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Assessing the Impact of Microplastics on Brain Chemistry: The Need for a Comprehensive Policy Framework to Mitigate Toxicity

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Abstract

Microplastics are pervasive pollutants that enter ecosystems and the human body. This review examines and synthesises the specific impact of microplastics on brain chemistry, exploring how exposure through ingestion and inhalation can affect neurological function and overall health, due to the growing evidence linking microplastic exposure to adverse biological effects, including neurotoxicity, oxidative stress, and inflammation, which may pose significant risks to human health. The conclusions indicate that microplastics, once in the human body, can cross biological barriers and accumulate in brain tissue, leading to changes in neurotransmitter systems and cognitive impairments. The findings suggest that understanding the mechanisms of microplastic-induced neurotoxicity is crucial for assessing the long-term health implications. This review also highlights the need for regulatory measures to limit microplastic pollution and calls for further research on its effects on human neurological health.

Keywords: *Microplastics, Bioaccumulation, Biomagnification, Brain Chemistry, Neurotoxicity, Regulatory Measures.*

INTRODUCTION

Could plastic pollution be changing the way you think?

The introduction of plastic in modern life has revolutionised convenience but has also introduced a silent, pervasive threat to the environment and human health. Among the most concerning of these threats are microplastics, tiny fragments of plastic less than five millimetres in size, which have now become ubiquitous in ecosystems worldwide (NOAA, 2021). According to various studies, microplastics have been detected in a wide array of environments, including the air we

breathe (Torres-Agullo et. al., 2021), the water we drink (Kye et. al., 2023), and the food we consume (Gamarro & Costanzo, 2024). Their small size allows them to be easily ingested by both wildlife and humans, leading to potential bioaccumulation in living organisms.

While the environmental impacts of microplastics on marine life and ecosystems have been extensively studied, their effects on human health are only beginning to be explored. In particular, the potential for microplastics to affect brain chemistry has raised alarming questions. In line with the aforementioned, this paper aims to answer the following research question: **How do microplastics impact brain chemistry and to what extent is a comprehensive policy framework required to reduce their toxicity?**

This research paper aims to argue that the infiltration of microplastics into the human body through inhalation and ingestion presents significant risks to brain chemistry by crossing protective barriers, causing neurotoxicity, and potentially exacerbating neurodegenerative diseases making a comprehensive policy framework essential to mitigate these effects and reduce human exposure to these pervasive environmental toxins.

Understanding Microplastics and Their Toxicity

Microplastics, as previously defined, are plastics less than five millimetres in diameter. They have emerged as a significant environmental contaminant due to their pervasive presence across various ecosystems. These particles are broadly classified into two categories based on their origins: Primary and Secondary [Fig.1].

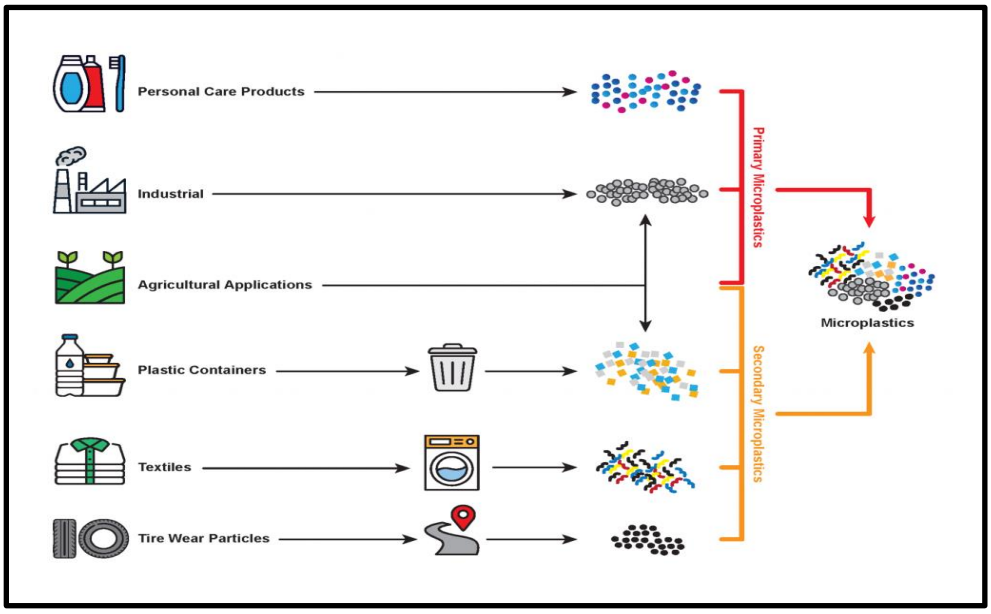

Figure 1. Primary and Secondary Microplastics alongside associated sources from ITRC (2022)

Primary microplastics are intentionally manufactured to be small and are incorporated into a range of products (Boucher & Friot, 2017). These include personal care items such as exfoliants in cosmetic products and industrial abrasives used in processes like sandblasting. In addition to cosmetic and industrial origins, microplastics are generated from the shedding of synthetic fibres during the laundering of textiles, the abrasion of tyres during road use, and the degradation of paints and coatings (Welden & Lusher, 2020). These are secondary microplastics and result from the fragmentation of larger plastic materials due to environmental factors, such as UV radiation, mechanical abrasion, and chemical degradation (Huber et al., 2022). According to (Duis & Coors (2016), these are more prevalent in the environment due to the widespread use and improper disposal of plastic products, which undergo degradation over time and disperse widely.

