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Review article

The potential impact of nano- and microplastics on human health: Understanding human health risks.

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ABSTRACT

Plastics are used all over the world. Unfortunately, due to limited biodegradation, plastics cause a significant level of environmental pollution. The smallest recognized to date are termed nanoplastics (1 nm [nm] up to 1 μm [μm]) and microplastics (1 μm–5 mm). These nano- and microplastics can enter the human body through the respiratory system via inhalation, the digestive tract via consumption of contaminated food and water, or penetration through the skin via cosmetics and clothes contact. Bioaccumulation of plastics in the human body can potentially lead to a range of health issues, including respiratory disorders like lung cancer, asthma and hypersensitivity pneumonitis, neurological symptoms such as fatigue and dizziness, inflammatory bowel disease and even disturbances in gut microbiota. Most studies to date have confirmed that nano- and microplastics can induce apoptosis in cells and have genotoxic and cytotoxic effects. Understanding the cellular and molecular mechanisms of plastics' actions may help extrapolate the risks to humans. The article provides a comprehensive review of articles in databases regarding the impact of nano- and microplastics on human health. The review included retrospective studies and case reports of people exposed to nanoplastics and microplastics. This research highlights the need for further research to fully understand the extent of the impact of plastics on human health.

1. Introduction

The world of plastics, or polymers, is extensive. The term encompasses materials primarily made of polymers - large chemical compounds produced through industrial polymerization processes. By introducing specific additives to these polymers or blending them with other substances, like carbon fibers, we can tailor their physicochemical and mechanical properties to fit particular applications. (Andrady and Neal, 2009)' (Kik et al., 2020) It's no surprise that global plastic production has surged since the 1960s. For instance, in 2023 alone, the world produced 400,3 million metric tons of plastic. (Rhodes, 2018)' (Plasticseurope.org) Typically, plastics are categorized based on the chemical composition of their primary polymer and any side chains. This includes acrylics, polyesters, silicones, polyethylenes (PE), polypropylenes (PP), polyurethanes, and halogenated plastics (Napper and Thompson, 2020). A comprehensive list of the most commonly used plastics can be found in Table 1.

Microplastics (MP) are any water-insoluble synthetic solid particles or polymer matrix of primary or secondary origin, with regular or irregular shape and size ranging from 1 μm to 5 mm. (Fig. 1) (Collignon et al., 2014)' (Filella, 2015a)' (Andrady, 2011a)' (Frias et al., 2019) While fragmentation can produce particles smaller than one μm, (Cózar et al., 2014a) the precise definition of nanoplastics (NP) remains a topic of debate. Some scholars define them as particles sized between 1 nm and 1000 nm (or 1 μm), while others align with the European Commission's description of engineered nanomaterials (ENMs) — particles that measure between 1 nm and 100 nm in at least one dimension. (Gigault et al., 2018a; Cole and Galloway, 2015; da Costa et al., 2016; Schwaferts et al., 2020).

The biodegradability of plastics is influenced by the raw materials used in their creation, their final chemical composition and structure, and the conditions under which they are expected to degrade. Recent focus has shifted to the environmental release of plastics, where they break down due to factors like sunlight (especially ultraviolet rays), water, wind, or biological processes involving bacteria, enzymes, or even attacks from rodents and insects (Filella, 2015b; Lehner et al., 2019a; Palm et al., 2019).

Given its ubiquitous use, plastic pollution poses a significant global challenge. Plastic waste is omnipresent, from oceans and rivers to soil, air, and even glaciers (Andrady, 2011b; Cózar et al., 2014b). NPs, detrimental to ecosystems, can be primary (originating directly from

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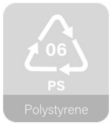
Abbreviations

BPA	bisphenol A
COOH	carboxyl group;
MPs	microplastics
NH2	amine group;
NPs	nanoplastics
PCV	polyvinyl chloride;
PE	polyethylene
PET	polyethylene terephthalate
PP	polypropylene
PS	polystyrene

Table 1
Standard classifications of plastics. Seven codes of plastics with their graphical counterpart (which make it easier to choose plastics and know which plastics to recycle), annual production and applications.

symbol	annual production in 2020 and estimation in 2040 [million metric tons] (www.statista.com)		applications
	27,5	36,4	packaging for water, drinks, oils, beer, fruit, nuts, mushrooms, pasta, groats, yoghurt containers, rice cooking bags, foil for wrapping cheese or meat
	48,5	66,4	toys for children, packaging for dairy products and cleaning products
	40,3	54,9	construction sector: pipes, veneers and casings for doors and windows, insulation of electric cables; medicine: syringes, catheters, sheaths for tubes and wires
	69,3	95,0	foil (also the one intended for food packaging), sacks and plastic bags
	75,4	98,8	storing food: bottle corks and, e.g., packaging for contact lenses; construction sector: pipes or as insulation for electric cables
	26,5	35,4	containers, lids, toys, bottles, trays, cups, disposable cutlery, and in the creation of models; construction sector: Styrofoam; medicine: material for diagnostic components, tissue culture trays, test tubes, Petri dishes, medical devices, a form of nanoparticles in pharmacological research
TOTAL:	398,2	545,9	

Abbreviations: **HDPE** = high-density polyethylene; **LDPE** = low-density polyethylene, **PET** = polyethylene terephthalate; **PVC** = polyvinyl chloride; **PP** = polypropylene; **PS** = polystyrene.

sources like clothing or industrial nanoparticles) or secondary (resulting from the breakdown of larger plastic items). Primary NPs enter the environment as particles with a diameter of less than 1 μm (Gigault et al., 2018b; Lambert and Wagner, 2016). As an illustration of the magnitude of the problem, in 2009, plastics constituted an estimated 10% of all waste (Barnes et al., 2009).

The research focus on quantifying human inhalation, ingestion, and dermal exposures, considering factors like exposure frequency, duration, plastic size, or intake rates. Different environments, spaces and diverse population groups are assessed to understand varying exposure levels. The primary concerns in this rapidly evolving field include the potential for physical damage, toxicity, and inflammatory responses. Risk assessment studies are essential to understand the implications of these particles on human health, but comprehensive risk assessments are still in the early stages. Given the emerging nature of the research, regulatory and policy responses are still being developed. There is increasing awareness among policymakers about the need to address plastic pollution and its potential impacts on human health.

This review discusses the biological impacts of nano- and microplastics on human health, factors affecting their toxicity, and potential mechanisms of action. Given the current limited understanding of nano- and microplastics' effects on human health, this article aims to discern if *in vivo* studies can shed light on the risks tied to human exposure. However, it's essential to note that the findings on human health impacts stem solely from retrospective studies. A deeper grasp of these materials' cellular and molecular interactions could offer insights into their potential human health risks.

This article reviews the effects of nano- and microplastics on human health. An exploratory strategy was used for the search. It was chosen not to follow the procedures used in Cochrane reviews because strict adherence to a combination of terms resulted in finding too many articles or leading to the exclusion of relevant articles. It has been observed that the search strategy for specific term combinations, although effective in studying human exposure, does not work well in studies involving any identified cell culture studies (Heddagaard and Møller, 2020). We searched PubMed for keywords such as: "nanoplastic", "microplastic", "human cell lines", "human digestive tract", "human intestine", "human respiratory system", "human lungs", "human skin", "human nervous system". Also, term combinations (such as two search terms) and multiple terms using logical operators "OR" and "AND" were used. We assessed 1230 titles and abstracts for inclusion in the review. The review included retrospective studies and case reports of people exposed to nanoplastics and microplastics. Some *in vitro* studies in human cell lines were also included in the review because understanding the cellular and molecular mechanisms of action of plastics may help extrapolate risks to humans. Along with the course of work, articles identified in the literature of previously found articles were also included.

