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Review

Impact of Microplastics on Human Health: Risks, Diseases, and Affected Body Systems

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Abstract: This review article aims to highlight the potential harm caused by microplastics (MPs) in different organs and systems and underscore the need for further investigation into their action mechanisms. MPs, such as polystyrene, polypropylene, and polyethylene, significantly impact human health, causing inflammation in the respiratory and gastrointestinal systems, compromising immune function, and increasing the risk of cardiovascular diseases and neurotoxicity. These effects are largely attributed to the role of MPs in disrupting hormonal regulation, which can lead to reproductive disorders and an elevated risk of cancer. These microscopic particles (less than 5 mm in size) are now ubiquitous in air, water, and food. However, much of the existing research on MPs focuses on their mechanisms of action and their association with health and disease, with limited emphasis on their direct impact on humans or long-term consequences. To effectively address plastic toxicity, it is crucial to understand the policy implications of MPs and their relevance to disease development. Recent research has highlighted the need for more stringent regulatory oversight of these materials to better understand and mitigate their impact on human health.

Keywords: human health; microplastics; diseases; environment

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1. Introduction

Microplastics (MPs), defined as plastic particles smaller than 5 mm, have emerged as a significant public health and environmental concern due to their widespread presence in ecosystems and potential to infiltrate human tissues [1,2]. While the environmental impact of MPs has been extensively reported, their effects on human health remain less understood, particularly regarding their role in disease progression across multiple organ systems [3–5]. Polystyrene and other synthetic polymers are well-known among other MPs for their ubiquity in standard products and harmful effects on biological systems. Extensive research on human health has impelled concern about the paraphernalia of MPs on various body organs and systems and the role of MPs in different disease progressions [6–9].

MPs enter the human body primarily through ingestion, inhalation, and transdermal absorption, with recent studies detecting their presence in vital organs such as the lungs, gastrointestinal tract, and even placental tissues [10,11]. They have infiltrated nearly every tier of the food chain, from bottled water to seafood, indicating that humans are probably consuming them often. This widespread exposure raises urgent questions about the long-term health implications of MPs, including their potential to induce inflammation,

disrupt immune homeostasis, and contribute to chronic diseases [12,13]. MPs induce considerable human immunotoxic effects by disturbing immunological homeostasis and eliciting persistent inflammation.

Humans are primarily exposed to MPs through ingesting contaminated water and food. MPs have been found in marine animals, such as fish, crustaceans, and mollusks, which are globally familiar sources of nutrition. When ingested, MPs may build up in the gastrointestinal tract (GIT), leading to inflammation, altering gut bacteria, and potentially entering the bloodstream under specific circumstances [14,15]. Disruptions in the balance of gut microbiota are detrimental to maintaining stable metabolism and a vigorous immune system [16]. In addition, prolonged exposure to MPs and resulting chronic inflammation of the GIT have been linked with conditions such as irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD) [17].

Airborne MPs from synthetic textiles, tire degradation, and urban dust endanger respiratory health. MPs may accumulate in pulmonary tissues upon inhalation, resulting in inflammation and compromised lung function [18]. Similarly, Prata [19] has reported that these inhaled MPs caused an alteration of the lungs, ultimately leading to asthma and COPD. Recent research indicates that MPs may bypass biological barriers, including the placental barrier, signifying prenatal exposure [20]. Hence, it advances considerable concerns for the development of fetal health along with long-term disease onsets. The exposure of pregnant women to MPs may pose risks to their unborn children [21]. Furthermore, recurrently identified polystyrene MPs harbor potentially deleterious compounds such as styrene monomers, which the International Agency for Research on Cancer (IARC, 2019) categorizes as possible human carcinogens that may be leaching from MPs, contributing a toxicity layer to the physical burden created by these particles [22,23]. Figure 1 shows the potential health effects of MPs on the human body system.

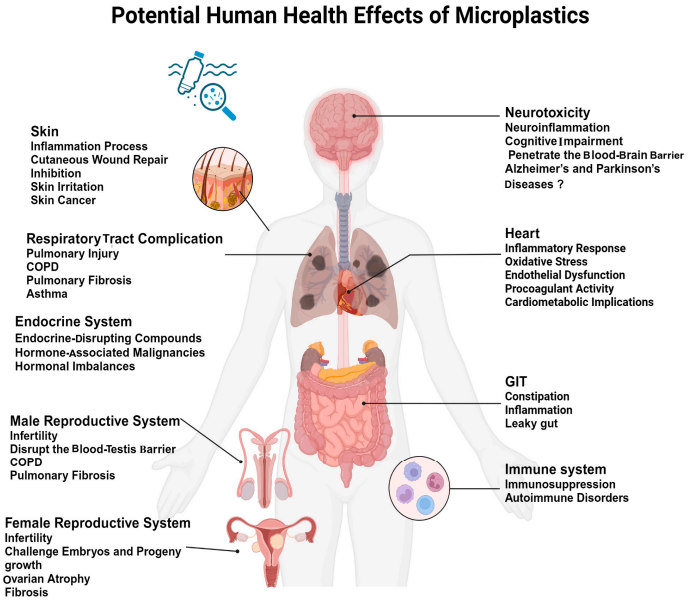

Figure 1. Potential human health effects of microplastic (MP) exposure across organ systems. This figure summarizes the adverse health effects of MPs on various human organ systems, including neurotoxicity (e.g., neurodegenerative diseases, cognitive impairment), cardiovascular diseases (e.g., oxidative stress, endothelial dysfunctions, cardiometabolic implications), respiratory complications (e.g., oxidative stress, inflammation), endocrine system interruption (e.g., hormonal imbalances, malignancies related to hormones), impairments of the gastrointestinal and reproductive system (e.g., leaky gut, infertility), and immune system dysregulation (e.g., autoimmune disorders, immunosuppression). Key mechanisms and associated health risks are highlighted. Created in BioRender. Ahmad, M. (2025) https://BioRender.com/y2xw9q6 (accessed on 3 March 2025).

Despite growing evidence of MPs' harmful effects, critical knowledge gaps remain. For example, the mechanisms by which MPs interact with biological systems, their role in the development of specific diseases (e.g., cardiovascular disorders, reproductive toxicity, and metabolic syndromes), and the long-term consequences of chronic exposure are poorly understood. This review aims to present recent findings on the health impacts of MPs, with a focus on their effects on the cardiovascular, respiratory, reproductive, and gastrointestinal systems. By identifying key knowledge gaps and proposing future research directions, we hope to provide a comprehensive resource for understanding the multifaceted risks posed by MPs to human health.

