Ethiopian Journal of Environmental Studies & Management 19(3): 283 – 294, 2026
ISSN:1998-0507 doi: https://ejesm.org/doi/v19i3.3
Submitted: March 20, 2026 Accepted: May 29, 2026

EFFECTS OF MICROPLASTIC CONTAMINATION ON BRAIN HEALTH: A REVIEW

ANGELE, C. AND *OSAWE, S.O.
Department of Biological Sciences, Biochemistry Programme, Faculty of Science, Benson Idahosa University, Benin City Nigeria
*Corresponding author: sosawe@biu.edu.ng

Abstract
Microplastics (MPs), are small plastic particles less than 5 mm. They are pervasive environmental contaminants with emerging neurotoxic potential. MPs are generated from daily used products such as food packaging, textiles, and tire debris and comprise of various synthetic polymers, plasticizing additives (e.g bisphenol A and phthalates), and adsorbed pollutants. Human exposure to MPs occurs daily through ingestion, inhalation, and dermal contact. The potential of MP to cause neurological defects emanates from its capacity to traverse the blood-brain barrier (BBB), to induce oxidative stress, neuroinflammation, and neurotransmitter dysregulation, ultimately compromising neuronal integrity and predisposing individuals to neurodegenerative disorders including Alzheimer's and Parkinson's diseases. This review consolidates current experimental and epidemiological evidence on the sources, exposure routes, and core neurotoxic mechanisms of MPs, and proposes targeted mitigation strategies. An integrated understanding of these dimensions is essential for developing evidence-based environmental and public health policies.

Keywords: *Microplastics, Brain health, Blood-brain barrier, Neuroinflammation, Neurodegeneration, Oxidative stress*

Introduction

Microplastics (MPs) are defined as synthetic plastic particles, less than 5 mm in diameter. They were first identified in surface waters of the Atlantic in the 1970s (Colton *et al*., 1974), and named 'microplastics' by Thompson *et al*. (2004). Ever since, these pollutants have emerged as a global environmental threat to human health. The ubiquitous nature of MPs is a result of the continuous production, use, and improper disposal of plastic materials. MPs exist in terrestrial, aquatic, and atmospheric environments (Moulin and van Egmond, 2019).

The exposure of humans to MPs is continuous and occurs via ingestion of contaminated food and drinking water, inhalation of airborne particles, and dermal contact with consumer products. Senathirajah *et al*. (2021) estimated that between 39,000 and 52,000 MP particles are consumed annually by individuals through food and water alone; This figure rises to about 74,000 particles if they are also exposed to air-borne particles. Once absorbed, MPs enter the systemic circulation and can infiltrate major organs, including the brain.

The neurotoxic potential of MPs has attracted increasing scientific attention. MPs can cross the blood-brain barrier (BBB), accumulate in neural tissue, and initiate a cascade of pathological events - including oxidative stress, chronic neuroinflammation, disruption of neurotransmission, and protein aggregation - that collectively mirror the early mechanistic hallmarks of neurodegenerative diseases (Zheng *et al*., 2024; Bhattacharyya *et al*., 2025). Despite growing pre-clinical evidence, mechanistic understanding of MP-induced neurotoxicity in humans remains incomplete. This review synthesizes current evidence on the sources, exposure routes, brain infiltration pathways, and neurotoxic mechanisms of MPs, and discusses prospective mitigation strategies.

Types and Properties of Microplastics

Primary and Secondary Microplastics

MPs are classified into two categories based on their origin. Primary MPs are intentionally manufactured at small sizes (< 5 mm) and are incorporated into cosmetics, personal care products (face washes, toothpaste, exfoliants), pharmaceuticals, and industrial abrasives (Guerranti *et al*., 2019). Secondary MPs arise from the physical, chemical, and biological degradation of larger plastic items, including bottles, bags, packaging, and fishing nets through UV radiation, mechanical abrasion, and weathering (Habib *et al*., 2022). Secondary MPs are the most predominant type of MPs that pollute the environment. They exhibit considerable diversity in their chemical composition, resulting from a wide variety of their constituent synthetic polymers.

The most common types of synthetic polymers in secondary MPS are polyethylene (PE), polystyrene (PS), polypropylene (PP), polyvinyl chloride (PVC), polyethylene terephthalate (PET), and polyamide (PA) – see Figure 1. Because PVC can release dangerous additives like bisphenol A (BPA), phthalates, lead, and dioxins, known endocrine disruptors with potentially carcinogenic qualities- it is of special toxicological concern (Abahussain *et al*., 2025).