2. Route of exposition and uptake of nano- and microplastics

Microplastics and nanoplastics can enter the human body through three primary routes: ingestion (via the digestive system), inhalation (via the lungs), and potentially direct skin contact.

Microplastics of varying colors and sizes, ranging from ≥800 nm to 5 mm, have been detected in numerous human samples, including the lungs, breast milk, liver, spleen, placenta, blood, sputum, colon, saliva, faces, urine, testis, and semen. Notably, microplastics were absent in the kidneys and lungs of stillborn (Kutralam-Muniasamy et al., 2023; Zhao et al., 2023).

2.1. Gastrointestinal tract

The gastrointestinal tract is believed to be the major entry point for nanoplastics into the human body. This ingestion primarily stems from consuming contaminated seafood, milk, beer, honey, sugar, salt, and bottled water (Kwon et al., 2020; Kutralam-Muniasamy et al., 2020;

Liebezeit and Liebezeit, 2013, 2014, 2015; Yang et al., 2015; Mason et al., 2018). Studies under controlled conditions reveal that animals can absorb and accumulate nanoplastics, with these plastics transferred to the following trophic level, increasing particle accumulation in the food chain and increasing human exposure (Cedervall et al., 2012).

A striking 81% of 159 global tap water samples contained microplastics, mainly fibres smaller than 5 mm (Kosuth et al., 2018). Other studies support these findings, noting the presence of microplastic particles (MPs) ranging from 5 to over 1350 μm in mineral water. Interestingly, the particle count was similar in both glass-bottled and polyethylene terephthalate (PET)-bottled water, with counts reaching up to 6292 particles per liter (Schymanski et al., 2018; Oβmann et al., 2018). Current research highlights the leaching of microplastics from plastic food packaging, especially at high temperatures, such as from teabags and plastic-based kettles and infant feeding bottles (Hernandez et al., 2019; Li et al., 2020). On average, global human consumption is estimated at 0.1–5 g of microplastics weekly, equivalent to the weight of a credit card (Senathirajah et al., 2021). Infants face greater exposure levels than adults (Zhang et al., 2021a). One particular study detected nine plastic types in human faeces, with PP and PET being predominant. All eight stool samples were tested positive for microplastics, with a median of 20 MPs (50–500 μm) found per 10 g of stool.(Schwabl et al., 2019) This result is in line with a study of 26 students, in which the fecal samples of 23 participants tested positive for MPs with a size of 20–800 μm; the summed mass of all MPs per participant ranged from 0.01 to 14.6 mg (Zhang et al., 2021b).

2.2. Lungs

The respiratory epithelium is a primary target for inhaled nano-sized particles - NPs, whose biological reactivity depends on their physicochemical properties. Airborne NPs and MPs sources include clothing, dried sludge, agricultural residues, and tire rubber dispersed due to mechanical wear during driving. The alveolar surface of the lungs spans approximately 150 m^2 and features a tissue barrier thinner than 1 μm. This barrier allows NPs to penetrate capillaries, facilitating systemic distribution (Lehner et al., 2019b). Autopsies on human lung tissue have evidenced the presence of MPs. Polymer particles and fibers were detected in 13 out of the 20 samples (Amato-Lourenço et al., 2021). To assess the airborne polystyrene (PS) absorption, a method was developed based on the concentration of styrene (StyU) and its metabolites, mandelic acid (MA) and phenylglyoxylic acid (PGA), in the urine. These main urinary metabolites in humans, MA and PGA, serve as biomarkers for styrene exposure. Increases in StyU and metabolites have been observed in workers exposed to large amounts of polystyrene inhalation. This level was influenced by work without respiratory protection (Persoons et al., 2018).

2.3. Skin

The skin serves as a protective barrier against various external particles, such as allergens, but also acts as a site for topical drug application due to its accessibility. Given its immunological significance, the skin could potentially be permeable to NPs and MPs. The prevalent use of these particles in cosmetics and textiles ensures considerable skin exposure (Hernandez et al., 2017). While data remains inconclusive, small particle size is probably crucial to skin penetration (Schneider et al., 2009). Only a few studies have reported low penetration of textile NPs through the skin barrier (Som et al., 2011). It is unlikely that NPs contained in polluted water can penetrate the stratum corneum. However, these particles might infiltrate the body via sweat glands, hair follicles, or skin injuries (Yee et al., 2021). Some experimental outcomes found that PS-NPs do not pass the stratum corneum or deeper layers of the porcine skin; instead, NPs could only penetrate the skin layer in the final stages of exfoliation (Campbell et al., 2012; Alvarez-Román et al., 2004). Other investigations using human skin samples visualised the accumulation of labelled PS-NPs in the stratum corneum and upper sections of hair follicles (Döge et al., 2018). However, there is a lack of more research using human skin. Another study illustrated that a significant number of epidermal Langerhans cells (cluster of differentiation [CD]-1a) located near the hair follicle on human skin absorbed NPs in varying sizes. After transdermal administration, it was shown that only

