[4,15]. Particles smaller than 150 µm may cross biological barriers, including the intestinal and pulmonary epithelia, and enter the bloodstream ^[17]. Larger particles are often excreted in feces, whereas smaller internalized particles may travel via the bloodstream and lymphatic system to various organs and tissues ^[17,18].

MPs may be present in ADFs intended for human consumption. They have been detected in edible tissues such as meat, liver and kidneys of livestock and poultry ^[19]. In milk-producing animals, these particles may pass into the mammary glands and enter raw milk. In poultry, they can enter developing eggs before they are laid ^[15,16]. Post-harvest contamination can also occur when food contacts plastic materials during production, processing, handling and packaging. Plastic equipment, worker gear and packaging may release MPs or additives into food, especially in fatty products where leaching is higher ^[20].

Exposure to MPs and NPs may contribute to health hazards through several interconnected pathways. These particles may enter circulation and accumulate within organs such as the liver, kidneys, lungs, brain and reproductive tissues after passing through biological barriers such as the intestinal, placental and blood-brain barriers. At the cellular level, exposure has been associated with endocrine disruption, oxidative stress, inflammation, microbiome imbalance, reproductive toxicity and neurotoxicity ^[19,21]. Additionally, pathogens and antibiotic resistance genes may be carried by MPs, raising the risk of microbial infections ^[21,22].

WELL-ESTABLISHED LINKS IN THE EVIDENCE CHAIN

Environmental contamination and entry into aquatic food chains are the most mature and substantiated parts of the current evidence chain. MPs are widely detected in major environmental matrices, including air, water and soil ^[23,24]. Consequently, the ingestion of MPs by marine and freshwater organisms, including commercially important fish and shellfish, is a recognized pathway for entry into the human food system ^[25]. This makes aquatic food-chain entry one of the strongest links in the current evidence chain. A second well-established link is the role of MPs as microbial scaffolds. A growing body of research shows that MPs may enrich microbial communities carrying antibiotic resistance genes (ARGs), representing an emerging One Health concern ^[26]. Third, the molecular mechanisms associated with MP-induced damage, particularly oxidative stress, chronic inflammation and cellular apoptosis, have been demonstrated in model organisms such as mice and zebrafish ^[17,27]. However, these studies have typically been conducted under controlled laboratory conditions using virgin polystyrene (PS) beads.

Finally, the presence of MP in human biological samples, including blood, placenta, feces and lung tissue, has been documented in analytical studies ^[21]. These findings support the view that environmental plastic particles can enter the human body. Taken together, these established links indicate that MP can enter the human food system via aquatic animals; however, evidence regarding terrestrial livestock products remains more limited.

PERSISTENT GAPS AND UNDERSTUDIED DOMAINS

Despite some established links, significant gaps remain in our understanding of the interfaces between environmental reservoirs, ADFs and clinical outcomes in humans. Compared with aquatic systems, the presence of MP in terrestrial farm animal tissues -including fresh meat, raw milk and eggs- and in raw ADFs has still not been sufficiently investigated ^[15,28]. The physiological mechanisms governing the transfer of MP from feed or contaminated soil to edible organs, milk and eggs are also not yet fully understood ^[27].

Most toxicity data are derived from studies using MP concentrations well above environmentally relevant levels. This limits the validity of these findings for real-world dietary exposure assessments ^[24,29]. The literature is also skewed; over 96% of studies focus on pure polystyrene spheres. This narrow focus fails to adequately represent other polymer types, such as polyethylene, polyvinyl chloride and polyethylene terephthalate, as well as the fragmented and fibrous morphologies commonly found in real food systems ^[13].

Another significant gap concerns the effects of food processing methods -including cooking, pasteurization and high-temperature treatments- on the physical properties, chemical composition and toxicological significance of MP in ADFs ^[4]. Although MP's have been detected in human biological samples, definitive evidence linking dietary MP exposure to specific clinical outcomes in humans is still lacking ^[24,29]. With regard to ADFs such as meat, milk and eggs, these gaps mean that real-world risk cannot yet be accurately quantified.

SYSTEMIC BARRIERS UNDERPINNING THE INCOMPLETE EVIDENCE CHAIN

There are several systemic barriers contributing to gaps in the chain of evidence. Unless these barriers are addressed, any assessment of MP contamination in ADFs will continue to be incomplete. First, no universally accepted protocol for the extraction and identification of MPs exists. Various analytical instruments, including Fourier-transform

infrared spectroscopy (FT-IR), Raman spectroscopy, and pyrolysis-gas chromatography/mass spectrometry (Py-GC/MS), produce inconsistent results regarding particle size and polymer composition ^[13,17,30]. Second, current quantification procedures typically overlook the most biologically active particles. Conventional filters and detection thresholds are inadequate for capturing NPs smaller than 1 micrometer or small MPs smaller than 150 micrometers ^[30,31]. Third, incompatible reporting units, such as particles per kilogram and milligrams per kg, hinder quantitative meta-analyses throughout the food chain ^[32]. Fourth, research endeavors continue to be divided between environmental ecotoxicologists, who primarily examine ecosystem loads, and biomedical clinical researchers, who focus on cellular reactions. This results in limited multidisciplinary integration in study design ^[33]. Fifth, at the institutional level, environmental health has only recently been formally incorporated into the Tripartite, now Quadripartite, One Health alliance. Thus, environmental surveillance infrastructure notably lags behind its human and animal clinical equivalents ^[25,34].

