QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

WINDYGA, Maria, SIECZKA, Anna, MAKOWSKA, Joanna, DARMOŃ, Jerzy, DOMAŃSKA, Karolina, PAPUGA, Aleksandra, SROKA, Maria, PIEGZA, Natalia and HEJDUK, Makary. Coffee and Heart. Association Between Brewing Methods and Cardiovascular Disease Risk: A Narrative Review of Current Evidence. Quality in Sport. 2026;68:74340. https://doi.org/10.12775/QS.2026.68.74340

ARTICLE TIMELINE
Received: 15.07.2026. Revised: 02.08.2026. Accepted: 02.08.2026. Published: 11.08.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Coffee and Heart. Association Between Brewing Methods and Cardiovascular Disease Risk: A Narrative Review of Current Evidence

Authors:

Maria Windyga

ORCID https://orcid.org/0009-0006-6937-909X

E-mail maria.windyga@poczta.fm

5th Military Hospital with Polyclinic in Kraków, Poland

Anna Sieczka

ORCID https://orcid.org/0009-0003-5509-3486

E-mail asieczka1517@gmail.com

Independent Public Health Care Facility in Myślenice, Poland

Joanna Makowska

ORCID https://orcid.org/0009-0000-6342-8379

E-mail joannamakowska10@gmail.com

Independent Public Health Care Facility in Myślenice, Poland

Jerzy Darmoń
ORCID https://orcid.org/0009-0008-3139-6366
E-mail darmonjerzy@gmail.com
Medical University of Silesia in Katowice, Poland

Karolina Domańska
ORCID https://orcid.org/0009-0009-1701-7961
E-mail karolina.domanska54@gmail.com
Independent Public Health Care Facility in Myślenice, Poland

Aleksandra Papuga
ORCID https://orcid.org/0009-0003-6851-7102
E-mail amp.papuga@gmail.com
Independent Public Health Care Facility in Myślenice, Poland

Natalia Piegza
ORCID https://orcid.org/0009-0002-9348-0113
E-mail nataliapiegza9@gmail.com
Independent Public Health Care Facility in Myślenice, Poland

Makary Hejduk
ORCID https://orcid.org/0009-0004-7579-4471
E-mail makaryhejduk@gmail.com
Independent Public Health Care Facility in Myślenice, Poland

Maria Izabela Sroka
ORCID https://orcid.org/0009-0002-8743-8963
E-mail marsrokaa@gmail.com
Frederic Chopin University Clinical Hospital in Rzeszów, Poland

Corresponding Author:

Maria Windyga, maria.windyga@poczta.fm

Abstract

Background

Coffee is one of the most frequently used central stimulants. It was commonly believed that its influence on cardiovascular health is poor. Recent research questions those beliefs suggesting neutral or protective associations - depending mostly on method of coffee preparation.

Aim

The aim of this narrative review is to synthesize current evidence on how type of brewing, roasting degree, quantity, and timing of coffee intake are associated with cardiovascular risk, and to connect these associations with the bioactive components of coffee.

Materials and Methods

A narrative review of cohort studies, dose-response and umbrella meta-analyses, a Mendelian randomization study, and a review of coffee's inflammatory effects, published predominantly within the last decade.

Results

Brewing method appears as the most significant factor influencing cardiovascular risk: filtered coffee was associated with neutral to even protective cardiovascular outcomes, while unfiltered were linked to higher LDL-cholesterol. These differences are the results of the diterpenes which are largely removed by paper filtration. Roasting degree influenced mostly antioxidants content. Quantity followed a J-shaped dose-response curve, with cardiovascular risk minimized at three to four cups per day. Morning coffee consumption was preliminary associated with lower cardiovascular mortality. Association with increased atrial fibrillation risk was excluded.

Conclusions

The association between coffee consumption and total serum cholesterol has been known since 1983. Later studies identified the lipid-raising components of coffee - the diterpenes, which concentration in the final brew depends strongly on the brewing method. A portion of unfiltered brewed coffee contains about 30 times the concentration of the diterpenes compared to filtered one. Filtered preparation, moderate intake, and morning consumption are associated with the most favorable cardiovascular profile.

