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Review

Micro- and Nanoplastics on Human Health and Diseases: Perspectives and Recent Advances

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Abstract

Micro- and nanoplastic (MNP) particles are constantly formed through plastic fragmentation by sunlight, friction, or oxidation. MNPs potentialize health risks when entering the human body by ingestion, infusion, inhalation, and skin absorption. Still, the translocation among intracellular compartments must also be considered because MNPs can reach the circulatory system and be found in virtually all body fluids, tissues, and organs, potentially causing significant health impacts. The ability of MNPs to interact with macromolecules and cause damage to intracellular structures results in several physiopathological conditions, such as inflammation, oxidative imbalance, apoptosis, and carcinogenesis. One major challenge in MNP research is the development of reliable detection and quantification methods and effective sample separation processes. Although there is evidence directly linking MNPs to heart disease, the same cannot be said for diseases such as cancer, respiratory conditions, and reproductive system disorders. Therefore, the impact of MNPs on human health was examined, and a careful evaluation of their effects was carried out. We reviewed the extensive scientific literature from the past years, focusing on exposure, aging, interactions, and effects on entering MNPs into human metabolism and the physiological systems, which makes these particles particularly hazardous.

Keywords: plastic additives; inflammation; apoptosis; carcinogenesis; neurotoxicity

Academic Editor: Javier Bayo

Received: 19 June 2025
Revised: 8 August 2025
Accepted: 2 September 2025
Published: 12 September 2025

Citation: de Souza, A.S.; Ferreira, P.G.; Pereira, P.R.; de Jesus, I.S.; de Oliveira, R.P.R.F.; de Carvalho, A.S.; Rodrigues, L.C.D.; Paschoalin, V.M.F.; Futuro, D.O.; Ferreira, V.F. Micro- and Nanoplastics on Human Health and Diseases: Perspectives and Recent Advances. *Microplastics* **2025**, *4*, 64. https://doi.org/10.3390/microplastics4030064

1. Introduction

Plastics were once considered inert materials, but recent reports have shown they represent health risks to humans because they can be degraded into micro- and nanoplastics (MNPs), becoming spreadable in air, water, and soil. Because plastic materials are light and durable, produced at low cost, and widely used for various purposes, the disposal of plastic materials is poorly managed and easily discarded. The manufacturing and utilization of plastics have increased annually due to irrational consumption driven by improved consumer purchasing power. Besides this, technological developments, allied with a lack

of awareness about plastic, a pollutant material, lead to poor waste management since less than 35% of plastic waste is recycled. Worldwide, more than 330 million tons of plastic are produced annually, with a considerable increase in production during the COVID-19 pandemic from 2019 to 2021 [1]. It is estimated that there are already 4.9 billion tons of plastic waste of varying sizes and chemical compositions, ubiquitous to all natural habitats, and these materials are spread in terrestrial and aquatic ecosystems [2]. In 2050, predictions indicate that this amount should increase by 12 billion metric tons [3].

Herein, we provide a comprehensive overview of the current evidence regarding micro- and nanoplastics (MNPs) in human tissues and biological fluids. Additionally, it explores the proposed mechanisms underlying MNP toxicity, including inflammatory responses, oxidative stress, and cellular dysfunction. Given the growing concern about the potential health implications of MNP exposure, we also discuss the limitations of current analytical methods and highlight key knowledge gaps that need to be addressed in future research. By synthesizing findings across different disciplines, this review seeks to clarify the biological relevance of MNP contamination and promote a more informed assessment of associated risks to human health.

MNP particles have been distributed worldwide for a long time, but they are being considered emerging pollutants, and their potential health risks have been assessed [4]. These particles are an emerging global environmental contaminant that affects living beings and ecosystems; however, little is known about the effects of MNP exposure and absorption by the human body [5]. The number of studies on the impact of these particles has been increasing, with emphasis on the polymers polyethylene (PE), polypropylene (PP), polyethylene vinyl chloride (PVC), polyethylene terephthalate (PET), and polystyrene (PS), which are the most produced plastics (Figure 1) [6,7]. Most discarded plastics are PS and PP, materials that could enter the circular economy [8]. However, the microparticles derived from biodegradable polylactic acid (PLA) also showed ecotoxicity against the mussel Mytillus coruscus, commonly used as a bioindicator organism [9].

Over 98% of plastics are produced from fossil sources. In addition, thousands of chemicals are added to polymers to impart specific properties such as color, flexibility, stability, water repellency, flame retardancy, and UV resistance. The critical environmental aspects of plastics led to the creation of the Minderoo-Monaco Commission that assesses these materials' impacts on human health [10]. Based on this, 175 nations have agreed to establish a legally binding international agreement to eliminate plastic pollution during the 2022 World Economic Forum [11].

The term "microplastics" (MPs) was first proposed by Thompson et al. in 2004 and refers to plastic particles with a size >1 μm and <5 mm, where nanoplastics (NPs), in turn, are defined by the European Food Safety Authority (EFSA) as <1 μm particles [12]. The primary source of MPs and NPs in the environment is the improperly discarded plastic devices, transported to rivers and oceans, and then fragmented mechanically or by solar irradiation in nature (Figure 1) [13].

One of the significant challenges in advancing our understanding of nanoplastics (NPs) lies in their detection, analysis, and classification. Unlike microplastics, which are relatively more straightforward to isolate and characterize, NPs often fall below the detection limits of conventional analytical techniques and require highly specialized equipment and protocols. Consequently, the number of studies focusing exclusively on nanoplastics remains limited.

Despite this analytical gap, current evidence strongly suggests that NPs coexist with microplastics in environmental and biological matrices and may pose even greater risks due to their smaller size, larger surface area, and enhanced cellular uptake and systemic distribution potential. The lack of robust and standardized methodologies for isolating and distinguishing NPs from complex matrices impedes comprehensive toxicological evalua-

tions and contributes to their underrepresentation in the scientific literature. Overcoming these analytical limitations is essential to fully elucidate the actual impact of nanoplastics on human health and the environment.

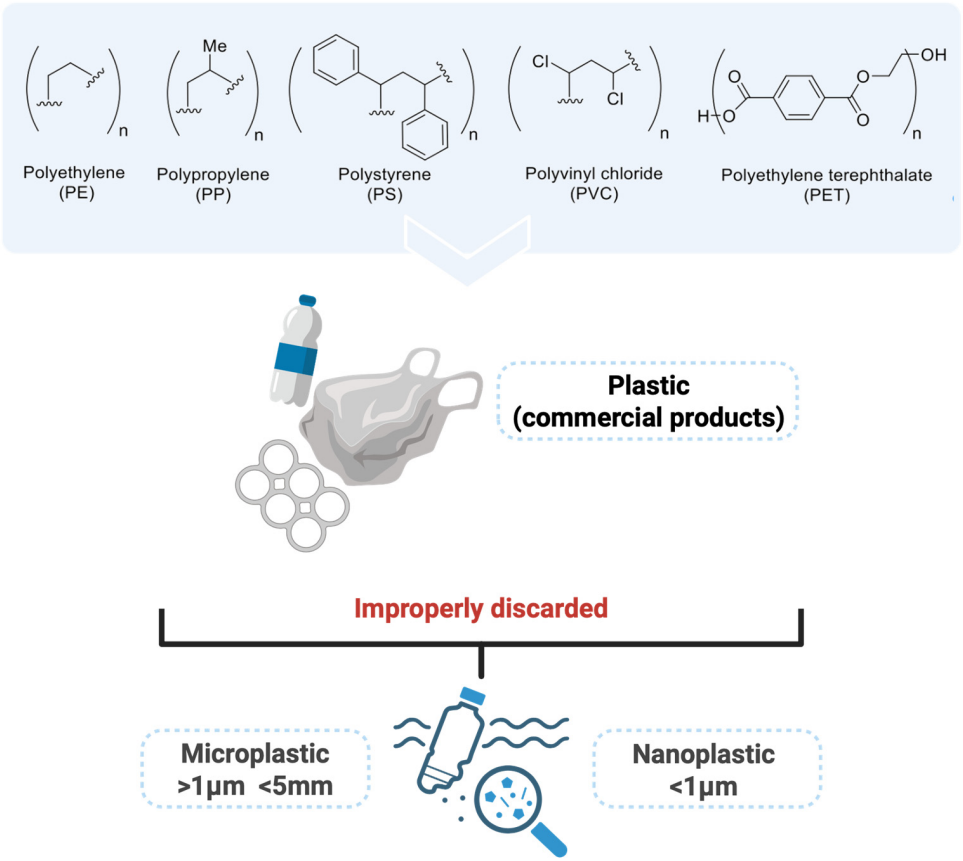

Figure 1. Main polymers that make up plastic materials and their classification by size. Created in https://BioRender.com.

There are large amounts of MNPs below the surface of the Atlantic Ocean, highlighting the need for an in-depth assessment of the risks of plastic pollution since these tiny particles are dangerous to the health of all living organisms, including humans [14]. Those MPs, distributed in the human body, can cause several physiopathological conditions and the development of diseases, requiring studies that reveal how MPs interact broadly with components of the biological systems (Figure 2) [15].