STRATEGIC ADVANTAGES OF THE ONE HEALTH EVIDENCE-CHAIN FRAMEWORK FOR FUTURE RISK ASSESSMENT

Despite the gaps and barriers noted above, the One Health evidence-chain framework itself offers several strategic advantages for future risk assessment. Implementation science approaches, exemplified by the CLEAN procedure (Cut, Lock, Extract, Amend, Network), move beyond descriptive environmental surveys. They offer actionable roadmaps that directly connect scientific evidence to policy initiatives and source control ^[25].

The framework facilitates the use of sentinel species, such as honeybees, as bioindicators of air and soil MP levels. These can function as early warning systems for human dietary exposure, allowing for pre-emptive intervention before contaminants enter the food chain ^[3].

The approach advocates for cumulative exposome evaluation through comprehensive dietary and environmental exposure modelling. This integrates combined loads from water, food and inhalation, rather than depending on discrete, compartmentalized studies ^[33].

It requires standardized data collection across air, water, soil, food and human tissue matrices. This is essential for pinpointing areas where MP pollution and antimicrobial resistance risks intersect ^[25,31]. By defining MPs as a population-level environmental and occupational hazard, the framework facilitates anticipatory governance. Nations

and regulatory bodies can tackle emerging threats through proactive design and manufacturing modifications instead of solely relying on reactive medical interventions ^[25].

ENVIRONMENTAL ENTRY POINTS INTO ANIMAL FOOD SYSTEMS

Environmental entry points of MPs and NPs into animal food systems are extensive, occurring at every stage from the abiotic environment to post-harvest processing. The main environmental entry points are outlined below.

Primary Environmental Sources of Microplastics

Primary MPs are intentionally manufactured at microscopic sizes for specific commercial and industrial purposes, with common examples including microbeads in personal care products such as exfoliating scrubs and toothpaste, industrial abrasives used in sandblasting and machinery cleaning, and plastic pellets known as nurdles, which serve as the raw material for producing nearly all plastic goods ^[35]. Unlike secondary MPs, which form from the environmental breakdown of larger plastic items via mechanical abrasion, photooxidation, and biological degradation, these primary MPs are deliberately created at a small scale, making them particularly problematic for wastewater treatment systems, especially microbeads, which can flow directly into oceans and waterways ^[28,36]. While many countries have banned rinse-off microbeads in cosmetics, nurdles and industrial abrasives continue to be released during manufacturing, transport, and industrial processes, contributing significantly to global plastic pollution. These sources are relevant to ADFs when released particles enter wastewater, agricultural soils, irrigation systems, feed production environments or aquatic food chains.

Entry Points into Aquatic and Terrestrial Environments

MPs enter aquatic and terrestrial environments through a variety of routes, which are becoming increasingly well documented, although they are still poorly regulated in comparison to traditional pollutants. MPs enter aquatic systems primarily through wastewater treatment plant (WWTP) effluents, surface runoff from contaminated land, and atmospheric deposition ^[9,37]. Although WWTPs can remove up to 99% of particles, large numbers of MPs may still be discharged into rivers and oceans each day ^[37,38]. Similarly, soils are contaminated through intensive farming practices, including the application of plastic mulch films, sewage sludge, organic compost containing plastic residues, and irrigation with contaminated wastewater ^[39,40].

The Role of Wastewater and Sewage Sludge

WWTPs efficiently trap MPs in the solid fraction, concentrating them in sewage sludge (biosolids), which

is frequently applied to farmlands as a nutrient-rich fertilizer ^[37]. Sewage sludge is considered a major driver of soil MP pollution, with some estimates suggesting that up to 430,000 tons of plastic are deposited on European farmlands annually via this route ^[28,41]. Plastic particles in wastewater and sludge have high surface areas that readily adsorb toxic organic and inorganic pollutants, such as heavy metals (e.g., cadmium), pesticides, and polycyclic aromatic hydrocarbons (PAHs) ^[9,42]. Reclaimed wastewater used for irrigation can introduce MPs directly onto crop surfaces and into the soil matrix.

Impact of Plasticulture and Agricultural Films

The term “plasticulture” refers to the systematic use of plastic materials in agricultural production to enhance crop yield, extend growing seasons, improve water use efficiency, and manage pests and weeds. Modern plasticulture is overwhelmingly dependent on three primary categories of plastics: mulch films, greenhouse covers, and protective nets ^[43].

Ramanayaka et al.^[44] reported that the degradation of non-biodegradable plastic mulch films over time leads to an increase in the release of plastic additives and MPs into agricultural soils, with di(2-ethylhexyl) phthalate (DEHP) detected at concentrations as high as 25.2 mg/kg in agricultural soils, indicating potential risks of uptake, translocation, and accumulation in plants. Greenhouse covers, constructed from polyethylene, polycarbonate, or ethylene-vinyl acetate (EVA), create controlled microenvironments that protect crops from extreme weather, but the weathering of these covers due to ultraviolet radiation leads to the gradual release of MP particles into underlying soils. Sorasan et al.^[45] demonstrated that naturally aged greenhouse cover films release more nano- and MP particles than new films under simulated sunlight exposure, with Raman spectroscopy confirming the lower crystallinity of released polyethylene NPs compared to new films. Protective nets, including anti-insect nets, bird nets, and shade nets made from high-density polyethylene or polypropylene, are deployed as physical barriers to exclude pests while reducing the need for chemical pesticides; however, these nets undergo UV degradation and mechanical abrasion during extended field use, generating MPs that are subsequently mobilized by wind, rain and irrigation. Consequently, there is a fundamental trade-off between short-term agricultural productivity and long-term soil health and environmental contamination due to modern plasticulture’s reliance on these three plastic forms ^[46]. This highlights the need for biodegradable alternatives, improved retrieval technologies and updated agricultural guidelines.

