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Plasticopathology: Multi-Organ Damage and One Health Implications of Micro- and Nanoplastics

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ABSTRACT

Microplastics (MPs) and nanoplastics (NPs) have emerged as pervasive environmental contaminants with increasing evidence of pathological consequences across biological systems. This study synthesizes current findings on histopathological alterations induced by MPs and NPs in multiple organ systems and animal species, highlighting their relevance to human health within a One Health framework. Analysis of recent experimental and field studies (2019-2025) revealed consistent lesions across the gastrointestinal, hepatobiliary, renal, reproductive, nervous, respiratory, cardiovascular, immune, and integumentary systems. Common pathological patterns include epithelial erosion, inflammatory infiltration, oxidative stress, cellular degeneration, and fibrosis, with severity often correlated to particle size, exposure route, and duration. The liver, kidney, and reproductive organs emerged as major targets of systemic toxicity, while translocation of particles across biological barriers underscores potential for multi-organ involvement. Evidence from animal models suggests trophic transfer of plastics through the food chain, posing direct and indirect risks to human health. Despite significant progress, knowledge gaps persist due to limited chronic exposure data, underrepresentation of veterinary and wildlife models, and lack of standardized exposure and detection methods. Hence, integration of omics technologies and exploring emerging areas such as transgenerational and epigenetic effects are essential for advancing mechanistic understanding. Overall, this review emphasizes the pathological continuum linking animal and human health impacts of plastic pollution and calls for coordinated global efforts-anchored in One Health principles to mitigate exposure, enhance surveillance, and inform evidence-based risk assessment and policy action.

KEYWORDS

Microplastics, nanoplastics, pathology, animal models, one health

INTRODUCTION

Microplastics (MPs) and nanoplastics (NPs) are fragments of synthetic polymers that arise either as intentionally manufactured small particles (primary MPs/NPs) or through the environmental breakdown of larger plastic debris (secondary MPs/NPs) by mechanical, chemical, and photolytic weathering. Microplastics are conventionally defined as plastic particles smaller than 5 mm, while particles in the sub-micrometre range (commonly ≤1 µm) are generally referred to as NPs. However, the field continues to debate exact lower limits for NPs and emphasises that particle size, shape, and surface chemistry all of which affect biological interactions span a continuum rather than discrete classes[1].

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The environmental burden of MPs/NPs is now global and multi-compartmental as particles have been detected throughout marine, freshwater and terrestrial ecosystems[1]. Also, studies have shown their presence in the atmosphere and in remote environments[2,3]. The MPs/NPs are partitioned among water, sediments and soils, where they can accumulate or be transported through hydrological and atmospheric pathways. However, their environmental ubiquity is driven by very large global plastic production, inefficient waste management, and the persistent nature of most polymer types. Hence, these widespread environmental reservoirs create continuous exposure sources for wildlife, livestock and laboratory animals[1-4].

Animals encounter MPs/NPs through multiple, biologically plausible exposure routes[8]. Ingestion is the best-documented pathway as aquatic organisms ingest MPs directly from water or indirectly via contaminated prey, while terrestrial animals may consume MPs in feed, soil or water and gastrointestinal uptake has been repeatedly shown in experimental models. Inhalation of airborne micro- and nano-sized particles is increasingly recognized as an important route of exposure for birds, small mammals and humans, particularly in urban and indoor environments where fibres and dust accumulate. Dermal contact is less well quantified but may be relevant for aquatic species, amphibians, or when skin barriers are compromised. The relative importance of each route depends on species ecology, particle size and behaviour, and exposure context[2].

A pathology-based synthesis is urgently needed because pathological examination links exposure to measurable tissue and organ injury, reveals lesion patterns and cellular mechanisms, and provides the biological evidence required for risk assessment and for extrapolating animal findings to human health within a One Health framework. Pathology integrates histological, ultrastructural, and biomarker data to show where particles deposit, what cells and compartments are affected, and which mechanistic pathways (such as oxidative stress, inflammation, barrier disruption, apoptosis, and genotoxicity) are engaged; such integration is essential to interpret heterogenous experimental results, identify susceptible species and life stages, and prioritize research and policy responses[5].

Therefore, this review aims to summarise current evidence on exposure routes and the translocation (toxicokinetics) of MPs/NPs in animals; document pathological findings across species and organ systems; analyze the principal mechanisms of tissue injury implicated by experimental and observational studies; and identify critical knowledge gaps and future research directions that will improve pathological assessment, inform risk evaluation for animal and human health, and guide One Health mitigation strategies.

