

REVIEW

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The role of microplastics exposure in Alzheimer's and Parkinson's disease

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Abstract

In recent times, both in vitro and in vivo studies have demonstrated toxicological and pathological evidence of microplastic exposure in neurological dysfunction and diseases. The mechanistic linkage of microplastics and nanoplastic exposure in Alzheimer's disease and Parkinson's disease—two of the most common neurodegenerative diseases in humans have not been explored. This gap in knowledge forms the basis of this review. Moreover, the global population keeps 'consuming' plastics through the skin, nostrils and mouth—which ultimately could result in intoxication, pathologies and contribute to the pathogenesis of Alzheimer's and Parkinson's. This narration examined the relationship between the risk of encountering microplastics and nanoplastics and the possible mechanisms of neurotoxicity and neurodegeneration, including oxidative stress, neuroinflammation, disruption of the blood–brain barrier, and protein clustering/ aggregation in both animal models and humans. Though strong linkage was found between microplastic exposure and the neurodegenerative diseases, further studies are required in this area—while public health scientist and policy makers are called to act to curb the global environmental challenge of plastic pollution.

1 Introduction

Microplastics are small plastic particles of sizes smaller than 5 mm. Microplastics (MPs) and its 'derivative' nanoplastics (NPs), which are typically defined as plastic particles smaller than 100 nm (and sometimes up to 1,000 nm depending on classification), continue to pose significant global- environmental and health challenge. These particles have been found in almost every part of the environment—including air, water and soil. They have even been found in the gastrointestinal tract of some animals and in the tissues of others [1]. They penetrate tissues by traversing (across) biological barriers through ingestion, inhalation or skin exposure, leading to their detection in lungs, breast milk, urine, blood, placenta and liver of humans [2–5]. Globally, the world has seen exponential increase in the use of plastics in the past 70 years—from 1.5 million tonnes in the 1950s to about 359 million tonnes now [2]. Moreover, some intestinal, cardiovascular, infectious and inflammatory diseases and cancers—of unknown 'traditional'



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etiology, have recently been known to be induced by MPs [2]. Once MPs and NPs enter the circulatory system and gets into the brain for example, they can traverse their immediate barriers due to their hydrophobicity and may elicit cytotoxicity, inflammation and oxidative stress in affected neurons, impairing signaling pathways leading to the progression to neurodegenerative diseases such as Alzheimer's and Parkinson's diseases [2]. Worldwide, neurological diseases continue to be the main source of disability, including neurodegenerative diseases such as Parkinson's disease, which is increasing in occurrence, resulting in disabilities and deaths [6]. On the other hand, Alzheimer's disease (AD) is the most prevalent neurodegenerative disorder and a leading cause of dementia in the aged population [7]. One major hallmark of neurodegenerative disease is misfolding of amyloid proteins, and MPs/NPs have been known to induce protein folding and accelerate the formation of abnormal amyloid proteins[8].

MPs and NPs promote the progression of neurodegenerative diseases through oxidative stress that generates reactive oxygen species (ROS), which disrupts antioxidant enzyme activities, leading to the formation of protein aggregates such as amyloid beta ($A\beta$), alpha-synuclein (α -syn) and Tau proteins [2]. Previous literature has cited microplastics in relation to recognized environmental factors (such as air pollution)—highlighting their capacity to activate common mechanisms associated with neurodegeneration. [9]. This narrative review will discuss the effects of the risk of exposure to MPs and NPs, to the potential mechanisms of neurotoxicity, such as oxidative stress, neuroinflammation, blood–brain barrier (BBB) disruption and protein aggregation in AD and PD, and synthesize some gaps for research and policy decisions. While the field is nascent, growing experimental and observational data indicate a plausible connection warranting further investigation.

2 Methodology

The review evaluates studies published in English in peer-reviewed journals or books from 2015 to 2025 to highlight the latest advancements in microplastics exposure in Alzheimer's and Parkinson's diseases. Online databases that were included Google Scholar (<https://scholar.google.com/>), PubMed (<https://pubmed.ncbi.nlm.nih.gov/>) Scopus (www.scopus.com) and guidelines from relevant health organizations were searched using the keywords microplastics, environmental pollution, human health, Alzheimer and Parkinson's diseases. The inclusion criteria were:

- Articles in peer-reviewed journals indexed in Google Scholar, PubMed, and Scopus were considered.
- Publications published in peer-reviewed journals indexed in Google Scholar, PubMed, and Scopus were evaluated. The search retrieved an extensive selection of literature, including experimental studies, systematic reviews, and policy recommendations.

2.1 Microplastics: sources, pathways, and exposure

The term Microplastics was coined by Richard Thompson who studied plastic waste in ocean bodies and described these tiny plastic particles that have become a global concern as microplastics, drawing attention of governmental and nongovernmental organizations and scientists [10]. Microplastics are tiny plastic particles with sizes less than 5 mm that are pervasive in the environment, with an estimated 400,000 tons released on

land and waterbodies [2] Microplastics are classified based on their origin into primary MPs, which are purposefully made for use in textiles, cosmetics and other industrial purposes, while secondary MPs describe the disintegration of larger plastics into minute particles due to heat, ultraviolet (UV) radiation and chemical degradation [2].

The main routes of exposure to MPs and NPs in Humans are via ingestion, inhalation and dermal contacts [2]. Humans ingest MPs through consumption of contaminated food and water, and dermal contact occurs using personal care products such as soap, scrubs and face.

wash [11]. Through oral ingestion (food, tooth paste etc.), MPs can move across the digestive epithelium into the circulatory system, a pathway described as translocation. Microplastics, therefore, are accumulated in the human body through translocation with the particles passing through the epithelial cells or absorbed by specific microfold cells [11]. Figure 1 shows how MPs and NPs get into the blood and other body systems through the 3 modes explained.

The small size of NPs facilitates their entry into the human body through inhalation (Fig. 1), which starts at the olfactory region and links the trigeminal nerve by penetrating the nasal mucosa lining and eventually ending up in the alveolar space and lung capillary bed [11]. The transport into the circulatory system is also enhanced because of the ability of NPs to attach to red blood cells (RBCs) and white blood cells (WBCs) in the systemic circulatory system [2]. The MPs and NPs which are airborne are inhaled through the olfactory nerve bulb, often described as nose-to-brain transport, leading to their accumulation in the olfactory epithelium [12].

