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Review

A review on microplastics and nanoplastics in the environment: Their occurrence, exposure routes, toxic studies, and potential effects on human health

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ABSTRACT

Microplastics (MPs) and nanoplastics (NPs) are emerging environmental pollutants, having a major ecotoxicological concern to humans and many other biotas, especially aquatic animals. The physical and chemical compositions of MPs majorly determine their ecotoxicological risks. However, comprehensive knowledge about the exposure routes and toxic effects of MPs/NPs on animals and human health is not fully known. Here this review focuses on the potential exposure routes, human health impacts, and toxicity response of MPs/NPs on human health, through reviewing the literature on studies conducted in different *in vitro* and *in vivo* experiments on organisms, human cells, and the human experimental exposure models. The current literature review has highlighted ingestion, inhalation, and dermal contacts as major exposure routes of MPs/NPs. Further, oxidative stress, cytotoxicity, DNA damage, inflammation, immune response, neurotoxicity, metabolic disruption, and ultimately affecting digestive systems, immunology, respiratory systems, reproductive systems, and nervous systems, as serious health consequences.

1. Introduction

Plastic products are durable, lightweight, resistant to degradation, strong, economically cheap, and insulation to heat and electricity, so these products have been used globally. Plastic manufacturing has rocketed in the last 70 years, from 1.7 million tons in the 1950s to over 348 million tons in 2017 (Revel et al., 2018; Mendoza and Balcer, 2019; PlasticsEurope, 2018). Currently, there are about 45 different types of plastics including polypropylene (PP), polyethylene (PE), polyethylene terephthalate (PET), polystyrene (PS), polyurethane (PU), polyvinyl chloride (PVC), and polycarbonate (PC) are commercially available (Lin et al., 2018). Because of its widespread use, plastic garbage has become a serious environmental challenge for management and majorly caused serious risks to the aquatic environment (Alimba and Faggio, 2019; Guzzetti et al., 2018; Kontrick, 2018; Lin et al., 2018; Prata et al., 2021; Prokić et al., 2021; Sruthy and Ramasamy, 2017; Strungaru et al., 2019). One study estimated globally, that about 5 trillion plastic particles weighing about 250,000 tons are floating in the marine (Eriksen et al., 2014).

Microplastics (MPs; size range 1 μm–5 mm) and nanoplastics (NPs;

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size range 1 nm–1 μm) are emerging plastics-related environmental pollutants (Gerdes et al., 2019). The toxic effects of MPs and NPs are determined by their characteristics, and the source of production *i.e.*, synthetic or natural sources. Majorly synthetic plastic fibers degrade very slowly and persist long as organic pollutants (Yang et al., 2022; Zhao et al., 2022), and pose long-term threats to the environment and human health (Chen et al., 2020, Eerkes-Medrano et al., 2015; Galloway et al., 2017; Law and Thompson, 2014; Li et al., 2019). The consequence of the environmental accumulation of MPs exposed to biota pose risk for bioaccumulation and biomagnification at the trophic level and causes multiple ecological repercussions (Barnes et al., 2009; Phuong et al., 2018). Several studies have shown that marine animals often ingest microplastics, which have serious effects on them since MPs collect in the epidermis, serve as bacterial transport channels, and absorb chemical compounds on their surface (Auta et al., 2017; Kumar et al., 2022; Roch et al., 2020; Rubin et al., 2021). According to many recent studies, MPs have been shown to accumulate in the gastrointestinal system of aquatic animals (Li et al., 2022; Ugwu et al., 2021). A next study reported, MPs increased catalase, glutathione reductase, glutathione-s-transferase, and induced antioxidant defenses in the liver of fish (*Sparus aurata*) (Capó et al., 2021). Inhalation exposure to polystyrene micro-(nano-) plastics was recently shown to cause inflammation in rats lungs (Lim et al., 2021). Further, one recent study reported the first evidence of MPs (polypropylene) in the human placenta (Ragusa et al., 2021). A next study conducted on plants reported the interiors of vascular plants may induce a variety of phytotoxic effects on growth, photosynthesis, and oxidative stress (Yin et al., 2021).

Therefore, MPs and NPs have a serious impact on environmental health, and public health through contaminated environments (van Emmerik and Schwarz, 2020). Understanding the primary intake route of MPs/NPs exposure is critical for probable reduction of risk; but comprehensive information about the exposure routes and toxic effects of MPs and NPs on human is lacking (Carbery et al., 2018). This review focuses on the potential impact of MPs and NPs on human health, exposure routes, and toxicity responses, through information collected from organisms, human cells, and the human experimental exposure models.

2. Methodology

The review focused on MPs and NPs studies conducted on an environmental and laboratory-scale for evaluating their hazardous consequences. Literature was searched on May 2022, using Web of Science (WoS) and Scopus databases. The search was restricted to peer-reviewed original papers written in English. As represented in Fig. 1; the following terms were used in WoS: (microplastic OR microplastic OR nanoplastic OR nano plastic) AND (animal toxicity OR human toxicity OR exposure OR human health), and in Scopus: microplastic OR microplastic OR nanoplastic OR nano plastic AND animal toxicity OR human toxicity. The information gathered from the literature was then divided into subtopics: occurrences, sources of exposure, toxicity responses, possible induced toxicity of MPs/NPs, and health impacts. The flow of thoughts in the assessment of MPs/NPs' possible impacts on human health is shown in Fig. 2.

3. Primary and secondary microplastics

Microplastics (MPs) are ubiquitous and originated from various anthropogenic sources (Anderson et al., 2017; Cole et al., 2011; de Sá et al., 2018; Kühn et al., 2015). Based upon their sources, MPs can be categorized into “Primary microplastics”, manufactured for indirect or direct use as raw materials for consumer polymer goods, and “Secondary microplastics” produced from the breakdown, cracking, and progressive deterioration of larger plastic fragments (Anderson et al., 2016; Andrady, 2011; Cole et al., 2011; Ryan et al., 2009; Thompson, 2015; Thompson et al., 2004). Primary MPs are commonly used in facial cleansers, cosmetics, scrubs, microbeads, toothpaste, exfoliants, and abrasives, as well as by industries (*e.g.*, air blasting) (Anderson et al., 2016; Browne et al., 2011; Cole et al., 2011; Prata et al., 2020a), or for washing synthetic clothes and rubbers (Laskar and Kumar, 2019), tea bags (Hernandez et al., 2019). They are also utilized in medicine as drug-production vectors (Patel et al., 2009).

The secondary MPs pose more public health risks than primary MPs (Ogonowski et al., 2016). Most environmental MPs are secondary MPs and originated from big plastic litter (Peng et al., 2020). Due to the chemical structure, plastic materials degrade extremely slowly.

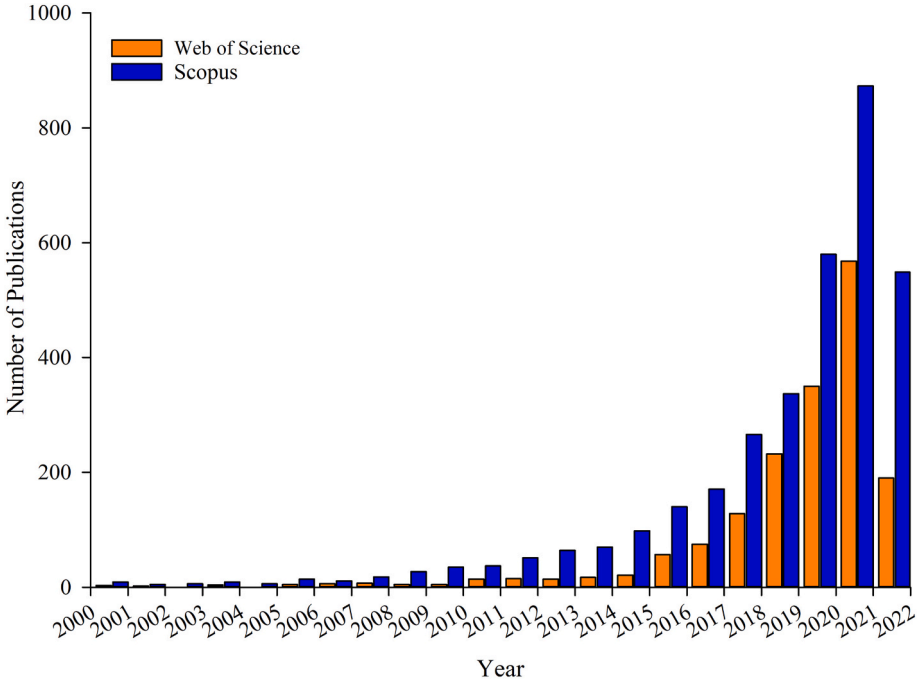

Fig. 1. Publication trends of microplastics and nanoplastics toxicity on animal and human research (until May 2022).

