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Editorial

Microplastics and nanoplastics in the brain: a review of the neurodevelopmental risks

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Abstract

Background: Microplastics (MPs, < 5 mm) and nanoplastics (NPs, 1-1,000 nm) – collectively referred to as MNPs – have become pervasive environmental contaminants. Their potential accumulation in the human body, particularly in the brain, has raised significant health concerns.

Objective: This review summarizes current evidence on the presence of MNPs in the brain, their potential routes of entry, mechanisms of action, and implications for neurological health.

Methods: We performed a critical review of recent literature concerning MNP exposure, uptake pathways, distribution in human tissues, especially the brain, and their neurotoxic effects, with a focus on developmental vulnerability.

Results: MNPs can reach the brain through ingestion, inhalation, and possibly via the bloodstream, crossing the blood-brain barrier (BBB) through mechanisms such as endocytosis, pinocytosis, or via immune cells. Animal studies suggest accumulation of MNPs in the brain induces oxidative stress, neuroinflammation, and behavioral changes. Particularly concerning is the perinatal period, where multipotent stem cells in breast milk may act as potential vectors of MNPs and endocrine-disrupting chemicals (EDCs) into the neonatal brain.

Conclusions: MNPs represent an emerging threat to neurodevelopmental health. Future research must clarify the long-term effects of MNP exposure, especially in early life, and identify potential strategies to mitigate their impact. The irony lies in the semantic shift: while "plasticity" once symbolized the brain's capacity for change, "plastic" now threatens that very adaptability.

Keywords

Microplastics, nanoplastics, neurotoxicity, plasticity, brain development, endocrine disruptors, breast milk, multipotent stem cells, blood-brain barrier, environmental pollution.

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Introduction

In the 1967 film *The Graduate*, a character famously advises the young protagonist, "There's a great future in plastics. Think about it." Decades later, this optimistic vision has taken a dramatic turn. In 2018, a striking title by Kontrick – "*Microplastics and human health: our great future to think about now*" – reflected growing concern over the unintended consequences of this once-revolutionary material [1].

Since 1950, global plastic production has surged from 2.3 million to 448 million tons in 2015. Alarmingly, 44% of all plastic ever produced was manufactured between 2000 and 2015 [2]. Plastics are now ubiquitous, found in every environment on Earth – from the peak of Mount Everest to the depths of the Mariana Trench. A 2021 study estimated that over 1,000 rivers account for 80% of plastic emissions into the oceans, with Asia contributing 81% of the total, followed by Africa (8%), South America (5.5%), North America (4.5%), Europe (0.6%), and Australia (0.4%). Notably, the largest contributing country estimated by that study was the Philippines [3].

This uncontrolled proliferation of plastic has resulted in extensive environmental contamination. Microplastics (MPs) and nanoplastics (NPs) – collectively referred to as MNPs – originate from two main sources: primary MNPs, intentionally produced for use in cosmetics or industry, and secondary MNPs, formed by the degradation of larger plastic debris via photodegradation, mechanical abrasion, and chemical weathering [4].

Humans are exposed to MNPs through ingestion (via contaminated food and water), inhalation, and dermal contact. Once internalized, MNPs can enter the circulatory system and potentially cross the blood-brain barrier (BBB). Proposed mechanisms of BBB penetration include clathrin-mediated endocytosis, macropinocytosis, transcytosis via immune cells, or disruption of tight junctions [5, 6]. Animal studies on *Daphnia magna* have confirmed that clathrin-dependent endocytosis facilitates intestinal absorption of NPs – a mechanism that may also be operative in brain tissues [6].

MNPs have been detected in terrestrial and aquatic ecosystems, the atmosphere, and even in human organs and tissues [7-9]. Of particular concern is their potential neurotoxicity, given that MNPs can accumulate in the brain and may trigger neuroinflammatory processes, oxidative stress, and cognitive impairment.

The term "plastic" derives from the Greek "*plastikos*," meaning "molded" or "shaped." In neuroscience, the concept of plasticity – introduced by psychologist William James in 1890 – refers to the brain's capacity to change in response to experience [10]. This concept laid the foundation for modern neuroscience. Eric Kandel, Nobel laureate and pioneer of synaptic plasticity, famously stated: "We are what we are by virtue of what we have learned and remembered" [10].

Until the late 20th century, it was believed that neurogenesis was confined to early childhood. However, landmark studies demonstrated that adult brains retain the ability to structurally and functionally remodel themselves – a phenomenon now known as adult neuroplasticity. One iconic study revealed that experienced London taxi drivers, who undergo intensive training in spatial navigation ("The Knowledge"), exhibited significantly larger hippocampal volumes compared to novices [11].