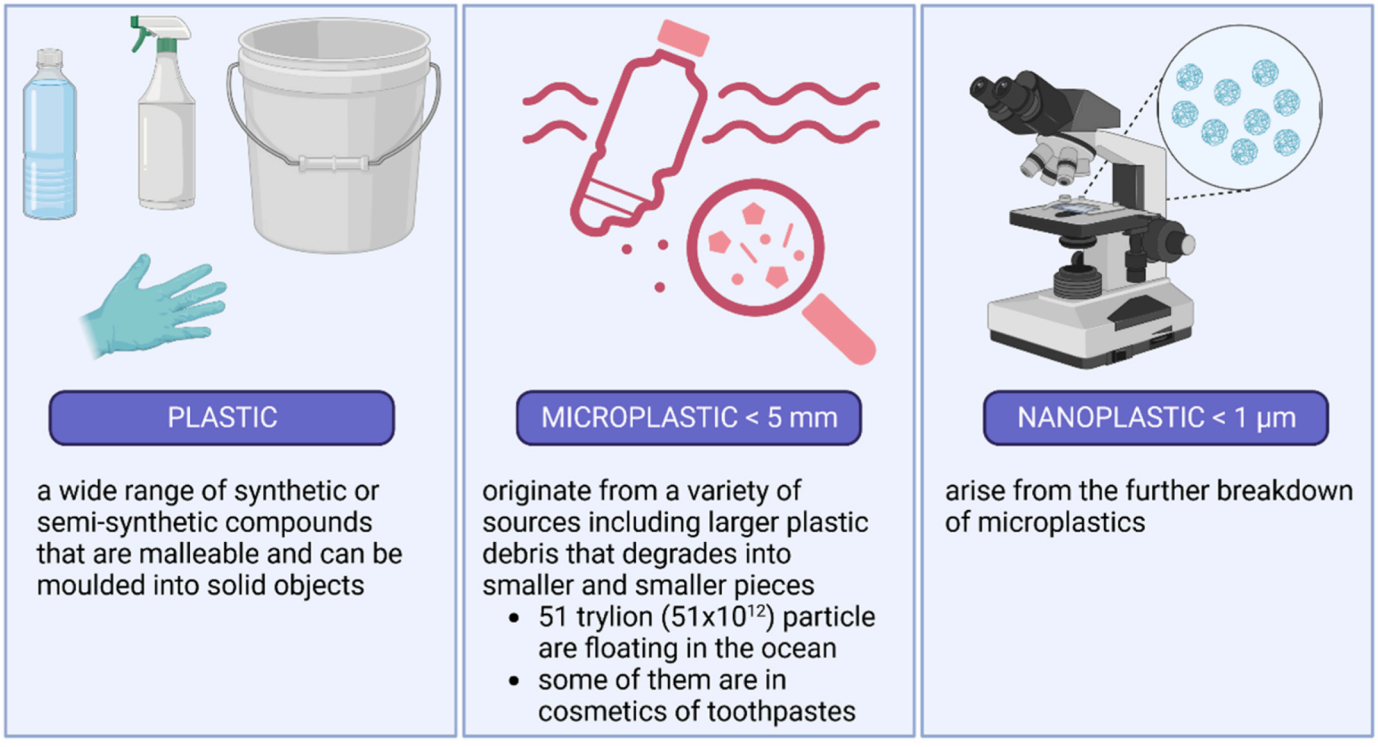


Fig. 1. Fragmentation and distribution of plastics due to size.

NPs with a diameter of 40 nm entered Langerhans cells. Visualisation techniques revealed the penetration of NPs with a diameter of 40 nm deep into the holes of the vellus hair and through the epithelium of the hair follicle. No NPs with 750 and 1500 nm diameter were detected in the Langerhans cells (Vogt et al., 2006).

3. Potential *in vivo* effects of plastic exposure

Certain plastics can pose health risks due to their inherent chemical composition or ability to absorb and transport other harmful substances. These potential dangers can be categorized into three main types: physical, chemical, and biological. The following section will delve into specific documented instances where plastic exposure resulted in diseases (Table 2). Moreover, it is currently unknown whether the diseases associated with plastic exposure are caused by nano- or microplastic particles, adding another layer of complexity to the assessment of these health risks. The exact mechanisms of how plastics react and whether nano- or microplastics can trigger reactions remain uncertain.

Table 2
Health impacts of nano- and microplastic exposure: organ-specific effects.

System affected/ health effect	Potential diseases/states	Notes
Skin	Allergic reactions, (Heilig et al., 2011; Rose et al., 2009; Yan et al., 2022) contact dermatitis58	Limited human studies; mostly allergic responses
Gastrointestinal tract	IBD,60 colorectal cancer,62 pancreatic cancer,63 gut barrier dysfunction, (Oddone et al., 2014; Chen et al., 2022; Auguet et al., 2022; Molina and Benedé, 2022; Cohen et al., 2002) metabolic disorders,65 non-alcoholic fatty liver disease66	Positive correlation between fecal MPs and IBD severity; lack of human studies on microbiota effects
Respiratory system	Lung cancer, (Ruder and Bertke, 2017; Coggon et al., 2015; Kogevinas et al., 1994; Sathiakumar et al., 2009; Collins et al., 2013; Sartorelli et al., 2020) occupational hypersensitivity pneumonitis, (Volkman et al., 2006; Nett et al., 2017; Cullinan et al., 2013) obstructive bronchiolitis, (Atis et al., 2005; Bertke et al., 2018) respiratory issues (coughing, dyspnea, wheezing), (Song et al., 2009; Zhou et al., 2017) pleural effusion81	Studies mainly in occupational settings; some diseases linked to specific plastics like styrene and polyacrylic NPs
Blood neoplasms	Leukemia, bladder cancer, lymphohematopoietic neoplasms (Daniels and Bertke, 2020; Christensen et al., 2018; Werder et al., 2018)	Associations found in occupational settings with long-term exposure
Neurological effects	Neurotoxic effects (headache, fatigue, dizziness, etc.), (Kolstad et al., 1995b) encephalopathy, unspecified dementia, degenerative neurological disorders (Shanbhag et al., 1994)	Correlation with environmental styrene exposure; more research needed for conclusive evidence
Osteolysis around bone prosthesis	Periprosthetic osteolysis (Orishimo et al., 2003; Gajski et al., 2014; Bitar and Parvizi, 2015; Yu et al., 2021)	Linked to PE particles in hip prostheses
Reproductive	Decreased fertility, (Chen et al., 2022) gonadal damage, (Sarasamma et al., 2020; Qiang and Cheng, 2021; Sun et al., 2021b; González-Acedo et al., 2021b) reduced offspring weight (Sun et al., 2021a; An et al., 2021)	Animal studies show effects; human impact has not yet been studied
Genotoxicity	DNA damage, increased sister chromatid exchange frequency (Brachner et al., 2020)	Observed in workers exposed to styrene; potential link to genotype

Abbreviations: **IBD** = inflammatory bowel disease; **MPs** = microplastics; **NPs** = nanoplastics, **PE** = polyethylene.

3.1. Skin

The literature contains limited publications concerning the effects of nano- and microplastics on human skin, with most studies predominantly focusing on potential allergic reactions. An early account of occupational allergy linked to plastic gloves dates back to 1985 (Estlander et al., 1986). In this study, allergic reactions were relatively rare, manifesting in only 5 out of the 542 patients examined. Another documented case involves a six-year-old female who developed persistent allergic contact dermatitis attributed to plastics present in both a toilet seat and a school chair (Heilig et al., 2011). While the plastics themselves can induce allergic responses, additives used in manufacturing plastic items, such as gloves, are also potential allergens (Rose et al., 2009). These additives can complicate the identification of exact allergenic substances. A notable example is bisphenol A (BPA). From 1998 onward, multiple instances of contact allergies to bisphenol A in individuals wearing polyvinyl chloride (PVC) gloves have been reported (Aalto-Korte et al., 2003).

3.2. Gastrointestinal tract

There is a positive correlation between the concentration of fecal MPs and the severity of inflammatory bowel disease (IBD). Also, patients with IBD have a higher concentration of MPs in their stool (41.8 pcs/stool g) compared to healthy individuals (28.0 pcs/g) (Yan et al., 2022). Research on IBD patients revealed a significant accumulation of microplastics in ulcerative lesions of the rectal mucosa. However, nanoplastics were only seen in trace amounts on mucosal surfaces, offering hope for developing precise drug delivery systems targeting the inflamed intestinal mucosa (Schmidt et al., 2013).

Of significant interest is the potential impact of nano- and microplastics on carcinogenesis. Given reports about environmental factors influencing the onset of colorectal cancer, scientific literature and meta-analyses were reviewed. The findings suggest an elevated risk of colorectal cancer among individuals working in various sectors, including the plastic and rubber industries (Oddone et al., 2014). There's also a noted increase in pancreatic cancer mortality among workers involved in producing reinforced plastics (Kolstad et al., 1995a).

Another research avenue is the effect of NPs and MPs on the microbiota. There's a lack of human studies in the current literature. However, *in vivo* experiments on mice showed disturbances in microbiota composition due to the presence of PVC-MPs, which were linked to weight loss and the accumulation of MPs and chemical additives in tissues (González-Acedo et al., 2021a). Exposure to PVC in adult mice led to significant gut barrier dysfunction, microbiota imbalance, and metabolic disorders (Chen et al., 2022). Also, mice exposed to MPs develop inflammation, endocrine disruptions, and lipid and energy metabolism alterations, participating in the pathogenesis of non-alcoholic fatty liver disease (Auguet et al., 2022). It is postulated that alterations in mammalian gut microflora after microplastics exposure can cause gut barrier breach and be associated with kidney disease, liver and neurological disruptions (Yong et al., 2020).

