REVIEW ARTICLE

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Uncovering the impact of nano- and microplastics on neurodegenerative diseases and strategies to mitigate their damage

Analiza wpływu nano- i mikroplastików na choroby neurodegeneracyjne oraz strategie łagodzenia ich toksyczności

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Abstract

Introduction. Microplastics (MPLs) are described as synthetic polymer particles with dimensions below 5 mm. Within this group, particles measuring less than 1 µm are referred to as nanoplastics (NPLs). These particles are extensively distributed in the environment and have been identified in human food sources as well as in both groundwater and tap water.

Objective. The aim of this review is to analyze the effect of micro- and nanoplastics on Alzheimer's and Parkinson's diseases. Furthermore, possible approaches to reducing the physiological effects of these particles are explored.

Brief description of the state of knowledge. Analysis suggests that the accumulation of nano- and microplastics may contribute to the progression of conditions such as Alzheimer's and Parkinson's diseases. Observed effects, such as neuroinflammation, mitochondrial dysfunction and blood-brain barrier disruption, suggest that neurotoxicity arises from multiple interacting pathways. Various bioactive substances have shown potential in reducing the neurotoxic impact of exposure to MNPs/NPLs. Melatonin, Fibroblast Growth Factor 1, and Camellia pollen have demonstrated promising effects in alleviating neurotoxicity. Furthermore, probiotics may play a role by restoring the natural balance of gut microbiota.

Summary. The reviewed studies suggest that microplastics and nanoplastics may have a significant impact on neurodegenerative diseases, particularly Alzheimer's and Parkinson's disease. Further investigations into pharmacological treatments and therapeutic approaches to counteract the toxic effects of these pollutants on the human body are essential. In the long term, such research will improve patients› quality of life and support the development of preventive strategies.

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Key words
neurotoxicity, neurodegeneration, microplastics, nanoplastics

Streszczenie

Wprowadzenie i cel pracy. Mikroplastiki (MPLs) to syntetyczne cząstki polimerowe o wymiarach poniżej 5 mm. Wśród nich cząstki o średnicy mniejszej niż 1 µm określane są jako nanoplastiki (NPLs). Cząstki te są powszechnie obecne w środowisku i zostały wykryte zarówno w żywności, jak i w wodzie gruntowej oraz wodzie z kranu. Celem niniejszego przeglądu jest analiza wpływu mikro- i nanoplastików na rozwój choroby Alzheimera i choroby Parkinsona. Ponadto omówiono możliwe strategie ograniczania fizjologicznych skutków działania tych cząstek.

Opis stanu wiedzy. Przeprowadzona analiza sugeruje, że akumulacja nano- i mikroplastików może przyczyniać się do progresji chorób neurodegeneracyjnych, takich jak choroba Alzheimera i choroba Parkinsona. Zaobserwowane efekty, takie jak neurozapalenie, dysfunkcja mitochondriów czy uszkodzenie bariery krew–mózg, wskazują na neurotoksyczność wynikającą z wielu współdziałających mechanizmów. Potencjał w łagodzeniu neurotoksycznego działania MNPs/NPLs mają różne substancje bioaktywne. Obiecujące działanie neuroprotekcyjne wykazały melatonina, czynnik wzrostu fibroblastów 1 (FGF1) oraz pyłek Camellia. Dodatkowo istotną rolę mogą odgrywać probiotyki, które przywracają naturalną równowagę mikroflory jelitowej.

Podsumowanie. Analizowane badania wskazują, że mikro- i nanoplastiki mogą mieć istotny wpływ na rozwój chorób neurodegeneracyjnych, a zwłaszcza choroby Alzheimera i choroby Parkinsona. Konieczne są dalsze badania nad leczeniem farmakologicznym oraz podejściami terapeutycznymi mającymi na celu przeciwdziałanie toksycznemu wpływowi tych zanieczyszczeń na organizm człowieka. W dłuższej perspektywie badania te mogą przyczynić się do poprawy

jakości życia pacjentów i opracowania skutecznych strategii prewencyjnych.