Exposure Pathways for Food-Producing Animals

Food-producing animals such as cattle, sheep, goats, pigs and poultry are exposed to environmental MPs via three main ingestion routes: contaminated water, contaminated

feed and biological vectors. Livestock often drink surface water from rivers, ponds and reservoirs contaminated by MPs from agricultural wastewater, wastewater discharge and atmospheric deposition ^[47]. In locations where treated wastewater is used for irrigation, grazing animals may consume MPs via pasture vegetation and standing water, and the presence of MPs in drinking water sources in areas of intensive agriculture has been quantified ^[48].

The second pathway, contaminated feed, occurs through two main mechanisms. Grazing animals often ingest contaminated soil in addition to the plants they consume, particularly soil-bound MPs that have accumulated from plasticulture practices and sewage sludge application ^[40]. Processed fodder and silage may also be contaminated during storage and handling through plastic packaging, silage wraps and bale nets ^[49].

The third important channel is biological vectors, which provide an indirect MP transmission pathway from soil to animal systems. Soil fauna, such as earthworms (*Eisenia fetida* and *Lumbricus terrestris*), ingest MPs from contaminated soils while digging and feeding, resulting in the build-up of particles in their gut tissues ^[12]. Free-range poultry, including chickens, turkeys and guinea fowl, may ingest MPs directly when they consume earthworms as a natural protein source, thereby introducing soil-derived plastic particles into the avian digestive tract. Compelling evidence for this sequence was provided by Huerta Lwanga et al. ^[12], who showed that chickens fed MP-contaminated earthworms accumulated significant loads of MPs in their gastrointestinal tracts, with MP concentrations in feces reaching up to 129.8 particles per gram dry weight. The soil-earthworm-chicken pathway is important because it demonstrates trophic transfer of MPs from soil invertebrates to higher vertebrates, suggesting that terrestrial food chains may be susceptible to MP transfer even in the absence of direct plastic exposure ^[50].

Contributions from Aquaculture Systems

Aquaculture systems serve as significant environmental entry points for MPs and NPs into animal food systems, acting as both a source of contamination and a conduit for trophic transfer. These contributions occur through the degradation of infrastructure, operational practices and the use of contaminated feed materials. Modern aquaculture relies heavily on plastic materials for fish cages, pens, pond linings and tanks; for example, gravity cages use high-density polyethylene pipes for structural frameworks, which can fragment due to UV exposure and mechanical stress ^[18]. Expanded polystyrene floats and buoys are also major contributors to MP pollution, with fragments from aquaculture debris accounting for over 90 per cent of MPs found on nearby shorelines in some regions.

Once introduced, contaminants are amplified and stabilized by specific environmental matrices within the farm environment. MPs serve as microbial scaffolds that may selectively enrich ARGs and antibiotic-resistant bacteria (ARB), while these “microplastomes” can facilitate horizontal gene transfer across different microbial species and environmental matrices ^[22,51]. Additionally, pond sediments function as long-term repositories for antibiotic residues and resistance genes, and these determinants can be resuspended into the water column during storms or routine farm operations, continuously re-exposing cultured animals ^[52].

The transmission from the environment into the food system occurs through several direct and indirect routes. Cultured species such as tilapia ingest MPs and associated resistant pathogens directly from the water and sediment, and research has tracked the movement of these resistomes from pond ecosystems into edible fish guts, indicating a potential risk for human dietary safety ^[14]. Therefore, the integration of these contaminants into the food system requires a source-to-table surveillance approach, as current governance often fails to address how environmental dynamics in aquaculture translate into human dietary exposure risks. The principal production environments, animal groups, ADFs consumed by humans, exposure sources and high-risk MP contamination sources are summarized in [Table 1](#).

POTENTIAL HEALTH IMPLICATIONS AND TOXICOLOGICAL UNCERTAINTY

Consequences on Animal Health

Building upon established environmental exposure pathways, the ingestion of MPs and NPs by livestock, poultry and aquatic species may induce systemic physiological stress. In vivo studies in terrestrial and aquatic organisms suggest that exposure can contribute to multi-organ toxic effects ^[4,10,17,19,27]. Owing to their small size, high surface-area-to-volume ratios and chemical persistence, these particulate contaminants may cross physiological barriers, translocate into tissues and contribute to systemic or organ-specific pathological changes ^[4,17,19]. These effects are relevant not only to veterinary health and animal welfare, but also to agricultural biosecurity ^[10,19,27].

