

Review Article

Impact of Microplastics on Human Health through the Consumption of Seafood: A Review

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Abstract

Microplastics (MPs) pose a significant risk to human health, particularly through seafood consumption. Once ingested, MPs can spread from the digestive system to other organs via phagocytosis and endocytosis, leading to toxicological effects. Accumulation of MPs in tissues causes swelling, blockages, oxidative stress, and Cytotoxicity. Studies show MPs alter metabolism, disrupt immune function, and contribute to autoimmune diseases. Chronic exposure has been linked to neurotoxicity, vascular inflammation, and increased cancer risk due to DNA damage. MPs can cross biological barriers, including the placenta, affecting fetal development. Additionally, they serve as vectors for pollutants and bacteria, further complicating health risks. MPs in the bloodstream can trigger inflammatory responses, endothelial adhesion, and red blood cell coagulation, leading to cardiovascular complications. *In vitro* studies indicate MPs impair renal function and cause long-term inflammation in distal tissues. Moreover, oxidative stress caused by MPs plays a critical role in carcinogenicity. Despite growing evidence of adverse health effects, further research is necessary to understand the full impact of MPs' exposure on human health and develop effective mitigation strategies.

Introduction

Microplastics in humans

Microplastics (MPs) pose a serious risk to human health after consuming seafood. There is a significant chance that MPs' contamination of the digestive system will spread to other parts of the body. Per sorption and endocytosis are two of the most popular routes via which MPs enter the human body. Given that humans eat fish as a major source of nutrition, but throw away, seafood microplastics enter in human body to cause toxicological effects [1-4]. Effects of Toxicology on human health, the accumulation of MPs, plastics in tissues results in swelling and blockage [5,6]. According to *in vitro* experiments, MPs cause adverse cellular changes in fish and concentrate in the gills, stomachs, and metabolic systems of crabs [7,8]. It has also been demonstrated that they carry pollutants and microorganisms [9]. Such negative effects were mostly determined by the individual's sensitivity and level of exposure [10]. Additionally, MP exposure has been connected to cytotoxicity, oxidative stress, and tissue

transfer, shown in Figure 1 [11-14]. In human consequently exposed to MPs for a prolonged amount of time, which may result in immune cell impairment, chronic discomfort, swelling, cell growth, and death [15]. Patients with MPs had a significantly higher rate of inflammatory bowel disease than the general population [16]. Over time, MPs inhibited

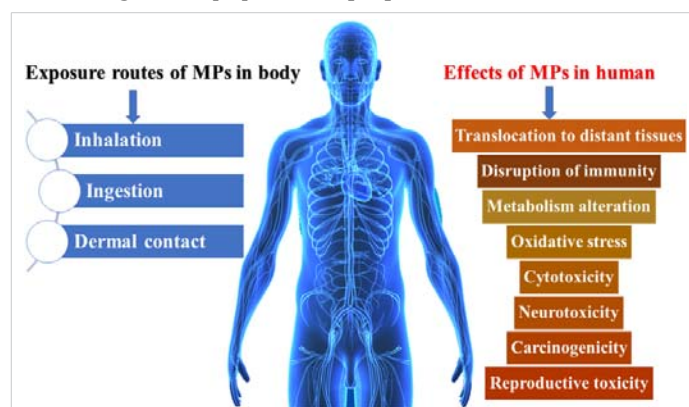


Figure 1: MP exposure has been connected to Cytotoxicity, oxidative stress, and tissue transfer.

More Information

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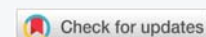
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the growth of Caco-2 cells [17]. A range of microbes may also use MPs as vectors [18]. They to absorb materials from their surroundings or expel substances from their matrices [19] (Figure 2).