2. Sources of Microplastics

This section provides an overview of the primary sources of MPs, their environmental distribution, and the mechanisms by which they enter human tissues. MPs have two broad categories of origins: primary and secondary sources. Primary sources are plastics designed to be manufactured in tiny sizes. Secondary sources are mainly textile fibers, plastic degradation, tire wear particles, and paints and coatings (Figure 2).

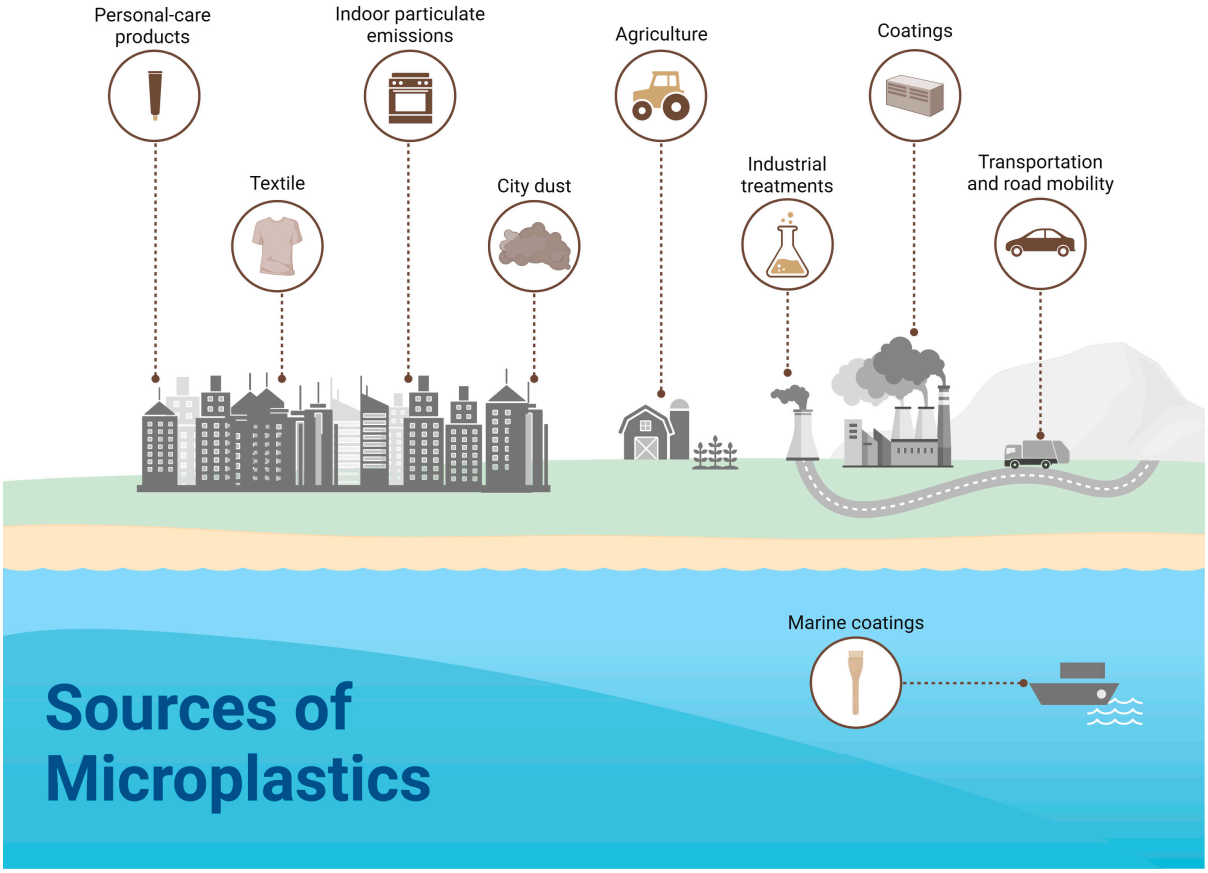

Figure 2. Primary sources of microplastic (MPs) include personal-care products: these MPs originate from the microbeads in various products such as scrubbing masks or toothpaste. Industrial abrasives: MPs are used in industrial processes like sandblasting, where they shoot small plastic pellets at a surface to clean it. These pellets are the type that can escape into the environment and contribute to MP pollution. Secondary sources of MPs include textile fibers: synthetic fibers from washing clothes made of polyester, nylon, and acrylic shed MPs. Plastic degradation: larger plastic debris, such as bottles, bags, and packaging materials, break down into MPs through environmental processes, such as exposure to UV radiation, physical abrasion, and biological degradation. Tire wear particles: abrasion of tires during driving releases MPs into the environment. Paints and coatings: marine and terrestrial paints can contain deletions of MPs as part of the dissolution or erosion process. Created in BioRender. Ahmad, M. (2025) https://BioRender.com/s41ly53 (accessed on 3 March 2025).

3. Human Exposure Pathways of Microplastics

Human exposure to MPs emerges via several routes, primarily linked with eating, inhalation, and dermal contact. Figure 3 illustrates the human exposure pathways of MPs.