Microplastics have been detected in a wide range of locations, from soils to aquatic environments, and digestive tracts of organisms. The diagram below [Fig. 2] shows how microplastics originate from urban and agricultural activities, spread through runoff and atmospheric deposition, and accumulate in soils across various environments, including remote areas. This cycle highlights the widespread contamination caused by microplastics.

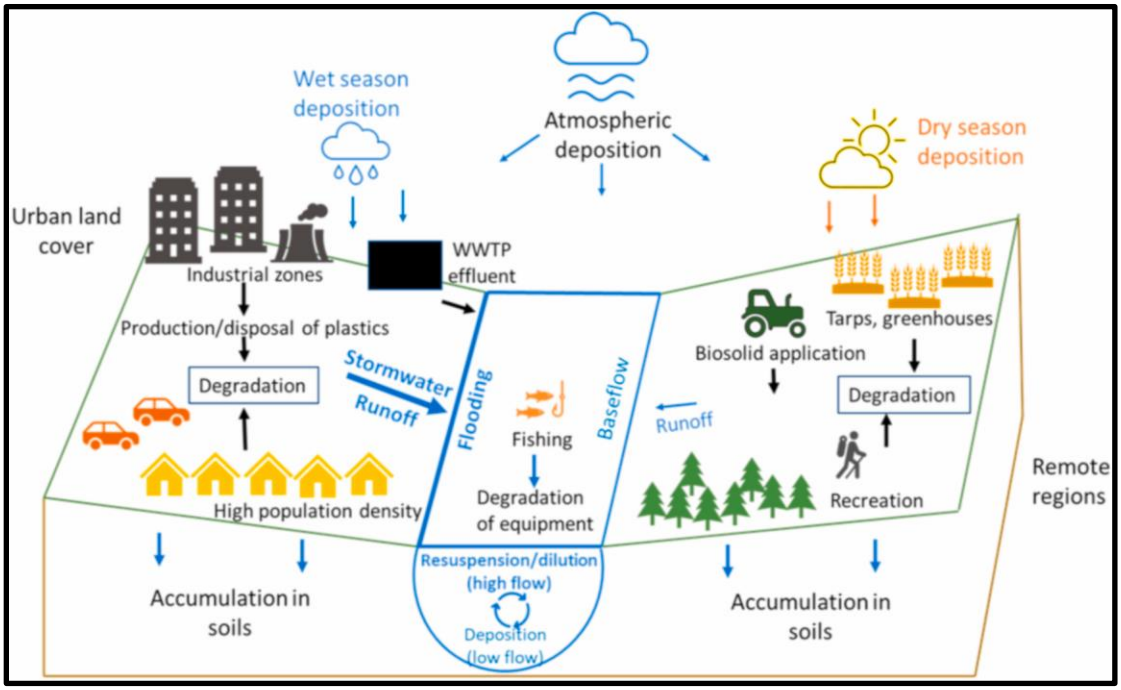

Figure 2. Spatial and temporal factors influencing the distribution and abundance of freshwater microplastics from Talbot & Chang (2022)

Oceans are the most well-documented repositories of microplastics. According to a study by The Ocean Cleanup (2021), the Great Pacific Garbage Patch alone contains approximately 1.8 trillion pieces of plastic, a significant proportion of which are microplastics. These particles are found not only on the ocean's surface but also in deep-sea sediments, where they have been detected at depths of over 11,000 metres in the Mariana Trench (Peng et al., 2019). This widespread presence poses a significant threat to marine life, as microplastics are ingested by a wide range of organisms, entering the food chain and potentially affecting human health through seafood consumption.

The food chain is also infiltrated via agricultural practices, including the use of plastic mulching, sewage sludge application, and the breakdown of plastic litter, contributing to the accumulation of microplastics in soil. According to Pérez-Reverón et al. (2022), the accumulation of microplastics may affect soil physical and chemical properties and processes, such as bulk density (Yadav et al., 2022) and cation exchange capacity (CEC) (Wen et al., 2022). Depending on their shape, type, size and quantity, microplastics can reduce or increase water-stable aggregates (Fang et al., 2024), which leads to changes in soil structures that are important for soil ecosystem functioning. Additionally, Plastic particles as large as 1 μm can enter the root tissue of rice seedlings and subsequently, driven by the pull of transpiration, be transported to the shoot (Liu et al., 2023). These microplastics can persist in soil for extended periods, potentially affecting soil structure, nutrient cycling, and the health of soil organisms, which play crucial roles in ecosystem functioning.