Impact on oxidative stress

The primary mechanisms for MPs' toxicity in inhalation exposure experiments were oxidative stress and the ensuing inflammatory and cytotoxic effects (Table 1). By producing oxidizing substances that adhere to their surface and reactive oxygen radicals produced by the host during inflammation, MPs can cause oxidative stress [20-22] employed microplastics to stimulate human stomach cancer cells and pro-inflammatory lung cells. Larger polyethylene particles

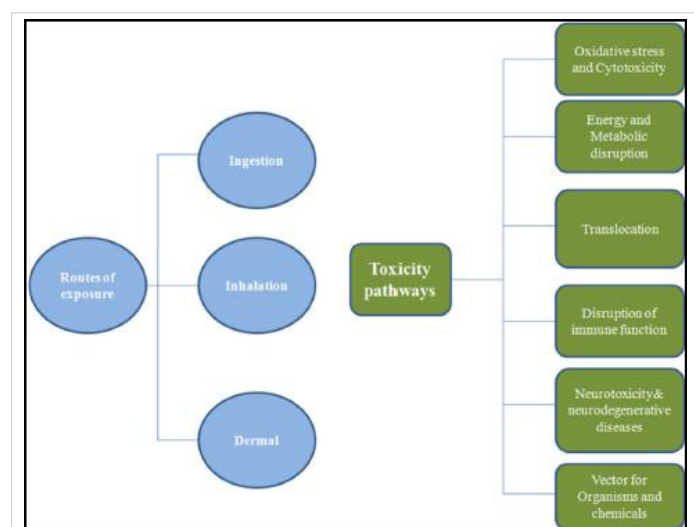


Figure 2: A Pictorial representation displaying the three primary paths of human exposure to MP, i.e., via the lungs, the gastrointestinal (GI) system, and skin.

(0.3–10 μm) have been shown to stimulate the production of cytokines, including TNF- α , IL-1 β , and IL-6, some of which are inflammatory agents. Reactive oxygen species are a part of MPs because of processing and polymerization. However, exposure to UV light or the presence of reactive metals can greatly increase the amount of these free radicals. Free radicals were created as MPs aged and oxidized the target tissues [23].

Impact on cytotoxicity and metabolism

According to Inkielewicz-Stepniak, polystyrene microplastics attached themselves to the intestinal epithelial surface. Because of the severe inflammatory response, it has been observed that human limbs and joint prostheses incorporating MPs release acute toxins and free radicals. It was demonstrated that those oxidants produced hydrolysis, which resulted in the breaking and leaking of polymers. The human organs may reject the prosthesis as a result of this free radical manufacturing process [24,25]. It was discovered that MPs' cytotoxicity resulted from inflammation and oxidative damage. *in vitro* studies on human brain and epithelial cells those MPs at concentrations of 0.05–10 mg/L generated reactive oxygen species to high levels, which contributed to cytotoxicity. Moreover, exposure of lung epithelial cells and macrophages to microplastics *in vitro* increased the production of reactive oxygen radicals, which in turn led to cytolysis and the agglomeration of unfolded protein particles in the endoplasmic reticulum. Modifying the Body's Metabolism and Energy Flow. MPs can modify metabolic enzymes to directly affect metabolism or indirectly affect metabolism by disrupting the energy balance. MPs exhibit metabolic impacts by increasing or decreasing energy consumption, reducing nutritional intake, and regulating

Table 1: Effects on oxidative stress, inflammation, and metabolic homeostasis in humans.