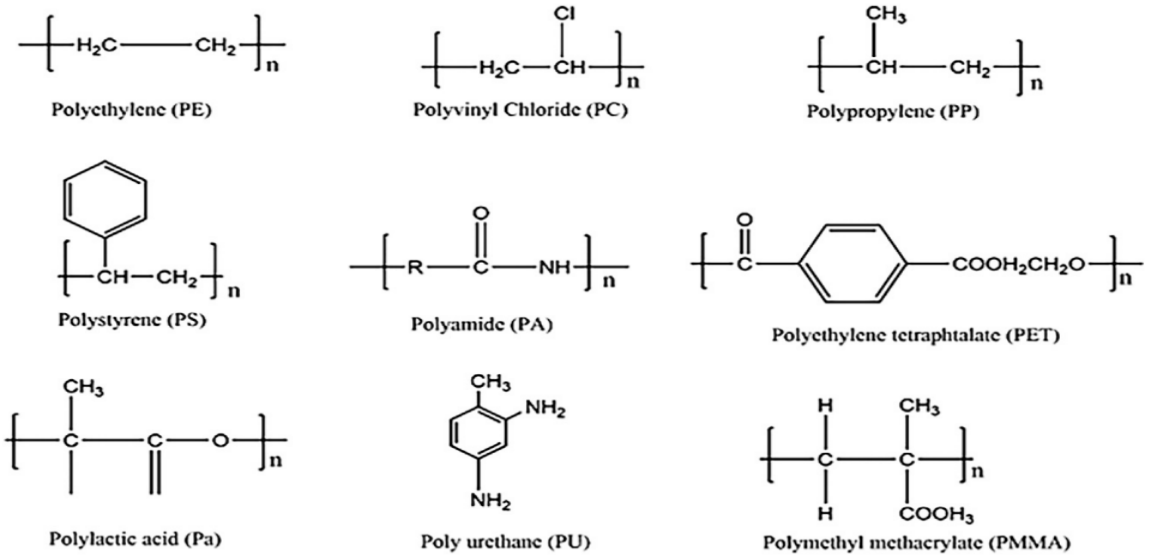

Fig. 1: Molecular structure of some polymers of microplastics (Bouwmeester *et al*., 2015)

Physical Properties and Morphology

Based on the diverse morphologies exhibited by MPs, they can be classified as fragments, fibres, films, foams, beads (microbeads), and nurdles as shown in Figure 2. Fibres originate during laundering of synthetic clothing and account for about 71% of MP pollution in aquatic environments (Allen *et al*., 2024).

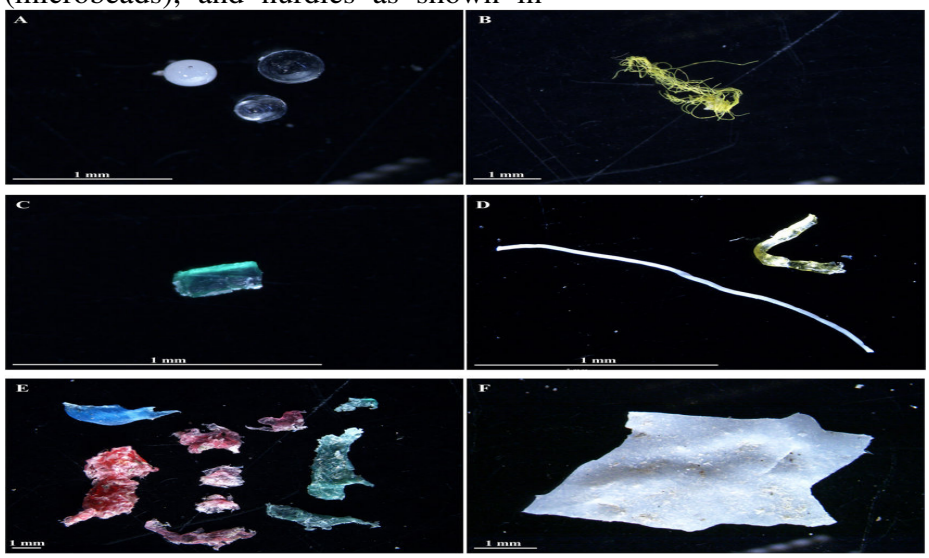

Fig. 2: Shapes of MPs A: Spheres (Pellets/Nurdles), B: Fibers, C: Fragments, D: Fibers (Long), E: Fragments (Colored), F: Film. *Adapted from* Dahl *et al*. (2021).

