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Microplastics Exposure and Redox Imbalance in Human Tissues: Mechanisms, Biomarkers, and Health Implications

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ABSTRACT

Microplastics-plastic particles smaller than 5 mm, have emerged as pervasive environmental contaminants detected in air, water, food, and consumer products. Human exposure occurs through ingestion, inhalation, and dermal contact, leading to measurable microplastic particles and plastic-associated chemicals in blood, lung tissue, placenta, and stool. Growing experimental and epidemiological evidence suggests that microplastics may disrupt redox homeostasis, triggering oxidative stress and downstream inflammatory and metabolic alterations. Redox imbalance arises when reactive oxygen species (ROS) generation exceeds antioxidant defense capacity, damaging lipids, proteins, and DNA. Microplastics can induce oxidative stress through particle surface reactivity, leaching of additives such as phthalates and bisphenols, adsorption of environmental pollutants, and activation of immune pathways. This review synthesizes current knowledge on human exposure pathways, cellular and molecular mechanisms linking microplastics to redox dysregulation, tissue-specific effects, biomarkers of oxidative injury, and potential long-term health consequences. It also highlights methodological challenges in exposure assessment and proposes research priorities to clarify dose–response relationships and vulnerable populations. Understanding how microplastics contribute to redox imbalance in human tissues is critical for risk assessment, regulatory action, and the development of preventive strategies.

Keywords: microplastics, oxidative stress, redox imbalance, reactive oxygen species, environmental toxicology

INTRODUCTION

Plastic production has increased exponentially over the past decades, resulting in widespread environmental accumulation of plastic debris across terrestrial and aquatic ecosystems[1]. Global reliance on plastic for packaging, construction, transportation, medicine, and consumer goods has led to persistent waste streams that degrade slowly over time[2]. Fragmentation through physical abrasion, ultraviolet radiation, chemical oxidation, and microbial activity generates microplastics (MPs), typically defined as particles less than 5 mm in size. These particles originate from primary sources, such as cosmetic microbeads, industrial resin pellets, and microfibers intentionally manufactured at small sizes, and secondary sources, including breakdown of larger plastic waste, tire wear particles, synthetic textiles, and fishing gear[3]. Human exposure to microplastics is now recognized as widespread and likely unavoidable. MPs have been detected in drinking water, bottled beverages, seafood, table salt, honey, fruits, vegetables, and indoor as well as outdoor air[4]. Food processing, packaging, and storage further contribute to contamination. Inhalation of airborne microfibers from textiles, furniture, and household dust represents a substantial exposure pathway, particularly in urban and indoor environments where people spend most of their time[5]. Recent advances in spectroscopic and microscopic analytical techniques have confirmed the presence of microplastic particles in human blood, lung tissue, colon samples, and placental tissue, raising concerns about systemic distribution, tissue accumulation, and long-term biological consequences[6].

Among the proposed mechanisms of toxicity, oxidative stress and redox imbalance have emerged as central themes[7]. Redox homeostasis maintains cellular integrity by balancing reactive oxygen species (ROS) production with antioxidant defenses such as glutathione, superoxide dismutase, catalase, and peroxiredoxins. Controlled ROS signaling is essential for normal cellular processes, but excessive ROS can damage lipids, proteins, and nucleic

acids[8]. Disruption of redox balance contributes to inflammation, mitochondrial dysfunction, genomic instability, and chronic diseases including cardiovascular and neurodegenerative disorders[9]. This review examines how microplastics may perturb redox equilibrium in human tissues and explores the implications for long-term health risk and disease susceptibility.

