Human Exposure, Biological Fate, and Oxidative-Stress Mechanisms of Micro- and Nanoplastics: A Critical Review

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Abstract. Microplastics and nanoplastics are widespread environmental contaminants that can plausibly reach humans through food, drinking water, inhaled air, and, under more limited conditions, dermal or medical routes. Reports of plastic particles in human blood, placenta, lung, and vascular tissue have intensified health concern, but tissue detection, persistent accumulation, and causal disease are different evidentiary claims. This critical review separates external exposure, internal dose, biological fate, experimental mechanism, and human outcome evidence. It examines gastrointestinal and respiratory entry, barrier crossing, biomolecular-corona formation, cellular uptake, distribution and clearance, and the modifying roles of particle size, shape, polymer chemistry, weathering, additives, and adsorbed pollutants. The mechanistic synthesis focuses on oxidative stress: particle-associated and cellular reactive oxygen species, mitochondrial dysfunction, disturbance of glutathione and antioxidant enzymes, redox-sensitive Nrf2 and inflammatory signaling, macromolecular injury, and regulated cell-death responses reported in experimental models. These pathways form a biologically plausible network, but much of the evidence derives from pristine polystyrene spheres, short exposures, and concentrations that are difficult to relate to human internal dose. Human biomonitoring remains vulnerable to contamination, polymer misidentification, incomplete particle-size recovery, and inconsistent mass-, number-, or surface-based dose metrics. Observational associations, including cardiovascular findings, therefore warrant investigation without being interpreted as proof of causation. We propose an evidence chain from external exposure to internal and biologically effective dose, molecular response, and adverse outcome. Progress requires harmonized quality assurance, realistic weathered-particle mixtures, reference materials, toxicokinetic studies, longitudinal cohorts, and studies that link validated internal dose to temporally ordered clinical outcomes.

Keywords: Microplastics, nanoplastics, human exposure, toxicokinetics, oxidative stress, mitochondrial dysfunction.

1. Introduction

Plastic particles are generated intentionally at small sizes or through fragmentation, abrasion, weathering, and degradation of larger products. They occur in water, food, indoor and outdoor air, dust, and consumer environments. “Microplastic” is commonly used for particles below 5 mm, while the lower boundary and the definition of “nanoplastic” vary across fields and analytical methods. Rather than treating a disputed diameter threshold as a biological law, this review uses micro- and nanoplastics (MNPs) as an operational continuum and states size-dependent claims explicitly. Smaller particles often have higher surface area per unit mass and may access different biological routes, but size interacts with shape, chemistry, surface condition, and agglomeration.

The World Health Organization (WHO) concluded that dietary and inhalation exposure is plausible while highlighting substantial uncertainty in exposure measurement and health-risk characterization [1]. Subsequent reports of polymer-associated particles in human blood, placenta, and lung tissue [2–4] have established an urgent analytical question: what was detected, how

confidently was it identified, and what does its presence mean biologically? Detection can indicate exposure and possible translocation. It does not by itself establish persistence, concentration at a target site, molecular damage, or causation of disease.

This distinction became especially important after an observational study reported that micro- and nanoplastic detection in excised carotid plaques was associated with subsequent cardiovascular events [5]. The result is clinically consequential and hypothesis-generating, but the study design cannot by itself exclude residual confounding, reverse pathways, or measurement-related bias. Recent systematic and scoping reviews similarly find heterogeneous human evidence with limitations in sampling, exposure assessment, and causal interpretation [6, 7].

Oxidative stress is frequently proposed as a unifying mechanism. In redox biology, damage does not follow from the mere presence of reactive oxygen species (ROS); ROS also participate in normal signaling. Toxicologically relevant oxidative stress arises when oxidant production and antioxidant or repair capacity become imbalanced, disturbing redox control and damaging cellular components. Experimental MNP studies report ROS generation, mitochondrial dysfunction, inflammation, lipid peroxidation, DNA damage, and cell death across models [8, 9]. Yet the plastic literature varies widely in particle characterization, dose, exposure duration, protein corona, and biological system.

