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Micro-and Nanoplastic-Induced Biochemical Toxicity: Emerging Mechanisms and Health Risks Across Biological Systems

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ABSTRACT

Micro- and nanoplastics (MNPs) - particles derived from the fragmentation of larger plastic debris or manufactured intentionally at microscopic scales - are now ubiquitous across terrestrial, freshwater, marine, and atmospheric environments. Their small size, high surface area, and capacity to carry additives and sorbed pollutants enable them to interact with biological systems in ways that larger plastic debris cannot. This review synthesizes current understanding of the biochemical mechanisms underlying MNP toxicity across biological kingdoms, emphasizing oxidative stress, membrane and protein interactions, immune activation and inflammation, genotoxicity, endocrine disruption, and microbiome perturbation. We discuss routes of exposure and internalization, particle physicochemistry that modulates bioactivity, cross-species comparability of effects (plants, invertebrates, vertebrates, and humans), and methodological challenges in detection and hazard assessment. Gaps are highlighted: standardization of particle characterization and dose metrics, elucidation of chronic low-dose effects and mixture interactions, and development of mechanistic biomarkers translatable between models and humans. The review concludes with priority research directions and implications for risk assessment and public health policy, arguing that addressing MNP toxicity requires coordinated advances in analytical methods, experimental design, and cross-disciplinary communication.

Keywords: Microplastics, Nanoplastics, Oxidative stress, Endocrine disruption, Microbiome

INTRODUCTION

Plastic production and the environmental persistence of discarded materials have led to the emergence of a new class of particulate contaminants: microplastics (generally <5 mm) and nanoplastics (<1 μm, often defined operationally) [1]. While initially regarded as posing mainly physical hazards such as entanglement or gastrointestinal blockage, recent evidence indicates that these particles exert subtler but significant biochemical toxicity [2]. Their small size, large surface area, and diverse physicochemical characteristics allow them to interact intimately with biological structures, disrupting cellular functions. In addition, they serve as carriers for a variety of chemical additives, heavy metals, and microbial communities, further amplifying their toxic potential [3]. Unlike conventional soluble pollutants, micro- and nanoplastics (MNPs) combine the properties of particles with chemical complexity [4]. Size, shape, surface charge, and environmental conditioning influence their internalization and fate within organisms. Once ingested or inhaled, they can cross biological barriers, accumulate in tissues, and induce oxidative stress, inflammation, membrane perturbation, or even genotoxic effects [5]. Moreover, their ability to leach endocrine-disrupting chemicals and alter microbiomes adds another layer of health concern. This review synthesizes mechanistic insights from molecular, cellular, and organismal studies to illustrate how MNPs induce biochemical toxicity across plants, invertebrates, vertebrates, and humans. We highlight four core areas: (1) particle properties and exposure pathways shaping internal dose, (2) fundamental biochemical mechanisms of harm, (3) cross-kingdom responses and human health implications, and (4) methodological and policy challenges in detection, risk assessment, and regulation. Understanding these processes is essential for advancing effective mitigation strategies and safeguarding ecosystem and public health.

2. Sources, properties, and exposure pathways

2.1 Origins and compositional diversity

MNPs originate from primary sources (manufactured beads, industrial abrasives, some cosmetics, and drug carriers) and secondary fragmentation of larger plastic items (packaging, textiles, fishing gear) [6]. Polymer types include polyethylene (PE), polypropylene (PP), polystyrene (PS), polyvinyl chloride (PVC), polyethylene terephthalate (PET), and others. Additives - plasticizers (phthalates), flame retardants (brominated compounds), UV stabilizers, and dyes - vary by polymer and influence toxic potential [7].

2.2 Size, shape, and surface chemistry

Size governs penetration and cellular uptake: nanoplastics can cross biological barriers and be internalized by endocytosis or passive diffusion, whereas microplastics are often taken up by phagocytosis or remain within gastrointestinal tracts [8]. Shape (fibers, fragments, spheres) modifies mechanical interaction with tissues. Surface charge, oxidation state, and biofilm formation (eco-corona or protein corona in biological fluids) alter particle identity and reactivity in situ.