2. Characteristics of Microplastics Relevant to Toxicity

The biological impact of microplastics depends heavily on their physicochemical properties, including particle size, shape, polymer composition, crystallinity, surface charge, and associated chemical additives[10]. These characteristics influence how particles interact with biological membranes, immune cells, and intracellular structures. Particle size is particularly critical. Smaller particles, especially those in the micrometer and nanometer range, have a greater surface area-to-volume ratio and are more likely to translocate across epithelial barriers in the gut and lungs[11]. Nanoplastics may penetrate cellular membranes via endocytosis and potentially reach organelles such as mitochondria, where they can interfere with redox processes. Shape also influences toxicity[12]. Fibrous particles may persist in lung tissue and resist clearance by macrophages, while irregular fragments and spheres may differ in how they activate inflammatory pathways. Microplastics are rarely chemically inert. They often contain additives such as plasticizers, antioxidants, stabilizers, flame retardants, and pigments introduced during manufacturing[13]. Under physiological conditions, these additives can leach from the polymer matrix, contributing to endocrine disruption and oxidative stress. Additionally, MPs act as vectors for environmental contaminants including heavy metals, persistent organic pollutants, and polycyclic aromatic hydrocarbons[14]. Adsorbed chemicals may desorb upon contact with biological fluids, enhancing oxidative potential and cumulative toxicity. Environmental weathering further modifies particle surfaces by introducing oxygen-containing functional groups and increasing roughness[15]. These changes can enhance chemical reactivity and catalyze ROS formation. Therefore, aging processes in the environment may significantly alter microplastic toxicity profiles and amplify their capacity to induce redox imbalance in human tissues.

3. Pathways of Human Exposure and Tissue Distribution

3.1 Ingestion

Dietary intake represents a primary route of exposure. Microplastics are present in seafood due to trophic transfer, in bottled and tap water, and in processed foods contaminated during packaging[16]. Ingested MPs may interact with the gastrointestinal epithelium, potentially crossing via paracellular transport or uptake by specialized cells.

Animal studies demonstrate translocation of small particles to the liver, spleen, and kidneys[17]. In humans, microplastics have been detected in feces and blood, suggesting partial absorption and systemic circulation.

3.2 Inhalation

Airborne microfibers from textiles, carpets, and urban dust are inhaled daily. Occupational settings such as textile manufacturing may present higher exposure levels[18]. Deposited particles in the respiratory tract can trigger oxidative stress in epithelial cells and alveolar macrophages. Persistent fibers may induce chronic inflammation and ROS generation, similar to other particulate pollutants.

3.3 Dermal Contact

Dermal exposure is less understood but may occur through cosmetics and personal care products. Although intact skin acts as a barrier, damaged or compromised skin may allow limited penetration of smaller particles or leached additives[19].

4. Mechanisms of Redox Imbalance Induced by Microplastics

4.1 Direct ROS Generation

Microplastics can directly generate ROS through surface redox reactions, particularly when containing transition metals or undergoing photochemical activation[20]. Particle–cell interactions may stimulate NADPH oxidase activity, increasing superoxide production.

4.2 Mitochondrial Dysfunction

Experimental studies indicate that exposure to MPs can impair mitochondrial membrane potential and electron transport chain function, leading to enhanced ROS leakage[21]. Mitochondrial oxidative stress disrupts ATP production and activates apoptotic signaling pathways.

4.3 Inflammatory Activation

Microplastics may activate innate immune receptors, including toll-like receptors and inflammasomes, triggering pro-inflammatory cytokine release[22]. Inflammation itself amplifies ROS production through immune cell activation, creating a feed-forward loop between oxidative stress and inflammation.

4.4 Leaching of Additives and Adsorbed Pollutants

Chemical additives such as phthalates and bisphenols are known endocrine disruptors capable of inducing oxidative stress[23]. When leached from MPs, these compounds can increase lipid peroxidation and DNA oxidation. Adsorbed pollutants may further contribute to redox imbalance.

5. Tissue-Specific Effects

5.1 Gastrointestinal Tract

The gastrointestinal tract represents a primary site of microplastic exposure through contaminated food and water[24]. In the gut, MPs may physically interact with the mucus layer and epithelial cells, potentially disrupting tight junction integrity. This disruption can compromise barrier function and increase intestinal permeability, sometimes referred to as a "leaky gut[25]." Alterations in gut microbiota composition have also been observed in experimental models, with shifts toward pro-inflammatory bacterial populations[26]. Dysbiosis may amplify oxidative stress and inflammatory signaling, facilitating systemic immune activation. Within enterocytes, excessive reactive oxygen species production can damage cellular membranes and mitochondria, impair nutrient absorption, and interfere with normal epithelial renewal processes.