As cattle and other livestock graze on fields contaminated with microplastics, either directly through ingestion of soil and plants or indirectly via contaminated water sources, these particles can accumulate in their tissues (Bahrani et al., 2023). This process, known as bioaccumulation, involves the gradual buildup of microplastics within an organism over time, as they are ingested faster than they are expelled (Chojnacka & Mikulewicz, 2014). As products from livestock are consumed by humans or other predators, the concentration of microplastics, along with any adsorbed toxic substances like persistent organic pollutants (POPs) and heavy metals (Aardema et al., 2024), increases further up the food chain in a process known as biomagnification (Merriam-Webster, 2009). This not only heightens the exposure risk for top-level consumers, including humans but also amplifies the potential health hazards.

An additional feature of microplastics is that they create new locations, protected from competition for nutrients in the water for bacterial and eukaryotic colonisation that might not proliferate otherwise (Zhai et al., 2023), nutrient cycling (Weig et al., 2021), and biofilm formation (Jia et al., 2024), leading to the formulation of the term plastisphere (Zettler et al., 2013). According to studies such Pathogens, such as Vibrio, Tenacibaculum, Pirellulaceae (Wright et al., 2020) and spirochetes (Xie et al., 2021), were commonly found on the plastisphere. Besides colonisation with pathogens, microplastics could act as a long-term reservoir for antibiotic resistance (Liu et al., 2021). A study by Schmidt et al. (2017) estimated that rivers also serve as conduits for between 1.15 and 2.41 million metric tons of plastic waste to the oceans annually. Results from a study by Kunz et al. (2023) show abundances ranging from 0 pcs/m³ in unpopulated rural areas up to 230 pcs/m³ in densely populated urban centres and are positively correlated with population density. This physical structure of microplastics also enables them to adsorb toxic substances from the environment, including persistent organic pollutants (POPs) and heavy metals. These chemicals, which are often hydrophobic, readily bind to the surfaces of microplastics, making these particles vectors for harmful contaminants as well (Zambrano-Pinto et al., 2024). When ingested, the toxic load carried by microplastics can be released into the organisms, leading to cellular damage, oxidative stress, and a range of health issues.

A relatively recent exploration is the atmospheric transport of microplastics, it highlights the far-reaching nature of this pollutant. Microplastics have been detected in the atmosphere in both urban and remote areas, indicating their ability to travel long distances through the air. Allen et al. (2019) reported the deposition of microplastics at a rate of 365 particles per square metre per day, suggesting that atmospheric transport is a significant pathway for the global distribution of microplastics. Once inhaled and lodged in the respiratory system, microplastics can induce a range of harmful effects. The physical presence of these particles can cause mechanical irritation, leading to inflammation of the respiratory tissues (Saha & Saha, 2024). Additionally, these substances can be released into lung tissues, contributing to local toxicity and potentially entering the bloodstream, where they can exert systemic effects (Campanale et al., 2020).

The implications of these findings extend beyond immediate health concerns, suggesting that microplastics may have far-reaching

effects, particularly on sensitive biological processes such as brain function.

Mechanisms of Microplastic Impact on Brain Chemistry

As previously discussed, microplastics have permeated a variety of environmental mediums, including air, water, and soil, making them almost impossible to avoid in everyday life. This widespread distribution facilitates multiple entrance pathways for microplastics into organisms.

Inhalation of airborne microplastics, as noted, can lead to the particles being lodged in the respiratory system, causing inflammation and potentially entering the bloodstream. Due to the degradation-resistant nature of microplastics, their accumulation in the respiratory tract may lead to local inflammation, exacerbation of existing respiratory conditions, and other health problems when translocated (Yang et al., 2023). This increases the potential for respiratory diseases and systemic toxicity. Notably, recent studies have detected microplastics in human pulmonary tissue (Ali et al., 2024) and sputum samples (Huang et al., 2022), highlighting inhalation as a significant route for plastic particles to enter the body. As the airway epithelium serves as the primary deposition site for airborne particulate matter (Cooper & Loxham, 2019), exposure to microplastics may compromise epithelial integrity. A recent study found that exposure to polystyrene microplastics (10 to 1000 μg/cm²) decreased the expression of tight junction proteins, such as Zonula Occludens, in human lung epithelial cells, indicating disruption of the epithelial lining (Dong et al., 2024). Furthermore, Shamsul Anuar et al. (2022) demonstrated that microplastic exposure significantly enhanced the contractile response of rat-isolated airway smooth muscle, possibly due to epithelial damage and subsequent dysfunction of the autonomic nervous system. The human lung, with its extensive alveolar surface area of approximately 150 m² and an exceptionally thin tissue barrier of less than 1 μm, presents a potential entry point for nanoparticles, including microplastics, into the bloodstream (Campanale et al., 2020). Once these toxic particles penetrate the bloodstream, they may circulate throughout the body, reaching various organs, including the brain.

