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Response

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Response: Ultra-Processed Food Consumption and Obesity: A Narrative Review of Their Association and Potential Mechanisms (J Obes Metab Syndr 2025;34:27-40)

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In the article entitled 'Ultra-processed food consumption and obesity: a narrative review of their association and potential mechanisms,'[1] we described a global surge in ultra-processed food (UPF) consumption, a trend that is projected to persist, despite variation across regions. By synthesizing epidemiological evidence from a broad range of observational and experimental studies, we confirmed a strong association between high UPF intake and increased risk of obesity and weight gain. This relationship is hypothesized to be driven not only by the obesogenic nutritional profile of UPFs but also the intrinsic properties of food processing itself. The current collective evidence provides a strong basis for limiting UPF consumption as a key component of obesity prevention and treatment, although further studies are needed to elucidate the precise etiology of UPF-induced obesity.

In this letter to the editor, the author concurred with the points raised in our review but emphasized the critical link between diet and brain health, advocating expanding the discussion on UPFs beyond obesity and cardiovascular outcomes to include cerebrovascular conditions such as dementia. We appreciate their comments and agree that the scope of UPF-related health concerns should be broadened. Indeed, UPF consumption has been strongly associated with increased risk of adverse cardiometabolic outcomes.[2] Accordingly, the health effects of UPFs have often been characterized by end-organ damage, particularly in the liver, heart, and kidneys.[3] Given that poor cardiometabolic health is a well-established risk factor for dementia,[4] recent studies have elucidated the association between UPF consumption and neurological sequelae.[3,5,6] Therefore, the brain is increasingly being recognized as a critical target of UPF-related end-organ damage.[3] Research exploring the relationship between diet and cognitive function, including neurodegenerative disease, has been conducted for over three decades, largely focusing on specific nutrients, food groups, and overall dietary patterns.[3,5,6] However, this body of evidence remains limited compared to the extensive research on obesity and cardiovascular outcomes. Epidemiological evidence specifically addressing the effects of food processing on cognitive decline or dementia risk only began to emerge around 2020.[3,5,6] As further evidence accumulates, a more comprehensive understanding of the relationship between UPF consumption and brain health will likely facilitate a more robust scientific discussion in this field.

The author also addressed how UPF-induced abdominal adiposity triggers systemic inflammation that affects the cerebral microvasculature. Furthermore, they described how the combined effects of harmful UPF components and a deficit in neuroprotective nutri-

ents may compromise the blood-brain barrier, leading to neurovascular disruption. These neurovascular alterations may accelerate cognitive decline and reinforce detrimental eating behaviors. The author further urged the adoption of robust, non-invasive neuroimaging techniques to better understand how UPF consumption may contribute to neurological sequelae, including functional and structural alterations in the brain. Specifically, they suggested that techniques such as arterial spin labeling (to quantify cerebral blood flow) and diffusion tensor magnetic resonance imaging (to assess structural brain changes) be incorporated as standard practice in longitudinal studies.

We concur that the assessment of brain function and structure can provide valuable insight into the mechanistic link between UPF consumption and brain health. Only recently did researchers begin to apply neuroimaging approaches to clarify the mechanistic pathways linking UPF consumption to adverse health outcomes. A randomized crossover trial evaluated the impact of a single ultra-processed meal on myocardial blood flow (MBF) and cognitive performance in healthy adults using positron emission tomography/computed tomography alongside neuropsychological testing.[7] That study demonstrated how consumption of a single ultra-processed meal increased MBF and myocardial flow reserve under intermediate-dose adenosine stress and was associated with reduced cognitive performance, suggesting the possibility of acute neurocognitive effects. In a population-based cohort study using data from the UK Biobank, investigators examined the long-term associations of sugary beverage consumption and its interaction with genetic risk in relation to incident dementia and brain structural markers derived from magnetic resonance imaging.[8] Higher consumption of sugar- and artificially sweetened beverages was associated with higher risk of dementia, whereas moderate natural juice consumption was associated with lower risk, greater grey matter volume, and lower white matter hyperintensity burden. Another study using data from the same cohort investigated the association between UPF consumption and neurodegeneration, including structural brain alterations.[9] Higher UPF consumption was associated with increased risk of dementia, Parkinson's disease, and multiple sclerosis. Moreover, higher UPF consumption was associated with reductions in subcortical grey matter volume and as well as in the volume, thickness, and surface area at the cortical level, suggesting a possible mechanistic pathway linking higher UPF consumption to neurodegenerative risk. In light of these findings, integrating the assessment of brain function and structure may improve our understanding of the neural consequences associated with UPF consumption. However, focusing solely on imaging biomarkers may not fully capture the broader clinical implications of UPF-related health effects. Rather than establishing universal standards, we believe that their application should be considered within the context of specific study aims and practical feasibility. Moreover, a uniform mandate may hinder diversity in study design, objectives, and available resources across studies. To establish clinical and public health relevance, research should address the entire continuum of outcomes, from subclinical functional and structural alterations to clinically meaningful endpoints, including cognitive impairment and incident dementia.

Collectively, we concur that the scope of UPF-related health concerns should extend beyond obesity and cardiovascular disease to encompass other outcomes including dementia. Moreover, assessing brain function and structure may help elucidate the mechanistic links between UPF consumption and brain health. However, future research should address the full continuum of outcomes, from subclinical, functional, and structural alterations to clinically meaningful endpoints.

CONFLICTS OF INTEREST

The author declares no conflict of interest.

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