On the other hand, microbeads are non-biodegradable particles ≤ 1 mm in diameter, commonly found in personal care products. Single-use plastic items are the source of fragments, which can be further degraded by UV radiation. The extent of toxicity exhibited by MPs is directly related to their size. The smaller the size, the greater the potential it has to be toxic as they are more likely to penetrate tissues with particles < 10 µm crossing cell membranes. Those < 20 µm are usually phagocytosed, and those up to 130 µm undergo paracellular transport in intestinal epithelial cells (Leslie *et al*., 2022).

Routes of Human Exposure to Microplastics

Ingestion

Humans are principally exposed to MPs through ingestion. MPs are present in drinking water, plastic-bottled water, seafood, salt, tea bags, and foods in plastic packaging. It is estimated that about 0.66 million MP particles enter babies' bodies during one year of normal bottle feeding through rubber baby teats made of silicone, which degrade during steam sterilization (Su *et al*., 2022). MPs also enter the food chain via agricultural contamination from sewage sludge, compost, and plastic mulching, subsequently migrating into crop root systems and edible tissues (Rillig *et al*., 2020; Schwab *et al*., 2020).

Inhalation

Inhalation constitutes an important secondary route of exposure to MPs, with road wear borne MPs accounting for approximately 84% of airborne MPs (Brahney *et al*., 2020). PE, PS, and PET fiber MPs (size range 10–8,000 µm) have been identified in human lung tissue by Lourenço *et al*. (2021).

Dermal Contact

Dermal exposure, while generally considered of lower systemic importance given the skin barrier, is a very likely route

of exposure by users of personal care products that contain MP components, and typical additives, such as brominated flame retardants, bisphenols, triclosan and phthalates, that can be absorbed through the skin (Wu *et al*., 2022).

Toxic Effects of Microplastics on Human Health

The effect of MPs on multiple organ systems occurs through both physical and chemical mechanisms. MPs may induce Physical irritation, intestinal microbiome dysbiosis, and gut inflammation in the gastrointestinal tract, which manifests as abdominal pain, bloating, and altered bowel habits (Jin *et al*., 2019). When inhaled, MPs induce oxidative stress in the airways and can promote chronic obstructive pulmonary disease (COPD), particularly following exposure to high concentrations of polystyrene (Dong *et al*., 2020).

Chemical additives, which make up about 4% of the finished plastic product, are a crucial aspect that defines the toxic nature of MPs. Phthalates, BPA, and polycyclic aromatic hydrocarbons (PAHs) are among the additives that migrate to the particle surface and are released during environmental fragmentation. These additives are endocrine disruptors, which bind to hormone receptors and interfere with hormone synthesis and regulation, resulting in disorders related to reproduction, development, and metabolism (Danopoulos *et al*., 2020; Vandenberg *et al*., 2017). Additionally, MPs serve as vectors for the adsorption of heavy metals and hydrophobic organic compounds, increasing their toxic burden upon tissue penetration, because of their high surface area-to-volume ratio (Brahney *et al*., 2020).

Dynamics of Microplastic Toxicity on Brain Health

Pathways of Brain Infiltration

Emerging evidence confirms that MPs not only traverse the gastrointestinal tract but also enter systemic circulation and infiltrate the central nervous system (CNS), raising serious concerns about their involvement in the growing prevalence of neurological disorders (Zheng *et al*., 2024). MPs cross the BBB through several mechanisms: (i) Systemic translocation: smaller particles (< 0.5 μm) cross via transcytosis or by disrupting tight junction proteins claudin-5 and occludin, increasing paracellular permeability (Ho *et al*., 2025); (ii) The olfactory route: inhaled nanoparticles bypass the BBB entirely through retrograde axonal transport from the nasal epithelium to the olfactory bulb (Sofield *et al*., 2024); (iii) The gut-brain axis: intestinal inflammation and dysbiosis caused by MPs communicate neuroinflammatory signals to the brain through the vagus nerve (Prüst *et al*., 2020); and (iv) Biomolecular corona formation a cholesterol-rich protein corona that forms around particles upon body entry has been shown to facilitate BBB crossing significantly compared to bare particles (Kopatz *et al*., 2023).