2.2 Towards nervous system: blood–brain barrier penetration and oxidative stress

The blood brain barrier (BBB) is a defensive barrier made up of endothelial cells that are tightly attached by junctions that protect the brain tissue from likely toxic substances circulating in the blood plasma [13]. The BBB therefore prevents the entry of most blood-borne pathogens and particulate matter (PM) from entering the brain,

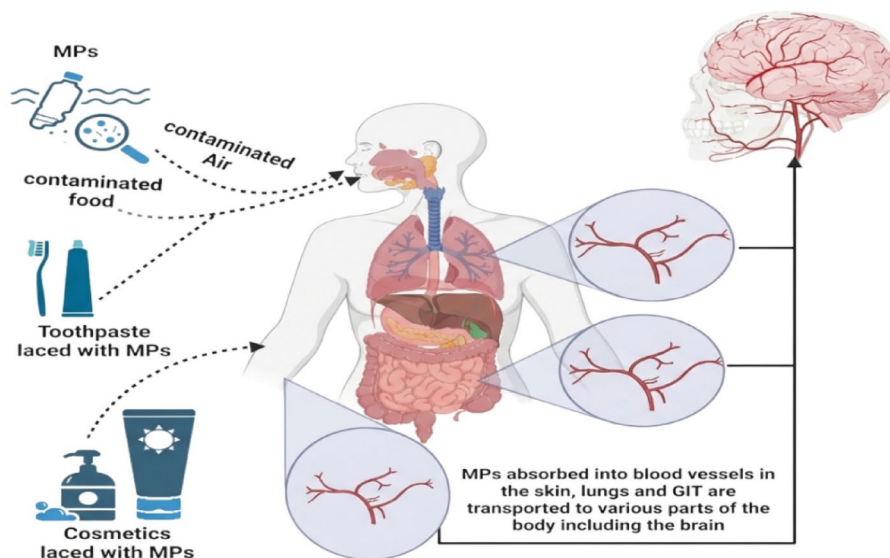


Fig. 1 Routes of Microplastic Exposure, (i) GIT into the blood and disseminated systematically, (ii) Dermal into the blood and disseminated systematically, (iii) Nasal and through the Pulmonary system

which ensures that there is a stable homeostatic microenvironment for normal functioning of the central nervous system [14]. There is much evidence demonstrating that a defect in the physiological structure of the BBB is manifested in the occurrence of several diseases, such as neurodegenerative diseases, ischemic stroke, multiple sclerosis and peripheral diseases [15]. Moreover, evidence suggests that MPs/NPs can cross biological barriers such as the BBB, eliciting neuroinflammatory responses and leading to progression to neurodegenerative diseases such as Alzheimer's and Parkinson's diseases [2]. Alzheimer's AD is characterised by the breakdown of the BBB, thereby permitting the infiltration of peripheral toxins and immune cells into the brain [16], and microplastics have the potential to either cross or compromise the integrity of the BBB, leading to the introduction of some of these foreign particles that initiate oxidative stress, promoting further inflammatory responses [16]. Polystyrene (PS), a polymer type of MP, can stimulate inflammatory responses and oxidative stress when present in the brain regions by activating pro-inflammatory cytokines such as tumour necrosis factor (TNF- α) and interleukins that cause neural damage [2]. Though direct human studies on the mechanistic overlap between Parkinson's disease, Alzheimer's disease, and microplastics are extremely limited, there are evidence from laboratory experiments and animal models, with human data largely restricted to biomonitoring. The work done by Alexander Chi Wang Siu et al. highlights biomonitoring evidence of microplastics in human blood and stool but notes the absence of direct mechanistic human studies [17]. Again, another study detected nanoplastics in human blood and raises concern about their ability to cross the BBB, but stresses that direct human mechanistic evidence is lacking [18].

Both Alzheimer's disease (AD) and exposure to microplastics share common mechanisms related to oxidative stress, neuroinflammation, and mitochondrial dysfunction. Microplastics exacerbate pathways that are already associated with AD, including the accumulation of reactive oxygen species (ROS), lipid peroxidation, activation of microglia, and pyroptosis, consequently intensifying neuronal damage and cognitive deterioration [19]. In Alzheimer's disease (AD), the excessive production of ROS results in damage of proteins, lipids, and DNA, which ultimately contributes to synaptic dysfunction and the death of neurons [20]. Microplastics, particularly polystyrene particles, also promote the accumulation of ROS, surpassing the capacity of antioxidant defenses and initiating further oxidative damage [21]. Both scenarios exhibit lipid peroxidation within neuronal membranes, which compromises cellular integrity and disrupts signaling [21].

2.3 Microglial activation & pyroptosis, amyloid beta aggregation, and neuroinflammation

Besides the action of ROS and the mechanism of oxidative stress in Alzheimer's disease (AD), microglia undergo chronic activation, leading to the release of pro-inflammatory cytokines and exacerbating neuronal damage [22]. Microplastics have been shown to induce microglial pyroptosis, a form of programmed cell death characterized by high levels of inflammation, thereby intensifying neuroinflammation as noted by Wang et al. [17]. This intersection indicates that microplastics could potentially hasten the progression of AD by conditioning microglia to adopt a harmful phenotype (Fig. 3). The hallmark of AD consists of amyloid- β plaques and tau tangles, which are aggravated by oxidative stress [20] (Fig. 2). Microplastics contribute to the worsening of protein misfolding indirectly by elevating ROS and inflammatory mediators, thereby destabilizing proteostasis [22]. These conditions exhibit compromised autophagy and lysosomal

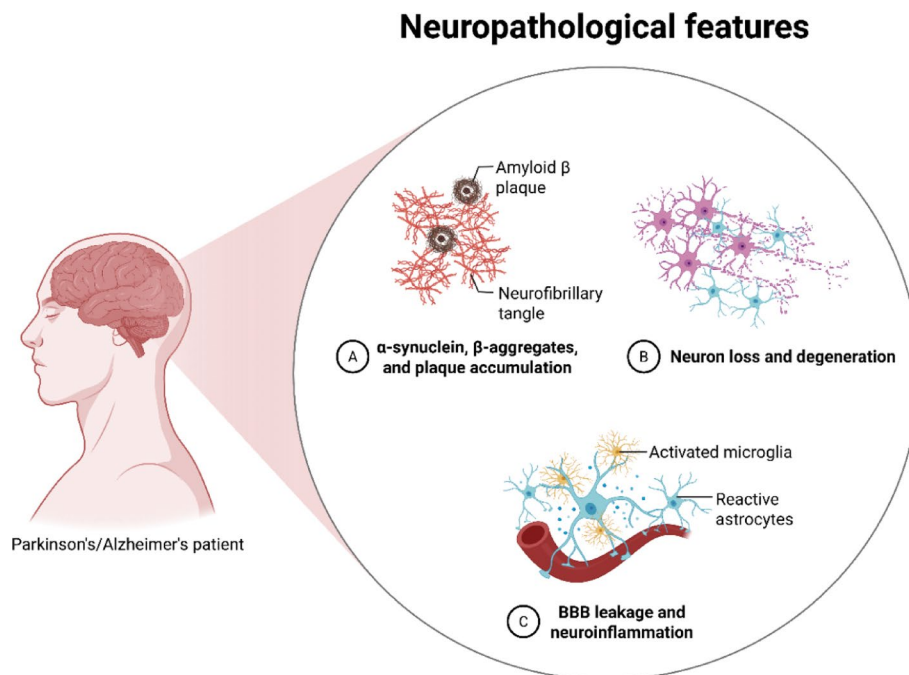


Fig. 2 Neuropathological features of Parkinson's and Alzheimer's Disease. Accumulation of α -synuclein, Amyloid β plaque and Tau protein **(A)**. Neuronal loss and degeneration **(B)**. Blood–Brain Barrier (BBB) leakage and neuroinflammation

clearance, resulting in the accumulation of toxic proteins. It is characterized by mitochondrial dysfunction, which leads to a decrease in ATP synthesis and an increase in the leakage of ROS [20]. Also, Microplastics interfere with the mitochondrial membrane potential and the electron transport chain, perpetuating the same detrimental cycle of oxidative stress [21]. There is an increasing amount of experimental research indicating that micro- and nanoplastics can interact with pathways relevant to AD, particularly those associated with neuroinflammation, disrupted processing and clearance of amyloid- β , as well as dysfunction of microglia.

