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Letter to the Editor

Response to the Letter to the Editor concerning "Associations of Individual Beverage Types and Substitution with Dementia Risk"

Dear Editor,

We sincerely thank Professor Wang for the thoughtful and constructive comments on our study [1] and for the opportunity to clarify several important methodological aspects. We fully agree that dietary exposure assessment based on a single 24-h recall may be subject to within-person variability and may not optimally reflect habitual intake, particularly for beverages that are not consumed daily. To address this concern, when participants had multiple Oxford WebQ dietary assessments available, we calculated the average intake across all measurements and used this mean value in the primary analyses. This approach was intended to better capture long-term habitual beverage consumption and to reduce random measurement error, thereby improving the stability and reliability of the exposure assessment.

We also acknowledge the important concern regarding potential reverse causation, given the long prodromal period of dementia and the possibility that early subclinical disease processes may influence dietary behaviors. To minimize this possibility, we conducted landmark analyses excluding participants who developed dementia within the first 1 year and within the first 2 years of follow-up. These analyses were performed using the Fine–Gray subdistribution hazard model with death treated as a competing event. The results of these landmark analyses were highly consistent with those of the main analyses. For example, in the 1-year landmark analysis, compared with non-consumers, individuals consuming more than one glass per day of sugar-sweetened beverages had a significantly higher risk of dementia (Model 4: subdistribution hazard ratio [SHR] 1.60, 95% CI 1.27–2.01, $P < 0.001$), and an identical estimate was observed in the 2-year landmark analysis (Model 4: SHR 1.60, 95% CI 1.27–2.01, $P < 0.001$). In contrast, coffee and tea consumption showed robust inverse associations across all landmark models. For example, in the 1-year landmark analysis, moderate coffee consumption (>0-1 glass/day) was associated with a 37% lower dementia risk (Model 4: SHR 0.63, 95% CI 0.52–0.76, $P < 0.001$), and a nearly identical result was observed in the 2-year landmark analysis (Model 4: SHR 0.64, 95% CI 0.53–0.77, $P < 0.001$). Tea consumption showed similarly consistent protective associations, with SHR values around 0.64–0.75 across models and landmark periods. These findings indicate that excluding early dementia cases did not materially change the observed associations, arguing against substantial reverse causation.

In addition, we agree that death is an important competing event in dementia research, especially in older populations with long follow-up. Therefore, all landmark analyses and primary analyses were conducted using the Fine–Gray subdistribution hazard model to explicitly account for competing risk due to mortality. The competing-risk results were highly consistent with those obtained from the main models. For instance, in the competing-risk analysis, compared with non-consumers, participants consuming more than one glass per day of sugar-sweetened beverages had a significantly higher dementia risk (Model 4: SHR 1.66, 95% CI 1.32–2.08, $P < 0.001$). In contrast, coffee and tea consumption remained strongly protective even after accounting for competing mortality (coffee, >0-1 glass/day in Model 4: SHR 0.63, 95% CI 0.52–0.76, $P < 0.001$; tea, >0-1 glass/day in Model 4: SHR 0.66, 95% CI 0.53–0.83, $P < 0.001$). These results demonstrate that competing mortality did not materially bias our findings and further support their robustness.

Taken together, by averaging repeated dietary assessments, performing landmark analyses at both 1 and 2 years, and applying Fine–-Gray competing-risk models, we have undertaken multiple complementary and rigorous analytical strategies to address exposure misclassification, potential reverse causation, and competing risks. The high consistency of results across all these analyses substantially strengthens the robustness, interpretability, and clinical and public health relevance of our findings regarding beverage consumption, substitution patterns, and dementia risk.

Once again, we are grateful to Professor Wang for the thoughtful and constructive comments, which have helped us to further clarify and strengthen the methodological rigor of our study. We also sincerely thank the editorial team for their careful handling of our manuscript and for providing us with the opportunity to address these important issues. Moving forward, we will continue to deepen our research on the associations between beverage consumption patterns, dietary quality, and cognitive health, with particular emphasis on frailty and dementia-related outcomes. We hope that our ongoing and future work will contribute more precise and comprehensive evidence to support the development of effective and personalized dietary interventions for aging populations.

CRediT authorship contribution statement

JHK, SJH, YJK and JWL contributed to the conception and design; acquisition, analysis, and interpretation of data; and drafting of the manuscript. All authors have read and approved the final version of the manuscript.

Consent for publication

All authors gave their consent for publication.

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[1] Kim JH, Kwon YJ, Lee Y, Han T, Lim MY, Heo SJ, et al. Associations of individual beverage types and substitution with dementia risk: a UK biobank cohort study. J Nutr Health Aging 2026;30:100740. https://doi.org/10.1016/j.jnha.2025.100740.

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