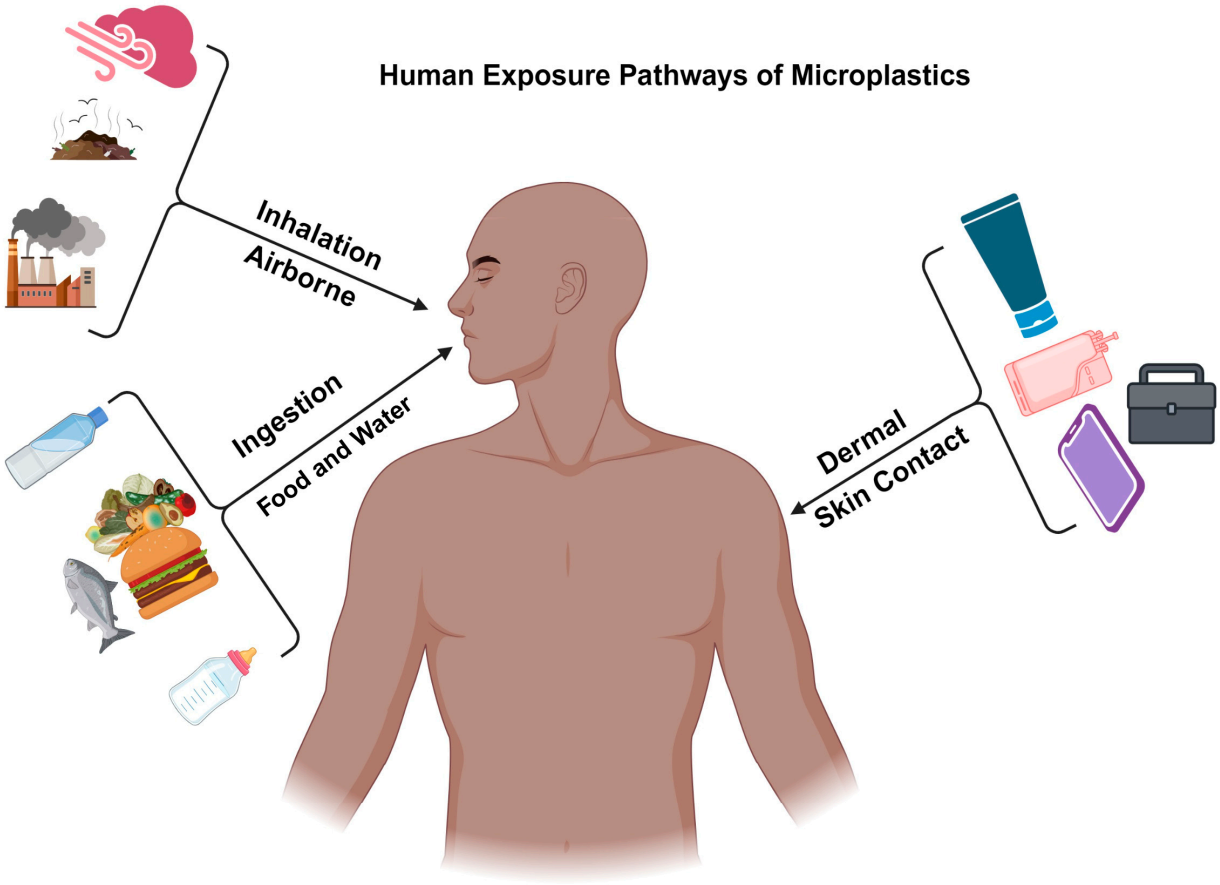

Figure 3. Human exposure pathways of microplastics (MPs). Human exposure to MPs emerges via several routes, primarily linked with inhalation, eating, and dermal contact. Key pathways include the following: inhalation: airborne MPs, particularly in indoor environments, can be inhaled, leading to potential respiratory complications; ingestion: MPs enter the human body through the consumption of contaminated food and water; dermal contact: MPs can come into contact with the skin through personal-care products and environmental exposure. Created in BioRender. Ahmad, M. (2025) https://BioRender.com/h88vxsd (accessed on 3 March 2025).

3.1. Ingestion

Food Consumption

Seafood: A primary avenue is the ingestion of seafood. Numerous marine creatures, such as fish, shellfish, and crabs, consume MPs, which can subsequently ascend the food chain to humans. Filter feeders such as mussels and oysters are especially vulnerable to the accumulation of MPs [24,25]. MPs have been found in various food items, including salt, sugar, honey, beer, and fresh fruit. Plastic particles are believed to contaminate crops through polluted soils or irrigation [26]. Plastic packaging and processing significantly contribute to MP contamination. Minuscule particles may transfer into food during handling, storage, and preparation, particularly with highly processed items or those stored in plastic for extended periods [27,28].

Research suggests that bottled water frequently has elevated levels of MPs compared to tap water, likely attributable to plastic packaging [29]. Regarding tap water, MPs have been globally identified; however, at levels typically lower than those seen in bottled water [30].

3.2. Inhalation

Airborne Microplastics

The indoor environment harbors substantial MPs, primarily from synthetic textiles, domestic dust, and deteriorated plastic materials. Tasks like vacuuming, dusting, and utilizing synthetic textiles and furnishings emit microplastic fibers that may subsequently be breathed in [31,32]. Considering the outdoor environment, MPs are prevalent in outdoor air, particularly in urban regions with significant plastic trash accumulation. Particles may derive from tire and road degradation, construction materials, industrial emissions, and deteriorated plastic waste. These airborne particulates may be deposited on food or be ingested by individuals in outdoor settings [33].

3.3. Dermal Contact

MPs are frequently included in cosmetics and personal-care items, including scrubs, toothpaste, and exfoliants. These microbeads may infiltrate the skin or be flushed into aquatic systems, increasing exposure [34,35].

Synthetic textiles, including polyester, nylon, and acrylic, may release microplastic fibers during use. Despite dermal contact being regarded as a less significant exposure pathway compared to ingestion and inhalation, there remains a potential danger of MPs adhering to the skin and being absorbed [36,37].

4. Potential Human Health Effects of Microplastics

MPs can impact multiple human bodily systems, including the immunological, respiratory, gastrointestinal, cardiovascular, neurological, and reproductive systems. Presented herein is a summary of the effects of each system.

4.1. MPs' Impact on the Immune System

MPs, such as polystyrene, can elicit immunological reactions within the body [38,39]. Immune activation: Introducing MPs into the bloodstream or tissues may elicit an inflammatory response. Chronic inflammation is associated with cardiovascular disease, autoimmune disorders, and cancer [40–42]. Macrophage overload: Inhaled MPs may aggregate in pulmonary tissues, resulting in overwhelmed immune cells and a heightened risk of respiratory illnesses [43,44]. Xu et al. found that MPs pose health risks to human tissues, with urban exposure assessed at 7.37×10^4 items/year for children and 1.06×10^5 items/year for adults. They highlighted the respiratory and immune systems as affected, linking atmospheric MPs to airborne pathogen risks, with *Sphingomonas* as a key mediator, and noted significant spatiotemporal variations driven by wind speed and rainfall [45]. Prolonged exposure to MPs, especially from substances like polystyrene, may lead to chronic inflammation linked to autoimmune illnesses and heightened cancer risk [45,46]. The oxidative stress pathways are activated, leading to the production of reactive oxygen species (ROS). Therefore, immune signaling pathways, such as the cGAS-STING pathway, upsurge the creation of pro-inflammatory cytokines through the NF-κB signaling pathway [47]. The over-activation of these pathways may lead to immune cell death, compromising immune defense and causing tissue damage. MPs interact with other environmental contaminants or microbial infections, enhancing their toxicity through surface binding, which amplifies immune responses [48,49]. The phenomenon of protein corona permits MPs to transport lethal substances further into the body system, which leads to chronic immune activation and systemic inflammation [50]. Furthermore, prolonged exposure to MPs is associated with insulted immune system organs, particularly the spleen and liver, which can lead to fibrosis and inflammation. Considering these verdicts, there is an increasing need for inclusive research on the synergistic effects of MPs with other

pollutants and the complex mechanisms of immune system dysregulation to augment understanding of long-term health risks.