Gastrointestinal Damage and Microbiome Dysbiosis

As the primary portal of entry in food-producing animals, the gastrointestinal tract is a key site of MP exposure and potential accumulation. Ingested plastics may exert mechanical stress on the intestinal mucosa, leading to villus damage, enterocyte degradation and disruption of the intestinal vascular barrier ^[57,58]. These alterations may increase intestinal permeability and

Table 1. Environmental exposure pathways and highrisk sources of MP contamination in animal production systems

Production or Exposure Environment	Source Animal Groups	Animal-Derived Foods Consumed by Humans	Common Environmental Feed and Exposure Sources	High-Risk Sources for MP Contamination	References
Terrestrial Livestock Systems	Cow, buffalo, goat, sheep, camel, yak, rabbit, pig	Beef, veal, buffalo meat, goat meat, mutton, lamb, pork, rabbit meat, milk, cheese, yogurt, butter, ghee, cream, curd, liver, kidney, heart, tongue, tripe, bone broth, animal fat, gelatin, collagen	Grasses, forbs, leaves, hay, silage, crop residues, straw, grains, roots, tubers, agricultural by-products, commercial feed, soil particles, and drinking water	Plastic mulch-contaminated soil, sewage sludge-amended soil, wastewater irrigation, contaminated grasses and forbs, feed packaging bags, plastic ropes, irrigation pipes, drinking water, airborne dust, and landfill-derived debris	[8,12,53]
Poultry Production Systems	Chicken, duck, turkey, goose, quail, pigeon	Chicken meat, duck meat, turkey meat, goose meat, quail meat, pigeon meat, eggs, liver, heart, gizzard, poultry fat, poultry broth	Grains, seeds, grasses, small plants, insects, worms, snails, kitchen scraps, commercial feed, soil particles, litter material, and drinking water	Contaminated commercial feed, plastic feed sacks, poultry litter, soil and dust particles, plastic bedding materials, contaminated drinking water, synthetic fibres from farm materials, and degraded plastic equipment	[27,53,54]
Freshwater Aquatic Systems	Freshwater fish, prawn, crab, snail	Freshwater fish, dried fish, smoked fish, fermented fish, fish roe, fish oil, prawn, freshwater crab, edible snails	Phytoplankton, zooplankton, algae, aquatic plants, detritus, insects, larvae, worms, small fish, sediment-associated organic matter, aquaculture feed, and freshwater	Polluted rivers, wastewater discharge, aquaculture ponds, plastic fragments in sediment, contaminated aquaculture feed, fishing nets, plastic ropes, plastic bags, domestic waste, and industrial effluents	[27,53,55]
Marine Aquatic Systems	Marine fish, shrimp, crab, lobster, squid, octopus, cuttlefish, shellfish	Marine fish, canned fish, salted fish, dried fish, fish roe, caviar, fish liver, cod liver oil, shrimp, crab, lobster, squid, octopus, cuttlefish, mussels, oysters, clams, scallops, abalone	Phytoplankton, zooplankton, algae, seaweed, detritus, small fish, crustaceans, mollusks, marine worms, suspended organic particles, sediment organic matter, and seawater	Marine plastic debris, MP-contaminated seawater, fishing nets, ropes, plastic packaging, ship waste, coastal wastewater discharge, contaminated sediments, and filter-feeding exposure in shellfish	[17,18,56]
Coastal and Brackish-Water Systems	Shrimp, prawn, crab, brackish-water fish, oysters	Shrimp, prawn, crab meat, brackish-water fish, oysters, dried shrimp, shrimp paste, fish sauce	Algae, phytoplankton, zooplankton, detritus, organic-rich sediment, small aquatic organisms, aquatic plants, aquaculture feed, brackish water, and coastal sediment	Coastal plastic waste, aquaculture pond liners, plastic nets, plastic ropes, contaminated sediment, wastewater runoff, agricultural runoff, plastic feed bags, and degraded fishing materials	[18,55,56]
Insect and Hive-Based Systems	Bees, crickets, grasshoppers, locusts, silkworms, ants, termites	Honey, royal jelly, bee pollen, propolis, crickets, grasshoppers, locusts, silkworm pupae, ants, termites	Nectar, pollen, plant leaves, grasses, crop plants, tree sap, fruits, seeds, decaying organic matter, fungi, wood material, plant residues, and environmental dust	Airborne MP particles, contaminated pollen, polluted plant surfaces, plastic mulch residues, agricultural dust, plastic greenhouse films, contaminated leaves, and degraded plastic materials near farms	[8,30]

suppress mucus secretion, thereby weakening normal gut barrier function ^[59,60]. MPs may also alter the diversity and taxonomic composition of the gut microbiome ^[60]. Experimental studies in avian and rodent models have reported reductions in beneficial, anti-inflammatory taxa, including *Clostridium* and *Faecalibacterium*, together with increases in opportunistic microorganisms ^[36,58]. In livestock and poultry, the combined effects of mucosal injury and microbial dysbiosis may impair nutrient digestion and assimilation. These disturbances may consequently be reflected in reduced final body weight, lower daily weight gain and poorer feed conversion ratios,

linking gastrointestinal toxicity directly to animal health and production performance ^[10,15].

Oxidative Stress and Programmed Cell Death

Following ingestion and systemic translocation, plastic particles may induce intracellular redox imbalances. MP exposure may trigger the overproduction of reactive oxygen species (ROS) while simultaneously exhausting endogenous antioxidant defenses. This is marked by a significant downregulation of key enzymes, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) ^[10]. The resulting oxidative

stress may cause damage to cellular DNA, proteins and lipids. This damage can activate programmed cell death pathways, including apoptosis, necrosis and pyroptosis, across multiple organs such as the liver, kidneys, spleen and heart ^[10,27].

