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Neurotoxicity of Microplastics and Nanoplastics in the Human Brain

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Abstract

Microplastics and nanoplastics are new environmental polluters that are hurting humans due to their effects on our human environment everywhere and the health of our world. Scientists are worried about the new particles because they could pass through your brain by passing through your blood. The presence of corrupted micro protein particles in the bloodstream is dangerous to the human brain. Studies have confirmed that these particles can corrupt neurotransmitter systems, cause cell damage, and stimulate inflammation, among other things. Because of their small size, plus surface reactivity they can just reach out and touch the stuff in your brain. Tests on animals have shown that exposing them to tiny plastic pieces can hurt them, while other tests exposed to limited plastic have harmed microscopic human cells. While research about human exposure to plastic remains limited, studies suggests it a risk factor to extensive damage in the brain. We have to start paying attention to where plastic exists all around the world because it is very harmful to the environment. Understanding the harms that toxic chemicals cause in our brains may aid public policy and the creation of less harmful materials.

Keywords: *Microplastics; Nanoplastics; Neurotoxicity; Blood-Brain Barrier; Neuroinflammation*

Introduction

Microplastics (MPs; < 5 mm) and nanoplastics (NPs; < 100 nm) are plastic fragments and particles that vary in size, shape, and polymeric composition. Since they first appeared as an emerging contaminant, MPs and NPs have been carried by soil, water, and air to many areas on the earth; as a result, these plastics can now be found in tables, foods, water, and the air [1]. The majority of previous research focused on how MPs and NPs are normally distributed, while more recent studies have examined their effects on human wellbeing [2]. Among these studies, special attention was placed on the effects of MPs and NPs on the nervous system, since MPs and NPs have a global spatial distribution that reaches even the brain. The NPs can reach the brain easily and interact with neurons, cells, and biomolecules [3]. MPs

and NPs can also penetrate the blood-brain barrier, causing various problems [4]. When mice are chronically exposed to MPs and NPs, their behavior weakens; their brains become inflamed; and their spatial learning ability, memory, cognition, and locomotor activity are significantly impaired [1].

Overview of microplastics and nanoplastics

Plastics are indispensable materials widely used by society and can be found in every corner on the planet due to inappropriate solid waste management practices [5]. So far, humans are the species most impacted by plastic pollution because of high exposure probabilities through inhalation, ingestion, and dermal contact [6]. After contact or entry, plastic particles can be absorbed and accumulated at different levels in the organism, posing a serious threat to human health [1]. The maximum diameter of plastic particles suspended in the air that participates in atmospheric transport varies between 0.1 and 200 μm, and strongly depends on the particles emitted during the urban contamination processes [6]. Furthermore, in the aqueous phase, new larger particles of more than 200 μm may be transported [7]. Therefore, suspended plastic particles that have an aerodynamic diameter of less than 10 μm can enter the human respiratory system through gas exchange processes, reach the blood circulatory system through the lungs, and then be transported to the brain through blood circulation, where the particles may be accumulated [8].

Plastics have been classified according to particle size into macroplastics (≥25 mm), mesoplastics (5-25 mm), microplastics (MPs; ≤5 mm) and nanoplastics (NPs; ≤100 nm) [9]. These polymers are fabricated either from petrochemicals or biomass, by various production methods and polymerization mechanisms [10]. However, most plastics are petrochemical polymers, present three-dimensional networks, and contain polymer chains [11]. Their main environmental sources include plastic production factories and wastewater treatment plants [12]. Small plastic particles (MPs and NPs) have been found to prevail in most compartments of the environment, including air, oceans and freshwater systems, sediments, soils, and even the most remote areas on the planet [13]. Pristine plastic particles can also be released to the environment during production and processing procedures for use in cosmetics and personal care products [14]. In general, plastic particles appear in various shapes, including pellets, fragments, films, fibers, and foams [15].

Definition and classification

Microplastics (MPs) are synthetic solid particles or polymeric matrices ranging in size between 1 μm and 5 mm, insoluble in water [9]. Nanoplastics (NPs) are generally defined as plastic particles ranging from 1 to 1000 nm, although for environmental relevance this size range may vary [16]. According to morphological criteria, MPs can be divided into fragments, films, foam, and pellets, while NPs can be classified either as spherical or irregular [16].