This review therefore addresses four questions: (1) through which routes can MNPs plausibly enter the human body; (2) how do particle and biological properties influence barrier crossing, uptake, distribution, and clearance; (3) which experimental pathways connect exposure to oxidative stress and downstream injury; and (4) what methodological limitations prevent firm human causal inference? The central contribution is an evidence-chain framework that keeps exposure plausibility, tissue detection, mechanistic hazard, and health risk analytically separate.

2. Review Scope and Evidence Logic

We conducted a structured narrative synthesis using an official health assessment, peer-reviewed human-detection and observational studies, systematic or critical reviews, analytical-method papers, and representative mechanistic research. Evidence was prioritized by relevance to human exposure or human-derived models. Animal and cellular evidence was used to assess biological plausibility, not to infer human risk directly. The review does not estimate a population attributable burden because measurement methods and internal-dose distributions remain insufficiently harmonized.

The proposed chain is:

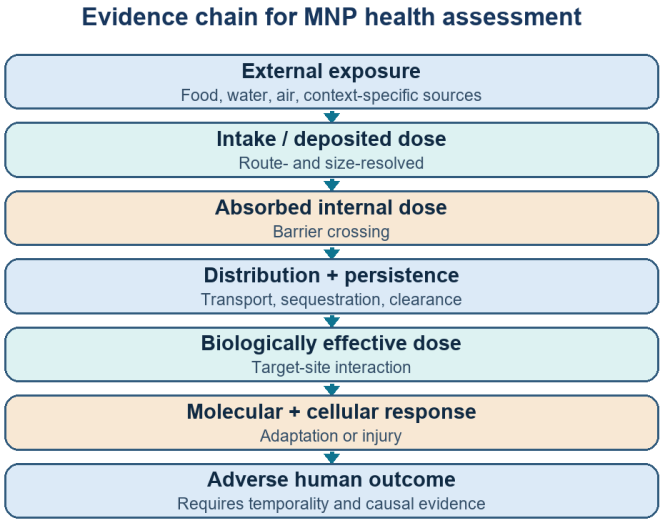


Figure 1. Evidence chain for MNP health assessment: external exposure → intake or deposited dose → absorbed internal dose → distribution and persistence → biologically effective dose at a target → molecular initiating events and adaptive responses → cellular or tissue injury → clinically meaningful adverse outcome. Particle sampling and characterization are required at every dose transition, while confounding and susceptibility influence the outcome transition.

Each arrow requires evidence. Environmental occurrence does not determine intake; intake does not determine absorption; tissue detection does not determine biologically effective dose; and a plausible oxidative pathway does not establish that exposure caused a human outcome. The chain also clarifies why "hazard" and "risk" differ. A particle may produce oxidative injury under experimental conditions, yet population risk depends on actual dose, duration, susceptibility, and exposure distribution.

3. Human Entry Routes

3.1 Gastrointestinal Exposure

Food, drinking water, swallowed dust, and particles cleared from the respiratory tract contribute to gastrointestinal exposure [1, 10]. Reported concentrations vary with sample collection, digestion, filtration, identification method, and the lower particle-size limit. Spectroscopic methods such as micro-Fourier-transform infrared or Raman microscopy identify polymer signatures for particles above method-dependent sizes; pyrolysis-based methods estimate polymer mass but generally sacrifice particle number and morphology. Consequently, estimates generated by different methods may describe different fractions of the same sample.

Ingestion exposes particles to changing pH, enzymes, bile salts, lipids, proteins, mucus, and microbiota. These conditions can agglomerate or disperse particles, leach additives, change surface charge, and create a biomolecular corona. Brouwer et al. showed in an in vitro digestion model that a gastrointestinal protein corona altered uptake of polystyrene nano- and microplastics by THP-1-derived macrophages in a size- and charge-dependent manner [11]. This finding demonstrates that pristine-particle exposure does not fully represent the biological identity encountered after digestion. It does not establish an absorption fraction in vivo.

Most larger particles are expected to remain in the lumen and be excreted, although retention in mucus and interaction with epithelium may occur. Smaller particles may cross through endocytosis, specialized epithelial pathways, or compromised barriers. The magnitude of each route in humans remains uncertain. Gastrointestinal inflammation, age, diet, and disease could alter permeability, but these modifiers require direct study rather than assumption.