Plastics might have an impact on unexplored health conditions, like food allergies. Plastics have the potential to alter how food allergens are digested, heighten the permeability of the intestines, induce inflammation within the gut, or lead to intestinal imbalances (changes in the microbiome), all of which could enhance sensitivity to food allergens (Molina and Benedé, 2022).

3.3. Respiratory system disorders

Research into the potential toxicity of styrene began in the late 20th century. In 1999, the Harvard Centre for Risk Analysis, commissioned by the Styrene Information and Research Centre (SIRC), investigated styrene's effects on human health. Occupational exposure to styrene varied but generally remained below 10 parts per million (ppm), with higher concentrations of over 20 ppm found in industries dealing with fiber-reinforced polymer composites. Studies in rodents confirmed carcinogenic effects, nervous system disorders, and respiratory toxicity. However, these studies did not conclusively link styrene to human health issues (Cohen et al., 2002). Notably, female mice exposed to 160 ppm of styrene in animal trials showed a significant increase in lung cancer compared to controls (Cruzan et al., 2001). In a retrospective analysis of 3704 boat-building workers exposed to styrene between 1959 and 1993 for over a year, 516 cases of invasive cancer were recognized. In contrast to the general population's statistics, a marginally elevated number of trachea, bronchus, and lung cancers were reported. The absence of data on lifestyle aspects, such as smoking or other employment, posed a significant limitation for this cohort. This is particularly important for lung cancer, given the slight rise observed compared to the reference population. The research's authors underscored that the subjects were relatively young (with a median age of forty-four in 1991), while the average age of cancer patients in the US is sixty-five years (Ruder and Bertke, 2017). Comparable findings were recorded from studies on a British cohort exposed to styrene (Coggon et al., 2015). Even though several other studies did not identify any association between lung cancer and exposure to styrene (Sathiakumar et al., 2009; Collins et al., 2013; Kogevinas et al., 1994).

Occupational inhalation of antigens can lead to an interstitial lung condition termed occupational hypersensitivity pneumonitis (OHP). In 2020, a clinical case report was published detailing hypersensitivity pneumonitis (HP) in a sixty-six-year-old individual who had been employed in the PET production industry from 1992 to 2013. This patient was under observation due to previous asbestos exposure. In 2012, she was diagnosed with asbestosis. By 2017, high-resolution computed tomography revealed a mild progression of pulmonary fibrosis. However, inspiratory clicks were detected upon auscultation, and the pulmonary function tests indicated a diminished diffusion capacity for carbon monoxide. Consequently, OHP was identified and believed to have been triggered by terephthalic acid and dimethyl terephthalate (DMP), a precursor in PET production. According to the authors, this is the first case of OHP related to employment in PET production (Sartorelli et al., 2020). Another case is an OHP likely caused by the effects of DMP and styrene. A forty-six-year-old employee of a yacht manufacturer reported shortness of breath, chest tightness, and coughing both day and night that had been progressing for two months. A clear correlation was observed between the manifestation of these symptoms and the individual's work environment. Treatment comprising systemic antibiotics, inhaled bronchodilators, and inhaled corticosteroids resulted in a slight symptom relief. The chest X-ray revealed a widespread interstitial pattern, while a spirometry examination revealed restriction loss. Only administering oral corticosteroids and avoidance from the work setting led to symptom resolution and bettered spirometry parameters (Volkman et al., 2006). In 2017, a review of available literature (comprising fifty-five articles and two unpublished case reports) evaluated the association between styrene exposure and the onset of non-malignant lung disorders. From the cases discussed, bronchial asthma was diagnosed in eight patients, while severe obstructive bronchiolitis was detected in ten individuals. In 75% of asthma cases, positive challenge tests were reported following inhalation exposure to styrene(Nett et al., 2017).

A series of cases of obstructive bronchiolitis have been documented in workers at companies producing glass-fiber-reinforced plastics. All six patients diagnosed with obstructive bronchiolitis were involved in producing and processing glass fibers using styrene resins. The disease progressed rapidly. Two patients underwent lung transplants, and a third died while awaiting a transplant. In four of the patients, obstructive bronchiolitis was confirmed through a biopsy. The appearance of the rare disease, bronchiolitis, in six individuals exposed to fiberglass with styrene resins across different workplaces may suggest a connection between the exposure and the disease's onset. However, the exact causative agent remains unidentified (Cullinan et al., 2013).

A study of Turkish plant workers found a 3.6-fold increased risk of respiratory issues in employees exposed to PP compared to the control group. The reported respiratory symptoms included coughing, dyspnea, and wheezing. Furthermore, deterioration was noted in pulmonary function tests, including the diffusion lung capacity for carbon monoxide, elevated serum levels of interleukin (IL)-8 and tumor necrosis factor-alpha (TNF-α), peribronchial thickening, and diffuse frosted glass suppression (Atis et al., 2005).

There are reports of dyspnea and pleural effusion necessitating hospitalization in a group of young women after inhaling polyacrylic NPs. Extensive diagnostic procedures were carried out, including video-assisted thoracic surgery. Histopathological examination of the lung tissues revealed non-specific inflammatory responses, fibrosis, pleural granulomas, and the presence of NPs within the cytoplasm of the lung epithelium and mesothelial cells (Song et al., 2009).

Another study conducted a comparative evaluation of the respiratory health of workers in a flocking plant over a decade. Nine cases of nylon flock (very short fibres of nylon that are cut or ground to a small, consistent length)-associated interstitial lung disease were detected. In five of these cases, symptoms subsided after a change in the workplace environment. A correlation was found between the severity and progression of nylon flock-associated interstitial lung disease and a low baseline of diffusion lung capacity for carbon monoxide (Turcotte et al., 2013).

The prenatal exposure to BPA is associated with a higher risk of allergic diseases in female infants (Zhou et al., 2017). There's also a correlation with low birth weight (Huo et al., 2015). Additionally, there's an increased risk of asthma symptoms, wheezing, and respiratory tract infections post-delivery (Spanier et al., 2012, 2014; Xie et al., 2016; Wu et al., 2021; Gascon et al., 2015). There has been a documented case of bronchial asthma following exposure to acrylonitrile butadiene styrene fibres, a thermoplastic commonly used in three-dimensional (3D) printing (House et al., 2017).

Numerous reports highlight the development of respiratory diseases following exposure to plastics, especially among individuals with significant exposure. A comprehensive study examining the potential health impacts of styrene exposure established reference inhalation toxicity levels at 20 ppm for workers and 3.7 ppm for the general population. There might be a need for legal regulations to safeguard workers from inhaling plastics.

3.4. Blood neoplasms

A cohort study examined cancer mortality among 5201 workers at two boat-building facilities. While there was no rise in mortality from hematopoietic neoplasms compared to the general population, a significant association was found between the duration and intensity of styrene exposure and the occurrence of neoplasms (Bertke et al., 2018).