5.2 Liver

The liver, as a central detoxification and metabolic organ, may accumulate microplastic particles that translocate from the intestine into the portal circulation[27]. Hepatocellular exposure to MPs has been associated in animal studies with increased lipid peroxidation, depletion of glutathione, and reduced antioxidant enzyme activity[28]. Persistent oxidative stress may promote lipid accumulation, inflammatory infiltration, fibrosis, and altered metabolic signaling pathways[29]. These changes raise concerns about potential contributions to nonalcoholic fatty liver disease and broader metabolic dysfunction.

5.3 Lung

Inhaled microplastics deposit in airway and alveolar regions, where they interact with epithelial cells and resident macrophages[30]. Chronic particle retention may overwhelm clearance mechanisms, leading to sustained oxidative stress and inflammatory responses. Repeated exposure can contribute to epithelial injury, fibrotic remodeling, and reduced pulmonary function.

5.4 Placenta and Reproductive Tissues

Detection of MPs in placental tissue suggests the possibility of maternal–fetal transfer. Oxidative stress within placental cells may disrupt nutrient and oxygen exchange, potentially influencing fetal development and pregnancy outcomes[31].

6. Biomarkers of Oxidative Stress Associated with Microplastics

Assessment of redox imbalance involves measuring biomarkers that reflect oxidative damage and antioxidant capacity[32]. Lipid peroxidation products such as malondialdehyde and 4-hydroxynonenal indicate membrane damage, while 8-hydroxy-2′-deoxyguanosine reflects oxidative DNA injury. Protein carbonyl levels provide insight into protein oxidation[33]. Evaluation of reduced and oxidized glutathione ratios, along with activities of antioxidant enzymes such as superoxide dismutase and catalase, helps determine cellular defense status. Experimental studies frequently report elevated oxidative markers and diminished antioxidant activity following MP exposure, although robust human biomonitoring data remain limited.

7. Vulnerable Populations

Children may experience higher exposure relative to body weight due to hand-to-mouth behavior and proximity to floors containing dust fibers[34]. Pregnant women and individuals with pre-existing inflammatory or metabolic disorders may be more susceptible to oxidative damage. Occupationally exposed workers in textile or plastic industries represent additional high-risk groups.

8. Methodological Challenges

Quantifying human exposure to microplastics remains technically challenging. Variability in sampling methods, contamination control, and analytical techniques complicates comparisons across studies[35]. Standardization of detection protocols is urgently needed. Establishing causal links between microplastics and redox imbalance in humans requires longitudinal studies with well-characterized exposure metrics and biomarker panels.

9. Public Health Implications

Chronic oxidative stress contributes to cardiovascular disease, neurodegeneration, metabolic syndrome, and cancer[36]. If microplastics consistently induce redox imbalance, long-term exposure could exacerbate these conditions. Risk communication should balance precaution with scientific uncertainty, avoiding undue alarm while promoting reduction of plastic pollution[37].

10. Future Directions

Research priorities include dose–response assessment, exploration of nanoplastic effects, investigation of combined exposures with other pollutants, and identification of molecular pathways mediating redox disruption[38]. Development of advanced in vitro models and human cohort studies will enhance understanding of systemic impacts.

CONCLUSION

Microplastics are emerging contaminants with the potential to disrupt redox homeostasis in human tissues. Through direct ROS generation, mitochondrial impairment, inflammatory activation, and chemical leaching, MPs may induce oxidative stress across multiple organ systems. Although experimental evidence is substantial, human

epidemiological data remain limited. Addressing knowledge gaps and implementing preventive strategies to reduce plastic pollution are essential steps toward protecting public health in an increasingly plastic-dependent world.

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