3.2 Inhalation Exposure

Airborne fibers and fragments arise from textiles, traffic-related abrasion, industrial processes, resuspended dust, and indoor materials. Inhaled dose depends on aerodynamic behavior rather than geometric diameter alone. Shape, density, and agglomeration influence whether a particle deposits in the nasopharyngeal, tracheobronchial, or alveolar region. Clearance through mucociliary transport can move deposited material into the gastrointestinal tract, while alveolar macrophages may engulf particles that reach deep lung regions.

Microplastics have been detected in surgically obtained human lung tissue using micro-FTIR methods [4]. This supports inhalation and retention plausibility, but the detected sample is not a representative population measure and cannot by itself identify source, residence time, or clinical effect. Fibers may behave differently from spheres because their aspect ratio affects deposition and clearance. Occupational settings with higher airborne concentrations deserve targeted study with rigorous background controls.

3.3 Dermal and Other Routes

Intact skin is generally a stronger barrier to particulate penetration than the gastrointestinal or respiratory epithelia. Entry may be more plausible through hair follicles, damaged skin, wounds, or very small particles in specific formulations. Current evidence does not justify treating ordinary dermal contact as equivalent to ingestion or inhalation. Medical devices, intravenous products, and procedural materials may create direct or bypass routes in particular contexts, but source attribution is difficult because plastic equipment can also contaminate collected samples.

Table 1. Exposure-route evidence and key uncertainties

Route	Main source classes	Relevant biological barrier	Principal uncertainty
Ingestion	Food, water, swallowed dust, mucociliary clearance	Mucus and intestinal epithelium	Intake distribution and human absorption fraction
Inhalation	Fibers, dust, abrasion particles, occupational aerosols	Airway mucus, epithelium, alveolar macrophages	Size-resolved deposited dose and clearance
Dermal	Cosmetics, dust, textiles, contaminated surfaces	Stratum corneum and appendages	Penetration under realistic intact/damaged skin conditions
Medical/direct	Devices, procedures, pharmaceutical pathways	Route-specific or bypassed barrier	Source attribution and clinical relevance

4. Biological Identity and In-Body Fate

4.1 Particle Properties and Biomolecular Corona

MNPs are not a single toxicant. Relevant attributes include size distribution, shape, polymer, crystallinity, density, surface charge, oxidation, weathering, additives, residual monomers, biofilm, and adsorbed environmental chemicals. A nominal polymer label such as "polystyrene" is therefore insufficient. Commercial fluorescent spheres commonly used in experiments are useful reference particles but differ from irregular, aged environmental fragments and fibers.

When particles contact saliva, gastrointestinal fluid, lung lining fluid, blood, or intracellular compartments, biomolecules adsorb to their surfaces. The resulting corona changes effective size, charge, receptor interactions, agglomeration, and clearance. The corona is dynamic and compartment-specific. It can mask the original surface or present new biological motifs. Uptake measured in serum-free culture may therefore overestimate or qualitatively differ from uptake in a protein-rich environment; conversely, a particular corona can enhance uptake, as the digestion-associated study illustrates [11].

4.2 Barrier Crossing and Cellular Uptake

Barrier crossing depends strongly on size but should not be reduced to a single cutoff. Cells internalize particles through phagocytosis, macropinocytosis, clathrin- or caveolae-associated endocytosis, and other pathways depending on cell type and particle identity. Professional phagocytes can engulf larger material, while smaller particles may enter non-phagocytic cells more readily. Fibers and irregular fragments can frustrate or alter uptake relative to spheres.

In the intestine, mucus can trap particles, transport them toward clearance, or mediate contact with epithelium. Specialized microfold cells and areas of barrier disruption are possible translocation sites. In the lung, macrophage uptake is protective when it supports clearance, yet persistent or difficult-to-clear particles can sustain inflammatory signaling. Cellular uptake alone is not synonymous with toxicity: it may result in sequestration, lysosomal processing, transcytosis, exocytosis, or retention.

4.3 Distribution, Tissue Detection, And Clearance

Leslie et al. reported quantification of several plastic polymers in human blood samples [2], while Ragusa et al. reported microplastic particles in placental specimens [3]. These studies provided important proof-of-detection observations but also illustrate method dependence and small-study uncertainty. Detection in blood is compatible with transport, yet a single measurement cannot establish kinetics or tissue source. Placental detection does not establish fetal exposure or adverse pregnancy outcome without validated localization, dose, and temporal evidence.