Sources and environmental distribution

Microplastics (MPs, 1 μm-5 mm) and nanoplastics (NPs, ≤1 μm) are widely distributed in oceans and freshwater, sediments, drinking water, foods and air [1]. MPs derive from primary or secondary sources [17]. Primary MPs enter the environment as small micro-sized particles, such as in cosmetic products, whereas secondary MPs accumulate via fragmentation and weathering of plastic debris [18]. NPs are either manufactured deliberately or generated by fragmentation of MPs or other manners of degradation [19]. Aberrant weather conditions, scratching, as well as premature degradation could generate fragmented particles as small as NPs [20].

Mechanisms of neurotoxicity

Microplastic particles with any dimension larger than 100 nm, and nanoplastic particles with all external dimensions between 1 and 100 nm, are ingested by cells through phagocytosis and pinocytosis, respectively [1]. reviewed multiple routes-direct or secondary-by which micro- and nanoplastics distribute themselves throughout the brain; for example, placental transfer and food chain accumulation facilitate such penetration, and they underscore that smaller particles achieve wider dissemination [21].

The filaments, fibers, and film fragments can deposit, accumulate, and permeate the brain, inducing oxidative damage and neuroinflammation [12]. Micro- and nanoplastics release their constituent monomers, additives, and pathogens-which include toxic agents- and abnormal mitochondrial energy metabolism and neural apoptotic pathways are activated, ultimately perturbing neurotransmission and cognitive function [22].

Cellular uptake and distribution

Micro- and nanoplastics (MNPs) can reach the brain via the circulation, through the olfactory nerve or potentially via the vagal nerve following uptake in the gastrointestinal tract [23]. Depending on their size, particles smaller than 100 nm typically enter the brain, whereas larger particles tend to deposit in peripheral organs [1]. Once in the tissue, they may enter specific brain-cell types either via diffusion or endocytosis [24]. Neurons highly exposed to the circulation-such as hippocampal granule cells, medial-striatal medium spiny neurons and cerebellar Purkinje cells-exhibit relatively high particle uptake rates [24]. Entering the cells, particles interact with intracellular macromolecules, singularly or in combination with leached additives and co-contaminants, and can trigger elevated oxidative stress and inflammation [25]. These effects tend to be more pronounced when the polymer particle is in the nanosize range [4]. Increased reactive oxygen species (ROS) production yields calcium dyshomeostasis, mitochondrial damage, and endoplasmic reticulum stress, while microglial activation and altered cytokine secretion underlie the initiation of an inflammatory response [18]. In brain cells, experiments primarily on nanopolystyrene indicate MNPs generally cause signs of oxidative stress and inflammation, mirrored by suppressed energy metabolism, apoptosis and mitochondrial damage [26]. These conditions induce deficits in neurotransmission at the synaptic level for GABAergic, dopaminergic and glutamatergic transmission [27].

Oxidative stress and inflammation

The largest cellular responses to micro- and nanoplastics concern oxidative stress and inflammation [28]. Reactive-oxygen species and inflammatory cytokines directly modify neurotransmission and thereby disrupt the central nervous system at a functional level [29].

Oxidative stress follows from an imbalance between the production of oxidants and the antioxidant capacity of the system. Micro- and nanoplastics induce an excess of free radicals that disturb the prevailing equilibrium in favour of a hazardous oxidative stress state [1]. An increased production of reactive-oxygen species leads to lipid peroxidation, protein carbonylation and DNA damage [30]. Exposure to micro- and nanoplastics therefore results in a pronounced upregulation of antioxidant enzymes and sensitizes cells to oxidative injury [1].

The inflammatory response consists of an increased expression and release of multiple pro-inflammatory mediators, including NF-κB, IFN1, IL-1β, IL-6, IL-8, iNOS and TNF-α [31]. These inflammation markers have been unequivocally observed in multiple *in vivo* studies, whereas *in vitro* experiments with multiple cell types describe similar upregulation [32].

Impact on neurotransmission

Microplastics (MPs) and nanoplastics (NPs) are plastic fragments smaller than 5 mm and 100 nm, respectively [27]. Their environmental omnipresence constitutes a major threat to human health, ultimately exerting neurotoxic effects after translocation and accumulation in brain tissue [33].