In 2020, a re-analysis was conducted on the relationship between styrene exposure and cancer mortality in a previously studied cohort of American boatbuilders. This cohort had occupational exposure to styrene from 1959 to 1978. Estimations of styrene exposure were based on workplace environments and air sampling data. The Cox proportional risk models were utilized to evaluate the relationship, considering factors such as gender, race, age, socioeconomic status, and the period of employment (exposure). A statistically significant association was found between styrene exposure and the onset of leukemia and bladder cancer. Working-life exposure to 0.05 ppm of styrene was linked to an additional death from blood cancer per 10,000 workers (Daniels and Bertke, 2020). The authors emphasized the need for more research to elucidate the

cancer risks associated with styrene exposure and call for guidelines to safeguard workers.

In Denmark, retrospective study with large sample sizes have been conducted. The study analyzed data from 73,036 employees across 456 small to medium-sized enterprises engaged in reinforced plastics production from 1968 to 2011. The study aimed to assess the link between styrene exposure and the incidence of lymphohematopoietic neoplasms. Estimates of styrene exposure were derived from employment records, survey data, and historical measurements of styrene exposure. Information on blood cancers was sourced from national cancer and patient registries. Among the study participants, a total of 665 cases of 21 different blood cancers (each with a minimum of 20 cases) were identified. When adjusting for exposure duration, the risk of acute myeloid leukemia was found to be twice as high for those with significant exposure in the previous 15–29 years. However, no elevated risks were observed for Hodgkin's lymphoma or T-cell lymphoma (Christensen et al., 2018).

3.5. Neurological effects

In 2011, a study was conducted to evaluate the potential neurological effects of styrene exposure among 21,962 residents of the United States (US) Gulf Coast. This region is believed to account for over half of the US's total styrene production. The study determined levels of styrene exposure by analyzing environmental styrene concentrations and measuring the styrene accumulation in the blood of 874 participants. Data on neurological symptoms, including headache, fatigue, dizziness, numbness, visual disturbances, and nausea, were collected. One-third of the participants reported experiencing at least one such symptom. There was a significant correlation between experiencing one or more neurological symptoms and being in the top quartile for estimated environmental styrene exposure. While findings from biomarkers were less definitive, elevated blood styrene levels were linked to nausea. Notably, non-white participants exhibited the most severe neurological symptoms. This evidence suggests that exposure to environmental styrene might lead to neurotoxic effects (Werder et al., 2018).

Retrospective studies utilizing Danish national registries investigated the risk of encephalopathy and unspecified dementia in individuals with documented styrene exposure. Of the 72,465 workers in the reinforced plastics sector from 1977 to 2011, 228 cases of encephalopathy and 565 cases of unspecified dementia were identified. Specific models were employed to estimate individual styrene exposure levels based on occupation, workplace styrene concentration, and years of employment. Although there was a noted increase in the risk of encephalopathy in recent years and a declining risk of unspecified dementia, no direct link between these two conditions and prior styrene exposure was established. Additionally, there was a reported uptick in mortality from degenerative neurological disorders, such as multiple sclerosis, parkinsonism, and motor neuron disease, among workers in the reinforced plastics industry (Kolstad et al., 1995b).

While individual studies have suggested a connection between exposure to nano- and microplastics and the onset of neurological symptoms, conclusive evidence remains elusive. More research is necessary to uncover the potential mechanisms that plastics might impact the nervous system.

3.6. Osteolysis around bone prosthesis

Periprosthetic osteolysis is a significant challenge in arthroplasty and is frequently observed as a complication following cementless total hip replacement. Particles phagocyted by macrophages from joint surfaces lead to the release of proinflammatory cytokines, accelerating osteoclast formation and subsequent bone resorption. Most hip prostheses currently used consist of a metal or PE-ceramic core. Evidence suggests that PE particles can contribute to hip osteolysis and that the quantity of PE particles shed from implant surfaces correlates with the severity of osteolysis. Analysis of interphase membranes from eleven unsuccessful total hip arthroplasties revealed a significant presence of PE particles (with an average diameter of 530 nm) alongside titanium particles, bone fragments, stainless steel, and silicate (Shanbhag et al., 1994). In another study involving eleven patients who underwent hip prosthesis revisions, tissues adjacent to periprosthetic osteolysis exhibited elevated expression of receptor activator of nuclear factor-kappa β (RANK), RANKL (RANK-Ligand), and TNF-α levels in cells containing PE debris. An increase in bone loss volume was also noted (Holding et al., 2006). An examination of fifty-six hip replacement patients suggested an implant wear rate of 0.2 mm/year is a "critical threshold" for osteolysis onset (Orishimo et al., 2003). Conversely, an *in vitro* study evaluating medical implants found no cytotoxic or genotoxic effects from PE-NPs smaller than 10 μm at the tested 10 μg/mL concentration (Gajski et al., 2014).

With an anticipated increase in life expectancy, the number of arthroplasty procedures is expected to rise, as is the risk of periprosthetic osteolysis due to elevated PE abrasion (Bitar and Parvizi, 2015). These might indicate the need to substitute PE in endoprosthetics with alternative materials, such as ceramics, particularly for individuals predicted to have a long lifespan.

3.7. Reproductive

Currently, there are no studies describing the impact of plastic MPs and NPs on human fertility and reproduction. However, some animal studies have been conducted. For instance, when the nematode *Caenorhabditis elegans* was exposed to NPs, a decrease in brood size (offspring) was observed across subsequent generations (Yu et al., 2021). In another study using *C. elegans*, both unmodified NPs and NPs-NH_2 (amine group) were tested, revealing that NPs-NH_2 had a more toxic effect on reproductive performance and gonad development in subsequent generations compared to unmodified NPs (Sun et al., 2021a). Additionally, polystyrene (PS) exposure has been reported to cause gonadal damage in zebrafish (Qiang and Cheng, 2021; Sarasamma et al., 2020). Moreover, zebrafish embryos developed severe pericardial edema after exposure to NPs. Above the no observed adverse effect level (NOAEL), nanoplastics significantly reduced cardiac output and blood flow velocity (Sun et al., 2021b).

Nanoplastics have been detected in mammalian offspring, indicating that they might cross the placental barrier and accumulate in fetal tissues. In a study involving pregnant Sprague Dawley rats intratracheally administered with PS-NPs (20 nm) were found in the mother's lungs, heart, and spleen, as well as in the fetus's organs, including the liver, lungs, heart, kidneys, and brain (Fournier et al., 2020). This exposure resulted in a significant reduction in the weight of both the fetus and the placenta. In female rats, microplastics were observed to induce apoptosis and fibrosis in ovarian granulosa cells (An et al., 2021). In male rats, exposure to microplastics was associated with decreased sperm viability (González-Acedo et al., 2021b).

The presence of plastic NPs and MPs has been confirmed in humans. In a study examining six placentas, twelve microplastic fragments, ranging from 5 to 10 μm in size, were found in four of them, with five located on the foetal side, four on the maternal side, and three on the chorioamniotic membranes (Ragusa et al., 2021). An *ex vivo* human placental perfusion model assessed whether PS-NPs of sizes 50, 80, 240, and 500 nm could cross the placental barrier and whether this process was influenced by particle size. The results showed that PS-NPs up to 240 nm in diameter could cross the placental barrier without affecting cell viability (Wick et al., 2010). Additionally, MPs larger than 50 μm were found in human placentas and meconium collected during two caesarean sections. In these samples, PP, PS, PE, and polyurethane were identified. However, polyurethane was also found in the air of the operating room, indicating potential sample contamination (Braun et al., 2021).

