

# Unraveling tea and coffee consumption effects on cardiovascular diseases risk factors: A narrative review

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## Abstract

**Backgrounds and Aims:** Daily lifestyle plays a vital role in modifying the risk for cardiovascular diseases (CVDs). Our daily life isn't inseparable from nutrition intake. As such, tea and coffee are often regarded as the most consumed beverages worldwide. There have been a lot of debates on the adverse effects and benefits of consuming these popular beverages. This comprehensive review explores the different types of tea and coffee and their mechanism of action. It delves deeper into their roles in reducing CVD risk, aiding CVD recovery, lowering CVD mortality, and their varying effects across populations and regions.

**Methods:** An extensive literature search was conducted on PubMed. Relevant articles were identified through cross-referencing and manual searches. Excluded from the study were commentaries, case reports, clinical vignettes, and non-English articles.

**Results:** Tea and coffee contain varying levels of caffeine and other bioactive compounds with cardioprotective effects against oxidative stress, inflammation, and more. Genetic factors further modulate their effects. Tea flavonoids benefit cholesterol, blood pressure, and endothelial function, while coffee constituents impact oxidative stress, metabolism, insulin sensitivity, and gut flora. Moderate consumption of both beverages may offer cardiovascular benefits, but outcomes vary depending on populations and conditions. Tea and coffee consumption may influence CVD recovery by reducing mortality and improving survival, however, it must be noted that it has the potential to be harmful to some individuals.

**Conclusion:** Evidence suggests that moderate consumption of these beverages may be linked to reduced cardiovascular mortality, although individual characteristics and pre-existing conditions can influence outcomes. Excessive caffeine consumption, found in both beverages, may pose risks such as arrhythmias, hypertension, and cardiovascular mortality in CVD patients, with a dose-dependent nature. Future research should delve into mechanisms, genetic factors, and diverse cultural impacts of its use. Health care providers should consider individual characteristics when advising on tea and coffee consumption in the context of cardiovascular health.

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## KEYWORDS

caffeine, cardiovascular disease, coffee, effects, influence, tea

## 1 | INTRODUCTION

Cardiovascular diseases (CVDs) take approximately 17.9 million lives annually. Globally, they are the leading causes of death, contributing to 32% of all global deaths. At least 75% of CVD deaths worldwide occur in low- and middle-income countries as they don't have many programs in disease early detection for people with related risk factors. Therefore, many are diagnosed in the late stage of the disease.<sup>1</sup>

The events of CVD are avoidable. Daily lifestyle plays a vital role in modifying the risk for CVDs. Our daily life is not inseparable from nutrition intake. As such, tea and coffee are the most consumed beverages worldwide. They have been part of traditions and involved in human social life for hundreds of years, such as to increase work productivity and wakefulness due to their caffeine content.

Previous studies mentioned that there have been several debates over the adverse effects and benefits of consuming these popular drinks daily in the context of CVD progression.<sup>2,3</sup> Furthermore, the study of their consumption has accumulated over the past few years. Hence, this review aims to provide comprehensive evidence focusing on the types and pharmacology of tea and coffee, their effects on reducing the risk of CVDs, their consumption effects on helping the recovery of CVD, their effects on reducing the mortality of CVDs, how their consumption differ in making effects on different populations in different geographical areas, and drawbacks of tea and coffee consumption on cardiovascular health.

## 2 | METHODS

A literature search was carried out on the "PubMed" database using the keywords: tea, coffee, cardiovascular system, heart failure, coronary artery disease (CAD), hypertension, and myocardial infarction. Additionally, cross-referencing and manual searches of articles were conducted following the initial search. Commentaries, case reports, clinical vignettes, and non-English articles were excluded from the study.

Data collection involved four authors reviewing the titles and abstracts of all retrieved records to identify articles that met the inclusion criteria. The results were thoroughly discussed to reach a final decision.

### 2.1 | Pharmacology of tea and coffee and the mechanistic action of their products on the human body, especially the cardiovascular system

In addition to having caffeine, coffee includes a lot of antioxidants and other bioactive substances. According to research, tea with

caffeine, catechin polyphenols, and flavonoids has cardioprotective effects against oxidative stress, inflammation, amyloid-beta aggregation, and apoptosis. In addition to having similar ingredients like caffeine, coffee, and tea also differ in their biologically active ingredients, such as epigallocatechin gallate (EGCG) and chlorogenic acid (CGA), which seem to have similar mechanisms. Additionally, genetic polymorphisms in enzymes involved in the absorption, metabolism, and excretion of tea and coffee components were linked to the two beverages' differing biological functions.<sup>4</sup>

