

Microplastics and mental health: The role of ultra-processed foods

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Ultra-processed foods now dominate the food supplies of high-income countries, with over 50% of energy intake coming from ultra-processed foods in the United States. Observational data has revealed that greater ultra-processed food consumption is associated with adverse mental health outcomes, while data from randomized controlled trials has demonstrated improvements to mental health following reduction in ultra-processed food intake. Ultra-processed foods are known to contain high concentrations of microplastics, largely due to both the processing and packing procedures. In light of recent findings which demonstrated alarming microplastic concentrations in the human brain, we propose that microplastics may partially mediate the adverse mental health effects of increasing ultra-processed food intake. In this viewpoint, we discuss the overlapping mechanisms for adverse mental health, paucity of research in the area, and propose a Dietary Microplastic Index (DMI) to study this potential relationship.

Using the Nova food classification system, ultra-processed foods are industrial formulations made almost entirely from substances extracted from foods, derived from constituents of foods, or synthesized in laboratories. Examples include instant noodles, carbonated drinks, and packaged foods (1). Ultra-processed foods now dominate the food supplies of high-income countries, such as the United States and Canada, and their consumption is rapidly increasing in middle-income countries (2). Particularly, the United States has one of the highest percent energy intake from ultra-processed foods at over 50% (3). This shift from whole foods to ultra-processed foods is largely driven by transnational food manufacturing, extremely profitable fast food corporations, and heavily promoted ultra-processed foods in the form of snacks (2).

A recent umbrella review in *The BMJ*—including nearly 10 million participants—found that people who consumed ultra-processed foods had a 22% higher risk of depression, 48% higher risk of anxiety, and 41% higher risk of poor sleep outcomes, among numerous adverse physical health outcomes (4, 5). On the contrary, evidence has demonstrated that those who adhere to a nutrient dense diet, primarily of unprocessed foods, are at a lower risk of adverse mental health outcomes (6). Beyond observational research, small randomized controlled trials have demonstrated moderate-to-large improvements in depressive symptoms with a Mediterranean diet compared to controls in those with depression (7). As such, from both observational and interventional research, there is a clear pattern between dietary intake and mental health.

The associations between ultra-processed foods and adverse mental health are complex and multifaceted. From a biological perspective, numerous mechanisms—largely identified through animal studies—are likely at play including inflammation, oxidative stress, epigenetics, mitochondrial dysfunction, the tryptophan–kynurenine metabolism, the hypothalamic–pituitary–adrenal axis, neurogenesis (via brain-derived neurotrophic factor), epigenetics, and chronic diseases such as obesity

(8). This likely arises from their poor nutrient profiles, energy density, and the physical/chemical properties associated with industrial processing and packaging methods, which introduce bisphenols and microplastics as contaminants (4). Interestingly, microplastics share similar mechanisms for their adverse health effects via oxidative stress, inflammation, immune dysfunction, altered biochemical/energy metabolism, impaired cell proliferation, abnormal organ development, disrupted metabolic pathways, and carcinogenicity (9). With particular attention to the central nervous system, microplastics and nanoplastics can induce oxidative stress, which may cause cellular damage and increase vulnerability to neuronal disorders. Particularly, microplastics have been demonstrated to influence neurotransmitters such as acetylcholine, γ -aminobutyric acid, and glutamate, which are commonly implicated in neuropsychiatric disorders (9). Although, the above mechanisms are largely based upon animal and cell culture studies.

High concentrations of microplastics are found within ultra-processed foods, largely due to both the processing and packing process (10, 11). For example, foods like chicken nuggets contain 30 times more microplastics per gram than chicken breasts—highlighting the impact of industrial processing on the content of the food (12, 13). Further, ultra-processed foods are often stored and heated in plastic, which independently serve as a significant source of microplastic exposure. Particularly, some plastic containers can release as many as 4.22 million microplastic and 2.11 billion nanoplastic particles from only one square centimeter of plastic area within 3 min of microwave heating (14). Beyond microplastics, Bisphenol A (BPA), a chemical compound used in the production of plastics, which is released when plastics degrade, is commonly found in packaging for ultra-processed foods (15). Therefore, the consumption of ultra-processed foods may serve as a significant risk factor for microplastic and BPA accumulation within humans.