3.8. Genotoxicity

The highest exposure to styrene occurs in the manufacturing of reinforced plastic products, where it is primarily metabolized in the liver. A study was conducted to assess the potential genotoxicity of occupational exposure to styrene. It evaluated styrene levels in workplace air, the concentration of its metabolites (MA and PGA) in urine, sister chromatid exchange (SCE), micronucleus formation, DNA damage, and genetic polymorphisms of metabolizing enzymes: cytochrome P450 2E1 (*CYP2E1*), epoxide hydrolase 1 (*EPHX1*), glutathione S-transferase Mu 1, theta 1 and Pi 1 (*GSTM1, GSTT1* and *GSTP1*). Fifty-two employees from five companies producing reinforced plastic items participated, all using the same production methods and materials, with no smoking history or other known toxic substance exposure. The control group comprised fifty-four workers not exposed to styrene, primarily performing office work. The results indicated that the average air concentration of styrene exceeded the time-weighted average limit value of 20 ppm and the biological exposure index for styrene (MA + PGA = 400 mg/g creatinine) set by the American Conference of Governmental Industrial Hygienists (ACGIH). The study group exhibited a significant increase in SCE frequency and DNA damage. The observation of increased microsomal epoxide hydrolase activity in styrene-exposed workers, correlating with a higher incidence of SCE, suggests a potential link between exposure and genotype (Teixeira et al., 2010).

4. Effects of cell exposure to plastics *in vitro*

Research on human cells has revealed that nano- and microplastics can trigger various cellular responses, including cytotoxicity, genotoxicity, inflammation, apoptosis, and oxidative stress (Fig. 2). An overview of *in vitro* studies is presented in Table 3. Among the plastic polymers studied, PS is most frequently used in research. Interestingly, PS does not rank within the top five polymers produced globally. There is a gap in data regarding other plastic types, particularly PET and PVC. The plastic particle sizes range from nano-through micro- and even millimeters, and some have different surface modifications, which are now seen as crucial for the cytotoxic and inflammation effects. Determining the duration of exposure is also challenging; it is unclear if the observed effects are acute or chronic. There's a lack of data representing long-term exposure, even though plastic bioaccumulation in the human body persists for a lifetime. Another problem is distinguishing and extracting plastic particles from environmental and biological samples. This impedes accurate dosage determination in studies (Brachner et al., 2020). Furthermore, there's no data exploring the cellular effects of extremely low plastic concentrations.

Upon entering the body, NPs interact with many biomolecules, such as proteins, lipids, and sugars. When these particles come into contact with biological fluids like serum or plasma, it is hypothesized that NPs change their original properties and acquire new biological properties. This is primarily due to the formation of a protein corona, a layer of adsorbed proteins on the NP surface (Park, 2020). The composition of this protein corona can vary based on the unique plasma proteomes associated with different physiological states (stress, diet, smoking) or diseases (diabetes, cancer) (Ahsan et al., 2018). This means that identical NPs can exhibit distinct protein corona compositions under different conditions. There are currently no personalized *in vitro* studies assessing cellular exposure to plastics. As a result, the impact of protein corona development on potential toxic effects remains uncertain.

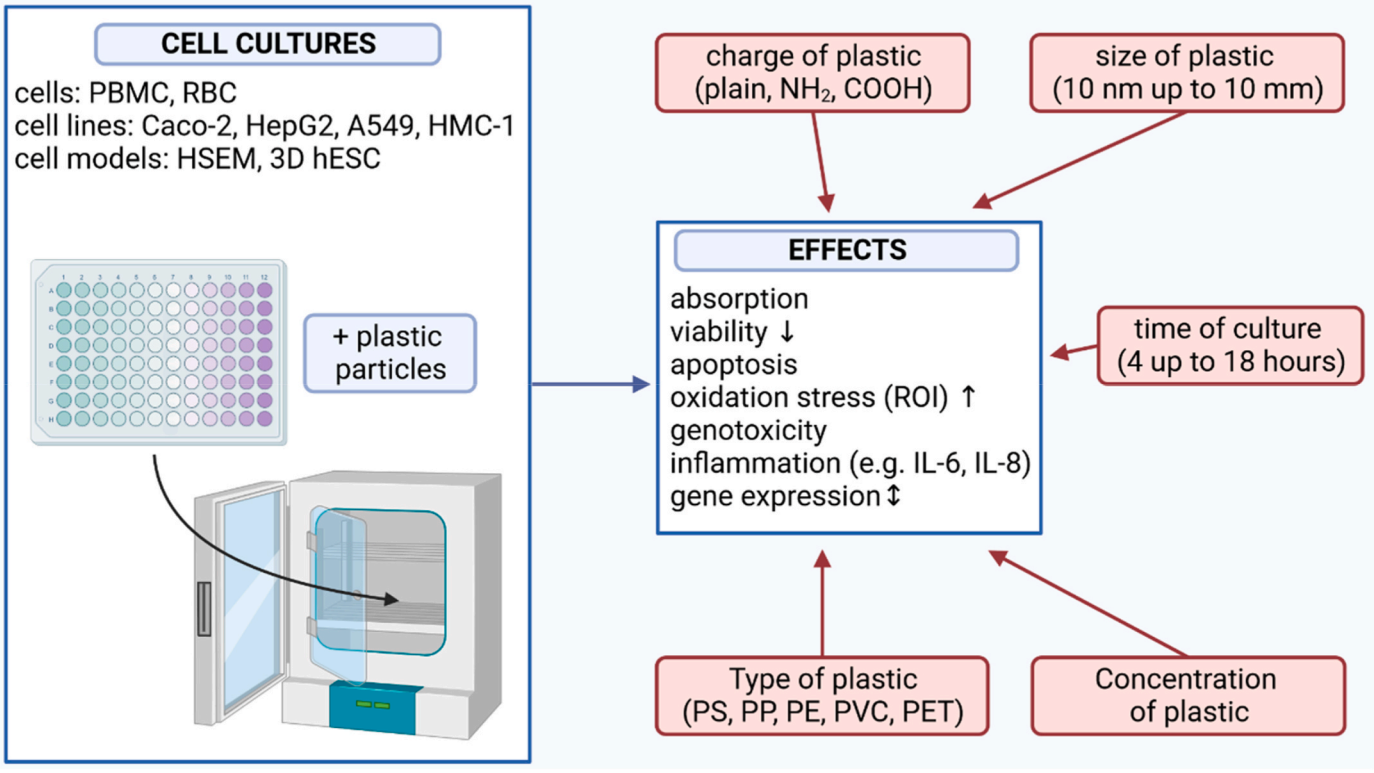


Fig. 2. Assessment of the nano- and microplastics' influence on human cells *in vitro* studies. Nano- and microplastics can enter the human body through various routes such as inhalation, dermal contact, or ingestion.
Abbreviations: **3D hESC** = three-dimensional human embryonic stem cells; **A549** = human alveolar type 2 epithelial cell line; **Caco-2** = human colon epithelial cell line; **COOH** = carboxyl group; **HepG2** = human hepatocellular carcinoma cell line; **HMC-1** = human mast cell line; **HSEM** = human skin equivalent model; **MP(s)** = microplastic(s); **NH_2** = amino group; **NP(s)** = nanoplastic(s); **PBMC** = peripheral blood mononuclear cell; **PE** = polyethylene; **PET** = polyethylene terephthalate; **PP** = polypropylene; **PS** = polystyrene; **PVC** = polyvinyl chloride; **RBC** = red blood cell.

