A Report on "Coffee and Tea Intake, Dementia Risk, and Cognitive Function" by Zhang et al. (2026)

Reviewer 2

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> I am wiser than this person; for it is likely that neither of us knows anything fine and good, but he thinks he knows something when he does not know it, whereas I, just as I do not know, do not think I know, either. I seem, then, to be wiser than him in this small way, at least: that what I do not know, I do not think I know, either.
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> Plato, *The Apology of Socrates*, 21d

> To err is human. All human knowledge is fallible and therefore uncertain. It follows that we must distinguish sharply between truth and certainty. That to err is human means not only that we must constantly struggle against error, but also that, even when we have taken the greatest care, we cannot be completely certain that we have not made a mistake.
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> Karl Popper, 'Knowledge and the Shaping of Reality'

Overview

Citation: Zhang, Y., Liu, Y., Li, Y., Li, Y., Gu, X., Kang, J. H., Eliassen, A. H., Wang, M., Rimm, E. B., Willett, W. C., Hu, F. B., Stampfer, M. J., and Wang, D. D. (2026). Coffee and Tea Intake, Dementia Risk, and Cognitive Function. *JAMA*.

Abstract Summary: This prospective cohort study investigated the long-term associations of caffeinated and decaffeinated coffee and tea intake with dementia risk and cognitive function in over 131,000 participants. The findings indicate that greater consumption of caffeinated coffee and tea is associated with a lower risk of dementia and modestly better cognitive function, with the most pronounced association observed at moderate intake levels.

Key Methodology: Prospective cohort study using data from the Nurses' Health Study (NHS) and Health Professionals Follow-up Study (HPFS) with up to 43 years of follow-up, repeated dietary measurements via food frequency questionnaires, and assessment of dementia (primary outcome) and subjective/objective cognitive function (secondary outcomes) using Cox proportional hazard models and generalized estimating equations (GEE).

Research Question: Is long-term intake of caffeinated and decaffeinated coffee associated with risk of dementia and cognitive outcomes?

Summary

Is It Credible?

This study by Zhang et al. presents a substantial analysis of two major prospective cohorts, the Nurses' Health Study (NHS) and the Health Professionals Follow-up Study (HPFS), involving 131,821 participants followed for up to 43 years. The authors report that higher consumption of caffeinated coffee and tea is associated with a lower risk of incident dementia and a lower prevalence of subjective cognitive decline. Specifically, they highlight a nonlinear dose-response relationship where moderate consumption—approximately 2 to 3 cups of caffeinated coffee or 1 to 2 cups of tea per day—is associated with the lowest risk (p. E1). The study also posits that this protection is specific to caffeinated beverages, as decaffeinated coffee showed no such benefit.

The credibility of these findings rests heavily on the validity of the outcome ascertainment, which presents a significant limitation. Incident dementia was identified through a review process where a physician blinded to exposure data confirmed the diagnosis using death certificates, medical records, and autopsy reports (p. E2). While this is more rigorous than relying solely on raw codes, the authors note that medical record confirmation was only used "when available" (p. E13). Dementia is notoriously underreported on death certificates, often appearing only in late stages or being omitted entirely in favor of cardiovascular or other causes of death. The authors attempt to bolster the validity of their outcome by demonstrating an association with plasma p-tau217 in a nested subset (p. E28). However, this validation analysis included only 103 participants, of whom just 27 developed incident dementia. This sample size is arguably too small to definitively validate the outcome measurement for the entire cohort of over 11,000 cases. Consequently, the reported hazard ratios may be influenced by detection bias if coffee drinkers differ from non-drinkers in

how their cause of death is recorded or diagnosed.

Furthermore, the study's central claim that caffeine is the primary neuroprotective agent warrants scrutiny due to inconsistencies in the data regarding different beverage types. The authors argue for caffeine specificity based on the lack of association with decaffeinated coffee (p. E2). However, tea consumption showed a protective effect comparable to caffeinated coffee (HR 0.86 for tea vs. 0.82 for coffee in fully adjusted models, p. E7). Given that tea typically contains significantly less caffeine per serving than coffee, the similarity in effect sizes suggests that other bioactive compounds, such as polyphenols or flavonoids, might play a substantial role. If caffeine were the sole driver, one might expect a clearer dose-response gradient correlating strictly with total caffeine intake across beverage types, rather than similar benefits from beverages with distinct caffeine profiles.

The analysis of decaffeinated coffee also introduces the challenge of reverse causation. The study found that higher decaffeinated coffee intake was associated with a higher prevalence of subjective cognitive decline (p. E4). The authors reasonably suggest this may be due to confounding by indication—individuals switching to decaf due to health concerns that also predispose them to cognitive issues (p. E12). While the authors performed sensitivity analyses excluding recent dietary changes and using lagged analyses to mitigate this, the "sick quitter" phenomenon remains a plausible complication (pp. E40, E42). If the decaf group is enriched with individuals at higher baseline risk despite these adjustments, the lack of a protective association might be an artifact of this selection bias rather than proof of caffeine's unique efficacy.

Finally, regarding objective cognitive function, the reported benefits are statistically significant but modest in magnitude. In the NHS cohort, the difference in TICS scores between the highest and lowest quartiles of caffeinated coffee intake was 0.11 units on a 41-point scale (p. E4). The authors acknowledge this represents a small difference, roughly equivalent to less than a year of age-related decline (p. E10).

While potentially meaningful at a population level, these effect sizes should be interpreted with caution regarding their clinical relevance for individuals. Additionally, the consistency of these cognitive findings is mixed; for example, while tea was associated with better scores across global cognition and verbal memory in fully adjusted models, caffeinated coffee's association with global cognition was borderline non-significant in the fully adjusted Model 2 ($p = 0.06$), though significant in the minimally adjusted Model 1 (p. E11).

