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Microplastic Particles and Neural Diseases: Mechanistic Links Between Environmental Exposure and Nervous System Dysfunction

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Abstract. Microplastic particles are widespread environmental contaminants with increasing concern for human neural health. Although direct causal links between microplastic exposure and specific neural diseases remain unproven, experimental evidence suggests that microplastics and nanoplastics may affect the nervous system through multiple interconnected pathways. After ingestion or inhalation, particles may interact with epithelial barriers, immune cells, vascular systems, and the gut microbiota, leading to systemic inflammation and barrier dysfunction that can influence neural homeostasis. At the cellular level, proposed mechanisms include oxidative stress, mitochondrial dysfunction, neuroinflammation, blood–brain barrier disruption, impaired proteostasis, altered neurotransmission, and synaptic dysfunction. These processes overlap with pathological features of neurodevelopmental disorders, neurodegenerative diseases, and cognitive or neuropsychiatric dysfunction. However, current studies are limited by inconsistent particle characterization, simplified exposure models, unrealistic doses, and limited human evidence. Future research should use standardized, disease-oriented, and physiologically relevant models to clarify whether microplastics contribute meaningfully to neural disease vulnerability or progression.

Keywords: Microplastics; Neurotoxicity; Neural diseases.

1. Introduction

Microplastics particles have become emerging issues in the environmental and biomedical fields because of their occurrence in water, air, soil, foods and in human biosamples [1]. Generally, microplastics are defined as plastic particles smaller than 5 mm in size; these can be further degraded into nanoplastics, which have a smaller size, larger surface area and, potentially, larger biological reactivity [2].

Human exposure to microplastics is continuous and occurs, for example, by ingestion, inhalation and, possibly, other routes; there are worries about their accumulation, distribution in organs or tissues and long-term effects for health [3]. Early studies of microplastics mainly addressed ecological toxicity and outcomes in the gastrointestinal tract and metabolism. Increasing interest lies in the nervous system, in which even minor disruptions in various forms of barrier function, immune responses, mitochondrial functions or synaptic activities can lead to functional disorder [4].

Associated possible impacts of microplastic exposure on neural diseases is an emerging and poorly understood area of research. Microplastics have so far not been established as direct causes of particular neurological diseases; however, experimental studies hint at interactions between microplastics and many of the processes underlying neural diseases, which are known to be involved in neurological dysfunction, such as oxidative damage, neuroinflammation, blood–brain barrier damage, mitochondrial dysfunction, altered neurotransmission and diseased protein homeostasis [5-7]. Some of these processes are closely related to neurodevelopmental disorders, neurodegenerative diseases and cognitive or mental dysfunctions. Therefore, this Review summarizes current knowledge on microplastic exposure, access to the nervous system, neurotoxicity mechanisms and disease-relevant consequences, and emphasizes the major limitations that exist that must be overcome for strong conclusions to be reached.

2. Exposure Routes, Biological Distribution, and Access to the Nervous System

Human exposure to microplastic particles is usually through ingestion and inhalation. Particles are found in food and drink water, air dust and other environmental sources. After entering into the body, the biology of microplastic particles is influenced by the particle size and shape, polymer composition, surface charge, additives, etc., and the biomolecules attached onto the surface. Larger microplastics, especially tens of micrometers to millimeter size microplastics, exist or are more easily eliminated on epithelium; small microplastics and nanoplastics are more easily to react with the epithelium, immune cells and biological barriers. Thus, exposure route is important but the most important factor for human neural health is whether these microplastic particles or inflammatory reactions elicited by these microplastics can enter or affect the nervous system [8-10].

Microplastics would have to bypass a number of biological barriers to affect the nervous system directly. The blood–brain barrier is the best-known one because it tightly controls the exchange of molecules and cells from blood into the brain parenchyma. Under normal conditions, this blood–brain barrier prevents the invasion of many circulating particles and inflammatory mediators by specialised endothelial cells, tight junctions, pericytes and astrocyte endfeet. However, microplastic exposure could also affect the nervous system indirectly by generating systemic inflammation, oxidative stress, endothelial dysfunction or gut microbiota dysbiosis, all impacting on the state of homeostasis of the nervous system without the need to accumulate large amounts of particles in the brain. Thus, nervous system toxicity should not be interpreted by direct penetration of particles into the body, but by barrier and immune-mediated communication between distal organs and the brain [11] [12] as well.