Keywords

coffee; coffee brewing method; cardiovascular risk; cholesterol; cafestol and kahweol; polyphenols; diterpenes; coffee roasting; coffee consumption timing; dose-response; inflammation; atrial fibrillation

1. Introduction

Coffee is one of the most widely consumed stimulants in the world. Only in the United States, 85% of adults consume caffeine daily and average caffeine intake is 135 mg per day, which is equivalent to about 1.5 standard cups of coffee (with a standard cup defined as 8 fluid oz [235 ml]) [1, 2]. It has been consumed for hundreds of years and has become an important part of cultural traditions and social life [2]. In XVII and XVIII century coffee was even said to be a panacea [1]. Its popularity was a breakthrough in development of democracy, science and popularized sobriety. Its influence on cardiovascular health has been the subject of scientific debate for decades. Early observational studies from the mid-20th century raised concern that coffee drinking increased the risk of coronary heart disease, while more recent, larger, and better-adjusted cohorts have reported neutral or even protective associations for moderate consumption [1, 3]. This alteration reflects the fact that coffee is not a single, uniform exposure. There were identified 130 species included in Coffea genus, although only two support cultivation and trade, accounting for 99% of global production. [4]. Coffea arabica is the most popular among these species, responding for 60% of global usage. [4]. Coffea canephora (Robusta) is contributing the remaining 40% [4]. These two species also show notable divergence in beverage quality attributes, namely body, aroma and taste with C. arabica being more appreciated, especially for its superior cup quality and aroma, whereas a robusta brew has harsher flavor and contains higher amounts of soluble solids, caffeine and phenolic compounds [4, 5]. The beverage delivered to a cup depends heavily on how the beans are roasted, how the brew is prepared (filtered, unfiltered/boiled, espresso, instant), how many cups of coffee are consumed, and even at what time of day it is drunk. Each of these variables changes the concentration of bioactive compounds: caffeine, chlorogenic acid, trigonelline, melanoidins and the diterpenes: cafestol and kahweol reaching the cardiovascular system [6, 7, 8]. It even contains modest amounts of magnesium, potassium, and vitamin B3 (niacin) [2].

Cardiovascular disease (CVD) remains the leading cause of death worldwide which is manageable. The primary prevention of it is increase of physical activity and dietary

modification. Because coffee is consumed daily by many adults, even small, preparation-dependent shifts in cardiovascular risk may be significant for public health relevance. Yet clinical and public guidance on coffee tends to treat it as a single category, overshadowing meaningful differences in risk between filtered brews and unfiltered or espresso-style preparations common in Southern Europe [1, 3, 9]. Large cohort studies illustrate this divergence: filtered coffee has been associated with lower cardiovascular and all-cause mortality relative to unfiltered brews in a Norwegian cohort of over 500,000 adults, whereas Italian cohorts drinking predominantly espresso have shown a dose-dependent increase in coronary heart disease risk with higher intake [1, 3]. A UK Biobank analysis further disaggregating ground, instant, and decaffeinated subtypes found that all forms were associated with reduced incident cardiovascular disease, but that only caffeinated forms reduced arrhythmia risk, and only within specific dose ranges [9].

Cafestol and kahweol, diterpenes largely preserved in unfiltered and espresso brew but mostly removed by paper filtration, suppress hepatic LDL-receptor activity and raise LDL-cholesterol [7, 8, 10]. Roasting, in turn, generates melanoidins with antioxidant properties while degrading some chlorogenic acid, and may independently influence blood pressure and metabolism even when filtration is held constant [10, 11]. Chlorogenic acid, trigonelline, and other polyphenols have been proposed as anti-inflammatory and antioxidant agents: a review of coffee's constituents by Frost-Meyer and Logomarsino found consistent reductions in pro-inflammatory mediators such as TNF-α, IL-1β, and MCP-1 in animal models [12]. Findings in human trials remain unclear, likely reflecting the same preparation-dependent variability seen in the cardiovascular literature [12]. Dose-response meta-analyses further suggest a non-linear, J-shaped relationship between coffee intake and cardiovascular outcomes, with the lowest risk typically observed at three to four cups per day and no clear evidence of harm at higher intakes [13, 14, 15]. Recently, attention has turned to chrononutrition: a 2025 analysis of NHANES data found that a morning-restricted coffee-drinking pattern, but not an all-day pattern, was associated with lower cardiovascular mortality, suggesting that timing may independently modulate cardiovascular risk [16]. At the same time, Mendelian randomization evidence has failed to demonstrate a predicted coffee consumption effect on cardiovascular outcomes, emphasizing that much of the observational literature may be created by confounding and reverse causation rather than a direct physiological effect of coffee itself [17].