2.3.1 Pyroptotic signaling in microglia

Polystyrene microplastics (PS) induce microglial pyroptosis in vitro, with findings indicating that gasdermin D (GSDMD) is a key mediator of this process; the silencing of GSDMD reducing the occurrence of pyroptosis. In vivo, exposure to PS intensifies neuroinflammation and deteriorates cognitive function in AD mouse models, thereby establishing a connection between plastic exposure and the inflammatory cell death pathways that are pivotal to Alzheimer's pathology [17]. (Fig. 3).

2.3.2 Impaired microglial clearance of amyloid- β

Nanoplastics obstruct the ability of microglia to uptake and eliminate amyloid- β , thereby interfering with the cellular processes that typically regulate extracellular A β levels. This disruption establishes a direct mechanistic connection between exposure to NPs and the pathology associated with the amyloids [17]. There are also changes in receptor-mediated phagocytosis and intracellular processing pathways, aligning with the noted decrease in A β clearance ability following exposure to nanoplastics [17].

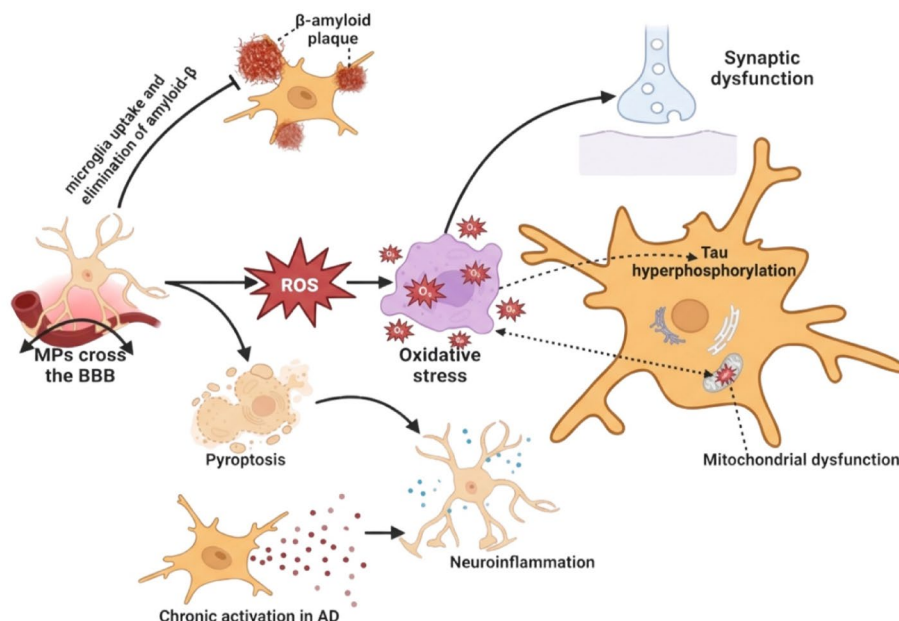


Fig. 3 Mechanisms of Microplastic intoxication, pathogenesis and pathophysiology in Alzheimer's Disease

In summary, micro- and nanoplastics have been experimentally demonstrated to trigger microglial pyroptosis and enhance neuroinflammation and hinder the clearance of amyloid- β by microglia, which are two fundamental mechanisms involved in the pathophysiology of AD [17]. This offer substantial mechanistic connections, while the diverse types of polymers, various exposure conditions, and tau-related pathways necessitate additional focused research.

2.4 Microplastics and Parkinson's disease: pathogenic and pathophysiologic mechanisms

Mitochondrial dysfunction is a key characteristic of Parkinson's disease (PD), with various microplastic-related impacts converging on identical mitochondrial pathways, including oxidative stress, susceptibility of complex I, compromised mitophagy, calcium imbalance, and a collapse in bioenergetics. It is characterized by the loss of dopaminergic neurons with the dysfunction of electron transport chain complex I, increased levels of ROS, and a disturbance in redox balance [21]. Both genetic and toxin-induced models highlight complex I impairment as a defining characteristic of PD [23]. It has been documented that exposure to MPs and NPs elevates cellular ROS levels and induces oxidative damage in both neural and peripheral models, thereby placing stress on mitochondria and enhancing electron leakage [21]. (Fig. 4). Moreover, NPs have been demonstrated to infiltrate neurons, disrupt the degradation of structures associated with Parkinson's disease (PD), and cause harm to brain areas in mice, indicating a molecular connection between plastics and α -synuclein pathology. This observation is consistent with fundamental mechanisms of PD, where compromised lysosomal and autophagic processes promote the accumulation of α -synuclein and the formation of Lewy bodies [21]. Other preliminary findings indicate that NPs could potentially be associated with Parkinson's disease; however, there is a lack of direct and reproducible evidence demonstrating accelerated misfolding or aggregation of α -synuclein or tau/amyloid- β in environmentally realistic conditions [24]. Moreover, other preliminary studies indicate that A β and α -syn may work together to facilitate their mutual aggregation and accumulation.

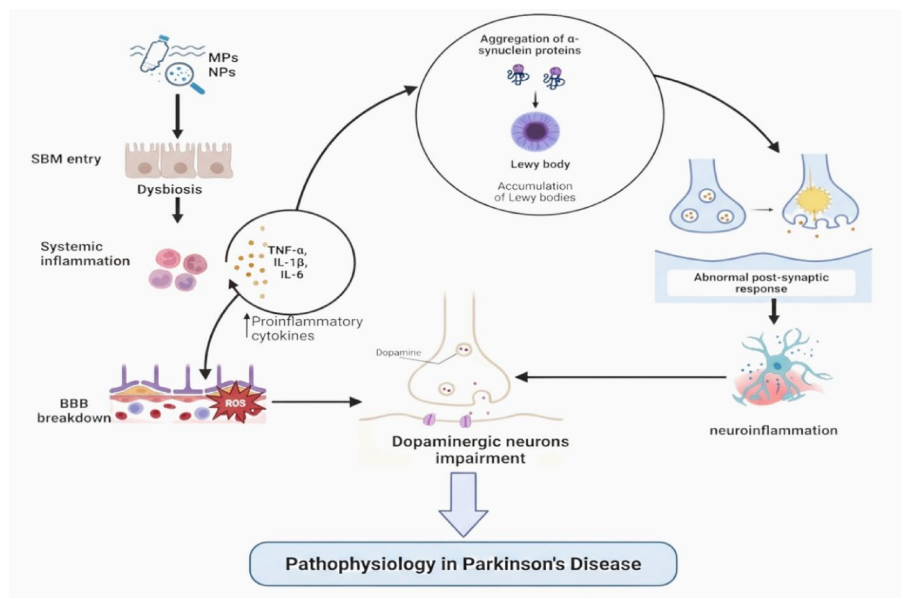


Fig. 4 Mechanisms of Microplastic and Nano intoxication, pathogenesis and pathophysiology in Parkinson's Disease

Although the precise mechanisms underlying the interaction between these proteins are not fully understood, an increasing body of evidence points to the possibility that A β could hinder protein clearance, trigger inflammatory responses, increase phosphorylation, or directly encourage aggregation [21].