Once in circulation, particles may interact with plasma proteins, blood cells, endothelium, liver, spleen, and kidney clearance pathways. Very small particles could be filtered or transported differently from micrometer-scale particles. Hepatobiliary excretion, renal elimination for

sufficiently small species, macrophage-mediated clearance, and redistribution are plausible, but quantitative human toxicokinetic data are sparse. Reported tissue concentration should therefore be described as a measurement at a time and site, not automatically as "bioaccumulation." Bioaccumulation requires evidence that uptake exceeds elimination over time.

The atheroma study by Marfella et al. found polyethylene in carotid plaque specimens and, in some samples, polyvinyl chloride; patients with detected particles experienced a higher rate of a composite cardiovascular outcome during follow-up [5]. The association persisted in adjusted analysis, but detection may correlate with unmeasured exposure, socioeconomic, behavioral, inflammatory, or clinical factors. The study cannot show whether particles initiated, accelerated, or merely co-occurred with atherosclerotic pathology. Its value lies in motivating replication, standardized quantification, and mechanistically connected prospective studies.

4.4 Additives and Adsorbed Chemicals

Plastic particles may contain plasticizers, stabilizers, pigments, flame retardants, catalysts, and residual monomers. Weathered surfaces can adsorb metals or organic pollutants. Biological effects may arise from the particle, leached chemicals, microbial material, or combinations. Experiments should distinguish these contributions using leachate controls, particle-free extracts, well-characterized reference particles, and environmentally aged materials. Without such controls, attributing oxidative effects to the polymer particle alone is premature.

5. Oxidative-Stress Mechanisms

5.1 Sources of Reactive Oxygen Species

Experimental reviews identify oxidative and inflammatory pathways as recurring responses to micro- and nanoplastics [8, 9, 12]. ROS can arise from several nonexclusive sources. Weathered particle surfaces may carry radicals or redox-active contaminants. Internalized particles can disturb membranes, lysosomes, mitochondria, or endoplasmic reticulum. Phagocytes can generate an oxidative burst as part of particle handling. Leached additives or adsorbates can undergo redox cycling. These mechanisms differ across particle type and cell context, so an observed fluorescent ROS signal should not be treated as proof of a particular source.

ROS assays themselves require caution. Fluorescent probes can be nonspecific, affected by light, particle adsorption, or assay interference. A stronger mechanistic design combines orthogonal measurements: redox couples, antioxidant-enzyme activity, lipid oxidation products, mitochondrial respiration, membrane potential, and damage markers. Time-resolved measurement helps distinguish early adaptive signaling from sustained oxidative distress.

5.2 Mitochondrial Dysfunction

Mitochondria are both a source and target of ROS. Particle exposure can alter electron-transport-chain function, mitochondrial membrane potential, fission–fusion balance, calcium handling, and mitophagy in experimental systems. Dysfunction can increase electron leakage and ROS production, creating a feedback loop in which oxidative damage further impairs respiration. A recent review specifically links environmental nanoplastic exposure to cellular mitochondrial mechanisms and associated disease hypotheses [13]. The strength of evidence varies by model, and disease attribution remains more speculative than the cellular mechanism.

Mitochondrial effects can reduce ATP availability and change cellular metabolism. This is particularly relevant for barrier cells, neurons, cardiomyocytes, and renal cells with high or specialized energy demands. Yet many in vitro studies expose cells to high particle concentrations over short intervals. Sedimentation and agglomeration can make the delivered cellular dose different from the nominal medium concentration. Mechanistic studies should quantify deposited or internalized dose and compare it with plausible human tissue levels.

5.3 Antioxidant Defenses And Nrf2 Signaling

Cells control oxidants using glutathione, superoxide dismutases, catalase, glutathione peroxidases, thioredoxin systems, and repair pathways. Moderate redox disturbance can activate adaptive responses, including the Keap1–Nrf2 pathway and transcription of antioxidant and detoxification genes. Increased antioxidant-enzyme activity is therefore not automatically evidence of damage; it can be a compensatory response. Conversely, depleted glutathione, impaired enzyme activity, or persistent oxidative products may indicate that defense capacity has been exceeded.