Coffee Compounds

Coffee is rich in antioxidants, including polyphenols which can inhibit oxidation of LDL, exert anti-thrombotic effects, and improve endothelial dysfunction [1].

CGA – chlorogenic acid

Chlorogenic acid is a main phenolic compound of coffee [5]. CGA hydrolyzes to caffeic and quinic acid. Its quantity is dependent on the type of bean and roasting - lighter roasts have the greatest amount of CGA and caffeic acid content whilst darker roasts contain 10% less CGA than the lighter ones, which is caused by intense heat. Heating also transforms CGA into quinolactones and melanoidins which may lead to increased antioxidant activity [12].

Caffeine

An alkaloid which content depends on the type of bean, brewing strength and roasting process. Arabica-brewed coffee may contain 36–112 mg/100 ml caffeine, while its quantity in Robusta may vary from 56 to 203 mg/100 ml. Caffeine is catabolized to xanthine and theobromine which decrease DNA degradation and reduce hydroxyl radicals. In a study carried out by Li et al. (2011) the authors reported less IL-6 and TNF-α in caffeine drinking rats. Caffeine also antagonizes adenosine receptors A1 and A2 resulting in stimulation of central nervous system, elevation of blood pressure and increased metabolism [12].

Cafestol and kahweol

Diterpenes, the most important compound of coffee regarding this narrative research. Their concentration is connected mostly with roasting and brewing process. They are antioxidants however their negative effect relates to increase in both total cholesterol and low-density lipoprotein cholesterol (LDL). Cafestol increases cholesterol esterase transfer protein (CETP) which, in turn, elevates LDL. Both cafestol and kahweol allow less LDL to be drawn in by cell receptors, thus increasing the serum load. Positive effects of diterpenes concentrate around reduction of the amount of COX-2, MCP-1, iNOS by kahweol – anti-inflammatory and anti-angiogenic effect [12].

The trolox antioxidant capacity (TEAC) of cafestol and kahweol is 3–4 times higher in instant than in ground coffee, giving instant coffee greater influence in reduction of hydroxyl radicals. Overall, a positive linear relationship exists between TEAC and coffee phenolics [12].

Trigonelline

Trigonelline is transformed to N-methylpyridinium during the roasting process improving its TEAC. Afterwards it is demethylated to nicotinic acid which reduces MCP-1 and upregulates adiponectin in adipocytes infused with TNF-α. The higher the roasting temperature, the greater the conversion to nicotinic acid [12].

Aim of the study

Summarizing, the base of evidence is large but divided across brewing method, roast degree, dose, and timing, with few studies integrating these dimensions or comparing them to cardiovascular outcomes on population-level. The aim of this narrative review is to synthesize current evidence on how brewing method, roasting type, quantity, and timing of coffee intake are associated with cardiovascular risk. Considering these results and connecting them to the underlying bioactive components of coffee it is possible to understand why the cardiovascular effects of coffee reported in the literature are so heterogeneous - and what this means for future research and practice.

2. Materials and methods

This narrative review is based on literature indexed in PubMed (MEDLINE), MDPI, ResearchGate, ScienceDirect databases up to July 2026. The search strategy included combinations of the following keywords: coffee, coffee brewing method, cardiovascular risk, cholesterol, cafestol and kahweol, diterpenes, polyphenols, antioxidants, coffee roasting, coffee consumption timing, dose–response, inflammation, atrial fibrillation. The search was limited to English publications between 2009 and 2026, mostly from journals with high impact factor. Data from older searches publicized between 2009 and 2012 were tailored to coffee compounds and roasting degrees. Selected articles were manually screened to identify the relevant ones, corresponding with the aim of the review. Abstracts without full-text availability and studies with insufficient methodological quality were excluded from the review.