4.2. Respiratory Health Issues in Response to MPs

These tiny MPs potentially are the reason for irritation and inflammation of the respiratory tract. Subsequently, this leads to coughing, sneezing, wheezing, shortness of breath, and aggravation of asthma [51,52]. Inhaled MPs, particularly those derived from polystyrene or tire wear particles, may result in pulmonary injury. Inhaling airborne MPs also causes lung inflammation and worsens asthma or COPD [53,54]. The fiber's size influences toxicity. Fibers with less thickness could be inhaled by the respiratory tract, whereas lengthy fibers enhanced the tenacity and noxiousness of cellular components of the lung. Thus, 15–20 μm fibers could not be efficiently cleared by alveolar macrophages and the mucociliary transport system [55]. Similarly, prolonged inhalation of MPs may lead to pulmonary fibrosis, a severe illness that induces scar formation on lung tissue [56,57]. Furthermore, synthetic clothing releases tiny particles everywhere, thus facilitating the inhalation of some of them. When they enter the respiratory organs, the lungs' fluid will likely catch these fibrous particles. However, some of these might cross the lung's natural barricade mechanisms [1,10,58].

Particle accumulation occurs when tiny MP particles become ingrained in the lungs' interior, resulting in chronic respiratory complications and causing lung dysfunction. Previous studies have reported the presence of synthetic fibers and respiratory irritation in the lung tissue of textile industry workers [59]. Therefore, the lungs of flock workers exposed to nylon showed persistent interstitial lung disease despite leaving the work environment. This slow decline in lung function progresses to secondary pulmonary hypertension and thereafter respiratory failure [60]. Danso et al. investigated the lung inflammation and toxicity of MPs such as polystyrene (PS), polypropylene (PP), and polyvinyl chloride (PVC) in mice. Mice were intratracheally inoculated with doses of 5 mg/kg of PS, PP, or PVC daily for two weeks. Results showed PS increased inflammatory cells in the bronchoalveolar lavage fluid (BALF) of C57BL/6 and ICR mice, whereas PP caused inflammatory cell infiltration in ICR mice. PS also increased NLRP3 inflammasome components and cytokines (e.g., IL-1, IL-6) in C57BL/6 mice, and PP increased NLRP3, ASC, and Caspase-1 in ICR mice [61]. Likewise, occupational exposure to polypropylene showed signs of respiratory symptoms and impaired lung function with a positive serum cytokine profile. This was consistent with the presence of mild interstitial lung disease, illustrating the need to use exposure controls when working with polypropylene in industry [62].

4.3. Gastrointestinal and Metabolic Impacts in Response to MPs

MPs' exposure to the GIT has become a notable concern linked to consuming particles mainly from contaminated food or water [63,64]. For example, GIT inflammation, constipation, dysbiosis, and alteration in intestinal absorbency [65,66]. Moreover, Osman et al. have reported that MPs accumulate in the digestive tract, causing irritation and obstructions [67]. The biological effects of MPs in the GIT are anticipated to result from their ancillary motion, which augments the immune response to adsorbed biomolecules on their surfaces [68]. Furthermore, MPs significantly impact the symbiotic association between gut bacteria and hosts. Hence, this phenomenon leads to an imbalance, which is called dysbiosis. Qiao et al. reported the toxicity and accumulation based on shape-dependent MPs such as beads, fragments, and fibers in the gut of zebrafish. Accumulation followed the order fibers (8.0 μg/mg) > fragments (1.7 μg/mg) > beads (0.5 μg/mg). The most severe intestinal toxicity, including mucosal damage, increased permeability, inflammation, and metabolism disruption, was caused by fibers. In addition, MP-induced gut microbiota

dysbiosis, with specific bacterial alterations, suggests a potential mechanism for intestinal toxicity [65]. Dysbiosis also negatively affects the host's immune system, possibly causing chronic illnesses, enhanced vulnerability to pathogen infections, and altered genetics and gut microbiota expression [69,70].

MPs, smaller than 50 μm, have posed substantial GIT health hazards due to their ability to accumulate in gut tissues and affect biological mechanisms [71]. Previous studies demonstrate that MPs breach the intestinal barrier upon ingestion, prompting stress at the cellular level. This causes endoplasmic reticulum stress and mitochondrial dysfunction, ultimately leading to inflammation and fibrosis in the GIT [72,73]. Thus, the interplay of these pathways with MP buildup stresses the demanding compulsion for further research into their extended impacts on human health [74,75].

Intestinal barrier compromise due to MPs has been shown to disrupt the integrity of the gut barrier, resulting in gut permeability (hence known as "leaky gut"). This could allow harmful bacteria and toxins to enter the bloodstream, worsening inflammation and conditions like IBS and IBD [76–78]. Similarly, in terms of altered gut microbiota, MPs disrupt the balance of gut bacteria, leading to metabolic problems, obesity, IBD, and type 2 diabetes [14,39,79,80].

4.4. Cardiovascular System Response to MPs

The cardiovascular system is complex and involves multiple pathways. MP exposure causes certain ailments, such as inflammation, oxidative stress, endothelial dysfunction, procoagulant activity, and cardiometabolic implications (Figure 4) [81,82]. Given the extensive occurrence in the human body and environment, the negative impact of MPs on cardiac health is alarming [83]. Past studies have revealed that polystyrene MPs may activate the Wnt/β-catenin signaling pathway in kidneys [84]. This leads to cardiac fibrosis, oxidative stress, and mortality in rats. The study carried out by Li et al. examined the cardiovascular toxicity of PSMPs in male Wistar rats. These rats were exposed to 0.5 μm PSMPs at concentrations of 0.5, 5, and 50 mg/L for 90 days. Results showed PSMPs increased Troponin I and creatine kinase-MB (CK-MB) levels in serum, causing myocardial structural damage, apoptosis, and collagen proliferation. PSMPs induced oxidative stress, activating the Wnt/β-catenin signaling pathway, leading to cardiac fibrosis. Thus, the findings discovered a potential mechanism for PSMPs-induced cardiovascular toxicity via oxidative stress and fibrosis [85]. The interaction of MPs with the circulatory system can cause endothelial damage, abnormal blood coagulation, and disrupted metabolic activities, exacerbating cardiovascular dysfunction. These particles may contribute to cardiomyocyte death, pericardial edema, and irregular heart rhythms by influencing oxidative balance and generating an inflammatory response [29,86]. Further research is needed to comprehend the vital mechanisms and evaluate the long-term MP risks to cardiovascular health.