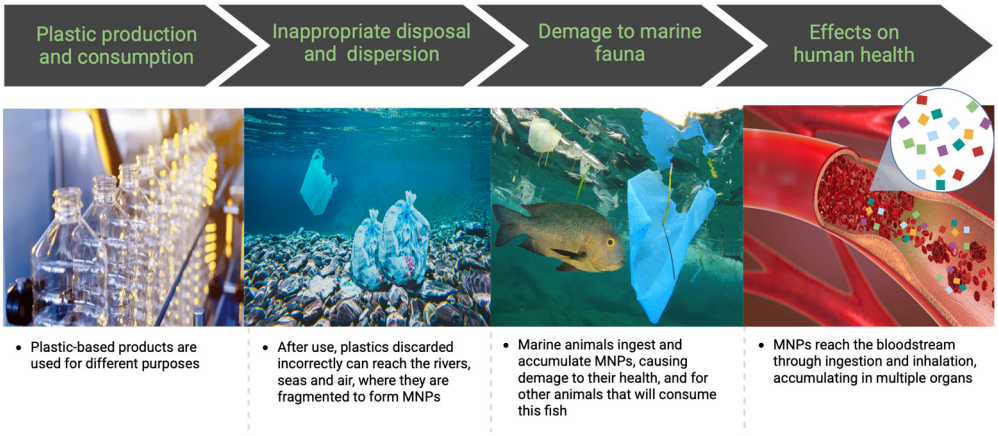

Figure 2. MP and NP chain impact on human health. Different color blocks represent MNPs. Created in https://BioRender.com.

2. MPs in the Environment

MPs have become a significant environmental concern as these contaminants accumulate and persist in all ecological compartments: air, water, soil, and even living organisms [16–19]. In addition, additives used to improve the properties of plastics and other toxic chemicals used for manufacturing are also carried by these particles, causing health problems [20]. Soil pollution by non-biodegradable MPs dates back to the widespread daily use of plastics. Over approximately 70 years (1950–2020), an enormous amount of plastic has accumulated on Earth, initially settling in the soil before being transported to water bodies and spreading in the air. There is now a concerted effort to trace better MPs in soils, including their sources, migration, distribution, biological effects, degradation, and the methodologies used to analyze them. The most frequently found MPs in soils are PE (78.8%), PP (78.8%), and PS (45.5%) [21]. In farming activities, MPs enter the agriculture chain through water, soil, silt, organic waste, fertilizers, and airborne precipitates, negatively impacting agricultural production as they alter the soil microbial population and reduce nutrients, affecting plant growth [22–26].

There are two main routes for particles of MNPs to reach the environment: (i) particles formed during the production of plastics following direct human activity, and (ii) products of the decomposition of plastics or larger plastic fragments incorrectly discarded after usage [27]. These particles cause enormous economic losses in various sectors, including maritime transportation and fishing activities. Five trillion particles of MPs float in rivers and oceans or are deposited on beaches. Due to their size, they enter the food chain of the human beings that live in these habitats, but more than this, they affect all people who consume fish or seafood [28,29]. Surprisingly, the skin-exfoliating polymeric microspheres, an ingredient found in cosmetic products such as shampoos, toothpaste, soaps, and creams, are the new source of environmental contamination by MNPs.

Despite the advantages of recycling MPs, recycling facilities can generate a significant load of MPs, estimated at 59 and 1184 tons annually [30]. Figure 3 shows some MPs of varying sizes collected from the sands of Praia das Dunas in Cabo Frio, RJ-Brazil, on 21 June 2024.

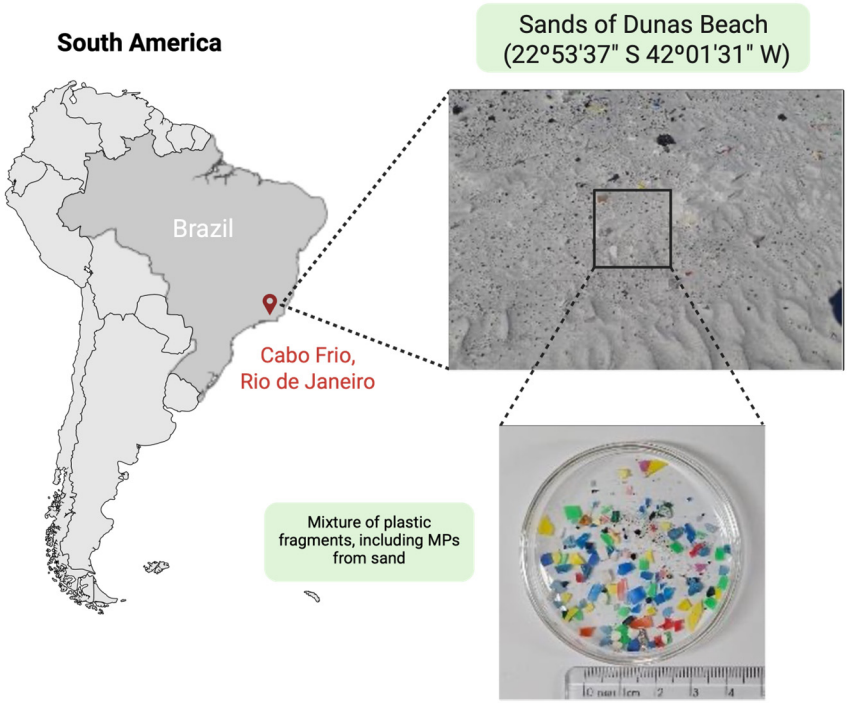

Figure 3. Plastic fragments, including MPs, collected from the sands at Praia das Dunas in Cabo Frio, RJ, Brazil (22°53′37″ S 42°01′31″ W). Created in https://BioRender.com.

The toxicity of MPs to humans has not yet been fully clarified. However, they are increasingly recognized as a significant public health concern, as a potentially hazardous factor associated with several physiopathological conditions and diseases, including disorders in lipid metabolism, inflammatory response, oxidative imbalance status in the organism, cancer, and heart diseases [31–35]. Once in a human body or even other living organisms with a circulation system, NPs can be indistinctly distributed to every organ or tissue. Still, these plastic particles can penetrate the core of lipid bilayers. As discussed, several hazardous chemical compounds associated with those NPs can be vectorized to plasmatic or intracellular membranes in living organisms [36].

Humans can be exposed to MPs through the inhalation of particles present in the air (especially in metropolitan cities), ingestion of contaminated water and or food, or through absorption by the skin, and also, less frequently, but not more critically, by tubing of transfusion therapies (Figure 4) [37–40]. After entering the human body, MPs and NPs reach the bloodstream, are distributed, and can accumulate in various tissues and organs [41]. MPs and NPs were detected in the oral, anal, and/or uterine/vaginal cavities, which are directly accessible because they are in contact with the external environment [42]. However, NPs can be translocated to any tissue or cell since their size allows them to be diffused into the lipid environment of both plasmatic and intracellular membranes.

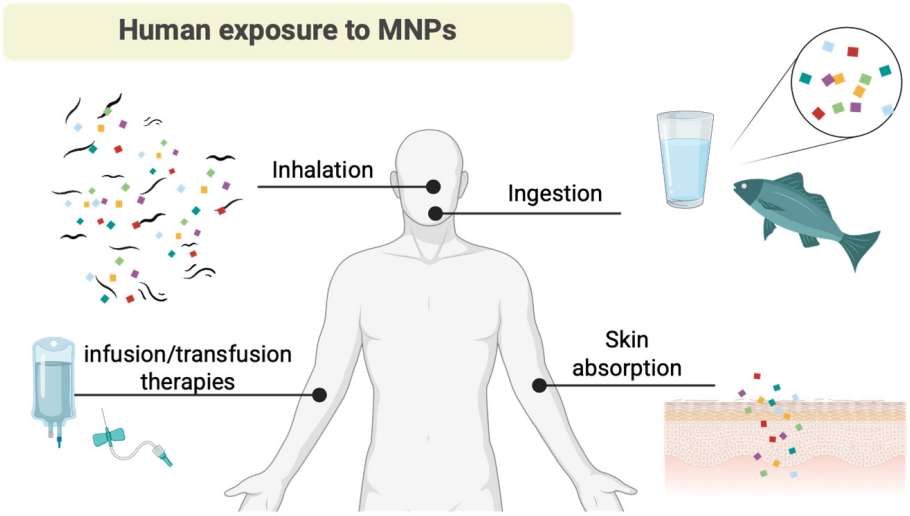

Figure 4. Routes of human contamination by MNPs. Different color blocks represent MNPs. Created in https://BioRender.com.