AI was used for language purposes in this research, enabling adherence to scientific writing standards, proper Englis grammar, style and clarity in the presentation of results. The final interpretation of data was purely conducted by human.

The aim of this narrative review is to synthesize current evidence on association between coffee preparation and cardiovascular risk.

3. Research results

Brewing method

The most important factor regarding influence on cardiovascular risk concerns brewing method. Basically, coffee could be brewed using two main methods: letting the coffee infused water through the paper filter resulting in a drip-brewed beverage or by directly letting the ground coffee beans simmer in close-to-boiling water (unfiltered method). Unfiltered coffee contains a substantial amount of the low-density lipoprotein (LDL)-cholesterol, increasing diterpenes kahweol and cafestol that might contribute, at least theoretically, to a higher IHD risk [1]. In espresso coffee, a more efficient extraction (solids, fine particles and oil droplets) is expected, which results in higher diterpene contents compared to filtered coffee brews. It is possible that the free forms of these compounds are more soluble in hot water, especially cafestol, considering the presence of hydroxyl groups in their structures and their small size. In addition, when esterified, kahweol may be bonded to long chain fatty acids, consequently it has a higher molecular weight and lower polarity, thus decreasing its extraction by hot water [18].

In a Norwegian cohort of 508,747 adults followed for a mean of 20 years, Tverdal et al. found that both filtered and mixed (filtered and unfiltered) coffee consumption was associated with lower cardiovascular and all-cause mortality relative to no coffee, whereas unfiltered brew showed an attenuated benefit. Moreover, among adults aged 60 and older, it showed an increased hazard ratio for cardiovascular disease (HR 1.19 in men) [1]. This contrasts with findings from the Italian EPICOR cohort (n = 43,249), in which espresso/mocha-style coffee - the dominant unfiltered preparation in that population - was associated with a dose-dependent increase in coronary heart disease risk, reaching HR 1.52 for more than four cups per day compared with fewer than one [3]. A UK Biobank analysis of 449,563 participants further disaggregated exposure into ground, instant, and decaffeinated subtypes: all three were associated with reduced incident cardiovascular disease at moderate intakes (lowest risk at 2–3 cups/day), but only ground and instant (caffeinated) coffee reduced arrhythmia risk, with decaffeinated coffee showing no such association [9].

Roasting type

There are three main roast degrees: light, medium and dark depending on temperature and amount of time the beans are spending on the stove. Coffee roasting is classified by the

bean's finishing temperature and total roast time relative to two audible markers, "first crack" (~196–202°C, when trapped moisture and CO_2 rupture the bean structure) and "second crack" (~224–226°C, when the bean's cellular structure breaks down further and surface oils emerge). Roasting proceeds through a drying phase (up to ~160°C), the Maillard reaction (~160–195°C), and development past "first crack" and "second crack". Longer, hotter roasts degrade chlorogenic acid while generating more melanoidins; lighter roasts preserve more original polyphenol content [19].

Light roast: total roast time of 6-9 min, maximum temperature is 196-205°C, finish at/just after the first crack [20].

Medium roast: total roast time of 7-11 min, maximum temperature is 210-219°C, finish past the first crack [20].

Dark roast: total roast time 10-15+ min, temperature is 220 - above 245°C, finish past second crack [20].

Kinetic modeling confirms this is a temperature- and time-dependent, first-order (Arrhenius-compliant) degradation process: roasting Coffea arabica at 230°C for 12 minutes reduced total chlorogenic acid content by roughly 50%, while 250°C for 21 minutes reduced it to near-trace levels [18, 20].

A controlled clinical trial by Cornelis et al. discovered that, once coffee was prepared using alternative methods, roasting degree was not independently associated with increases in total or LDL-cholesterol, although both medium-light and medium roasts raised LDL-cholesterol relative to baseline [7]. Roast-dependent quantification of cafestol and kahweol across different brewing types confirms that diterpene content is driven primarily by filtration rather than roast level [10]. Separately, a controlled intervention comparing green and roasted coffee blends found reductions in body weight, abdominal adiposity, and blood pressure in hypercholesterolemic subjects, with lipid and insulin-resistance improvements limited to that subgroup - suggesting roast-related polyphenol content may modulate metabolic, rather than purely lipid, pathways [11].