2.3 Environmental conditioning and co-contaminants

In the environment MNPs accumulate aged surfaces, oxidized functional groups, and sorbed pollutants (persistent organic pollutants, polycyclic aromatic hydrocarbons, heavy metals) and host microbial communities [9]. These acquired compounds can be transferred to organisms on contact or ingestion, complicating attribution of observed toxicity to the particle itself versus its chemical payload.

3. Cellular uptake, biodistribution, and persistence

Particle size and surface properties determine cellular internalization mechanisms: macropinocytosis and phagocytosis for larger particles; clathrin- and caveolin-mediated endocytosis and direct translocation for smaller nanoparticles [10]. Once internalized, MNPs can persist in endolysosomal compartments, interfere with organelle function, or translocate to the cytosol and other organelles (mitochondria, nucleus) depending on size and coating [11]. Biodistribution studies in model organisms show that nanoplastics can reach liver, kidney, brain, and reproductive tissues; microplastics often remain in the gut but can induce systemic responses through inflammatory signaling [12]. Clearance pathways (exocytosis, fecal elimination, renal excretion) are particle- and species-dependent and frequently inefficient, leading to accumulation with chronic exposure [13].

4. Core biochemical mechanisms of toxicity

4.1 Oxidative stress and redox imbalance

One of the most consistently observed responses to MNP exposure is increased production of reactive oxygen species (ROS) and perturbation of antioxidant defenses (reduced glutathione, altered activities of superoxide dismutase and catalase) [14]. ROS can arise via surface redox reactions on particles, leaching of redox-active additives or metals, mitochondrial dysfunction following internalization, and activation of immune cells that generate oxidative bursts. Oxidative stress links to lipid peroxidation, protein carbonylation, and DNA oxidation - early events in cellular damage cascades [15].

4.2 Membrane interactions and protein dysfunction

Direct contact between particles and cell membranes can disrupt membrane integrity, fluidity, and transporter function. Nanoplastics may insert into lipid bilayers or destabilize membranes via mechanical stress [16]. Surface chemistry influences binding to membrane proteins and receptors, potentially altering signaling cascades. Protein corona formation can mask or expose moieties that interact with receptors, leading to unintended receptor activation or inhibition [17].

4.3 Inflammation and immune activation

MNPs activate innate immune responses: phagocytes recognize particles as foreign, leading to cytokine release (IL-1β, TNF-α, IL-6) and recruitment of immune cells [18]. Chronic, unresolved inflammation is a central pathway linking MNP exposure to tissue damage, fibrosis, and altered organ function. In some models, inflammasome activation (e.g., NLRP3) has been implicated after nanoparticle exposure [19].

4.4 Genotoxicity and DNA damage

Evidence from cellular and in vivo studies points to DNA strand breaks, micronuclei formation, and chromosomal aberrations following exposure to certain MNPs and their associated chemicals. Mechanisms include direct physical interactions with DNA (less likely for larger particles), oxidative DNA lesions mediated by ROS, and indirect genotoxicity via inflammatory mediators [20].

4.5 Endocrine and developmental disruption

Plastic additives and sorbed chemicals (phthalates, bisphenols, flame retardants) are well-known endocrine disruptors. When transported by MNPs, these compounds can be delivered to sensitive tissues (gonads, developing embryos), perturbing hormone signaling, reproductive development, and neuroendocrine regulation [21]. There is growing concern about low-dose and nonmonotonic dose responses typical of endocrine disruptors.

4.6 Microbiome perturbation and secondary biochemical effects

MNPs can alter microbial communities in soil, water, and animal guts by providing substrates for biofilm formation, preferentially selecting for plastic-tolerant microbes, or adsorbing signaling molecules. Dysbiosis in the gut is tied to altered metabolite production (short-chain fatty acids, bile acids) that can have systemic biochemical consequences, including immune modulation and metabolic dysfunction [22].