Another route to the brain from microplastics that is of biologically relevant indirect relevance is the gut–brain axis. Microplastics can interact with the intestinal epithelium, mucus layer, immune cells and gut microbiota, potentially shifting microbial composition, gut permeability and local inflammatory signalling. These downstream effects, in turn, can impact the brain by circulating cytokines, microbial metabolites, vagal signalling and immune–endocrine signalling. This route is of special relevance as the gut is likely to be one of the primary sites of chronic microplastic exposure even if their direct translocation into the brain is limited. Gut-mediated inflammation and microbiota-mediated metabolic shifts might, therefore, be major drivers through which microplastics lead to neurological dysfunction [12].

Overall, whether microplastics are able to impact the nervous system depends on both direct and indirect routes. Direct effects might arise if small enough (e.g., nanoplastics) microplastics cross biological barriers and accumulate in the nervous system. However, indirect effects will be at least as important, and include gut dysbiosis, pulmonary inflammation, systemic immune activation, oxidative stress, vascular and epithelial barrier perturbations. These may ultimately culminate in the nervous system even without extensive microplastic penetration into the brain. Evaluating microplastic-induced neurotoxicity thus requires attention not only to microplastic biodistribution, but also to the wider biological reactions that may emanate from peripheral exposure sites.

3. Cellular and Molecular Mechanisms of Microplastic-Induced Neurotoxicity

Most studies investigating microplastic-induced neurotoxicity are unlikely to reflect one phenomenon. Rather, available data suggest that microplastics and nanoplastics are disturbing neural functioning via several connected cellular and molecular routes: oxidative stress, mitochondrial failure, neuroinflammation, disruption of the blood–brain barrier, altered neurotransmission, impairment of synapses and altered protein homeostasis [5, 13]. Many of these mechanisms are not specific to microplastic exposure, but represent common pathways present in different neural disorders. Hence, the neurotoxic relevance of microplastics should be understood not only by asking whether particles have 'directly' built up in the brain, but also how microplastics perturb cellular stress pathways, glial activation, communication between neurons and integrity of the barrier.

3.1. Oxidative Stress and Mitochondrial Dysfunction

Although oxidative stress is one of the most proposed mechanisms that link microplastic exposure to neural injury, microplastics can drive formation of reactive oxygen species by directly interacting with cells, surface chemical compounds, surface adsorbed environmental compounds or inflammatory responses in peripheral tissues. Neurons are particularly sensitive to oxidative stress because of their metabolic demands, high lipid-rich content of membranes and poor ability to regenerate. Excessive reactive oxygen species can damage lipids, proteins and nucleic acids, compromising the integrity of membranes, enzymes and cellular signalling. In neural tissue, such impacts could affect the survival of neurons, axon function and synaptic activity, establishing a general mechanism by which microplastic exposure could explain neural disease effects [14].

Strong oxidative stress and mitochondrial dysfunction are closely related and are likely to further promote microplastic-induced neurotoxicity. Because neurons depend on mitochondrial oxidative phosphorylation to support maintenance of ion gradients, synaptic activities, axonal transport and neurotransmitter recycling, mild mitochondrial impairment may induce several functional impairments. Microplastic- and nanoplastic-induced mitochondrial dysfunction can affect mitochondrial membrane potential, diminish mitochondrial ATP production, change the concentration of Ca^{2+} and activate apoptosis signalling pathway [15]. Thus, a vicious cycle can occur when mitochondrial damage leads to production of reactive oxygen species (ROS), and oxidative stress worsens mitochondrial dysfunction. This sort of mitochondrial vulnerability is particularly pertinent to neurodegenerative disorders, whose characteristic pathological features are impaired energy metabolism and an eventual accumulation of oxidative damage [16].