Core Mechanisms of Neurotoxicity

Once accumulated in neural tissue, MPs initiate several converging pathological mechanisms:

Oxidative Stress and Mitochondrial Dysfunction: The most consistently documented mechanism of MP-induced brain damage is oxidative stress. MPs deplete cellular antioxidant enzymes, such as superoxide dismutase (SOD) catalase (CAT), and reduced glutathione (GSH), while inducing excess production of reactive oxygen species (ROS). This results in the peroxidation of the lipid-rich portions of membranes of neurons (Zheng *et al*., 2024). Even at low concentrations (0.05–10 mg/L) of MPs, human cerebral and epithelial cells (Schirinzi *et al*., 2017) are damaged by ROS generated. Furthermore, mitochondrial complexes I and III in neurons, are disrupted by MPs. This results in energy deficits (ATP loss) and initiates

apoptotic cascades (Bai *et al*., 2025), which leads to the ultimate death of neurons.

Chronic Neuroinflammation: Microglia, the brain's resident immune cells, identify MPs as foreign particles and attempt to phagocytose them. The inherent resistance of plastic to enzymatic degradation results in 'frustrated phagocytosis,' driving chronically activated microglia to continuously secrete pro-inflammatory cytokines, including Tumor Necrosis Factor-alpha (TNF-α) and interleukin - 1β (IL-1β), promoting a sustained neuroinflammatory state that accelerates neuronal death (Marcellus *et al*., 2024).

Disruption of Neurotransmission: MPs impairs the activity of acetylcholinesterase (AChE), causing it to accumulate in the synaptic cleft, causing motor and cognitive dysfunction (Hamed *et al*., 2022). The activation of microglia, inhibition of AChE, and elevation of circulating pro-inflammatory cytokines collectively culminate in *in vivo* neurotoxicity (Jayavel *et al*., 2024).

Links to Neurodegenerative Diseases

The molecular mechanisms underlying MP-induced neurotoxicity, oxidative stress, protein aggregation, and chronic inflammation overlap substantially with the pathogenic hallmarks of Alzheimer's disease (AD) and Parkinson's disease (PD). High MP concentrations have been associated with fibrillization of α-synuclein (central to PD pathology) and deposition of amyloid-β plaques (central to AD pathology) (Da Silva *et al*., 2025). A notable post-mortem study by Bhattacharyya *et al*. (2025) reported a 50% increase in brain MP concentrations compared to samples from 2016, with significantly higher MP loads found in individuals who died from dementia. In experimental models, polystyrene nanoplastics (50 nm) were shown to accumulate in the placenta and impair neurodevelopment in fetal mice (Liang *et al*., 2025), while multi-polymer MP exposure in rats caused loss of dopaminergic neurons and Parkinson-like motor deficits (Ho *et al*., 2025).

MPs also serve as significant carriers of heavy metals, whose biofilms accumulate metal ions at the particle surface. Disruption of metal homeostasis in the CNS initiates cascades resulting in mitochondrial dysfunction, protein misfolding, and neurodegeneration (Jurek, 2024).

Experimental and Epidemiological Evidence

Various experimental models support pre-clinical evidence that MP-induces neurotoxicity across multiple species. Deng *et al*. (2017) confirmed that MPs accumulate in the liver, kidneys, and gut of mice in a size-dependent manner, leading to disrupted energy and lipid metabolism and inducing neurotoxic responses. Sincihu *et al*. (2023) demonstrated that low-density polyethylene MPs significantly reduced SOD activity and elevated malondialdehyde (MDA) levels in Wistar rats, causing neuronal membrane and DNA damage alongside reduced serum amyloid beta 1-42 (Aβ42) levels, a cognitive biomarker implicated in Alzheimer's disease. Human cerebral organoid studies have further shown that nano-PVC (< 1 μm) inhibits neural progenitor cell proliferation, induces premature cellular aging markers, and disrupts synaptic network formation (Adamiak *et al*., 2025).

Table 1. Cross-Species Analysis of Microplastic Exposure on Brain Function and Behavioural Phenotypes (2019–2025)

Experimental Model	Particle Type & Size	Brain Region	Observed Effects	References
Velvet crab	PS, 0.5 µm	Brain tissue	Altered gill-to-brain signalling	Crooks *et al*. (2019)
Zebrafish	PE, 1–10 µm	Whole brain / Optic tectum	Reduced locomotion; decreased AChE and MAO activity; neuronal degeneration	Hamed *et al*. (2022)
Adult mice	PS, < 5 µm	Hippocampus & cerebral cortex	Impaired spatial memory; anxiety-like behaviour; reduced nesting drive	Zhou *et al*. (2023)
Honeybees	PS/PMMA, 1–5 µm	Mushroom bodies	Impaired learning; reduced homing efficiency	Pasquini *et al*. (2024)
Fetal mice	PS-NPs, 50 nm	Whole brain	Placental accumulation; impaired neurodevelopment of offspring	Liang *et al*. (2025)
Rats	Multi-polymer mix	Striatum	Loss of dopaminergic neurons; Parkinson-like tremors; defective motor coordination	Ho *et al*. (2025)
Human organoids	Nano-PVC, < 1 µm	Neural progenitor cells	Inhibited proliferation; premature aging markers; disrupted synaptic networks	Adamiak *et al*. (2025)