MNP studies report both induction and suppression of antioxidant markers, reflecting differences in dose, duration, cell type, and sampling time [8, 9]. A defensible interpretation requires a response trajectory. Early Nrf2 activation with restored redox balance differs from transient activation followed by glutathione depletion and damage. Repeated low-dose designs are especially important because environmental exposure is likely chronic and intermittent rather than a single high bolus.

5.4 Inflammation and Redox Crosstalk

Oxidative stress and inflammation amplify one another. ROS and damaged organelles can activate redox-sensitive transcription and inflammasome-related pathways. NF-κB signaling can increase pro-inflammatory cytokine expression; lysosomal injury, mitochondrial ROS, and danger signals can contribute to NLRP3 inflammasome activation in susceptible models. Activated immune cells in turn generate ROS and reactive nitrogen species. Reviews of micro- and nanoplastic adverse-outcome pathways describe this oxidative–inflammatory coupling [12].

The existence of pathway activation in a cell or animal model does not establish chronic human inflammation at measured exposure levels. Endotoxin contamination is a particular confounder because it can produce strong inflammatory responses. Studies should report endotoxin testing, sterility handling, corona conditions, and particle interference with cytokine assays.

5.5 Macromolecular Damage and Cell Fate

If oxidant production exceeds defense and repair, lipid peroxidation can compromise membranes and generate reactive aldehydes. Protein oxidation can alter enzymes, receptors, and cytoskeleton. Oxidative DNA lesions and strand breaks can activate repair or checkpoint responses. The microplastic oxidative-stress literature reports these endpoints across a variety of organisms and cellular models [8]. Their interpretation depends on validated assays, temporal ordering, and evidence that effects are not caused solely by leachates or assay artifacts.

Downstream cell outcomes include growth arrest, senescence-like states, apoptosis, necrotic injury, autophagy changes, and, in some experimental contexts, ferroptosis-related lipid peroxidation. These pathways overlap. Caspase activation supports apoptosis more directly than loss of viability alone; ferroptosis requires more than ROS or lipid oxidation and should be supported by iron dependence, pathway markers, and rescue with appropriate inhibitors. Mechanistic labels should therefore match the evidence actually measured.

5.6 Integrated Mechanism and Susceptibility

The integrated hypothesis is a network rather than a linear universal pathway. Particle identity and corona influence deposition and uptake; uptake and leachates disturb membranes, lysosomes, mitochondria, or redox enzymes; ROS and inflammatory signaling alter macromolecules and organelle function; repair, antioxidant response, and clearance determine recovery; and sustained imbalance can shift cell fate. Host susceptibility—including age, pre-existing respiratory or cardiovascular disease, metabolic state, barrier integrity, and co-exposure—may change thresholds.

Table 2. Mechanistic evidence and interpretation limits

Mechanistic node	Representative measurements	Stronger inference requires	Common overinterpretation
ROS increase	Fluorescent probes, redox couples	Orthogonal assays and interference controls	ROS signal proves mitochondrial origin
Mitochondrial dysfunction	Respiration, ATP, membrane potential, morphology	Time course and delivered/internal dose	Any viability loss is mitochondrial toxicity
Antioxidant response	GSH/GSSG, SOD, CAT, GPx, Nrf2 targets	Adaptive-versus-decompensated trajectory	Enzyme induction always means damage
Inflammation	Cytokines, NF-κB, inflammasome markers	Endotoxin controls and pathway perturbation	Cytokine change predicts human disease
Cell death	Caspases, membrane integrity, lipid peroxidation	Pathway-specific markers and rescue	ROS alone proves apoptosis or ferroptosis

6. Evidence Gaps and Research Priorities

6.1 Contamination Control And Polymer Identification

MNP analysis is unusually vulnerable to background contamination from clothing, air, laboratory plasticware, filters, reagents, and clinical equipment. Quality assurance should include field blanks, procedural blanks, filtered reagents, nonplastic or characterized equipment, covered processing, airborne deposition controls, recovery tests, and documented blank correction. Kutralam-Muniasamy et al. describe progress and persistent problems in human microplastic diagnostics [14]. A recent QA/QC screening framework found that none of the 101 assessed human-organ, tissue, and fluid studies met all essential criteria, underscoring the need to improve rather than dismiss the evidence base [15].