Also, the effects of microplastics on gastrointestinal environments indicate a possible alteration of the enteric-nervous system and gut inflammation. This observation is significant considering the prodromal gut involvement in PD and the proposed propagation of α -synuclein from the gut to the brain through the vagus nerve pathway [2]. Studies in mouse models have shown that polystyrene NPs are capable of infiltrating nerve cells in mice, disrupting the degradation of α -synuclein-associated structures, and causing harm to brain areas pertinent to PD [21]. This offers clear evidence that environmental nanoplastics may facilitate the accumulation of α -synuclein, which is a defining characteristic of Parkinson's Disease.

An examination of rodent models reveals that exposure to MPs induces oxidative stress, neuroinflammation, and a decrease in dopaminergic neurons, which culminates in motor dysfunction that mirrors the symptoms of Parkinson's disease [21] (Fig. 4). These investigations tell a mechanistic connection between the pathology induced by microplastics and the degeneration of the nigrostriatal pathway associated with PD. In humans, nanoplastics have been detected in blood, lungs, placentas, and even brain tissue [3–5, 12], thereby confirming that environmental exposure can penetrate the central nervous system. This provides biological plausibility for the epidemiological correlation with neurodegenerative diseases such as PD [2]. In another study, nanoplastics were found to be capable of infiltrating nerve cells, disrupting the degradation of α -synuclein-associated structures, and causing harm to brain areas – characteristics pertinent to PD in mice [21]. Although this information is derived from animal studies, the National Institutes of Health (NIH) of the United States of America (USA) underscored the public

health concern that increasing plastic pollution could elevate the risk of developing Parkinson's disease [21].

2.4.1 Susceptibility of α -syn to surface-induced misfolding, including NP interactions

It is proposed that the aggregation of α -Syn is significantly linked to the pathogenesis of Parkinson's Disease (PD). The pivotal involvement of α -Syn aggregation in PD is further corroborated by the discovery of point mutations in the α -Syn protein that are related to rare familial variants of PD. Nevertheless, the relationship between the aggregation tendencies of α -Syn mutants and their connection to the PD phenotype is complex. Recent findings indicate that oligomers, which are produced during the early phases of aggregation, represent the highly neurotoxic entities responsible for neuronal death in PD. However, the diverse and unstable characteristics of these oligomers hinder their comprehensive analysis. In contrast, α -Syn fibrils have been demonstrated to act as infectious agents, propagating in a manner akin to prions [25].

2.4.2 Significant involvement of the gut-brain axis

Recent studies indicate a possible connection between the commensal gut microbiota and the brain, which may affect neurodevelopment, cognitive function, and overall health. This two-way communication is commonly known as the microbiome–gut–brain axis. Increasing evidence points to the fact that the emergence of non-motor symptoms, including gastrointestinal issues, frequently occurs before the appearance of motor symptoms and the formal diagnosis of the disease, thereby reinforcing the potential significance of the microbiome–gut–brain axis in the fundamental pathological processes associated with PD [26].

2.4.3 Uptake of nano plastics into the nigra region of the brain

Researchers investigated the interactions between alpha-synuclein fibrils and nanoplastics in cultured brain cells, specifically neurons. Their findings revealed that both fibrils and plastics can penetrate neurons through a process known as endocytosis, where the cell's outer membrane envelopes specific substances. Once internalized, both fibrils and plastics were found to enter the lysosomes, which are membrane-bound organelles functioning as the cell's waste disposal system. The study indicated that NPs interfered with lysosomal functions, thereby hindering the degradation of toxic aggregates of alpha-synuclein [21]. Subsequently, the research team examined the interactions of polystyrene NPs and alpha-synuclein within the mouse brain. They discovered that these NPs and alpha-synuclein fibrils also interacted in this context, leading to an increased dissemination of pathological features throughout interconnected brain areas. Notably, neurons in the substantia nigra region of the brain were particularly impacted. This area is crucial for motor skills and movement regulation, and is known to be compromised in PD, besides other related dementias [21].

2.5 Effect of microplastics on target proteins in Alzheimer and Parkinsons diseases

Micro- and nanoplastics (MNPs) function as catalytic surfaces, driving up the misfolding and aggregation of proteins associated with Alzheimer's and Parkinson's disease. These particles penetrate past the blood–brain barrier and interact directly with proteins through hydrophobic and electrostatic forces.

In Parkinson's disease and alpha-synuclein (α -syn) interactions, nano plastics have a strong affinity for the amphipathic and NAC domains of α -syn. Alpha-synuclein aggregates are intracellular protein clumps primarily associated with Parkinson's disease. Toxic fibrils form rapidly as a result of this interaction, which produces a "nucleation center" that decreases the energy barrier for protein aggregation [27]. Microplastics converge with α -syn in the lysosomes after translation. Plastic prevents these aggregates from breaking down, which encourages their buildup and subsequent prion-like distribution throughout the brain. [21] Research reports that prolonged exposure to micro/nano-plastics triggers the phosphorylation of α -synuclein at Ser129, a characteristic of Parkinson's disease, resulting in the targeted degeneration of dopaminergic neurons in the substantia nigra [27].

The relationship between Amyloid-Beta ($A\beta$) and Alzheimer's disease is influenced by microplastics, which stabilize the toxic oligomeric forms of Amyloid-beta. The hydrophobic layer on microplastics draws in the hydrophobic regions of $A\beta_{42}$ monomers. This interaction facilitates the conversion from random coils to β -sheet structures, which are the fundamental components of amyloid plaques [22]. Reports demonstrate that $A\beta$, "seeded" on nanoplastic surfaces produces aggregates that are considerably more cytotoxic than those generated without plastics, resulting in increased neuronal apoptosis rates [u,v]. Exposure to MNPs induces oxidative stress and activates kinases, such as GSK-3 β , resulting in the hyperphosphorylation of Tau proteins. Hyperphosphorylated Tau dissociates from microtubules, resulting in their collapse [2, 22] This impairs axonal transport, resulting in the production of neurofibrillary tangles, a major cause of cognitive impairment in Alzheimer's [28]. Beyond direct binding, microplastics can also affect these target proteins through cellular disruption through decreased mitochondrial membrane potential, limiting the ability of "chaperone" proteins to refold or remove damaged proteins [28]. Microplastics can cause microglia to develop a pro-inflammatory M1 phenotype. Chronic inflammation leads to protein misfolding and hinders the removal of $A\beta$ plaques and α -syn aggregates by phagocytes [28]. $A\beta$ plaques are extracellular clumps of protein fragments primarily associated with Alzheimer's disease.

2.6 Physicochemical characteristics of micro plastics in the environment

The toxicity of microplastics is substantially influenced by their physical and chemical characteristics, including polymer type, particle aging and weathering, size, and additive composition. These factors collectively affect the ability of microplastics to penetrate biological systems and induce adverse effects like oxidative stress, inflammation, and neuroinflammation.

2.6.1 Polymer type

The kind of polymer greatly affects toxicity, determining the interaction of micro and nanoplastics with biological systems, the induction of oxidative stress, and the leaching of dangerous additives. Polyethylene terephthalate (PET) and polyvinyl chloride (PVC) nanoplastics are typically more cytotoxic than polystyrene (PS) due to their innate chemical composition. They cause oxidative stress, cell death, and disruption of metabolic pathways [29, 30].

Polyethylene (PE) and polypropylene (PP) are polyolefins characterized by extensive linear hydrocarbon chains that are resistant to spontaneous decomposition. Polyethylene

(PE) and polypropylene (PP) polymers may require over a century to decompose [31]. Polypropylene and polyethylene have strong hydrophobicity, making them more effective at adsorbing and transporting organic pollutants (such as triclosan or heavy metals) than other materials [32].