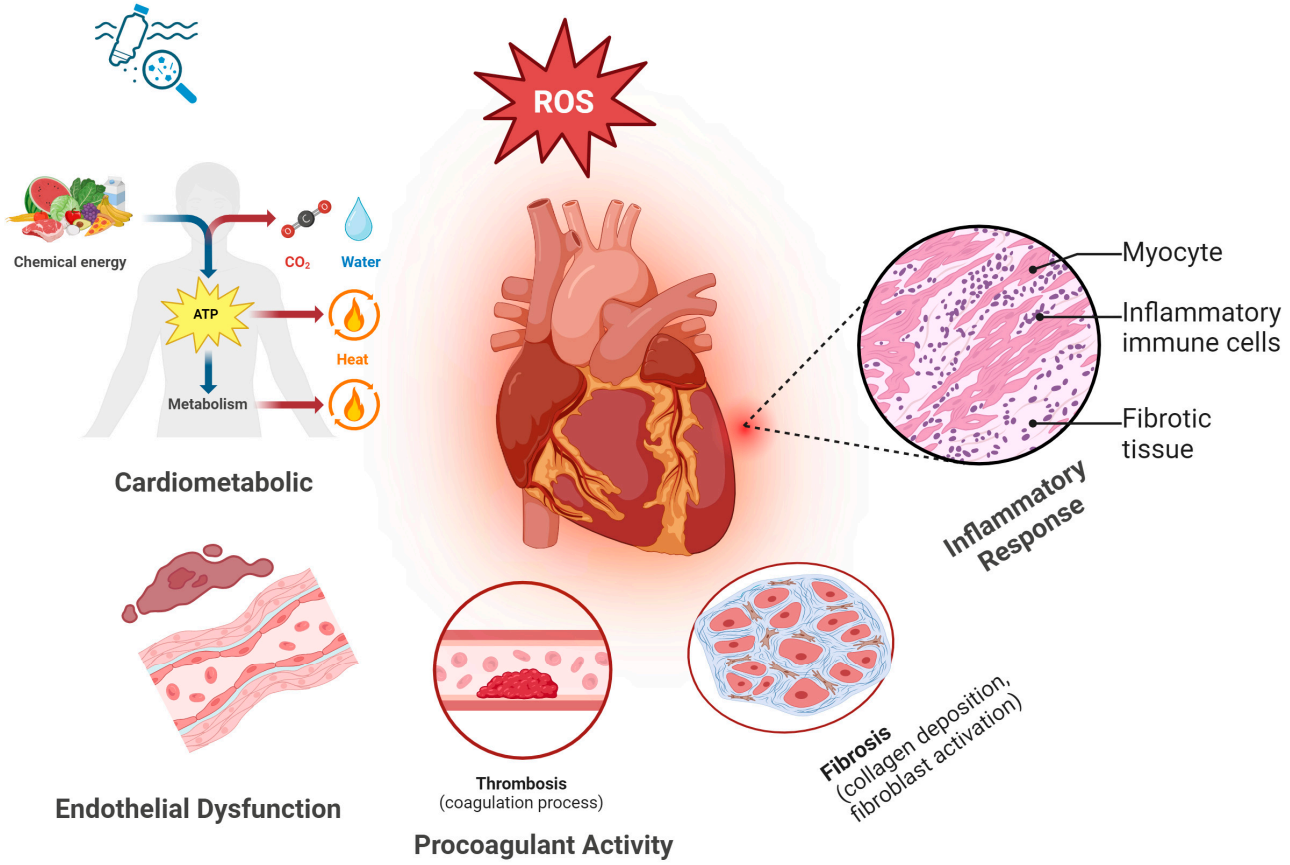

Figure 4. Pathophysiological pathways associated with microplastic (MP) exposure to cardiovascular diseases. MP exposure disrupts chemical energy metabolism (e.g., ATP production in myocytes), induces oxidative stress (e.g., ROS generation), and triggers inflammatory responses (e.g., immune cell activation). These mechanisms contribute to cardiovascular diseases by promoting endothelial dysfunction, procoagulant activity, and fibrotic changes, ultimately driving conditions such as atherosclerosis, hypertension, and heart failure. Created in BioRender. Ahmad, M. (2025) https://BioRender.com/fohq2ti (accessed on 3 March 2025).

4.4.1. Inflammatory Response

MPs induce a systemic inflammatory response that affects cardiovascular function [87]. These MPs can infiltrate the bloodstream and circulate throughout the body upon ingestion or inhalation. Previous research indicates that exposure to MPs could stimulate immune cells, mainly macrophages, and augment pro-inflammatory cytokine secretion, such as IL-6 and TNF-α [88]. Chronic inflammation is known as a contributor to endothelial dysfunction, a critical factor in the onset of atherosclerosis and relevant cardiac disorders [87,89].

4.4.2. Oxidative Stress

MPs can induce oxidative stress by producing ROS in vascular cells [90–92]. Elevated ROS levels can induce oxidative damage to endothelial cells, compromising the integrity and functionality of the arterial lining. This dysfunction significantly contributes to the onset of hypertension and atherosclerosis [93,94]. Oxidative stress influences the expression of nitric oxide synthase, thereby diminishing nitric oxide (NO) availability, which is crucial for vasodilation and the regulation of appropriate blood pressure levels [95,96].

4.4.3. Endothelial Dysfunction

The endothelium, which coats the inner surface of blood arteries, is especially susceptible to microplastic exposure. Studies indicate that MPs may directly engage with

endothelial cells, resulting in cell death and disturbing vascular homeostasis [97]. Compromised endothelial function is a pivotal initial occurrence in the onset of cardiovascular disorders, including atherosclerosis and thrombosis [98,99]. A hypothesized method entails the transport of MPs into the bloodstream, leading to their accumulation in vascular tissues, where they may disrupt cellular signaling pathways.

4.4.4. Procoagulant Activity

MPs may elevate the risk of thrombosis by enhancing procoagulant activity [100,101]. Microplastics can be a substrate that induces platelet aggregation and coagulation when interacting with plasma proteins. The first visual photograph and Raman spectrum of MPs in thrombi were presented by Wu et al. [102]. This indicates that perhaps the risk of microparticle exposure was under-recognized, and not just for non-soluble particles like synthetic material. Therefore, there is an urgency to re-evaluate its health effects. This procoagulant action may elevate the incidence of cardiovascular incidents, including myocardial infarction or stroke. MPs have been shown to modify platelet morphology and elevate the expression of clotting components, hence augmenting the coagulation cascade [103,104].