Caffeine (1,3,7-trimethylxanthine or 3,7-dihydro-1,3,7-trimethyl-1H-purine-2,6-dione) chemical formula is  $C_8H_{10}N_4O_2$ . The human small intestine rapidly absorbs caffeine after its oral administration within 45 min and reaches peak value at 30 min; subject to food intake and pH. Caffeine's metabolic half-life is three to 5 h and readily crosses the blood-brain barrier. Eighty percent of consumed caffeine goes through metabolism into paraxanthine, theobromine, and theophylline, which occurs via isoenzyme CYP1A2 in the liver.<sup>5</sup>

Phenolics in tea are often associated with preventive effects against CVDs.<sup>6</sup> Unsweet tea also provides consumers with bioactive compounds, namely flavonoids, which reduce low-density lipoprotein (LDL) cholesterol, systolic, and diastolic blood pressure among healthy populations and those at risk. Flavonoids are phytochemicals and ligands that bind nuclear receptor 4A (NR4A) which plays a role in inflammatory-related diseases such as cancer, fibrosis, and injury when overexpressed.<sup>7</sup> NR4A receptors are demonstrated to be highly expressed in macrophages and smooth muscle cells of human atherosclerotic lesions and during the response to injury of the arterial wall.<sup>8</sup> Flavonoids are also thought to improve blood flow by increasing endothelial nitric oxide bioavailability.<sup>9</sup>

Studies in animals and clinical studies of cardiovascular patients showed that resting heart rate is an emerging indicator and predictor of cardiovascular health and mortality, in which reduced heart rate reduction is associated with prolonged maximum lifespan.<sup>10</sup> A meta-analysis by Han et al.<sup>11</sup> showed that 3–6 cups of daily coffee consumption per day had no effect on resting heart rate after 2–24 weeks, even after subgroup level analysis. The net changes in resting heart rate between the coffee intervention and control groups varied from  $-5.04$  to  $3.60$  bpm. The overall outcome of the random-effects model revealed that the coffee intervention increased resting heart rate by  $0.40$  bpm compared to the control group, but not statistically significant (95% confidence interval [CI]:  $-0.78$  to  $1.57$ ;  $p = 0.506$ ), with no substantial heterogeneity among the studies ( $I^2 = 0.0\%$ ,  $p = 0.756$ ). The leave-one-out sensitivity analysis indicated a narrow range of  $0.12$  bpm (95% CI:  $-1.24$  to  $1.48$ ;  $p = 0.864$ ) to  $0.59$  bpm (95% CI:  $-0.62$  to  $1.80$ ;  $p = 0.342$ ), indicating that the pooled impact size of coffee consumption on resting heart rate was not dependent on a single study.

## 2.2 | Effects and pathophysiology of tea and coffee consumption on reducing the risk of CVDs

### • Hypertension

Hypertension is a major risk factor for CVD, especially in the adult population,<sup>12</sup> but its prevalence can be significantly decreased by some basic dietary modifications.<sup>13</sup> A recent study has shown that consuming three to four cups a day of coffee lowers the risk of hypertension among adults, with an odds ratio (OR) of 0.41 for men and 0.59 for women.<sup>14</sup> This could be related to the presence of CGA, and its effect on vascular activity.<sup>15</sup> CGA is found in green tea. It improves vascular reactivity by increasing the reactive hyperemia ratio and decreasing serum homocysteine.<sup>16</sup> CGA reduced NAD(P)H-dependent superoxide production and inhibited the expression of the p22<sup>phox</sup> gene, which in turn reduced free radical production and directly scavenged free radicals. Furthermore, CGA metabolites, ferulic acid work as a nonselective NAD(P)H oxidase antagonist to affect the BP reduction greatly. By endothelial-dependent pathway, it also stimulates nitric oxide production. CGAs also inhibit angiotensin-converting enzyme activity. All these mechanisms, combined with the CGA anti-inflammatory effect, play a role in the integrity of overall vascular function and well-regulated BP.<sup>17</sup> However, it is barely present in roasted coffee.<sup>18</sup>

In contrast, the presence of caffeine does not seem to have any impact on vascular activity or blood pressure in habitual coffee drinkers.<sup>19</sup> A prospective study in Singaporean Chinese people showed that there is a U-shaped relationship between the consumption of coffee and the risk of hypertension. Individuals who drink less than one cup of coffee weekly, or more than two cups of coffee weekly, seem to have a reduced risk of developing hypertension with a hazard ratio (HR) of 0.87.<sup>20</sup> Antihypertensive effects of tea are related to flavonoids, which modulate endothelial function, inducing vasodilation. They also have mild anti-inflammatory and antioxidant effects.<sup>21</sup> Another study on Singaporean Chinese people, claims that the parallel consumption of green tea and British tea significantly lowers the risk of hypertension with an OR of 0.63 for people aged 40 years old or more.<sup>2</sup>