Mitigation Strategies and Future Directions

Several complementary strategies have been proposed for managing MP exposure and environmental pollution, each addressing different points in the plastic life cycle. Reduction of plastic production and usage remains the most upstream and effective intervention. Minimizing microbeads in personal care products and pharmaceuticals reduces the volume of primary MPs entering aquatic systems (Prata, 2018). This approach follows a summarized reverse pyramid model and revolving wheel as shown in Figure 3, encompassing the 7 R's: Refuse, Reduce, Reuse, Recycle, Rethink, Regift, and Recover; a hierarchical framework for minimizing plastic waste release at source (Reis *et al*., 2023; Glavič, 2021).

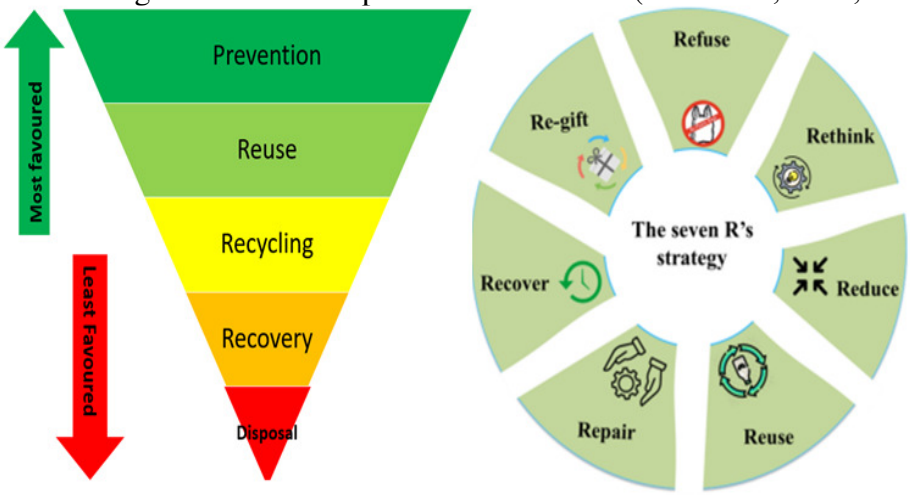

Fig. 3: Mitigation against Microplastics Contamination. *Adapted from:* Kumar *et al*. (2021)

Behavioural and lifestyle modifications can substantially curtail individual MP exposure. These include selecting natural fibre clothing (cotton, wool) over synthetic alternatives (nylon, acrylic, polystyrene), installing MP-capture filters in domestic washing machines, choosing glass over plastic containers for food and beverages, and using natural-ingredient cosmetics and personal care products (De Falco *et al*., 2019; Osman *et al*., 2023).

Biodegradable polymer substitution represents a promising technological frontier. Bioplastics, including polyhydroxyalkanoates, are already deployed in agricultural mulching films, pharmaceutical packaging, and automotive components (Filiciotto and Rothenberg, 2021). Complementary emerging technologies include sunlight-activated photocatalytic degradation, plastic-to-fuel conversion, and microbial biodegradation pathways, which collectively represent the next generation of MP remediation tools (Lamichhane *et al*., 2023). From a neurological research perspective, future investigations should prioritize: (i) longitudinal human epidemiological studies examining MP brain burden and neurodegenerative disease incidence; (ii) mechanistic elucidation of MP-induced BBB disruption and neuroinflammatory signalling at physiologically relevant exposure concentrations; (iii) investigation of synergistic effects between MP-adsorbed pollutants (heavy metals, POPs) and primary neurotoxicity; and (iv) development of standardized

biomarkers for assessing MP-associated neurological risk in clinical settings.