The ingestion of MNPs is a particular concern because of the presence of plastic particles in fish and seafood, which are an essential part of a balanced human diet, as already mentioned, but also for birds and mammals, which can be a source of MNPs for human beings. Fish tend to ingest plastic particles of specific colors, such as white, yellow, and blue [43]. However, the shape of MPs also plays an essential role in ingestion by aquatic animals since some of them are very attractive; for example, fiber-type MPs resembling worms and eggs are often accidentally ingested by fish [44]. When consumed by other animals, these contaminated fish transfer their load of MPs and NPs to their predators, including humans. MPs were found in the stomach content of neotropical fishes in Brazil from the Paraná river basin. Microscopic analysis of 220 individuals belonging to 14 species was conducted, and the results indicated the presence of small amounts of plastic particles of different shapes measuring from 1 mm to 3 mm [45]. MPs were also found in the gastrointestinal tract of fish specimens (18.1–34.5%) harvested in the middle Uruguay River in southern South America, and several species were collected in the rivers from the Amazon Basin, Brazil [46].

It is important to note that the qualitative and quantitative analyses of MPs in waterbodies still require optimization. No international standards are established for sampling, extraction, or detection of these contaminants, leading to inconsistent results in several reports [47,48]. After reviewing several studies, Koelmans and colleagues have proposed more appropriate methodologies for MPs' quantitative evaluation in waterbodies [49]. In the later study, these researchers quantified the exposure limits and effects using probability density functions that describe the diversity of the MP particles [50].

Quantitative data, including information on MPs, were compiled from 26 studies, yielding 402 data points representing over 3600 processed samples. Microplastic contamination via inhalation was evaluated using reported airborne MP concentrations and respiration rates, as provided by the U.S. Environmental Protection Agency (EPA) [51]. Regarding dietary exposure, MPs were found in food items consumed daily, such as seafood, sugar, salt, honey, alcohol, and tap and bottled waters. Based on the collected data, it was estimated that humans consume between 39,000 and 52,000 MPs annually, depending on their age and sex. If the inhalation route is considered, the estimate increases to between 74,000 and 121,000 MNPs annually [52]. However, the authors believed that these numbers are underestimated due to the methodology used and highlighted that individuals who consume bottled water may be ingesting nearly 90,000 additional MPs, compared to the 4000 MPs ingested by those who consume piped water.

The zebrafish (Danio rerio), an animal model organism sharing 70% of its genome with humans, is widely used to study blood diseases and carcinomas, serving as a toxicological model. Zebrafish exposed to MPs had their metabolome and gut microbiome evaluated, revealing gut inflammation, unbalanced oxidative conditions, and lipid metabolism disorders [53]. Within this context, Rahman et al. assessed the toxicity and cell viability of lung epithelial cells from mice under the effect of 11 types of MPs, prepared in the laboratory from commercial plastics [54]. The small-sized particles (1–5 μm) from PE and PET, prepared from disposable water bottles, induced maximum toxicity. These PET microparticles induced activation of the interferon signaling pathway because they were perceived by immune cells similarly to pathogens. PET-MPs with the smallest size and heterogeneous shapes induced cell injury, triggering the inflammatory cascade, DNA damage, and cell death, which are characteristic of tissue injury in vivo.

A significant concern is the MPs' potential to cause teratogenic effects. The teratogenic impact of exposure to plastic particles on the diatom Cocconeis placentula and the cnidarian Hydra vulgaris belongs to different trophic levels. A moderate teratogenic risk index was calculated for diatoms, even at a concentration of 0.1 $\mu g \cdot mL^{-1}$ of plastic particles; however, a low teratogenic risk was estimated for *H. vulgaris* even at the highest concentrations, but a slower regeneration was observed for these organisms. The study highlighted that the teratogenic effect depends on the concentration of plastic particles and can vary according to the habitat of the trophic level. However, the correlation between specific types of NPs or MPs and particular teratological forms was poorly evaluated [55].

The association of MPs with pesticides poses a significant threat to soil organisms such as earthworms (*Eisenia fetida*) that have been individually exposed to PE and PP, causing adverse effects on earthworms' digestive tract and side effects on soil ecosystems [56].

In addition to the concern about the effects of MPs and the human health risks, the additives used in plastic manufacture are also of concern. Additives are chemical compounds added to improve functionality and/or loading and are waste by-products of plastics production. The chemical bonds between these additives and polymers are weak supramolecular interactions and are, therefore, released unintentionally during environmental exposure. There are approximately 2712 known plastic additives, some of which are considered carcinogenic, causing DNA damage, apoptosis, immune system

impairment, and certain types of cancers, including endocrine, biliary tract, pancreatic, and hepatocellular carcinomas. However, many of these compounds remain untested. Human exposure to MNPs presents risks from the plastic particles and the toxic additives they carry, which can spread throughout the body [57].

3. Biological Effects of Polystyrene Particles (PS-MPs and PS-NPs)

Styrene, the monomer used to produce PS, is classified as a potentially carcinogenic substance (carcinogenicity class B2) by the International Agency for Research on Cancer (IARC), and for this reason, the micro- and nanoparticles originating from PS are considered [58]. Indeed, the binomial particle size and composition/surface modifications must also be considered to evaluate the toxicity of plastic micro- or nanoparticles. Several studies were conducted to estimate the hazardous effects of micro- and nanoplastic particles, as the most dangerous types of MNPs, mainly due to their ability to interact with a wide range of organic macromolecules.

Recent studies involving MPs and NPs of PVC were tested on a triple culture model (Caco-2/HT29-MTX-E12/THP-1), mimicking the inflamed human intestine. The exposure to PVC particles during an active inflammatory process on gut cells was found to augment the release of IL-1β cytokine, causing the death of epithelial cells (Figure 5). No acute toxic effects were seen in the model of the healthy intestine following PS or PVC exposure. Busch et al. and Hesler et al. concluded in their respective works that PS-MPs did not present acute effects in the healthy gut model since they do not cross the intestinal and placental barriers and are weakly embryotoxic and non-genotoxic in vitro [59,60]. Although the polymer has different chemical characteristics from the styrene monomer, its use has raised several concerns. The PS-MPs and PS-NPs accumulate along the food chain, promoting adverse effects on living organisms [61]. The photodegradation of PS-MPs increases its toxicity to marine organisms, disrupting hepatic lipid homeostasis and consequent liver injury, besides causing inhibition of growth [62]. Similarly, earthworms were more sensitive to freeze–thaw aging MPs that caused oxidative imbalance in those organisms, changing their microbiota and possibly impacting the benefits of these invertebrates to the soil [63].

The tools used to predict the harmful effects of organic compounds are essential for the design of substances and are very efficient in predicting the toxicological profile of MPs. Three artificial intelligence algorithms that can predict the behavior of these particles on cells have identified particle size as the most critical characteristic contributing to the adverse effects [64].

The most recent data indicate that PS is among the most dangerous MPs, as it can be associated with several organic macromolecules. Jones et al. conducted a critical literature review to address the question, "Are micro- and nanoplastics toxic when ingested?" [65]. Their findings indicate that PS-MPs are among the most toxic in vitro and in vivo, causing inflammation, altered cell proliferation, dysregulation of cell membrane permeability, and impairments in lipid and amino acid metabolism, ultimately leading to apoptosis. It is important to note that PE and PP also disrupt lipid metabolism and that PS is often ingested alongside other polymers.

Regarding size, particles < 10 μm pose the greatest danger, both in vitro and in vivo, suggesting that the chemical composition and size of MNPs are critical factors in their toxicity. This observation aligns with the known risks associated with other types of particles. In addition to the significant biological potential of MNPs, Awet et al. identified the harmful effects of PS-NPs on the environment [66]. Their study, which evaluated the antimicrobial impact of PS-NPs on the native soil microbiome, suggests that PS-NPs may pose potential environmental risks. For 28 days, the activities of dehydrogenases, N-(leucine-aminopeptidase), P-(alkaline phosphatase), and C- (β-glucosidase and cellobio-

hydrolase) were significantly reduced, demonstrating the impact on the physiology and functionalities of soil microbiota.