Chemical confirmation is essential because visual classification alone cannot reliably distinguish plastic from biological or mineral material. Spectroscopy, thermal analysis, and mass spectrometry provide different outputs and size windows [16]. Studies should report the fraction of a sample analyzed, spectral match criteria, detection limits, recovery by size and polymer, and whether results are blank-corrected. Imaging should demonstrate localization where tissue context matters.

6.2 Realistic Test Materials And Dose Metrics

Pristine polystyrene spheres dominate experimental studies because they are available, uniform, and fluorescent. Environmental MNPs are mixtures of fragments, fibers, films, weathered surfaces, additives, and coronas. Research needs well-characterized reference materials that span realistic shapes and aging states, plus field-derived mixtures with contamination and composition controls. Fluorescent labels require stability tests because free dye can be mistaken for particle uptake.

Dose should be reported by mass, particle number, size distribution, surface area, and—where feasible—delivered or internalized amount. A mass-equivalent dose can contain radically different particle numbers across sizes. Nominal medium concentration does not describe sedimentation or agglomeration. For inhalation, deposited dose per surface area and aerodynamic properties are relevant; for ingestion, gastrointestinal transformation and apical dose matter. Human biomonitoring needs harmonized units that permit comparison across methods.

6.3 Human Causal Inference

Systematic review evidence indicates that human studies remain heterogeneous and at moderate to high risk of bias [6]. Cross-sectional tissue detection lacks temporal ordering. Case–control designs may be vulnerable to differences in procedures or sample storage. Observational cohorts improve temporality but still need repeated exposure or internal-dose measures, relevant confounders, prespecified outcomes, and transparent missing-data handling. The carotid-plaque study [5] provides

a model for linking tissue detection to follow-up, but replication should include external quality assurance and stronger source and dose characterization.

Prospective cohorts could combine personal air or diet sampling, standardized blood or other validated matrices, repeated measurements, inflammatory and redox biomarkers, and clinical outcomes. Nested mechanistic studies could test whether internal dose precedes molecular response. Natural experiments or occupational contrasts may improve exposure variation, provided co-exposures are measured. Causal inference should triangulate across methods rather than rely on one biomarker or model.

6.4 Toxicokinetics and Effect Thresholds

Human risk assessment needs absorption, distribution, metabolism-like transformation, and excretion data across particle classes. Isotope-labeled or otherwise stable, validated tracers in appropriate models can quantify mass balance. Physiologically based models may then connect external exposure to tissue dose, but only if parameterized with empirical data and particle-specific behavior. Repeated low-dose studies should examine whether clearance reaches steady state or whether particular compartments retain particles.

Mechanistic thresholds should be related to internal dose and susceptibility. Omics can discover pathways, but pathway enrichment should be followed by targeted validation and functional rescue. Mixture studies should separate particle, additive, and adsorbate contributions. Reporting null results and full dose–response curves will reduce publication bias toward dramatic high-dose effects.

7. Conclusion

Humans plausibly encounter micro- and nanoplastics through ingestion and inhalation, with dermal and direct routes relevant in narrower conditions. Reports in blood, placenta, lung, and vascular plaque support exposure and translocation hypotheses, but detection does not by itself demonstrate persistent bioaccumulation, target-organ dose, or disease causation. Biological fate depends on size, shape, polymer, weathering, additives, agglomeration, and the compartment-specific biomolecular corona.

Experimental evidence supports a plausible oxidative-stress network involving ROS, mitochondrial dysfunction, antioxidant responses, inflammatory crosstalk, macromolecular injury, and cell-fate changes. Most mechanistic evidence, however, comes from models and doses that are not yet securely linked to human internal exposure. The strongest conclusion is therefore one of structured concern rather than quantified clinical risk.

Future work should follow the full evidence chain from external exposure to absorbed and biologically effective dose, temporally ordered molecular response, and adverse outcome. Harmonized blanks, recovery controls, chemical identification, realistic particles, multidimensional dose reporting, toxicokinetic mass balance, and longitudinal human studies are the priorities. These practices will allow the field to preserve legitimate warning signals while replacing inference by proximity—particle present, therefore disease caused—with evidence capable of supporting prevention and policy.

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