CONCLUSION

The persisting nature of microplastics in the environment constitutes a growing threat with significant implications for brain health. They can cross the BBB and, through diverse routes, accumulate in neural tissue, initiating a cascade of oxidative stress, chronic neuroinflammation, dysregulated neurotransmission, and protein aggregation, which converge in the pathogenesis of Alzheimer's and Parkinson's diseases. Post-mortem evidence of elevated brain MP loads, particularly in dementia patients, lends clinical urgency to pre-clinical experimental findings. While there is a paucity of information regarding dose-response relationships and long-term effects in humans, the existing body of evidence warrants the adoption and implementation of immediate preventive and remediation strategies. Preserving brain health throughout life requires integrating aspects of MP neurotoxicology and mitigation strategies into environmental health policy, as well as reducing primary plastic production while expediting the development of biodegradable alternatives.

REFERENCES

Abahussain, A.A.M., Nasr, F.A., Bin Jumah, A., Saravanan, P., Kumar, N.S., Al-Zharani, M., Guganathan,L., Sasikumar, G., Alsalamah, S.A., Qurtam, A.A., Senthilkumar, J. and Tamizhdurai, P. (2025). Toxic threats from plastic waste: Human health impacts, challenges, and policy solutions. *RSC Advances*, 15: 40761–40788.

Adamiak, K., Sidoryk-Węgrzynowicz, M., Dąbrowska-Bouta, B., Sulkowski, G. and Strużyńska, L. (2025). Primary astrocytes as a cellular depot of polystyrene nanoparticles. *Scientific Reports*, 15(1): 6502.

Allen, E., Henninger, C. E., Garforth, A. and Asuquo, E. (2024). Microfiber pollution: A systematic literature review. *Environmental Science and Technology*, 58(9): 4031–4045.

Lourenço, L.F.A., Oliveira, R.C.D., Ribeiro Júnior, G., Galvão, L.D.S., Ando, R.A. and Mauad, T. (2021). Presence of airborne microplastics in human lung tissue. *Journal of Hazardous Materials*, 416: 1-6.

Bai, H., Wu, Y., Li, H., Zhu, Y., Che, R., Wang, F. and Zhang, C. (2024). Cerebral neurotoxicity of amino-modified polystyrene nanoplastics in mice. *Science of the Total Environment*, 912: 169511.

Bouwmeester, H., Hollman, P.C. and Peters, R.J. (2015). Potential health impact of environmentally released micro- and nanoplastics in the human food production chain: experiences from Nano toxicology. *Environmental Science Technology*, 49 (15): 8932-8947.

Brahney, J., Hallerud, M., Heim, E., Hahnenberger, M. and Sukumaran, S. (2020). Plastic rain in protected areas of the United States. *Science,* 368(6496): 1257–1260.

Colton, J.B., Knapp, F.D. and Burns, B.R. (1974). Plastic particles in surface waters of the northwestern

Atlantic. *Science*, 185(4150): 491–497.

Crooks, N., Parker, H., and Pernetta, A. P. (2019). Brain food? Trophic transfer and tissue retention of microplastics by the velvet swimming crab (*Necora puber*). *Journal of Experimental Marine Biology and Ecology*, 519: 151187.

Dahl, M., Bergman, S., Björk, M., Diaz-Almela, E., Granberg, M., Gullström, M., Leiva-Dueñas, C., Magnusson, K., Marco-Méndez, C., Piñeiro-Juncal, N. and Mateo, M.Á. (2021). A temporal record of microplastic pollution in Mediterranean seagrass soils. *Environmental Pollution*, 273: 116451.

Danopoulos, E., Twiddy, M. and Rotchell, J.M. (2020). Microplastic contamination of drinking water: A systematic review. *PLoS ONE*, 15(7): e0236838.

De Falco, F., Di Pace, E., Cocca, M. and Avella, M. (2019). The contribution of washing processes of synthetic clothes to microplastic pollution. *Scientific Reports*, 9: 6633.

Deng, Y., Zhang, Y., Lemos, B. and Ren, H. (2017). Tissue accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure. *Scientific Reports*, 7(1): 46687.

Filiciotto, L., and Rothenberg, G. (2021). Biodegradable plastics: Standards, policies, and impacts. *ChemSusChem*, 14(1): 56–72.

Glavič, P. (2021). Evolution and Current Challenges of Sustainable Consumption and Production. *Sustainability*, 13(16): 9379.

Guerranti, C., Martellini, T., Perra, G., Scopetani, C. and Cincinelli, A. (2019). Microplastics in cosmetics: Environmental issues and needs for global bans. *Environmental Toxicology and Pharmacology*, 68: 75–79.