A crucial milestone in the field was the discovery of nerve growth factor (NGF) by Rita Levi-Montalcini, which earned her the Nobel Prize in 1986. NGF is a small dimeric protein of 118 amino acids that orchestrates axonal growth and regeneration, playing a key role in neuronal development and plasticity [12, 13].

Neuroplasticity enables the brain to adapt, recover, and reorganize following injury or disease [14]. However, this same plasticity may render the brain more vulnerable to foreign agents such as MNPs, particularly during critical developmental windows. The current review explores this paradox:

the brain is plastic, but increasingly contains plastic – with profound implications for public health.

The brain is plastic, but it also contains a lot of plastic

Recent studies have confirmed the presence of MNPs in human brain tissue, raising significant concerns about their potential neurotoxicity [15]. Animal models have shown that MNP exposure can induce behavioral changes, neuroinflammation, oxidative stress, and mitochondrial dysfunction in neuronal cells. Key concerns include:

- neuroinflammation: MNPs may activate microglia and astrocytes, promoting chronic inflammatory responses implicated in neurodegenerative disorders;
- oxidative stress: MNPs can increase the production of reactive oxygen species (ROS), leading to lipid peroxidation, DNA damage, and neuronal apoptosis;
- cognitive impairment: accumulation of MNPs in the brain may disrupt synaptic transmission and plasticity, contributing to learning and memory deficits.

A recent study by researchers at the University of New Mexico analyzed brain, liver, and kidney tissues from 28 deceased individuals using pyrolysis gas chromatography – mass spectrometry, attenuated total reflectance – Fourier transform infrared spectroscopy (ATR-FTIR), electron microscopy, and energy-dispersive spectroscopy [16]. Their findings are noteworthy:

- polyethylene dominance: MNPs detected in all organs were predominantly composed of polyethylene, the most widely used plastic polymer globally; in Europe alone, polyethylene accounts for nearly 30% of total plastic demand [17];
- higher brain accumulation: brain tissues contained significantly higher concentrations of MNPs – up to 30 times more – compared to the liver and kidneys; this is particularly notable given the lower cerebral blood flow compared to renal and hepatic circulation; the kidneys receive approximately 400 mL/100 g/min, the liver around 100-130 mL/100 g/min, and the brain about 50-60 mL/100 g/min overall (80 mL/100 g/min in gray matter, 20 mL/100 g/min in white matter) [18, 19].

While the kidneys and liver are responsible for excreting many xenobiotics, the brain may act as a "sink," with limited capacity to eliminate MNPs once they cross the BBB. This phenomenon warrants further investigation [20].

Interestingly, no significant correlation was found between MNPs concentration and the age of individuals, suggesting the possibility of partial elimination or biological adaptation mechanisms. However, data also showed that MNPs content in the brain and kidneys increased over time: samples from 2024 exhibited approximately 50% higher levels than those from 2016, paralleling the rise in global plastic production and waste. Plastic production doubled from 234 million tonnes (Mt) in 2000 to 460 Mt in 2019, while waste generation rose from 156 Mt to 353 Mt during the same period. Only 9% of plastic waste is currently recycled [21].

Perhaps the most alarming finding was the 3- to 5-fold higher concentration of MNPs in the brains of individuals diagnosed with dementia, compared to those without. While this association does not establish causality, it raises serious concerns and highlights the urgent need for mechanistic studies to explore this potential link [16].

Although some quantitative aspects of these findings were questioned in a recent *Nature* editorial [22], other evidence supports the plausibility of such a connection. For example, seabird studies have shown that ingestion of plastic induces lesions in the stomach lining, liver, kidneys, and brain, including cellular changes resembling early neurodegenerative disease such as Alzheimer's [23].

Further support comes from preliminary data presented at the 2025 American Academy of Neurology (AAN) conference, showing that coastal populations in the United States exposed to higher environmental concentrations of MNPs exhibited lower cognitive performance and a higher incidence of cognitive disability [24].

Currently, no effective strategies exist to promote MNPs clearance from the human brain. However, in fish models, removal from a plastic-contaminated environment results in a progressive decline in cerebral MNPs: after 75 days, brain MNP concentrations decreased by 75% [25, 26]. Whether this degree of clearance is achievable in humans remains unknown, particularly given the ubiquitous presence of MNPs in air, food, and water. Inhalation is now considered a major source of exposure, with adults estimated to inhale up to 62,000 MNPs annually [27].

Taken together, these findings underscore the growing threat posed by MNPs to brain health. Understanding the pathways of entry, bioaccumulation, and physiological interaction with

brain tissue is critical for developing mitigation strategies and assessing public health risks.