3.2. Neuroinflammation and Glial Activation

Another important pathway through which microplastic exposure could affect neural function is via neuroinflammation. In the CNS, microglia are a group of immune cells residing within the brain that regularly monitor the local microenvironment and react to injury, infections or abnormal signal molecules. Microplastic exposure can induce inflammatory signalling directly if the particles or the particle-bound molecules make it to neural tissue, or indirectly via the systemic cytokines produced in the peripheral organs, such as the gut or lung, for instance [14]. Once activated, microglia can secrete pro-inflammatory cytokines, oxygen radicals, nitric oxide and other products that change neuronal survival and neuronal synaptic activity. Acute activation of glial cells is indeed protective, but persistent or uncontrolled activation could induce a state of chronic inflammation, leading to neuronal damage and progress towards chronic neural dysfunction [17].

Astrocytes might also contribute to microplastic-associated neuroinflammation. In physiological conditions, astrocytes clear neurotransmitters, maintain ion homeostasis, provide metabolic support, support synapse development and maintain the blood–brain barrier. In response to inflammatory or oxidative stimuli, however, astrocytes can become reactive, expressing cytokines, providing inadequate metabolic support and altering the extracellular homeostasis, impairing neuronal communication and causing neuronal vulnerability to injury. As astrocytes can communicate closely with neurons, endothelial cells and microglia, astrocyte activation can also be an important link in the bridge between barrier damage, inflammatory signalling and synaptic dysfunction after exposure to microplastics [18].

3.3. Blood–Brain Barrier Disruption

Blood–brain barrier disruption represents an important pathway by which microplastic exposure can enhance neural injury. Microplastics and nanoplastics can compromise blood–brain barrier integrity through endothelial oxidative stress, inflammatory signalling, disruption of tight junctions and vascular leakiness. Once blood–brain barrier function is compromised, circulating cytokines, immune cells, toxic molecules or even smaller particles can more readily penetrate neural tissue, contributing to neuroinflammation and neuronal stress [19].

Barrier dysfunction also produces a feedback loop with neuroinflammation. Peripheral inflammation may dampen endothelial tight junction function and boost the blood–brain barrier disruption. Increased disruption then permits additional inflammatory mediators to access the neural environment, which can lead to further activation of microglia and astrocytes and production of pro-inflammatory mediators, such as cytokines and reactive oxygen species, that act to exacerbate endothelial dysfunction. In this setting, the blood–brain barrier is not only a passive target of microplastic-associated toxicity but also an active regulator of disease. This is pertinent to chronic neural diseases for which sustained low-grade inflammation and vascular dysfunction progressively interfere with the neural homeostasis [19].

4. Potential Links Between Microplastics and Neural Diseases

Present evidence does not allow us to make a simple conclusion of microplastic causing a specific neural disease directly. Nevertheless, microplastic exposure could interact with a number of pathologies that are already linked to a neurological dysfunction. Oxidative stress, neuroinflammation, mitochondrial damage, barrier impairment, synaptic dysfunction, dysregulated proteostasis are shared features of many neurological diseases. Thus, microplastics disease relevance must be understood in this context through shared mechanisms rather than single-disease process pathways. In this view, microplastics can be seen as an environmental stressor that increases neuronal vulnerability in susceptible periods, such as development, ageing or disease before onset conditions [4].

That the developing nervous system is especially vulnerable to stress from microplastics may be explained because neurogenesis, neuronal migration, synaptogenesis, myelination, refinement of circuit wiring happen during certain time windows. In early development, biological barriers and detoxification systems can also be less mature, making early development sensitive to inflammatory, oxidative or metabolic stresses. If microplastics or microplastic-induced signals from the periphery interfere with these development processes, they will interfere with normal developmental neuronal circuit wiring and long-term neurofunctions. In experimental studies, microplastic exposure during early life has shown effects on the development of behaviours, learning and memory, and anxiety-like effects. Such observation in animal models cannot readily be translated to humans' neurodevelopmental disorders. Thus, microplastics should be considered as environmental modifiers for neurodevelopmental risk, not the cause of a specific neurodevelopmental disorder per se [20].