Microplastics in drinking water and food sources, including seafood and crops, represent a significant route of human exposure. The contamination of water bodies has led to microplastics entering drinking water, which is consumed by millions globally. Additionally, the food chain poses another major pathway for exposure, with microplastics found in various consumables. In seafood, these

particles accumulate in the digestive tracts of fish and shellfish, while in agriculture, they are present in vegetables and grains grown in polluted soil. Ingested microplastics can cause physical damage to the gastrointestinal (GI) tract, alter the gut microbiota, and facilitate the absorption of harmful chemicals associated with the particles. The GI tract is closely linked to the central nervous system (CNS) through the gut-brain axis [Fig.3], a network that includes the enteric nervous system (ENS). This network, embedded within the intestinal mucosa, is highly responsive to various stimuli and could be adversely affected by microplastics.

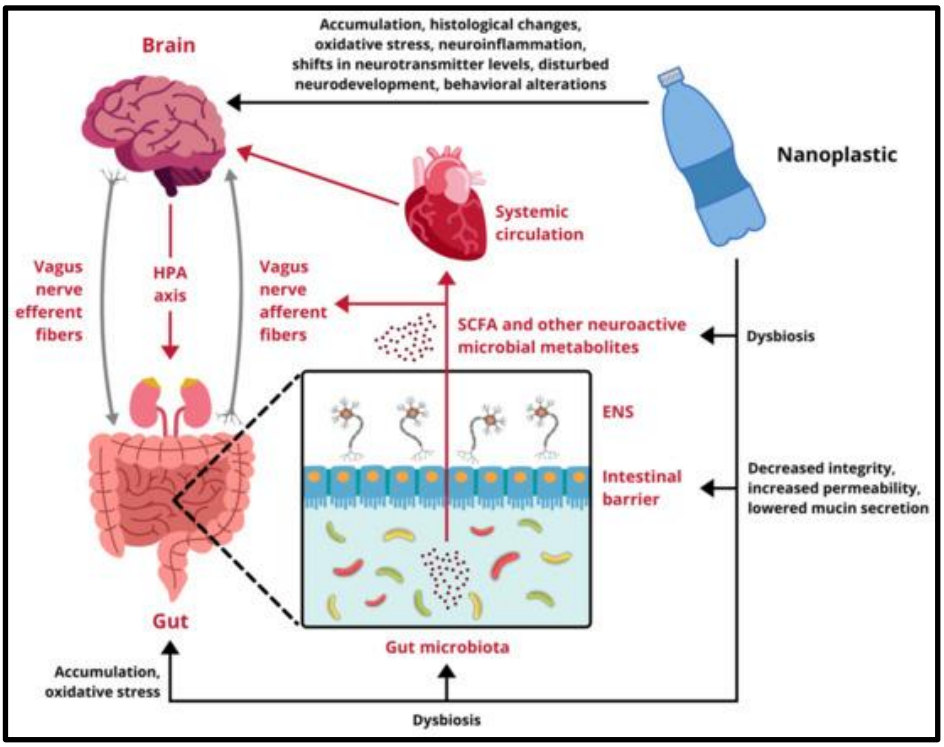

Figure 3. Impact of nano plastic exposure on the gut-brain axis from Grodzicki et al. (2021)

Jung et al. (2020) found that 100 nm polystyrene microplastics were taken up by murine astrocytes and neurons, particularly affecting the cell bodies near the nuclei. This uptake resulted in reduced neuronal viability and disrupted neuronal development, as indicated by changes in gene expression related to neuronal function. Increased proinflammatory signalling in astrocytes was also observed, with upregulation of TNF-α and IL-1β genes, which could lead to inflammation and neuronal damage. Schirinzi (2020) demonstrated that exposure to various micro and nanoplastics, including polyethylene (PE)

and polystyrene, led to increased production of reactive oxygen species (ROS) in human glioblastoma T98G cells, with polystyrene showing more pronounced effects. Similarly, Hoelting et al. (2012) used an embryonic stem cell-derived 3D model of human neural development to demonstrate the impact of 33 nm PE microplastics. Short-term exposure at high concentrations caused cytotoxicity and oxidative stress, while long-term exposure revealed that even low concentrations could accumulate and alter neurodevelopmental gene expression.

