COMMENTARY

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Coffee and tea consumption in relation to chronic disease: An epidemiological perspective

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Coffee and tea are deeply embedded in daily life, yet medical advice has long been cautious or even contradictory. Many clinicians have traditionally regarded coffee and tea as non-essential dietary indulgences and potential cardiovascular hazards, largely on account of their caffeine content. Over the last decade, however, a substantial body of epidemiological research, including large prospective cohorts, meta-analyses and newer genetic and biomarker-based studies has shifted this perspective. Taken together, these data suggest that, for most adults, moderate consumption of coffee and tea is associated with lower risks of several major chronic diseases and with reduced all-cause mortality, particularly when these beverages displace sugar-sweetened drinks. We synthesize recent epidemiological and mechanistic evidence on coffee and tea consumption in relation to chronic disease. A schematic summary of beverage types, bioactive components, intermediate pathways, dose–response relationships, and chronic disease outcomes is captured in Figure 1, and this commentary focuses on what the evolving evidence means for clinical and public-health practice.

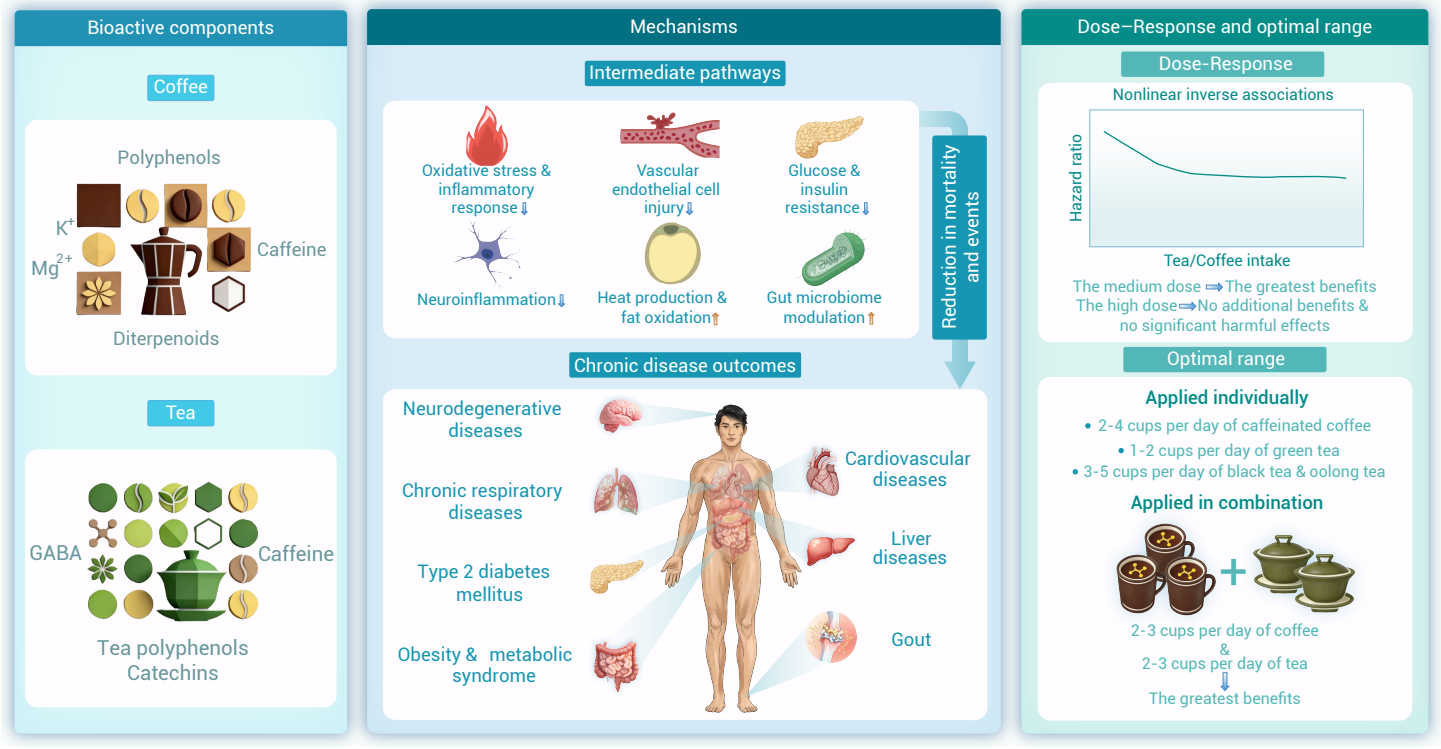

Figure 1. Proposed epidemiological framework linking coffee and tea consumption with intermediate pathways, dose–response patterns and major chronic disease outcomes

FROM SAFETY TO SUBSTITUTION

Traditional evaluations of coffee and tea consumption have centered on potential adverse cardiovascular outcomes, such as hypertension and arrhythmogenesis. Yet, current epidemiological evidence indicates that evaluating these beverages solely in terms of direct cardiotoxicity is incomplete. A more clinically relevant inquiry involves assessing their displacement of alternative fluids within the broader context of dietary habits.