5. Cross-kingdom effects: evidence and implications

5.1 Plants and agricultural systems

Root uptake of nanoplastics can occur via apoplastic and symplastic pathways; particles may alter root architecture, nutrient uptake, and photosynthetic efficiency. MNPs adsorbed to soil aggregates change water retention and nutrient cycling, indirectly influencing plant biochemistry and crop yield [23]. Translocation to edible tissues raises concerns about human dietary exposure.

5.2 Aquatic invertebrates and fish

Filter feeders and deposit feeders readily ingests microplastics, resulting in gut blockage, reduced feeding efficiency, and altered energy budgets. Biochemical endpoints include oxidative stress markers, disrupted ion regulation across gills, and impaired detoxification enzyme systems (e.g., cytochrome P450). Developmental abnormalities and reproductive impairment have been reported under chronic exposures [24].

5.3 Mammals and humans: routes and observed responses

Laboratory rodents exposed to nanoplastics show accumulation in liver, spleen, and brain, with correlated oxidative stress, inflammatory cytokine induction, and metabolic disruptions (lipid and glucose metabolism) [25]. Human evidence is emerging: microplastics have been detected in human stools and some tissue samples; however, causal links to disease remain to be rigorously established. Occupational and high-exposure groups (waste handlers, plastic manufacturing workers) may face elevated risk [26].

6. Biomarkers, detection, and methodological challenges

6.1 Analytical challenges

Accurate detection and quantification of nanoplastics in complex matrices are technically demanding [27] Distinguishing synthetic polymers from biological particulates and quantifying particle size, shape, concentration, and surface chemistry require high-resolution techniques (e.g., electron microscopy, Raman and FTIR spectroscopy, thermal analysis), often combined with labor-intensive sample preparation that risks contamination [28].

6.2 Dose metrics and exposure realism

Studies vary widely in particle types, sizes, surface treatments, and dose metrics (mass, number, surface area). Environmental concentrations are often low but chronic; laboratory studies frequently use high acute doses that may not reflect realistic exposure scenarios [29]. There is an urgent need for standardization of reference materials and adoption of dose metrics that reflect biologically relevant interactions (e.g., surface area or particle number).

6.3 Biomarkers of effect

Candidate biochemical biomarkers include oxidative stress markers (lipid peroxides, oxidized DNA bases), inflammatory cytokines, stress protein expression (heat shock proteins), and metabolomic signatures [30]. To be useful across species and for human risk assessment, biomarkers must be validated for sensitivity, specificity, and temporal dynamics following exposure.

7. Mixture effects and co-exposures

MNPs do not act in isolation in the environment. Their role as carriers for chemical contaminants and microbial pathogens can create additive or synergistic toxicities [31]. For instance, sorbed hydrophobic organics may desorb in the gut microenvironment, increasing internal chemical exposure. Similarly, plastic-associated microbes may include pathogens or antibiotic-resistance genes, presenting combined microbiological and biochemical risks [32]. Understanding mixture dynamics requires integrated experimental and modelling approaches.

9. Implications for risk assessment and policy

Current regulatory frameworks are not yet well suited to evaluate particulate contaminants that both carry chemicals and act as particles. Risk assessors will need frameworks that combine particle toxicology, additive chemical hazards, and exposure pathways (ingestion, inhalation, dermal) [33]. Precautionary measures include reducing primary microplastic release (e.g., industrial beads, textile fibers), improving waste management to limit fragmentation, and monitoring high-risk pathways (drinking water, seafood supply chains). Public health messaging should be evidence-based and balanced acknowledging uncertainties while promoting source reduction.

CONCLUSIONS

Micro- and nanoplastics present a multifaceted challenge at the intersection of materials science, environmental chemistry, toxicology, and public health. Biochemical toxicity arises from a tapestry of mechanisms - oxidative stress, membrane perturbation, inflammation, genotoxicity, endocrine disruption, and microbiome changes - modulated by particle physicochemistry and environmental conditioning. While experimental evidence indicates plausible pathways to harm across biological systems, translating findings to human health risk requires standardized methods, environmentally realistic exposure models, and robust biomarker validation. Coordinated

research and policy action are needed to close critical gaps and mitigate potential long-term impacts of MNPs on ecosystems and human well-being.

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