Immunotoxicity and Chronic Inflammation

The host immune system may recognize MPs and NPs as foreign bodies, provoking sustained immunological responses ^[4]. Exposure may drive pathological alterations within primary and secondary lymphoid organs, particularly the thymus and spleen, resulting in structural tissue disruption and inflammatory cell infiltration ^[10,27].

Particle exposure may activate key inflammatory cascades, including the NF- κ B and NLRP3 inflammasome pathways. This activation can trigger an overproduction of pro-inflammatory cytokines, such as tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), and interleukin-1 beta (IL-1 β) ^[10,61]. Over time, chronic inflammation may contribute to immune dysregulation and increased susceptibility to secondary infections ^[61].

Hepatic Toxicity and Metabolic Disruption

As a major metabolic and detoxification organ, the liver may accumulate translocated plastic particles and undergo metabolic disruption ^[4,57]. MPs may disrupt hepatic lipid and energy homeostasis by altering the transcription of critical metabolic genes, such as peroxisome proliferator-activated receptor gamma (PPAR γ), and by contributing to intracellular ATP depletion. This metabolic shift may promote the aberrant accumulation of intracellular lipid droplets, cholestasis and parenchymal damage. The presentation has been compared with the progression of non-alcoholic fatty liver disease (NAFLD) in mammalian models ^[4,10].

Reproductive and Transgenerational Toxicity

In experimental animal models, exposure to MPs and NPs has been associated with reproductive toxicity in both sexes. In male animals, accumulation within testicular tissue has been linked to spermatogenic cell death, testicular inflammation and interstitial fibrosis, together with reductions in sperm count, viability and motility ^[4,31]. In female animals, exposure has been associated with ovarian fibrosis, depletion of primordial and mature follicle pools and alterations in reproductive hormone levels, including reduced estradiol and progesterone concentrations ^[4,62]. Experimental evidence also indicates that NPs may cross the placental barrier during gestation. Maternal exposure has been associated with vertical transfer to fetal tissues and with adverse developmental outcomes, including intrauterine growth restriction, altered offspring sex ratios and metabolic disturbances in subsequent generations ^[4,61,62]. These findings indicate

that reproductive toxicity may have implications not only for individual animal health but also for breeding performance and population sustainability.

Neurological and Cardiovascular Disorders

Beyond metabolic and reproductive systems, plastic particles may exert toxic effects on the neuro-cardiovascular axis. NPs capable of traversing the blood-brain barrier may accumulate within central nervous system tissues. This localized accumulation may be associated with microglial activation, cerebral and cerebellar hemorrhages, acetylcholinesterase (AChE) inhibition and neurobehavioral changes ^[4,10,32]. Concurrently, the myocardium may be sensitive to MP-induced oxidative stress. Reported cardiovascular outcomes include myocardial dysplasia, bradycardia, cardiomyocyte apoptosis and progressive cardiac fibrosis ^[10,27,61].

Environmental Vectors for Pathogens and Xenobiotics

Beyond their intrinsic particle toxicity, MPs may act as environmental vectors because of their hydrophobic surfaces and high specific surface areas, increasing the risks associated with co-occurring contaminants ^[4,10]. As chemical carriers, MPs can partition and concentrate heavy metals, including cadmium and lead, persistent organic pollutants (POPs), polycyclic aromatic hydrocarbons (PAHs), and veterinary antibiotics such as chlortetracycline from the surrounding environment ^[63,64]. When co-ingested, these contaminants may exacerbate toxic effects. For instance, avian models co-exposed to polyvinyl chloride (PVC) MPs and cadmium show compounded hepatic atrophy and necrotic damage compared with single-contaminant exposure ^[65,66]. Additionally, weathering plastics may leach intrinsic manufacturing additives, including endocrine-disrupting phthalates and bisphenol A (BPA) ^[67].

MPs may also act as biological vectors within the “plastisphere,” providing a stable substrate for the formation of complex microbial biofilms. These biofilms may harbor and transport zoonotic pathogens, including *Vibrio* spp., *Helicobacter pylori* and *Salmonella*, potentially contributing to gastric infections and mucosal damage upon ingestion ^[68,69]. The dense microbial ecology of the plastisphere may also facilitate horizontal gene transfer of ARGs, accelerating the emergence and dissemination of multidrug-resistant bacteria within intensive livestock operations ^[1,10].

Ultimately, multi-organ damage, systemic inflammation and the accumulation of xenobiotics within edible animal tissues may present a dual concern. These effects can reduce agricultural yield and animal welfare, while also contributing to the movement of plastic particles and associated chemical or biological contaminants through

the food chain. These veterinary consequences may contribute to human exposure through the consumption of ADFs containing plastic particles and associated contaminants.

IMPLICATIONS FOR HUMAN HEALTH

Human exposure to MPs and NPs is now widespread, occurring primarily through three main routes: ingestion of contaminated food and water, inhalation of airborne particles and dust, and dermal contact [32]. Under certain conditions, these particulate contaminants may cross biological barriers and enter systemic circulation, where they may contribute to multi-organ effects [17,70,71].