A children's perspective on nanoplastics and microplastics

A particularly urgent dimension of MNPs research concerns their impact on children's health. Due to developmental immaturity, children – including fetuses and neonates – are more vulnerable to environmental toxins, including endocrine-disrupting chemicals (EDCs) and synthetic polymers such as MNPs.

A 2022 review by Sripada et al. emphasized the specific vulnerabilities of pediatric populations, from *in utero* exposure to postnatal stages, highlighting how early plastic exposure may have lifelong health consequences [28]. This has been reinforced by additional reviews addressing plastic pollution during pregnancy and neonatal development, a period often described as "beyond the cradle" [29].

MNPs have been shown to infiltrate the human food chain, airways, water, and biological fluids [30]. One of the authors of the present paper (A.R.) was the first to document the presence of MPs in the human placenta [31]. Following this discovery, MNPs have also been detected in human breast milk [32], amniotic fluid, and even in meconium – the first stool of newborns – demonstrating prenatal and immediate postnatal exposure.

A recent study presented in abstract form at the Society for Maternal-Fetal Medicine reported higher levels of NPs in the breast milk of mothers of preterm infants, raising concerns about differential exposure in vulnerable populations. Concurrently, a 2025 systematic review synthesized data from animal and human studies and concluded that MNPs may negatively impact female reproductive health, including ovarian function, hormonal balance, and fetal development [33].

One of the authors of the present paper (V.F.) also conducted the first metabolomic analysis of human breast milk, demonstrating its complex biochemical composition [34]. Both authors are currently collaborating on the European LIFE MILCH project, using metabolomics to compare breast milk samples with and without MNP contamination. Preliminary findings suggest that the presence of plastic may alter energy-related metabolic pathways, with potential implications for metabolic syndrome and developmental programming [35-37].

Breast milk contains multipotent stem cells capable of migrating to neonatal tissues, including the brain, where they differentiate into neurons, astrocytes, and oligodendrocytes. This unique biological feature supports brain development and plasticity during a critical window of neurodevelopment. A study on intranasal administration of breast milk in preterm infants with severe intraventricular hemorrhage reported reductions in major complications, including porencephaly and posthemorrhagic hydrocephalus [38].

However, this same mechanism may also present a route for MNPs and EDCs to reach sensitive target tissues. Multipotent stem cells are particularly susceptible to chemical disruption, and may inadvertently act as vectors, delivering contaminants directly to developing organs. MNPs are known to exert a "Trojan horse" effect, adsorbing and transporting additional pollutants such as bisphenols and phthalates, thereby amplifying their toxicity [39].

The role of EDCs in altering stem cell function is an emerging field of concern. Mechanisms include interference with hormone signaling pathways, oxidative stress induction, epigenetic modifications, and disruption of gene expression. These effects may interfere with neurogenesis, myelination, and synaptogenesis, ultimately increasing the risk for neurodevelopmental disorders [40].

Conclusions

This review highlights the growing body of evidence suggesting that MNPs can accumulate in the human brain and may exert neurotoxic effects. These particles, once considered inert environmental pollutants, now appear capable of disrupting essential neurobiological processes – especially during sensitive developmental windows such as the perinatal and neonatal periods.

Evidence from human autopsy studies, *in vivo* animal models, and population-based data all point to the brain as a vulnerable target for MNPs. Findings include their detection in brain tissues, their association with oxidative stress and neuroinflammation, and their possible link to neurocognitive deficits and neurodegenerative diseases. Moreover, emerging studies suggest that individuals with dementia may harbor significantly higher levels of MNPs in the brain, although causality remains to be established.

The risk appears especially pronounced in neonates, where the presence of MNPs in breast milk, amniotic fluid, and meconium reflects early-life exposure. Multipotent stem cells in breast milk may serve as vectors for MNPs and EDCs, potentially transporting them into the neonatal brain and disrupting neurodevelopmental processes. Given the known sensitivity of the developing brain to environmental insults, these findings raise concerns about long-term consequences.

To advance understanding and protect public health, especially in children, several research priorities are essential:

1. longitudinal studies to assess long-term neurological effects of early-life MNPs exposure;
2. mechanistic investigations into how MNPs interact with neural cells, particularly in the context of inflammation, epigenetics, and oxidative stress;
3. development of standardized detection methods for MNPs in biological matrices, including neural tissues, placenta, breast milk, and blood;
4. policy actions aimed at reducing plastic production and human exposure – especially in vulnerable populations.

Ultimately, our goal should be to increase brain plasticity while decreasing plastic in the brain. As the neuroscience community continues to explore the brain's remarkable adaptability, it must also confront the environmental factors that threaten its integrity.

Declaration of interest

The Authors declare that there is no conflict of interest.

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