SOURCES AND ROUTES OF EXPOSURE

Primary and secondary sources of MPs/NPs: Microplastics and nanoplastics originate from both primary and secondary sources. Primary sources refer to purposely manufactured small plastic particles, such as microbeads used in cosmetics, exfoliants, or engineered nanoparticles for industrial applications (e.g., in coatings or drug delivery systems)[5]. Secondary sources arise from the fragmentation, abrasion, and weathering of larger plastic items (e.g., bottles, films, fishing nets, packaging) under environmental forces such as UV irradiation, mechanical shear, and chemical degradation. Over time, larger macroplastics shed smaller fragments, which further degrade to micro- and nano-scales[5]. Several reviews have emphasized that a major proportion of environmental MPs/NPs derive from degradation of consumer and packaging plastics, textiles, tires, and plastic waste in terrestrial and aquatic settings[6]. Moreover, synthetic fibers shed from textiles during washing, abrasion of vehicle tires, and wear of polymer coatings are significant contributors to microplastic release into the environment. Sewage sludge or biosolids used in agriculture further act as conduits: Microplastics concentrated during wastewater treatment become applied to soils, releasing MPs into terrestrial environments[5].

Environmental distribution: Once released, MPs/NPs disperse widely across environmental compartments. In aquatic systems, plastics enter via runoff, riverine transport, wastewater effluents, and direct dumping; they distribute through water columns, settle into sediments, and may be resuspended[5]. The heterogeneity in density, shape, and biofouling influences their vertical position (floating, suspended, or settled) in aquatic systems. In terrestrial soils, MPs accumulate from atmospheric deposition, irrigation with treated effluent, sludge application, plastic mulching, litter, and degradation of *in situ* plastics[5]. Soils can act as reservoirs and sources for groundwater and runoff transport. In the atmosphere, microplastics appear as particles and fibers in airborne dust and can undergo long-distance transport. Ambient air studies report microplastic concentrations in urban and remote settings, attributing much atmospheric MP burden to road dust, fiber shedding, wind erosion, and sea spray. The shape of particles, particularly fibers, affects their settling velocity and thus potential for long-range travel[5].

Routes of exposure in animals: Animals are exposed to MPs/NPs via multiple routes, the predominance of which depends on species ecology, habitat, and particle characteristics. Ingestion constitutes the most common route of exposure. Aquatic organisms ingest MPs directly from the water column or indirectly via contaminated prey (zooplankton, benthos). Terrestrial animals ingest MPs via contaminated feed, drinking water, soil ingestion (geophagy), or consumption of prey that have accumulated particles. In several experimental animal studies MPs are administered per os (e.g. in food or water) to mimic natural ingestion exposure[5]. Also, the inhalation of airborne particles is increasingly recognized, especially in terrestrial and aerial species. Microplastics and fibers suspended in ambient air or dust can deposit in respiratory tracts, especially in animals inhabiting indoor or urban environments. The presence of MPs in atmospheric dust, deposition layers and aerosols supports inhalation as a realistic exposure route[2].

Furthermore, dermal (contact) absorption or adhesion is less well documented but may contribute, particularly in aquatic or amphibious species whose skin is in direct contact with plastic-laden water or substrates. In such settings, MPs and NPs might adhere to or penetrate mucous or partially permeable integument, especially if skin integrity is compromised. The significance of this route likely depends on particle size (smaller NPs being more capable of permeation) and duration of contact[6-8].

Bioaccumulation and trophic transfer across food chains: After entry into organisms, MPs/NPs may accumulate (bioaccumulate) in tissues, especially if elimination is slow, and can be transferred along food chains (trophic transfer). Evidence supports that many organisms exhibit internal MP burdens exceeding ambient concentrations (i.e. bioaccumulation) across multiple trophic levels. A systematic review of marine species found that MP bioaccumulation occurs within trophic levels, though clear biomagnification (increased concentration at higher trophic levels) remains unproven in field settings. Laboratory studies, however, have documented trophic transfer from prey to predator (e.g., microplastics from copepods to jellyfish)[7]. In freshwater contexts, evidence is more limited and ambiguous, and more data are needed to confirm transfer and accumulation patterns in terrestrial food webs[8]. A mass-balance modeling study in coastal systems suggested that trophic transfer might contribute to redistribution of MPs in food webs, though with caution about unrealistic exposure assumptions[9]. Overall, while bioaccumulation is well supported, the extent to which plastics or associated chemical additives biomagnify in natural systems remains an active area of investigation (Table 1).