Gastrointestinal Toxicity and Gut Microbiome Dysbiosis

In humans, the gastrointestinal tract represents a major site of exposure to ingested plastic particles and may serve as an important interface between dietary exposure and systemic effects. Particles smaller than 150 µm may penetrate the intestinal mucus layer, while smaller fractions can be internalized through endocytosis, including uptake mediated by specialized microfold cells (M-cells) located within the Peyer's patches of the gut-associated lymphoid tissue [61]. Persistent exposure to MPs within the intestinal lumen may alter both the taxonomic composition and metabolic activity of the gut microbiota [60]. Such alterations may contribute to dysbiosis and compromise intestinal barrier integrity by increasing gut permeability. Unlike the predominantly experimental evidence available for food-producing animals, human evidence is currently based mainly on observational findings. Patients with inflammatory bowel disease have been reported to exhibit significantly higher levels of MPs in fecal samples than healthy controls [72,73]. Although this does not establish causality, it suggests a possible association between intestinal MP burden, microbiome disturbance and chronic intestinal inflammation.

Damage to the Respiratory and Pulmonary Systems

Beyond dietary ingestion, inhalation of airborne MPs, particularly synthetic microfibers shed from textiles and suspended indoor dust, represents another important exposure route for the human respiratory system [74]. Inhaled particles may penetrate deep into the lower airways and provoke a pathological response known as "dust overload". Under these conditions, alveolar macrophages may be unable to clear the foreign material effectively, contributing to chronic airway inflammation, alveolitis and progressive interstitial lung disease [61,75]. In vitro models using human lung epithelial cells indicate that MP exposure may downregulate α1-antitrypsin expression, a biomarker associated with increased COPD risk. These particles may also induce DNA damage, which

could contribute over time to malignant transformation under certain exposure conditions [61,69].

Systemic Translocation and Cardiovascular Risks

Once MPs and NPs cross the intestinal or pulmonary epithelial barriers, they may enter systemic circulation, as suggested by their detection in human blood samples [70,71]. Circulating plastic particles may contribute to systemic inflammatory cascades, cytotoxic effects on blood cells and vascular occlusion [17]. Recent studies have identified MPs and NPs embedded within human thrombi (blood clots) and atheromas (arterial plaques) [76]. The presence of these particles within vascular tissues has been associated with an increased risk of cardiovascular events, including coronary artery disease, myocardial infarction and stroke [75,76].

Cellular Damage, Oxidative Stress, and Genotoxicity

At the cellular level, the large surface area and structural reactivity of NPs may disrupt baseline metabolic homeostasis. Intracellular accumulation of plastic particles may result in the overproduction of reactive oxygen species (ROS) while exhausting endogenous antioxidant pools. This redox imbalance can cause oxidative damage to cellular proteins and membrane lipids [67,77].

Oxidative stress may also contribute to DNA damage, mitochondrial dysfunction and activation of programmed cell death pathways. These processes can promote downstream cellular changes that may contribute to carcinogenesis under sustained or high-exposure conditions [78,79].

Reproductive Toxicity and Transgenerational Effects

Evidence concerning reproductive effects in humans remains more limited than the experimental animal literature. MPs and NPs have been detected in several human reproductive matrices, including placenta, amniotic fluid, breast milk, semen and testicular tissue, demonstrating that reproductive organs and pregnancy-related tissues may be exposed to these particles [80]. Emerging studies have also raised concerns regarding possible effects on male reproductive parameters and the integrity of the blood-testis barrier [81,82]. However, much of the evidence concerning ovarian damage, hormonal disruption and developmental effects of prenatal exposure is still derived from experimental models rather than direct human clinical studies [83-85]. Accordingly, the detection of plastic particles in human reproductive tissues supports biological plausibility, but current evidence remains insufficient to establish causal relationships with specific reproductive or transgenerational outcomes in humans.

Neurotoxicity and Cognitive Effects

Due to their small size, NPs may cross the blood-brain barrier (BBB) and enter the central nervous system.

Accumulation within cerebral tissues may trigger microglial activation. This activation can drive elevated ROS generation and sustained neuroinflammation ^[32,86].

NPs have been implicated in structural neuronal damage, amyloid-beta (A β) oligomer formation and abnormal protein aggregation. These findings raise concerns that chronic low-dose exposure may contribute to the pathogenesis or progression of neurodegenerative disorders, including Alzheimer's disease, Parkinson's disease and multiple sclerosis ^[13,87].

Biological and Chemical Transport of Hazardous Pollutants

MPs may act as environmental vectors for co-occurring hazardous contaminants and may increase their toxicological relevance inside human tissues. Due to their hydrophobic surfaces, MPs can partition and concentrate persistent organic pollutants (POPs), including polychlorinated biphenyls (PCBs) and polycyclic aromatic hydrocarbons (PAHs), from surrounding aquatic and terrestrial environments ^[50].

MPs may also leach intrinsic manufacturing additives, including bisphenol A (BPA), phthalates and brominated flame retardants. These compounds may interfere with endocrine signaling pathways and have been associated with adverse metabolic, reproductive and developmental effects. In addition, MPs provide a stable substrate for the colonization of complex microbial biofilms. This "plastisphere" may host and transport virulent human pathogens, including *Vibrio* spp. and *Helicobacter pylori*, potentially contributing to gastrointestinal infection, mucosal damage and localized inflammation upon ingestion ^[61].

WELL-CALIBRATED RISK INTERPRETATION

Although numerous *in vitro* and animal studies support the biological plausibility of MP and NP toxicity, the extrapolation of these data to real-world human health hazards must be approached with a high degree of caution and balance. To date, no empirical evidence demonstrates a causal link between the ingestion of animal products contaminated with MPs and specific adverse human health outcomes.