Uptake of polystyrene (PS) particles by neuronal cells has been documented, and MP exposure gives rise to oxidative stress and inflammatory responses in the brain [22]. PS-MPs and PS-NPs induce a Parkinsonian syndrome in rats upon chronic exposure, an effect explained by the inhibition of dopamine synthesis [34]. NPs tend to inflict more damage than MPs [1].

Human exposure pathways

Humans are exposed to micro- and nanoplastics mainly through inhalation, ingestion, and dermal contact [1]. The presence of microplastics in indoor air and airborne fallout renders inhalation a worldwide concern [5]. Additionally, microplastics enter the air from road traffic, synthetic textiles, construction materials, and incineration at a local level [35]. Furthermore, microplastics accumulate in water, sediment, and marine organisms that are eventually consumed by humans [36]. Experiments in animal models and *in vitro* organoids demonstrate that nano-sized plastics translocate to the brain via the lung-to-blood-to-brain and gut-to-blood-to-brain routes; microplastics apparently do not [35]. Research on microplastics is therefore of utmost importance in the search for links to human pathologies related to brain dysfunction and exposure to airborne environmental pollutants [37].

Inhalation and ingestion

Microplastics (MPs, < 5 mm) and nanoplastics (NPs, < 1 μm) represent an emergent environmental contaminant of potential concern for neurotoxicology [38]. Human epidemiology and experimental data consistently demonstrate that the neurotoxicity of these particles increases markedly as particle size declines from the micro- to the nano-scale range [1]. Long-term airborne MPs and NPs exposure causes cognitive deficits and neural damage in both laboratory models and exposed workers [39]. Experimental studies reveal substantial size-dependent particle uptake by neurons, poor capacity for cellular elimination and extensive brain distribution, particularly in the case of NPs [14]. Adsorbed environmental pollutants and (due to their physicochemical composition) persistent oxidative stress and inflammation represent important mediators of MP- and NP-induced damage; neurotransmission is also strongly affected [40]. Unlike larger particles, nano-sized MPs and NPs readily cross the blood-brain barrier from the respiratory tract, as a result of chronic exposure at relatively low airborne concentrations [17]. Ambient air near busy roadways and in many indoor environments contains at least several thousand MP- and NP-sized particles per m3 [41]. Inhalation constitutes a central human exposure pathway for neural MP and NP uptake and damage [42]. Particles can also enter the body exclusively via ingestion, e.g. hand-to-mouth transfer of airborne MPs and NPs deposited on groceries and in food preparation areas [43]. An initial human exposure event will therefore generally involve a combination of inhalation, ingestion and dermal contact, as well as potential occupational routes, e.g. heat generation during welding leading to polymer release for operators using plastic-coated steel wire [44].

Dermal contact

Human skin offers a significant barrier against environmental agents, protecting against ultraviolet radiation and limiting the penetration of particulate matter [44]. The skin is composed of three principal layers-epidermis, dermis, and subcutaneous tissue-each exhibiting structural differences that govern permeability [1]. Generally, particulates larger than 100 nm are unable to traverse the skin barrier under normal conditions [45].

The occasional presence of microplastics within human tissue suggests that skin penetration is not a major exposure pathway [46]. Nonetheless, nanoplastics can directly enter the dermis, especially in cases of skin disease or wound healing [20]. Empirical evidence indicates that 20-nm polystyrene nanoparticles readily permeate various animal and human skin models [47]. *In vivo*, dermal exposure leads to accumulation of 20-nm particles in organs including the pancreas, brain, and heart [48]. Additionally, 100-nm nanoparticles have the capacity to penetrate compromised human skin models [24].

Occupational exposure

For the general population, inhalation, ingestion, and dermal contact provide opportunities for hazardous exposure to microplastics and nanoplastics, while occupational exposure may additionally occur in industrial and laboratory settings [1]. Several plastic-worker cohort studies have reported neurological and neurodegenerative problems [49]. Likewise, neurological outcomes were also evident in rats treated with polychlorinated biphenyls (a plasticizer), triggering neurotoxicity via mechanisms such as oxidative stress [50].

Long-term health implications

The increasing prevalence of microplastics and nanoplastics in aquatic environments constitutes a potential threat to water safety as well as to human health [20]. Microplastics and nanoplastics readily cross biological barriers of cells and are consequently widely distributed throughout the human body and tissues, including the brain [37]. Microplastics have been detected in human blood as well as in lung tissue and placenta, yet nanoplastics are of greatest concern because of their ability to cross the blood-brain barrier and penetrate cells more readily than their larger counterparts [1]. Chronic exposure to microplastic and nanoplastic particles is likely to cause brain damage that manifests itself primarily in the form of neurodegenerative diseases [51].