Up until recently, most research on microplastic accumulation and human health has focused on correlations between physical health outcomes such as myocardial infarction, stroke, irritable bowel disease, and death (9, 16, 17). It was not until a study in *Nature Medicine* found that the human brain contains approximately a spoon's worth of microplastics, with levels three to five times higher in those with a documented dementia diagnosis (although this does not demonstrate causality) (18). The microplastics in the brain were smaller (<200 nm), most often polyethylene, and were 7 to 30 times higher than those in other organs such as the liver or kidney. This study also found a 50% increase in microplastic concentration based on time of death, from 2016 to 2024, which parallels the ongoing rise of ultra-processed foods available. For BPA, associations have been found with mental disorders such as autism, depression, and anxiety (19, 20). No evidence currently exists (in humans) for microplastic accumulation and other mental health outcomes, partially due to the difficulty in quantifying microplastic exposures from an observational perspective and ethics surrounding microplastic exposure from an interventional perspective.

The accumulation of a substantial quantity of microplastics in the brain and throughout the body raises significant health concerns. Emerging evidence suggests potential effects on immune function, genetic





stability, and endocrine regulation, making it reasonable to expect that such widespread deposition could have adverse impacts on both mental and physical health (21, 22). As ultra-processed foods, which contain significant microplastic content, represent over half of energy intake in the United States, with simultaneous rise in the rates of depression, it is imperative that this link be further examined (3, 23).

The first study that propelled the field of Nutritional Psychiatry was the SMILES trial (24). It was a 12-week, parallel-group, randomized controlled trial of adjunctive dietary intervention for the treatment of moderate to severe depression. Beyond depression, the participants were confirmed to have a “poor” dietary quality through the use of the Dietary Screening Tool (DST) (25). In this study, the dietary intervention largely focused on replacing nutrient poor, ultra-processed foods with nutrient dense, unprocessed alternatives. Sixty-seven people were randomized to a dietary intervention ($n = 33$) or control ($n = 34$) setting, with depression symptomatology, as measured by the Montgomery-Åsberg Depression Rating Scale (MADRS), serving as the primary endpoint at 12 weeks. The dietary support group eliminated 21.76 processed foods per week and demonstrated significantly greater improvements at 12 weeks on the MADRS than the control group, whereby remission was achieved for 32.3% and 8.0% of the intervention and control groups, respectively, with a number needed to treat of 4.1. While the aim of this study was to improve the overall nutritional value as an intervention, there was likely a direct reduction in microplastic intake as a result of these aforementioned substitutions. However, as microplastic exposure was not directly measured, this remains a hypothesis requiring further investigation.

Retroactively, for the SMILES trial (24) or the numerous other randomized controlled trials (7) that have since been conducted in the field of nutritional psychiatry, it would be of utmost value if post-hoc analyses could be conducted estimating the change in microplastic content due to dietary interventions, and their subsequent effect on various mental health outcomes. This may become increasingly possible as more research quantifying the microplastic content of various ultra-processed food items is readily available (12, 13). From an observational perspective, no nutritional population-based surveys currently estimate or track microplastic intake via diet, which precludes analysis of long-term microplastic exposure via diet and adverse mental health outcomes. Perhaps, similar to the Dietary Inflammatory Index (DII), which is used to assess the inflammatory potential of a person's diet based on the foods they consume (26) or the Nova food classification system, which categorizes foods based on the extent and purpose of industrial processing (1), a Dietary Microplastic Index (DMI) can be developed or integrated into existing dietary-based risk indices, to assess the microplastic content and risk of accumulation based on the foods consumed.

Overall, as the levels of ultra-processed foods, microplastics, and adverse mental health outcomes simultaneously rise, it is imperative that we further investigate this potential association. After all, you are what you eat.

Author Contributions

Nicholas Fabiano was responsible for the conceptualization and writing the original draft. All authors participated in review/editing and approved the final version of the manuscript.

Author Disclosures

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