Polypropylene's high molecular weight of 42 g/mol, tensile strength of 31–41 mPa, and hardness of 98% make it more resistant to cracks, acids, organic solvents, and electrolytes, as well as having greater hydrophobic characteristics. PE has a lower molecular weight than PP (28 g/mol), tensile strength of 17–33 mPa, and hardness of 60–69% [33, 34]. Furthermore, PP has a triple bond (alkyne, $C\equiv C$) in its chain structure, making it stronger than PE, which only has a double bond (alkene, $C=C$) [35]. These properties confer PE and PP with high stability and strong resistance, which is the primary reason for their use in industries. The presence of several plastics in human brain tissues, including polyethylene and polystyrene, has been demonstrated to be related to higher levels of neuroinflammatory markers [36]. Polyethylene and polystyrene typically have higher translocation rates across biological barriers, such as the blood–brain barrier (BBB), which results in more severe neurological impacts such as acetylcholinesterase inhibition and neuroinflammation [36]. Plastic polymers, particularly in the form of microplastics and nanoplastics can significantly affect Parkinson's disease (PD) by accelerating the misfolding and aggregation of the protein alpha-synuclein. These particles can enter the brain, causing damage to dopaminergic neurons, particularly in the substantia nigra region [21]. Polymer type has a significant impact on Alzheimer's disease (AD) in two ways: as environmental contaminants (microplastics), that could contribute to disease progression via neurotoxicity, and as therapeutic carriers (biopolymers), which are designed to deliver drugs across the blood–brain barrier (BBB) to treat the disease [37].

2.6.2 Particle aging and weathering

Particle aging and weathering are transformative processes that alter the physical, chemical, and biological properties of microplastics (MPs) when they reach the environment. After various aging processes, MPs' physicochemical characteristics alter, making them easily absorbed by organisms and having biological impacts. The aging reaction breaks down MPs into smaller particles, affecting their absorption by organisms. Because of the mouth's limited size, creatures (such as oyster larvae and nematodes) were easier to absorb tiny MPs, which might ultimately result in toxic effects [38]. Weathering-induced photo-oxidation of plastics degrades their material properties, causing decreasing strength, fragility, and, when exposed to environment mechanical stress, there is plastic fragmentation into smaller pieces [39].

Studies indicate that weathered PS microplastics (MPs) can facilitate the indirect photolysis of organic contaminants in water by forming reactive species under sun irradiation and functioning as heterogeneous photosensitizers [40].

2.6.3 Size heterogeneity and barrier penetration

Size is the most critical determinant of whether a particle remains localized or becomes a systemic neurotoxicant. The polydispersity of MNPs in the environment is a primary barrier to translational research, as toxicity is highly size-dependent. Microplastics can cross the blood–brain barrier (BBB) and affect the central nervous system; with

Table 1 Overview of Research Studies on Polymer Exposure and Neurological Effects

Study (date)	Polymer type	Exposure dose & route	Key neurological outcomes
Han et al. (2025) [43]	Polystyrene (PS-NP & PS-MP)	20 nm (NP) / 0.25 µm (MP); 20 µg/mouse/day (16 weeks, Inhalation)	PS-NP showed 2.9× higher brain accumulation; induced α-synuclein clumping, dopamine neuron loss, and motor deficits (Parkinson's pathology)
Siu et al. [44]	Polystyrene (PS-NPs)	100 pM (Low concentration)	Accelerated amyloid-beta (Aβ40/42) nucleation, promoting oligomer formation and neurotoxicity (Alzheimer's progression)
Gasper et al. [45]	Nano-microplastics (Polystyrene)	Environmental dose in drinking water [3]	Induced sex-dependent cognitive changes; ApoE4-risk mice (genetically predisposed to Alzheimer's) showed severe memory and spatial processing deficits
Liu et al. [46]	Anionic Polystyrene	Nanosize particles; In vitro & mouse models	Strong interaction with α-synuclein; significantly promoted protein clumping in neurons and subsequent dopamine loss
Jeong et al. [47]	Polystyrene (PS-MP)	0.3–56.5 particles/mm ³ (Ambient concentration, Inhalation)	Linked to chronic neuroinflammation and microglial activation, exacerbating pathways identical to PD-related neurodegeneration

decreasing particle size, polystyrene nanoplastics (~ 200 nm) exhibit higher trans endothelial transport and permeability than larger microplastics (1.0 µm) (p) Nanoplastics (< 1 µm) possess a high surface-area-to-volume ratio, allowing them to penetrate deep into organs (brain, liver, testes) and even enter individual cells via endocytosis [41, 42]. Microplastics ranging from 5.5 to 26.4 µm have been detected in the human olfactory bulb, suggesting their potential to induce neurotoxicity upon reaching brain tissue [12].

2.7 Primary experimental studies from 2024–2026 have increasingly linked microplastic (MP) and nanoplastic (NP).

In animals’ models, exposure microplastics has been linked to the acceleration of Alzheimer’s and Parkinson’s disease pathologies. Table 1 highlights key experimental studies on Alzheimer’s & Parkinson’s (2024–2025) in relation to microplastics exposure.

2.8 Limitations of current literature, gaps and recommendations for research & practice

2.8.1 Quantifying population exposure and access to the brain in humans

There is a deficiency in standardized methodologies for assessing chronic, lifelong exposure to microplastics and NPs through inhalation, ingestion, and dermal pathways, particularly among vulnerable populations and in diverse geographical contexts. Although current biomonitoring techniques (such as the detection of plastics in blood) show potential, they have not yet been standardized or validated for inferring neurotoxic risks [24]. Research conducted on animals and in vitro indicates that NPs have the potential to penetrate the blood–brain barrier; however, evidence about humans is still indirect, scarce, and methodologically diverse [24].

2.8.2 Particle characterization, mechanistic clarity and causal pathways

Most studies frequently employ polystyrene beads, which fail to represent the diversity found in the environment (including variations in polymer type, size range, shape, additives, and weathering). In the absence of realistic particles and appropriate doses, the translational relevance to neurodegeneration remains questionable [2]. Although microplastics can elicit inflammatory responses, the specificity of cell types (microglia, astrocytes, and neurons), the temporal dynamics, and the thresholds for chronic neuroinflammation in vivo remain inadequately understood [24].

2.8.3 Oxidative stress, mitochondrial dysfunction and Gut-brain axis

Suggested connections between these factors necessitate dose–response mapping and the demonstration of downstream neurodegenerative phenotypes in intact models that accurately represent human ageing and comorbid exposures [24]. Microplastics have the potential to modify gut physiology and microbiota; however, the causal relationships linking alterations in the gut to central proteinopathy, neuroinflammation, and clinical phenotypes (motor/cognitive) are predominantly hypothetical and have not been rigorously examined in longitudinal studies [24].

2.8.4 Prospective cohorts with exposure metrics, confounding factors, exposures, and clinical heterogeneity

Currently, there are no substantial, prospective human cohorts that incorporate validated measures of micro/nanoplastic exposure, neuroimaging, fluid biomarkers (such as p-tau and neurofilament light), and clinical endpoints related to Parkinson's or Alzheimer's diseases. The lack of temporality and measurable exposure impedes causal inference [24]. Air pollution, metals, and organic pollutants can accompany microplastics. Understanding their individual contributions and interactions regarding the risk of neurodegeneration represents a significant gap [24]. Parkinson's and Alzheimer's exhibit significant biological diversity. It is essential to conduct subtype-specific analyses (for instance amyloid-driven versus tau-driven AD) to identify variations in susceptibility to microplastic exposure [16, 24].