The Gap in Environmental Relevance in Laboratory Studies

A significant challenge in current toxicological risk assessment is the wide disparity between laboratory experimental designs and real-world exposure scenarios. Many laboratory toxicity experiments expose animals or cell cultures to MP concentrations that are 2 to 7 orders of magnitude, up to 100,000 times, higher than those

normally detected in pristine or urban environments ^[24]. In addition, the vast majority of experimental research relies on pristine, perfectly spherical, synthetic polystyrene (PS) beads ^[60,88]. In reality, environmental MPs consist of a diverse mixture of irregular fragments, fibers and films composed of different polymers, including polyethylene (PE), polypropylene (PP) and polyethylene terephthalate (PET), which may have undergone prolonged weathering and microbial colonization within the plastisphere ^[89]. Therefore, idealized laboratory models may not fully reflect actual environmental exposure scenarios.

The Vector Effect and Chemical Contribution Dynamics

While MPs can adsorb and accumulate persistent organic pollutants (POPs), heavy metals and manufacturing additives, their contribution to total human chemical exposure through dietary intake appears limited. Worst-case exposure models for the consumption of highly contaminated seafood, such as bivalves and mussels, indicate that chemical transfer from ingested MPs accounts for less than 0.1% of total human dietary intake for toxicants such as polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), DDT, bisphenol A (BPA), and polybrominated diphenyl ethers (PBDEs) ^[18].

Baseline exposure to harmful chemicals from non-plastic food matrices, drinking water, indoor dust and conventional environmental pathways may therefore represent a larger contribution than the quantities introduced via MPs in both human and animal hosts ^[24].

Bioavailability, Clearance, and the Gastrointestinal Barrier

Not all consumed plastics accumulate within host tissues, as the gastrointestinal tracts of humans and animals function as selective protective barriers. The majority of ingested MPs, particularly those with dimensions exceeding 150 μm , are unable to cross the intact intestinal epithelium and are cleared from the body via fecal excretion ^[10]. For sub-micron plastic fractions, including NPs, that are small enough to breach the intestinal mucosal barrier, the estimated rate of systemic translocation into the bloodstream remains low, falling below 0.3% ^[90].

The Nanoplastic Analytical Blind Spot

NPs are theoretically expected to carry the highest toxicological risk because of their capacity to cross restrictive biological barriers and penetrate intracellular compartments ^[91]. However, this remains a substantial blind spot in conventional risk assessment. Characterizing and quantifying particles below the 1 μm threshold using standard spectroscopic techniques, such as micro-Fourier Transform Infrared (μ -FTIR) and micro-Raman spectroscopy, remains highly challenging.

Consequently, reliable and validated data on the actual occurrence, steady-state concentration and chemical composition of NPs in complex food matrices such as meat, milk and shellfish are still lacking. Without advanced, high-resolution analytical tools, true systemic NP risk remains difficult to quantify epidemiologically.

Methodological Inconsistencies and Lack of Standardization

The broader field of MP research is constrained by methodological variation, which limits cross-study comparisons and weakens the basis for regulatory conclusions^[31,53]. Differences in filter pore sizes, chemical digestion agents, including enzymatic and alkaline protocols, and reporting units, such as particles per gram and micrograms per liter, make results difficult to compare across studies^[92].

Procedural contamination also remains an important concern. Many published studies have lacked rigorous procedural blanks, creating a risk of false positives from background airborne microfibers or laboratory equipment during sample preparation^[93]. These limitations help explain the current absence of legally enforced and scientifically validated safety thresholds for MPs in human tissues or commercial food supplies.

Absence of Longitudinal Epidemiological Proof

The current body of literature supporting MP toxicity relies almost exclusively on short-term, acute animal exposure models or high-dose *in vitro* cell lines. To date, there are no long-term, prospective cohort, or longitudinal epidemiological studies evaluating the chronic health impacts of low-dose MP exposure on human populations across multiple decades^[91].

Balanced Nutritional Public Health Perspectives

From a public health and nutritional standpoint, the potential hazards of dietary MP exposure should be weighed against the established nutritional benefits of these foods. Animal-derived whole foods -including fish, lean meat, and dairy products- are critical sources of highly bioavailable proteins, essential micronutrients (e.g., Vitamin B₁₂, iron, zinc), and bioactive fatty acids vital for human growth, neurological function, and cardiovascular health. Current evidence suggests that the nutritional and socio-economic benefits of consuming these products should be weighed against the low-level and still insufficiently quantified risks associated with dietary exposure to MPs.

ONE HEALTH SURVEILLANCE AND GOVERNANCE SYSTEM

Plastic pollution has interconnected impacts across ecological matrices, wildlife populations, and human

public health. To respond effectively to this escalating global crisis, governance structures must transition from theoretical conceptualization to rigorous implementation science-the systematic translation of empirical evidence into realistic, cross-sectoral initiatives, regulatory frameworks, and operational practices.