Słowa kluczowe
neurotoksyczność, neurodegeneracja, mikroplastiki, nanoplastiki

INTRODUCTION

There is no universally accepted definition of microplastics and nanoplastics. These terms are used to describe a broad spectrum of plastic particles with varying densities, diverse in form, size, and chemical arrangement including, for example, polyvinyl chloride (PVC), polystyrene (PS) and polyethylene (PE). Microplastics (MPLs) are generally described as plastic particles smaller than 5 mm. A fraction of these, smaller than 1 µm, is categorized as nanoplastics (NPLs) [1, 2, 3, 4]. These particles are extensively distributed in the environment, and have been detected in human food, and in both ground and drinking water [1, 2, 4]. Many studies suggest that humans encounter MNPs/NPLs mainly through swallowing them via the digestive tract, absorbing them through the skin, and breathing them in through the lungs [4, 5]. There are multiple pathways through which these particles can enter the human body, and similarly, there are various methods for biomonitoring their presence within the organism. Examples of biomonitoring approaches for micro- and nanoplastics are summarized in Table 1.

The cited studies employed meticulous procedures aimed at minimizing the risk of microplastic contamination. The impact of MNPs/NPLs on human health remains insufficiently explored. This paper focuses on summarizing current findings on neurotoxicity and their potential contribution to neurodegenerative disorders.

OBJECTIVE

The aim of the review is to examine the influence of micro- and nanoplastics on the two most widespread neurodegenerative disorders – Alzheimer's and Parkinson's diseases. Additionally, possible strategies aimed at mitigating the physiological consequences of these particles are discuss.

MATERIALS AND METHOD

The selection of materials for th study was based on a search conducted through open-access platform PubMed. A literature search was carried out 12 March 2025 with the search terms: 'microplastic' and 'Parkinson' – 23 results; 'microplastic' and 'Alzheimer disease' – 15 results; "(microplastic) AND (nanoplastic) AND (neurodegenerative)" – 11 results, 'microplastic', 'biomonitoring' and 'methods' – 270 results. The search focused on articles published between 2020–2025. To extend the scope, the reference sections of the retrieved articles were also reviewed, leading to the inclusion of other relevant works. This process resulted in the identification of 31 articles. The eligibility criteria included the type of publication, such as original research, reviews, systematic reviews, case reports, and observational studies, as well as being published in English and being thematically related to the subject of the review. The final manuscript underwent a thorough review, verification, and revision process by all contributing authors. Any unclear content was addressed through group discussion and modified for clarity and precision.

DESCRIPTION OF THE STATE OF KNOWLEDGE

Parkinson's disease (PD). A multifaceted neurodegenerative condition with a complex pathogenesis and a wide spectrum of clinical expressions. This progressively increasing disorder is mainly identified based on bradykinesia, often accompanied by rigidity and rest tremor. Nevertheless, the clinical presentation is broad and also involves multiple non-motor manifestations. From a pathological perspective, PD is identified by the presence of α-synuclein aggregates in Lewy bodies and neurites, which results in the degeneration of dopaminergic neurons [13, 14]. Emerging evidence points to the gut-brain axis, a reciprocal communication system linking the brain and the digestive system, which could

Table 1. Basic forms of biomonitoring microplastics in the human body

Study	Form of biomonitoring	Description
Leslie et al. [6]	Human blood	Four widely-used plastic polymers were found in blood. The most common were polyethylene terephthalate, polyethylene, and styrene-based polymers. Average plastic particle concentration – 1.6 µg/ml.
L.C. Jenner et al. [7]	Human lung tissue	Microplastics were found in 11 out of 13 human lung tissue samples, (39 in total). The most common types were polypropylene (23%), polyethylene terephthalate (18%), and resin (15%). Average concentration – 0.69 ± 0.84 MP/g, after background correction.
Pironti et al. [8]	Human urine	Urine from 6 volunteers (3 men, 3 women) was analyzed using Raman microspectroscopy, detecting microplastics (4–15 µm) in 4 samples: vinyl acetate and polyvinyl chloride (1 woman), polypropylene and polyethylene (3 men).
N. Zhang et al. [9]	Human faeces	Of 24 recruited male students (18–25 years, Beijing), 24 finished the study. Microplastics (MPs) were detected in 23 faecal samples (95.8%). Polypropylene was the most common.
Ragusa et al. [10]	Human breast milk	Microplastics were found in 26 of 34 breast milk samples, with polyethylene (38%) and polyvinyl chloride (21%) as the most common types.
Braun et al. [11]	Human placenta and meconium	Microplastics > 50 µm were detected in placenta and meconium from 2 Caesarean sections, polyethylene and polypropylene were in the peripheral placenta, polyethylene and polyurethane in the central area.
Q. Zhao et al. [12]	Human testis and semen	6 testis and 30 semen samples were analyzed for microplastics (MPs). Both semen and testis samples contained MPs. Polystyrene (67.7%) dominated in testis, whereas polyethylene and polyvinyl chloride were more common in semen.