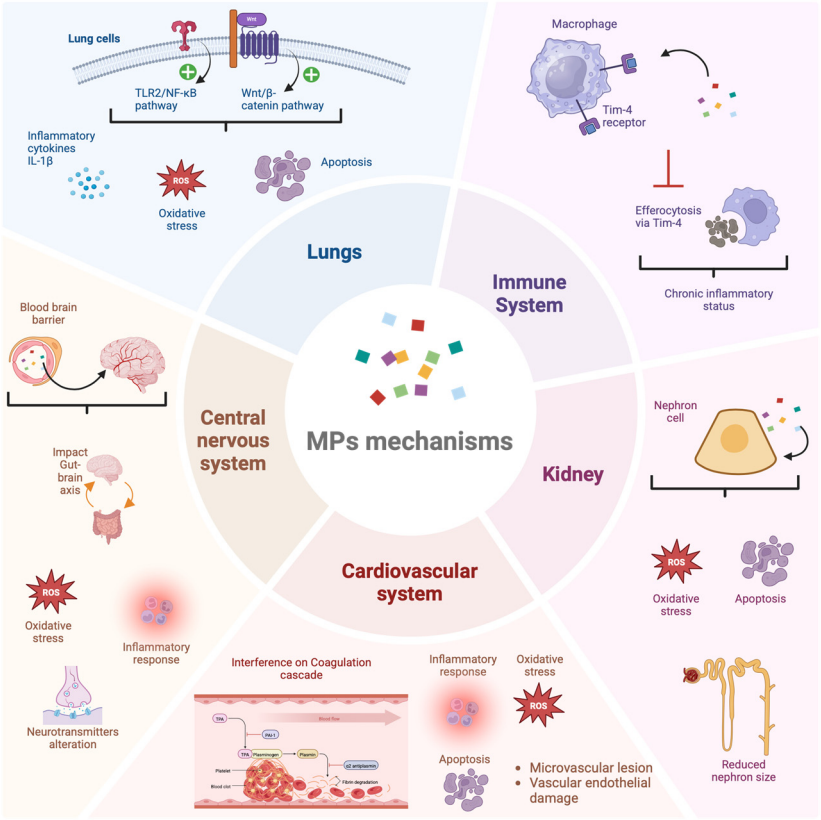

Figure 5. Some biological mechanisms triggered by MNPs in the cardiovascular system, the central nervous system, the immune system, the kidney, and the lungs, based on in vitro and in vivo studies. Arrows in immune system and kidney panels indicate MPs interaction with receptor or internalization. Created in https://BioRender.com.

Dong et al. used healthy human lung epithelial cells (BEAS-2B) in culture to investigate the relationship between lung toxicity and PS-MPs [67]. The results showed that these MPs cause cytotoxic and inflammatory effects on epithelial cells by increasing the production of reactive oxygen species and decreasing transepithelial electrical resistance, raising the risk of chronic obstructive pulmonary disease (COPD). In a further study by Danso et al. on the pulmonary toxicity of PS, PP, and PVC using three strains of mice (C57BL/6, BALB/c, and ICR), it was found that PS exposure led to an increase in inflammatory cells by stimulating the release of inflammatory cytokines in the bronchoalveolar lavage fluid of C57BL/6 and ICR mice (Figure 5) [68]. These findings suggest that inhaling PS-MPs poses a significant risk to pulmonary function, as Cao et al. described lung lesions in mice chronically exposed to PS-MPs. The lesions were induced by activation of the TLR2/NF-κB pathway, triggering inflammation and oxidative stress in lung cells, which aggravates apoptosis and induces pulmonary fibrosis in mice (Figure 5) [69]. Wu et al. proved the protective antioxidant capacity of N-acetylcysteine against PS-NPs in mice by reversing the pulmonary toxicity of PS-MPs [70]. Li et al. evaluated different approaches to induce pulmonary fibrosis in mice through oxidative stress and activation of the Wnt/β-catenin signaling pathway (Figure 5) [71]. Their findings demonstrated that inhalation of these MPs triggered fibrosis dose-dependently by elevating oxidative stress in the lungs. Notably, treatment with melatonin (50 mg/kg) mitigated the lung damage caused by PS particles.

Kuroiwa et al. studied how phagocytes recognize MPs using Tim4, a surface protein receptor that plays an important role in the immune system [72]. Phagocytes absorb MPs,

and it is possible that, in the case of PS-MPs, a supramolecular pi-stacking interaction occurs between the benzene aromatic rings of PS and mucin 4 of Tim4 immunobiological cells. This may represent a new interface between MPs and biological systems through aromatic–aromatic interactions. As Tim4 mediates efferocytosis through its PtdSe-binding site, the macrophage engulfment of apoptotic cells can be blocked entirely by PS-MPs because the aromatic cluster of Tim4 is fully occupied by the PS-MP binding, perturbing efferocytosis (Figure 5). Conversely, the authors have considered that continued exposure to PS microparticles can generate a chronic inflammatory status, predisposing individuals to autoimmune diseases.

Garcia et al. investigated the ingestion of various concentrations of MPs and how they affect the functioning of metabolic pathways within the colon, liver, kidney, and brain in mice [73]. PS was administered in separate groups at concentrations of 0.2 or 4 mg/week, and light microscopy and spectroscopy were used to analyze the serum and tissues of the brain (prefrontal cortex), liver, kidney, and colon for the presence of MPs. The liver was the organ that concentrated the particles the most, while the kidneys had the lowest concentration. These results suggest that the particles pass the intestinal epithelium, enter the circulatory system, and deposit in the organs.

Bingrui et al. studied the effect of exposure to PS-MPs at various stages of nephrogenesis using human renal organoids, three-dimensional structures grown in the laboratory, i.e., in vitro [74]. MPs with a size of 1 µm adhered to the cell surface during the Nephron Progenitor Cell (NPC) stage and accumulated within glomerulus-like structures of renal organoids. This resulted in organoids with reduced nephron size, increased production of reactive oxygen species, apoptosis, decreased cell viability, and reduced NPC (Figure 5). However, the adverse effects of exposure to these PS-MPs on the human renal system remain uncertain, as there is no model for a proper comparison.

All these works undoubtedly point to the real danger that MNPs derived from PS pose to human health and the environment. In this way, a discussion can be opened about the alternatives for replacing this material and its gradual removal from contact with humans and the environment, especially as packaging or containers for food and plastic products with a very short shelf life.

Table 1 summarizes the data related to the interactions of PMs and NPs with the mammal organisms, discussed in this review, such as reported sample types, MP/NP polymers, and detection methods.

Table 1. Occurrence of MPs and NPs in mammalian organs, tissues, and/or fluids.

Sample	MP/NP Polymers	Analysis Method	Concentration or Particle Size	Reference
Sperm mice	PS	-	80 nm (NP) 5 µm (MP)	Wen et al. [75]
Human blood from a transfusion therapies	PE, PA, PS, and PC	-	4 to 148 µm (MP)	Zhu et al. [76]
Human amniotic fluid and/or placenta	PVC and calcium–zinc PVC	Attenuated total reflectance–Fourier transform infrared spectroscopy (ATR-FTIR)	10 to 50 µm (MP)	Halfar et al. [77]
Human placenta and fecal meconium	Ten types; mainly PE, PP, and PS	Fourier transform infrared microspectroscopy	Qualitative	Braun et al. [78]
Human placenta	PP, PVC, and PBS	Laser direct infrared spectroscopy (LD-IR)	0.28 to 9.55 particles/g; 20.34 to 307.29 µm	Zhu et al. [79]
Human breast milk	PE, PVC, and PP	Raman Microspectroscopy Detection	2 to 12 µm (MP)	Ragusa et al. [80]

Table 1. *Cont.*

Sample	MP/NP Polymers	Analysis Method	Concentration or Particle Size	Reference
Human endometrium	PA, PU, PET, PP, PS, and PE	Raman Microspectroscopy Detection	2 to 200 µm (MP)	Qin et al. [81]
Residues from human cardiac surgery	Nine types of MPs detected	Laser direct infrared chemical imaging and scanning electron microscopy	184 to 469 µm (MP)	Yang et al. [82]
Human cirrhotic liver	Six types of MPs detected	Fluorescence microscopy and Raman spectroscopy	4 to 30 µm (MP)	Horvatits et al. [83]
Sperm from dogs and humans	PE	Pyrolysis gas chromatography associated with mass spectrometry (Py-GC/MS)	112.63 mg/g in dogs; 328.44 mg/g in humans	Hu et al. [84]
Human colectomy	Polycarbonate, PA, and PP	Stereo and FTIR microscopes	28.1 particles/g of tissue	Ibrahim et al. [85]
Human colorectal adenocarcinoma	PE, MMA, and PA	ATR-FTIR and Raman spectroscopy	1 nm (NP) to 1.2 mm (MP)	Cetin et al. [86]
Neuronal tissues	PS	-	5 µm to 20 µm	Prüst et al. [87]
Human pulmonary tissues	PS	-	20 to 100 µm (MP); 2.2 particles per gram of lung	Bishop et al. [88]
Human pulmonary tissues	PE and PP	Raman spectroscopy	<5.5 µm (MP particles); 8.12 to 16.8 µm (MP fibers)	Amato-Lourenço et al. [89]
Human bronchoalveolar lavage fluid (BALF)	PP, PE, and PS	LD-IR combined with scanning electron microscopy	<20 µm (MP); 4.31 particles per 10 mL	Qiu et al. [90], Chen et al. [91]
Human pulmonary tissue	PET and PP	µFTIR spectroscopy	3 µm (MP); 1.42 particles per gram of lung	Jenner et al. [92]
Nasal and pulmonary samples	-	-	-	Zha et al. [93]
Human blood	Amino-polystyrene and PS	Thrombin/fibrinogen clot model, characterized by turbidity and thromboelastography	<100 µg/mL 50, 100, and 500 nm (NP)	Tran et al. [94], Christodoulides et al. [95]
Cardiovascular system	PET, PA-66, PE-vinyl chloride, and PE	Py-GC/MS	118.66 µg MP/g of tissue	Liu et al. [96]
Atheroma plaque from the human carotid arteries	PE	Py-GC/MS, stable isotope analysis, and electronic microscopy	21.7 µg/mg of plaque (MP)	Marfella et al. [97], Kozlov et al. [98]
Colorectal cancer in rats	80 µg/kg/day	-	1 µg	Li et al. [99]
Human pancreatic, esophageal, lung, stomach, colon, and cervical tumors	PS, PVC, and PE	Gas chromatography and mass spectrometry (CG/MS)	MPs detected; 18.4 to 427.1 ng/g	Zhao et al. [100]