Quantity (dose–response)

The relationship between coffee intake and cardiovascular risk is consistently non-linear. Ding et al.'s meta-analysis of 36 prospective cohorts (>1.2 million participants) found

the lowest cardiovascular risk at approximately 3.5 cups/day, with no evidence of increased risk at higher intakes [13]. The BMJ umbrella review by Poole et al., synthesizing 201 meta-analyses of observational data and 17 of randomized trials, similarly found the greatest reduction in relative risk of death from all causes and from heart disease at around three cups/day, with benefit plateauing above that threshold [14]. The Moli-sani study, in a large Italian population, validated a protective dose-response pattern for cardiovascular and total mortality [15].

Timing of intake

The most recent research concerns when coffee is consumed. Wang et al., analyzing NHANES data (n = 40,725) alongside a validation cohort, identified a "morning-type" drinking pattern (before ~11:30 a.m.) associated with significantly lower all-cause mortality (HR 0.84) and cardiovascular mortality (HR 0.69) relative to non-drinkers. This association was not observed for an "all-day-type" pattern consuming equivalent total quantities. However, timing-based evidence remains preliminary relative to the brewing-method and dose-response literature [16].

Arrhythmia as a specific endpoint

Caffeine's sympathomimetic action was thought to be leading to arrhythmia, resulting in inclusion of atrial fibrillation (AF) as a distinct outcome. In the Physicians' Health Study (n = 18,960 male physicians), a lower AF risk was observed only within a narrow window of 1–3 cups/day, with no association below or above this range [21]. A subsequent dose - response meta-analysis pooling 723,825 participants and over 30,000 AF events found no overall increase in AF risk with higher coffee consumption, consistent with the UK Biobank finding that coffee containing caffeine reduced arrhythmia incidence at moderate doses [22, 9].

Causal evidence

Observational studies of this kind are vulnerable to confounding regarding countless number of factors affecting it like diet, socioeconomic status, and smoking. Nordestgaard and Nordestgaard addressed this using Mendelian randomization, analyzing coffee consumption with twelve genetic variants in UK Biobank and FinnGen data. Predicted coffee consumption was not associated with any of fifteen cardiovascular outcomes examined, a null result that persisted after adjustment for predicted BMI and smoking [17].

4. Discussion

Summarizing studies used in this review; it is hard to support a single directional claim about cardiovascular risk. Brewing method appears to be the dominant modifier: cohorts dominated by filtered consumption (Norway, UK Biobank ground/instant subtypes) report neutral or protective associations, while cohorts dominated by unfiltered, espresso-style preparation (Italy) report a dose-dependent increase in coronary risk [1, 3, 9]. The easiest explanation is diterpene exposure: cafestol and kahweol, retained in unfiltered and espresso preparations but largely removed by paper filtration, suppress hepatic LDL-receptor activity and inhibit bile-acid synthesis, raising LDL-cholesterol which is not attributable to roast degree once filtration is held constant [7, 10]. Considering this roast degree more accurately modulates the antioxidant (melanoidin, chlorogenic acid) content of the brew, which may explain metabolic effects such as those seen with green/roasted blends without materially changing lipid outcomes for which the brewing method holds responsibility [11, 7].

This component-level framing allows to reconcile the divergence Frost-Meyer and Logomarsino identified between animal and human inflammatory-marker studies [12]. Their review found that coffee constituents consistently reduced TNF-α, IL-1β, and MCP-1 in animal models, but human trial results were markedly inconsistent - the authors attributed it to uncontrolled variation in preparation method, roast, and dose across studies. This is unsurprising considering most of a human study pooling filtered and unfiltered, light- and dark-roast coffee under a single "coffee consumption" exposure variable. In effect, averaging over populations with materially different diterpene and polyphenol intake blurs any true anti-inflammatory signal from chlorogenic acid and trigonelline.