Table 3
Impact of nano- and microparticles, their properties and effects on cells in *in vitro* studies.

Properties of MPs/NPs	Cell type	Effects
40 nm NPs 750 nm NPs 1500 nm NPs	Human skin samples	Only 40 nm NPs enter Langerhans cells, into the holes of the vellus hair and through the epithelium of the hair follicle5 (Napper and Thompson, 2020)
50 nm PS-NPs-NH_2	HSEM HaCaT	Significant phototoxicity, sensitisation, or skin irritation (Park et al., 2011)
100 nm PS-NPs	Hs27	Stimulation of ROS production and the induction of genotoxic stress (including DNA destruction) (Poma et al., 2019)
60 nm PS-NPs 60 nm PS-NPs-NH_2 60 nm PS-NPs-COOH	Caco-2 HT29-MTX-E12 LS174T	Only PS-NPs-NH_2 show the ability to strongly interact and aggregate mucin and induce apoptosis in all cell lines (Inkielewicz-Stepniak et al., 2018)
50–100 nm PS-NPs	Caco-2	The cytotoxic effect at higher concentrations (more than 150 μg/mL), concentration-dependent uptake of PS-NPs (Cortés et al., 2020)
1 μm PS-MPs 4 μm PS-MPs 10 μm PS-MPs	Caco-2, thereof-derived co-cultures mimicking intestinal M cells and goblet cells	Slight uptake of PS-NPs by cultured cells, but no inflammatory response; without activation or interference with Mφ (Stock et al., 2019)
10 nm PS-NPs 5 μm PS-MPs	Caco-2	Disruption of the mitochondrial membrane potential and inhibition of the ATP binding cassette (ABC) transporter activity in the plasma membrane without affecting the viability and integrity of the cell membrane (Wu et al., 2019)
50 nm PS-NPs-COOH 500 nm PS-NPs-COOH	Caco-2 HT29-MTX-E12	PS-NPs 50 nm: no effect on cell viability up to 50 μg/mL, at high doses (10 μg/mL), a significant increase in metabolic activity; PS-NPs 500 nm: low concentration reduces the metabolic activity of the cells (0.01 μg/ml) (Hesler et al., 2019)
50 nm PS-NPs	Caco-2 HT29-MTX-E1 Raji-B	Penetration of PS-NPs through the epithelial barrier of the gastrointestinal tract without causing dangerous effects (Domenech et al., 2020)
44 nm PS-NPs 100 nm PS-NPs	AGS	Increase in the level of IL-6 and IL-8; 44 nm PS-NPs accumulate in the AGS cytoplasm faster than 100 nm PS-NPs (Forte et al., 2016)
58 nm PS-NPs-NH_2	Huh7	Cell death pathway dependent on lysosomal membrane permeabilization only at low dose of NP (25 μg/mL) (Murschhauser et al., 2019)
50 nm PS-NPs 50 nm PS-NPs-NH_2 50 nm PS-NPs-COOH	HepG2	PS-NPs- NH_2, and PS-NPs-COOH internalize faster and induce inhibition of cell viability and oxidative stress, compared to unmodified particles (He et al., 2020)
50 nm PS-NPs	Triple culture model: Caco-2, HT29-MTX-E12 and THP-1	No acute toxicological effects, including cytotoxicity, cell layer integrity or DNA damage; release of pro-inflammatory cytokines (IL-1β, IL-6, IL-8, and TNF-α)(Busch et al., 2021a)
<50 μm PVC	Triple culture model: Caco-2, HT29-MTX-E12 and THP-1	No changes in cell viability, cytotoxicity, or mucus distribution; 50 μg/cm^2 PVC particles induce a significant increase in IL-1β(Busch et al., 2021b)
300 nm-10 μm PE-MPs	NHBC differentiated into the mature osteoblast phenotype	Increase expression of the mRNA of the osteocyte E11 markers, DMP-1 of matrix dentin and SOST, increase expression of genes related to osteoclast activity (RANKL, CXCL8 and M-CSF), decrease expression of osteoclast antagonists(Atkins et al., 2009)
10 μm PS-MPs 3–16 μm PE-MPs	T98G	PS-MPs increase ROS production at the highest concentration; PE-MPs do not affect ROS production (Schirinzi et al., 2017)
33 nm PE-NPs	3D *in vitro* model derived from hESC	PE-NPs penetrate the 3D structure and decrease the expression of genes (including the NOTCH pathway, neuron precursor markers (NEUROD1 and ASCL1), the neuron calibration gene, FOXG1)(Hoelting et al., 2013)
20 nm PS-NPs 500 nm PS-NPs 1 μm PS-NPs	Human Mo obtained from peripheral blood	20 nm PS-NPs are passively absorbed, with a high cytotoxicity effect; 500 nm and 1 μm PS-NP stimulate the pro-inflammatory cytokines (IL-6 and IL-8) secretion, possible respiratory bursts (Prietl et al., 2014)
6.5–8.5 μm PE-NPs	U937 cells to study the differentiation of Mo	Increase in the expression of IL-8, MIP-1β and CXCR4 genes (Matsusaki et al., 2007)
20 μm PP-NPs	PBMCs	Cytotoxicity (related to ROS release), induction of cytokine (IL-6 and TNF-α) and histamine release (Hwang et al., 2019)
29, 44 and 72 nm PS-NPs	PBMCs	Diameter-dependent toxicity and oxidation properties(Kik et al., 2021)
~120 nm PS-NPs-NH_2 ~120 nm PS-NPs-COOH	Human Mφ	PS-NPs-NH_2 and PS-NPs-COOH reduce the expression of scavenger receptors CD163 and CD200R and increase the release of IL-10 in M2 Mφ; PS-NH_2-NPs inhibit the M1 and M2 Mφ phagocytosis(Fuchs et al., 2016)
50 nm PS-NPs	Raji-B TK6 THP-1	Highest internalization in THP-1 cells; mild toxicity, ROS production and genotoxicity in Raji-B and TK6 cells(Rubio et al., 2020)
50, 108 and 243 nm PS-NPs	RBC HBMEC	The formation of aggregates and increased adhesion to cells. The smaller size of NP and the higher concentration exacerbate the above effects(Barshtein et al., 2016)
64, 202 and 535 nm PS-NPs	A549	Significant increase in calcium ion concentration, moreover, oxidative capacity and increased production of IL-8 induced only by PS-NP with a diameter of 64(Brown et al., 2001)
25 nm PS-NPs 70 nm PS-NPs	A549	Significant inhibition of the S-phase of the cell cycle, activation of the transcription of inflammatory genes and changes in the expression of proteins

(*continued on next page*)

Table 3 (*continued*)

Properties of MPs/NPs	Cell type	Effects
		associated with the cell cycle and pro-apoptosis (Xu et al., 2019)
100 nm PS-NPs in co-culture with various PAE	A549	Oxidative stress (ROS production) and inflammatory responses (release of IL-1α, IL-6, IL-8, TNF-α) (Shi et al., 2021)
Bap-NPs	A549	Persistent binding of crown mucin to NP-Bap increases particle uptake but decreases cytotoxicity(Ji et al., 2021)
50 and 100 nm PS-NPs PS-NPs-NH_2 PS-NPs-COOH	MAC AT2 TT1	PS-NPs-NH_2 cause cell death, ROS production, mitochondrial disruption and release of cytochrome C; PS-NPs and PS-NPs-COOH cause a significantly lower rate of cytotoxicity, (Ruenraroengsak and Tetley, 2015; Ruenraroengsak et al., 2012)
50 nm PS-NPs	BEAS-2B	An increase in the number of amino acids and intermediate metabolites of the tricarboxylic acid cycle, which indicates metabolic changes related to stress in the autophagous and endoplasmic reticulum (Lim et al., 2019)
60 nm PS-NPs 60 nm PS-NPs-COOH	BEAS-2B	Much greater cytotoxic effect and increased uptake in Mφ of positively charged PS-NH_2 than negatively charged particles (Xia et al., 2008; Chiu et al., 2015)
4.8–5.8 μm PS-MPs	HRT-18	Increased in cytotoxicity and in oxidative stress, release of IL-8(Mattioda et al., 2023)