### • CAD

CAD is a major public health problem, and a leading cause of mortality around the world, especially in high-income countries.<sup>23</sup> Studies show that high flavonoid intake in coffee and tea can reduce the risk of CAD by 50% compared to low-intake individuals, due to their antioxidant, and anti-inflammatory effects. In vitro, flavonoids inhibit the oxidation of LDLs, stimulate nitrous oxide secretion, and have minor antithrombotic effects. These findings are not well established in the human body, and their effect on atheromatous plaques is not identified.<sup>24,25</sup> The latest research showed that the increase in adiponectin concentration is associated with the prevention of atherosclerosis, but their clinical relevance remains uncertain.<sup>26</sup> Recent studies found that greater tea consumption among Japanese people reduces the risk of CAD, especially myocardial infarction, with an OR of 0.54 (95% CI

[0.30–0.98]). The same study reported a lower incidence of 3-vessel CAD in the same patient category with an OR of 0.49 (95% CI [0.24–0.98]). However, there is no significant effect of coffee consumption.<sup>27</sup> Regular tea consumption also decreases the progression of coronary artery calcifications over 1 cup a day and is associated with fewer cardiovascular events (adjusted HR 0.71; 95% CI [0.53–0.95]). However, coffee intake does not appear to affect the evolution of coronary calcifications (adjusted HR 0.97, 95% CI [0.78–1.20]).<sup>28</sup>

### • Heart failure

Heart failure is a major health problem in both low and high-income countries.<sup>29</sup> Recent studies show no significant correlation between coffee consumption and heart failure risk in Swedish and Finnish populations, with moderate coffee consumption (1–6 cups daily) reportedly decreasing the risk.<sup>30,31</sup> Although caffeine is known for its positive inotropic effects by inhibiting adenosine receptors and stimulating adrenergic response,<sup>32,33</sup> a recent dose-response meta-analysis reported a J-shape relationship between habitual coffee consumption and the risk of heart failure, with the minimal risk at four cups a day.<sup>34</sup> The exact mechanism is yet to be discovered. Still, it is probably related to the modulatory effects of flavonoids on inflammatory pathways, especially the NF-kappa-B pathway, which is a transcription factor implicated in CVD.<sup>35</sup> As for tea consumption, a novel study showed that a regular intake reduces the risk of heart failure by 28%.<sup>36</sup> This could be related to EGCG, a flavonoid found in green tea, which decreases oxidative stress, inflammation, and telomerase activity in mice suffering from heart failure.<sup>37</sup>

### • Atrial fibrillation (Afib)

Afib is the most common arrhythmia in adults.<sup>38</sup> Dietary habits have an essential impact on cardiac rhythm. Regular intake of tea and coffee seems to have a slight antiarrhythmic effect.<sup>39</sup> An observational population-based study of 130,054 people, showed an inverse relationship between caffeine intake and arrhythmia (HR highest vs. lowest quartile = 0.6;  $p = 0.03$ ). The role of caffeine in enhancing cardiac rhythm is explained by the blockage of calcium reuptake by the sarcoplasmic reticulum during diastole by inhibiting intracellular phosphodiesterase.<sup>40</sup> In contrast, its antiarrhythmic properties are due to the inhibition of adenosine receptors.<sup>41</sup> Coffee has antiarrhythmic properties by inhibiting adenosine A1 and A2A receptors. These adenosine increases arrhythmia risk by shortening refractory periods in the atrium and ventricle. The effects of endogenous adenosine are mitigated by blocking the adenosine receptor through caffeinated coffee.<sup>14</sup>

However, recent studies do not find any association between coffee consumption and the risk of Afib in healthy adults.<sup>42,43</sup> On the other hand, green tea consumption seems to be a protective factor against Afib in the Chinese population (OR: 0.349, 95% CI: [0.253–0.483],  $p < 0.001$ ).<sup>44</sup> This effect is also related to the implication of EGCG flavonoid, which reduces inflammation, oxidative stress, and myocardial remodeling by modulating the NF-kappa-B pathway on rabbit models.<sup>45</sup>