4. Occurrence of MNPs in the Bloodstream, Reproductive System, and Gastrointestinal Tract

PS-MPs affect the male reproductive system by decreasing sperm count and damaging testicular structures. Wen et al. studied the effects of NPs (80 nm) and MPs (5 µm), specifically PS, on the spermatogenesis of male C57BL/6 mice that orally ingested these particles for 60 days [75]. It was observed that different molecular mechanisms were

affected. NPs affected the regulation of retinoic acid metabolism, while MPs mainly influenced pyruvate metabolism and thyroid hormone metabolism, which compromised the spermatogenesis of mice and, consequently, their reproduction.

A frequent but unexpected source of MP contamination is the disposable materials used for serum infusion or blood transfusion therapies, where the infusion bottles, infusion bags, and infusion tubes are made of plastic. Zhu et al. identified eight MPs, ranging from 4 µm to 148 µm, at a concentration of 1–2 MNPs/unit [76]. The MNPs were found in PP-bottled, PE-bagged, and glass-bottled, but no particles were found in infusion tubes. The MP samples accounted for 11.66% of the total samples evaluated. This finding indicates that plastic materials used for infusion/transfusion therapies can be a source of MNPs in the bloodstream. Besides this, MPs absorbed from contaminated food can be secreted into saliva, gastric juice, and intestinal fluids (Figure 6), releasing toxic compounds into the gastrointestinal tract and distributing them to other organs through the bloodstream (Figure 6) [101].

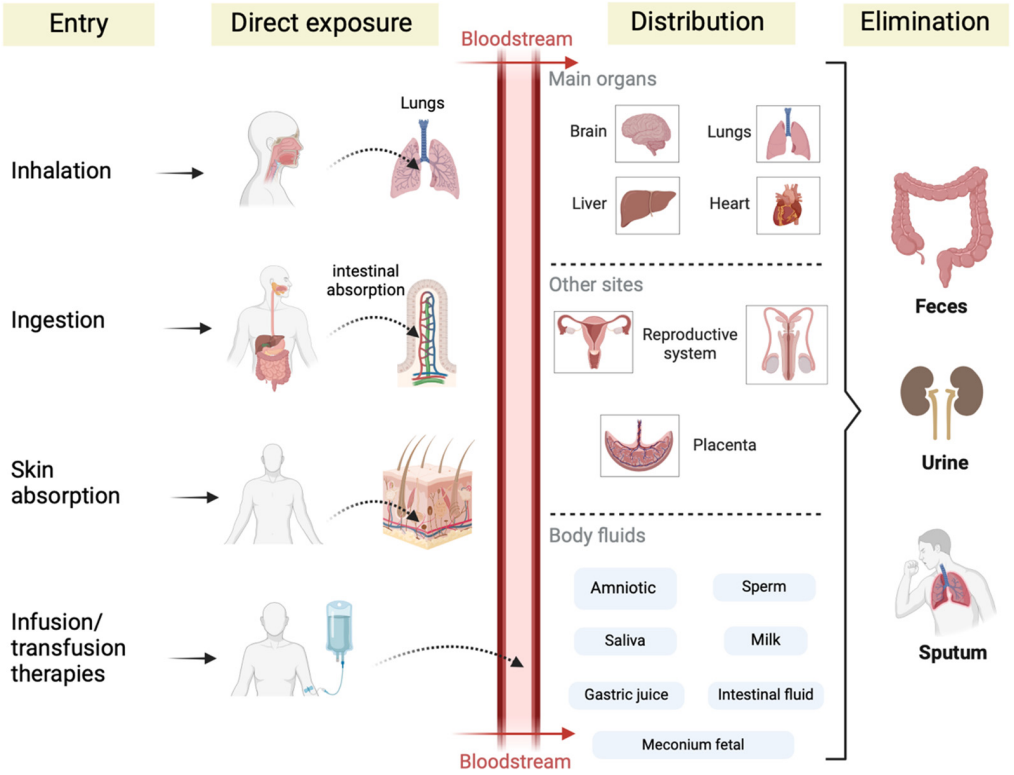

Figure 6. Trajectory representation of the absorption of MNPs through various entry points and distribution into human organs, systems, and fluids before elimination. Dotted-line arrows indicate the absorption pathway. Created in https://BioRender.com.

The pioneering study of Halfar and coworkers showed the presence of MPs in the amniotic fluid and placenta (Figure 6) by employing attenuated total reflectance–Fourier transform infrared spectroscopy (ATR-FTIR) after alkaline digestion with KOH [77]. From 20 samples of 10 patients, 44 MPs or plastic additives were found in the amniotic fluid, placenta, or both in 90% of those patients. PVC and the calcium–zinc PVC stabilizer predominated with granulometry between 10 and 50 µm. It is important to note that if MPs were found in the placenta, both mother and fetus were, or are, being exposed to risks. Indeed, all women who participated in the study have experienced pregnancy complications, such as pregnancies with prelabour rupture of membranes before the onset of labor. However, no evidence correlates the breakage of the amniotic sac with the presence of MPs in the placenta.

In a qualitative study using the Fourier transform infrared microspectroscopy technique (FTIR) in a clinical setting to detect MPs in the human placenta and fetal meconium (Figure 6), ten types of MPs (PE, PP, PVC, PS, polyethylene terephthalate, PA, PU, polycarbonate, polymethylmethacrylate, polyoxymethylene) were detected. Braun et al. detected polyurethane (PU) particles in the operating room following atmospheric precipitation [78].

Zhu et al. recently conducted a quantitative and qualitative study on MPs in 17 placenta samples using laser direct infrared spectroscopy (LD-IR) [79]. Twelve spherical or irregular-shaped MP types were found in four placenta samples, and three were identified as PP. In all of them, there were MPs at concentrations ranging from 0.28 to 9.55 microparticles per gram. Eleven distinct polymers were found, where PVC was the most abundant (43.27%), PP at 14.55%, and polybutylene succinate (PBS) at 10.90%. While the study provided data on the size and types of polymers present, there was no correlation between the presence of plastic particles and potential diseases in mothers and fetuses. Employing the Raman microspectroscopy technique, Ragusa et al. also studied the presence of MPs in human breast milk samples (Figure 6) from 34 women [80]. MPs were found in 26 samples, and the most abundant polymers identified were PE, PVC, and PP, with sizes ranging from 2 to 12 µm. Transmission to babies through breast milk is another form of vertical transmission of MPs. Considering another part of the female reproductive system (Figure 6), Qin et al. evaluated the presence and entry mode of MPs into the human endometrium [81]. After the investigation of the endometrial tissues of 22 patients, the contamination of MPs of the polyamide (PA), PU, PET, PP, PS, and PE classes was found with sizes ranging from 2 µm to 200 µm. Tests with mice to determine whether contamination occurs via blood circulation, vaginal contamination, or vaginal–uterine contamination proved that blood circulation is the route of contamination of the endometrium, with particles acquired during the diet. In addition, MP caused reduced fertility of the mice (significance level with probability ($p < 0.05$). After 3.5 months of intragastric exposure, a significant inflammatory response occurred in the endometrium ($p < 0.05$). Studies have indicated that MP contamination in the human uterus (Figure 6) can also have harmful effects on reproductive health.

An investigation into MPs in samples from 15 patients who underwent cardiac surgeries (Figure 6) used a laser direct infrared chemical imaging system and scanning electron microscopy [82]. The analysis detected several of these particles in five tissue types, with the largest fragment being 469 µm in diameter. In addition, nine types of MPs, with a maximum diameter of 184 µm, were detected in blood samples collected before and after the surgeries.

A recent study investigated MPs in healthy and cirrhotic liver tissues (Figure 6) [83]. The analysis involved chemical digestion of the tissues, application of the fluorescent dye red of the Nile (an azo dye), followed by fluorescence microscopy and Raman spectroscopy. The samples from healthy livers did not show MPs. However, the cirrhotic liver tissues tested positive for MPs. Six different polymers were identified, ranging from 4 to 30 µm in MPs. The authors claim that they do not have sufficient evidence to confirm the MPs in cirrhotic liver tissues as the cause of cirrhosis.