| Toxic effects | Characteristics of Plastic Particles | Particle size | Details | References |
|--------------------------------|---|--|--|---|
| Inflammation | Polystyrene particles | 202 and 535 nm | Expression of IL-8 is increased. | Deng, et al. [37]; Forte, et al. [22]; Fuchs, et al. 2016; Prietl, et al. 2014; Nich and Goodman 2014; Brown, et al. [39]; Green, et al. 1998; Devane, et al. 1995a,b |
| | Carboxylated polystyrene particles | 20, 44, 500, and 1000 nm | Inflammation was induced in human lung cells. Expression of IL-6 and IL-8 is increased. | |
| | Carboxylated and amino-modified polystyrene particles | 120 nm | Multiple human cancers have increased. Inflammation. | |
| | Polystyrene MP particles | 5 and 20 μm | The liver is inflamed as a result of the inflammation. Neurotransmission has been harmed as a Result of the induction. | |
| Oxidative stress and apoptosis | Amine-modified polystyrene particles | 60 nm | Mucin has strong interaction and Aggregation. Was induced in all intestinal epithelial cells. | Mahadevan and Valiyaveetil 2021; Inkielewicz-Stepniak, et al. [24]; Liu, et al. 2018; Chiu, et al. 2015 [28]; Ruenraroengsak and Tetley 2015; Paget, et al. 2015; Thubagere and Reinhard 2010; Xia, et al. 2008 |
| | Cationic polystyrene particles | 60 nm | Autophagic cell death in mouse macrophages and lung epithelial cells has been induced. | |
| | Functionalized polystyrene, polyvinyl chloride (PVC), and poly (methyl methacrylate) (PMMA) | 20, 40, 50, and 100 nm 120 nm, 140 nm | Apoptosis was induced in a variety of human cell types, resulting in reduced cell viability due to a decrease in ATP. | |
| Metabolic Homeostasis | Pristine and fluorescent polystyrene MPs | 5 μm | Amino-addition and bile-addition metabolism changes. Induced dysbiosis of the gut microbiota and intestinal barrier failure. | Luo, et al. 2019a; Jin, et al. 2019; McCarthy, et al. 2011 Stock, et al. 2019; Luo, et al. 2019a,b; Wang, et al. 2020; Deng, et al., 2017 [37]; Xia, et al. 2016. |
| | Polystyrene particles | 30 nm | Ionic homeostasis and altered ion channel Function The distribution of cytokinesis-associated proteins and blocked vesicle transit. | |
| | MPs | 0.5 and 5 μm | Increased the chances of metabolic disorders in children | |

metabolic enzymes. In the study, humans have more complex metabolic processes and a greater energy requirement, which may have an impact on the metabolic effects [26-28].

Impact on immune system dysfunction

Based on their dispersion and human reaction, MPs have been reported to elicit either local or systemic immune responses upon exposure. Conversely, in biologically susceptible individuals, environmental exposure to MPs was sufficient to compromise immune systems, leading to autoimmune disorders or immune suppression [10,29,30] suggesting that autoimmune disorders caused by MPs may be influenced by immune modulators, chronic cell damage, and improper immune cell stimulation. This series of events would result in the production of antibodies against self-antigens. Furthermore, systemic lupus erythematosus and autoimmune rheumatic illnesses have been related to MP exposure [31,32]. Further investigation into the effects on human immune systems is therefore necessary. Transfer of Cells to Different Tissues Following exposure, MPs may move through the circulatory system to distal tissues. Internalisation of MPs caused vascular swelling, blockages, an inflammatory response, blood cell cytotoxicity, and elevated blood pressure in the respiratory system [13,33].

Impact on neurotoxicity

Chronic exposure to particulate matter, especially MPs, has been linked to *in vivo* neurotoxicity; this may be because of immune cell activation and oxidative stress in the brain [34]. These occurrences may be the consequence of direct contact with teleported particles or long-term neuronal damage caused by pro-inflammatory cytokines in the bloodstream. It has been demonstrated that MPs influence neuronal behavior and function [35]. Exposure to MPs changed serum neurotransmitter levels and increased AChE activity in the brain. Because of the high concentration of bioactive chemicals in microplastics, an *in vitro* study found that they could cause toxicity and reduce metabolic activity in different types of brain cells. Carcinogenicity Human contact with plastic products has long been linked to cancerous growths. However, up until now, no solid evidence has been established. Consuming MPs can cause long-term inflammation and irritation, which can damage DNA and lead to cancer [36]. Found evidence of pro-inflammatory agents that stimulated the vasculature and caused oxidative stress and persistent irritation caused by microplastics, which in turn led to the development and formation of cancers. These effects included toxicity and impaired metabolic activity in different types of brain cells.

Impact on the immune system

When MPs' structural materials were broken, it was claimed that the materials moved steadily along the concentration gradient. Contaminants such as these may be created when MPs come into contact with body surfaces

and are carried to deeper tissues [37]. Even at low doses, those substances demonstrated the ability to interact with endogenous hormones. As vectors, MPs carried bacteria to their intended targets, shielding them from the immune system, inducing pro-inflammatory responses, and possibly even aiding infections. Due to their large surface area, MPs were susceptible to becoming vectors when they came into contact with chemicals and bacteria. MPs may have a negligible effect on exposure to hazardous chemicals when weighed against daily food and dust consumption [38] (Figure 3).