Habib, R.Z., Aldhanhani, J.A.K., Ali, A.H., Ghebremedhin, F., Elkashlan, M., Mesfun, M., Kittaneh, W., Al Kindi, R. and Thiemann, T. (2022). Trends of microplastic abundance in personal care products in the United Arab Emirates. *Environmental Science and Pollution Research,* 29: 89614–89624.

Hamed, M., Soliman, H., Osman, A., and Sayed, A. (2022). Microplastics and nanoplastics toxicity in fish: A review. *Environmental Science and Pollution Research*, 29: 51231–51247.

Ho, K. H., Yeh, J.-Y., Tu, T.-Y., Lin, C.-W., Lo, Y.-C., Wang, H.-C., Yang, Y.-F., Huang, C.-W., and Park, C. (2025). Exposure to nanoplastics disrupts neurotransmitter release in rat hippocampal neurons. *Environment and Health*. https://doi.org/10.1021/envhealth.5c00477.

Jayavel, S., Govindaraju, B., Michael, J. R., and Viswanathan, B. (2024). Impacts of micro and nanoplastics on human health. *Bulletin of the National Research Centre*, 48: 110.

Jin, Y., Lu, L., Tu, W., Luo, T. and Fu, Z. (2019). Impacts of polystyrene microplastic on the gut barrier, microbiota and metabolism of

mice. *Science of the Total Environment*, 649: 308–317.

Jurek, A. (2024). Metal homeostasis disruption as a mechanism of microplastic-induced neurodegeneration. *Neurotoxicology*, 101: 1–12.

Kopatz, V., Wen, K., Kovács, T., Keimowitz, A.S., Pichler, V., Widder, J., Vethaak, A.D., Hollóczki, O. and Kenner, L. (2023). Micro-and nanoplastics breach the blood–brain barrier (BBB): Biomolecular corona's role revealed. *Nanomaterials*, 13(8): 1404.

Kumar, R., Verma, A., Shome, A., Sinha, R., Sinha, S., Jha, P.K., Kumar, R., Das, S., Sharma, P. and Prasad, P.V.V. (2021). Impacts of plastic pollution on ecosystem services, sustainable development goals, and need to focus on circular economy and policy interventions. *Sustainability*, 13(17): 9963.

Lamichhane, S., Bal Krishna, K.C. and Sarukkalige, R. (2023). Microplastics in environment: Global concern, challenges, and controlling measures. *Science of the Total Environment*, 845: 157247.

Leslie, H.A., van Velzen, M.J.M., Brandsma, S.H., Vethaak, A.D., Garcia-Vallejo, J.J., and Lamoree, M.H. (2022). Discovery and quantification of plastic particle pollution in human blood. *Environment International*, 163: 107199.

Liang, X., Zeng, Y., Zhang, P., Zhu, B., Feng, J., Deng, T., Fu, Z., Liu, C., Chen, C. and Zhang, Y. (2025). Polystyrene nanoplastics trigger pyroptosis in dopaminergic neurons in Parkinson's disease. *Journal of Translational Medicine*, 23(1): 631.

da Silva, I.A.N., Paulus, A., Skoryk, V., Su, K.Y., Herranz-Trillo, F. and Klementieva, O. (2025). Microplastic Exposure Promotes Amyloid Misfolding and Metabolic Impairment at Sub-Lethal Doses in an In Vitro Cellular Model of Alzheimer's Disease. *bioRxiv*, 2025-12.

Marcellus, T., Rivera, E. and Jiang, W. (2024). Frustrated phagocytosis and chronic neuroinflammation: Microglial responses to non-degradable plastic particles. *Neuroinflammation Research*, 11(2): 45–58.

Moulin, L. and van Egmond, R. (2019). Microplastics in the environment: Occurrence, distribution, and potential impacts. JRC Technical Report. Luxembourg: Publications Office of the European Union.

Osman, A.I., Hosny, M., Eltaweil, A.S., Omar, S., Elgarahy, A.M., Farghali, M., Yap, P.S., Wu, Y.S., Nagandran, S., Batumalaie, K., Gopinath, S.C.B., John, O.D., Sekar, M., Saikia, T., Karunanithi, P., Hatta, M.H.M. and Akinyede, K.A (2023). Microplastic sources, formation, toxicity and remediation: A review. *Environmental Chemistry Letters*, 21: 2125 - 2160.

Pasquini, E., Ferrante, F., Passaponti, L., Pavone, F.S., Costantini, I. and Baracchi, D. (2024). Microplastics reach the brain and interfere with honey bee cognition. *Science of the Total Environment*, 912: 169362.