4.4.5. Potential Cardiometabolic Implications

Emerging evidence indicates that MP exposure may also indirectly impact cardiometabolic health. MPs may interfere with lipid metabolism, resulting in dyslipidemia, a recognized risk factor for cardiovascular illnesses [105,106]. Previously, studies involving animal models have established that MP exposure may modify cholesterol storage and transport, leading to plaque accumulation in arteries [107–109].

4.4.6. Mechanism of Action of MPs in the Cardiovascular System

The processes via which MPs may affect the cardiovascular system are under ongoing investigation, but they may be hard to comprehend. There are several molecular pathways by which MPs can exert detrimental effects on the cardiovascular system (Figure 5). These mechanisms include the following:

Direct physical contact between MPs and endothelial cells can result in mechanical damage and inflammation and subsequently endothelial dysfunction. In addition, this interaction triggers a cascade of events such as the p53 signaling pathway and mitogen-activated protein kinases (MAPKs). The release of pro-inflammatory cytokines such as IL-1β and TNF-α exacerbates inflammation in the vascular wall, causing heart damage.

MPs frequently comprise chemical additives such as bisphenol A (BPA), phthalates, or polybrominated diphenyl ethers (PBDEs), which leach and interfere with biological transmission. These substances are endocrine disruptors interfering with hormone signaling pathways that can distress lipid metabolism and vascular function. For example, BPA binds to estrogen receptors, activating signaling pathways that disrupt endothelial function, promote oxidative stress, and impair nitric oxide production, which is essential for vasodilation. Similarly, MPs may alter the function of peroxisome proliferator-activated receptors (PPARs), which regulate lipid homeostasis. This disruption could lead to lipid accumulation in the vasculature, leading to plaque formation and increasing the risk of atherosclerotic cardiovascular disease.

Immune activation: The immune system's response to MPs, for example, macrophage activation, results in cytokine secretion and inflammatory mediators, which impair endothelial dysfunction. These inflammatory mediators contribute to endothelial dysfunction by increasing oxidative stress and inducing vascular smooth muscle cell proliferation.

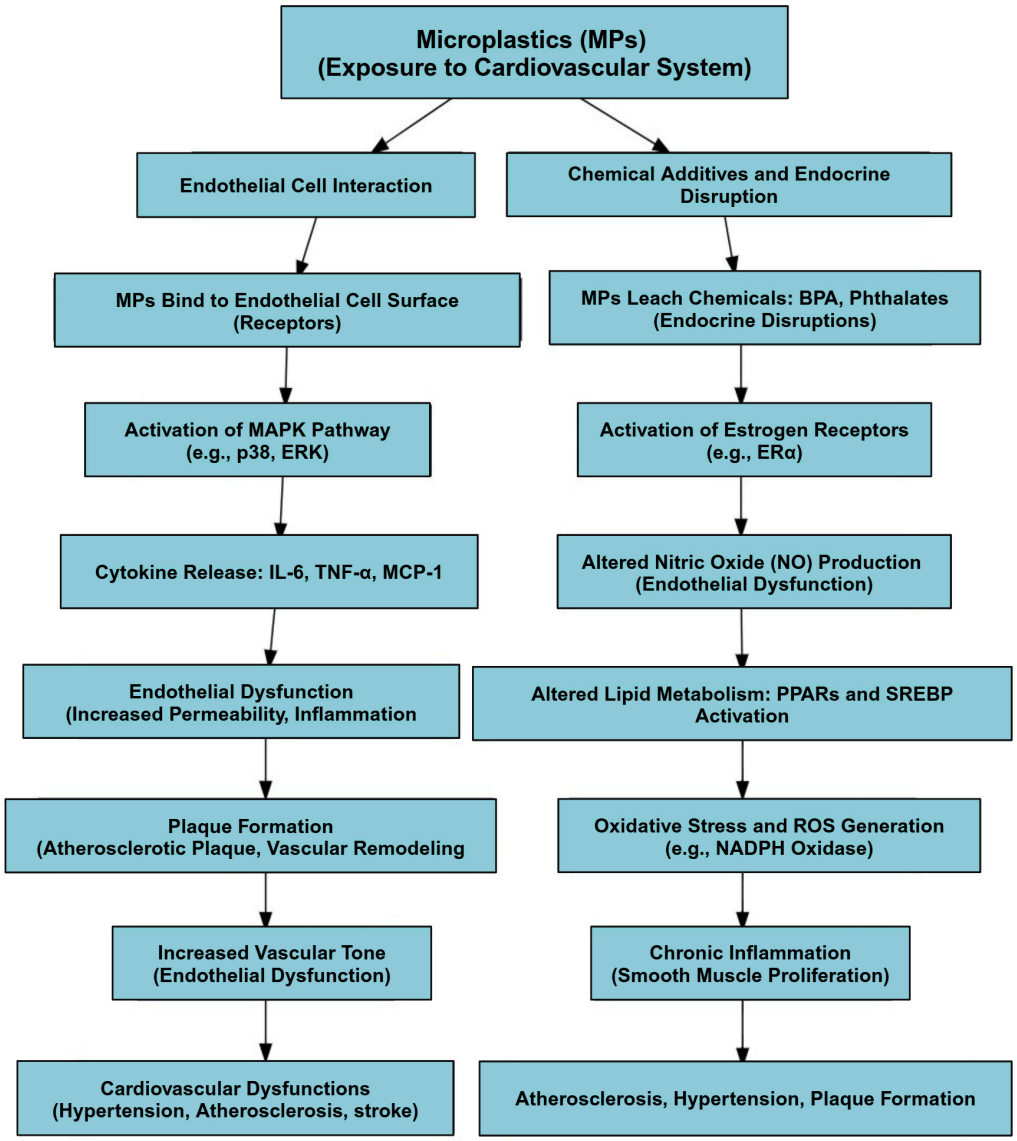

Figure 5. The schematic representation of multifaceted key molecular mechanisms of MP-induced cardiovascular dysfunction. Key processes include the binding of MPs to endothelial cell receptors and the leaching of chemical additives (e.g., BPA, phthalates) that activate molecular pathways such as MAPK (e.g., p38, ERK) and estrogen receptors (e.g., ERα). Release of pro-inflammatory cytokines (e.g., IL-6, TNF-α, MCP-1), altered nitric oxide (NO) production, and oxidative stress (e.g., ROS generation via NADPH oxidase). Dysregulation of lipid metabolism (e.g., PPARs, SREBP activation), plaque formation, and vascular remodeling lead to cardiovascular dysfunction such as atherosclerosis, hypertension, and stroke.