be involved in Parkinson's disease [14, 15]. This axis is also of significance in the context of exposure to microplastics and nanoplastic. Liang et al. examined how the murine digestive system influences the transformation, distribution, and toxicity of microplastics polylactic acid (PLA) polymer and oligomer. The 28-day oral gavage experiment in mice showed that microplastics PLA polymer degrade partially, forming oligomer nanoplastics that enhance bioavailability and intensifying overall toxic impacts. Furthermore, the study underscores common mechanisms of toxicity linked to Parkinson's disease-like neurotoxicity, induced by both microplastics PLA polymer and oligomer, with an increase in MICU3 (Mitochondrial Calcium Uptake 3 protein) expression in the midbrain, leading to mitochondrial calcium overload in neurons [16].

A few papers have also been published discussing the direct impact of micro- and nanoplastics on the α-synuclein protein [17, 18, 19]. Liu et al. found that anionic nanoplastics promote α-synuclein fibril formation by interacting with its amphipathic and non-amyloid segments and disrupting lysosomal function. They enter neurons via clathrin-mediated endocytosis. In mice, they accelerate pathology spread, particularly in dopaminergic neurons of the substantia nigra [17]. In another paper, Liang et al., using ESI-TOF-MS (electrospray ionization time of flight mass spectrometry), show that polystyrene nanoplastics promote Non-Amyloid Component core (NACore) oligomerization and increase its toxicity in microglial cells. Simulations suggest hydrophobic interactions drive this process, and zebrafish studies show developmental impairments [18]. It is worth mentioning the study by Ghosal et al., which explored the binding interactions between human α-synuclein and microplastics (polyethylene, polyvinyl chloride, and polystyrene). Multi-spectroscopic techniques, such as UV-vis, circular dichroism and Fourier transform infrared (FTIR), demonstrated structural modifications in α-synuclein. The study indicates that polystyrene microplastics (particularly at 100 nm) are the most potent in triggering amyloidogenic oligomerization [19]. These findings link nanoplastics to amyloidogenesis and potential neurological damage [17, 18, 19]. The detrimental effects of polystyrene nanoplastics have also been highlighted by Huang et al. They demonstrated that these particles impair mitochondrial activity, possibly by affecting complex I (CI), which sets off mitophagy through the AMPK/ULK1 signalling route, culminating in the loss of dopaminergic neurons, a mechanism potentially linked to Parkinson's disease-like neurodegeneration [20]. Other research has also indicated the possible neurotoxicity of polystyrene nanoplastics [21].

Alzheimer's disease. Another disease that may be affected by exposure to micro- and nanoplastics is Alzheimer›s disease. Alzheimer's disease is the predominant neurodegenerative disorder responsible for the majority of dementia cases (50–70%), with risk increasing significantly after the age of 65. This disorder is linked to structural alterations in the brain, such as the accumulation of fibrous amyloid-beta plaques. These processes are associated with excessive tau protein phosphorylation [22, 23].

Exposure to micro- and nanoplastics may contribute to an increased risk of Alzheimer›s disease through various molecular pathways. One of which is the acceleration of amyloid-β (Aβ) peptide aggregation [24, 25]. Gou et al. report that polystyrene (PS) nanoplastics accelerate the nucleation of amyloid-β subtypes (Aβ40 and Aβ42), enhancing the generation of toxic oligomers and causing neurotoxic effects. While PS nanoparticles alone do not display neurotoxicity, their hydrophobic surface promotes Aβ monomer interactions, leading to oxidative stress, membrane disruption, and dysregulated Ca^{2+} homeostasis [24]. A similar study was conducted by Gabbrielli et al., who investigated several types of nanoparticles and reported that the interaction between plastic nanoparticles and β-amyloid fibrils depends on nanoparticle type and surface charge, with polystyrene nanoparticles potentially accelerating fibril formation, while polyethylene and polypropylene can hinder fibril growth, potentially increasing toxic oligomer accumulation [25]. Additional studies have also examined the impact of polystyrene nanoplastics, with insulin acting as a model to analyze the protein fibrillation pathway [26].

Another mechanism by which exposure to micro- and nanoplastics may induce neurotoxicity and contribute to Alzheimer›s disease is microglial pyroptosis, an inflammatory type of cell death. A study by Wang et al. in mice showed a connection between this mechanism and polystyrene microplastics [27].

Alleviation of micro- and nanoplastic-induced effects. In light of the documented toxic effects of micro- and nanoplastics in many studies, an increasing number of investigations are exploring methods to reduce exposure and mitigate the adverse biological effects in the human organism.