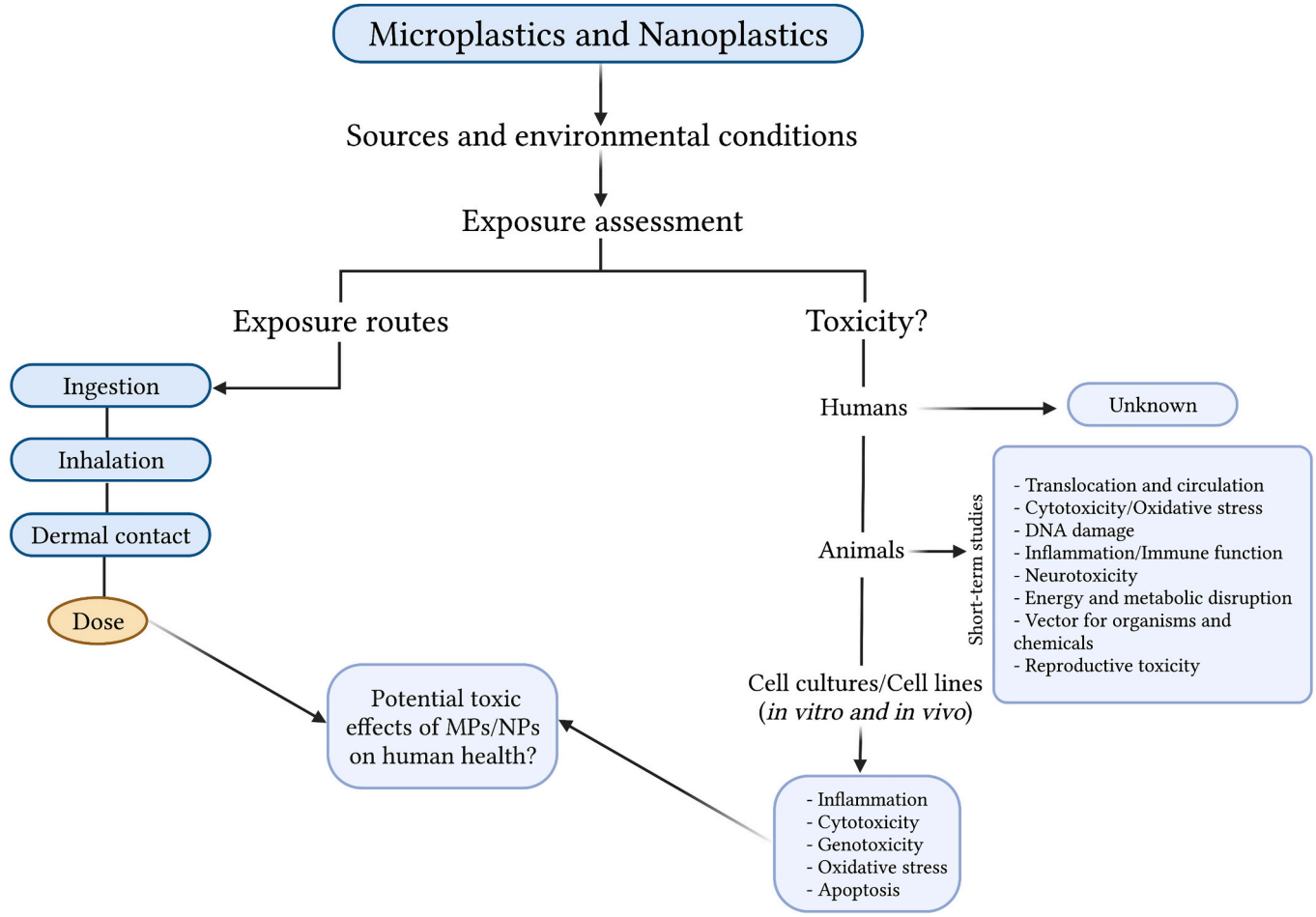

Fig. 2. Concept of human health effects of MPs/NPs (modified from Heddagaard and Møller, 2020).

Physical, chemical, and (or) biological action break large pieces of plastics into microplastic fragments (1 μm–5 mm) and nanoplastics (NPs) with a size range 100–1000 nm and <1000 nm (<1 μm) (Auta et al., 2017; EFSA CONTAM Panel, 2016; Huang et al., 2021; Jahnke et al., 2017; Lehner et al., 2019; Pirsaheb et al., 2020). As listed in Table 1, based on plastic size, and shape, environmental plastic debris can be grouped into four categories; nanoplastics, microplastics, mesoplastics and macroplastics (Alimba and Faggio, 2019; Andrady, 2011, 2017; Cole et al., 2011; Eriksen et al., 2014; Galgani et al., 2015; GESAMP, 2015; Helm, 2017; Koelmans et al., 2017; Lebreton et al., 2018; Tibbetts et al., 2018; Wu et al., 2019).

Table 1
Plastic debris is classified based on its size and shape in the environment.

Plastic fragments categorizations	Diameter sizes	Shapes
Nanoplastic (NPs)	1–1000 nm and <20 μm	Fragments, pellet, nurdles, microbead, spherical bead, irregular bead, granule, foam, fibers, film, and commercial fragments (melted plastic appearance)
Microplastics (MPs)	1–5 mm (0.1–0.5 cm)	
Mesoplastics (MSPs)	5–25 mm (0.5–2.5 cm)	
Macroplastics (MCPs)	> 25 mm (>2.5 cm)	

4. Exposure routes

Ingestion or intake of contaminated water or food, inhalation of contaminated air with MP, and dermal contact with contaminated water, air, textiles, and cosmetics are the three major routes of MPs and NPs exposure (Alimba et al., 2021; Prata et al., 2020a; Prokić et al., 2021; Revel et al., 2018). The details of each exposure route are explained below.

4.1. Ingestion

Ingestion (oral consumption) is one of the major exposure routes for MPs and NPs (Galloway, 2015; Prata et al., 2020a). Microplastics have been reported in drinking water (Koelmans et al., 2019), foods from fish farms and marine culture zones (Feng et al., 2019), mussels (Li et al., 2018; Li et al., 2016), and salts from lakes and oceans (Gündoğdu, 2018; Yang et al., 2015), in commercial salts (Iñiguez et al., 2017; Karami et al., 2017) and in bottled water (Praveena and Laohaprapanon, 2021). PET and PP were the most common polymers reported in bottled water (Danopoulos et al., 2020). One survey had reported MPs and NPs from a majority (88 %) of tap water in both developed and developing countries (Kosuth et al., 2018). Furthermore, MPs and NPs were reported in tea-bags from grocery stores and coffee and tea from cafes in Montreal, Canada (Hernandez et al., 2019), and food stores in Germany (Schymanski et al., 2018). A Mexican study reported MPs in milk with concentrations of 1–14 particles/L and sizes ranging from 0.1 to 5 mm (Kutralam-Muniasamy et al., 2020). Philipp et al. (2020) reported microplastic particles in the intestines of harbor seals (*Phoca vitulina*) and grey seals (*Halichoerus grypus*). Consumption of such fish

contaminated with MP in the long-term may result in a significant risk of biphenyl exposure and negative health consequences (Barboza et al., 2020). These types of MPs contamination in food and drinks need to be investigated throughout the world, currently such information is confined to a few geographical regions mostly from developed countries (Barboza et al., 2018). Consumption of contaminated food and drinks poses a serious public health risk for long-term exposure (Barboza et al., 2018). The intake of 0.5 mg/day of MPs of size 5–20 μm can accumulate in the stomach, kidney, and liver. The MPs particle size was linked to tissue accumulation kinetics and distribution patterns (Revel et al., 2018). Endocytosis allows MPs and NPs to enter the body's circulatory system, including the Peyer's patch microfold (M) cells (Alimba et al., 2021). More studies are needed for evaluating the effect of MPs exposure on human health (Prata et al., 2020a).

4.2. Inhalation

Inhalation (breathing route) is the next major exposure route for MPs and NPs (Chen et al., 2020). MPs and NPs have been reported from ambient air (Chen et al., 2020; Dris et al., 2017). Still, atmospheric MPs and NPs can be directly inhaled due to their small size and pose human health risks by accumulating in the respiratory tracts and potentially crossing the blood-brain barrier (BBB) (Chen et al., 2020; Prata, 2018). MP fibers size larger than 250 μm has been reported in human lung tissue (Pauly et al., 1998). The chemical composition of these particles may cause acute and chronic respiratory problems in the short and long-term. Some fibrous MPs may be inhaled, and the majority of them will be cleared by mucociliary clearance; nevertheless, some may linger in the lungs, triggering a localized biological response, including inflammation, particularly in those who have limited clearance systems (Gasperi et al., 2018). An investigation employing a Breathing Thermal Manikin (BTM) to simulate human exposure to indoor airborne microplastics revealed that MPs might be swallowed due to probable direct human exposure to microplastic pollution through indoor air (Vianello et al., 2019). Recently, Lim et al. (2021) reported that inhalation exposure to polystyrene MPs/NPs increased inflammation and the expression of the inflammatory protein (TGF-β) and TNF-α in the lung tissue. More additional studies are needed on the effects of MP and NP inhalation exposure on human health and their features and concentrations to assess and avoid future harm. To fully assess the danger of these MPs and NPs, further research is needed to obtain exposure and inhalation levels in both indoor and outdoor situations. However, investigating the inhalation exposure routes of MPs in humans can have an ethical challenge and also for experimental design.

The average concentration of airborne MPs in the United States of America (USA) was 5.4 n/m^3 (outdoor) and 0.9 n/m^3 (indoor); 33 % were MPs with the size of 50–3250 μm and in Shanghai, China, was 1.42 ± 1.42 n/m^3 with a size range of 23–5000 μm (Liu et al., 2019a; Liu et al., 2019b; Liu et al., 2019c). The next study in Hamburg, Germany, reported an average concentration of 275 n/m^2/day with a size range of 63–5000 μm (Klein and Fischer, 2019). There can be various sources of MPs and NPs in the atmosphere including, synthetic textiles, flock industry (Atis et al., 2005; Prata, 2018), vinyl chloride (VC), and polyvinyl chloride (PVC) industries are major sources of atmospheric microplastics (Dris et al., 2017; Dris et al., 2016; Xu et al., 2004), release from solid waste dumping sites, and intensive agricultures (Cai et al., 2017). Further, sea breeze and sea spray can also be an important source of MPs' nearer to coastal areas. A study, estimated about 136,000 tons/year MPs are emitted from marine to the atmosphere as sea spray (Allen et al., 2020). As the atmosphere does not have any physical boundary, MPs and NPs pollutants released or produced in a geographical location can be easily transported to the next place. For example, an air mass trajectory study revealed that MPs travel up to 95 km in the atmosphere (Allen et al., 2019). This study reported, MP particles in marine boundary layer air samples on the French Atlantic coast, both onshore winds, which had an average of 2.9 MP/m^3, and offshore winds, which had 9.6 MP/m^3 (Allen et al., 2020). Atmospheric transportation of MPs can be affected by many environmental conditions such as wind speed, initial concentration, and wind direction impact MP transportation. The MPs on the air and particulate matter were mostly polyethylene (PE), polystyrene (PS), polyethylene terephthalate (PET), and other fibers with a size range of 10–8000 μm (Dris et al., 2016; C. Liu et al., 2019; K. Liu et al., 2019; Kumar et al., 2022).