Table 1: Representative studies on microplastics/nanoplastics exposure and bioaccumulation

Species/model	Exposure route	Particle type and size	Bioaccumulation/trophic transfer (summary)	References
General (multiple taxa)	Dermal, inhalation, ingestion (review)	Polystyrene nanoplastics (focus)	Review summarized evidence for penetration via skin, respiratory and digestive tracts and subsequent accumulation	Gupta *et al.*[2]
Farm chickens (Gallus gallus domesticus)	Dietary (feed) ingestion	Environmental MPs (various sizes recovered from crop/gizzard)	First reports quantifying MPs in crop and gizzard confirms dietary exposure and internal presence in poultry	Bilal *et al.*[3]
BALB/c mice	Oral gavage (oral ingestion)	Polystyrene particles: 50, 100 and 500 nm	Size-dependent biodistribution shown; smaller NPs (50-100 nm) reached multiple organs, indicating systemic translocation and tissue accumulation	Du *et al.*[4]
Copepods (Tigriopus)→Jellyfish (Aurelia)	Trophic transfer (indirect ingestion)	Polyethylene microbeads (environmental MP)	Demonstrated trophic transfer from contaminated copepods to jellyfish (experimental)	Costa *et al.*[7]
Freshwater biota (review across taxa)	Waterborne ingestion, trophic interactions	Varied MPs reported across studies	Comprehensive review showing bioaccumulation potential in freshwater species; trophic transfer reported experimentally but field biomagnification remains uncertain	Bhatt and Chauhan[8]
Rodent pregnancy models (maternal exposure)	Pulmonary exposure (inhalation)	Nanopolystyrene (nanoplastics)	Demonstrated lung-to-placenta and fetal tissue translocation of nanoplastics after maternal inhalation	Fournier *et al.*[10]

TOXICOKINETICS OF MICROPLASTICS AND NANOPLASTICS

Microplastics and nanoplastics exhibit size-dependent absorption, distribution, metabolism, and excretion (ADME) behaviors that critically determine their organ targeting and pathological potential[11]. An experimental mammalian study showed that smaller particles particularly those in the nanoplastic range are more readily taken up across epithelial barriers (intestinal mucosa and alveolar epithelium), enter the systemic circulation or lymphatics, and distribute to distal organs including liver, kidney, spleen, and brain.

Whereas larger microplastics tend to be retained in the gastrointestinal tract or cleared in faeces[2]. In a controlled *in vivo* biodistribution study, fluorescent or radiolabelled polystyrene particles demonstrated that 50-00 nm particles penetrate tissues and accumulate in multiple organs after oral or inhalation exposure[12]. While imaging (e.g., PET tracing) of inhaled micro-/nanoplastics confirms pulmonary deposition with subsequent translocation to extrapulmonary sites in rodent models[12]. These experimental data, together with systematic reviews, indicate that while elimination via faecal and, to a lesser extent, renal routes occurs, significant tissue persistence and organ retention can follow repeated or high-dose exposures a toxicokinetic profile that underpins chronic tissue exposure and risk[2,12].

MECHANISMS OF TOXICITY AND PATHOGENESIS

Multiple, often interacting, mechanisms have been implicated in micro- and nanoplastic toxicity across cell types and species. The dominant pathways reported include oxidative stress (overproduction of reactive oxygen species and consequent lipid, protein and DNA oxidation), activation of innate inflammatory cascades (local cytokine release, immune cell recruitment and inflammasome activation), disruption of epithelial and endothelial barrier integrity (tight junction alteration and increased permeability), induction of programmed cell death pathways (apoptosis and dysregulated autophagy), endocrine perturbation through adsorbed or leached additives, and direct or indirect genotoxic effects

detectable by comet and micronucleus assays[13]. The severity and combination of these mechanistic outcomes vary with particle physicochemistry (size, shape, polymer type, surface charge and weathering state), exposure route, and the biological context (cell type, species, developmental stage)[14]. Moreover, studies now show that NPs elicit stronger systemic and cellular responses than larger MPs at comparable mass doses. Collectively, these mechanistic insights explain how retained particles and their associated chemical burdens produce the cellular and tissue-level injuries that are subsequently observed histologically across organ systems[14].

PATHOLOGICAL FINDINGS ACROSS ORGAN SYSTEMS

Gastrointestinal system: Experimental and field studies have consistently identified the gastrointestinal (GI) tract as a primary site of MP/NP interaction and injury. Histopathological alterations reported across fish, rodents, poultry and mammalian models include mucosal irritation, epithelial erosion, villus shortening or atrophy, goblet-cell depletion, and infiltration of inflammatory cells in the lamina propria[15]. These structural changes commonly coincide with functional disturbances such as increased intestinal permeability and microbiota dysbiosis. In addition to direct physical abrasion, MPs/NPs can provoke oxidative stress and local cytokine upregulation in intestinal tissues, which exacerbates epithelial injury and may impair nutrient absorption shown in Table 2.