The physiology of the male reproductive system is sensitive to several environmental conditions, especially concerning sperm production [102,103]. Based on a study that evaluated the effect of MPs on the sperm from dogs and humans (Figure 6) using sensitive pyrolysis gas chromatography associated with mass spectrometry (Py-GC/MS), 12 types of MP were detected, with PE being the most prevalent, in the testicles of 47 dogs and 23 humans, reaching, in total, an average concentration of 122.63 mg/g in dogs and 328.44 mg/g in humans the testicle sizes was also increased [84].

Eleven human colectomy samples (Figure 6) were analyzed from adult patients in Peninsular Malaysia (mean age 45.7 years) [85]. After chemical digestion, the samples were analyzed under stereo and FTIR microscopes in search of MPs (composition, abundance, length, shape, and color). The results showed the detection of MPs in all 11 samples, with an average of 331 particles/individual sample or 28.1 ± 15.4 particles/g of tissue. Filaments or fibers accounted for 96.1% of the particles, 73.1% of all filaments, 90% were polycarbonate, 50% were PA, and 40% were PP. Cetin et al. conducted a study to investigate the effects of MPs in colorectal adenocarcinoma tissues compared to normal tissues [86]. They employed FTIR-ATR and Raman spectroscopy for detection and analysis. The study found that MPs were more prevalent in tumor tissues than in normal or control tissues, with particle sizes ranging from 0.001 to 1.2 mm and composed of polymers such as PE, methyl methacrylate (MMA), and PA. However, the study did not establish a definitive connection between the presence of MPs and colorectal cancer.

Other in vivo and in vitro studies have investigated the potential for MPs to cross the blood–brain barrier and cause neurotoxicity by promoting oxidative stress, disrupting inflammatory balance, altering neurotransmitters (synaptic plasticity), inhibiting acetylcholinesterase activity, and impacting the gut–brain axis [104]. The analyses showed that lower M and NPs cause more severe damage to neuronal diseases. There are still no relevant reports of the effects in humans, but there is evidence that they can disrupt neurons and alter the memory and behavior of organisms (Figure 6). Urgency is needed to elucidate the neurotoxic danger of exposure to MPs and NPs [87,105].

5. Occurrence of MPs in the Respiratory Tract via Inhalation

MP particles (>1 μm up to <5 mm) dispersed in the air also represent the most important source of exposure of the human body through respiration, aggravated by the fact that smaller particles ensure their prolonged permanence in the atmosphere [106,107]. MP particles lodged in the lungs can impair gas exchange. Moreover, sustained exposure to airborne MPs can aggravate pulmonary diseases by compromising airway barrier integrity. This effect is thought to arise from alterations in the biophysical properties of pulmonary surfactants, leading to structural disruptions of the alveoli and impaired epithelial cell proliferation (Figure 7) [108–110]. Current mechanistic investigations remain early, underscoring the need for further in-depth studies.

Patients with a history of exposure to MPs had significantly higher levels of MPs in their lung tumors than those with lower exposure histories [111]. This suggests a potential link between MP inhalation and the development of Ground-Glass Nodules (GGNs) visible on CT scans (Figure 7). Individuals most affected reside in heavily polluted urban areas, particularly near major highways, as shown by Wang and coworkers [112]. For instance, a study of 12 nonsmoking women living close to major highways found elevated levels of MPs in their lung tissues. Analyses using infrared laser imaging spectroscopy and scanning electron microscopy identified 108 MPs, of which 12 were composed of various polymers, with an average concentration of 2.2 particles per gram of lung tissue. Particle sizes ranged from 20 to 100 μm, with PP being the most prevalent polymer (34.2%), followed by PET (21.3%). Notably, MP contamination was positively correlated with elevated platelet and fibrinogen levels and negatively correlated with direct bilirubin levels, a degradation product of hemoglobin [112].

The SARS-CoV-2 coronavirus infection, which led to the COVID-19 pandemic, has as its main characteristic a pulmonary disease that can vary from mild to severe. It was predictable that lungs contaminated with particles of MPs could interfere with the course of this infection. Bishop et al. showed that MPs can dysregulate lung inflammation by

SARS-CoV-2 by suppressing innate immune responses after two days of infection and increasing the release of proinflammatory cytokines six days after infection [88].

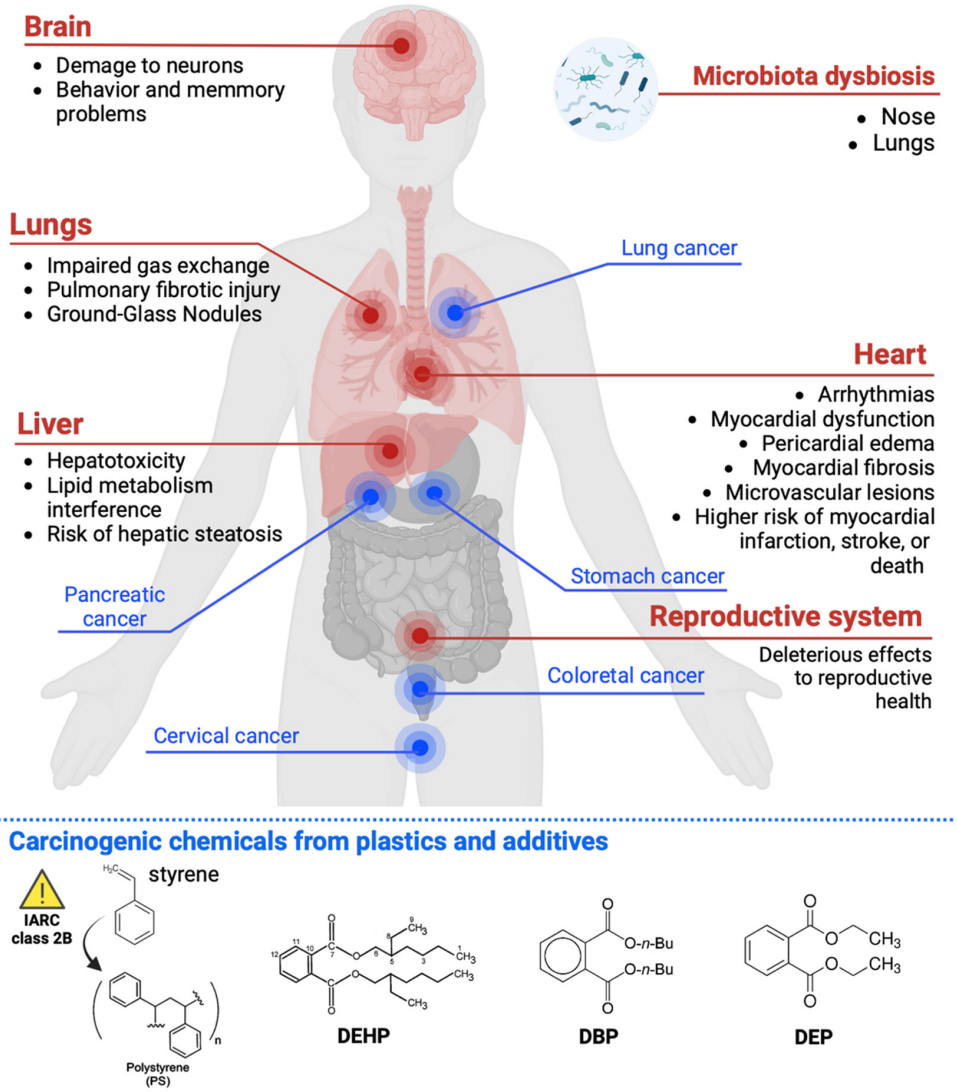

Figure 7. Health damage that might be associated with MNP absorption and potential carcinogenic contributors. Created in https://BioRender.com.

Human lung tissues obtained from autopsies were evaluated by Raman spectroscopy. MPs were observed in 13 of the 20 tissue samples; all were smaller than 5.5 µm, and the fibers ranged from 8.12 to 16.8 µm. The present polymers were PE and PP. Although they do not find a direct correlation between MP and some diseases, they can still harm the respiratory system (Figure 7) [89].

One way to obtain samples is to investigate the deposition of MPs in bronchoalveolar lavage fluid (BALF). This fluid is a source of samples of the material present in the pulmonary alveoli and bronchi. Qiu et al. conducted experiments to detect MPs in the respiratory tract. BALF of 18 nonsmoking individuals was analyzed by LD-IR spectroscopy combined with scanning electron microscopy [90]. Thirteen types of MPs were detected in the 18 samples of the liquid, with PE being the most frequent (86.1%), showing that MPs penetrate deep into the respiratory tract. MPs were also found in Chinese children's bronchoalveolar lavage fluid (BALF), with 89.6% of the samples containing MPs, averaging 4.31 ± 2.77 particles per 10 mL [91]. These data confirm inhalation as a significant route of exposure to MPs in the pediatric lungs. The MPs were made of PP (41.9%), PE (19.4%),

and PS (13.6%), most of which were smaller than 20 µm, indicating a possible relationship between MPs and pediatric lung diseases.