Although research on nanoplastic toxicity to the GI tract and CNS cells is still limited, existing studies indicate that nanoplastics can penetrate gut cells, potentially leading to broader systemic distribution. In neural models, nanoplastics have been shown to induce oxidative stress and reduce cell viability, especially in models that more accurately reflect long-term exposure conditions. The paper by Gałęcka et al. (2024) confirms that microplastics, as foreign substances, affect neuronal populations in the ENS of the jejunum. However, the exact mechanisms by which these particles impact neuronal and other cellular functions are not fully understood. Potential mechanisms of neurotoxicity may include mitochondrial dysfunction, oxidative stress, direct physical damage, changes in neurotransmitter levels, or disruptions to the gut microbiome. These effects may lead to increased expression of pro-apoptotic proteins and inflammatory cytokines, contributing to neurodegenerative processes. The available evidence suggests that microplastics could have multiple impacts on both the ENS and CNS, potentially exacerbating neurodegenerative and gastrointestinal diseases. Conditions such as Parkinson's and Alzheimer's disease, which may originate in the ENS, could be influenced by age-related changes in the gut microbiome and exposure to toxic agents like microplastics. This highlights the need for further research into the long-term neurochemical effects of microplastic exposure.

The study by Gałęcka et al. (2024) also suggested that the decrease in neurons might be a defensive mechanism against microplastics. Neurotransmitters, such as acetylcholine, which is primarily excitatory, can cause vasodilation. In the case of inhibited intestinal motility, partly due to the activity of galanin, these conditions may promote the absorption of microplastics from the intestines into the bloodstream. According to Nezami and Srinivasan (2010), acetylcholine-secreting neurons stimulate smooth muscles, enhance the secretion of intestinal juices, dilate blood vessels, and release intestinal hormones. In the

current study, PET microplastics were found to reduce the population of these neurons, potentially leading to negative effects on intestinal peristalsis and digestive functions, as discussed by Rytel et al. (2020). Additionally, the reduction in this activity might weaken the immune response by inhibiting macrophage activity.

In a study by Lee et al. (2022), exposure to polystyrene microplastics has been shown to impair learning and memory in mice, with polystyrene microplastics detected in the hippocampus. This region exhibited reduced levels of immediate-early genes, abnormal increases in synaptic glutamate AmicroplasticA receptors, and heightened neuroinflammation—key factors in synaptic plasticity and memory. Interestingly, vagus nerve ablation, which influences the gut-brain axis, improved memory in these mice, suggesting that polystyrene-microplastics alter neuronal gene expression and synaptic proteins through a vagus nerve-dependent pathway, leading to neuroinflammation and behavioural changes (Ali et al., 2024). Further evidence of microplastic-induced neurotoxicity comes from animal studies where male mice orally exposed to fluorescent polystyrene-microplastics (5 μm and 20 μm) exhibited reduced acetylcholinesterase (AchE) activity (Deng et al., 2017), thereby impairing cholinergic neurotransmission efficiency and causing oxidative stress. Exposure to microplastics also altered serum levels of essential amino acids, such as threonine, aspartate, and taurine, while reducing phenylalanine, a precursor to neurotransmitters. Although evidence remains limited, these findings underscore the need for further investigation into the potential neurotoxic risks posed by micro- and nano-plastics. Complementary studies by Donkers et al. (2022) using advanced in vitro models of the lungs and intestines have demonstrated that micro- and nano-plastics can induce cytotoxic and pro-inflammatory effects, translocate across epithelial barriers, and activate immune cells. This translocation suggests that ingested microplastics can spread through the bloodstream, potentially accumulating in tissues-a condition termed "plasticemia," which could lead to organ dysfunction.

Recent research has raised significant concerns about the ability of microplastics to penetrate the blood-brain barrier (Fig.4), a critical protective mechanism that typically shields the brain from harmful substances. Liu et al. (2024) demonstrated that microplastics can indeed cross this barrier, accumulating in brain tissue and suggesting potential neurotoxic effects. This discovery underscores the need to

further investigate the implications of microplastics on brain health.

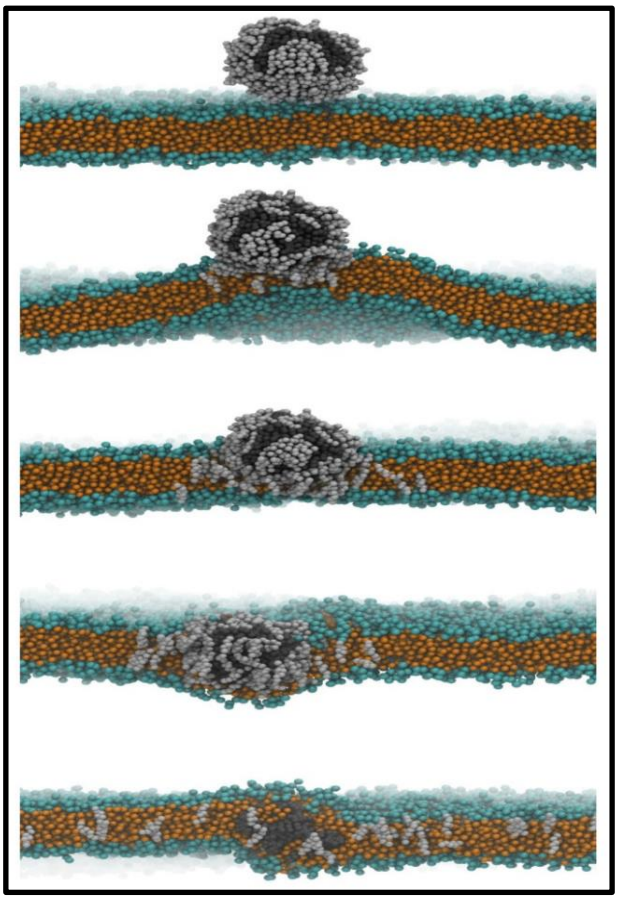

Figure 4. Snapshots of the simulations showing the entry of the plastic particle with a corona of 150 cholesterol molecules into the DOPC bilayer, a model blood-brain barrier from Kopatz et al. (2023)