To comprehend the risk, it is necessary to first divide current exposure concentrations into inhalable, thoracic, and respirable particles (Wright and Kelly, 2017). Currently, the atmospheric accumulation source of MPs/NPs has received little attention, and there is not sufficient information about the fate and movement of airborne MPs in the environment (Akdogan and Guven, 2019; Chen et al., 2020).

4.3. Dermal contact

In addition to ingestion and inhalation, dermal contact is an additional route for the exposure of MPs and NPs. Microplastics containing consumer products such as face creams, and face wash can increase the exposure risk of polyethylene MPs (Fendall and Sewell, 2009; Hernandez et al., 2017; Revel et al., 2018). Such customer products can have acrylic components, which can have allergic interaction with chemicals of the epoxy resin system of our body (Tosti et al., 1993). However, absorption *via* the skin is improbable due to the physiochemical properties of MPs and the fact that the uptake of nanoparticles across the skin needs penetration of the stratum corneum, which is restricted to particles below 10 and 100 nm (Schneider et al., 2009). However, some other studies suspected the possibility of NPs can permeate human skin (Revel et al., 2018; Sykes et al., 2014). Further, a possibility of MPs of exposure from air fallout as a cutaneous exposure through deposition on the skin was reported earlier (Prata, 2018; Wright and Kelly, 2017). As a consequence of various tissues, the total number of microplastics on the skin was 800 pieces (Prata, 2018; Wright and Kelly, 2017).

Further, microfibers and MPs/NPs from cosmetics and toothpaste may be absorbed by the skin. In addition, Schirinzi et al. (2017) showed *in vivo* that MPs and NPs may induce oxidative stress in epithelial cells by cutaneous contact. However, in humans, the mechanism of the human epidermis is primary and responsible for the stratum corneum (SC) effectiveness as the main physicochemical barrier, both again the permeation of exogenous compounds into the skin, and endogenous compounds out of the skin (Jepps et al., 2013; Schneider et al., 2009). This suggests that MPs and NPs may not penetrate the deeper layer of skin in humans, however, may settle on the epidermis. On the other hand, exposure to MPs/NPs with surface-adsorbed chemical compounds might result in discomfort and deeper absorption. Therefore, it is necessary to investigate the possible detrimental effects of nanoplastics and the extensive skin contact with plastic particles (*e.g.*, from dust, microbeads, and liquid hand-cleansers).

5. Toxic effects of microplastics and nanoplastics on animals

5.1. Translocation and circulation

Microplastic translocation through the gastrointestinal tract (GIs) has been reported in laboratory studies using mussels (Browne et al., 2008) and crabs (Watts et al., 2016). MPs and NPs in tissues other than the GI tract have yet to be determined in fish (Bouwmeester et al., 2015). Depending on their size and surface charge, NPs might enter the circulatory system following translocation over the gut barrier (Revel et al., 2018). Avio et al. (2015) reported the exposure of PE and PS to absorb pyrene in mussels, *Mytilus galloprovincialis* L. They demonstrated tissue localization of MPs in hemolymph, gills, and especially the digestive tissues markedly accumulated pyrene. Microplastic exposure was measured in young planktivorous fish (*Acanthochromis polyacanthus*), a common and abundant species on Indo-Pacific coral reefs. The number of plastics in the GIs vastly increased when the size of plastic particles

was reduced to approximately one-quarter of the size of the food particles, with a maximum of 2102 (300 μm) particles present in the gut of an individual fish after one week of plastic exposure under five different plastic concentration treatments, with the plastics being the same size as the natural food particles (mean of 2 mm diameter) (Critchell and Hoogenboom, 2018). Increased permeability of the gastrointestinal mucosa may occur due to malnutrition and diets high in saturated fats and high-fructose carbohydrates (West-Eberhard, 2019). Furthermore, particles smaller than 5 μm might pass through the gastrointestinal tract wall, resulting in bioaccumulation when absorption exceeds release or particles are digested in tissues or organs (Roch et al., 2020). MPs and NPs have the potential to translocate, exposing distant tissue. After inhalation and ingestion, MPs/NPs have been shown to translocate into the circulatory system and distant tissues such as the liver in rats (Eyles et al., 1995; Eyles et al., 2001). Browne et al. (2008) evaluated the consumption, transport, and accumulation of this debris in Mussels (*Mytilus edulis*). Microplastic accumulated in the intestines after consumption in this first trial. Mussels were also treated with saltwater and microplastic treatments (Browne et al., 2008). Microplastic translocation through the gastrointestinal system has been established in crabs and mussels in the laboratory. After 30 days of exposure, microplastics altered the expression levels of CYP1A transcription, and GSTa rose at first, then dropped at increasing microplastic concentrations, and histological abnormalities occurred in the livers of fish exposed PVC-MPs (Xia et al., 2020a; Xia et al., 2020b). Because epithelial barriers are more permeable during inflammation, translocation happens more often (Prata et al., 2020b).

5.2. Oxidative stress and cytotoxicity

An increase in free radicals and associated products and a lack of protection against oxidative injury due to the reduced number of antioxidant enzymes responsible for such reactions or malfunction produce oxidative stress, while cytotoxicity is defined by cell viability is mediated through the release of cytotoxic granule-related molecules (Dusinska et al., 2017; Leval and Gaulard, 2014). The majority of the knowledge comes from cellular research. Oxidative stress has been seen in mice (Deng et al., 2017) and zebrafish (*Danio rerio*) following exposure to MPs (Lu et al., 2016). These findings show that MPs consumption induces oxidative stress and microplastic-induced toxicity in organisms (Rochman et al., 2013). To prevent buildup in the body, once harmful compounds should be swiftly digested and eliminated by organisms (Sureda et al., 2006). Two stages are involved in one major process of hazardous metabolism. Compounds are metabolized in the first phase by the cytochrome P450 system, which speeds up the addition of oxygen atoms to the molecule. In the second phase, cytochrome P450 system molecules are coupled with foreign chemicals like glutathione, glucuronic acid, or sulfates to make more hydrophilic compounds (Capó et al., 2021; Falfushynska et al., 2019; Uno et al., 2012). On the other hand, an *in vitro* investigation found that MPs were detrimental to human health. Although exposure to polystyrene nanoplastics (PS-NPs) at concentrations of 0.001–10 ng/L did not cause cytotoxicity, data showed that they could alter the toxicity of human pharmaceuticals in marine fish cell lines (Almeida et al., 2019), and antioxidant activities were altered after exposure to PVC microplastics (X. Xia et al., 2020). Consequently, only cytotoxic PS-NPs with the greatest concentration potential were exposed (Lim et al., 2019). In mice (*Mus musculus*), PS and PE beads (0.5–1.0 μm) with organophosphorus flame retardants (OPERs) increased OPFR-induced oxidative stress compared to OFPR alone (Deng et al., 2018). Although common blood proteins were responsible for the protein-induced coalescence of NPs and grain development, when NPs entered the circulation, the in-and-out flow of bodily fluids might be disturbed (Gopinath et al., 2019). Furthermore, the peppery furrow shell clam, *Scrobicularia plana*, displayed antioxidant (superoxide dismutase, catalase, and glutathione peroxidase) and biotransformation (glutathione-S-transferases) enzyme activity following exposure to low-density polyethylene (LDPE) microplastics (11–13 μm) (O'Donovan et al., 2018). Furthermore, 70 nm and 50 μm MPs accumulated in large quantities in the digestive tracts of goldfish (*Carassius auratus*) larvae, perhaps causing oxidative stress (Yang et al., 2020a). Overproduction of reactive oxygen species (ROS) and mitogen-activated protein kinase (MAPK)-hypoxia-inducible factor 1 (HIF-1)/nuclear factor-kappaB (NFκB) in *Daphnia pulex* exposed to polystyrene nanoplastic (Liu et al., 2020). According to recent research, long-term exposure to microplastics in *Sparus aurata* produced liver oxidative stress after 90 days (Capó et al., 2021). Thus, exposure to MPs and NPs may produce histological changes and oxidative stress induction, and cytotoxicity might be a significant mechanism of MPs and NPs toxicity responses in marine and aquatic animals.

5.3. Deoxyribonucleic acid damage

Alterations in the expression of stress-related genes (*i.e.*, HSP60, HSP70 & GST), and other genes implicated in body function and body structure (*i.e.*, SERCA), were identified for adult Daphnia 48 h after exposure (Imhof et al., 2017). González-Soto et al. (2019) studied on effects of dietary exposure to polystyrene MPs of 0.5 and 4.5 μm alone, with absorbed benzo[*a*]pyrene (BaP) in mussels, to elucidate the effects of MP size and the presence of absorbed BaP on the organism with higher toxicity of small MPs compared to larger ones, were recorded for deoxyribonucleic acid damage (DNA damage) and cell composition of digestive tubules (DTs). For the exposure to low-density polyethylene (LDPE) microplastics (11–13 μm) and without absorbed contaminants (BaP) and perfluorooctane sulfonic acid (PFOS) in the peppery furrow shell clam, *Scrobicularia plana*, it was able to induce genotoxicity with single and double-strand DNA breaks (O'Donovan et al., 2018). Meanwhile, polystyrene nanoplastic exposure altered cell variability, induced cell cycle S phase arrest, stimulated inflammatory gene transcription and altered the expression of cell cycle proteins (Xu et al., 2019). Similarly, in the marine animal *Chlamys farrier* (Xia et al., 2020a), organism exposure to PS did not enhance BDE-209 DNA damage on hemocytes, and changed antioxidant-related gene expression was found in the livers of larvae exposed to PVC microplastics (Xia et al., 2020b). At aquatic exposure, the exposure route impacts the distribution and toxicity of polystyrene nanoplastics in zebrafish and antioxidant gene expression and hypoactivity (Zhang et al., 2020).