In mice, Sofield *et al.*[16] observed microplastic-induced inflammation manifested as edema, vacuolization, crypt depth increases, villi damage, and splitting of enterocytes in mouse models. These histopathological changes also included alterations in mucus-producing goblet cells and mucus secretion, which are crucial for gut barrier function and host immunity. Further study by Zhou *et al.*[17] documented that exposure to MPs and NPs led to oxidative stress, inflammation, and apoptosis in the GI tissues, which disrupted intestinal barrier function and promoted colonization by pathogens such as *Helicobacter pylori*.

Hsu *et al.*[18] demonstrated that polystyrene NPs affected specific bacterial taxa by interfering with extracellular vesicle-mediated communication and mucin expression in the gut, ultimately impairing the intestinal barrier integrity. This disruption of host-microbiota interplay by nanoplastics underlines a molecular pathway for NP-induced gut injury and microbiome dysbiosis. In a zebrafish model, Abdul Rehman *et al.*[19] found significant changes in gut microbiota composition and function alongside evidence of intestinal inflammation following chronic exposure to carboxylated polystyrene NPs. This study added to the evidence that nano-sized plastics can induce toxic effects in the digestive system through microbiome alteration and inflammation, thus supporting findings in mammalian models that indicate gut barrier weakening and immune dysfunction.

Hepatobiliary system: The liver is a frequent deposition site for systemically translocated MPs/NPs and has shown a spectrum of histopathological responses. Reported lesions include hepatocellular degeneration and vacuolation, lipid accumulation (steatosis), Kupffer cell activation and aggregation, inflammatory cell infiltration, and, in some models, early fibrotic changes driven by macrophage activation and stellate-cell signaling. Wang *et al.*[20] established that polystyrene microplastics induce macrophage extracellular trap formation, which, via ROS/TGF-β/Smad2/3 signaling pathways, contributes to liver fibrotic injury. The lesions documented by Fan *et al.*[21] were mitochondrial ROS-dependent macrophage necroptosis, macrophage-hepatocyte crosstalk driving hepatocyte damage, and elevated liver injury markers. Yilmaz *et al.*[22] demonstrated in mice that dietary microplastics combined with a high-fat diet exacerbate liver damage with inflammatory cell infiltration, ballooning degeneration, and steatosis progression to metabolic-associated steatohepatitis (MASH). Han *et al.*[23] reported that chronic NP exposure aggravated lipotoxic hepatocellular injury and fibrosis in a mouse model of MASH. Nanoplastics promoted hepatocyte Sonic Hedgehog (SHH) production, which activated hepatic stellate cells (HSCs) and macrophage-driven fibrotic signaling, thereby worsening liver fibrosis and disease progression.

Renal system: The kidney pathology documented following MP/NP exposure includes tubular epithelial degeneration and necrosis, glomerular alterations (including podocyte injury and mesangial changes), interstitial inflammation, and molecular signatures of oxidative stress and apoptosis. Studies have reported corresponding functional disturbances such as elevated serum urea and creatinine or electrolyte imbalances following subacute or chronic exposures[24,25]. Also, organoid and *in vivo* models indicated that nephrogenesis and tubular integrity may be particularly vulnerable to MP[26]. These renal effects appear to be mediated by a combination of direct particle accumulation, inflammation, and oxidative injury.

Evidence from *in vivo* animal models demonstrates that MPs accumulate in kidney tissues, where they induce oxidative stress by increasing reactive oxygen species (ROS) production, leading to mitochondrial dysfunction and cellular damage. For instance, Huang *et al*.[24] reported that MPs trigger oxidative stress, inflammation, apoptosis, and endoplasmic reticulum stress in renal cells, thus, implicating pathways such as NF-κB and inflammasomes like NLRP3 in driving kidney inflammation and injury. Also, the review by Aditya *et al*.[25] showed that exposure to polystyrene MPs resulted in histological changes in renal morphology, impaired renal function biomarkers, and activation of pyroptosis pathways involving Gasdermin D and caspase-1. A study by Lee *et al*.[27] confirmed that nanoplastics can damage glomeruli and renal tubules, causing podocyte degeneration and tubular necrosis via multifactorial processes, including oxidative stress and fibrosis. Furthermore, research by Chen *et al*.[28] showed that airborne NPs can deposit in kidneys after inhalation and exacerbate nephrotoxicity through mitochondrial and inflammatory pathway disruptions, ultimately contributing to renal dysfunction and fibrosis.