The dose-response and timing literatures complicate this picture. The consistent J-shaped curve, with risk minimized around three to four cups per day, is compatible with a model in which the antioxidant and vasodilatory benefits of polyphenols and caffeine-mediated adenosine antagonism dominate at moderate intake, while at higher intake, cumulative diterpene exposure (in unfiltered preparations) and sympathetic activation from caffeine become proportionally more influential - though the absence of a clear harm signal at high intakes in filtered-coffee-dominant cohorts suggests this trade-off is preparation-dependent rather than fixed [13, 14, 15]. The 2025 finding that morning-restricted intake, but not equivalent all-day intake, predicts lower cardiovascular mortality is less resolved [16]. The possible explanations include circadian variation in cortisol and inflammatory tone, or

interaction with sleep timing, but this evidence currently weak - based on a single major analysis (already subject to published methodological critique) and it should be treated as hypothesis.

The atrial fibrillation literature is the clearest: across both the Physicians' Health Study and larger pooled meta-analyses, coffee does not increase AF risk nevertheless it may modestly reduce it within a moderate dose window, directly countering older assumptions that caffeine's sympathomimetic effects would be arrhythmogenic in habitual drinkers [21, 22].

The most important limitation cutting across this entire work is that nearly all of it is observational. The null result from Nordestgaard and Nordestgaard's Mendelian randomization analysis is a necessary counterweight: it does not disprove the associations described above, but it does mean that residual confounding by diet, socioeconomic patterning, or health-related behavior change (reverse causation, where illness precedes and causes reduced coffee intake) cannot be excluded as a partial explanation for the protective associations reported in cohort studies [17].

5. Conclusion

The evidence assembled in this review does not support treating coffee as a single, uniform cardiovascular exposure. Its relationship with cardiovascular risk is better understood as a function of four interacting variables, which depend on preparation: brewing method, roast degree, quantity, and timing - each of which changes the balance of bioactive compounds, principally caffeine, chlorogenic acid, trigonelline, melanoidins, and the diterpenes cafestol and kahweol, that reach the cardiovascular system.

Among these, brewing method emerges as the strongest modifier: paper filtration removes the diterpenes responsible for LDL-cholesterol elevation, which explains why filtered-coffee-dominant cohorts in Norway and the UK report neutral-to-protective associations while an espresso-dominant Italian cohort reports increased coronary risk with higher intake [1, 3, 7, 9, 10]. Contrary roast degree appears to act less on lipid pathways and more on the antioxidant and metabolic profile of the brew, via melanoidins and chlorogenic acid [10, 11]. Quantity follows a consistent J-shaped curve, with cardiovascular risk minimized around three to four cups per day and no evidence of harm at higher intakes in the populations studied, while timing - specifically, morning-restricted consumption - has emerged as a promising but still preliminary independent factor [13-16]. Atrial fibrillation, once a central concern regarding

caffeine's cardiovascular safety, is not meaningfully increased by moderate coffee intake in either individual cohorts or pooled meta-analyses [21, 22].

This preparation-dependent model also conciliates inconsistencies in the literature: Frost-Meyer and Logomarsino's finding of robust anti-inflammatory effects in animal studies but inconsistent effects in human trials [12] is well explained by human studies' typical failure to stratify by brewing method, roast, and dose - the same variables this review identifies as decisive for cardiovascular outcomes. At the same time, the null finding from Mendelian randomization analysis [17] is an essential caution, meaning that causality has not been definitively established for coffee's cardiovascular associations at the population level.

To sum up, if any general guidance can cautiously be drawn from current evidence, it favors filtered preparations, moderate intake (roughly three to four cups per day), and morning-type consumption, over unfiltered or espresso-style preparations, very high or erratic intake, or all-day drinking patterns. The clearest gap for future research is the very limited number of studies that jointly measure brewing method, roast degree, dose, and timing within a single cohort, and of randomized trials isolating individual coffee components (chlorogenic acid, trigonelline, cafestol/kahweol) against both lipid and inflammatory endpoints. Until such studies exist, statements about coffee's protective or harmful cardiovascular effects should be understood as recommendations about a particular way of preparing and consuming coffee.

Author contributions:

Conceptualization: Maria Windyga, Anna Sieczka, Joanna Makowska.

Methodology: Maria Windyga, Karolina Domańska, Jerzy Darmoń, Anna Sieczka.

Formal analysis: Maria Windyga, Karolina Domańska, Anna Sieczka, Aleksandra Papuga, Joanna Makowska, Natalia Piegza.

Investigation: Maria Windyga, Makary Hejduk, Aleksandra Papuga, Natalia Piegza, Anna Sieczka, Maria Sroka.