Cognitive decline

Cognitive decline is characterized by a gradual loss of cognitive abilities, often associated with neurodegenerative diseases such as Alzheimer's and Parkinson's [27]. Extrinsic factors contribute significantly to the onset of such diseases [52]. The persistence of micro- and nanoplastic particles in the environment raises the question of whether their potential neurotoxic effects could promote cognitive decline in humans [1].

Engineered and environmental nanoplastics readily diffuse across membranes during treatment and are primarily incorporated by endocytosis in particle models [53]. Microplastic exposure in aquatic invertebrates results in blockage of neuronal uptake of synaptic vesicle (SV) proteins SYT1 and SV2, likely due to competitive membrane binding by particles [54]. Although microplastic exposure induces increased basal levels of reactive oxygen species (ROS) in invertebrates *in vivo*, engineered and environmental nanoplastics do not increase ROS or alter oxygen consumption in neuronal cell-line models [55].

Microplastics in the digestive tract distribute across all organ tissues and cross the blood-brain barrier to reach neurons, triggering oxidative stress via free radicals that reduce antioxidant enzymes and damage cells [56]. Neuronal damage is implicated in neurodegenerative diseases such as Alzheimer's, with early symptoms including impaired learning and memory [2]. Chronic toxicity requires sustained dose and duration for cellular damage to manifest [57]. Component chemicals are expected to exert limited effects due to low concentrations and slow degradation [15]. Model particles confirm that toxicity arises primarily from incidental particle effects rather than chemical composition [7]. Primary toxicity correlates inversely with particle size. Among plastic types of a given polymer class, polyvinyl chloride (PVC) exerted the strongest effects, possibly due to residual monomer content and inherent toxicity [28].

Neurodegenerative diseases

Microplastic and nanoplastic exposure is significantly linked to neurodegenerative pathologies. Microplastics notably disrupt neurotransmitter expression and activity, affecting dopamine, glutamate, serotonin, GABA, and acetylcholine systems [27]. Enzymatic activities of acetylcholinesterase and acetylcholine transferase, essential for cholinergic neurotransmission, are altered by exposure [58]. Corresponding brain tissue changes mirror chronic degenerative diseases such as Parkinson's and Alzheimer's [59]. In exposed mice, the brain coefficient inversely correlates with the microplastic dose. Histological abnormalities include irregular cellular arrangements, cytoplasmic vacuolisation, neuronal degeneration, and mitochondrial rupture [26]. Such cellular damage is associated with oxidative stress, demonstrated by elevated reactive oxygen species and related gene expression [37]. In neuronal cultures, microplastics induce apoptotic cell death accompanied by depletion of cholinergic, GABAergic, dopaminergic, and glutamatergic neurons [27]. Amplification of pathways involved in neurodegenerative diseases further underscores the neurodegenerative potential of exposure [3]. *In vivo* models of Alzheimer's and Parkinson's diseases reveal that microplastic exposure exacerbates neuroinflammation, microglial pyroptosis, and dopaminergic neuron degeneration. Microplastics additionally promote α-synuclein aggregation, a hallmark of neurodegenerative processes [35].

Conclusion

Microplastic (MP) and nanoplastic (NP) debris have been recognized as emerging contaminants of increasing concern due to potential accumulation in biological tissues following long-term human exposure via different routes [18]. Polymers are deposited in the environment during manufacturing, use and disposal, then fragmented into smaller plastic particles via physical and chemical processes with external stimuli such as corrosion from wind, sunlight (UV), water and microbes [27]. Due to their dispersive and persistent character, MPs and NPs easily penetrate the atmosphere, hydrosphere and biosphere, representing worldwide environmental risks [60]. In particular, inhalation and ingestion are the main entry pathways into the human body, leading to biodistribution of plastic microparticles into the bloodstream and accumulation in many tissues, mainly the lungs, liver, heart, spleen and brain [61]. MPs and NPs can cause a succession of pathological conditions depending on their routes of exposure, dose and time of exposure. MP/NP pollutants are present in various environmental compartments [1].

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