2.8.5 Methodology, standards gaps, and environmentally realistic dosing; Outcome measures, toxicological and translational models

There is currently no agreement on the protocols for the detection, quantification, and attribution of micro/nano plastics within human tissues (including the brain), nor is there a consensus on artefact control during sampling and imaging.[16, 24]. Numerous studies employ supra-physiological concentrations or monodisperse particles. It is essential to establish exposure paradigms that accurately reflect the complexity and chronic nature of real-world particles [16, 24]. The comparability of outcomes across studies is constrained by the variability in endpoints (cell viability versus behavior, or versus neuropathology). There is a notable absence of harmonised, tiered outcomes that range from molecular signatures to clinical phenotypes.[24, 48].

2.8.6 Ageing, kinetics, BBB and disease models

The majority of experiments are conducted using young animals and involve brief exposure periods. However, neurodegeneration develops over many years. There is a lack of ageing models that incorporate long-term, low-dose exposure [24, 48].

In-vitro BBB models require validation when compared to in vivo kinetics for a variety of particles and additives. This includes the examination of transport mechanisms (transcytosis versus paracellular transport for instance) as well as the dynamics of retention and clearance within the brain parenchyma [24, 48].

2.8.7 Additives, leachates and risk assessment, policy translation and Intervention evidence

The neurotoxic potential of plasticizers, flame retardants, and degradation byproducts may surpass that of the polymer itself. Their effects, both individually and in

combination, remain insufficiently characterized [2, 48]. Currently, there are no established threshold levels for neurotoxic risk associated with micro/nano plastics. It is unfeasible to determine safe exposure benchmarks without reliable, cross-scale dose–response data [24, 48]. There are also deficiencies of trials or quasi-experimental studies that demonstrate that decreasing microplastic exposure (whether dietary, occupational, or environmental) results in alterations to neurobiomarkers or decelerates the progression of neurovegetative conditions in populations affected by PD or AD [24, 48].

2.9 Gaps and future directions: research, practice and policy

2.9.1 Confirmed human biomonitoring and longitudinal studies

Human biomonitoring (HBM) research has underscored the extensive daily exposure to environmental chemicals. Certain chemicals are believed to play a role in negative health effects, including reproductive, neurological, and metabolic disorders, as well as various developmental and chronic impairments. A key aim of the H2020 European Human Biomonitoring Initiative (HBM4EU) was to create informative effect biomarkers that could be utilized in a more systematic and standardized manner across large-scale European HBM studies [49]. Standardized assays for the detection of micro/nanoplastics and additives in blood, cerebrospinal fluid, and postmortem brain should be implemented, along with appropriate contamination controls [24, 48]. It is essential to integrate exposure metrics with neuroimaging techniques (such as dopaminergic terminals and amyloid/tau PET), fluid biomarkers, and clinical outcomes [24].

Furthermore, independent laboratories ought to replicate research on particle–protein interactions (including α -synuclein, tau, and amyloid- β) under realistic conditions, while considering the effects of additives. Furthermore, there have been suggestions to employ both rodent and non-rodent models that accurately represent life-course exposures to particle mixtures reflective of environmental conditions, thus demonstrating the mechanistic validation of aging and chronic exposures [24, 48].

2.9.2 Human studies

Research involving direct human studies on the mechanistic connections between Parkinson's disease, Alzheimer's disease, and microplastics is notably scarce. The majority of the evidence is derived from laboratory and animal studies, while human data is predominantly confined to biomonitoring [50]. The investigation conducted by Zuri et al. emphasises the biomonitoring findings of microplastics present in human blood and stool, yet it highlights the lack of direct mechanistic studies involving humans [51]. Further, the research by Elif Gecegelen et al. in 2025 advocates prospective human cohort studies; at present, the evidence remains limited to indirect exposure estimates and biomonitoring [52].

2.9.3 Biomarker development

2.9.3.1 Neurodegenerative disease biomarkers (PD & AD) Despite consistent advancements in the field of neurodegenerative research, several significant gaps persist. The identification of reliable biomarkers for monitoring disease progression poses a challenge, especially when motor dysfunction overlaps with neuroinflammatory processes. The objective of elucidating genotype–phenotype relationships and creating effective, personalized interventions continues to be a considerable challenge within the realm of movement and motor neuron disorders. In the meantime, dependable techniques for the

early detection of subtle neuropathological alterations in Alzheimer's disease (AD) and associated dementias have not yet been established, thereby restricting opportunities for prompt therapeutic intervention [48, 53]. In another research done by Katherine Gong et al., the generated Novel plasma assays enabled accurate blood-based biomarkers for neurodegenerative diseases with minimally invasive options for clinical use. They identified proteins associated with disease status and AD-related phenotypes [54].

2.9.3.2 Microplastic exposure biomarkers Microplastics have been found in various biological samples, including blood, stool, placenta, and, most recently, in postmortem human brains [3, 5, 12]. Reviews indicate that oxidative stress markers (such as ROS and glutathione depletion), neuroinflammatory cytokines, and signatures of protein aggregation (including amyloid- β and α -synuclein) may serve as potential points of overlap [48, 55]. Although these pathways intersect with the biology of PD and AD, there currently exists no standardized biomarker assay that directly correlates microplastic burden with the progression of neurodegenerative diseases [24].

2.9.4 Regulation and public health

Microplastics are increasingly recognised as a worldwide pollutant that poses potential systemic health risks, including neurological hazards, thereby acknowledging microplastics as a significant public health issue [2, 56]. Regulatory and allied organizations such as the EU, WHO, as well as Centre for the Understanding of Sustainable Prosperity (CUSP) stress the necessity for risk assessment frameworks; however, they have yet to provide disease-specific guidelines for PD or AD, with regards to MPs and NPs [57, 58]. Also, Public health organizations advocate for the development of harmonized detection methods to accurately quantify exposure and establish relevant thresholds for chronic diseases [24, 58]. Further, regulatory frameworks have yet to incorporate these mechanistic overlaps of oxidative stress, neuroinflammation, and protein misfolding into their risk assessment processes [59].

3 Conclusion

The pervasive infiltration of microplastics (MPs) and nanoplastics (NPs) into the human body represents a critical environmental risk factor for neurodegenerative diseases. Evidence suggests these particles can breach the blood–brain barrier, triggering oxidative stress, neuroinflammation, and protein aggregation—pathologies central to both Alzheimer's and Parkinson's diseases.

While animal models provide strong biological plausibility for these mechanisms, direct mechanistic evidence in humans remains limited to biomonitoring data. Future research must prioritize standardized quantification methods and longitudinal studies to confirm causality and define risk thresholds. Concurrently, immediate public health policy is essential to mitigate the escalating global burden of plastic pollution and its potential neurological consequences.

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Author contributions

ENYN conceptualized the review, wrote the first draft, involved in methodology, data collection and editing of the final draft JK and JG were involved in methodology, data analysis, reviewing and editing the final draft SPS, SH and SYL and MO were involved in methodology, data collection and analysis DNDD, ADD, MA and HAA reviewed and edited the final draft All Authors read and approved the final version of the manuscript.