## • Dyslipidemia

Hyperlipidemia increases myocardial infarction risk, necessitating aggressive management. Catechins in tea can prevent LDL particle oxidation by incorporating tea catechins into LDL molecules. The EGCG component of green tea inhibits the Jagged-1/Notch signaling pathway, reducing endothelial dysfunction caused by oxidized LDL. Green tea can inhibit micelle formation in the intestine and ultimately lead to decreased lipid absorption. Also, catechins increase LDL receptor binding activity in HepG2 cells through the SREBP-1 (sterol regulatory binding protein-1) pathway. Even though strong epidemiological evidence exists to support the beneficial effects of green tea on lipid profile, randomized clinical trials (RCT) produced inconsistent results. A meta-analysis by Xu et al included 31 RCTs with a total of 3321 subjects. Primary outcome measurements included LDL, triglyceride (TG), total cholesterol (TC), and high-density lipoprotein (HDL). Weighted mean differences (WMD) were then measured between the intervention and control groups. Results showed that green tea significantly reduces LDL (WMD:  $-4.55$  mg/dL;  $p < 0.0001$ ) and TC (WMD:  $-4.66$  mg/dL;  $p < 0.0001$ ). There wasn't a statistically significant decrease in TG level (WMD:  $-3.77$  mg/dL;  $p = 0.15$ ), but it favored green tea consumption. Also, HDL level (WMD:  $0.23$  mg/dL;  $p = 0.50$ ) was not statistically significant and did not favor green tea with an overall statistically significant result for the heterogeneity test ( $I^2 = 34.8\%$ ;  $p = 0.035$ ).<sup>46</sup> However, Huang et al. performed a longitudinal cohort study over 6 years that proved that tea consumption decreases HDL. Baseline tea consumption was collected and HDL concentrations were measured every 2 years in a group of 80,182 individuals with no CVD, cancer, or lipid-lowering agents. The adjusted mean difference in the HDL-C decreased rate was  $0.010$  ( $p < 0.0001$ ) mmol/L per year for tea consumers versus nonconsumers (never or less than once/month group). Variables such as age, sex, and presence or absence of metabolic syndrome were also considered. Men, people over 60 years old, and those with lower lifestyle scores and higher incidence of metabolic syndrome showed a slower decrease in HDL-C concentrations with tea consumption ( $p < 0.0001$ ).<sup>47</sup> Another meta-analysis performed by Zhao et al on black tea consumption showed favorable outcomes on lipid concentrations. The meta-analysis included 10 studies with no significant heterogeneity and 411 patients. Results illustrated decreased LDL ( $-4.64$  mg/dL;  $p < 0.036$ ). No statistically significant change was detected in TC ( $-2.04$  mg/dL;  $p < 0.363$ ) or HDL ( $-1.15$  mg/dL;  $p < 0.236$ ). The effect of black tea consumption on LDL levels was shown to be more significant in subjects with higher cardiovascular risk.<sup>48</sup>

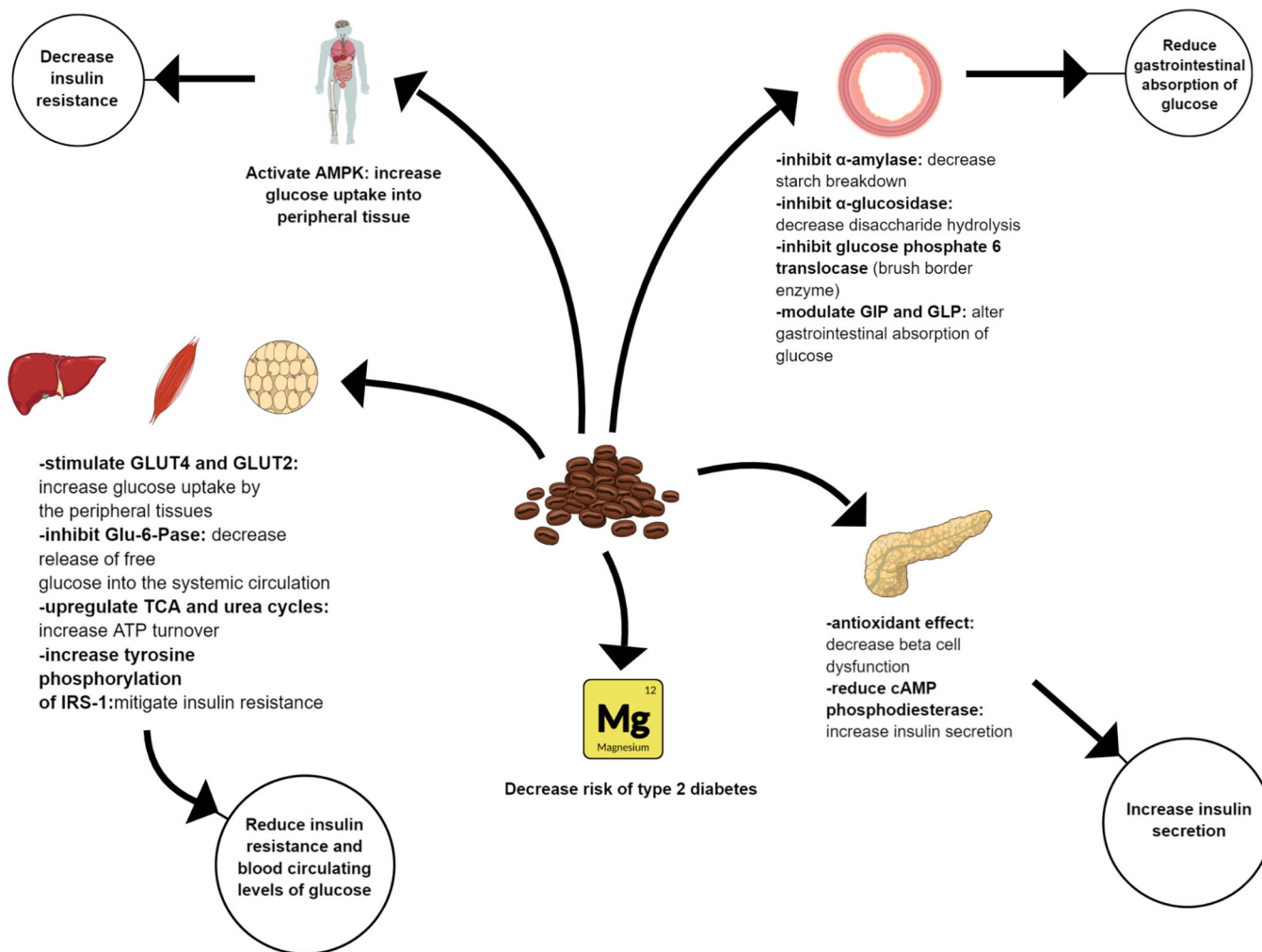
Several epidemiological studies and RCTs generated mixed results in demonstrating the effect of coffee consumption on lipid concentrations. Coffee contains oils/fats known as diterpenoid alkaloids (kahweol and cafestol). They are postulated to be responsible for increasing blood lipids by reducing LDL receptor activity. LDL would then accumulate outside the cells, leading to the development of atherosclerosis.<sup>49</sup> Diterpene cafestol can act as an