5.4. Inflammation and disruption of immune function

Meanwhile, PE causes significant histological changes and a severe inflammatory response in blue mussels (*Mytilus edulis L.*) (von Moos et al., 2012). PET particles of a diameter of 0.5 to 20 μm are kept in the cytoplasm of histiocytes in the joint capsule, while bigger particles with a diameter of up to 100 μm are found extracellularly in the tissue (Wright and Kelly, 2017). Particulate matter exposure has also been connected to autoimmune rheumatic illness (Bernatsky et al., 2016) and systemic lupus erythematosus (Fernandes et al., 2015). In a study of mussels subjected to PE and PS to absorb pyrene, researchers discovered that cellular impacts included changes in immunological response, lysosomal compartment, and peroxisomal proliferation (Avio et al., 2015). In another research, PS particles of different sizes were collected in zebrafish's livers, gills, and intestines, causing inflammation (Lu et al., 2016). For instance, PS MPs increased the expression of IL-1α, IL-1β, and interferon in the gut, indicating microplastic dysbiosis and inflammation in adult male zebrafish (Jin et al., 2018). Similarly, mice were inflamed in the small intestine after being exposed to PS MPs at a concentration of 10–150 μm (Li et al., 2020). Meanwhile, González-Soto et al. (2019) showed a small increase in the prevalence of inflammatory reactions in mussels *Mytilus galloprovincialis* at the tissue level following exposure to polystyrene MPs of 0.5 and 4.5 μm alone and with sorbed benzo[*a*]pyrene (BaP). For example, changes in hemocyte count, blood cell composition, phagocytic activity, intracellular content of ROS, Ca^{2+}

concentration, and lysozyme activity, which were revealed by microplastics, indicate immunotoxicity in the bivalve *Tegillarca granosa*, and exposure to MPs may hamper immune responses (Tang et al., 2020). Furthermore, following exposure to polystyrene particles, MDT-15-SBP-1 signaling triggered both the endoplasmic reticulum (ER) unfolded protein response (UPR) and the innate immunological response in the worm (*Caenorhabditis elegans*) (Yang et al., 2020b). Therefore, polystyrene microplastic particles were potential immune boosters, causing cytokine and chemokine synthesis in a diameter- and concentration-dependent manner (Hwang et al., 2020).

5.5. Neurotoxicity and neurodegenerative diseases

Data on neurotoxicity and neurodegenerative illnesses in live beings are still scarce. The researcher attempts to gather and analyze previous studies, such as the study effect after exposure to PS–NPs with 50 nm (1 mg/L) in zebrafish (*Danio rerio*) (Chen et al., 2017) and 0.1 μm in red tilapia (*Oreochromis niloticus*) (Ding et al., 2018). Similarly, O'Donovan et al. (2018) found that exposure to low-density polyethylene (LDPE) microplastics (11–13 μm) in the Clam, *Scrobicularia plana*, might cause neurotoxicity due to changes in acetylcholinesterase activity. This is because the compounds in MPs/NPs might activate AChE activity due to additive toxicity. MP particles may also reach the circulatory system and subsequently the brain by absorption through the gills, intestines, and the lungs or directly through the nasal cavity. Oxidative stress may occur in the brain, which can lead to cellular damage and neuroinflammation, increasing the risk of neurodevelopmental and neurodegenerative illnesses (Prüst et al., 2020). The only information available on MPs and NPs is their neurotoxicity effects. In the future, it will be necessary to research the effects of MPs and NPs on neurotoxicity response and illness in organisms.

5.6. Disruption of energy metabolism

The findings revealed MP dispersion and accumulation across mouse tissues and significant metabolomic changes in numerous biomarkers (creatine, 1-oxoglutarate, and citrate reduced), indicating an energy shortfall and probable toxicity to exposed MPs (Deng et al., 2017). PS was also able to collect PS microplastics in mouse guts, resulting in decreased intestinal mucus secretion, impairment of gut barrier function, metabolic problems (Jin et al., 2019), *etc.* Furthermore, the findings provided a mechanistic foundation for the putative protective role of the lipid metabolic response in nano-polystyrene-exposed *Caenorhabditis elegans* worms (Yang et al., 2020b). Therefore, microplastics that act as carriers of chemicals, proteins, and exogenous toxins existing on the particles in live organisms can cause chemical toxicity (Vethaak and Legler, 2021). Another mechanism for disrupting energy and metabolism could be this. However, the current data is insufficient to summarize the impact of MPs and NPs on the mechanisms of metabolic diseases in people and animals.

5.7. Vector for organisms and chemicals

The introduction of chemicals into a lower trophic level organism increases the likelihood that these chemicals will be biomagnified by predators (Rochman, 2015). Exposure to NP with 50 nm co-phenanthrene had an additive impact in a case study on the crustacean *Daphnia magna*, with higher immobilization of organisms than exposure to compounds alone (Ma et al., 2016). After 96 h of exposure to PE microspheres with 0, 18.4, and 184 μg/L in the presence of pyrene (20 and 200 g/L), it was discovered that pyrene decreased the energy available through the aerobic pathway of energy production and that pyrene modulated the bioavailability of pyrene and simultaneous exposure to PE MPs (Oliveira et al., 2013). MPs may also operate as a pollutant transport vector for other harmful components like hexachlorobenzene and dichloro-diphenyl-trichloroethane (DDT), finally ending up in the body of a living thing that consumes it (Laskar and Kumar, 2019). Furthermore, toxicologically heavy metal interactions between microplastics represent a substantial route into groundwater and surface water in the coastal south Indian region (Selvam et al., 2021). Furthermore, Cd, MPs, and their combinations produced oxidative stress, damage, and neurotoxicity in *Corbicula fluminea*. Superoxide dismutase (SOD), glutathione S-transferase (GST), acetylcholinesterase (AChE), and lipid peroxidation (LPO) levels could also be used as Cd pollution indicators (Parra et al., 2021). MPs also had rapid sorption kinetics (<24 h), and preloaded MPs with natural organic matter (NOM) demonstrated higher micropollutant uptake due to the formation of a complex with NOM and/or co-absorption (Ateia et al., 2020). Plastic debris was discovered to have an impact on metals in coastal ecosystems by i) providing an absorption site (Cu and Pb), particularly for PVC; ii) plastic desorption, *i.e.*, the 'inherent' load (cadmium and zinc); and iii) serving as a point source for acute trace metal exposure to coastal ecosystems (Munier and Bendell, 2018). Similarly, plastic types' long-term metal absorption was similar; implications for plastic litter in aquatic ecosystems where discovered, and high-density polyethylene (HDPE) accumulated lower metal concentrations than the other four polymers. Chemicals in the surrounding marine and aquatic water can be absorbed by smaller MPs and NPs (Sørensen et al., 2020); consequently, they act as vectors of toxic chemical dispersion and transport throughout the ecosystem while also being a mixture of dangerous chemicals themselves (Li et al., 2022). For example, Yang et al. (2022) investigated the influence of PS MPs on Pb bioaccumulation in crabs. They found that MPs are a potential vector of heavy metals and that co-exposure has more severe effects on crabs, and that the toxicity of PS–MPs was induced by the development of numerous pathogenic bacteria (Liu et al., 2022). This could be due to the increased toxicity of pollutants due to bacterial biodegradation, particularly on the skin's surface or in the digestive system of aquatic animals where bacteria thrive. In comparison, all-metal concentrations improved over time throughout the 12-month study period, and none of the metals chromium (Cr), manganese (Mn), cobalt (Co), nickel (Ni), zinc (Zn), or lead (Pb) exceeded saturation for at least one kind of plastic for the whole 12-month exposure. The longer it remains at sea, the higher the metal concentrations become. Overall, a complex mixture of metals, including those recognized by the US EPA as priority pollutants (Cd, Ni, Zn, and Pb), could be observed on plastic debris made up of various types of plastic (Rochman et al., 2014). As a result of their chemical heterogeneity, MPs and NPs could be vectors for hazardous chemicals into aquatic organisms *via* adsorption, release, and transport (Li et al., 2022). In addition, *S. colias* had the highest liver BPA levels (302 ng/g-dry weight) while *T. trachurus* had the lowest (5 ng/g-dw). In *S. colias*, however, the bisphenol with the highest concentration in the muscle was BPE (272 ng/g-DW). Fish with microplastics had somewhat greater bisphenol concentrations than fish without microplastics. Such findings highlight the need for more research into the toxicity of MP and related compounds to fish (Barboza et al., 2020).