The Bottom Line

The study provides credible observational evidence that moderate long-term consumption of caffeinated coffee and tea is associated with a reduced risk of dementia and subjective cognitive decline. However, the reliance on death records, self-reports, and only sometimes-available medical record confirmation for dementia ascertainment introduces potential misclassification bias. Additionally, the claim that caffeine is the sole protective agent is challenged by the strong protective effects observed for tea, which has lower caffeine content. While the findings support a healthy dietary pattern including these beverages, the specific biological mechanism remains difficult to isolate from the data, and the absolute magnitude of cognitive benefit appears small.

Potential Issues

Validity of the primary dementia outcome is uncertain due to ascertainment methods: The study's primary outcome, incident dementia, was identified through death records, self-reported physician diagnoses, and medical record confirmation "when available" (p. E13). This methodology is susceptible to misclassification, as dementia is often underreported on death certificates and self-reports can be inaccurate. While the authors note that the participants are health professionals, which may increase reporting accuracy, and cite high validity for death ascertainment in the cohorts generally, the lack of systematic cognitive screening for the entire participant pool suggests that dementia cases were likely identified at later stages (p. E2). The authors attempt to validate the outcome by showing an association with plasma p-tau217, a known biomarker, but this analysis was conducted on a small nested subset of 103 participants, of whom only 27 developed dementia. Although this validation analysis found a strong association (HR 2.78), its small size limits the generalizability of this finding (pp. E27–E28). The authors acknowledge in the limitations section that "misclassification of dementia status remains possible" (p. E13).

The analysis may underestimate the total effect of coffee due to potential overadjustment: The study's primary statistical model (Model 2) adjusts for several clinical comorbidities, including histories of diabetes, hypertension, and hypercholesterolemia (pp. E7, E39). While these factors can be confounders, they may also lie on the causal pathway between coffee consumption and dementia risk. For instance, the authors' own discussion posits that caffeine may exert a protective effect by improving insulin sensitivity and reducing the risk of type 2 diabetes (p. E12). By statistically controlling for these potential mediators, the analysis risks underestimating the total protective effect of coffee. The authors mitigate this issue by presenting a minimally adjusted model (Model 1) alongside the fully adjusted model. The results are very similar across both models (e.g., pooled HR for highest vs. low-

est caffeinated coffee intake was 0.81 in Model 1 and 0.82 in Model 2), suggesting that the mediation effect through these specific pathways is minimal in this analysis (p. E7). However, the primary interpretation focuses on the fully adjusted model, which likely estimates an effect of coffee that is independent of its influence on these major vascular and metabolic pathways.

The pattern of results across different outcomes and beverages is inconsistent, challenging a simple caffeine-centric conclusion: The study's narrative suggests a protective effect driven primarily by caffeine, but the detailed results show a more complex picture. For objective cognitive measures in the Nurses' Health Study cohort, higher tea intake was associated with statistically significant benefits across all three reported domains (TICS score, verbal memory, and global cognition) in the fully adjusted model (p. E11). In contrast, higher caffeinated coffee intake was significantly associated with a better TICS score, but its association with the global cognition score was borderline non-significant ($p = 0.06$) and its association with verbal memory was not significant in the same fully adjusted model (p. E11). This broad association for tea, which typically contains less caffeine than coffee, suggests that non-caffeine components may play a significant role. Furthermore, the findings for decaffeinated coffee are contradictory: while it showed no association with dementia risk, it was associated with a significantly higher prevalence of subjective cognitive decline and modestly worse objective verbal memory scores (pp. E4, E6). The authors propose "confounding by indication" to explain the negative findings for decaffeinated coffee, but this complex pattern of results is not fully reconciled and weakens the parsimony of the conclusion that caffeine is the primary protective agent (p. E12).

Measurement limitations for key exposures may affect the interpretability of some findings: The study contains measurement limitations for some of its key exposures, which the authors acknowledge. First, the analysis of tea is complicated because the food frequency questionnaires did not distinguish between specific

types, such as green versus black tea, or caffeinated versus decaffeinated varieties (p. E12). While the authors used a standard caffeine content for tea to calculate total caffeine intake, the inability to isolate different tea types makes it difficult to attribute the observed protective effect of tea solely to caffeine versus other potentially neuroprotective compounds, such as flavonoids or L-theanine (p. E2). Second, the questionnaire used to assess subjective cognitive decline was not administered consistently across all waves in the Health Professionals Follow-up Study, with specific items being omitted in certain years (p. E30). This variation in the measurement instrument over time introduces potential measurement error into this secondary outcome.

Future Research

Improved outcome ascertainment: Future work should prioritize cohorts that utilize active surveillance and standardized clinical adjudication for dementia, rather than relying heavily on death records or self-reports. While the NHS cohort included telephone-based cognitive testing for a subset of older participants, implementing systematic cognitive screening at regular intervals for *all* participants in future cohorts would reduce detection bias and provide a more accurate estimate of incidence, particularly for milder or earlier-stage cases that may not appear on death certificates.

Granular exposure assessment: To better disentangle the effects of caffeine from other bioactive compounds, future studies should collect detailed data on specific tea types (e.g., green vs. black) and preparation methods. Analyzing these subtypes separately could help determine if the protective effects align with caffeine content or if they track more closely with specific polyphenol profiles, thereby clarifying the underlying mechanism.

Investigation of reverse causation in decaffeinated intake: Research designs should explicitly model the timing and reasons for changes in beverage consumption. Analyzing a sub-cohort of "never-caffeine" drinkers who consume decaffeinated coffee versus those who switched from caffeinated to decaffeinated beverages could help isolate the "sick quitter" effect from a true lack of biological efficacy, providing a fairer test of the caffeine hypothesis.

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