agonist of the farnesoid X receptor (FXR), which elevates the cholesterol level in blood serum. FXR is involved in the regulation of cholesterol homeostasis. Furthermore, the competition of cafestol with the endogenous ligand chenodeoxycholic acid for binding to FXR also contributes to the increased cholesterol levels.<sup>50</sup> Another mechanism is increasing cholesterol synthesis as coffee oil decreases the excretion of bile acids and neutral sterols necessary for cholesterol absorption in the intestine. A meta-analysis performed by Cai et al. included 12 studies of a total of 1017 subjects, which showed a TC increase of  $8.1$  mg/dL ( $p < 0.001$ ), TG increase of  $12.6$  mg/dL ( $p < 0.007$ ), and LDL increase of  $5.4$  mg/dL (95% CI:  $1.4, 9.5$ ;  $p < 0.009$ ). Some trials showed that unfiltered coffee had a stronger cholesterol-raising effect than filtered coffee. The process of filtering reduces the amount of coffee oil present. Also, boiled coffee is prepared at a higher temperature, which raises the concentration of coffee oils. This in turn leads to a greater increase in lipid concentrations. Finally, the meta-analysis also showed that there was a dose-response relationship between the increase in TC, LDL, TG, and quantity of coffee consumed. Individuals with hyperlipidemia are especially sensitive to this effect.<sup>51</sup> Data from the Tromso study further validate the negative impact coffee consumption has on lipid concentrations. Compared to individuals who did not consume espresso, TC increased by  $0.09$  mmol/L (95% CI  $0.01$  to  $0.17$  for women and  $0.16$  mmol/L, 95% CI  $0.07$  to  $0.24$  for men) in those who drank 3–5 cups of espresso daily. TC exhibited a greater increase ( $0.30$  mmol/L, 95% CI  $0.13$  to  $0.48$  for women and  $0.23$  mmol/L, 95% CI  $0.08$  to  $0.38$  for men) in individuals who consumed  $\geq 6$  cups of boiled/plunger coffee daily compared with participants drinking 0 cups. Drinking of  $\geq 6$  cups of filtered coffee daily was correlated with  $0.11$  mmol/L (95% CI  $0.03$  to  $0.19$ ) higher S-TC levels for women but not for men. There were significant sex differences for all coffee types except boiled/plunger coffee.<sup>52</sup>

## • Other metabolic disorders that contribute as comorbidities in CVDs

Type 2 diabetes (DM2) is a chronic disease that is increasing in prevalence. By the year 2030, an estimated global number of people with diabetes is expected to reach 366 million.<sup>53</sup> Many animals, cohorts, and RCTs have studied the effect of coffee consumption on diabetes and obesity. The mechanism through which the components of coffee decrease the risk of diabetes is summarized in Figure 1.<sup>49,54</sup> Ding et al's systematic review and meta-analysis of 45,355 diabetes cases found a strong inverse dose-dependent association between coffee consumption and diabetes risk, with a 33% lower risk in participants who drank 6 cups/day. The association was consistent among men and women and across European, US, and Asian populations, suggesting coffee components are responsible for its antidiabetic effect. However, due to the observational nature of these studies, a causal relationship between coffee consumption and lower risk of diabetes cannot be concluded.<sup>53</sup>