Reproductive system: Microplastic/nanoplastic exposure has been associated with adverse reproductive pathology, which manifests as reduced gametogenesis, gonadal degeneration, disrupted folliculogenesis, testicular atrophy, and altered reproductive hormone profiles[29]. Lesions reported by Jin *et al*.[30] in male mice were testicular histological alterations, including tubular degeneration/atrophy, germ-cell loss, decreased sperm viability, reduced testosterone, and altered steroidogenic enzyme expression. Experimental rodent and fish studies showed decreased sperm count and motility, structural damage to seminiferous tubules and ovarian follicles, and evidence of endocrine perturbation. Also, some studies have reported developmental and transgenerational effects offspring of exposed parents exhibiting reproductive and developmental abnormalities. This suggested that germ-line or early life exposures may have lasting, multigenerational consequences[29].

The study by Bai *et al*.[31] revealed that female mice that received daily gavage with polystyrene MPs during gestation (GDs 0-14) had embryonic growth retardation, reduced maternal weight gain, and impaired reproductive performance. Examination of placentas revealed inflammatory cell infiltration, downregulation of tight junction proteins, and induction of Endoplasmic Reticulum (ER) stress via the GRP78/IRE1α/JNK axis, leading to increased apoptosis in the placental tissue[31]. These findings illustrate direct damage to the placental barrier, disruption of maternal-fetal exchange, and emphasise the potential for microplastics to impair gestational outcomes.

Nervous system: Microplastics/nanoplastics have been shown to induce notable neurotoxic effects across the nervous system, raising significant health concerns. Studies reveal that MPs/NPs can cross the Blood-Brain Barrier (BBB), leading to neuronal damage, synaptic dysfunction, and neuroinflammation. Yu *et al*.[32] demonstrated that exposure to MPs/NPs in *Caenorhabditis elegans* caused impairments in neural development and behavior, including disrupted neurotransmitter levels such as glutamate, serotonin, and GABA. This was associated with altered expression of synaptophysin and CREB, key proteins regulating synaptic repair and memory formation, pointing to potential long-term cognitive deficits. In Zebrafish embryos exposed to polystyrene NPs, Zhou *et al*.[33] reported neurodevelopmental defects such as neuronal loss, axonal defects, apoptotic and developmental gene expression changes, leading to behavioral

impairments. The study by Nihart *et al.*[34] found MPs/NPs accumulate in human brains postmortem, thus suggesting persistent exposure and bioaccumulation within central nervous tissue. Hence, this accumulation may exacerbate protein aggregation and inflammatory responses in neural tissues, correlating with neurodegenerative disease development

Respiratory system: Evidence from inhalation exposure studies and occupational/ambient deposition assessments supports the respiratory tract as a credible target for MP/NP pathology. Inhaled particles can deposit throughout the airways and alveoli and elicit alveolitis characterized by epithelial injury, inflammatory cell infiltration. In some experimental models, there was progression to interstitial fibrosis with collagen deposition, particularly after chronic exposures to respirable-sized particles[35]. These respiratory lesions reflect a combination of particle-induced oxidative stress, persistent macrophage activation, and impaired clearance mechanisms. Yang *et al.*[36] reported airway/alveolar inflammation (alveolitis), macrophage infiltration, elevated BALF cytokines, epithelial injury in mice following exposure to NPs via inhalation; while chronic exposures produced COPD-like lesions and fibrosis in some models. Lesion reported by Danso *et al.*[37] included increased inflammatory cells in BALF, upregulated TLR/NF-κB signaling, lung inflammation, and injury.

Cardiovascular system: Evidence indicates that systemically circulating MPs/NPs can directly injure the vascular compartment, producing endothelial structural damage and provoking pro-coagulant changes. A controlled *in vivo* study in mice exposed to polystyrene nanoplastics (pristine and surface-functionalized forms) reported histological and ultrastructural endothelial injury in small arteries, an inflammatory vascular infiltrate, and a shift toward a prethrombotic state; molecular analyses implicated activation of the JAK1/STAT3/tissue-factor axis as a plausible mechanistic link between particle exposure and coagulation dysfunction[38]. Complementing these primary data, Lee *et al.*[39] synthesized *in vitro* findings of nanoplastic-induced endothelial leakiness (reduced tight-junction integrity, increased vascular permeability) and oxidative-inflammatory activation that together provide a plausible pathway linking chronic MP/NP exposure to endothelial dysfunction and atherogenic processes. While cardiovascular histopathology in higher-order mammals (and clinical validation in humans) remains underdeveloped, these mammalian and *in vitro* data justify including cardiovascular endpoints (endothelial injury, perivascular inflammation, coagulation markers) in comparative pathology assessments of MPs/NPs.