5.8. Reproductive toxicity

According to the study's findings, oysters' oocyte quantity diameter and sperm velocity decreased after exposure to PS (Sussarellu et al., 2016). At the same time, African catfish (*Clarias gariepinus*) exposed to virgin or phenanthrene-loaded (10 or 100 μg/L) LDPE fragments were able to modify the expression of the reproductive axis genes (Karami et al., 2016). Meanwhile, PS nanoparticles (10 μm, at 2–200 μg/L) and Medaka (*Oryzias melatigma*) caused sex-dependent reproductive endocrine disruption (Wang et al., 2019). Furthermore, zebrafish (*Danio rerio*) subjected to PE MPs with a wavelength of 38.26 ± 5.64 μm were able to cause significant alterations in morphometric parameters of larvae, as well as a reduced larval survival rate (Malafaia et al., 2020). In addition, a study in *Oryzias latipes* demonstrated that PS MPs induction dose-dependency lowered egg counts in adult females after exposure to

PS MPs (Zhu et al., 2020). Furthermore, research on chronic microfiber exposure in an adult Japanese medaka (*Oryzias latipes*) discovered that polyester (PES) exposure did not affect reproduction, whereas females exposed to PP MFs produced more eggs over time (Hu et al., 2020). Within 90 min of infusion, fluorescently labeled 20 nm polystyrene nanoparticles overcame the mouse placental barrier and reached the fetal compartment into the maternal uterine artery through the umbilical vein (D'Errico et al., 2019).

Based on the review, the authors summarized the results of studies on the selected toxic effects of MPs and NPs in organisms, animal tests, and cell line cultures, which were divided into eight systems and can be classified according to the nature of the effects of exposure according to the organ system, as detailed in Table 2. Section 6 evaluates and examines the possibility of MPs and NPs having adverse effects on people.

6. Potential toxic effects of microplastics and nanoplastics on human health

As indicated in Table 2, this review summarizes known research on MPs and NPs and their potential toxicity. Humans are exposed to MPs/NPs by ingestion, inhalation, and skin contact, which may result in adverse health consequences. Recent research has provided compelling evidence that exposure to MPs/NPs poses dangers to human health (Noventa et al., 2021; Vethaak and Legler, 2021). Thence, we intend focused on *in vitro* and *in vivo* investigations of cytotoxic effects in rats and human cells. The section next summarizes several impacts that are anticipated to result in varying degrees of human effects due to exposure to MPs and NPs:

6.1. Gastrointestinal and urinary tract system

Prior research explored the translocation through the mammalian stomach into the lymphatic system of various kinds and sizes ranging from 0.1 to 150 μm, and it has been established in human investigations that these MPs ranging from may pass into the lymphatic system (Hussain et al., 2001; Schirinzi et al., 2020). Schirinzi et al. (2017) demonstrated that both MPs and NPs-PS might produce oxidative stress *in vivo* in human epithelial cells. In addition, research conducted on the stomachs of mice following exposure to PS and PS (0.5 and 50 μm) revealed a decrease in intestinal mucus production, as well as harm to the gut barrier function and metabolic consequences (Jin et al., 2019; Lu et al., 2018). In addition, it was shown that polystyrene with a particle size range of 10–150 μm and a high concentration of MPs caused inflammation in the small intestines of mice (Li et al., 2020). The most recent investigation done by Liu et al. (2022) on mice exposed to 500 mg/L PS-MPs revealed that intestinal immunological imbalance was associated with enhanced inflammatory damage (TNF-α, IL-1 β, and IFN-γ). Jin et al. (2019) determined that PS particles of 5 μm at 100 and 1000 μg/L promote intestinal barrier failure and microbial dysbiosis in the gut. Another investigation of human intestinal cells exposed to PS and NPs demonstrated histological and inflammatory effects *in vivo* (Stock et al., 2019); therefore, NPs indicated cell viability and promoted apoptosis in all cell lines (Inkielewicz-Stepniak et al., 2018). Moreover, a study conducted on human intestinal epithelial cell lines Caco-2 and derived co-cultures, mimicking intestinal M-cell/goblet cells by different sizes of spherical fluorescent polystyrene particles (1, 4, and 10 μm) after exposure *in vivo*, revealed a potential impact on intestinal immune cells, despite the absence of histologically detectable lesions and inflammatory responses (Stock et al., 2019). PS-NPs also affected cell viability and inflammatory gene expression, cell morphology was strongly induced, and there was an up-regulation of IL-6 and IL-8 genes in gastric adenocarcinoma cells (Forte et al., 2016), especially when exposed to a small size that is used in experiments can cause inflammatory damage (Elizalde-Velázquez and Gómez-Oliván, 2021). Human renal cortical epithelial (HRCE) cells exposed in the laboratory to 44 nm polystyrene nanoparticles demonstrated that NPs entered HRCE cells by many processes, either energy-dependent (endocytosis) or energy-independent of kidneys (Monti et al., 2015). In addition, an *in vitro* human digestive model research found that microplastics released more Cr than in freshwater. The gastrointestinal phase generated the most bioaccessible hexavalent chromium (Cr^{+6}), but trivalent chromium (Cr^{+3}) may provide greater non-carcinogenic dangers to humans (Liao and Yang, 2020). Therefore, it collected information from a review of both *in vitro* and *in vivo* studies. It may first corroborate that exposure to MPs and NPs in the human body through the stomach may aggravate cellular inflammation. However, nothing is known about the impact of exposure to these MPs and NPs on human health at this time. Therefore, more research on exposure in both laboratory animals and human cells is required.

6.2. Respiratory tract system

The respiratory tract is a key component of oxygen exchange and cellular respiration in higher species. Therefore, the exposure of the respiratory system to foreign chemicals will result in irritation and inflammation. Particularly, exposure to particle matter affects the respiratory system of humans. Dong et al. (2020) recently studied the exposure of PS MPs (4.06 ± 0.44 μm) to human lung epithelial cells (BEAS-2B) at concentrations ranging from 1 to 1000 μg/cm^2. Experiment results revealed that PS MPs generated cytotoxic effects, oxidative stress, and inflammatory response. Moreover, it was shown that the particle size and concentration of PS MPs affected the induction of cellular damage. Human bronchial epithelial cells treated with PS nanoparticles at extremely high concentrations exhibited cytotoxicity and endoplasmic reticulum stress-related metabolic changes, according to Lim et al. (2019). PS with a length of 60 μm induced ROS and endoplasmic reticulum stress in macrophage and lung epithelial cell cultures, resulting in autophagic cell death (Chiu et al., 2015). Paget et al. (2015) subjected human lung epithelial cells (Calu-3) and macrophages (THP-1cell lines) to PS nanobeads (50 nm) to exhibit DNA damage and produce cytotoxic and genotoxic effects on Calu-3 epithelial cells and THP-1 macrophages. In addition, the examination of human alveolar type II epithelial cell lines exposed to PS nanoparticles (25 and 70 nm) revealed an influence on the elevation of NS-kB and inflammatory cytokines transcripts and a correlation between cell cycle and protein expression (Xu et al., 2019). Factors influencing the possible toxicological impact of PS–NPs on alveolar epithelial cells include exposure concentration, exposure size, and exposure duration (Xu et al., 2019). PS-MPs induce pulmonary cytotoxicity and enhance respiratory symptoms as a result (Dong et al., 2020; Prata et al., 2020b). Moreover, human lung adenocarcinoma cells (A549) investigate the effects of PS (amino-functionalized 100 nm) on the under-fluid shear stress when cellular uptake of particles is enhanced (Kang et al., 2016). Currently, the research of rats exposed to PS-MPs triggered the production of the inflammatory protein (TGF-β), and TNF-α increased in the lung tissue for 14 days, which is a greater effect at the molecular level than at the organismal level (Lim et al., 2021). An *in vivo* (female mice) PS NPs injection investigation indicates that PS and silica model NPs persist in the lung without apparent removal over 28 days, and the long-term accumulation can cause a greater health risk owing to non-degradation (Zhao et al., 2022). According to the ATSDR (2010), inhalation of PS-MPs may cause cancer. Based on the discussion in this part, it was determined that the size and high concentration of MPs/NPs may inhibit cell viability, cytotoxic impact, and inflammatory response. Consequently, these results give scientific evidence to assist explain the compelling human impacts in situations of MPs/NPs inhalation exposure. However, the present study's findings come from an experiment in which MPs/NPs were directly exposed to experimental cells. Therefore, it is essential to investigate the control elements and settings for MPs/NPs absorption in animal testing and human cell cultures.

Table 2
The selected toxic effects of MPs and NPs on various organisms, animal tests, and cell cultures.