Moon et al.'s meta-analysis of RCTs found no significant effect of long-term coffee consumption on insulin resistance and sensitivity, but noted limitations such as short duration, confounding



**FIGURE 1** Figure showing the different mechanisms of action of the components of coffee in decreasing the risk of diabetes. AMPK, AMP-activated protein kinase; cAMP, cyclic AMP; GIP, gastric inhibitory polypeptide; GLP, glucagon-like peptide; GLUT 2/4, Glucose transporter 2/4; Glu-6-Pase, glucose-6-phosphatase; IRS-1, insulin receptor substrate 1; TCA, tricarboxylic acid cycle.

**TABLE 1** Summary of the possible mechanism of action of green tea on glucose metabolism as proven by animal studies.

Effect of green tea on glucose metabolism (proven by animal studies)
EGCG increases tyrosine phosphorylation of the insulin receptor and insulin receptor substrate-1 in H4IIE leading to decreased hepatic glucose production
Increases insulin sensitivity and glucose metabolism
EGCG enhances glucose tolerance and preserves islet structure
EGCG promotes GLUT-4 translocation in skeletal muscle thus increasing glucose uptake
EGCG increases anti-inflammatory markers (interleukin-10)

Abbreviations: EGCG, epigallocatechin; GLUT-4, glucose transporter-4.

variables, and small sample size.<sup>55</sup> Therefore, more well-constructed RCTs must be performed to validate or reject the hypothesis demonstrated by cohort studies. Another meta-analysis by Kondu et al further validates the one prepared by

Moon et al. However, it showed that there is a statistically significant decrease in fasting blood glucose in individuals <55 years of age only with green tea. The postulated mechanisms of action of green tea on reducing blood glucose are summarized in Table 1.<sup>56</sup> Green tea supplementation reduces fasting glucose but does not affect insulin or HbA1c. Short-term trials validate these findings but long-term trials are needed to assess the effects of green tea consumption on glycemic control.<sup>57</sup> Zhang et al's study on high-risk diabetic Chinese populations found worsening glucose metabolism due to lower beta cell functional capability.<sup>58</sup> These results demonstrate that the relationship between tea consumption and diabetes remains controversial and requires further studies.

As for obesity, many of the effects of coffee are through its relation to lipid metabolism, which is summarized in Table 2. Coffee is also ergogenic and thermogenic<sup>54</sup> and has the ability to inhibit the multiplication of adipocytes and affect gastrointestinal microbiota.<sup>59</sup> A meta-analysis by Lee et al showed that from 12 epidemiological studies, particularly in men, higher coffee intake might be modestly

**TABLE 2** Summary of possible mechanism of action of coffee on obesity.

Mechanism of action of coffee on obesity
Downregulates of SREBP-1: decrease lipogenesis
Upregulates of PPAR-alpha
Inhibits of HMGCoA/FAS: inhibit fatty acid and cholesterol synthesis
Stimulates CPT: stimulate lipid beta-oxidation
Inhibits of LDL oxidation

Abbreviations: CPT, carnitine palmitoyltransferase; FAS, fatty acid synthase; HMGCo-A reductase, 3-hydroxy-3-methylglutaryl coenzyme A reductase; PPAR- $\alpha$ , peroxisome proliferator-activated receptor alpha; SREBP-1, sterol regulatory element-binding protein.

**TABLE 3** Summary of possible mechanism of action of tea on obesity.

Mechanism of action of tea on obesity
Reduces food consumption by modulating neuroendocrine metabolic regulators
Reduces absorption of lipids and protein in the gastrointestinal tract
Acts on gastrointestinal microbiota which ultimately increases the rate of lipid metabolism
Inhibit differentiation and proliferation of pre-adipocytes
Reduce lipid production, promote lipolysis, induce lipid metabolism
Stimulate conversion of white into brown adipose tissue
Promote fecal lipid excretion

associated with reduced adiposity.<sup>60</sup> The mechanism of action of tea on obesity is summarized in Table 3. There has been a heterogeneity of results on the effect of green tea on obesity. Lin et al. demonstrated in a most recent meta-analysis of 26 randomized trials that green tea supplementation significantly decreases body weight (BW) and body mass index but has no effect on weight circumference. Also, they illustrated that green tea supplementation follows a dose-response pattern for BW ( $p < 0.001$ ). Obvious outcomes were recorded for dosages  $< 500$  mg/day for 12-week duration.<sup>61</sup>