Microplastics may also be relevant to neurodegenerative diseases because their proposed neurotoxic mechanisms are similar to several disease processes involved in age-related loss of brain function. Neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease and others are associated with chronic oxidative stress, mitochondrial dysfunction, neuroinflammation, proteostasis defect, synaptic failure and progressive loss of neurons. Exposure to microplastics could further exacerbate these processes through increasing cellular stress, initiating glial inflammatory responses, affecting barrier function or impeding protein degradation. For instance, oxidative and inflammatory stress may enhance neuronal sensitivity, mitochondrial impairment may dampen the ability of neurons to support long-term demand of energy-intensive neuron functions (for example, for axon transport or synaptic transmission). However, experimental data are still sparse and there is no clear experimental or direct causative relation between exposure to microplastics and the progression of a human neurodegenerative disease [21] .

For Parkinson's disease-related pathways, special attention has been given to dopaminergic vulnerability and α-synuclein pathology. Dopaminergic neurons are very sensitive to mitochondrial damage, oxidative stress and inflammatory injury, which are also found after microplastic exposure in experimental studies. In addition, the gastrointestinal tract may be important because of gut inflammation, microbiota alterations and the gut–brain axis signalling, all of which are increasing in relevance to Parkinson's disease pathophysiology. The exposure to microplastics could, in theory, aggravate these peripheral and neural stress factors even under chronic exposure, but this link should

be stated cautiously, because most of the available evidence have mainly mechanistic plausibility, and not direct pathogenesis evidence for disease [21].

But for Alzheimer's disease-related exposure to microplastics, it might be relevant because of their possible impact on oxidative stress, neuroinflammation, blood–brain barrier function and protein homeostasis. These processes are related to amyloid-β accumulation, tau, synaptic dysfunction and cognitive decline. Microplastic-induced inflammation or mitochondrial damage can induce greater neuronal stress and impair clearance machinery, which makes the cells much more 'permissive' to neurodegeneration. It is currently unclear whether microplastics stimulate amyloid-β or tau aggregation directly, so microplastic involvement in Alzheimer's disease should be discussed as an indirect and disease-modifying effect rather than a causal one [22].

More indirect outcomes such as cognitive and neuropsychiatric changes may be functional effects of microplastic-induced neural stress. Learning and memory, mood control and anxiety-related behaviours require a coordinated input from synapses, neurotransmitters, the mitochondrial energy supply, glia and other cell types. Exposure to microplastics can affect these coordinated processes through oxidative stress, neuroinflammation, alteration of neurotransmitter activity, gut–brain axis interference and disruption of blood–brain barrier integrity. Experimental animal studies have reported behavioural changes such as impairments in memory performance, reduced exploratory behaviours, anxiety-like phenotype and others after exposure to microplastics. However, such outcomes of behaviours are highly dependent on particle type, dose, exposure route and animal model, and cannot be directly attributed to human psychiatric diseases [23]. Another relevant consideration is that cognitive and neuropsychiatric outcomes might occur without considerable neuronal death. Effects of synaptic dysfunction, glial reactivity, neurotransmitter balance and low-grade inflammation can alter neural circuit activity long before any visible structural damage does. This is particularly relevant to chronic low-dose exposure to cumulative subtle functional changes. In this way, microplastics behave like persistent environmental insults that impair neural resilience, not as direct insults leading to defined psychiatric disease. Future studies will therefore have to link findings at behavioural level with cell-specific neural mechanisms, concentrations of relevant particles in the exposure and long-term functional implicationss [23].

5. Current Evidence, Limitations, and Future Directions

Current understanding of microplastic-related neurotoxicity is based largely on experimental models, such as neural cell cultures, models of the blood–brain barrier, animal studies and fewer organoid or advanced in vitro models. Cell models are of use for investigations of direct effects on neurons, astrocytes, microglia or endothelial cells, mostly in the context of oxidative stress, inflammatory signalling, mitochondrial function and cell viability. However, these models also oversimplify complex interactions among peripheral exposure sites, systemic immune response, vascular barriers, brain tissue. In vitro studies are therefore valuable for the mechanistic understanding but cannot fully reflect how microplastics are absorbed, distributed, transformed and eliminated in vivo.