MP particles embedded within pulmonary tissue can potentially compromise gas exchange (Figure 7). Employing µFTIR spectroscopy, their presence was identified in digested lung samples from 13 individuals, with a detection threshold of 3 µm [92]. A total of 39 MP particles in 11 of the 13 samples, with an average concentration of 1.42 ± 1.50 MPs per gram of lung tissue. The primary plastic components found were PET and PP, which were present in critical areas for gas exchange, including terminal bronchioles, alveolar ducts, and alveoli. Xu et al. demonstrated that MPs impair lung biophysical function through the hetero-aggregate formation of this interface, negatively affecting gas exchange [113]. Further evidence of the effect of MPs on the respiratory system was reported by Li et al., demonstrating that the particles of MPs dispersed in the air, resulting from tire wear, induced pulmonary fibrotic injury (Figure 7) through the rearrangement of proteins that provide structural support to the cytoskeleton of epithelial cells [114].

MPs and NPs can potentially disrupt the composition of nasal and lung microbiota (Figure 6), including bacteria, viruses, and fungi, though their effects are not yet fully understood [93]. Studies have shown that bacteria from *Staphylococcus* spp. and *Roseburia* spp. are more frequently associated with MPs, while *Prevotella* spp. are more commonly linked to NPs. These findings suggest that airborne MPs and NPs could contribute to microbial dysbiosis in both nasal and pulmonary environments (Figure 7).

Jiang et al. studied MPs' contamination of the human upper respiratory tract, differentiating between internal and external workers [115]. It has been shown that indoor workers are more exposed to MP particles, while outdoor workers are more exposed to MP fibers. The latter ones can wear respirator masks as personal protective equipment, but they have the advantage of the wind dispersing MP particles.

6. MP Occurrence and the Cardiovascular System Treats

MP particles' size and chemical composition can significantly impact the cardiovascular system. The bloodstream can transport MPs to various organs and tissues, potentially leading to severe physiopathological conditions and diseases (Figure 6). For example, MPs can induce platelet aggregation and thrombus formation. However, there remains uncertainty regarding the full extent of pathologies associated with environmental exposure to MPs and the various routes [116]. Data published over the past decade establishes the toxicity of MPs and their effects on cardiovascular mechanisms by impairing cardiac function. Direct effects of MPs on myocardium include arrhythmias, pericardial edema, and myocardial fibrosis [117]. Moreover, microvascular lesions were also reported to induce hemolysis, thrombosis, blood clotting, and vascular endothelial damage. As mentioned, MNPs can trigger oxidative stress, inflammation, and apoptosis in the cardiovascular system.

MPs' impact on fibrin clot formation was investigated using a simplified ex vivo human thrombin/fibrinogen clot model and characterized by turbidity and thromboelastographic analyses [94]. When the particles were pre-incubated with fibrinogen, a reduction in the efficiency of coagulation for amino-polystyrene and PS of up to 100 µg/mL was observed. The results demonstrated a significant inhibitory effect on fibrin clot formation, impacting the formation rate and clot strength (Figure 5).

Further evidence of MP interference in the dynamics of blood coagulation was reported using the technique of thromboelastography, detecting particles of 50, 100, and 500 nm from three types of plastics [95]. Carboxypolystyrene (cPS) was found to activate the coagulation cascade by enhancing the rate of fibrin polymerization, while PS particles in general had minimal effects on coagulation dynamics, except for the 50 nm particles that triggered the coagulation cascade. Additional studies are necessary to elucidate better the effects of

MPs on both thrombin- and platelet-mediated clotting; however, it seems that the binomial particle size and composition-surface modifications must be considered to evaluate the role of MPs on coagulation.

Environmental factors may elicit cardiovascular diseases. Exposure to pollutants and chemicals (e.g., arsenic, lead, cadmium, polluting gases, airborne particles, solvents, pesticides, and MP particles) through the air, water, and food [118]. Daily exposure to these external agents has been extensively related to increased risk of cardiovascular diseases [119,120]. Recently, the Py-GC/MS technique has emerged to detect MPs in organs and tissues and was employed to investigate MPs in the atherosclerotic plaques of 17 samples from human vessels, coronary and carotid arteries, and aorta, with a mean concentration of 118.66 μg/g of tissue. PET was found in 73.70% of samples, PA-66 in 15.54%, PE-vinyl chloride in 9.69% and PE in 1.07% [96].

A prospective, multicenter, observational study has proven a direct link between MPs and cardiovascular disorders. Marfella and coworkers conducted a historic study involving 304 patients, of whom 257 completed the mean follow-up of 33.7 ± 6.9 months [97]. The patients underwent a surgical procedure to remove the accumulation of atheroma plaque from the carotid arteries (asymptomatic carotid endarterectomy) [98]. It was proposed that MPs interact with fat molecules and can accumulate in atheromatous plaques in the epithelial lining of blood vessels. Analyses to identify the type and size of the MPs were performed using Py-GC/MS, stable isotope analysis, and electronic microscopy analysis. The irregular bubbles of MPs mixed with cells and other residues were found in 150 patient samples. Chemical analyses revealed that PE was found in 58.4% samples, with an average level of 21.7 ± 24.5 μg/mg of plaque. The detection of inflammatory biomarkers was evidenced by enzyme-linked immunosorbent and immunohistochemical tests. Patients with higher concentrations of MPs in atheroma plaque samples displayed higher levels of inflammation biomarkers. These data suggest that MPs may contribute to plaque rupture in patients with carotid atheroma plaques. Those in whom MPs and NPs were detected presented higher risks of myocardial infarction, stroke, or death from any cause at 34 months of follow-up compared to those in whom these particles were not detected.

Research into the relationship between MPs and heart disease is still emerging. But preliminary evidence suggests a potential cause-and-effect relationship where MPs may directly contribute to the development of heart diseases. The primary challenge in this field has been accurately detecting and quantifying MPs and establishing this relationship using analytical methods.

7. MPs and the Development of Cancer

As previously mentioned, plastics may contain various additives, including phthalic esters, polycyclic aromatic hydrocarbons, polychlorinated biphenyls, plasticizers (particularly bisphenol A), and dyes. Phthalic esters (PAEs), such as di-2-ethylhexyl-fatalate (DEHP), di-n-butyl phthalate (DBP), and diethyl phthalate (DEP), are among the most systemic toxic compounds displaying carcinogenic potential (Figure 7) [121–123]. They can be adsorbed by MPs and released when they reach the living organisms [124]. Deng et al. demonstrated for the first time that MPs can transport and release PAEs in the intestines of mice. As a result, MPs can adsorb and transport PAEs to the gastrointestinal tract, where they accumulate and cause toxic effects [125].

Bisphenol A is another plasticizer widely used in plastics manufacture and, hence, has been the subject of major toxicological concerns for human health. In this context, Cheng et al. investigated bisphenol's hepatotoxic effects (Figure 7) combined with PS particles [126]. It was observed that adding both to a culture medium decreased bisphenol levels, suggesting an interaction between the two substances, which resulted in a syner-

gistic adverse effect, including hepatotoxicity and genomic interference related to lipid metabolism. This study reveals a pronounced metabolic health risk, notably the development of hepatic steatosis, arising from the co-exposure to PS and bisphenol, even at low doses that approximate human internal exposure levels.

There is still little knowledge about the correlation between exposure to MPs and their additives and various types of human cancer. Kumar et al. discuss the possibilities of inhalation, ingestion, and dermal exposure causing genotoxicity, cell division and viability, cytotoxicity, induction of oxidative stress, metabolism disruption, DNA damage, and human immune responses [127]. However, they conclude that with current knowledge about the potential risks to human health, there are still many gaps and recommend further investigation.

Rawle et al. investigated the potential of MP particles to induce colonic inflammation in rats that consumed approximately 80 µg/kg/day of 1 µm PS through drinking water [128]. They observed that colonic inflammation was prolonged in rats exposed to MPs, suggesting that disturbances in the colon activate lymphoid cells and promote inflammatory responses. Regarding the effects of MPs on the colon, Li et al. reviewed the incidence of colorectal cancer with MPs [99]. The occurrence of this type of cancer is increasing in people under 50 years of age, and this epidemiological change suggests that there may be a prevalent environmental cause, possibly contamination by MPs. This hypothesis is based on substantial evidence of the cumulative presence of these MPs in the gastrointestinal tract from contaminated food. Particles of MPs transiting the gastrointestinal tract can interfere with the protective mucus layer of the colon and rectum, reducing its protective effect and increasing the likelihood of colorectal cancer (Figure 7).

A recent study revealed the presence of MPs in several human tumors (Figure 7) through qualitative and quantitative analysis using gas chromatography and mass spectrometry (CG/MS) [100]. Among the 26 samples analyzed, PS, PVC, and PE were found in six types of tumors. The distribution of MPs varied among the different organs. In lung, stomach, colon, and cervical tumors (Figure 7), the detection rates of MPs were 80%, 40%, 50%, and 17%, respectively, with concentrations ranging from 7.1 to 545.9 ng/g of tumoral tissue. In pancreatic tumors (Figure 7), MPs were detected in 70% of the samples, with concentrations between 18.4 and 427.1 ng/g of tumoral tissue, while in esophageal tumors, no MP was detected. MPs may affect the tumor immune microenvironment (TIME), accelerating tumor progression and treatment resistance, which is still unclear.