Data curation: Maria Windyga, Joanna Makowska, Jerzy Darmoń, Maria Sroka.

Writing rough preparation: Maria Windyga, Karolina Domańska, Jerzy Darmoń, Joanna Makowska, Anna Sieczka, Maria Sroka, Natalia Piegza, Aleksandra Papuga, Makary Hejduk.

Writing review and editing: Maria Windyga, Anna Sieczka, Karolina Domańska, Joanna Makowska, Jerzy Darmoń, Aleksandra Papuga.

Visualization: Maria Windyga, Joanna Makowska, Maria Sroka, Makary Hejduk.

Supervision: Maria Windyga.

All authors have read and agreed with the published version of manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: The authors confirm that the data supporting the findings of this study are available within the article's bibliography.

Conflict of interest: None declared.

Declaration of AI Use: Artificial intelligence (AI) was used only for language enhancement purposes, such as grammar correction and stylistic refinement.

References

1. Tverdal, A., Selmer, R., Cohen, J. M., & Thelle, D. S. (2020). Coffee consumption and mortality from cardiovascular diseases and total mortality: Does the brewing method matter?. European journal of preventive cardiology, 27(18), 1986–1993. https://doi.org/10.1177/2047487320914443
2. van Dam, R. M., Hu, F. B., & Willett, W. C. (2020). Coffee, caffeine, and health. New England Journal of Medicine, 383(4), 369–378. https://doi.org/10.1056/NEJMra1816604
3. Grioni, S., Agnoli, C., Sieri, S., Pala, V., Ricceri, F., Masala, G., Saieva, C., Panico, S., Mattiello, A., Chiodini, P., Tumino, R., Frasca, G., Iacoviello, L., de Curtis, A., Vineis, P., & Krogh, V. (2015). Espresso coffee consumption and risk of coronary heart disease in a large Italian cohort. PloS one, 10(5), e0126550. https://doi.org/10.1371/journal.pone.0126550

4. Domingues, D. S., Ramalho, J. C., & Partelli, F. L. (2023). Coffee—From plant to cup. Agronomy, 13(9), Article 2346. https://doi.org/10.3390/agronomy13092346
5. Ludwig, I. A., Mena, P., Calani, L., Cid, C., Del Rio, D., Lean, M. E., & Crozier, A. (2014). Variations in caffeine and chlorogenic acid contents of coffees: what are we drinking?. Food & function, 5(8), 1718–1726. https://doi.org/10.1039/c4fo00290c
6. Mendoza, M. F., Sulague, R. M., Posas-Mendoza, T., & Lavie, C. J. (2023). Impact of Coffee Consumption on Cardiovascular Health. Ochsner journal, 23(2), 152–158. https://doi.org/10.31486/toj.22.0073
7. Correâ, T. A., Rogero, M. M., Mioto, B. M., Tarasoutchi, D., Tuda, V. L., César, L. A., & Torres, E. A. (2013). Paper-filtered coffee increases cholesterol and inflammation biomarkers independent of roasting degree: A clinical trial. Nutrition, 29(7–8), 977–981. https://doi.org/10.1016/j.nut.2013.01.003
8. Miranda, A. M., Steluti, J., Fisberg, R. M., & Marchioni, D. M. (2017). Association between Coffee Consumption and Its Polyphenols with Cardiovascular Risk Factors: A Population-Based Study. Nutrients, 9(3), 276. https://doi.org/10.3390/nu9030276
9. Chieng, D., Canovas, R., Segan, L., Sugumar, H., Voskoboinik, A., Prabhu, S., Ling, L. H., Lee, G., Morton, J. B., Kaye, D. M., Kalman, J. M., & Kistler, P. M. (2022). The impact of coffee subtypes on incident cardiovascular disease, arrhythmias, and mortality: long-term outcomes from the UK Biobank. European journal of preventive cardiology, 29(17), 2240–2249. https://doi.org/10.1093/eurjpc/zwac189
10. Orrje, E., Fristedt, R., Rosqvist, F., Landberg, R., & Iggman, D. (2025). Cafestol and kahweol concentrations in workplace machine coffee compared with conventional brewing methods. Nutrition, Metabolism and Cardiovascular Diseases. Advance online publication. https://doi.org/10.1016/j.numecd.2025.103933
11. Sarriá, B., Sierra-Cinos, J. L., García-Diz, L., Martínez-López, S., Mateos, R., & Bravo-Clemente, L. (2020). Green/Roasted Coffee May Reduce Cardiovascular Risk in Hypercholesterolemic Subjects by Decreasing Body Weight, Abdominal Adiposity and Blood Pressure. Foods (Basel, Switzerland), 9(9), 1191. https://doi.org/10.3390/foods9091191
12. Frost-Meyer, N. J., & Logomarsino, J. V. (2012). Impact of coffee components on inflammatory markers: A review. Journal of Functional Foods, 4(4), 819–830. https://doi.org/10.1016/j.jff.2012.05.011