Supporting these findings, a study by Kopatz et al. (2023) utilised computer models to show that the ability of polystyrene particles to cross the blood-brain barrier depends on their surface corona-specific molecular coatings that influence their interaction with biological membranes. These in silico results were confirmed in vivo, with mouse models showing that nanometer-sized microplastic particles could be detected in brain tissues as soon as two hours after exposure (Fig.5). This rapid accumulation highlights the urgent need to understand how microplastics interact with brain tissues and what long-term effects they may have.

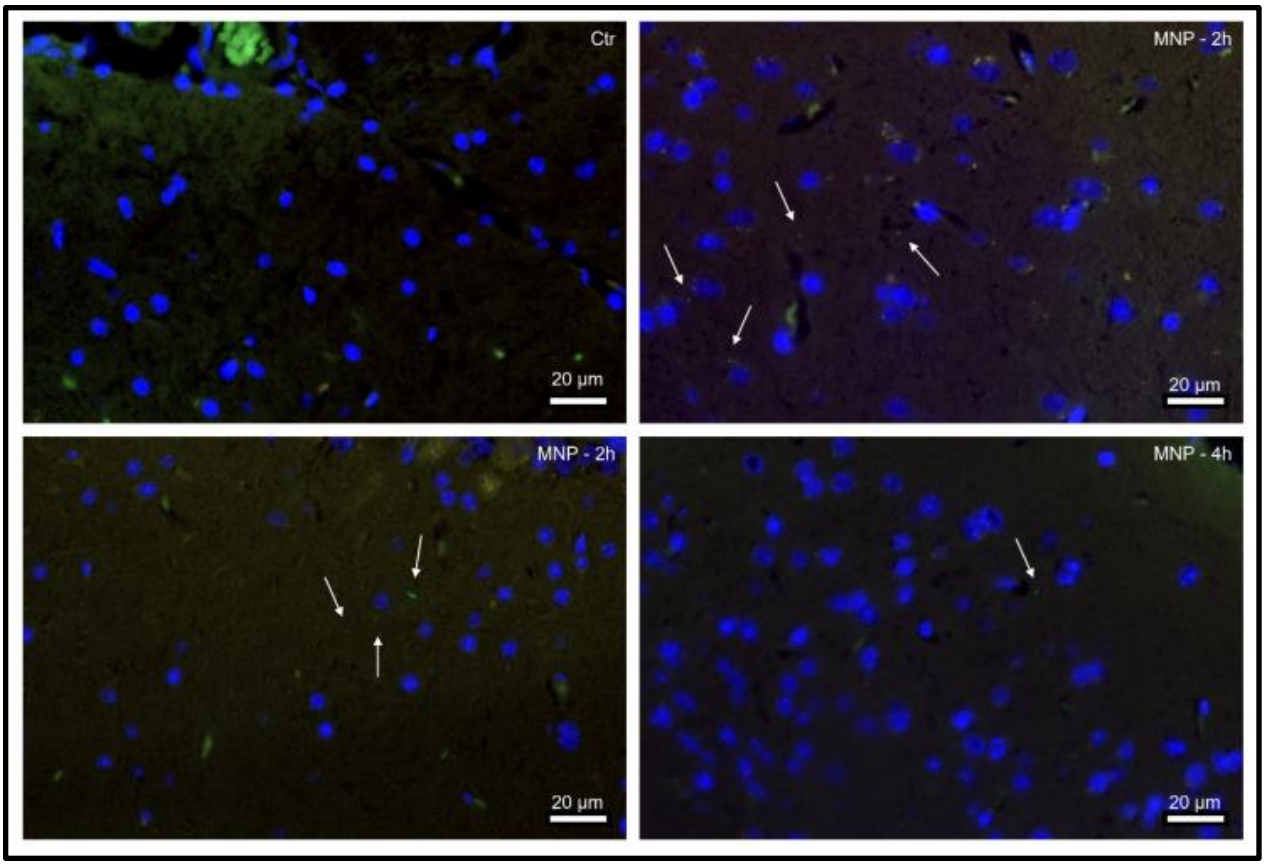

Figure 5. Nanometer-scale plastic particles in the mouse brain from Kopatz et al. (2023)

Furthermore, Gaspar et al. (2023) investigated the effects of microplastic exposure on astrocytes, a type of glial cell in the brain that typically responds to neural stress or injury. Contrary to the usual increase in GFAP expression associated with neuroinflammation, the study found a decrease in GFAP levels, suggesting astrocyte atrophy. This pattern is consistent with early-stage neurodegenerative conditions like Alzheimer's disease (AD) or Major Depressive Disorder (MDD), where decreased GFAP expression has been observed. These results indicate that exposure to polystyrene microplastics may induce age-dependent neurochemical changes, potentially contributing to the early onset of neurological disorders.