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Competing Interests

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References

1. Alziny N, Elgarhy FM, Mohamed AM, Shalaby HY, Nounou N, Soliman O, et al. The impact of microplastics in food and drugs on human health: a review of the MENA region. *Int J Environ Res Public Health*. 2025;22(3):380.
2. Campanale C, Massarelli C, Savino I, Locaputo V, Uricchio VF. A detailed review study on potential effects of microplastics and additives of concern on human health. *Int J Environ Res Public Health*. 2020;17(4):1212. <https://doi.org/10.3390/ijerph17041212>.
3. Leslie HA, van Velzen MJ, Brandsma SH, Vethaak AD, Garcia-Vallejo JJ, Lamoree MH. Discovery and quantification of plastic particle pollution in human blood. *Environ Int*. 2022;163:107199.
4. Jenner LC, Rotchell JM, Bennett RT, Cowen M, Tentzeris V, Sadofsky LR. Detection of microplastics in human lung tissue using FTIR spectroscopy. *Sci Total Environ*. 2022;831:154907.
5. Ragusa A, Svelato A, Santacroce C, Catalano P, Campolo V, English M, et al. Plasticenta: first evidence of microplastics in human placenta. *Environ Int*. 2021;146:106274.
6. Global, regional, and national burden of Parkinson's disease, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016 - PubMed [Internet]. [cited 2025 Nov 18]. Available from: <https://pubmed.ncbi.nlm.nih.gov/30287051/>
7. Alzheimer's Association. (2023). 2023 Alzheimer's disease facts and figures. *Alzheimer's & Dementia*, 19(4), 1598–1695
8. Windheim J, Colombo L, Battajni NC, Russo L, Cagnotto A, Diomedea L, et al. Micro- and nanoplastics' effects on protein folding and amyloidosis. *Int J Mol Sci*. 2022;23(18):10329. <https://doi.org/10.3390/ijms231810329>.
9. Mir RH, Sawhney G, Potttoo FH, Mohi-ud-din R, Madishetti S, Jachak SM, et al. Role of environmental pollutants in Alzheimer's disease: a review. *Environ Sci Pollut Res*. 2020;27(36):44724–42.
10. Thompson RC, Olsen Y, Mitchell RP, Davis A, Rowland SJ, John AWG, et al. Lost at sea: where is all the plastic? *Science*. 2004;304(5672):838.
11. Prata JC, da Costa JP, Lopes I, Duarte AC, Rocha-Santos T. Environmental exposure to microplastics: an overview on possible human health effects. *Sci Total Environ*. 2020;702:134455. <https://doi.org/10.1016/j.scitotenv.2019.134455>.
12. Amato-Lourenço LF, Dantas KC, Júnior GR, Paes VR, Ando RA, de Oliveira Freitas R, et al. Microplastics in the olfactory bulb of the human brain. *JAMA Netw Open*. 2024;7(9):e2440018. <https://doi.org/10.1001/jamanetworkopen.2024.40018>.
13. Daneman R, Prat A. The blood–brain barrier. *Cold Spring Harbor Perspect Biol*. 2015;7(1):a020412. <https://doi.org/10.1101/cshperspect.a020412>.
14. Patabendige A, Janigro D. The role of the blood–brain barrier during neurological disease and infection. *Biochem Soc Trans*. 2023;51(2):613–26.
15. Ma Y, Xu D, Wan Z, Wei Z, Chen Z, Wang Y, et al. Exposure to different surface-modified Polystyrene nanoparticles caused anxiety, depression, and social deficit in mice via damaging mitochondria in neurons. *Sci Total Environ*. 2024;919:170739.
16. Gou X, Fu Y, Li J, Xiang J, Yang M, Zhang Y. Impact of nanoplastics on Alzheimer's disease: enhanced amyloid- β peptide aggregation and augmented neurotoxicity. *J Hazard Mater*. 2024;465:133518.
17. Wang G, Lin Y, Shen H. Exposure to Polystyrene Microplastics Promotes the Progression of Cognitive Impairment in Alzheimer's Disease: Association with Induction of Microglial Pyroptosis. *Mol Neurobiol*. 2024;61(2):900–7.