13. Ding, M., Bhupathiraju, S. N., Satija, A., van Dam, R. M., & Hu, F. B. (2014). Long-term coffee consumption and risk of cardiovascular disease: a systematic review and a dose-response meta-analysis of prospective cohort studies. Circulation, 129(6), 643–659. https://doi.org/10.1161/CIRCULATIONAHA.113.005925
14. Poole, R., Kennedy, O. J., Roderick, P., Fallowfield, J. A., Hayes, P. C., & Parkes, J. (2017). Coffee consumption and health: umbrella review of meta-analyses of multiple health outcomes. BMJ (Clinical research ed.), 359, j5024. https://doi.org/10.1136/bmj.j5024
15. Ruggiero, E., Di Castelnuovo, A., Costanzo, S., Persichillo, M., De Curtis, A., Cerletti, C., Donati, M. B., de Gaetano, G., Iacoviello, L., Bonaccio, M., & Moli-sani Study Investigators (2021). Daily Coffee Drinking Is Associated with Lower Risks of Cardiovascular and Total Mortality in a General Italian Population: Results from the Moli-sani Study. The Journal of nutrition, 151(2), 395–404. https://doi.org/10.1093/jn/nxaa365
16. Wang, X., Ma, H., Sun, Q., Li, J., Heianza, Y., Van Dam, R. M., Hu, F. B., Rimm, E., Manson, J. E., & Qi, L. (2025). Coffee drinking timing and mortality in US adults. European heart journal, 46(8), 749–759. https://doi.org/10.1093/eurheartj/ehae871
17. Yuan, S., Carter, P., Mason, A. M., Burgess, S., & Larsson, S. C. (2021). Coffee Consumption and Cardiovascular Diseases: A Mendelian Randomization Study. Nutrients, 13(7), 2218. https://doi.org/10.3390/nu13072218
18. Rendón, M. Y., Dos Santos Scholz, M. B., & Bragagnolo, N. (2018). Physical characteristics of the paper filter and low cafestol content filter coffee brews. Food research international (Ottawa, Ont.), 108, 280–285. https://doi.org/10.1016/j.foodres.2018.03.041
19. Perrone, D., Donangelo, R., Donangelo, C. M., & Farah, A. (2010). Modeling weight loss and chlorogenic acids content in coffee during roasting. Journal of agricultural and food chemistry, 58(23), 12238–12243. https://doi.org/10.1021/jf102110u
20. Moon, J. K., Yoo, H. S., & Shibamoto, T. (2009). Role of roasting conditions in the level of chlorogenic acid content in coffee beans: correlation with coffee acidity. Journal of agricultural and food chemistry, 57(12), 5365–5369. https://doi.org/10.1021/jf900012b

21. Bodar, V., Chen, J., Gaziano, J. M., Albert, C., & Djoussé, L. (2019). Coffee Consumption and Risk of Atrial Fibrillation in the Physicians' Health Study. Journal of the American Heart Association, 8(15), e011346. https://doi.org/10.1161/JAHA.118.011346
22. Cao, Y., Liu, X., Xue, Z., Yin, K., Ma, J., Zhu, W., Liu, F., Luo, J., & Sun, J. (2022). Association of Coffee Consumption With Atrial Fibrillation Risk: An Updated Dose-Response Meta-Analysis of Prospective Studies. Frontiers in cardiovascular medicine, 9, 894664. https://doi.org/10.3389/fcvm.2022.894664