Integrated Cross-Sectoral Surveillance

An operational One Health paradigm requires continuous and coordinated surveillance across environmental health, animal production, food safety and public health domains. Historically, the environmental domain has received disproportionately low priority within the One Health triad. However, ecosystems serve as the ultimate primary pathway, sink and reservoir for MPs and NPs^[34]. The formal incorporation of the United Nations Environment Programme (UNEP) into the Quadripartite One Health coalition underscores the need to integrate environmental monitoring into One Health surveillance. Continuous monitoring of aquatic bodies, terrestrial soils, air, municipal wastewater streams and agricultural sludge is vital for establishing responsive early-warning networks^[34,61].

Domesticated livestock and wild populations act as sensitive environmental sentinels for anthropogenic contamination^[24]. Long-term monitoring of companion animals and livestock exposed to pastures, commercial feed and agricultural water supplies can provide early-warning indicators of environmental contamination. Tracking these animal cohorts allows researchers to map MP bioaccumulation patterns and systemic toxicities before clinical manifestations become evident in human populations^[24,91].

Surveillance must also encompass the entirety of the global food supply chain to generate accurate dietary exposure assessments and support advanced human biomonitoring strategies, including the screening of human blood, placental tissues and fecal matter^[91]. Standardized and validated analytical techniques are needed across public health and veterinary laboratories to ensure cross-domain data comparability, harmonized reporting and data interoperability.

PILLARS OF A ONE HEALTH GOVERNANCE FRAMEWORK

Human medicine, veterinary sciences and environmental management have historically operated separately. This fragmentation creates structural silos that impede multi-directional data exchange and limit institutional coordination^[24]. Overcoming these barriers requires the formalization of multi-sectoral coordination mechanisms, such as dedicated inter-ministerial task forces or specialized One Health Secretariats, to

explicitly delineate organizational roles, institutionalize accountability and cultivate cross-stakeholder trust ^[94]. Environmental, zoonotic and toxicological surveillance networks must be technologically integrated. Establishing unified, secure data-sharing infrastructure enables real-time epidemiological analysis by linking environmental mass spectrometry data, animal necropsy records and human clinical admissions. This cross-referencing facilitates coordinated and targeted interventions during contamination events or periods of elevated exposure ^[95].

Cultivating a competent One Health workforce requires unified, multidisciplinary educational pathways. Joint training frameworks, such as cross-disciplinary Field Epidemiology Training Programmers (FETPs) that bring together medical clinicians, veterinary pathologists and environmental toxicologists, are essential. These initiatives can reduce communication barriers, align scientific terminology and support collaborative problem-solving from the outset ^[96].

Collaborative governance is heavily constrained by an ongoing deficiency in dedicated financial resources; historical funding streams have remained highly reactive and crisis-driven ^[34,97]. Operationalizing a resilient One Health architecture demands pooled, non-siloed and sustainable budget allocations. Secure funding provides separate state departments with the systemic motivation to collaborate transparently rather than compete for limited, isolated financial resources.

COMPREHENSIVE LEGISLATION AND MITIGATION POLICIES

Ultimately, cross-sectoral surveillance data must be directly leveraged to drive proactive, binding public policy. Robust national and international legislation is required to mitigate MP pollution at its source.

The One Health Mitigation Strategy

Transitioning to an integrated, systems-based framework requires a multi-tiered policy approach. National policies should promote sustainable waste management strategies grounded in circular economy principles, including Refuse, Rethink, Reduce, Reuse and Recycle ^[98].

Legislation should support the gradual phase-out of non-essential single-use plastics, restrict intentional MP additives in commercial goods and encourage the development of marine-degradable bioplastics. Governance frameworks should also prioritize targeted funding for innovative engineering practices, including microbially mediated plastic bioremediation and advanced wastewater filtration technologies capable of capturing sub-micron particles.

By shifting away from isolated food-safety testing and toward this integrated systems approach, global governance can link environmental source tracking, animal sentinel data and human biomonitoring to support sustainable, cross-sectoral legislative reform ^[91,97].

CONCLUSION

This review shows that MPs and NPs represent an emerging One Health concern at the intersection of environmental contamination, animal production and human dietary exposure. The evidence is strongest for environmental contamination, aquatic food chains and the biological plausibility of particle translocation. However, the evidence remains more limited for terrestrial ADFs, particularly meat, milk and eggs. This gap is important because these products occupy a central place in human nutrition and may also serve as pathways for environmental contaminants.

Current toxicological studies indicate that MPs and NPs can contribute to oxidative stress, inflammation, microbiome disruption, reproductive effects and multi-organ toxicity under experimental conditions. However, many of these studies rely on high exposure concentrations, pristine polymer particles and laboratory models that do not fully represent real-world dietary exposure. At present, there is no longitudinal epidemiological evidence establishing a causal relationship between MP exposure from ADFs and specific human diseases.

Future research should therefore prioritize standardized analytical protocols, comparable reporting units, reliable detection of NPs, and contamination data from terrestrial ADFs. Particular attention should be given to the effects of food processing, post-harvest contamination, animal feed, and the transfer of MPs from environmental matrices into edible tissues and secondary products. Prospective cohort studies and integrated biomonitoring will be necessary to clarify whether dietary exposure from ADFs has measurable long-term health consequences.

A balanced One Health approach is essential. MP and NP contamination should be addressed as a genuine food-safety and environmental health concern, but the current evidence does not justify overstating the risk or undermining the established nutritional value of ADFs. The most appropriate path forward is integrated surveillance linking environmental monitoring, animal sentinels, food-chain testing and human biomonitoring. Such an approach can support evidence-based regulation while avoiding unnecessary alarm.

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