In many high- and middle-income settings, a substantial proportion of daily fluid intake consists of sugar-sweetened beverages (SSBs) such as soft drinks, sweetened juices, ready-to-drink coffees and energy drinks, which are strongly associated with weight gain, type 2 diabetes (T2D), cardiovascular diseases (CVD) and premature mortality.[1] When analyses are framed in terms of substitution rather than absolute intake, a clear pattern emerges. Importantly, recent high-quality prospective studies have utilized robust methodological approaches, such as isocaloric substitution models and have rigorously adjusted for overall diet quality scores, physical activity, and socioeconomic status to isolate the specific effect of beverage replacement from the broader "healthy user bias". In these models, exchanging one daily serving of SSBs for unsweetened coffee, tea or plain water is linked to meaningful reductions in incident diabetes, CVD events and all-cause mortality, particularly among people already living with T2D. In these cohorts, long-term outcomes are consistently better in those who choose coffee, tea and water instead of sugary drinks.[1]

This way of looking at the data has practical consequences. It shifts the clinical discussion from generalized caution about caffeine to specific, achievable changes in what patients drink. Asking someone to swap an afternoon cola for plain tea, or to choose filtered coffee with a little milk instead of a large sweetened latte builds on existing preferences rather than demanding entirely new behaviors. From an epidemiological standpoint, these kinds of substitu-

tions have a much larger impact on long-term risk than fine-tuning the nutrient content of an otherwise sugar-laden beverage pattern.

DOSE–RESPONSE PATTERNS AND MAJOR HEALTH OUTCOMES

Large cohort studies show a clear non-linear association between coffee intake and major outcomes: as intake rises from none to about two to four cups per day (typically standardized in meta-analyses as ~200 mL or 5–8 oz per cup, yielding ~75–100 mg of caffeine), risks of all-cause and cardiovascular mortality fall by roughly 15–20%; beyond this level, the curve plateaus, with little extra benefit and a potential for adverse effects at very high doses.[1-3] It is important to acknowledge that excessive caffeine intake can precipitate sleep architecture disruption, anxiety, and tachycardia, particularly in susceptible subgroups such as those with underlying anxiety disorders or genetic predispositions. Tea follows a similar pattern, with about two to four cups of green tea or three to five cups of black/oolong tea daily linked to a lower incidence of CVD, stroke and T2D, and to slower biomarker-derived biological ageing.[1,3] New data extend this non-linear pattern to brain outcomes: in two large US health-professional cohorts with more than 40 years of follow-up, higher intakes of caffeinated coffee and tea were associated with lower dementia incidence and less subjective cognitive decline, with the strongest differences observed at roughly 2–3 cups of coffee or 1–2 cups of tea per day; decaffeinated coffee was not protective, but the same moderate intakes were also related to slightly better objective cognitive performance. Similar intake ranges are associated with slower overall cognitive ageing for tea and with reduced risks of Parkinson's disease and chronic liver disease for coffee.[3] Another study reported that adolescent intake of caffeinated coffee was significantly associated with a 14% lower T2D risk in adulthood among women who consumed ≥ 1 serving/day compared with non-consumers.[4] These findings align with broader evidence on flavonoids and other polyphenols, indicating that individuals who obtain these compounds from diverse sources (e.g., tea, coffee, fruits, and other plant foods) have lower risks of major chronic diseases than those relying on one or two sources. Overall, the epidemiologic signal is consistent: for most non-pregnant adults, moderate coffee and tea intake appears to fall within a "safe and beneficial" range for cardiovascular, metabolic, and cognitive health. Pregnant individuals are excluded from this guidance because gestation reduces maternal caffeine clearance and higher caffeine intake has been linked to low birth weight and pregnancy loss. Moreover, evidence remains limited for patients with established chronic diseases who are receiving pharmacologic treatment or other medical therapies, and potential interactions between coffee or tea and commonly used medications are not well characterized. The proposed "safe and beneficial" ranges derive from standardized epidemiologic thresholds, but clinical recommendations should be individualized according to caffeine sensitivity, the caloric load of additives, and the presence of pre-existing diseases and ongoing treatments.