Animal models provide important evidence for a link between exposure to microplastics and neural and behavioural outcomes. Rodent, zebrafish and other in vivo animal models allow researchers to measure particle biodistribution, blood–brain barrier integrity, neuroinflammation, changes in neurotansmitters and behavioural performance in a whole animal setting. These studies are particularly relevant to study developmental exposure, chronic exposure, effects of exposure via the gut–brain axis, and so on. However, their interpretation is often confounded by differences in exposure dose, particle size, polymer type, exposure route and duration. Many studies examine particle concentrations that are not likely to represent realistic human exposures and it is difficult to assess whether the observed neural effects are ecologically or clinically relevant [24].

Human evidence is the weakest part of the current field. Microplastics have been found in several human biological samples, but evidence that microplastic burden causes neurological or psychiatric disease outcomes is still very scarce. Such gaps reflect many challenges, such as how to accurately

measure microplastics in human tissues, how to distinguish internal exposure from sample contamination, and how to link particle exposure to later disease progression. In addition, the neural diseases mentioned usually result from complex interactions between genetics, ageing, lifestyle, inflammation, vascular dysfunction and environmental exposures. Future human studies will have to adopt consistent detection methods, careful sampling procedures and rigorous epidemiological designs to assess whether exposure to microplastics affects measurable neural risk [25].

Another strong limitation of studies across the field is the lack of a standardized approach to particle characterisation. The toxicity of microplastic particles depends strongly on particle size, particle shape, polymer type, particle surface charge, particle age, polymer additives and pollutants attached as adsorbed biomolecules or pollutants. However, several studies only employ available (commercial) spherical polystyrene particles, which are unlikely to represent the heterogeneous, weathered and chemically complex particles present in the environment [26]. In addition, distinctions between microplastic and nanoplastic differences are not always properly distinguished, and can confound comparisons between studies. Future work should, therefore, report particle properties in more detail and use particle exposure models that are closer to the environmental and biological complexity encountered.

Advanced experimental platforms can help overcome these limitations. Human iPSC-derived neural cells, brain organoids, blood–brain barrier-on-chip and gut–brain axis models can offer more physiologically relevant tools to explore the effects of microplastics-associated neurotoxicity [27]. These platforms allow researchers to assess cell-type-specific vulnerability, human-relevant barrier responses, and interactions among neurons, glia, endothelial cells and peripheral inflammatory signals. They can also be combined with transcriptomic, proteomic, metabolomic and high-content imaging studies to probe early molecular signatures of exposure to microplastics. However, they have to be complemented with carefully controlled in vivo and epidemiological studies because no single model can fully recapitulate the complexity of chronic human exposure and neural disease progression in humans.

Future studies need to further focus on chronic, low-dose and mixture-based exposures. In the real world, people will not just be exposed to a single uniform type of plastic particle; exposure involves mixtures of different polymers, sizes, shapes, additives, plastic degradation products and pollutant add-on contaminants. These mixed exposures may interact with other disease relevant stressors, including ageing, infection, metabolic disorder, genetic predisposition and existing neuroinflammation. Thus, future studies should move away from acute high-dose exposure studies and consider whether long-term microplastic exposure could gradually alter neural resilience, speed up disease and associated pathways, or increase vulnerability to other neurological insults.

6. Summary

Microplastic particles are emerging environmental contaminants relevant to neural health but their relationship to neural diseases is not fully understood. Current knowledge points to microplastics and nanoplastics that can affect the nervous system via direct and indirect mechanisms (such as potential interactions with biological barriers, systemic inflammation, gut–brain axis perturbations, oxidative stress, mitochondrial dysfunction, neuroinflammation, impaired proteostasis, and synaptic malfunction), which overlap with key features of the pathogenesis of neurodevelopmental disorders, neurodegenerative diseases and cognition or neuropsychiatric dysfunction, indicating that microplastics act as environmental stressors that increase neural vulnerability but are not proven direct causes of specific diseases. However, the field is still constrained by poor particle characterization, unrealistic exposure models, inadequate human evidence and weak links between general toxicological outcomes and disease-relevant mechanisms.

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