Prata, J.C. (2018). Airborne microplastics: Consequences to human health? *Environmental Pollution*, 234: 115–126.

Prata, J.C., da Costa, J.P., Lopes, I., Duarte, A. C. and Rocha-Santos, T. (2020). Environmental exposure to microplastics: An overview on possible human health effects. *Science of the Total Environment*, 702: 134455.

Prüst, M., Meijer, J. and Westerink, R.H. S. (2020). The plastic brain: Neurotoxicity of micro- and nanoplastics. *Particle and Fibre Toxicology*, 17: 24.

Reis, W.F., Barreto, C.G. and Capelari, M.G.M. (2023). Circular Economy and Solid Waste Management. *Sustainability*, 15(22): 15715.

Rillig, M.C. and Lehmann, A. (2020). Microplastic in terrestrial ecosystems. *Science*, 368(6498): 1430–1431.

Sangkham, S., Faikhaw, O., Munkong, N., Sakunkoo, P., Arunlertaree, C., Chavali, M., Mousazadeh, M. and Tiwari, A. (2022). A review on microplastics and nanoplastics in the environment. *Marine Pollution Bulletin*, 181: 113832.

Schirinzi, G.F., Pérez-Pomeda, I., Sanchís, J., Rossini, C., Farré, M. and Barceló, D. (2017). Cytotoxic effects of commonly used nanomaterials and microplastics on cerebral and epithelial human cells. *Environmental Research*, 159: 579–587.

Schwab, F., Zhai, G., Kern, M., Turner, A., Schnoor, J.L. and Wiesner, M.R. (2016). Barriers, pathways and processes for uptake, translocation and accumulation of nanomaterials in plants–Critical review. *Nanotoxicology*, 10(3), pp.257-278.

Senathirajah, K., Attwood, S., Bhagwat, G., Carbery, M., Wilson, S. and Palanisami, T. (2021). Estimation of the mass of microplastics ingested–A pivotal first step towards human health risk assessment. *Journal of Hazardous Materials*, 404:124004.

Sincihu, Y., Lusno, M.F.D., Mulyasari, T.M., Elias, S.M., Sudiana, I.K., Kusumastuti, K., Sulistyorini, L. and Keman, S. (2023). Wistar rats' hippocampal neurons response to blood low-density polyethylene microplastics. *Neuropsychiatric Disease and Treatment*, 19: 73–83.

Sofield, C. E., Anderton, R. S., and Gorecki, A. M. (2024). Mind over microplastics: Exploring microplastic-induced gut disruption and gut-brain-axis consequences. *Current Issues in Molecular Biology,* 46(5): 4186–4202.

Su, Y., Hu, X., Tang, H., Lu, K., Li, H., Liu, S., Xing, B. and Ji, R. (2022). Steam disinfection releases micro (nano) plastics from silicone-rubber baby teats as examined by optical photothermal infrared microspectroscopy. *Nature nanotechnology*, 17(1): 76-85.

Thompson, R.C., Olsen, Y., Mitchell, R. P., Davis, A., Rowland, S.J., John, A.W.G., McGonigle, D. and Russell, A.E. (2004). Lost at sea: Where is all the plastic? *Science*, 304(5672): 838.

Vandenberg, L.N., Colborn, T., Hayes, T.B., Heindel, J.J., Jacobs, D.R., Jr, Lee, D.H., Shioda, T., Soto, A.M., vom Saal, F.S., Welshons,

W.V., Zoeller, R.T. and Myers, J.P. (2012). Hormones and endocrine-disrupting chemicals: low-dose effects and nonmonotonic dose responses. *Endocrine Reviews*, 33(3), 378–455.

Wu, P., Huang, J., Zheng, Y., Yang, Y., Zhang, Y., He, F., Chen, H., Quan, G., Yan, J., Li, T., Gao, B. and Pan, X. (2022). Environmental occurrences, fate, and impacts of microplastics. *Journal of Hazardous Materials*, 424: 127337.

Zheng, Y., Xu, S., Liu, J. and Liu, Z. (2024). The effects of micro- and nanoplastics on the central nervous system. *Toxicology*, 504: 153799.

Zhou, Y., Wu, Q., Li, Y., Feng, Y., Wang, Y. and Cheng, W. (2023). Low-dose of polystyrene microplastics induce cardiotoxicity in mice and human-originated cardiac organoids. *Environment International*, 179: 108171.