18. Revel M, Châtel A, Mouneyrac C. Micro(nano)plastics: a threat to human health? *Curr Opin Environ Sci Health*. 2018;1:17–23.
19. Kwon W, Kim D, Kim HY, Jeong SW, Lee SG, Kim HC, et al. Microplastics cause self-assembling of α -synuclein and lead to neurotoxicity. *J Hazard Mater*. 2022;424(Pt C):127284.
20. Butterfield DA, Halliwell B. Oxidative stress, dysfunctional glucose metabolism and Alzheimer disease, and the therapeutic relevance of antioxidants. *Free Radic Biol Med*. 2019;144:143–56.
21. Kwon W, Kim D, Kim HY, Jeong SW, Lee SG, Kim HC, et al. Microplastics cause self-assembling of α -synuclein and lead to neurotoxicity. *J Hazard Mater*. 2022;424(Pt C):127284.
22. Windheim J, Colombo L, Battajni NC, Russo L, Cagnotto A, Diomedea L, et al. Micro- and nanoplastics' effects on protein folding and amyloidosis. *Int J Mol Sci*. 2022;23(18):10329.
23. Przystupski D, Kadej I, Kulbacka J. Nanoplastics as emerging mitochondrial toxins: implications for neurodegenerative diseases. *J Hazard Mater*. 2024;465:133481.
24. Weiss N, Vethaak AD. Microplastics and nanoplastics: an emerging threat for the human brain? *Environ Int*. 2021;155:106686.
25. Mehra S, Sahay S, Maji SK. α -Synuclein misfolding and aggregation: Implications in Parkinson's disease pathogenesis. *Biochim Biophys Acta BBA - Proteins Proteomics*. 2019;1867(10):890–908.
26. Sampson TR, Debelius JW, Thron T, Janssen R, Shastri GG, Huang ZE, et al. Gut microbiota regulate motor deficits and neuroinflammation in a model of Parkinson's disease. *Cell*. 2016;167(6):1469–80.
27. Liu Z, Sokratian A, Duda AM, Xu E, Stanhope C, Fu A, et al. Anionic nanoplastic contaminants promote Parkinson's disease-associated α -synuclein aggregation. *Sci Adv*. 2023;9(46):ead8716. <https://doi.org/10.1126/sciadv.adi8716>.
28. Siu ACW, Paudel KR, Singh G, Gupta G, Singh SK, Chellappan DK, et al. Do microplastics play a role in the pathogenesis of neurodegenerative diseases? Shared pathophysiological pathways for Alzheimer's and Parkinson's disease. *Mol Cell Biochem*. 2025. <https://doi.org/10.1007/s11010-025-05428-3>.
29. Hamilton BM, Jantunen LM, Rochman CM. Polyurethane microplastics and associated tris(chloropropyl)phosphate additives both affect development in larval fathead minnow **Pimephales promelas**. *Environ Toxicol Chem*. 2025;44(2):460–9. <https://doi.org/10.1093/etojnl/vgae040>.
30. Marfella R, Prattichizzo F, Sardù C, Fulgenzi G, Graciotti L, Spadoni T, et al. Microplastics and nanoplastics in atherosclerosis and cardiovascular events. *New England J Med*. 2024;390(10):900–10.
31. Chamas A, Moon H, Zheng J, Qiu Y, Tabassum T, Jang JH, et al. Degradation rates of plastics in the environment. *ACS Sustain Chem Eng*. 2020;8(9):3494–511.
32. Rochman CM, Browne MA, Halpern BS, Hentschel BT, Hoh E, Karapanagioti HK, et al. Classify plastic waste as hazardous. *Nature*. 2013;494(7436):169–71.
33. Galeski A. Strength and toughness of crystalline polymer systems. *Prog Polym Sci*. 2003;28(12):1643–99.
34. Khoironi A, Hadiyanto H, Anggoro S, Sudarno S. Evaluation of polypropylene plastic degradation and microplastic identification in sediments at Tambak Lorok coastal area, Semarang, Indonesia. *Mar Pollut Bull*. 2020;151(110868):1–10. <https://doi.org/10.1016/j.marpolbul.2019.110868>.
35. Sutar H, Sahoo PC, Sahu PS, Sahoo S, Murmu R, Swain S, et al. Mechanical, thermal and crystallization properties of polypropylene (PP) reinforced composites with high density polyethylene (HDPE) as matrix. *Mater Sci Appl*. 2018;9(5):502–15.
36. Shan S, Zhang Y, Zhao H, Zeng T, Zhao X. Polystyrene nanoplastics penetrate across the blood-brain barrier and induce activation of microglia in the brain of mice. *Chemosphere*. 2022;298:134261. <https://doi.org/10.1016/j.chemosphere.2022.134261>.
37. Zhu Y, Xu H, Yu C, Jiang W, Hou X, Ma M, et al. Polymers for the treatment of Alzheimer's disease. *Front Pharmacol*. 2025;16:1512941.
38. Cole M, Galloway TS. Ingestion of nanoplastics and microplastics by Pacific oyster larvae. *Environ Sci Technol*. 2015;49(24):14625–32.
39. Sommer C, Nguyen J, Menzel T, Prume JA, Ruckdäschel H, Koch M. Weathering-induced oxidation: an investigation of artificially aged polystyrene samples using Laser-induced Breakdown Spectroscopy. *Polym Test*. 2022;112:107623.
40. Zhu K, Jia H, Zhao S, Xia T, Guo X, Wang T, et al. High-efficiency photolysis of 2,2',4,4'-tetrahydroxybenzophenone mediated by weathered microplastics: role of reactive species and influential factors. *Water Res*. 2020;173:115574.
41. Vojnits K, De León A, Rathore H, Liao S, Zhao M, Gibon J, et al. ROS-dependent degeneration of human neurons induced by environmentally relevant levels of micro- and nanoplastics of diverse shapes and forms. *J Hazard Mater*. 2024;469:134017. <https://doi.org/10.1016/j.jhazmat.2024.134017>.
42. Wu M, Goh YL, Mowe MAD, Todd PA, Yeo DC. The effects of microplastics size and type on entrapment by freshwater macrophytes under vertical and lateral deposition. *J Limnol*. 2025. <https://doi.org/10.4081/jlimnol.2025.2218>.
43. Han Y, et al. Nanoplastics cause an increased risk of Parkinson's disease compared to microplastics at environmental exposure levels. *J Hazard Mater*. 2025;484:136974.
44. Siu ACW, Paudel KR, Singh G, et al. Do microplastics play a role in the pathogenesis of neurodegenerative diseases? Shared pathophysiological pathways for Alzheimer's and Parkinson's disease. *Mol Cell Biochem*. 2025. <https://doi.org/10.1007/s11010-025-05428-3>.
45. Gaspar L, Bartman S, Tobias-Wallingford H, Coppotelli G, Ross JM. Short-term exposure to polystyrene microplastics alters cognition, immune, and metabolic markers in an apolipoprotein E (APOE) genotype and sex-dependent manner. *Environ Res Commun*. 2025;7(8):085012.
46. Liu X, Zhao Y, Zhang T, et al. Chronic exposure to anionic polystyrene nanoplastics triggers cognitive decline and Alzheimer's-like neurotoxicity through the TLR2/MMP9 and p53/Bax/Bcl-2 pathways. *J Hazard Mater*. 2025;482:136125.
47. Jeong H, Shanmugiah J, Jang J, Lee DH, Choi J, Kim JS. Nanoplastics cause an increased risk of Parkinson's disease compared to microplastics at environmental exposure levels. *J Hazard Mater Adv*. 2025;20:100941. <https://doi.org/10.1016/j.hazadv.2025.100941>.
48. Prosperi M, Vethaak AD, Weiss N. (Nano)microplastics and neurodegeneration: navigating the transition from biological plausibility to clinical relevance. *Sci Total Environ*. 2026;967:178340.
49. Ganzleben C, Antignac JP, Barouki R, Castaño A, Fiddicke U, Garrido G, et al. Human biomonitoring as a tool to support chemicals regulation in the European Union. *Int J Hygiene Environ Health*. 2017;220(2 Pt A):142–56.

50. Baker TB, Smith SS, Bolt DM, Loh WY, Mermelstein R, Fiore MC, et al. Implementing clinical research using factorial designs: a primer. *Behav Ther.* 2017;48(4):567–80.
51. Zuri G, Karanasiou A, Lacorte S. Human biomonitoring of microplastics and health implications: a review. *Environ Res.* 2023;231(Pt 2):116172.
52. Gecegel E, Khan HA, Khan ST. A novel risk factor for dementia: chronic microplastic exposure. *Front Neurol.* 2025;16:1581109.
53. Tanaka M, Battaglia S, Liloia D. Navigating neurodegeneration: integrating biomarkers, neuroinflammation, and imaging in Parkinson's, Alzheimer's, and motor neuron disorders. *Biomedicines.* 2025;13(5):1045.
54. Gong K, Timsina J, Ali M, Chen Y, Liu M, Wang C, et al. High-sensitivity plasma proteomics reveals disease-specific signatures and predictive biomarkers of Alzheimer's disease phenotypes in a large mixed-dementia cohort. *Mol Neurodegener.* 2025;20(1):120.
55. Li Y, Tao L, Wang Q, Wang F, Li G, Song M. Potential health impact of microplastics: a review of environmental distribution, human exposure, and toxic effects. *Environ Health.* 2023;1(4):249–57.
56. Landrigan PJ, Raps H, Cropper M, Bald C, Brunner M, Canonizado EM, et al. The Minderoo-Monaco Commission on plastics and human health. *Ann Glob Health.* 2023;89(1):23. <https://doi.org/10.5334/aogh.4056>.
57. Hansen, S. F., Hernandez Bonilla, A., & Mata Marciano, V. (Eds.). (2024, April 23). CUSP policy brief 2–Micro and nanoplastics and public health: a reasonable concern. Zenodo. Available from: <https://zenodo.org/doi/10.5281/zenodo.11035612>
58. Wareham LK, Liddelow SA, Temple S, Benowitz LI, Di Polo A, Wellington C, et al. Solving neurodegeneration: common mechanisms and strategies for new treatments. *Mol Neurodegener.* 2022;17(1):23. <https://doi.org/10.1186/s13024-022-00524-0>.
59. Sakowski SA, Koubek EJ, Chen KS, Goutman SA, Feldman EL. Role of the exposome in neurodegenerative disease: recent insights and future directions. *Ann Neurol.* 2024;95(4):635–52.

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