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COMMENTARY

Human microplastic removal: what does the evidence tell us?

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The increased levels of microplastics and nanoplastics (MNPs) found in human brain tissue are alarming, particularly in patients with dementia. Although total avoidance of MNP exposure will likely remain an unattainable endpoint in light of their ubiquity in the environment, new studies indicate feasible pathways by which dietary intake may be decreased or clearances improved. This commentary reviews the evidence on human exposure to MNPs, their tissue penetration, and potential health effects, particularly on neurotoxicity. We will explore evidence-based strategies for reducing exposure through dietary and lifestyle changes while addressing key gaps in our current knowledge calling for additional research.

A recent paper in *Nature Medicine* by Nihart et al. found that the human brain contains approximately a spoon's worth of microplastics and nanoplastics (MNPs), with levels 3–5 times higher in those with a cohort of decedent brains with a documented dementia diagnosis (with notable deposition in cerebrovascular walls and immune cells) (1). Particularly, brain tissues were found to have 7–30 times higher amounts of MNPs than other organs such as the liver or kidney. Also of note, the microplastics in the brain were of a smaller size (<200 nm) and most often polyethylene. Although MNP concentration was not influenced by factors such as age, sex, race, or cause of death, there was a worrisome 50% increase in MNP concentration based on the time of death (2016 versus 2024).

This aligns with the observed exponential increase in MNP environmental concentrations over the past half-century (2). Particularly, 10 to 40 million tonnes of emissions of microplastics to the environment are estimated per year, with this figure expected to double by 2040 (3). Wind and water can redistribute microplastics and have since been reported in diverse locations from the deep sea sediments to our highest mountains (3). Microplastics are pervasive in the food we eat, the water we drink, and the air we breathe (3). Humans are exposed to MNPs through various routes, but their impact on various organ systems is not fully understood (4).

The current evidence base (largely based upon animal and cell culture studies) suggests that MNP exposure can lead to adverse health impacts via oxidative stress, inflammation, immune dysfunction, altered biochemical/energy metabolism, impaired cell proliferation, abnormal organ development, disrupted metabolic pathways, and carcinogenicity (4). These can lead to direct or indirect consequences to various organ systems, including respiratory, gastrointestinal, cardiovascular, hepatic, renal, nervous, reproductive, immune, endocrine, and muscular (4). Particularly, a recent study in *The New England Journal of Medicine* found that people with a carotid artery plaque in which MNPs were detected had a higher risk of myocardial infarction, stroke, or all-cause mortality (5). Additionally, inflammatory bowel disease (IBD) patients' stool contained about 1.5 times more microplastics than healthy controls, averaging 41.8 vs. 28.0 particles per gram of dry stool (6). However, the underlying mechanisms and whether long-term exposure to MNPs is associated with disease susceptibility is an area that requires further investigation.

Due to the higher concentration in the brain, the 3–5 times higher amount in brains with dementia, specific attention should be given to the nanoparticles <200 nm, predominantly polyethylene, found in the brain. In a study on fish, nanoplastics reduced swimming activity and hunting (predatory) performance (7). A study of mice exposed for 8 weeks led to learning and memory deficits, lower levels of synaptic proteins, and neuroinflammation (8). In human studies, the significance of elevated microplastic levels in patients with dementia remains unclear. Is dementia weakening the blood-brain barrier, allowing more microplastics to enter? Or do microplastics, once inside, trigger microinflammation and make it harder for the brain to clear proteins, potentially worsening neurodegeneration?

Given the widespread presence of microplastics in the environment, completely eliminating exposure is unrealistic. A more practical approach is to reduce the most significant sources of microplastic intake. Switching from bottled water to tap water could reduce microplastic intake from 90,000 to 4,000 particles per year, making it an impactful intervention (9). However, while reducing intake is a logical approach, it remains unclear whether this translates into a measurable reduction in microplastic accumulation within human tissues. Beyond bottled water, significant dietary sources of microplastics are alcohol and seafood.