Systems	Samples	Microplastics	Main finding	References
Gastrointestinal tracts	**Fishes**			
	Fish (*Sparus aurata*)	MPs	- Increase was significantly higher in catalase (CAT), glutathione reductase (GRd), and glutathione-s-transferase (GST) in the seawater exposed MPs. - MPs exposure induced an increase of antioxidant defenses in the liver of *S. aurata*.	(Capó et al., 2021)
	Fish farm and maricultural area	MPs	The total number of MPs in the gill (746) was higher than that in the gut (514), implying the potential high risk of scaleless fish consumption for human health through the food chain by seafood consumption.	(Feng et al., 2019)
	Freshwater fish (*Squalius cephalus*)	Fibers	Eighteen anthropogenic particles were found in fish stomachs, with a length of 2.41 mm.	(Collard et al., 2018)
	Freshwater fish	Fibers	- Villi cracking and splitting of the enterocyte	(Lei et al., 2018)
	Zebrafish (*Danio rerio*)	PA, PE, PP, PVC, and PS	- Intestinal damage to the adult fish gut	
	Adult male zebrafish	PS	- PS MPs increased the expression of IL-1α, IL-1β and interferon in the gut, indicating microplastics dysbiosis and inflammation.	(Jin et al., 2018)
	Zebrafish (*Danio rerio*)	PS and PE NPs	- Alteration in intestinal mucosa and gill epithelial with higher neutrophil infiltration	(Limonta et al., 2019)
	Mussels			
	(*Mytilus galloprovincialis* L.)	PE, PS to adsorb pyrene	- Tissue localization of MPs occurred in hemolymph, gills, and especially digestive tissue that were a marked accumulation of pyrene. - Cellular effects included alteration of immunological response, lysosomal compartment, and peroxisomal proliferation. - The evidence that MPs adsorb PAHs, emphasizing a high bioavailability of these chemicals after the ingestion	(Avio et al., 2015)
	Blue mussel (*Mytilus edulis* L.)	PE	- Notable histological change and a strong inflammatory response	(von Moos et al., 2012)
	Mice or Mouse			
	Mice	500 μg/L polystyrene microplastics (PS-MPs)	- An increased expression of inflammation factors (TNF-α, IL-1 β, and IFN-γ) and intestinal immune imbalance. - Exposure to PS-MPs aggravated induce the histopathological damage in colonic mucosa.	(Liu et al., 2022)
	Male mice	PS	- An accumulation of PS microplastics among mice guts consequently caused the reduction of intestinal mucus secretion damage to gut barrier function, and metabolic disorders in mice.	(Jin et al., 2019)
	Mouse	PS MPs (10–150 μm)	- High concentration of MPs induced inflammation of the small intestine.	(Li et al., 2020)
	Mouse	PS particles 5 μm at 100 and 1000 μg/L	- Intestinal barrier dysfunction was caused. - Gut microbiota dysbiosis was induced.	(Jin et al., 2019)
	Mouse	PS particles (0.5 and 50 μm)	- Decrease in mucus secretion in the gut - Alteration in gut microbiota	(Lu et al., 2018)
	Human cells			
	Human intestinal epithelial cell lines Caco-2 and there of derived co-cultures mimicking intestinal M-cell/goblet cells *in vivo*	PS	- The absence of histologically detectable lesions and inflammatory responses was found *in vivo*. - The particles did not interfere with the differentiation and activation of the human macrophage model. - The present result suggests that oral exposure to PS microplastic particles under the chosen experimental conditions does not pose a relevant acute health risk to mammals.	(Stock et al., 2019)
	Cell culture (intestinal epithelial cell lines, LS174T, HT-29, and Caco-2)	NPs	- The effect of NPs on mucin and its potential role during NP–cell interaction was investigated. - Positive NPs affected cell viability and induced apoptosis in all cell lines independently of their ability to produce mucin.	(Inkielewicz-Stepniak et al., 2018)
	Human gastric adenocarcinoma epithelial cell (AGS)	PS–NPs	- This study evaluated the kinetics of uptake of 44 nm and 100 nm unmodified PS–NPs in gastric adenocarcinoma cells. - PS–NPs also affected cell viability, inflammatory gene expression, and cell morphology. - NP44 strongly induced an up-regulation of IL-6 and IL-8 genes, two of the most important cytokines involved in gastric pathologies.	(Forte et al., 2016)

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Table 2 (*continued*)

Systems	Samples	Microplastics	Main finding	References
	Human intestinal epithelium *in vitro*	PS	- The nanoparticle-induced apoptosis in individual cells, which then propagated across the cell monolayer through "bystander killing effects." - This study confirmed that ingested nonbiodegradable nanoparticles represented a potential health risk due to their detrimental impact on the intestinal membrane by destroying their barrier protection capability overtime.	(Thubagere and Reinhard, 2010)
Kidney	Human renal cortical epithelial (HRCE) cell	44 nm, polystyrene NPs	- NPs entered HRCE cells through multi mechanisms either energy-dependent (endocytosis) or energy-independent.	(Monti et al., 2015)
Respiratory tract	**Human cells** Alveolar lung organoids cultured from epithelial cell progenitors, isolated both from murine lungs and human lung tissue.	Nylon (11 × 30 μm) or PE (15 × 53 μm) microfibers	- Exposure to nylon or PE microfiber resulted in significantly fewer human and rat respiratory organs after 14 d of culture. - The organoids in the alveoli are not affected by these fibers, and exposure of developed organoids from day 14–21 day to fibers or a fiber-conditioned medium did not have any effect on organoid sizes or numbers.	(Song et al., 2022)
	Human lung epithelial cells (BEAS–2B)	PS MPs (4.06 ± 0.44 μm) at 1–1000 μg/cm^2	- Cytotoxic effects - Oxidative stress and inflammatory response - Disruption of the epithelial layer	(Dong et al., 2020)
	Human alveolar type II epithelial cell line	PS nanoparticles (25 and 70 nm)	- Viability decreased, and cell cycle arrest was induced. - Upregulation of transcript for NS-kB and some pro-inflammatory cytokines - Alteration of cell cycle and apoptosis-regulation was related to protein expression.	(Xu et al., 2019)
	Human bronchial epithelial cells	PS nanoparticles	- Cytotoxicity of PS and NPs was at very high concentrations. - Metabolomics analyses revealed autophagic and endoplasmic reticulum (ER) stress-related metabolic changes.	(Lim et al., 2019)
	Human lung adenocarcinoma cells (A549)	PS, amino-functionalized 100 nm	- Increased cellular uptake of particles under fluidic shear stress	(Kang et al., 2016)
	Human lung epithelial cells (Calu-3) and macrophages (THP-1cell lines)	PS nanobead (50 nm)	- Increased DNA damages and depletion of reduced glutathione in both cell lines - Induced similar cytotoxic and genotoxic effects on Calu-3 epithelial cells and THP-1 macrophage	(Paget et al., 2015)
	Female mice	PS-NPs	- Indicated that PS-NPs were retained in lung organ without apparent excretion within 28 d.	(Zhao et al., 2022)
	Rats	PS-MPs	- Inspiratory time was decreased in male rats. - Respiratory frequency was increased, and inspiratory and expiratory time were decreased in female rats. - Inflammation increased. - The expression of the inflammatory protein (TGF-β) and TNF-α increased in the lung tissue. - Overall, the findings showed that exposure of PS-MPs to rats to 14-day inhalation had a more pronounced impact at the molecular level than at the organismal level.	(Lim et al., 2021)
Reproductive	**Fishes** Zebrafish (*Danio rerio*)	PE MPs, 38.26 ± 15.64 μm	- MPs induced significant changes in morphometric parameters of larvae. - MPs caused a lower larval survival rate after egg hatching.	(Malafaia et al., 2020)
	Oryzias latipes	PS MPs, 10 μm	- Dose-dependent decreased egg number in mature females.	(Zhu et al., 2020)
	Medaka (*Oryzias melatigma*)	PS nanoparticles (10 μm, at 2–200 μg/L)	- Reproductive endocrine disruption in a sex-dependent manner	(Wang et al., 2019)
	African catfish (*Clarias gariepinus*)	Virgin (50 or 500 μg/L) or phenanthrene-loaded (10 or 100 μg/L) low-density polyethylene (LDPE) fragments	- Changes in the expression of reproductive axis genes	(Karami et al., 2016)
	Mussels Oysters	PS	- Oocyte number diameter and sperm velocity decreased in oysters.	(Sussarellu et al., 2016)
Serum or blood	**Fishes** Fathead minnow (*Pimephales promelas*) neutrophil extracellular	PS (41.0 nm) and polycarbonate (PC), 58.7 nm-NPs	- Significant increases in innate immune responses (in terms of degranulation of primary granules and trap release)	(Greven et al., 2016)
	Gilthead seabream (*Sparus aurata*) & European sea bass (*Dicentrarchus labrax*)	Virgin polyvinyl chloride (PVC) and polyethylene (PE), 40–150 μm	- Increased oxidative stress burst in leukocytes of *Sparus aurata* - Upregulation of the redox regulation nuclear factor erythroid 2-related factor 2 (Nrf2) in leukocytes of *Sparus aurata*	(Espinosa et al., 2018)

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Table 2 (*continued*)