Impact on translocation of cells to other tissues

Microplastics may move through the circulatory system to distal tissues. The cardiovascular system experienced an inflammatory response, blood cell cytotoxicity, vascular swelling, blockages, and elevated blood pressure in the respiratory system upon of MPs [33]. Microplastics can result in endothelial wall adhesion and red blood cell coagulation. MPs induce hemolysis and aid in the synthesis of the pro-inflammatory chemical histamine, to increase porosity of the epithelial membrane due to inflammation is the most important means of MP transfer [39].

Impact on placenta

In a perfusion model of the human placenta, found that 240 nm microplastics may readily cross the placental barrier. An *ex vivo* human placental perfusion model absorbed polystyrene particles, which subsequently passed through the placental barrier without compromising the stability of the explant. Particles were transported by diffusion in addition to extracellular and intracellular transporter proteins. The findings demonstrate that polystyrene microplastic can cross the placenta in a manner akin to that of other nanoparticles. Human renal cortical epithelial cells ingested microplastic (44 nm) without washing them, even after 90 minutes,

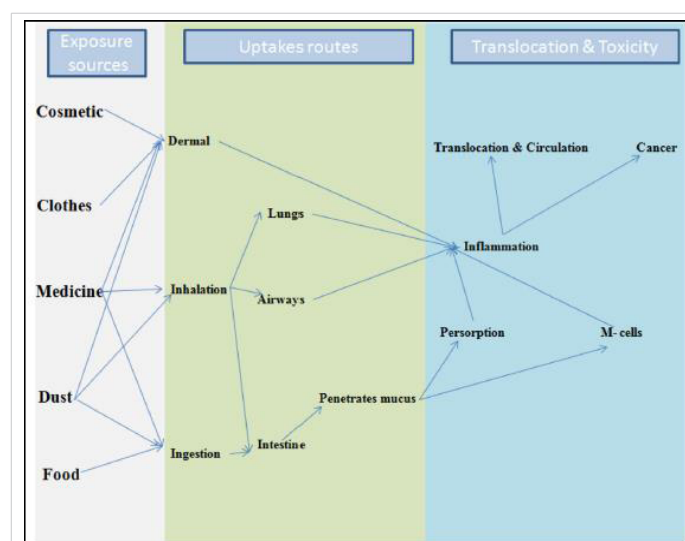


Figure 3: Potential routes of exposure and toxicity pathways for microplastics in the human body.

according to *in vitro* experiments. Conversely, the accumulation of particles resulted in significant impairments to renal active function. When MPs are transferred to distal tissues, they may result in long-lasting inflammation, compromised organ function, and an increased risk of neoplasia [40,41].

Conclusion

Microplastics (MPs) have significant toxicological effects on human health, particularly through inhalation and ingestion exposure. The primary mechanisms of MPs' toxicity include oxidative stress, inflammation, cytotoxicity, and metabolic disruptions. MPs can generate Reactive Oxygen Species (ROS) both intrinsically and through interactions with environmental factors such as UV radiation and reactive metals. This oxidative stress triggers inflammatory responses, leading to cellular damage and dysfunction. MPs also interfere with metabolic processes by altering energy consumption, reducing nutrient absorption, and modifying metabolic enzyme activity. Systemic immune responses have been observed, with MPs potentially contributing to immune suppression and autoimmune disorders such as systemic lupus erythematosus and autoimmune rheumatic diseases. Furthermore, MPs can migrate through the circulatory system, affecting distal tissues. Their presence in the bloodstream can lead to vascular inflammation, blockages, increased blood pressure, and neurotoxicity, potentially affecting neurotransmitter levels and neuronal function. There is also emerging evidence linking MPs' exposure to carcinogenic effects, as chronic inflammation and oxidative stress may contribute to DNA damage and tumor formation. Additionally, MPs can act as carriers for harmful contaminants, bacteria, and endocrine-disrupting chemicals, increasing their toxicity. They have been shown to penetrate biological barriers such as the placental barrier and renal tissues, posing risks to fetal development and kidney function. The long-term accumulation of MPs in human tissues may result in chronic inflammation, organ dysfunction, and increased risks of neoplastic diseases.

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