Immune and haematopoietic system: Microplastics/nanoplastics modulate immune and haematopoietic tissues both structurally and functionally. The reported effects include splenic architectural changes, altered leukocyte distributions, lymphoid depletion or hyperplasia depending on exposure context, and dysregulated cytokine profiles indicative of either immunosuppression or inappropriate hyperactivation[40]. *In vitro* and *in vivo* studies have demonstrated particle uptake by macrophages and dendritic cells with consequent inflammasome activation, altered antigen-presentation capacity, and changes in haematopoiesis that can translate into compromised host defence or chronic inflammatory states[41].

Integumentary system: Dermal pathology due to MP/NP exposure is less frequently reported than internal organ lesions but includes skin irritation, epidermal inflammation, and secondary inflammatory responses where prolonged contact or abrasive particles occur. Although intact mammalian skin is a robust barrier to particulate entry, experimental and field data indicate that integumentary pathology is credible under environmental or biological circumstances that compromise barrier integrity. In aquatic species, especially teleost fish, exposure studies demonstrate clear epidermal and gill lesions lamellar fusion, epithelial lifting, mucous cell hyperplasia, and deposition of particles within lamellar structures consistent with mechanical abrasion and inflammatory responses. In mammals and human-relevant models, a review of *ex vivo* and *in vitro* studies showed that nanoscale particles (particularly <200 nm) can penetrate or be taken up by keratinocytes and fibroblasts, and that penetration is enhanced when the stratum corneum is compromised or when transappendageal routes (hair follicles, sweat glands)[42]. Thus,

while classical dermal histopathology from environmental MP/NP exposure in terrestrial mammals is limited, the fish histopathology and mechanistic skin-penetration evidence support the integumentary system as a plausible, if under-studied, target for pathology in contaminated environments.

Multi-organ system: In *Cirrhinus mrigala* fingerlings, dietary exposure to polystyrene microplastics over prolonged periods resulted in negative growth and digestive performance effects, in addition to histopathological alterations within the digestive tract[43]. These showed that exposure adversely affected the gastrointestinal morphology and hepatic function in juvenile fish. Hence, the results emphasise that aquatic species remain vulnerable to MP-mediated damage in organs involved in nutrient absorption and metabolism.

In an acute exposure model in mice, Chen *et al.*[44] provided further insight into early tissue damage and distribution following a single high-dose gavage of fluorescent 200 nm microplastics. Fluorescence imaging detected particles not only in the stomach and caecum, but also in the lungs, liver, kidney, spleen and brain. Histological examination showed mild congestion in the liver and lungs and epithelial shedding in the stomach and caecum, indicating that particles translocated beyond the gut to multiple organs[44]. These observations highlight that even a single bolus of nanoplastics can cross the gut barrier, enter systemic circulation, and evoke early tissue responses in multiple organs.

A study by Wang *et al.*[45] developed a lactational exposure model in which maternal mice received polystyrene microplastics (PS-MPs) via daily gavage from day 1 to day 21 after giving birth. Their findings indicated that PS-MPs accumulated predominantly in the faeces, colon, liver, and mammary tissue. Additional investigations revealed that this exposure disrupted both the gut and blood–milk barriers, leading to inflammatory responses and structural damage in the liver, intestine, and mammary glands. Metabolomic and metagenomic analyses revealed disturbed bile acid metabolism in the liver and major changes in gut microbiota, which disrupted the intestine-liver connection. Interestingly, when gut bacteria were removed using antibiotics or faecal microbiota transplantation, inflammation decreased, and barrier function improved. These results suggest that PS-MPs worsen intestine-liver axis disorders by causing colon damage, disturbing gut balance, and altering liver metabolism. They also move through this axis to affect mammary tissues, thus, revealing a possible gut-liver-mammary pathway for PS-MP toxicity in mice[45]. Lee *et al.*[46] reported that exposure to microplastics primarily triggers widespread degenerative alterations including apoptosis, inflammatory damage, and fibrotic changes across multiple organ systems, with the nervous system being a notable exception. Their findings also indicated that these tissue changes were largely driven by MP-induced inflammation, which subsequently progressed to fibrosis and functional decline.