Chemicals linked to MPs are recognized as endocrine disruptors, which can indirectly impact cardiovascular health by modifying the hormone equilibrium of aldosterone and cortisol, influencing blood pressure regulation, and contributing to metabolic diseases.

4.5. Neurotoxicity of the Nervous System in Response to MPs

According to specific research, MPs, such as polystyrene, may affect the nervous system [110–112] and penetrate the blood–brain barrier, potentially inducing neuroinflammation and contributing to cognitive impairment [113]. Similarly, research on animals indicates that extended exposure to MPs may result in cognitive alterations and behavioral deficiencies [114,115]; however, further studies are required to elucidate these consequences in people. Neurodegenerative risk is high. Prolonged exposure to MPs may be associ-

ated with the onset of neurodegenerative disorders, mainly Alzheimer's and Parkinson's diseases [116,117].

4.6. Endocrine System Disruption in Response to MPs

Endocrine disruption is acknowledged as a possible consequence of MPs. They can encapsulate and adsorb various substances from their environment, known as endocrine-disrupting compounds (EDCs) [118,119]. These EDCs are exogenous compounds or mixtures that can disrupt the regular endocrine-related mechanism, leading to adverse health consequences [120]. Common EDCs include phthalate esters, bisphenol A (BPA), octylphenol, and nonylphenol, produced as reaction agents or additives produced from plastic [121,122]. MPs produce these EDCs upon ingestion or interaction with organisms and can interfere with the endocrine system. This disruption may harm hormonal balance, reproductive functionality, development, and general health [12,123]. Hormonal imbalances: These MPs disrupt hormone homeostasis, resulting in reproductive health complications, developmental difficulties, and hormone-associated malignancies, e.g., breast or prostate cancer.

4.7. Carcinogenic Potential of MPs

MPs may increase cancer risk through chronic inflammation. Persistent inflammation caused by MP accumulation in tissues may increase cancer risk, particularly gastrointestinal or lung cancers [124]. Chemical exposure: Some plastics, including polystyrene, contain carcinogenic chemicals like styrene, which has been classified as a possible carcinogen. Our data highlight the potential of MNPs as covert catalysts for tumor growth, particularly by augmenting cell migration and perhaps facilitating metastasis. This insight illuminates a crucial and previously underexamined concern [125,126]. MPs impair regular pulmonary cell activity, potentially facilitating the development of cancer. Following that modification, cellular metabolism decelerates processes and undermines tissue integrity. Moreover, MPs alter the microarchitecture of the lung, impairing tissue and promoting infiltration of cancer cells and tumor dissemination [127–129]. Jin et al. reported that in lung epithelial cells, MPs can provoke oxidative stress, generating ROS and pro-inflammatory cytokines [130], resulting in mitochondrial dysfunction. This surplus of generated ROS subsequently engages with cellular constituents, including proteins, lipids, and DNA, resulting in oxidative impairment and eliciting an inflammatory process [131].

4.8. Reproductive System Response to MPs

MPs, particularly those infused with chemical compounds, may adversely affect reproductive health [132,133]. Studies indicate that these MPs interfere with the neuroendocrine system, affecting the synthesis of sex hormones via the hypothalamic–pituitary–gonadal (HPG) axis. The effects of MPs on reproductive health vary depending on the type of MPs, necessitating a more nuanced understanding of their mechanisms of action. Zhang et al. reviewed recent animal studies and determined that the minimum human equivalent dose of MPs associated with impaired male semen quality is 0.016 mg/kg per day. Therefore, MPs disrupt the blood–testis barrier in the male reproductive system and lead to impaired spermatogenesis [134]. Similarly, in females, they contribute to placental dysfunction, endometrial hyperplasia, ovarian atrophy, and fibrosis [133]. Lin et al. studied the impact of polystyrene MPs (PSMPs) on the absorption efficacy and instabilities of the reproductive system induced by microcystin-LR (MC-LR) in zebrafish. When zebrafish were exposed to combined MC-LR (0, 1, 5, and 25 μg/L) and PSMPs (100 μg/L) for 60 days, increased MC-LR accumulation in gonads and exacerbated reproductive injuries were reported. Moreover, PSMPs enhanced MC-LR-induced reproductive toxicity, as evidenced by abnormal increases in 17β-estradiol (E2) and testosterone (T) levels and altered mRNA levels

of key genes in the hypothalamic–pituitary–gonadal (HPG) axis [135]. Regarding fertility concerns, endocrine-disrupting substances released by MPs may compromise reproductive function, potentially resulting in reduced fertility in both genders [136]. Virtanen et al. have reported that a notable reduction in male semen analysis parameters has been documented over the last 80 years [137]. The environmental pollutants, predominantly environmental endocrine-disrupting chemicals (EEDCs), were posited as a potential contributing factor. Spermatogenesis may be disrupted by either single or multiple exposures to endocrine-disrupting EEDCs, including di(2-ethylhexyl) phthalate (DEHP), heavy metals, and various phosphorus pesticides [138].

Similarly, developmental concerns in offspring are caused by exposure to MPs during gestation, which may impact fetal development [139,140]. Research using animals indicates that maternal MP exposure may reduce birth weights and developmental complications in progeny. For instance, the dose rate used at 1.357 μg/g bodyweight was administered via gavage to study the effects of 1 μm PSMPs across multiple generations (F0, F1, and F2). This resulted in disrupted reproductive cycles, diminished fertility, hormonal imbalances, and structural and functional abnormalities [141]. MPs have been found to disrupt the reproductive endocrine system by disrupting the HPG axis [142]. Following exposure to PSMPs, male mice exhibited decreased serum levels of follicle-stimulating hormone, luteinizing hormone, and testosterone, alongside increased estradiol levels. Conversely, the serum levels of FSH and T in female mice showed a reversal of these changes [143]. As a result, the potential reproductive endocrine disorder caused by MPs may be due to interference on the HPG axis and reduced steroidogenesis that could distort sex hormone balance, thereby delaying gonad maturation. This may reflect a possible way by which MPs challenge embryos and progeny growth. Despite the alarming findings concerning the latent detrimental impacts of MP compounds, there is limited knowledge of the leaching properties of these different polymers and their probable adverse effect on human health. Future research should focus on elucidating the mechanisms of MP-induced reproductive toxicity and identifying strategies to mitigate these effects.