A BRIEF MECHANISTIC PERSPECTIVE

Both coffee and tea deliver complex mixtures of bioactive compounds. Coffee contains caffeine, chlorogenic acids, diterpenoids and diverse polyphenols. Tea offers caffeine plus catechins, theaflavins, other flavonoids, γ-aminobutyric acid, and tea polysaccharides.[5] Experimental and human studies suggest that these constituents act on several intermediate pathways relevant to chronic disease: they may improve endothelial function, reduce oxidative stress and low-grade inflammation, enhance insulin sensitivity and glucose handling, modestly increase thermogenesis and fat oxidation, and modulate the gut microbiome (e.g., promoting short-chain fatty acid-producing bacteria) and dampen neuroinflammatory processes.[2,5] Moreover, brewing method affects bioavailability. Unfiltered coffee (e.g., French press, boiled) retains lipid-raising diterpenes (cafestol, kahweol) that paper filters remove, partly explaining cohort differences in cardiovascular risk.[2] These mechanisms also support a non-linear dose–response: at higher intakes, sympathetic activation and cortisol rises from excess caffeine may offset polyphenols' benefits, so gains plateau beyond about two to four cups daily.[2]

Genetic and epigenetic work further supports biological plausibility, and provides critical insights into interindividual variability. Mendelian-randomization analyses using variants that proxy for higher plasma caffeine concentrations link life-long higher caffeine exposure to lower adiposity and reduced risk of T2D, consistent with a causal thermogenic or metabolic effect. However, Individual responses are strongly shaped by pharmacogenetics, particularly *CYP1A2* variants that determine "fast" versus "slow" caffeine metabolism. Among slow metabolizers, prolonged caffeine clearance means that even moderate intake may elicit adverse cardiovascular effects (e.g., elevated blood pressure and palpitations), underscoring the limitations of universal safety thresholds and highlighting the importance of genetics-informed, personalized guidance on beverage consumption.

LIMITATIONS AND FUTURE DIRECTIONS

Despite the encouraging consistency of current epidemiological evidence, several limitations warrant caution and define priorities for future work. Most of the data come from high-income countries and from populations of European or East Asian ancestry; patterns of coffee and tea preparation, co-consumed foods, and underlying risk profiles may differ substantially in other regions, thereby limiting generalizability. Exposure assessment relies almost entirely on self-reported intake, typically captured at long intervals and with limited detail on cup size, brewing strength, additives, and changes over time. Residual confounding remains a concern. Even after careful adjustment, moderate green tea and filtered coffee drinkers often have healthier diets, more physical activity, and higher socioeconomic status that may not be fully captured in analyses. In addition, the evidence base may be subject to publication bias, whereby null or negative findings are less likely to be reported, thereby complicating the interpretation of heterogeneous results across cohorts. Reverse causation is also a concern, as individuals with early or subclinical disease may reduce caffeine intake following medical advice, potentially inflating the apparent benefits observed among remaining consumers. Although genetic and epigenetic data contribute to strengthening causal inference, current evidence predominantly focuses on caffeine, leaving the roles of other bioactive compounds and broader beverage consumption patterns less well characterized.

Future research should move in several directions. Randomized or quasi-experimental beverage-substitution trials, particularly in high-risk groups such as individuals with pre-diabetes, established T2D or CVD, are needed to quantify the impact of replacing sugar-sweetened beverages with coffee or tea on clinical outcomes. Future prospective cohorts should incorporate more granular and repeated assessments of beverage intake, capturing preparation methods, additives, and timing, while incorporating objective biomarkers of exposure. Mechanistic studies that integrate metabolomics, microbiome profiling, and epigenetic analyses are needed to elucidate the specific components and biological pathways most relevant to cardiometabolic and neurodegenerative outcomes, as well as to identify subgroups that may respond differentially according to genetic predisposition or baseline risk. Finally, expanding the inclusion of diverse populations across ethnic, socioeconomic, and cultural contexts will be essential to ensure that public health recommendations on coffee and tea are both evidence-based and broadly generalizable.

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AUTHOR CONTRIBUTIONS

XW: conceptualization, methodology, supervision and writing–original draft preparation. CL: visualization, and writing–reviewing and editing. CL, YZ, YY, KS: writing–reviewing and editing. All authors contributed to the article and approved the submitted version.

DECLARATION OF INTERESTS

The authors declare no competing interests.