Systems	Samples	Microplastics	Main finding	References
	African catfish (*Clarias gariepinus*)	Virgin (50 or 500 μg/L) or phenanthrene-loaded (10 or 100 μg/L) low-density polyethylene (LDPE) fragments	- Change in blood chemistry	(Karami et al., 2016)
	Mouse	PS MPs (0.5 and 5 μm)	- Microplastic exposure caused a change in serum.	(Luo et al., 2019)
		PS MPs (10 and 150 μm)	- The secretion of IL-1α in serum increased, and the Th17 and Treg cells among CD^{4+} cells decreased.	(Li et al., 2020)
	Immune cells	Acrylonitrile butadiene styrene (ABS) and PVC	- Larger PVC tended to induce interleukin 6 (IL-6) release and tumor necrosis factors-α (TNF-α). - Smaller ABS induced the production of IL-6 at high concentrations.	(Han et al., 2020)
	Human serum albumin	NPs	- The coronated-NPs with increased protein conformation changes caused a higher genotoxic and cytotoxic effect in human blood cells.	(Gopinath et al., 2019)
Brain and nervous	**Fishes** Zebrafish (*Danio rerio*)	PS-NPs (50 nm, 1 mg/L)	- Acetylcholinesterase activity was inhibited.	(Chen et al., 2017)
	Red tilapia (*Oreochromis niloticus*)	PS-NPs (0.1 μm, at 1, 10 and 100 μg/L)	- Brain acetylcholinesterase (AchE) activity was inhibited.	(Ding et al., 2018)
	Corbicula fluminea	Cadmium (Cd), to microplastics (MPs) and their mixtures.	- The results showed that Cd, MPs, and their mixtures caused oxidative stress, damage, and neurotoxicity. - Exposure to MPs induced an increase in reduced/oxidized glutathione (GSH/GSSG) ratio and increased AChE activity. - The combined exposure to Cd and MPs caused a synergetic effect in the gill and gonad, while an antagonism response was recorded in the digestive gland.	(Parra et al., 2021)
	Mice (*Mus musculus*)	PS and PE beads (0.5–1.0 μm) + organophosphorus flame retardants (OPERs)	- MPs enhanced OPFR-induced oxidative neurotoxicity compared to OFPR alone.	(Deng et al., 2018)
		PS-MPs of 5 μm and 20 μm	- In the liver, dose-dependent increase in AChE, LDH, GSH-Px, and SOD activity; a dose-dependent decrease in ATP and CAT in the liver (≥ 0.01 mg/day, both 5 and 20 μm).	(Deng et al., 2017)
	Human cells T98G and Hela cerebral & epithelial human cells	NMs and MPs/NPs	- Oxidative stress of the mechanism of cytotoxicity was at the cell level.	(Schirinzi et al., 2017)
Placental	Human placental perfusion model	Carboxylate modified polystyrene particles (50 and 300 nm)	- The fate transport of polystyrene particles in the fetal to the maternal direction - All polystyrene particles accumulated in the syncytiotrophoblast of the placental tissue. - The syncytiotrophoblast was the key player in regulating nanoparticle transport across the human placental. - These findings were important for reproductive toxicology.	(Grafmueller et al., 2015)
Skin	Human dermal fibroblasts (HDFs), human peripheral blood mononuclear cells (HPBMCs), and the human mast cell line (HMC-1)	PS particle	- Human intake of PS particles from personal care products can occur *via* absorption through the skin. - PS particles induced cytokine and chemokine production.	(Hwang et al., 2020)
	Fish farm and mariculture area in six major wild fish species (including *Thryssa kammalensis*, *Amblychaeturichthys hexanema*, *Odontamblyopus rubicundus*, *Cynoglossus semilaevis*, *Chaeturichthys stigmatias*, and *Collichthys lucidus*)	MPs	- The total number of MPs in the skin or gills was higher than in the gut. - As to different tissues, the total number of MPs on the skin (800) or in the gills (746) was higher than that in the gut (514). - In terms of skin, MPs' abundances in three species of scaleless fish with mucus (*A. hexanema*, *C. stigmatias*, and *O. rubicundus*) were generally higher than the other three fishes with scales (*C. lucidus*, *C. semilaevis*, and *T. kammalensis*).	(Feng et al., 2019)
Other Intracellular development	Human embryos and human pluripotent stem cells (hiPSCs)	PS-NPs	- Two pluripotency genes, LEFTY1 & LEFTY2, which encoded secreted ligands of the transforming growth factor-beta, were downregulated while CA4 and OCLM were related to eye development. - They used the HiPathia method, which uncovered the diseased mechanism and the APOC3 circuit responsible for increased risk for ischemic cardiovascular disease.	(Bojic et al., 2020)

6.3. Blood and immune system

PE wear particles may be plentiful in the lymph nodes around joint replacements in humans (Leugering and Püschner, 1978; Morawski et al., 1995), such that macrophages harboring PE particles entirely replace the lymph nodes (Urban et al., 2000). Changes in serum levels were seen in rats exposed to PS-MPs at concentrations of 0.5 and 5 μm (Luo et al., 2019). Recently, Li et al. (2020) found that rats exposed to PS MPs with 10 and 150 μm secreted more IL-1 in their blood, whereas Th17 and Treg cells were reduced among $CD4^+$ cells. Han et al. (2020) found that exposure of immune cells to acrylonitrile butadiene styrene (ABS) and polyvinylchloride (PVC) stimulated the production of interleukin 6 (IL-6) and tumor necrosis factors-α (TNF-α) from PVC. In addition, exposure to tiny ABS generated massive quantities of IL-6 products. In addition, exposure to nanoplastics in human serum albumin induces protein conformational changes and genotoxic and cytotoxic consequences (Gopinath et al., 2019). Experiments on human-derived cells employing microplastics have recently shown that exposure to PVC and ABS induces immunological responses and short-term immunotoxicity (Han et al., 2020). To define the future threshold limit, the effects of MPs and NPs on inflammation and laboratory animals' immune systems and human cells will be evaluated (Burgos-Aceves et al., 2021a, 2021b; Burgos-Aceves et al., 2018a; Pagano et al., 2019). It was demonstrated that both MPs and NPs were exposed to the blood system. Consequently, there is a potential for significant changes if small sizes and high concentrations can stimulate the level of immune cells and blood cells, necessitating the urgent development of a risk assessment framework and concepts for human exposure to microplastics.

6.4. Brain and nervous system

One *in vitro* investigation found that MPs are hazardous to human health. Exposure to PS MPs (10 μm) and PS NPs (40 and 250 nm) at 10 ng/mL to 10 μg/mL in cerebral and epithelial human cells was able to induce oxidative stress, contributing to cytotoxicity at the cell level (Schirinzi et al., 2017). Furthermore, the tissue distribution, accumulation, and tissue-specific health risk of PS MPs in mice were also investigated (Deng et al., 2017). To learn more about the effect of nanomaterials (NMs) and MPs/NPs, T98G and Hela cerebral human cells were exposed to various concentrations (50 μg/mL to 10 mg/mL). Oxidative stress and cytotoxicity were induced at the cell viability (Schirinzi et al., 2017) because of reactive oxygen species (ROS), which were significantly increased in T98G cells after exposure to both MPs (Elizalde-Velázquez and Gómez-Oliván, 2021). It was shown that PS and PE beads with organophosphorus flame retardants (OPERs) increased OPFR-induced neurotoxicity in mice relative to OFPR alone (Deng et al., 2018). In addition, the research by Deng et al. (2018) on mice exposed to PS and PE beads (0.5–1.0 μm) with organophosphorus flame retardants (OPERs) revealed that PS and PE increased OPFR-induced oxidative neurotoxicity in comparison to OFPR alone. In addition, exposure to MPs and NPs may decrease acetylcholine esterase activity and modify neurotransmitter levels, which may contribute to observed behavioral abnormalities (Prüst et al., 2020). Polymeric nanoparticles proved that the therapeutic medication delivery method might access the central nervous system (Patel et al., 2012). In contrast, the smaller NPs are identical to the treatment nanoparticles when exposed. Therefore, it is plausible that these NPs may disrupt neuronal systems and lead to persistent central nervous system (CNS) illnesses or neurodegenerative disorders in the future. These findings imply that MPs/NPs may generate oxidative stress, which may be a key mechanism for oxidative neurotoxicity on the cellular level. However, the neurotoxic danger and health risk assessment of exposure to nano-and microplastics (NMPs) need immediate clarification and investigation.

6.5. Embryos and placental barrier

The placenta is one of the most vital organs for life support during pregnancy (Arumugasaamy et al., 2020). It is hemochorial, meaning there is direct contact between maternal blood and fetal chorion, and it has a discoid form, which is unique to non-human primates, rabbits, and rodents (Arumugasaamy et al., 2020). There is currently evidence of MPs in the human placenta with a range of 5 to 10 μm (in the fetal side, maternal side, and chorioamnionitis membranes); all MPs were pigmented three were stained with polypropylene (Ragusa et al., 2021), and detected polyethylene (PE), polypropylene (PP), and polystyrene (PS) with a size >50 μm in placenta and meconium acquired from cesarean delivery (Braun et al., 2021). Recent research on human embryos and pluripotent stem cells (hiPSCs) by Bojic et al. (2020) has shown that carbonic anhydrase–IV (CA4) and ovarian cancer liver metastases (OCLM) are associated with ocular development. In previous research by Grafmueller et al. (2015), the *ex vivo* human placenta fusion model (HPFM) was used to examine the transport pathways behind the placental transfer of PS-NPs (50 to 300 nm). In addition, a human placenta perfusion model revealed that 240 nm PS may pass the placental barrier (Wick et al., 2010). This revealed that PS-NPs were collected in the syncytiotrophoblast tissue of the placenta. Thus, it has been proven that the syncytiotroplast regulates the trafficking of PS-NPs into the human placenta (Elizalde-Velázquez and Gómez-Oliván, 2021). Moreover, this process may result in unfavorable pregnancy outcomes and fetal development limitations (Ilekis et al., 2016). Due to the vital function of the placenta in facilitating fetal development and serving as an interface between the fetus and its external environment, the presence of exogenous and potentially dangerous MP particles is of major concern (Ragusa et al., 2021). These data will aid in elucidating the process for a better comprehension of the passage of MPs/NPs into the placenta and may lead to the idea of fetal developmental defects, which may result in deformities and embryotoxicity.

Microplastics and nanoplastics may result from the degradation of plastic products, and their potential consequences on human health are as follows: MPs come into touch with epithelial linings in the colon or lungs, which may result in physical, chemical, and biodegradable toxicity and may also be stored (Vethaak and Legler, 2021). Several *in vitro* (*i.e.*, human cell culture) and *in vivo* (mice) studies indicate the potential for ingested or inhaled microplastics to cause a variety of toxic effects, including induced oxidative stress, secretion of cytokines, cytotoxic effects, inflammatory and immune responses, DNA damage, metabolic disorders, and oxidative neurotoxicity. In general, it is believed that the features of microplastics, such as chemical composition, size, form, surface charge, and concentration, impact human health (Wright and Kelly, 2017). Moreover, MPs/NPs with chemical makeup, pose health concerns to humans (Rahman et al., 2021). Therefore, the harmful effects of microplastics and nanoplastics on animals and cell cultures are dependent on their size, shape, dose, and chemical composition. Particularly, human and animal exposure to persistent organic pollutants (POPs) such as polycyclic aromatic hydrocarbons (PAHs) cause toxicity, and it is essential to pay attention to the combined biotoxicity induced by the interaction of PAHs and MPs (Sun et al., 2021). In addition, have papers described the influence of MPs/NPs on gene set enrichment after exposure of hiPSCs to PS-NPs, such as induced pluripotent stem cells, atrioventricular heart valves, and cellular dysfunction? (Bojic et al., 2020). In a recent study, Kumar et al. (2022) discovered that extended exposure to PS and PVC, frequent MPs, and NPs found in human implants *via* ingesting, inhalation, and cutaneous exposure, may induce exposure carcinogenesis. However, there are currently no laws to manage and monitor this risk and its implications for secondary MPs (Karbalaei et al., 2018). During this review, these are all crucial topics that will aid in elucidating the practicality and exchanging information about the influence of MPs/NPs on human health. Based on this review, we may summarize in Fig. 3 the potential exposure pathways that lead to potential human health toxicity.