4.9. Skin and Dermal System

While cutaneous exposure to MPs is deemed a less critical route of exposure relative to ingestion and inhalation, possible issues predominantly remain when the context is occupational and cosmetic. This section examines the mechanisms and outcomes of dermal exposure to MPs. Research on MPs reveals several mechanisms contributing to oncogenic potential over the dermal system. These tiny MPs exhibit extraordinary capability to penetrate cellular and physiological blockades, inducing toxicity effects across multiple levels [144,145]. Mechanistic experiments involving MPs at the micro-level have demonstrated that they may induce elevated amounts of ROS in carcinogenic cells of the skin. This ROS is demonstrated to induce danger-associated molecular patterns. Additionally, it is related to the interruption of the inflammation process, toll-like receptors, and cytokine synthesis [146,147]. Shi et al. observed that concurrent exposure of mice to polystyrene and DEHP inhibited cutaneous wound repair and induced inflammatory response and oxidative stress [148].

Chronic exposure to MPs elicits persistent inflammation all over the body systems, involving numerous cytokines, thereby fostering a pro-carcinogenic milieu [77,78]. Inflammatory responses not only facilitate the initiation and proliferation of tumors but also enhance invasion, angiogenesis, and metastasis. Hence, these are critical characteristics of destructive skin cancer morphologies. Furthermore, MPs have been demonstrated to interfere with immune-related mechanisms, hindering the immune system's capacity to identify and eradicate cancer cells [149,150]. MPs in cosmetics can induce skin irritation,

especially in those with sensitive skin or those subjected to elevated concentrations of MPs [151–153].

4.10. Scenarios with Negligible Health Impacts and Adaptive Responses

4.10.1. Scenarios with Negligible Health Impacts

The potential of MP hazards is well-reported, but it is critical to recognize that not all MP exposure scenarios could lead to significant health impacts. For instance, under certain circumstances, MPs might have negligible effects, or biological systems may adapt to mitigate damage. Hence, it is important to understand these scenarios to assess the MP toxicity better. Considering low MP exposure, MPs may not have sufficient accumulation in tissues. For instance, research carried out by Zheng et al. reported that tissue accumulation of PSMPs in *Litopenaeus vannamei* confirmed the sublethal impacts [154]. In human kidney cells, though PSMP exposure reduced cellular proliferation, cell viability was not significantly decreased [155]. Similarly, in humans, occasional exposure to MPs through food or water might not exceed the thresholds required to trigger adverse effects, mainly when the immune system effectively clears those particles [156].

4.10.2. Adaptive Responses to MP Exposure

Antioxidant defense in response to MP exposure plays a critical role. To counter the oxidative stress induced by MPs, antioxidant enzymes, like catalase and superoxide dismutase (SOD), are upregulated by cells of aquatic organisms [10]. Similarly, the immune system could adapt to MP exposure to modulate inflammation. For example, chronic low levels of PSMPs could lead to an immune response, which reduces the excess inflammatory progress [157]. Furthermore, the tissue repair mechanism, like cell proliferation, is activated in response to MPs, which counters the oxidative stress and inflammation [158].

As MPs have posed potential health risks, it is significant to identify those biological systems that have evolved to handle environmental challenges such as exposure to foreign particles. Thus, adaptive responses might effectively mitigate the risks associated with MP exposure. But the need for caution is not negated by this. Therefore, to acknowledge the risks and resilience of the biological system, a balanced approach is much needed.

5. Regulations on MPs and Health Protection

Regulations aimed at mitigating the MPs' environmental and health effects have been globally implemented, encompassing polystyrene, polyethylene, polypropylene, and other prevalent varieties. While these regulations represent important steps forward, their effectiveness varies, and more comprehensive measures are needed to address the global crisis caused by MPs.

Regulations on Polystyrene Bans on Expanded Polystyrene (EPS): Various jurisdictions, including the European Union, Canada, and U.S. states such as New York and California, have prohibited EPS in food containers due to its role in MP pollution.

Alternative Categories of MPs Regulations

Microbead prohibitions: Nations such as the United States, United Kingdom, and Canada have enacted bans on microbeads in personal-care items to mitigate primary MP pollution. This enactment was placed when the Microbead-Free Waters Act (MFWA) of 2015 banned microbeads in personal-care products. However, MFWA varied significantly across different regions, leading to inconsistencies in implementation timelines, potential loopholes, and restrictions [159].

Packaging and waste regulations: The European Union (EU), Canada, and some U.S. states have limited single-use plastics, specifically polyethylene and polypropylene goods, to mitigate the degradation of secondary microplastics into the environment. The EU

introduced EU Directive 2019/904, known as the Single-Use Plastics Directive (SUPD), to prohibit certain single-use plastic items. The SUPD imposes rules like extended producer responsibility and mandatory product redesign for other plastic products [160].

Regulations on tires and textiles: Current research and policy aim to mitigate MPs from tire degradation and synthetic fabrics by enhancing road runoff management and implementing washing machine filtration systems. The current regulations represent a critical way forward; addressing the MP crisis requires a comprehensive approach that combines policy, technology, and public engagement.

6. Conclusions and Future Perspective

In conclusion, this review highlights the universal presence of microplastics (MPs) in the environment, their potential human health impacts, and the urgency for coordinated action to mitigate these risks. MPs, tiny plastic particles measuring less than 5 mm, have emerged as a significant environmental and public concern due to their widespread presence in ecosystems and potential to infiltrate human tissue. MPs originate from various sources, including plastic waste, synthetic textiles, and personal-care products, and have been increasingly detected in human environments (including the food chain), water, and air. MPs may harm human health, trigger inflammation, and cause cellular stress. Evidence is mounting that MPs can have global consequences on the general population by possibly inciting inflammatory responses leading to chronic diseases. They can interfere with endocrine function, suppress immune responses, and serve as vectors for harmful chemicals and pathogens. Though the concrete links to certain diseases are still in limbo, it follows that MPs accumulating on and within you is not good. Reduction in plastic pollution and additional research into toxicity will be vital for reducing potential health risks that may not yet be fully understood with regard to its impact on human well-being.

It is important for potential future studies to further investigate the impact of MPs on human health as well as to elucidate their mechanisms in biological systems and assess possible risks regarding certain diseases. Improved identification will help in better tracking of MPs within the human body, and long-run studies may establish additive health effects. Moreover, investigation of the association between MP exposure and long-term ailments such as cancer or cardiovascular pathologies will be crucial.

To address this growing global threat of MPs, a multifaceted approach is needed, combining technological innovation, policy interventions, and public engagement. The use of alternative eco-friendly materials and strategies that reduce plastic consumption could provide solutions to environmental MP pollution. Society as a whole can take steps to combat the potential long-term health effects of MPs by focusing on prevention and educating the public. Thus, with the implementation of robust policies, technological advancements, and fostering public awareness, we could be able to reduce MP pollution and safeguard humans. The time to act is now, before the consequences of MP exposure become irreversible.

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