### 2.3 | Effects of tea and coffee consumption on helping in the recovery from CVDs

The impact of tea and coffee consumption on the recovery of CVDs has been extensively studied, yielding mixed results.<sup>62–66</sup> Green tea consumption reduces all-cause and CVD mortality,<sup>64</sup> with three cups per day improving survival. Coffee also reduces CVD all-cause mortality,<sup>63</sup> but caution is needed as higher consumption may increase risk for pre-existing CVD.<sup>66</sup>

The impact of tea and coffee consumption on CVD recovery is uncertain, but polyphenols may have antioxidant and anti-inflammatory

benefits. Consuming three or one cup daily may lower mortality risk, in comparison with those who don't consume them at all. Chen et al. prospective cohort study demonstrated the combination of 1–2 cups/day of coffee and 2–4 cups/day of tea exhibited decreased mortality risks for all-cause (HR, 0.78; 95% CI: 0.73–0.85) and CVD (HR, 0.76; 95% CI: 0.64–0.91) mortality in combined analyses compared to neither coffee nor tea use.<sup>65</sup> Moreover, caffeine has the potential to raise blood pressure and heart rate, which could be harmful to some individuals' cardiovascular health.

### 2.4 | Effects of tea and coffee consumption on reducing the mortality of CVDs

Coffee consumption may reduce cardiovascular risks like metabolic syndrome, obesity, and depression risk, but its impact on lipid profiles may be unfavorable. However, evidence suggests habitual coffee consumption can neutrally or positively impact cardiovascular events like coronary heart disease, congestive heart failure, arrhythmias, and stroke. Regular coffee consumption of 2–3 cups daily is safe, with neutral to favorable effects across most health outcomes.<sup>67</sup> Even with those who have already developed diabetes, consuming coffee might exhibit an inverse correlation with mortality risk.<sup>68</sup> Prior large prospective cohort studies have demonstrated this inverse relationship in elderly patients<sup>69</sup> even after smoking was attributed as an important confounding variable.<sup>70</sup> Ding et al. prospective cohort show higher coffee consumption is associated with lower CVD mortality risk,<sup>71</sup> but in hypertension patients, heavy coffee consumption increases the risk, unlike in those without or with grade 1 hypertension.<sup>72</sup>

Although animal experiments have yielded encouraging findings concerning tea's impact on blood pressure and various biomarkers related to CVD, the results observed in human studies are limited and somewhat less satisfying. A prospective cohort study executed in Japan showed that green tea consumption is associated with reduced all-cause mortality and CVD-related mortality but not cancer-related mortality.<sup>73</sup> Chen et al. performed a prospective cohort study on 498,158 participants to identify the association between tea consumption and all-cause and cause-specific mortality. The results showed an inverse J curve between quantity of tea consumption and mortality. The outcome did not support the notion that excessive coffee consumption is linked to reduced mortality. The study highlights that incorporating a moderate intake of less than 1–2 cups per day of coffee or 2–4 cups per day had an inverse relationship with CVD-related mortality.<sup>65</sup> Compared to coffee, green tea remains to have a beneficial effect on mortality in those who have hypertension.<sup>72</sup> Finally, an umbrella review of 23 systematic reviews further validates that moderate consumption of two cups of tea will decrease CVD risk and mortality and may be encouraged to be incorporated into individuals' diets as the side effect profile of tea is minimal in moderate amounts.<sup>9</sup> Additional research is essential to elucidate the potential mechanisms underlying the association between combined coffee and tea consumption and mortality.

In CVD populations as demonstrated by Zheng et al. study,<sup>74</sup> once pertinent confounders were taken into account ( $p < 0.05$ ), the findings showed that patients with CVD who drank four or more cups of coffee (HR = 1.389, 95%CI: 1.385–1.393), iced tea (HR = 1.639, 95%CI: 1.632–1.645), or hot tea (HR = 1.529, 95%CI: 1.520–1.538) per day had a higher chance of dying from all causes. However, decaffeinated coffee, decaffeinated iced tea, decaffeinated hot tea, and one to three cups of hot tea per day may lower the risk of all-cause death in the population with CVD. The risk of CVD death in the CVD population may be increased by coffee consumption (1–3 cups per day, HR = 1.266, 95% CI: 1.262–1.271;  $\geq 4$  cups per day, HR = 2.027, 95%CI: 2.018–2.036), iced tea consumption ( $\geq 4$  cups per day, HR = 1.773, 95%CI: 1.763–1.784), hot tea consumption (1–3 cups per day, HR = 2.005, 95% CI: 1.998–2.012), decaffeinated iced tea (frequency always, HR = 1.434, 95% CI: 1.430–1.439), and decaffeinated hot tea (HR = 1.725, 95% CI: 1.719–1.731). On the other hand, individuals with CVD had a lower risk of dying from the disease when they drank iced tea (1–3 cups daily, HR = 0.870, 95% CI: 0.866–0.873), decaffeinated coffee, and decaffeinated iced tea (approximately 25% to 75% of the time, HR = 0.728, 95% CI: 0.725–0.730).