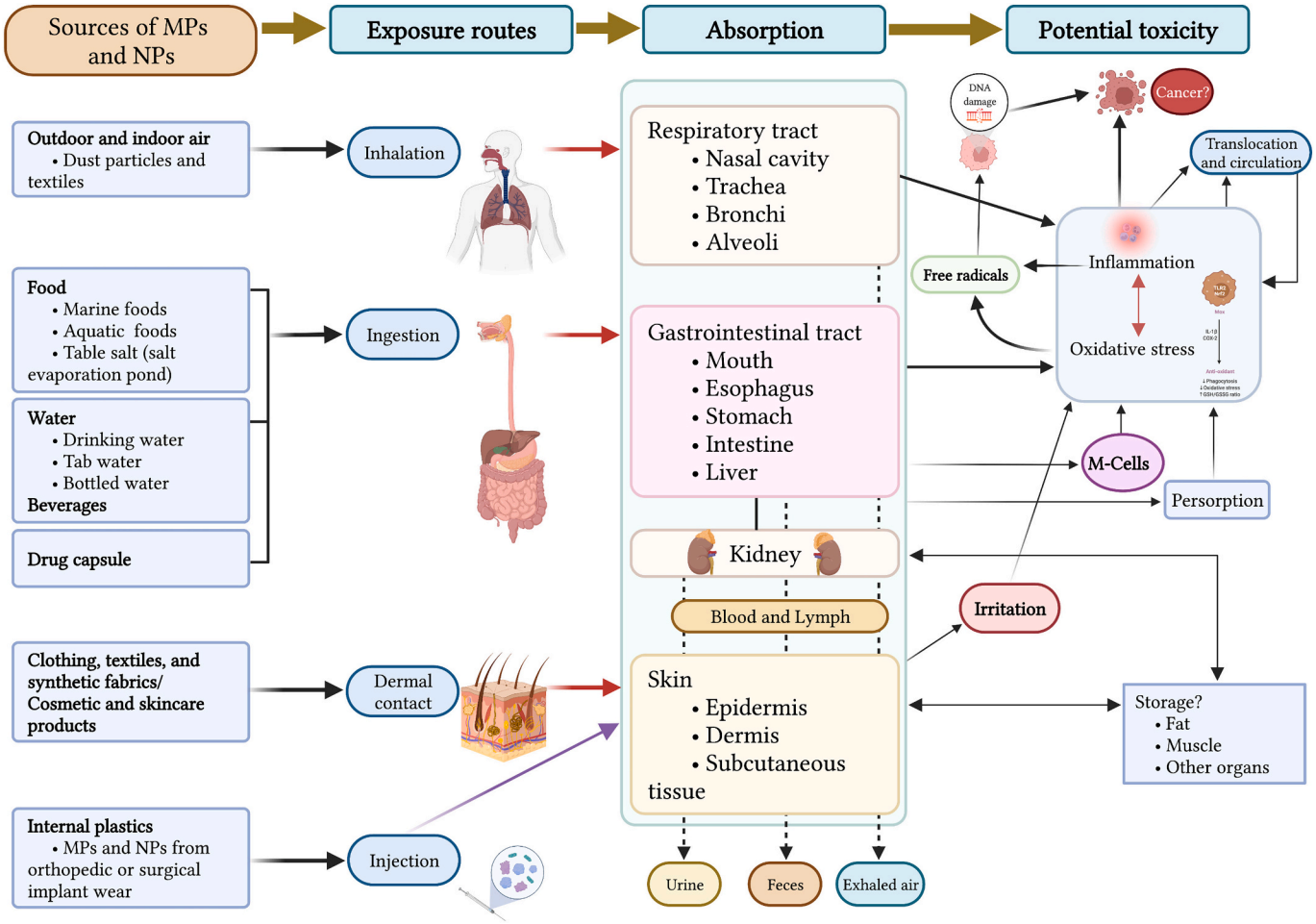

Fig. 3. Potential pathways and routes of exposure to MPs/NPs and potential toxic effects on humans.

7. Conclusions and perspectives

MPs/NPs are emerging pollutants, pervasive in the environment from many primary and secondary sources. The primary route of exposure to these pollutants is through ingestion (oral intake) of contaminated food, drinks, and drugs; inhalation of contaminated air; and dermal contact with contaminated exposure. Major toxic effects of MPs and NPs on animals are cell viability, oxidative stress, decreased immune response, inflammation, cytotoxicity, DNA damage, disruption of metabolism, neurotoxicity, impaired reproductive activity, and tumors in animals and human cells. Further, more detailed and comprehensive studies of MPs and NPs on human health risks from various exposure routes are needed (Kumar et al., 2022). This review has indicated that the possible human health consequences of MPs and NPs have potential effects on the digestive, respiratory, circulatory, and immunological systems, as well as the nervous, embryonic, and placental systems; however, additional multicellular response research is required. Future research must investigate measuring the exposure length, chemical additives, size, and concentration that are detrimental to organisms, and people participating in the health risk assessment of MPs and NPs in food products must also be monitored.

Furthermore, research about the environmental exposure to MPs and NPs *via* many sources such as shellfish and fish, or drinking water from plastic bottles, is required. MPs/NPs particles may penetrate organs, and those of this size can overcome cell membranes, and the blood-brain barrier, and reach the placenta, assuming particle dispersion in secondary tissues such as the liver, muscles, and brain is conceivable. Furthermore, there is insufficient information to understand the health implications of MPs and NPs; however, effects could be caused by physical properties such as size, shape, length, chemical properties (presence of chemical additives and polymer type), concentration, or microbial biofilm growth. Several investigations on pathogenic processes at the cellular and tissue levels and the long-term implications of tissue/organ accumulation are required. More research is needed to understand better MPs/NPs pollutants in the environment and their consequences on human health (Akdogan and Guven, 2019; Baechler et al., 2020; Haegerbaeumer et al., 2019; Horton et al., 2017; Sui et al., 2020).

In summary, the most important unanswered questions are as follows: 1) Long-term assessments of multiple species, such as model ecosystems and mammals, should be conducted to examine effects with greater human relevance; 2) standardization of MPs/NPs concentrations and exposure conditions, as well as quality assessments, are required to obtain more reliable and comparable data; 3) A standard procedure or protocol for MPs/NPs analysis and exposure assessment tool must be established to measure health risk assessment, particularly exposure to ingestion and inhalation pathways; and 4) and research on the protocol, toxicokinetics, and toxicity of MPs/NPs are required to improve understanding of their potential health impacts on seafood, aquatic food, tap and drinking water, and human health. Furthermore, a food safety study is required to assess the hazards and health concerns that microplastic-contaminated food products, particularly fish, crabs, mussels, shellfish, and other seafood, pose to consumers, as well as drinking water (Abo-Al-Ela and Faggio, 2021; Burgos-Aceves et al., 2018b; Burgos-Aceves and Faggio, 2017; Burgos-Aceves et al., 2019). To offer a solid foundation for risk assessment, experimental designs must be able

to distinguish between the effects of dietary restriction and plastic particle toxicity (PPT), as well as to show if MPs/NPs have effects that vary from those generated by natural particles (Kögel et al., 2020; Ogonowski et al., 2016). Therefore, monitoring should include not only the gastrointestinal tracts but also muscle and liver, and possibly the kidneys, as (rats and dogs) experiments revealed that MPs/NPs that crossed the intestinal barrier and entered the bloodstream are received by the liver and kidneys (Kögel et al., 2020).

There is evidence of increased manufacture of single-use plastic, notably face masks and plastic bags, during the continuing COVID-19 epidemic and beyond, raising environmental hazards (Aragaw, 2020; Fadare and Okoffo, 2020; Sangkham, 2020, 2022; Stojkovic et al., 2021). These are now strewn in several locations, contaminating soil, freshwater, and marine habitats. As a result, it is critical to push and invest in research on the impact of plastic trash on ecosystems and human health to react appropriately in the future to a similar problem. To examine the bioaccumulation of MPs and NPs in humans through the food chain in diverse geographical localities, planning and cooperation efforts involving ecologists, pathologists, and epidemiologists, as well as in fields addressing environmental health and medicine, are required. With the expected rise of synthetic details in our environment, more research is required to properly comprehend the health risks associated with MPs and NPs exposure, which necessitates understanding human exposure, etiology, and impacts. Reliable exposure data is essential for risk assessment and management. This knowledge will aid in understanding their influence on creatures and health impacts, and policy-makers should cooperate and implement plastic waste reduction policies at the national and international levels as soon as possible.

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Sarawut Sangkham: Conceptualization, Methodology, Resources, Validation, Formal analysis, Investigation, Writing – Review & Editing, Writing – Original Draft, Visualization, Supervision, and Project administration. **Orasai Faikhaw:** Validation, Writing – Review & Editing. **Narongsuk Munkong:** Validation, Writing – Review & Editing. **Pornpun Sakunkoo:** Validation, Writing – Review & Editing. **Chumlong Arunlertaree:** Writing – Review & Editing. **Murthy Chavali:** Writing – Review & Editing. **Milad Mousazadeh:** Writing – Review & Editing. **Ananda Tiwari:** Validation, Writing – Review & Editing.

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