The neurotoxic potential of microplastics is further highlighted by Jin et al. (2022), who explored the impact of microplastics on the hippocampus, a brain region critical for learning and memory. Their study found that exposure to microplastics led to an increase in pro-apoptotic proteins in the hippocampus, which is a key feature of neurodegenerative diseases. Additionally, the study observed a decrease in the density of dendritic spines, which are essential for synaptic growth and

plasticity, with implications for cognitive function and intellectual ability. Furthermore, levels of synapsin 1, synaptophysin, and other proteins critical for maintaining synaptic homeostasis and memory function, were found to decrease in a concentration-dependent manner following polystyrene-microplastic exposure. The study also reported lower expression of Syt 1 and Bdnf, genes necessary for neurodevelopment and synaptogenesis, further indicating the detrimental impact of microplastics on brain function (Fig.6).

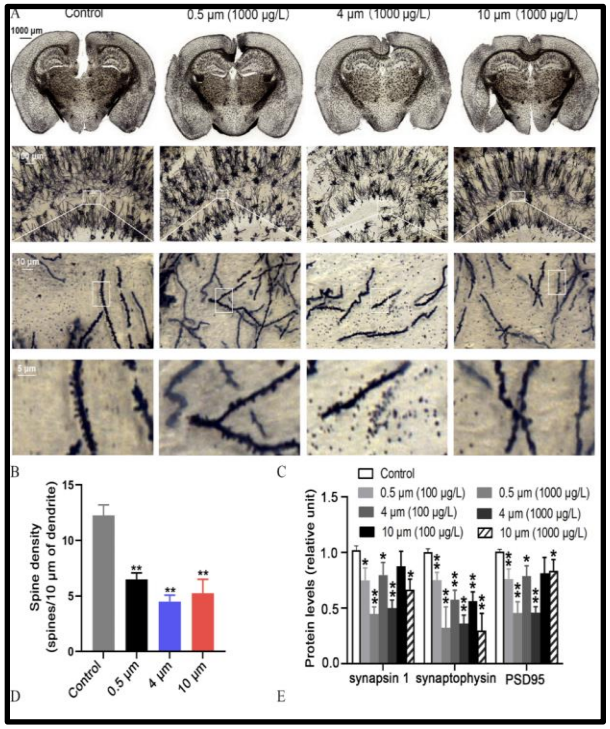

Figure 6. The density of dendritic spines and expression of proteins related to synaptogenesis in the hippocampus of polystyrene microplastics from Jin et al. (2022)

This is also backed up by a study on animal behaviour. Crump et al. (2020) found that microplastic exposure impaired shell selection behaviour in hermit crabs—a complex task that requires the processing of information about shell quality. This impairment suggests that microplastics may inhibit cognitive functions, raising concerns about their potential impact on human cognition if similar mechanisms occur in humans.

Additionally, Schirinzi et al. (2017) explored the cellular effects of microplastic exposure, finding that human cerebral (T98G) cells and epithelial (HeLa) cells produced increased levels of reactive oxygen species (ROS) after 24 hours of exposure to polystyrene microplastics at the highest concentrations tested. However, polyethylene microplastics did not induce the same effect. The generation of ROS is a key indicator of oxidative stress, which can lead to cellular damage and is a known contributor to neurodegenerative diseases.

Taken together, these studies highlight the potential for microplastics to disrupt neurochemistry by crossing the blood-brain barrier, inducing neuroinflammation, affecting glial cell function, and promoting oxidative stress. The implications for human health are profound, suggesting that chronic exposure to microplastics could contribute to the

development of neurodegenerative diseases and cognitive impairments. Further research is crucial to fully understand the long-term neurochemical effects of microplastics and to develop strategies to mitigate their impact on brain health.

Policy Strategies to Mitigate Human Exposure and Neurotoxicity from Microplastics

Given the mounting evidence of the neurotoxic effects of microplastics, a comprehensive policy framework and other measures are essential to mitigate these risks and protect public health.

A foundational step in addressing the microplastic crisis is educating the public about the sources of microplastic pollution and the associated risks, particularly the brain, which is essential for encouraging change and supporting regulatory measures. The effectiveness of public awareness campaigns in other domains offers a blueprint for similar efforts targeting microplastics. For instance, anti-smoking campaigns, such as those implemented by the World Health Organization (WHO) and national governments, have helped prevent an estimated 129,000 early deaths (Shrestha et al., 2021) and helped save an estimated $7.3 billion in smoking-related healthcare costs (Davis et al., 2022). These campaigns combined education, media outreach, and policy interventions to shift public behaviour. A comparable approach for microplastics could involve comprehensive educational programs in schools, public service announcements, and targeted social media campaigns to raise awareness of the issue and promote sustainable practices. Informed citizens are more likely to support and comply with regulatory measures, thereby amplifying the impact of policy interventions.