## 2.5 | Effect of tea and coffee on different populations in different geographical areas

The literature on tea and coffee consumption's impact on various populations is limited, but studies suggest green tea reduces all-cause and CVD mortality in Japanese populations,<sup>75</sup> while higher intake may increase CVD mortality risk.<sup>76</sup> Coffee and tea consumption patterns in European adults vary significantly, influenced by geographical regions and cultural differences, potentially contributing to diverse health effects. People consume more diluted coffee in Denmark, while stronger coffee in lesser quantities (such as Turkish coffee or ristretto coffee) is consumed in Greece and Italy. The average coffee cup in Italy weighs 55 g, while in Denmark it's 182 g.<sup>77</sup> However, uncertainties remain about the effects of tea and coffee consumption on different populations and regions, necessitating further research.<sup>78</sup>

## 2.6 | Drawbacks of tea and coffee consumption on cardiovascular health

The risk of tea and coffee consumption on cardiovascular death is dose-dependent. In a retrospective cohort study by Zheng et al. the high consumption of caffeinated coffee and tea was associated with an increased risk of death in CVD patients. Therefore, those with CVDs should limit their intake of caffeine, in which  $< 3$  cups per day were found to be the safe-zone dose and simultaneously can reduce the risk of all-cause and CVD mortality. Caffeine promotes the release of catecholamines and activates sympathetic nerves and renin-angiotensin systems.<sup>74</sup> Elevated levels of catecholamines in the

blood are thought to cause lethal ventricular arrhythmias and, ultimately sudden cardiac death. Catecholamines are thought to cause such arrhythmogenic effects by acting on  $\alpha$ -adrenoceptors in coronary arteries to cause coronary spasm and subsequent myocardial ischemia as well as on  $\beta$ -adrenoceptors in cardiomyocytes to excessively increase the concentration of cyclic AMP and produce defects in intracellular  $\text{Ca}^{2+}$  handling.<sup>79</sup> Sympathetic nervous system and  $\beta 1$ -receptor activation may result in susceptibility to arrhythmias, due to its inotropic and chronic effects. Higher caffeine concentrations increase intracellular cAMP and cyclic guanosine monophosphate by inhibiting nonspecific phosphodiesterases, which influence cardiac contractility due to calcium release.<sup>80</sup>

Consumption of caffeine should be limited to a single dose of 200 mg in healthy people without comorbidities, as it will not cause any toxic effects.<sup>81</sup> Caffeine intoxication can happen in the dose consumption of 300 mg or above. Symptoms related to the cardiovascular system include tachycardia and arrhythmia.<sup>82</sup> In people with severe hypertension, except those with grade 1 hypertension, heavy coffee consumption was associated with an increased risk of cardiovascular death. On the contrary, green tea consumption doesn't have any association with CVD deaths in all blood pressure categories.<sup>72</sup>

## 2.7 | Future recommendations and conclusions

While coffee and tea consumption has traditionally been regarded as one of the most common risk factors contributing to cardiovascular deaths, recent evidence has shown that their consumption has been associated with many benefits. The role of coffee and tea in reducing CVDs is due to their antioxidant and bioactive substances, such as phenolics, flavonoids, CGAs, and EGCG. Furthermore, caffeine's effects on inhibiting adenosine receptors and stimulating adrenergic response play a big role in decreasing the risk of CVDs. However, one must be careful when consuming them (maximum single dose of 200 mg) as overdoses of caffeine (300 mg single dose) may also result in an increased risk of CVD mortality. More future research that focuses on the impact of tea and coffee consumption based on geographical area and different cultures, the synergistic effect of being consumed together, and the role of genetic factors in the effect are needed.

### AUTHOR CONTRIBUTIONS

Laurentia Fidella Averina Setia Santoso, Kristina Nasr, Adam Maamar Roumani, and Hadi Khaled Abou Zeid performed the literature search and wrote the manuscript. Mohammed Shawki Dabbous gave input critically as a mentor. Laurentia Fidella Averina Setia Santoso led this review-making process and had the idea of this review topic, Kristina Nasr also made the figures and tables on this manuscript; therefore, they are both regarded as first authors. Laurentia Fidella Averina Setia Santoso is the corresponding author.

All authors have read and approved the final version of the manuscript. Laurentia Fidella Averina Setia Santoso had full access to

all of the data in this study and takes complete responsibility for the integrity of the data and the accuracy of the data analysis.

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## CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.


## DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no data sets were generated or analyzed during the current study.

## TRANSPARENCY STATEMENT

The lead author Laurentia Fidella Averina Setia Santoso affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

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