Probiotics are considered in both prophylaxis and therapy of MNPs/NPLs-induced effects. They can alleviate gut dysbiosis and intestinal permeability, decrease proinflammatory markers, and stop the immune system from overreacting [28]. Qiao et al. found that exposure to polystyrene micro- and nanoplastics caused gut injury and microbiota imbalance in mice. The toxicity was highest for aminated polystyrene (PS-NH2). The study emphasizes how gut microbiota plays a key role in the toxicity of MNPs/NPLs and the potential and their possible health impacts [29]. Introducing probiotics could possibly mitigate the harmful effects of these substances in humans [28].

Melatonin is a substance investigated Huang et al. who focused on the role of mitophagy in polystyrene nanoplastics (PS-NPs) induced mitochondrial impairment and evaluated the possible protective effects of melatonin in vitro. The researchers assessed the impact of melatonin treatment (10 mg/kg/d, administered intraperitoneally) to mice at a dose of 250 mg/kg/d of PS-NPs over a period of 28 days, confirming its effectiveness. By modulating autophagy of mitochondria, melatonin helped improve motor function and counteract mitochondrial damage [20].

The beneficial properties of Camellia pollen as a functional food were also highlighted. Bai et al. exposed that aminomodified polystyrene nanoplastics (APS-NPs) impaired occluding junctions and the blood-brain barrier in mice, leading to neuronal damage and Alzheimer-like symptoms. Camellia pollen alleviated these effects by preventing neurotoxicity, protecting the blood-brain barrier, inhibiting pathological Tau acetylation through modulation of deacetylase Sirtuin 1 and the acetyltransferase CBP, and reducing apoptosis of neurons via regulation of the p53/Bax/Bcl-2 pathway. The study highlights potential of Camellia pollen in treating neurodegenerative diseases [30].

The last molecule discussed with potential protective effects is Fibroblast Growth Factor 1 (FGF1). The neurotoxicity of polystyrene nanoplastics (PS-NPs) was examined by Qian et al., who found that PS-NPs interaction stimulated lipophagy-mediated lipolysis, driving neuroinflammation through mediators such as 2-arachidonoylglycerol and prostaglandin E2. Therapy with fibroblast growth factor (FGF1) reduced lipid accumulation and inflammation, improving cognitive function in mice, which suggests FGF1 as a potential neuroprotective agent against polystyrene nanoplastics [31].

Beyond the examples discussed above, it is worth considering the potential application of other compounds, including anti-inflammatory agents, which may offer protection against the harmful effects of micro- and nanoplastics by inhibiting neuroinflammatory pathways. However, since there are no experimental data confirming their effectiveness in this area, more focused research is needed to better understand their potential.

SUMMARY

The findings provide critical insights into the role of nano- and microplastics in neurodegenerative diseases. Analysis suggests that the accumulation of these particles in neural tissues leads to cellular disruptions, inflammation, and neuronal damage, and contribute to the progression of such conditions as Alzheimer's and Parkinson's diseases. The observed effects of neuroinflammation, mitochondrial dysfunction, oxidative stress, and blood-brain barrier disruption, suggest that neurotoxicity arises from multiple interacting pathways. The most frequently examined substances were polystyrene (PS) microplastics and nanoplastics. Therefore, the neurotoxic potential of these substances has been most thoroughly described [19, 20, 21, 24, 25, 27, 29, 30, 31].

Various bioactive substances have shown potential in reducing the neurotoxic impact of exposure to MNPs/NPLs. Melatonin, Fibroblast Growth Factor 1 and Camellia pollen, have demonstrated promising effects in alleviating neurotoxicity [20, 30, 31]. Furthermore, probiotics may play a role by restoring the natural balance of gut microbiota, which may mitigate potential the neurological damage caused by microplastics and nanoplastics [28, 29]. However, more research is needed to discover and characterize additional protective compounds with similar or greater neuroprotective capabilities.

While micro- and nanoplastic research is expanding, the majority of experimental work to date has involved non-human subjects, such as mice [16, 17, 20, 27, 29, 30, 31]. The translation of these findings to human health remains a critical challenge. Future research is required to validate these findings in human-relevant models.

CONCLUSIONS

Microplastics and nanoplastics may have a significant impact on neurodegenerative disorders, particularly Alzheimer's and Parkinson's disease. Given the growing presence of plastic pollutants in the environment, further investigations into pharmacological treatments and therapeutic approaches to counteract the toxic effects of these pollutants on the human body are essential. In the long term, such research will improve the quality of life of patients, and enable the development of strategies for prevention and risk mitigation strategies.

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