Stopping the practice of heating food in plastic could be one of the most effective ways to reduce microplastic consumption. Tea bags are often plastic, and a study found that despite being labeled food grade released a total of 16 μm of micro and nanoplastics (2.4 million micron-sized particles 1–150 μm and 14.7 billion submicron plastic particles <1 μm) (10).

Additionally, food storage may contribute to microplastic exposure. A randomized crossover trial of canned foods showed a more than 1000% rise in urinary bisphenol A (BPA) levels after five days of daily canned soup intake (11). This suggests that limiting canned food consumption and opting for non-plastic or BPA-free packaged alternatives can effectively reduce exposure. These BPA spikes' duration and health impact remain unclear, warranting further research.

Highly processed foods, like chicken nuggets, contained 30 times more microplastics per gram than chicken breasts, highlighting the impact of industrial processing, which often uses plastics at some point (12). One RCT looking at depression outcomes showed that eliminating 21.76 servings/week of highly processed foods per week (often stored in plastics) had a reduction in depression >1 effect size (13). The study concluded that the reduction in depression was due to a more Mediterranean diet. However, it is possible that the diet also lowered microplastic intake, contributing to improved brain health, though this was not directly examined in the study (14).

Inhalation is another substantial source of exposure, with up to 62000 particles in male adults per year. A High-Efficiency Particulate Air (HEPA) filter removes up to 99.97% of airborne particles as small as 0.3 μm, which

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includes a significant amount of airborne microplastics, though data on whether this translates to meaningful changes in absorption and outcomes humans is lacking (15).

Avoiding plastic and opting for glass or stainless steel containers may reduce intake. Heating food in plastic containers, especially in the microwave, can release staggering amounts of microplastics and nanoplastics—up to 4.22 million and 2.11 billion particles per square centimeter in just three minutes (16). Even long-term storage at room temperature or in the fridge leads to significant plastic shedding. These plastics show toxic potential, with in vitro studies revealing up to 77% cell death in human kidney cells after prolonged exposure (16).

There is scarce evidence on the effective removal of microplastics once they have been ingested. One study of 20 individuals measured BPA in blood, sweat, and urine. BPA is a chemical compound used in the production of plastics, which is released when plastics degrade. 16 of the individuals had BPA identified in their sweat, with this being the only identified source of BPA in some individuals. This suggests that induced sweating could facilitate the removal of BPA, though further studies are required to investigate its efficacy and long-term implications (17). While some evidence suggests that sweat may facilitate the excretion of certain plastic-derived compounds like BPA, no direct research currently confirms its role in reducing the microplastic burden in humans. Further studies are needed to determine whether these strategies are effective in eliminating microplastics from the body.

Future research should prioritize establishing clear exposure limits and assessing the long-term health consequences of microplastic intake. Large-scale human studies are needed to determine the dose-response relationship between microplastic exposure and chronic health outcomes such as endocrine disorders and cognitive disease. Standardizing biomonitoring methods to track microplastic accumulation in tissues will also be essential for understanding their physiological impact and association with other diseases, ideally in cohort studies controlling for both intake variables like use of plastics, types of foods consumed, but also excretion (frequency of sweating in sauna and exercise). In parallel, studies should further evaluate the effectiveness of various reduction and elimination strategies.

One of the most hopeful aspects of the findings to date is the lack of correlation between age and microplastic accumulation, suggesting that despite ongoing environmental exposures, the body has mechanisms to clear these particles over time through sweat, urine, and feces. As methods for measuring microplastics in living humans improve, we can test the common-sense hypothesis that reducing intake of microplastics (e.g., drinking tap water, avoiding plastic tea bags, using metal or glass for cooking and storage, minimizing highly processed foods stored in plastic) and enhancing elimination may reduce accumulation in humans. In fish models, it takes approximately 70 days to clear 75% of accumulated brain microplastics, suggesting that decreased inputs and increased outputs must both be maintained for long enough durations to see measurable changes (18). As the knowledge increases, government-wide initiatives will help us reduce exposure.

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