The interactions between MPs and toxic heavy metals and their effects on human and animal health have also been studied. In aquatic environments, MPs can adsorb metals, glycolipids, pesticides, long-chain perfluoroalkyl substances (PFAS), and other contaminants, potentially leading to harmful biological effects [129]. MPs act as carriers of these toxic substances [130,131]. MPs undergo aging due to exposure to UV radiation and air, increasing the surface roughness and, consequently, enhancing the contact surface of the material. Godoy and colleagues investigated MPs made from various polymers and their ability to carry metals, pesticides, and pharmaceutical products [132]. Metals, originating from plastic manufacturing processes, can associate with MPs through environmental adsorption and be transferred along the food chain. PE, PS, and PVC MPs quickly adsorb various metals, but chromium and lead are absorbed in more significant quantities. Co-exposure to PVC-derived MPs and cadmium damages the liver of female ducks, but the mechanism is still unknown. Cadmium is a toxic metal that interacts strongly with various polymers and can be carried into organisms [133]. Sun and colleagues studied the association of PVC with cadmium and its biological effects on female ducks over two months [134]. The ducks were treated with pure water, isolated components, or were co-exposed to both. PVC with cadmium offered to ducks led to liver shape changes,

hepatocyte dysfunction, ultrastructural damage, and alterations in glycolipid metabolism, ultimately leading to apoptosis. Another study showed that hepatotoxicity caused by PP-MPs, artificially aged with UV radiation, was tested in hepatic organoids, generating oxidative stress at low doses and interfering with the mitochondrial respiratory chain [135].

8. Elimination and Clearance of MNPs

As mentioned, the main routes MPs and NPs can take to enter the human body are infusion tubing, inhalation, absorption by the skin, and ingesting food, following distribution via the bloodstream, accumulating in the respiratory and digestive system, reproductive system, liver, spleen, and brain (Figure 6) [136]. However, the accumulation of MPs and NPs does not necessarily imply direct damage; part of the MPs absorbed can be engulfed by macrophages and digested by gut microbiota. It may undergo an inflammatory reaction and a fibrous encapsulation. Most of them will be removed by the liver and spleen and excreted in the feces. The most common route for the MP elimination is absorbed from drinking water and food ingestion. Schwabl et al. were the first to examine the human feces of eight healthy volunteers for the presence of MPs (Figure 6) [137]. The analysis was conducted after the chemical digestion of the fecal samples, infrared microspectroscopy with a Fourier transform, focusing on the 10 common MP types. The characteristics of the participants included the following: none of them were vegetarians; six consumed seafood, and food was commonly wrapped, packaged, or stored in plastic; seven participants consumed beverages in plastic bottles daily; and three used cosmetics with synthetic polymers. All eight feces samples confirmed that particles between 50 and 500 µm were found in all stool samples. Nine of the ten most common MP types were identified, and the most abundant were PP and PET. Subsequently, Yan and coworkers analyzed MPs in the feces of patients with inflammatory bowel disease and compared them with those of healthy people [138]. The fecal concentration of MPs in compromised patients (41.8 particles/g DM) was higher than in healthy individuals (28.0 particles/g DM). The study found a possible positive correlation between fecal MP concentration and inflammatory bowel disease. MPs were analyzed in the feces of both groups, with 15 types of non-MP sheets and fibers detected, the majority being PET at 22.3–34.0%.

Urine is another route for MP elimination, particularly after the biodegradation of polymers (Figure 6) [139]. The urine samples of six volunteers from different cities in southern Italy (three men and three women) were analyzed by Raman microspectroscopy, which identified MPs and NPs ranging from 4 to 15 µm, with irregular shapes and different colors. Polymers such as PP and PE were found in four samples, while polyvinyl acrylate (PVA) and PVC were observed in one female sample.

Inhalation is one of the involuntary routes of absorption of MPs; however, sputum secretion can eliminate part of this material (Figure 6), and analysis of human sputum can indicate the amount of these polymers absorbed by the lungs. Huang et al. investigated the sputum of 22 patients with different respiratory diseases employing laser infrared imaging spectrometry and Fourier transform infrared microscopy [140]. The analyses showed 21 types of MP < 500 µm, with PU being the most prevalent, followed by polyester, chlorinated polyethylene, and alkyd varnish, which comprised 78.36% of the total MP. The types of MPs detected may be related to smoking, thus increasing the risks to human health.

9. Conclusions

Plastic is a relatively recent invention in human history, and it has become ubiquitous in our everyday lives due to its versatility, durability, and low cost. However, these same characteristics that have made plastic so popular are the ones that now pose a colossal challenge to the planet's health. Plastics are widespread in the environment at an alarming rate,

and the proliferation of plastics is not only a problem of visual pollution but a profound threat to the health of ecosystems and living beings, with unpredictable consequences for the entire ecological system of our planet. MNPs can be found in every ecosystem, terrestrial, aquatic, and atmospheric, and they have already reached all human body organs through different routes. However, quantifying MNP particles by non-invasive methods in human tissues has not yet been available to determine the safe limits of exposure and accumulation in the body organs. Despite the increasing number of studies on MP effects in the reproductive, cardiovascular, and respiratory systems, comprehensive data regarding their toxicological effects are still limited and fragmented. This gap underscores the need for further investigation into the mechanisms of action, dose–response relationships, and long-term impacts of MPs on biological systems. Consequently, there are still few direct correlations between their presence and the development of diseases, with only suggestions and hypotheses of positive occurrence of certain physiopathological conditions such as inflammatory and oxidative status, besides some diseases such as cancer or intestinal, pulmonary, and cardiovascular affections, that seem to be induced or aggravated by these materials.

Plastics visible to the naked eye are just a small chapter in this story. They slowly break down into MPs and NPs, enabling them to penetrate almost any environment, from the depths of the oceans to the most remote and pristine regions of the Earth, and living organisms, including human beings, since they reach the bloodstream, the different organs and tissues, even the human placenta, raising concerns about the long-term health effects. MNPs can interact with other toxic chemicals such as pesticides, heavy metals, and persistent organic pollutants, which are then ingested or inhaled, and finally released into the body, potentially causing cellular damage, inflammation, oxidative imbalance, and changing the gene expression pattern.

It is possible to say that this part of the Anthropocene era could be called the era of plastics. Changing individual behaviors, corporations, and government institutions may be the key to solving the global crisis caused by plastics. Many actions can be implemented, but international politics will be mandatory, including expanding plastics collection and recycling, reducing production, reducing consumption, higher taxes and fees on single-use products, and encouraging the use of biodegradable plastic. Minimizing plastics should follow an approach that starts with prevention, reduction, reuse, recycling, and recovery, and ends with elimination as the final option.

However, solving the plastics crisis requires a multi-pronged approach. Education and public awareness are crucial to changing consumer behavior, encouraging sustainable consumption practices, and reducing single-use plastic use. Corporations also play an essential role by innovating in sustainable packaging and promoting the circular economy, where products are designed to be reused and recycled rather than discarded.

The relevance of biodegradable plastics such as PLA should be raised as potential substitutes for the hazardous plastic-derived MNPs. Still, the focus was primarily on evaluating materials currently produced on a large scale and economic feasibility, and addressing the immediate and necessary investigation of the threats from those micro- and nanoplastic particles to living beings.

Governments should implement strict policies encouraging the reduction of plastic use, promoting recycling, and penalizing waste. Measures such as banning single-use plastics, imposing taxes on plastic products, and subsidizing biodegradable alternatives can accelerate the transition to a society that is less dependent on plastics. International cooperation is essential, as plastic pollution is a global problem that transcends borders.

Author Contributions: A.S.d.S. conducted the literature searches, delineated the main topics, wrote the original draft, and created figures; P.G.F., I.S.d.J., R.P.R.F.d.O., A.S.d.C., and L.C.D.R. helped in the data investigation and writing the original draft; P.R.P. helped in the data investigation, writing the original draft, and created figures; V.M.F.P. and D.O.F. worked in the writing—review and editing and supervision; V.F.F. was responsible for project administration, resources, funding acquisition, writing—review and editing, and supervision. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded by FAPERJ grant numbers E-26/200.911/2021 (FAPERJ CNE), and CNPq 1A 301873/2019-4; SEI-260003/001178/2020 (thematic FAPERJ); E-26/210.093/2023 (thematic FAPERJ); E-26/201.016/2022 (FAPERJ CNE); 300860/2025-0 (CNPq); E-26/200.756/2023 (FAPERJ postdoc fellowship).

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Data are contained within the article.

Conflicts of Interest: The authors declare no conflicts of interest.

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