Abbreviations: **μg** = microgram; **μm** = micrometer; **3D** = three dimensional; **A549** = human alveolar type 2 epithelial cell line; **AGS** = gastric adenocarcinoma cell line; **ASCL1** = achaete-scute homolog 1 protein; **AT2** = primary human alveolar type 2 epithelial cells; **ATP** = Adenosine triphosphate; **BEAS-2B** = bronchial epithelial cells, **Caco-2** = human colon epithelial cell line; **CD** = cluster of differentiation; **COOH** = carboxyl group; **CXCL8** = IL-8 or chemokine (C-X-C motif) ligand 8; **CXCR4** = C-X-C chemokine receptor type 4; **DMP-1** = acid phosphoprotein; **DNA** = deoxyribonucleic acid; **FOXG1** = forkhead box protein G1; **HaCaT** = human immortalized human keratinocyte cell line; **HBMEC** = human bone marrow endothelial cells; **HepG2** = human hepatocellular carcinoma cell line; **hESC** = human embryonic stem cells; **Hs27** = human fibroblast cell line; **HRT-18** = Human colorectal epithelial cell line; **HSEM** = human skin equivalent model; **HT29-MTX-E12** = human adenocarcinoma colonic epithelial cells; **Huh7** = human hepatocarcinoma cell line; **IL** = interleukin; **LS174T** = human Caucasian colon adenocarcinoma; **M1/M2** = type 1/2 macrophages; **MAC** = primary human alveolar macrophages; **M-CSF** = macrophage colony-stimulating factor; **MIP-1β** = macrophage inflammatory proteins 1 beta; **mL** = millilitre; **Mo** = monocyte, **MPs** = microplastics; **mRNA** = messenger ribonucleic acid; **Mφ** = macrophages, **NEUROD1** = neurogenic differentiation 1, transcription factor; **NH_2** = amine group; **NHBC** = normal human bone-derived cells; **nm** = nanometer; **NOTCH** = family of type-1 transmembrane proteins that form a core component of the Notch signaling pathway; **NP-Bap** = polystyrene-benzopyrene; **NPs** = nanoplastics; **PAE** = phthalate esters; **PBMC** = human peripheral blood mononuclear cells; **PCV** = polyvinyl chloride; **PE** = polyethylene; **PS** = polystyrene; **Raji-B** = human B lymphoblast-like cell line; **RANKL** = receptor activator of nuclear factor kappa-B ligand; **RBC** = red blood cells; **ROS** = reactive oxygen species; **SOST** = sclerostin; **T98G** = human brain glioblastoma cells;**THP-1** = leukemic monocyte cell line; **TK6** = human lymphoblast cells; **TNF** = tumor necrosis factor; **TT1** = human alveolar epithelial type I-like cells; **U937** = histiocytic lymphoma cells.

5. Concluding remarks

Plastic pollution is a pressing global issue. Current literature does not conclusively highlight the detrimental effects of plastic on human health, nor does it confirm its safety. Research on human cells has demonstrated that nano- and microplastics can lead to cytotoxicity, genotoxicity, inflammation, apoptosis, and oxidative stress, among other concerns. However, *in vitro* studies have certain limitations, such as the duration of nanoplastic exposure and the accurate determination of plastic particle sizes and their concentrations. *In vivo* studies, which primarily focus on individuals who work in close contact with plastics, also highlight the potential effects of nano- and microplastics on various human systems like the skin, digestive, respiratory, nervous, and reproductive systems. They also indicate possible links to carcinogenesis and genotoxicity. It's important to note that all existing *in vivo* research consists of retrospective studies or individual case reports. Given that conducting other forms of research might pose ethical challenges, it is advisable to standardise the methods for assessing human exposure to plastics and further investigate to establish safe concentration levels for humans.

A multi-faceted approach is essential in advancing research and methodology related to nano- and microplastics. Firstly, it is crucial to develop standardized methods for collecting and analyzing human samples, which include identifying and quantifying microplastic fibers in human tissues like the lungs and accurately measuring environmental plastic concentrations. Enhancing *in vitro* testing is also key, requiring diversification to encompass a broader range of exposure times, plastic types, and cell models, thereby aiding in the understanding of the varied effects of different plastics. Personalized research is important too, recognizing that nano- and microplastics may interact differently with individuals, necessitating tailored research to comprehend these variations among diverse groups. When it comes to data quality and analysis, investigating the long-term, chronic effects of plastic exposure is paramount, given the extensive exposure time and continuous bioaccumulation of plastics in the body. Additionally, studying the impact of plastics on cells at very low concentrations is essential, as there is a lack of current data in this area. Finally, in assessing the impact on human health, it is necessary to develop *in vivo* testing methods to measure the concentration of different types of plastics in the human body, thus establishing a direct link between plastic exposure and potential health disorders. Gaining a deeper understanding of how nano- and microplastics interact at the cellular and molecular levels is crucial to extrapolating the potential risks these materials pose to human health. Additionally, a multi-disciplinary approach in research is crucial. Collaboration across various scientific fields, including toxicology, environmental science, medicine, and public health, is essential for a holistic understanding of the multifaceted impact of plastics.

In parallel with the above-mentioned research and methodology enhancements, it is important to address several aspects of daily life. To mitigate exposure to nano- and microplastics, individuals can implement preventive strategies including the reduction of single-use plastics, opting for products with minimal plastic packaging, and prioritizing natural materials over synthetic alternatives in daily consumption.

Regarding advancements in materials science, significant progress is being made in the development of biodegradable plastics (Rai et al., 2021; Narancic and O'Connor, 2019; Zubair and Ullah, 2020). These alternatives have the potential to significantly mitigate the environmental and health impacts associated with traditional plastics. The biodegradability factor plays a critical role in reducing long-term environmental accumulation and associated health risks.

Lastly, public awareness campaigns are vital in educating the populace about the potential health risks associated with plastics. The media, as a powerful tool, can significantly contribute to this effort by disseminating accurate and scientifically-backed information, thus fostering a well-informed public capable of making conscious decisions about plastic usage.

CRediT authorship contribution statement

Ewa Winiarska: Writing – review & editing, Writing – original draft,

Conceptualization. **Marek Jutel:** Writing – review & editing. **Magdalena Zemelka-Wiacek:** Writing – review & editing, Writing – original draft, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: MJ reports a relationship with ALK-Abello, Allergopharma, Stallergenes, Anergis, Allergy Therapeutics, Leti, and HAL, GSK, Novartis, Teva, Takeda, Chiesi that includes: consulting or advisory and speaking and lecture fees. MJ reports to be the Allergy journal Deputy Editor. MZW reports to be the European Academy of Allergy and Clinical Immunology Knowledge Hub Deputy Editor. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

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