Table 2: Representative studies showing histopathological findings of microplastics and nanoplastics across organ systems and animal species

Organ system	Species/model	Particle type and size	Key histopathological findings	References
Gastrointestinal and hepatic	C57BL/6 mice (DSS colitis model)	Polystyrene MPs, 5 µm (oral gavage)	Exacerbation of DSS colitis: shortened colon, aggravated histopathological damage, reduced mucus secretion, increased permeability; associated hepatic inflammatory infiltration and risk of secondary liver injury	Lou *et al.*[47]
Hepatobiliary	Mouse (*in vivo*) and macrophage/ hepatocyte models	Polystyrene nanoplastics (20 nm PSNPs) and 1 µm PSMPs (acute exposure)	Acute liver injury: mitochondrial ROS-dependent macrophage necroptosis, macrophage-hepatocyte crosstalk driving hepatocyte damage and elevated liver injury markers	Fan *et al.*[21]

Table 2: Continue

Renal	Juvenile rats (weanlings)	Polystyrene MPs (~1000 nm), oral 28 day	Nephrotoxicity: histological renal lesions, increased BUN/creatinine, ER stress, oxidative stress, inflammation, apoptosis; amelioration by NAC/ER-stress blocker	Wang *et al.*[20]
Reproductive (male testis)	Male mice (C57BL/6)	Polystyrene MPs, 0.5-10 μm (chronic drinking-water exposure)	Testicular histological alterations: tubular degeneration/atrophy, germ-cell loss, decreased sperm viability, reduced testosterone and altered steroidogenic enzyme expression (LH/LHR/cAMP/PKA/ StARpathway)	Jin *et al.*[30]
Reproductive	Pregnant mice	Polystyrene MPs gavage at 0, 25, 50, 100 mg/kg body weight during gestational days 0-14	Embryonic growth retardation; placental morphological changes; dysfunction via GRP78/IRE1α/JNK axis induced apoptosis and ER stress in placenta	Bai *et al.*[31]
Nervous system (development)	Zebrafish embryos (*Danio rerio*)	Polystyrene nanoplastics (various sizes; environment-relevant)	Neurodevelopmental effects: Neuronal loss, axonal defects, apoptotic and developmental gene expression changes→ behavioral impairments	Zhou *et al.*[33]
Respiratory/ pulmonary	C57BL/6 mice (inhalation/instillation)	Polystyrene nanoplastics or microplastic fragments (inhalation/intratracheal)	Airway/alveolar inflammation (alveolitis), macrophage infiltration, elevated BALF cytokines, epithelial injury; chronic exposures produced COPD-like lesions and fibrosis in some models	Yang *et al.*[36]
Pulmonary (instillation)	C57BL/6 mice (intratracheal instillation)	PP, PS, PE microplastic fragments (5 mg/kg intratracheal, 14 day)	Increased inflammatory cells in BALF, upregulated TLR/NF-κB signaling, lung inflammation and injury; PS showed marked responses	Danso *et al.*[37]
Cardiovascular	Mice (*in vivo*)	Polystyrene nanoplastics (PS, PS-NH , PS-COOH); i.v./oral/inhalation paradigms (experimental)	Structural damage to vascular endothelial cells, vascular inflammation, coagulation dysfunction and a prethrombotic state; JAK1/STAT3/tissue-factor signaling implicated	Wang *et al.*[38]
Cardiovascular	Rats (*in vivo*)	Polystyrene microplastics (PS-MPs), oral gavage (0.5-50 mg/kg/day for 90 days)	Metabolic perturbation and markers of myocardial injury; dose-dependent increases in myocardial enzyme levels, oxidative stress and inflammation in myocardial and aortic tissues; metabolomics showed dysregulated lipid metabolism and pathways linked to cardiac injury	Song *et al.*[48]
Immune/ hematopoietic	Mice (*in vivo*)	Polystyrene MPs (oral/parenteral exposures)	Spleen histopathology, lymphoid modulation, altered cytokine profiles (e.g., reduced S100A8), immune suppression or dysregulated immune activation depending on model	Wang *et al.*[40]

Table 2: Continue

Integumentary (gill/skin-fish)	Nile tilapia (*Oreochromis niloticus*), early juveniles (15 d exposures)	Environmental MPs (mixed sizes; aqueous exposure, 1-100 mg/L)	Gill lamellar fusion and degeneration, epithelial lifting, mucous cell hyperplasia and deposition of MPs between lamellae; epidermal inflammation; systemic lesions in liver, kidney, intestine documented by whole-body histology	Hamed *et al.*[49]
Integumentary/ dermal penetration (mechanistic evidence)	*Ex vivo* human/animal skin models and *in vitro* skin cells (review)	Nanoplastics and nanoparticles (<200 nm)	Systematic review and synthesis: Nanoscale particles can penetrate compromised skin or access via transappendageal routes; size (<200 nm), surface chemistry and skin condition are key determinants; supports plausibility of dermal uptake under certain conditions	Menichetti *et al.*[42]
Multi-organ	Fingerlings of *Cirrhinus mrigala* (fish)	Diets with graded concentrations (up to 2.5%) of PS-MPs over 90 days	Negative histopathological changes (unspecified but observed) together with reduced growth, digestibility, body composition, hematological alterations	Rashid *et al.*[43]
Multi-organ	Mice	200 nm fluorescent microplastics, single gavage at 200 mg/kg	Detected MP particles in multiple tissues (stomach, cecum, lung, kidney, liver, spleen, brain); mild congestion in liver and lungs; epithelial shedding in stomach and cecum	Chen *et al.*[44]