Furthermore, there should be significant attention paid to the establishment of standardised monitoring guidelines for microplastic contamination in food, water, and air. Currently, monitoring efforts are inconsistent and lack uniformity, hindering our ability to accurately assess human exposure and associated risks. The development of global monitoring protocols that can be applied across different regions and environments should be prioritised. The success of similar monitoring systems in the regulation of lead and mercury provides a strong precedent for this approach. For example, the Centers for Disease Control and Prevention (CDC) in the United States established standardised blood lead level guidelines, which have been instrumental in monitoring and reducing lead exposure across the population (CLP Prevention, 2024). A similar framework for

microplastics could involve the regular monitoring of contamination levels in critical media, followed by reporting and public disclosure. In addition to monitoring, setting permissible exposure limits (PELs) for microplastics based on current scientific understanding is essential. These would serve as benchmarks for determining the safety of food and water supplies. For instance, the regulation of airborne particulate matter (PM2.5 and PM10) by the Environmental Protection Agency (EPA) in the United States has significantly reduced air pollution and associated health risks (EPA, 2023). By establishing clear exposure limits for microplastics, governments can enforce stricter controls on sources of pollution and take immediate corrective measures when contamination levels exceed safe thresholds.

One of the most effective strategies is to implement bans on the use of microbeads in personal care products-a measure that has already proven successful in several countries. For instance, the Microbead-Free Waters Act of 2015 in the United States prohibited the manufacture and sale of rinse-off cosmetics containing plastic microbeads, leading to a significant reduction in microplastic pollution (FDA, 2020). Expanding similar bans globally and extending them to other products, such as industrial cleaning agents and abrasive materials, would further reduce microplastic emissions. Moreover, governments should incentivize the development and adoption of biodegradable alternatives to conventional plastics. This approach has been successfully employed in the promotion of biodegradable packaging materials. Such as legislation that mandates the use of compostable bags for fruits, vegetables, and other loose items in supermarkets implemented in Italy (Newman, 2021). This policy has not only reduced plastic waste but also spurred innovation in the development of biodegradable materials. A similar strategy could be applied to microplastics, with subsidies or tax incentives provided to companies that invest in research and development of sustainable alternatives.

Finally, addressing the microplastic crisis requires a significant increase in funding for research into the health impacts of microplastics, particularly their effects on brain chemistry, as well as the development of innovative solutions to mitigate these impacts. The current state of research is still in its infancy, and there is a pressing need for more comprehensive studies to understand the full extent of the risks posed by microplastic exposure. The success of funding initiatives for environmental research, such as the Horizon 2020 program in the European Union, which supported research and innovation in areas like

climate change and sustainable development, demonstrates the impact that targeted funding can have (EEAS, 2018). Governments should prioritise funding for research into microplastics, particularly focusing on their long-term effects on human health and potential interventions. This could include international collaborations to pool resources and expertise, as well as public-private partnerships to accelerate the development of sustainable alternatives to conventional plastics.

CONCLUSION

Given the increasing prevalence of microplastics in various environments, understanding their potential risks, particularly to human health, is crucial. This review aimed to explore the impact of microplastics on brain chemistry and to assess the necessity of a comprehensive policy framework to mitigate their toxicity.

The findings suggest that both primary microplastics and secondary microplastics can enter the human body through ingestion, inhalation, and dermal absorption. Once inside, these particles can cross biological barriers, including the blood-brain barrier, leading to potential neurotoxicity. Research indicates that exposure to microplastics can cause inflammation, oxidative stress, and alterations in neurotransmitter levels, contributing to neurological disorders and cognitive impairments. Furthermore, the processes of bioaccumulation and biomagnification increase the potential for microplastics to disrupt brain chemistry over time.

The implications of findings from the cited studies emphasise the urgent need for further research into the mechanisms by which microplastics affect brain chemistry. Investigations into the long-term effects of microplastic exposure, the impact on vulnerable populations, and the interaction between microplastics and other environmental toxins would be beneficial. Identifying dose-response relationships and biomarkers of exposure is essential for evaluating the risk posed by microplastics.

To address these concerns, a comprehensive policy framework is crucial to regulate the production, use, and disposal of plastics. Policies that focus on reducing plastic waste, promoting sustainable alternatives, and enforcing stricter regulations on microplastic emissions are shown to be most impactful. Additionally, public education campaigns are vital to raise awareness about the dangers of microplastic pollution and encourage responsible disposal practices. By implementing these measures, we can reduce

the potential neurotoxic effects of microplastics, thereby safeguarding human health and the environment.

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