ONE HEALTH IMPLICATIONS

The pervasive presence of MPs and NPs across environmental matrices has profound One Health implications, emphasising the interconnectedness of human, animal, and ecosystem health. Experimental and field studies demonstrate that animals ranging from aquatic species to terrestrial livestock accumulate MPs/NPs in critical organs such as the liver, kidney, and reproductive tissues, leading to histopathological and physiological disruptions[30,47]. These animal findings serve as sentinel indicators of potential human health risks, as similar exposure routes (dietary, inhalational, dermal) occur in people. The bioaccumulation and trophic transfer of MPs through the food chain from contaminated water and plankton to fish, livestock, and ultimately humans raise concerns about chronic ingestion and long-term systemic effects[21]. Moreover, plastic-associated additives and adsorbed pollutants (e.g., heavy metals, persistent organic contaminants, antibiotic residues) may exacerbate zoonotic and toxicological risks, influencing antimicrobial resistance and endocrine disorders that cut across species boundaries[31,42].

From an environmental and ecological health perspective, the continuous deposition of plastic particulates alters soil and aquatic microbiota, disrupts ecosystem functions, and threatens biodiversity changes that indirectly affect food security and planetary health[45]. Within this One Health continuum, veterinarians and pathologists could play pivotal roles in surveillance, diagnosis, and risk assessment by characterizing tissue lesions, identifying sentinel species, and tracing exposure pathways between animals, humans, and the environment[44]. Their integrative expertise would support the development of bio-monitoring frameworks, regulatory thresholds, and mitigation strategies aimed at curbing plastic pollution and safeguarding cross-species health. Collaborative efforts among environmental scientists, medical professionals, and policymakers are therefore essential to translate animal data into actionable interventions that protect human populations and sustain ecological resilience[48,49].

KNOWLEDGE GAPS AND FUTURE DIRECTIONS

Despite growing evidence of the pathological effects of microplastics and nanoplastics, significant knowledge gaps remain that constrain comprehensive risk assessment. Most studies to date focus on acute or short-term exposures, leaving a critical void in understanding the consequences of chronic exposure and cumulative multi-organ effects across life stages and species. Also, there is an underrepresentation of veterinary and wildlife models, which limits the ecological and comparative pathology perspectives necessary for One Health assessments. The lack of standardized exposure protocols and detection methods including particle size characterization, quantification, and histological localization continues to hinder reproducibility and cross-study comparison. Future research must therefore adopt harmonized methodologies and leverage omics technologies such as toxicogenomics, proteomics, and metabolomics to elucidate molecular mechanisms and biomarkers of plastic-induced toxicity. Moreover, emerging areas like transgenerational inheritance, epigenetic regulation, and developmental programming deserve focused investigation to understand how plastic exposure influences health outcomes beyond directly exposed individuals and across generations.

CONCLUSION

The accumulating evidence demonstrates that MPs and NPs induce multi-organ histopathological alterations across animal species, with implications that extend to human and environmental health. These findings highlight the pervasive nature of plastic pollution as a shared biological threat, bridging ecosystems through the food chain and exposing both animals and humans to comparable risks. While the gastrointestinal, hepatic, renal, reproductive, and nervous systems appear most vulnerable, the broader One Health perspective emphasises the interconnectedness of these effects. Therefore, addressing the identified knowledge gaps through chronic exposure models, standardized detection methods, and advanced molecular approaches will be crucial for defining exposure thresholds and guiding effective global mitigation strategies.

SIGNIFICANCE STATEMENT

This study discovered consistent multi-organ histopathological injury patterns induced by microplastics and nanoplastics across diverse animal models, which can be beneficial for improving environmental health risk assessment and translational toxicology within a One Health framework. By linking particle characteristics with organ-specific pathology and systemic translocation, this study will help researchers to uncover the critical areas of chronic exposure, trophic transfer, and mechanistic pathways that many researchers were not able to explore. Thus, a new theory on plastic-driven pathological continuity across ecosystems may be arrived at.

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